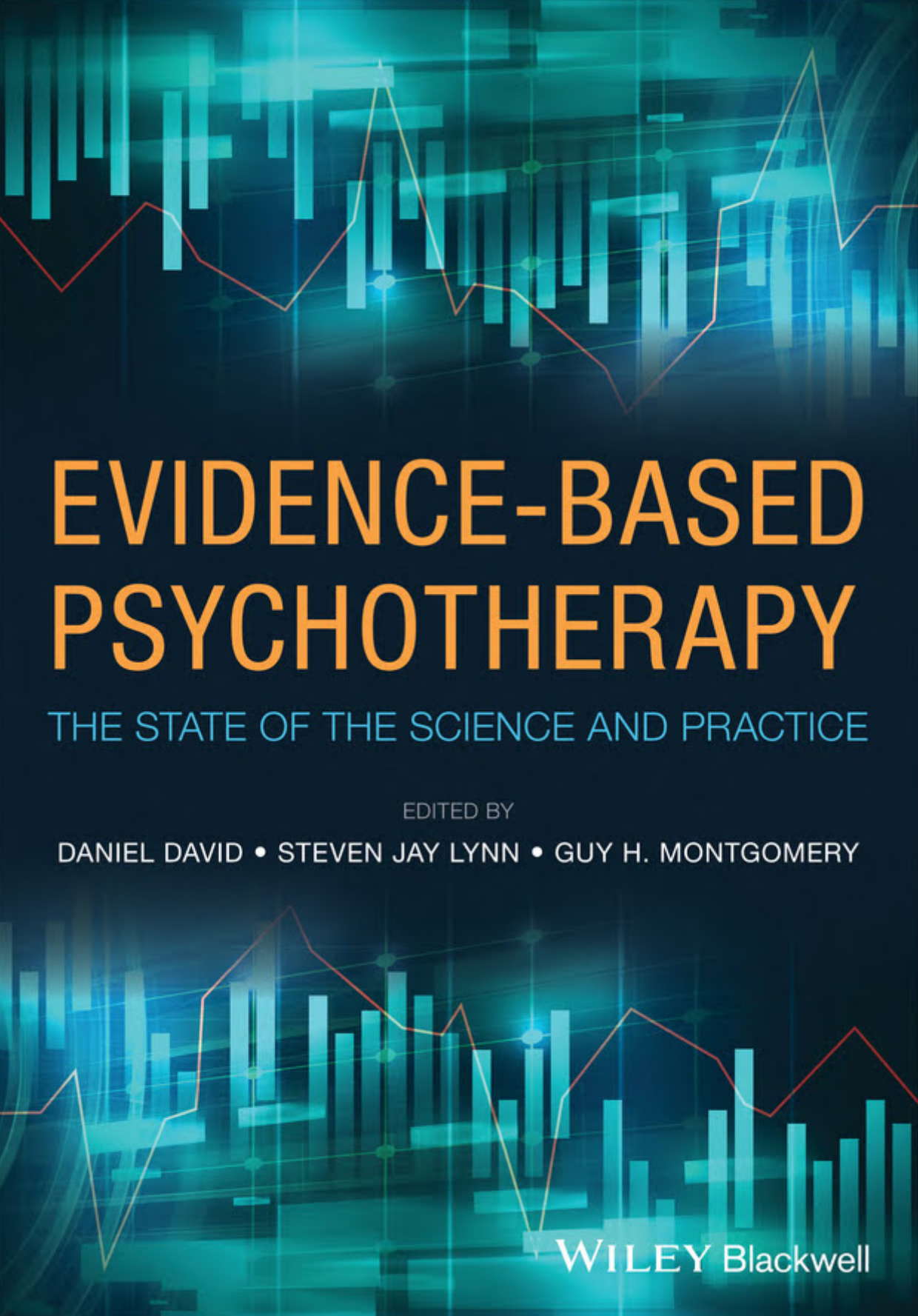
EVIDENCE-BASED PSYCHOTHERAPY
THE STATE OF THE SCIENCE AND PRACTICE
EDITED BY
DANIEL DAVID • STEVEN JAY LYNN • GUY H. MONTGOMERY
WILEY Blackwell

Evidence-Based Psychotherapy

Evidence-Based Psychotherapy

The State of the Science and Practice

Edited by

Daniel David, Steven Jay Lynn, and Guy H. Montgomery

WILEY Blackwell

This edition first published 2018

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

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

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

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

Library of Congress Cataloging-in-Publication Data

Names: David, Daniel, 1972– editor. | Lynn, Steven J., editor. | Montgomery, Guy, 1967– editor.
Title: Evidence-based psychotherapy: the state of the science and practice / edited by Daniel David, Steven Jay Lynn, Guy Montgomery.
Other titles: Evidence-based psychotherapy (David)
Description: First edition. | Hoboken, NJ, USA: Wiley, 2018. | Includes bibliographical references and index. |
Identifiers: LCCN 2017037695 (print) | LCCN 2017038625 (ebook) | ISBN 9781118625538 (pdf) | ISBN 9781118625583 (epub) | ISBN 9781118625521 (cloth)
Subjects: | MESH: Mental Disorders–therapy | Psychotherapy | Evidence-Based Practice
Classification: LCC RC480 (ebook) | LCC RC480 (print) | NLM WM 420 | DDC 616.89/14–dc23
LC record available at https://lccn.loc.gov/2017037695

Cover Design: Wiley
Cover Image: © monsitj/Gettyimages

Set in 10/12pt WarnockPro by Aptara Inc., New Delhi, India

10 9 8 7 6 5 4 3 2 1

Contents

List of Contributors *xvii*

1 An Introduction to the Science and Practice of Evidence-Based Psychotherapy: A Framework for Evaluation and a Way Forward *1*
Daniel David, Steven Jay Lynn, and Guy H. Montgomery
1.1 Evidence-Based Psychotherapies and Clinical Practice *1*
1.2 Classifying Psychotherapies: Tricky Business *3*
1.3 A New Evaluation Scheme for Psychotherapy: Efficacy and Mechanisms *5*
1.4 What We Aim to Accomplish *6*
1.5 Conclusions *6*
References *7*

2 Varieties of Psychotherapy for Major Depressive Disorder in Adults: An Evidence-Based Evaluation *11*
Daniel David, Ioana A. Cristea, and Aaron T. Beck
2.1 Description of the Disorder *11*
2.2 Classification of Psychotherapies According to David and Montgomery's (2011) Evaluative Framework *12*
2.2.1 Category I: Evidence-Based Psychotherapies *13*
2.2.1.1 Cognitive therapy *14*
2.2.2 Category II: Intervention-Driven Psychotherapies *17*
2.2.2.1 Interpersonal psychotherapy *17*
2.2.2.2 Behavioral therapies *18*
2.2.3 Category III: Theory-Driven Psychotherapies *20*
2.2.3.1 Cognitive bias modification: Concreteness training *21*
2.2.3.2 Rational emotive behavioral therapy *21*
2.2.4 Category IV: Investigational Psychotherapies *22*
2.2.4.1 Short-term psychodynamic psychotherapy *22*
2.2.4.2 "Third-wave" cognitive–behavioral psychotherapies *23*
2.2.4.3 Behavioral couple therapy *24*
2.2.5 Categories V, VI, VII, VIII, and IX *25*
2.3 Conclusions and Discussion *26*
2.3.1 Implications for Research *26*
2.3.2 Implications for Practice *27*
References *29*

3 Evidence-Based Psychological Interventions for Bipolar Disorder *37*
Aurora Szentágotai-Tătar and Daniel David
3.1 Overview of Bipolar Disorder *37*
3.2 Evidence-Based Interventions for Bipolar Disorder *39*
3.2.1 Category I: Evidence-Based Psychotherapies *40*
3.2.2 Category II: Intervention-Driven Psychotherapies *40*
3.2.3 Category III: Theory-Driven Psychotherapies *40*
3.2.4 Category IV: Investigational Psychotherapies *40*
3.2.4.1 Cognitive–behavioral therapy *40*
3.2.4.2 Mindfulness-based cognitive therapy *43*
3.2.4.3 Psychoeducation *44*
3.2.4.4 Family interventions *45*
3.2.4.5 Interpersonal and social rhythm therapy *49*
3.2.5 Categories V, VI, VII, VIII, and IX *50*
3.3 Conclusions and Future Directions *50*
References *53*

4 The Treatment of Panic Disorder and Phobias *63*
Barbara Depreeuw, Leigh A. Andrews, Sharon Eldar, and Stefan G. Hofmann
4.1 Panic Disorder *63*
4.1.1 Defining Features *63*
4.1.2 Treatments *64*
4.1.2.1 Category I: Therapeutically and theoretically well supported *64*
4.1.2.2 Category II: Therapeutically well supported and theoretically equivocal *66*
4.1.2.3 Category III: Therapeutically equivocal and theoretically well supported *66*
4.1.2.4 Category IV: Therapeutically and theoretically equivocal *69*
4.1.2.5 Category V: Therapeutically well supported and theoretically contradictory *70*
4.1.2.6 Category VI: Therapeutically contradictory and theoretically well supported *70*
4.1.2.7 Category VII: Therapeutically equivocal and theoretically contradictory *70*
4.1.2.8 Category VIII: Therapeutically contradictory and theoretically equivocal *70*
4.1.2.9 Category IX: Therapeutically and theoretically contradictory *71*
4.2 Social Anxiety Disorder *71*
4.2.1 Defining Features *71*
4.2.2 Treatments *72*
4.2.2.1 Category I: Therapeutically and theoretically well supported *72*
4.2.2.2 Category II: Therapeutically well supported and theoretically equivocal *74*

4.2.2.3 Category III: Therapeutically equivocal and theoretically well supported *74*
4.2.2.4 Category IV: Therapeutically and theoretically equivocal *75*
4.2.2.5 Category V: Therapeutically well supported and theoretically contradictory *77*
4.2.2.6 Category VI: Therapeutically contradictory and theoretically well supported *77*
4.2.2.7 Category VII: Therapeutically equivocal and theoretically contradictory *77*
4.2.2.8 Category VIII: Therapeutically contradictory and theoretically equivocal *78*
4.2.2.9 Category IX: Therapeutically and theoretically contradictory *78*
4.3 Specific Phobia *78*
4.3.1 Defining Features *78*
4.3.2 Treatments *79*
4.3.2.1 Category I: Therapeutically and theoretically well supported *79*
4.3.2.2 Category II: Therapeutically well supported and theoretically equivocal *81*
4.3.2.3 Category III: Therapeutically equivocal and theoretically well supported *81*
4.3.2.4 Category IV: Therapeutically and theoretically equivocal *81*
4.3.2.5 Category V: Therapeutically well supported and theoretically contradictory *81*
4.3.2.6 Category VI: Therapeutically contradictory and theoretically well supported *81*
4.3.2.7 Category VII: Therapeutically equivocal and theoretically contradictory *81*
4.3.2.8 Category VIII: Therapeutically contradictory and theoretically equivocal *81*
4.3.2.9 Category IX: Therapeutically and theoretically contradictory *81*
4.4 Implications for Research *81*
4.5 Implications for Practice *82*
4.6 Conclusions *82*
References *83*

5 The Psychological Treatment of Generalized Anxiety Disorder *95*
Hanna McCabe-Bennett, Katie Fracalanza, and Martin M. Antony
5.1 Features and Prevalence of Generalized Anxiety Disorder *95*
5.2 Review of Theory and Evidence Supporting GAD Psychotherapies *96*
5.2.1 Category I *96*
5.2.1.1 Intolerance of uncertainty model of GAD and intolerance of uncertainty therapy *96*

5.2.1.2 Metacognitive model of GAD and metacognitive therapy *98*
5.2.1.3 Acceptance-based theory and acceptance-based behavior therapy for GAD *100*
5.2.2 Category II *102*
5.2.2.1 Applied relaxation *102*
5.2.2.2 Transdiagnostic model *103*
5.2.3 Category III *104*
5.2.3.1 Emotion dysregulation model of GAD and emotion regulation therapy *104*
5.2.3.2 Other mindfulness interventions for GAD *105*
5.2.3.3 Cognitive/contrast avoidance theories of GAD and interpersonal/emotional processing therapy *106*
5.2.4 Category IV *109*
5.2.4.1 Psychodynamic model and treatments *109*
5.3 Implications for Research *111*
5.4 Implications for Practice *111*
5.5 Conclusions *111*
Notes *112*
References *112*

6 The Treatment of Obsessive–Compulsive Disorder *123*
Lindsey M. Collins, Laura B. Bragdon, and Meredith E. Coles
6.1 Exposure and Ritual Prevention *125*
6.1.1 Support for Treatment *125*
6.1.2 Support for Theory *127*
6.1.3 Summary *127*
6.2 Cognitive Therapy *128*
6.2.1 Support for Treatment *128*
6.2.2 Support for Theory *129*
6.2.3 Summary *130*
6.3 Cognitive–Behavioral Therapy *131*
6.3.1 Support for Treatment *131*
6.3.2 Support for Theory *131*
6.3.3 Summary *132*
6.4 Acceptance and Commitment Therapy *132*
6.4.1 Support for Treatment *133*
6.4.2 Support for Theory *133*
6.4.3 Summary *134*
6.5 Metacognitive Therapy *134*
6.5.1 Support for Treatment *135*
6.5.2 Support for Theory *135*
6.5.3 Summary *136*
6.6 Attention Bias Modification Training *136*
6.6.1 Support for Treatment *138*
6.6.2 Support for Theory *138*
6.6.3 Summary *138*
6.7 Interpretation Bias Modification Training *139*

6.7.1 Support for Treatment *139*
6.7.2 Support for Theory *140*
6.7.3 Summary *140*
6.8 Eye Movement Desensitization and Reprocessing Therapy *140*
6.8.1 Support for Treatment *141*
6.8.2 Support for Theory *141*
6.8.3 Summary *142*
6.9 Implications for Research *142*
6.10 Implications for Practice *145*
6.11 Conclusions *146*
Note *146*
References *146*

7 Evidence-Based Practice for Posttraumatic Stress Disorder *157*
B. Christopher Frueh, Anouk L. Grubaugh, Alok Madan, Sandra M. Neer, Jon D. Elhai, and Deborah C. Beidel
7.1 Overview *157*
7.1.1 Clinical Features *159*
7.1.2 Diagnostic Considerations *160*
7.1.3 Epidemiology *160*
7.1.4 Psychological and Biological Assessment *161*
7.2 Etiological Pathways and Causal Mechanisms *161*
7.2.1 Behavioral and Molecular Genetics *161*
7.2.2 Neuroanatomy and Neurobiology *162*
7.2.3 Learning, Modeling, and Life Events *163*
7.2.4 Cognitive Influences *163*
7.2.5 Sex and Racial/Ethnic Considerations *164*
7.2.6 Course, Prognosis, and Treatment *164*
7.3 Evidence-Based Practices *166*
7.3.1 Psychosocial Treatments for PTSD *166*
7.3.1.1 Exposure therapy *166*
7.3.1.2 Cognitive therapy *170*
7.3.1.3 Summary of psychotherapy for PTSD *174*
7.3.2 Psychopharmacological Therapy for PTSD *175*
7.3.2.1 Neuromodulation and neurostimulation for PTSD *175*
7.3.2.2 Variability in treatment response *176*
7.3.3 Interventions for PTSD and Comorbid Disorders *176*
7.3.3.1 Substance abuse *176*
7.3.3.2 Severe mental illness *177*
7.3.4 Telemedicine for PTSD *177*
7.4 Conclusions *178*
References *178*

8 Evidence-Based Psychological Interventions for Eating Disorders *189*
Diana M. Cândea, Daniel David, and Aurora Szentágotai-Tătar
8.1 Description of the Disorders *189*
8.2 David and Montgomery's (2011) Evaluative Framework *190*
8.3 Classification of Psychological Interventions for Anorexia Nervosa *190*

8.3.1 Category I: Evidence-Based Psychotherapies *190*
8.3.2 Category II: Intervention-Driven Psychotherapies *190*
8.3.3 Category III: Theory-Driven Psychotherapies *191*
8.3.4 Category IV: Investigational Psychotherapies *191*
8.3.4.1 Family-based therapy *191*
8.3.4.2 Cognitive–behavioral therapy *193*
8.3.4.3 Cognitive analytical therapy *194*
8.3.4.4 Interpersonal therapy *195*
8.3.4.5 Maudsley model of anorexia nervosa treatment for adults (MANTRA) *195*
8.3.4.6 Ego-oriented individual therapy *195*
8.3.4.7 Focal psychodynamic therapy *196*
8.3.4.8 Behavioral therapy *196*
8.3.4.9 Nutritional counseling *196*
8.3.5 Categories V, VI, VII, VIII, and IX *196*
8.4 Classification of Psychological Interventions for Binge Eating Disorder *197*
8.4.1 Category I: Evidence-Based Psychotherapies *197*
8.4.1.1 Cognitive–behavioral therapy for binge eating disorder (CBT-BED) *197*
8.4.1.2 Guided self-help cognitive–behavioral therapy *198*
8.4.1.3 Pure self-help cognitive–behavioral therapy *199*
8.4.2 Category II: Intervention-Driven Psychotherapies *199*
8.4.3 Category III: Theory-Driven Psychotherapies *199*
8.4.4 Category IV: Investigational Psychotherapies *200*
8.4.4.1 Interpersonal psychotherapy *200*
8.4.4.2 Behavioral weight control *200*
8.4.4.3 Dialectical behavior therapy *201*
8.4.5 Categories V, VI, VII, VIII, and IX *202*
8.5 Classification of Psychological Interventions for Bulimia Nervosa *202*
8.5.1 Category I: Evidence-Based Psychotherapies *202*
8.5.1.1 Cognitive–behavioral therapy *202*
8.5.1.2 Guided self-help cognitive–behavioral therapy *203*
8.5.1.3 Pure self-help cognitive–behavioral therapy *204*
8.5.2 Category II: Intervention-Driven Psychotherapies *205*
8.5.2.1 Focal supportive psychotherapy *205*
8.5.3 Category III: Theory-Driven Psychotherapies *205*
8.5.4 Category IV: Investigational Psychotherapies *205*
8.5.4.1 Interpersonal therapy *205*
8.5.4.2 Dialectical behavioral therapy *206*
8.5.4.3 Behavior therapy *206*
8.5.4.4 Family-based therapy *206*
8.5.4.5 Dietary counseling *207*
8.5.4.6 Psychodynamically oriented supportive psychotherapy *207*
8.5.4.7 Psychoanalytic psychotherapy *208*
8.5.5 Categories V, VI, VII, VIII, and IX *208*
8.6 Discussion and Conclusions *208*

8.6.1 Implications for Research *209*
8.6.2 Implications for Practice *209*
References *210*

9 Evidence-Based Treatment for Alcohol Use Disorders: A Review Through the Lens of the Theory × Efficacy Matrix *219*
James MacKillop, Monika Stojek, Lauren VanderBroek-Stice, and Max M. Owens
9.1 A Brief History of Alcohol Use Disorder Treatment *220*
9.1.1 Psychiatric Nosology of Alcohol Use Disorders *221*
9.1.2 Epidemiology of Alcohol Use Disorders *222*
9.2 Treatment Reviews *224*
9.2.1 Reinforcement-Based Treatments *224*
9.2.2 Motivational Interviewing *227*
9.2.3 Cognitive–Behavioral Therapy *229*
9.2.4 Behavioral Couples Therapy *231*
9.2.5 Alcoholics Anonymous *235*
9.2.6 Confrontational Interventions *237*
9.3 Implications for Research and Practice *239*
9.4 Conclusions *240*
Acknowledgments *240*
Note *240*
References *241*

10 Psychotherapeutic Treatments for Male and Female Sexual Dysfunction Disorders *253*
Jessica C. Emanu, Isabelle Avildsen, and Christian J. Nelson
10.1 Treatments for Sexual Dysfunction *254*
10.2 Category I *254*
10.2.1 Cognitive–Behavioral Therapy *254*
10.2.1.1 Evidence for therapy *255*
10.2.1.2 Theory of disorder *259*
10.2.1.3 Summary *260*
10.3 Category III *260*
10.3.1 Sex Therapy *260*
10.3.1.1 Evidence for therapy *261*
10.3.1.2 Theory of disorder *262*
10.3.1.3 Summary *262*
10.4 Category IV *262*
10.4.1 Behavioral Therapies *262*
10.4.1.1 Evidence for Therapy *263*
10.4.1.2 Theory of Disorder *264*
10.4.1.3 Summary *264*
10.5 Category VIII *265*
10.5.1 Psychoeducation *265*
10.5.1.1 Evidence for Therapy *265*
10.5.1.2 Theory of Disorder *265*
10.5.1.3 Summary *265*

10.6 Other Therapies *266*
10.7 Implications for Research *266*
10.8 Implications for Practice *267*
10.9 Conclusions *268*
References *268*

11 The Psychological Treatment of Psychopathy: Theory and Research *271*
Lisa K. Hecht, Robert D. Latzman, and Scott O. Lilienfeld
11.1 Conceptualizing and Measuring Psychopathy *272*
11.2 Is Psychopathy Treatable? A History of Negative Opinion *275*
11.3 Methodological Issues in Treatment Investigations *276*
11.3.1 Lack of Contemporary, Empirically Rigorous Studies *276*
11.3.2 Lack of Theoretically Grounded Treatments *277*
11.3.3 Lack of Consensus Regarding Conceptualization and Assessment of Psychopathy *278*
11.4 Evaluation of Psychopathy Treatments *278*
11.4.1 Cognitive–Behavioral Approaches *279*
11.4.2 Therapeutic Communities *283*
11.4.3 Psychodynamic Approaches *285*
11.5 Implications for Research *287*
11.6 Implications for Clinical Practice *289*
11.7 Conclusions *290*
References *290*

12 The Treatment of Borderline Personality Disorder *299*
Lorie A. Ritschel, Colleen M. Cowperthwait, Lindsay M. Stewart, and Noriel E. Lim
12.1 Treatments for Borderline Personality Disorder *300*
12.2 Category II *301*
12.2.1 Dialectical Behavior Therapy *301*
12.2.1.1 Evidence for therapy *302*
12.2.1.2 Theory of the disorder *303*
12.2.1.3 Theory of change *304*
12.2.1.4 Summary *305*
12.2.2 Mentalization-Based Therapy *305*
12.2.2.1 Evidence for therapy *306*
12.2.2.2 Theory of the disorder *307*
12.2.2.3 Theory of change *308*
12.2.2.4 Summary *308*
12.3 Category IV *308*
12.3.1 Schema-Focused Therapy *308*
12.3.1.1 Evidence for therapy *309*
12.3.1.2 Theory of the disorder *310*
12.3.1.3 Theory of change *311*
12.3.1.4 Summary *311*
12.3.2 Cognitive–Behavioral Therapy *312*
12.3.2.1 Evidence for therapy *312*
12.3.2.2 Theory of the disorder *312*

12.3.2.3 Theory of change *313*
12.3.2.4 Summary *313*
12.3.3 Transference-Focused Psychotherapy *313*
12.3.3.1 Evidence for therapy *313*
12.3.3.2 Theory of the disorder *314*
12.3.3.3 Theory of change *315*
12.3.3.4 Summary *315*
12.4 Other Therapies *315*
12.5 Implications for Research *316*
12.6 Implications for Practice *317*
12.7 Conclusions *319*
References *319*

13 The Treatment of Dissociation: An Evaluation of Effectiveness and Potential Mechanisms *329*
Reed Maxwell, Harald Merckelbach, Scott O. Lilienfeld, and Steven Jay Lynn
13.1 A Review of the DSM-5 Dissociative Disorders *331*
13.2 Treatments for Depersonalization/Derealization Disorder *332*
13.3 Treatments for Dissociative Identity Disorder *337*
13.4 Treatments for Dissociative Fugue and Dissociative Amnesia *346*
13.5 Innovative Treatments for Dissociation *347*
13.5.1 Sleep-Related Interventions *348*
13.5.2 Emotion Regulation Interventions *349*
13.6 Conclusions *350*
References *351*

14 Psychotherapy for Schizophrenia-Spectrum Disorders *363*
Stacy Ellenberg, Steven Jay Lynn, and Gregory P. Strauss
14.1 Treatment for Schizophrenia *365*
14.2 Category II *366*
14.2.1 Cognitive–Behavioral Therapy *366*
14.2.1.1 Components of the therapy *367*
14.2.1.2 Evidence for the therapy *368*
14.2.1.3 Theory of the disorder *375*
14.2.1.4 Theory of change *378*
14.2.1.5 Summary *380*
14.2.2 Social Skills and Social Cognition Training *380*
14.2.2.1 Evidence for the therapy *381*
14.2.2.2 Theory of the disorder *384*
14.2.2.3 Theory of change *384*
14.2.2.4 Summary *385*
14.3 Category IV *385*
14.3.1 Metacognitive Therapy *385*
14.3.1.1 Evidence for the therapy *386*
14.3.1.2 Theory of the disorder *390*
14.3.1.3 Theory of change *390*
14.3.1.4 Summary *391*

14.4 Other Therapies *391*
14.5 Implications for Research *392*
14.6 Implications for Clinical Practice *393*
14.7 Conclusion *393*
References *394*

15 Psychotherapy and Autism Spectrum Disorder: Conceptual and Pragmatic Challenges *407*
Raymond G. Romanczyk, Rachel N. S. Cavalari, and Jennifer M. Gillis
15.1 History of the Diagnosis of Autism *407*
15.2 Heterogeneity *408*
15.3 Core Characteristics *408*
15.4 Prevalence *409*
15.5 Etiology and Theoretical Approach: The Context of Etiological Complexity *409*
15.6 Defining Psychotherapy in the Context of Autism Spectrum Disorder *410*
15.7 Evidence-Based Treatment Review *414*
15.7.1 Results *420*
15.7.2 Framework Categorization *420*
15.7.2.1 Worst evaluation category *420*
15.7.2.2 Best evaluation category *420*
15.7.2.3 Mixed evaluation categories *423*
15.7.2.4 Empty categories *426*
15.8 Discussion *427*
15.8.1 Implications for Practice and Research *427*
15.9 Conclusion *428*
Notes *429*
References *429*

16 Varieties of Psychotherapy for Attention-Deficit Hyperactivity Disorder: An Evidence-Based Evaluation *435*
Anca Dobrean, Costina R. Păsărelu, and Manfred Döpfner
16.1 Description of the Disorder *435*
16.2 Classification of Psychotherapies *437*
16.2.1 Category I: Evidence-Based Psychotherapies *437*
16.2.1.1 Combined treatment *437*
16.2.2 Category II: Intervention-Driven Psychotherapies *439*
16.2.2.1 Cognitive–behavioral therapy *439*
16.2.2.2 Psychoeducation *441*
16.2.2.3 Organizational skills training *442*
16.2.3 Category III: Theory-Driven Psychotherapies *444*
16.2.3.1 Behavioral parenting training *444*
16.2.4 Category IV: Investigational Psychotherapies *446*
16.2.4.1 Social skills training *446*
16.2.4.2 Cognitive training interventions *447*
16.2.4.3 Neurofeedback training *448*
16.2.4.4 Mindfulness-based interventions *450*
16.2.5 Categories V, VI, VII, VIII, and IX *451*

16.3 Conclusions and Discussion *452*
16.3.1 Implications for Research *452*
16.3.2 Implications for Practice *453*
References *453*

17 The Treatment of Insomnia *465*
Elisabeth Hertenstein, Bogdan Voinescu, and Dieter Riemann
17.1 Description of the Disorder *465*
17.1.1 Definitions *465*
17.1.2 Subtypes *466*
17.1.3 Assessment *466*
17.1.4 Prevalence and Risk Factors *466*
17.2 Psychological Theories and Mechanisms of Change *467*
17.2.1 Classical Conditioning *467*
17.2.2 Behavioral Model *468*
17.2.3 Hyperarousal Model *469*
17.2.4 Attention Intention Effort Model *470*
17.2.5 Cognitive Model *472*
17.2.6 Metacognitive Model *474*
17.2.7 Mediators of Treatment Effects *474*
17.3 Therapeutic Packages *475*
17.3.1 Cognitive–Behavioral Therapy *475*
17.3.2 Brief Behavioral Treatment *476*
17.3.3 Computerized Cognitive–Behavioral Therapy *477*
17.3.4 Sleep Hygiene Education *480*
17.3.5 Stimulus Control *483*
17.3.6 Sleep Restriction *484*
17.3.7 Relaxation *485*
17.3.8 Cognitive Therapy *486*
17.3.9 Paradoxical Intention *486*
17.3.10 Intensive Sleep Retraining *487*
17.3.11 Mindfulness *487*
17.3.12 Biofeedback *488*
17.4 Implications for Research and Practice *489*
17.5 Conclusions *496*
References *496*

18 The Scientific Status of Evidence-Based Psychotherapies: Concluding Thoughts *507*
Daniel David, Steven Jay Lynn, and Guy H. Montgomery
References *511*

Index *513*

List of Contributors

Leigh A. Andrews is a doctoral student at the University of Delaware. He graduated *cum laude* from Bowdoin College in 2015 and worked for 2 years as a research coordinator in the Psychotherapy and Emotion Research Laboratory under Dr. Stefan G. Hofmann. He is interested in mechanisms of treatment in emotional disorders, particularly in novel forms of treatment. He is particularly interested in studying how some transdiagnostic factors, such as negative affect, emotional reactivity, and coping strategies including suppression, interact with treatment mechanisms.

Martin M. Antony is Professor of Psychology at Ryerson University in Toronto. He also holds faculty appointments in the Department of Psychiatry at the University of Toronto and the Department of Psychiatry and Behavioural Neurosciences at McMaster University. His expertise is in the area of anxiety and related disorders (e.g., panic disorder, social anxiety disorder, phobias, obsessive–compulsive disorder) as well as perfectionism.

Isabelle Avildsen is an expert in the field of sexual disorder at the Graduate Center, City University of New York, approaching the disorders in an evidence-based paradigm.

Aaron T. Beck is a Professor Emeritus in the Department of Psychiatry at the University of Pennsylvania. He is the father of cognitive therapy and the grandfather of cognitive–behavioral therapy. Professor Beck is considered one of the "Americans in history who shaped the face of American psychiatry" and one of the most influential psychotherapists of all time (e.g., by *American Psychologist*, 1998). Professor Beck is a key world expert in the in the theory, research, and treatment of depression.

Deborah C. Beidel is Trustee Chair and Pegasus Professor of Psychology and Medical Education at the University of Central Florida (UCF), where she is also Director of UCF RESTORES, a clinical research center dedicated to anxiety, stress, and trauma. Her recent work focuses on developing effective treatments for PTSD for veterans, active duty personnel, and first responders, using technology to enhance effective treatments for use in standard clinical practice.

Laura B. Bragdon is a graduate student in the Coles lab at the Department of Psychology, Binghamton University. She received her BS in psychology, with a minor in studio arts/art history, from Trinity College in 2008, and holds an MA in psychology from

Columbia University. Before joining the Coles lab, she worked at the Anxiety Disorders Center at Hartford Hospital's Institute of Living as a full-time research assistant. Her areas of research interest focus on anxiety disorders and obsessive–compulsive disorder, specifically the varying mechanisms underlying the motivational domains of incompleteness and harm avoidance, the mediators and moderators involved in the treatment of anxiety disorders, and dissemination of evidence-based treatments for anxiety disorders more broadly.

Diana M. Cândea is an Assistant Professor in the Department of Clinical Psychology and Psychotherapy, Babeş-Bolyai University, and a member of the International Institute for the Advanced Studies of Psychotherapy and Applied Mental Health (Cluj-Napoca, Romania). Her expertise is related to self-conscious emotions, emotion regulation, and evidence-based psychotherapies.

Rachel N. S. Cavalari is Director of Services of the Children's Unit for Treatment and Evaluation at the Institute for Child Development and adjunct Assistant Professor in the Department of Psychology at Binghamton University. Her expertise is related to assessment, diagnosis, and treatment approaches for autism spectrum disorders, child safety and caregiver supervision, and staff supervision practices.

Meredith E. Coles is Professor of Psychology at Binghamton University and has served as the director of the Binghamton Anxiety Clinic since 2004. She is a fellow of the Association for the Advancement of Behavioral Therapy. Her expertise is in the nature and treatment of obsessive–compulsive disorder and anxiety disorders in both children and adults. Dr. Coles' work spans basic mechanisms (e.g., information-processing biases, circadian rhythms) to treatment utilization (e.g., mental health literacy).

Lindsey M. Collins is a graduate student in the Coles lab in the Department of Psychology, Binghamton University. Her current areas of interest include the etiology of obsessive–compulsive disorder (OCD), cognitive control deficits in OCD, and mechanisms of cognitive–behavioral therapy (CBT) for anxiety. Lindsey is currently involved in research projects investigating the development of OCD-related beliefs in children and the relation between electroencephalography indices, cognitive control, and OCD symptoms. Lindsey's clinical interests include CBT for child anxiety and behavior disorders. She is currently a clinician in the Binghamton Anxiety Clinic and the Binghamton Psychological Clinic. She interns twice a week at the Children's Home of Wyoming Conference.

Colleen M. Cowperthwait is a Clinical Associate and Fellow in the Clinical Psychology Fellowship Program in the Department of Psychiatry and Behavioral Sciences at Duke University Medical Center. She has clinical and research experience in cognitive–behavioral therapy and dialectical behavioral therapy, and studies how to adapt and implement evidence-based treatment programs. She is especially dedicated to adaptations of dialectical behavioral therapy for teens, college students, and young adults.

Ioana A. Cristea is Associate Professor in the Department of Clinical Psychology and Psychotherapy, Babeş-Bolyai University. Her research focuses on critically appraising

the efficacy of psychological and pharmacological interventions for mental disorders, as well as issues related to transparency and rigor in clinical research.

Daniel David is Professor of Clinical Cognitive Sciences at Babeş-Bolyai University, Director for Research at the Albert Ellis Institute, Adjunct Professor at the Icahn School of Medicine at Mount Sinai, and Director of the International Institute for the Advanced Studies of Psychotherapy and Applied Mental Health. His expertise is related to clinical cognitive neurogenetic sciences and evidence-based psychotherapies.

Barbara Depreeuw is based at the Department of Psychological and Brain Sciences, Boston University, and is an internationally trained clinical psychologist with wide clinical experience. She has worked at various universities and in clinical settings in the United States (University of California, Los Angeles; Boston University), the Netherlands, and Belgium (The Human Link; University of Leuven). She has over 10 years of experience in primary care, clinic, and corporate settings. Her main expertise is related to cognitive–behavioral therapy for anxiety disorders, work-related stress, and traumatic stress.

Anca Dobrean is Professor in the Department of Clinical Psychology and Psychotherapy at Babeş-Bolyai University. Her main research interests are related to evidence-based assessment and treatment of children's and adolescents' emotional and behavioral problems.

Manfred Döpfner is Professor of Psychotherapy in the Department of Child and Adolescent Psychiatry and Psychotherapy, Medical Faculty of the University of Cologne, Germany. He is a supervisor in behavior therapy and Director of the School of Child and Adolescent Cognitive Behavior Therapy at the University of Cologne and of the Institute of Clinical Child Psychology of the Christoph-Dornier-Foundation for Clinical Psychology at the University of Cologne. He received the German Psychology Prize for his scientific work in 2005.

Sharon Eldar is based at the Department of Psychological and Brain Sciences, Boston University, and is a clinical psychologist with vast experience as a clinical therapist, specializing in anxiety disorders. She received her PhD from Tel-Aviv University and was a postdoctoral fellow at Boston University. Her research is focused on the development of new therapeutic methods to treat anxiety and related disorders. These methods include attention bias modification, and enhancement of existing cognitive–behavioral therapy via positive affect training. In her clinical work, she works with all age groups and uses integrative methods in treating a wide range of mental disorders.

Jon D. Elhai is Professor in the Department of Psychology and in the Department of Psychiatry at the University of Toledo in Toledo, Ohio. His areas of research include posttraumatic stress disorder and cyberpsychology.

Stacy Ellenberg is a clinical psychology graduate student in the Department of Psychology at Binghamton University. Her research interests include technology-based interventions and psychosis.

Jessica C. Emanu is based at Long Island University and is an expert in the field of sexual disorders, approaching them from an evidence-based paradigm.

Katie Fracalanza is a clinical instructor in the Department of Psychiatry and Behavioral Neurosciences at Stanford University. Her areas of expertise include cognitive–behavioral therapy, exposure and response prevention, and other empirically supported treatments for adults with mood and anxiety disorders. She is actively involved in research and in teaching psychotherapy to graduate students and psychiatry residents.

B. Christopher Frueh is Professor in the Department of Psychology at the University of Hawaii. His areas of research include posttraumatic stress disorder among combat veterans, and he also works extensively with retired and active duty members of the military special operations community.

Jennifer M. Gillis is Associate Professor and a member of the Clinical Psychology Faculty at Binghamton University. She is Codirector of the Institute for Child Development and Adjunct Assistant Professor of Psychiatry at SUNY Upstate Medical University. Her expertise is related to evidence-based treatment approaches for autism spectrum disorders throughout the lifespan, applied behavior analysis, and organizational behavior management.

Anouk L. Grubaugh is Professor in the Department of Psychiatry at the Medical University of South Carolina and a staff psychologist at the Veterans Affairs Medical Center in Charleston, South Carolina, USA. Her areas of research include posttraumatic stress disorder among combat veterans and people with severe mental illnesses.

Lisa K. Hecht is a doctoral student in clinical neuropsychology in the Department of Psychology at Georgia State University. Her research interests focus on the relationship between executive functioning, social cognition, and psychopathy.

Elisabeth Hertenstein is a psychologist and Research Associate at the University Medical Center Freiburg and member of the workgroup "Sleep, Insomnia and Mental Disorders." She is a cognitive–behavioral therapist in training and currently works as a clinical psychologist at the sleep laboratory of the University Medical Center Freiburg.

Stefan G. Hofmann is Professor of Psychology in the Department of Psychological and Brain Sciences at Boston University. He has been the president of numerous national and international cognitive–behavioral therapy associations and has been the editor of various professional journals, including *Cognitive Therapy and Research*. His research focuses on the mechanism of treatment change, translating discoveries from neuroscience into clinical applications, emotion regulation, and cultural expressions of psychopathology.

Robert D. Latzman is an Associate Professor and Associate Director of Clinical Training in the Department of Psychology at Georgia State University. His research focuses on characterizing neurobehavioral mechanisms that underlie the development and

persistence of psychopathological behaviors in both human and nonhuman primates, particularly externalizing-related behaviors (e.g., aggression, substance use, psychopathy).

Scott O. Lilienfeld is Samuel Candler Dobbs Professor of Psychology at Emory University and Visiting Professor at the University of Melbourne. His research focuses on personality disorders (especially psychopathy), dissociative disorders, psychiatric classification, pseudoscience in psychology, evidence-based practice, and the application of scientific thinking to psychology.

Noriel E. Lim is Assistant Professor of Psychiatry and Behavioral Sciences at Emory University School of Medicine, where he directs Emory's Child, Adolescent, and Young Adult Pre-doctoral Internship Training Program as well as the children's anxiety treatment program. He also serves on the Board of Convention Affairs for the American Psychological Association and on the Board of Directors for the Asian American Psychological Association.

Steven Jay Lynn is a Distinguished Professor of Psychology at Binghamton University, a licensed clinical psychologist, and the editor of *Psychology of Consciousness: Theory, Research, and Practice*. He has published extensively in the areas of psychopathology, psychotherapy, memory, hypnosis and consciousness, and trauma.

James MacKillop is the Peter Boris Chair in Addictions Research, Director of the Peter Boris Centre for Addictions Research, and Professor of Psychiatry and Behavioural Neurosciences at McMaster University/St. Joseph's Healthcare Hamilton. Dr. MacKillop is also a Senior Scientist at Homewood Research Institute.

Alok Madan is an Associate Professor in the Menninger Department of Psychiatry and Behavioral Sciences at the Baylor College of Medicine, and McNair Scholar and Senior Psychologist at the Menninger Clinic. His clinical and research interests include mental health quality and outcomes, psychosomatic and behavioral medicine, and minimally invasive neuromodulation for neuropsychiatric disorders.

Reed Maxwell is based at the Psychiatry Department, Weil-Cornell Medical College, and is an expert in clinical, health, and positive psychology, working in the framework of the evidence-based paradigm.

Hanna McCabe-Bennett is a graduate student at Ryerson University in Toronto. She is currently completing her predoctoral internship at St. Joseph's Healthcare Hamilton with rotations in anxiety disorders, dialectical behavior therapy, and neuropsychology. Her expertise is in the area of anxiety and related disorders and hoarding.

Harald Merckelbach is Professor of Psychology and Law in the Departments of Forensic Psychology and Clinical Psychological Science, Faculty of Psychology and Neuroscience, Maastricht University. His expertise is related to clinical psychology and law and he has been awarded the "Nobel Prize for Psychology."

Guy H. Montgomery is an Associate Professor and Director of the Center for Behavioral Oncology at the Icahn School of Medicine at Mount Sinai. Dr. Montgomery is also a licensed clinical psychologist, specializing in behavioral medicine. He has published more than 100 peer-reviewed empirical articles and has received research funding from the National Cancer Institute and the American Cancer Society.

Sandra M. Neer is a clinical psychologist on the faculty of the Psychology Department at the University of Central Florida (UCF). She is Director of Clinical Services at UCF RESTORES, a clinical research center for trauma. Her research focuses on the outcome of evidence-based treatments for posttraumatic stress disorder in combat veterans and first responders.

Christian J. Nelson is a clinical psychologist in the Department of Psychiatry and Behavioral Sciences at Memorial Sloan Kettering Cancer Center. He specializes in the treatment of patients with genitourinary malignancies and older patients with cancer. As the psychological liaison to the genitourinary and sexual medicine services, he helps men experiencing treatment-related sexual dysfunction, and their partners, optimize intimacy before, during, and after treatment. He also works with the geriatric disease management team in the hospital, providing psychological support for patients aged 65 and over coping with the combined issues of cancer and aging.

Max M. Owens is a doctoral candidate in the Department of Psychology at the University of Georgia.

Costina R. Păsărelu is Assistant Professor at Babeș-Bolyai University in the Department of Clinical Psychology and Psychotherapy and a member of the International Institute for the Advanced Studies of Psychotherapy and Applied Mental Health. Her expertise is related to evidence-based assessment and interventions for children and adolescents. Her main research interests are transdiagnostic treatments delivered via technology for youth populations and parents.

Dieter Riemann has been tenured Professor of Clinical Psychophysiology at the University Medical Centre Freiburg since 1993. He is also head of the Department of Clinical Psychology at the Centre for Mental Disorders, and since 2015 he has been a Visiting Professor at the University of Oxford (Nuffield Department of Clinical Neurosciences). He served as a member of a DSM-5 workgroup for the American Psychiatric Association and is Editor in Chief of the *Journal of Sleep Research*. His research interests encompass the etiology, pathophysiology, and treatment of insomnia as well as neurophysiological and behavioral correlates of sleep and mental disorders.

Lorie A. Ritschel is Clinical Assistant Professor in the Department of Psychiatry at the University of North Carolina Chapel Hill School of Medicine. She is a consultant and trainer in dialectical behavior therapy (DBT) with Behavioral Tech, LLC, and she directs the adolescent DBT program at her private practice in Durham, North Carolina. Her areas of expertise are borderline personality disorder, emotion dysregulation, suicide and self-harm behavior, and a variety of empirically supported psychotherapies.

Raymond G. Romanczyk is a SUNY Distinguished Service Professor and a member of the clinical psychology faculty at Binghamton University. He is Codirector of the Institute for Child Development and Adjunct Professor of Psychiatry at SUNY Upstate Medical University. His expertise is related to evidence-based treatment approaches for autism spectrum disorders, large-scale service delivery, and information-processing for clinical decision-making.

Lindsay M. Stewart is Assistant Professor within the Department of Psychiatry and Behavioral Sciences at Emory University. She specializes in the assessment and treatment of mood and anxiety disorders.

Monika Stojek is based at the Department of Psychology, University of Georgia and Medical College of Georgia, Georgia Regents University. She is also a Postdoctoral Research Fellow at Eunice Kennedy Shriver National Institute of Child Health and Human Development.

Gregory P. Strauss is Assistant Professor at the Department of Psychology, University of Georgia, and is an expert in the fields of psychosis and emotions. He takes a translational approach to studying various aspects of schizophrenia, relying on theoretical frameworks and methods from the field of affective neuroscience (e.g., functional magnetic resonance imaging, electroencephalography, event-related potentials, eye tracking, electrocardiography, electrodermal activity, and electromyography).

Aurora Szentágotai-Tătar is Professor of Clinical Psychology and Psychotherapy at Babeş-Bolyai University, a fellow of the Albert Ellis Institute, and a member of the International Institute for the Advanced Studies of Psychotherapy and Applied Mental Health. Her expertise is related to emotion regulation and evidence-based psychotherapies.

Lauren VanderBroek-Stice is a doctoral candidate in the Department of Psychology at the University of Georgia.

Bogdan Voinescu is a clinical research fellow at the Institute of Psychiatry, Psychology and Neuroscience, King's College London, and a lecturer in Psychiatry at the Department of Clinical Psychology and Psychotherapy, Babeş-Bolyai University. His research interests have mainly been related to insomnia and circadian rhythms in various psychiatric and somatic conditions.

1

An Introduction to the Science and Practice of Evidence-Based Psychotherapy

A Framework for Evaluation and a Way Forward

Daniel David,[1] Steven Jay Lynn,[2] and Guy H. Montgomery[3]

[1] *Department of Clinical Psychology and Psychotherapy, Babeș-Bolyai University, Cluj-Napoca, Romania*

[2] *Psychology Department, Binghamton University, Binghamton, USA*

[3] *Department of Population Health Science and Policy, Icahn School of Medicine at Mount Sinai, New York, USA*

The terrain of contemporary psychotherapy is vast. In fact, patients can choose from more than 500 brands of psychotherapy. The challenges in navigating this bewildering landscape of psychotherapeutic interventions can be daunting (Lilienfeld, 2007). In this volume, we guide consumers of psychotherapy, clinicians, researchers, and students in the task of ascertaining the psychological treatments that are most rigorously evaluated, the treatment mechanisms that are best established, and the interventions that are most likely to be associated with positive outcomes for an array of disorders.

1.1 Evidence-Based Psychotherapies and Clinical Practice

Many psychotherapies in vogue today have never been subjected to rigorous scientific scrutiny, and there is no guarantee that a consumer of psychotherapy will receive an effective, evidence-based treatment. Although researchers have demonstrated that some psychotherapeutic interventions are successful, many individuals with major mental disorders still fail to receive treatments grounded in rigorous research (see Lynn & Lilienfeld, 2017). As Lilienfeld (2007) points out, surveys of clinical practitioners reveal that "substantial pluralities or even majorities do not treat patients with empirically supported methods" (p. 63). One such survey (Kessler et al., 2003) revealed that only about a fifth of individuals with clinical depression received adequate, empirically based clinical treatment in the year in which they were interviewed (see also Wang, Berglund, & Kessler, 2000, reporting similar findings for anxiety disorders). A more recent representative community household survey from 21 countries found that, among respondents who received treatment for depression, only 41% received treatment that met even minimal standards (Thornicroft et al., 2017). Most people with depression receive no psychological treatment, grossly suboptimal treatment, or ineffective treatment (Kessler

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

et al., 2003; Shim, Baltrus, Ye, & Rust, 2011). Much the same can be said for anxious individuals. In a study of 582 patients with anxiety disorders treated in community mental health settings, only 13.2% received cognitive–behavioral therapy, an empirically based treatment for anxiety (Sorsdahl et al., 2013; Wolitzky-Taylor, Zimmerman, Arch, De Guzman, & Lagomasino, 2015).

There is reason for equal, if not more, pessimism regarding treatment of disorders other than anxiety and depression. About one-third of individuals with autism receive nonvalidated interventions (Romanczyk, Turner, Sevlever, & Gillis, 2015); the majority of therapists who treat posttraumatic stress disorder fail to implement exposure and response prevention, one of the consensus treatments of choice for this condition (Freiheit, Vye, Swan, & Cady, 2004; Lilienfeld, 2007; Russell & Silver, 2007; see also Chapter 7); most therapists who treat eating disorders fail to capitalize on scientifically based treatments (Lilienfeld, Ritschel, Lynn, Brown et al., 2013); and as many as three-quarters of licensed social workers deliver one or more interventions with no research grounding whatsoever (Pignotti & Thyer, 2009).

Other interventions (e.g., attachment therapies, memory recovery techniques, critical incident stress debriefing, grief counseling for normal bereavement) not only lack empirical support but are also potentially harmful. Several produce "deterioration effects" in as many as 3% to 10% of patients, in which patients become worse after psychotherapy (see Lilienfeld, 2007). Moreover, a quarter or more of therapists report they use highly suggestive techniques (such as guided imagery or repeated prompting of memories) that are known to increase the risk of false memories of abuse (see Lynn, Krackow, Loftus, Locke, & Lilienfeld, 2015). Thomas Insel, the director of the National Institute of Mental Health, framed the situation this way: "Mental health care in America is ailing" (Insel & Fenton, 2009).

Unfortunately, many mental health professionals administer scientifically questionable or pseudoscientific techniques (see Lilienfeld, Lynn, & Lohr, 2015). For example, a large national survey by Kessler and associates (2001) revealed that substantial numbers of clinically depressed and anxious individuals receive such interventions as "energy therapy," massage therapy, aromatherapy, acupuncture, and even laughter therapy (see also Lee & Hunsley, 2015; Lilienfeld et al., 2015; Lilienfeld, Ruscio, & Lynn, 2008). Even if treatments such as equine assisted therapy (i.e., animal-assisted therapy), which lack rigorous empirical support (Anestis, Anestis, Zawilinski, Hopkins, & Lilienfeld, 2014), do little or no harm, mental health consumers who engage in them may forego effective interventions. Economists term this little-appreciated adverse effect an "opportunity cost." Such unsupported techniques also deprive mental health consumers of valuable time, money, and energy, sometimes leaving them with precious little of all three (see Lynn & Lilienfeld, 2017; Lynn, Malakataris, Condon, Maxwell, & Cleere, 2012). Nonscientific practices can also tarnish the reputation and credibility of mental health professionals, rendering members of the general public more reluctant to turn to them for greatly needed psychological help (Lynn & Lilienfeld, 2017).

In the main, psychotherapy is helpful. Scientists have established that many interventions—those that focus on directly changing people's thoughts, feelings, behaviors, and interpersonal relationships—are superior to no therapy, and often work as well as, or even better than, medications for common psychological conditions such as depression and anxiety (Barlow, Gorman, Shear, & Woods, 2000; Butler, Chapman,

Forman, & Beck, 2006; Dimidjian et al., 2006; Lemmens et al., 2015; Stewart & Chambless, 2009; Weitz et al., 2015). Moreover, psychotherapy combined with medication produces better outcomes in the treatment of depression than medication alone (Cuijpers, De Wit, Weitz, Andersson, & Huibers, 2015).

Still, implementing interventions, maximizing their outcomes, and getting them to patients in need are by no means without challenges. Although evidence-based therapies are available for a diversity of clinical conditions, there exists a pressing need to more widely disseminate (by teaching, training, and practice) and increase the accessibility of such services (Barnett, Rosenberg, Rosenberg, Osofsky, & Wolford, 2014; Karlin & Cross, 2014; Stewart et al., 2014). For example, as many as 70% of individuals with anxiety and mood disorders do not use or have access to psychological services (Kazdin & Rabbitt, 2013; Lilienfeld, Lynn, & Namy, 2018). Moreover, there is much room for improvements in evidence-based therapies, as many patients with clinical conditions do not respond satisfactorily to treatment, and, even when they do respond, they often relapse months to years after treatment (Steinert, Hofmann, Kruse, & Leichsenring, 2014).

1.2 Classifying Psychotherapies: Tricky Business

As David and Montgomery (2011) argued, the meaning of the term "evidence-based psychotherapy" is a moving target that varies considerably among (a) researchers, (b) classification schemes that identify therapies as "empirically supported," and (c) international organizations. A particular therapy may be considered empirically supported vis-à-vis one classification system, yet not be listed as supported in another classification system. Indeed, multiple evaluative frameworks for evidence-based psychotherapies have generated conflicting views and diverging standards regarding the status of individual psychological interventions. For example, the National Institute for Health and Care Excellence's guidelines (http://www.nice.org.uk) are not always consistent with those stipulated by Division 12 (the Society of Clinical Psychology) of the American Psychological Association (https://www.div12.org/psychological-treatments) or the American Psychiatric Association (http://www.psych.org), or with the conclusions of typically comprehensive Cochrane Reviews (http://www.cochrane.org). This lack of consistency instills confusion among professionals and patients alike, both of whom are seeking to select empirically validated treatments, and strongly supports the need for a unified, more scientifically oriented system for categorizing psychological treatments.

Most of the abovementioned classification systems are limited to a focus on the empirical status of the therapy package. Typically, the schemes evaluate the intervention package by comparing it with various control conditions (e.g., no intervention, waitlist, placebo/attention control, treatment as usual, active treatment, evidence-based treatment). Nevertheless, a treatment package is typically allied with a hypothesized underlying theory/mechanism of change, which should, we contend, impact the evidence-based status of the treatment delivered. Unfortunately, as David and Montgomery (2011) have argued, the current evaluative psychotherapy frameworks ignore the support, or lack thereof, for underlying theory and mechanism of change. Conceivably, a technique based on voodoo practices could be classified as "probably

efficacious" in current evaluative frameworks of psychotherapy, based on a clinical trial comparing voodoo therapy with a waitlist control condition.

The lack of a concerted focus on mechanisms of change is not surprising given that science (Kuhn, 1962), and the science of psychotherapy in particular, can be described as evolving in loosely demarcated stages or phases. Acknowledgment of the need to consider potential mechanisms that moderate or mediate treatment success is only of recent origin. DiGiuseppe, David, and Venezia (2016) have argued that the psychotherapy field can be described in terms of the following phases: (1) a preparadigmatic phase (e.g., schools of psychotherapy proliferated, often based on who would "shout the loudest" to attract attention, rather than based on rigorous controlled studies); (2) a paradigmatic phase (e.g., the first science-based paradigm was arguably behavior therapy); (3) crisis (e.g., behavior therapy was strongly challenged by new learning theories that emphasized cognitive processes); (4) new paradigms (e.g., cognitive therapies emerged as contenders to behavior therapy); (5) paradigm clashes (e.g., behavior and cognitive paradigms competed for ascendancy); and (6) normal science (e.g., the integration of cognitive and behavioral paradigms yielded the cognitive–behavioral paradigm), which is being challenged again by emerging (so-called third-wave) approaches such as acceptance and commitment therapy (which are in the process of a new integration into the cognitive–behavioral family).

In the early phases of the evolution of psychotherapy, a psychotherapy school was often successful because a charismatic or highly influential founder (e.g., Freud, Rogers) forcefully promoted its practice and the hypothesized underlying theory as useful and even scientific, as viewed through the lens of prevailing scientific standards. In this period of "grand psychotherapy systems" (e.g., psychoanalysis, humanistic–existential), most of the evidence invoked by a particular school was based on examples derived from successful cases. During this period, advocates of a particular system often did not place a premium on careful evaluation of treatment efficacy or effectiveness, and rarely, if at all, on empirical evaluation of mechanisms presumed to be associated with treatment gains.

A notable, contrasting, and welcome development, then, was the emergence of the relatively recent movement toward evidence-based practice (Lilienfeld, Ritschel, Lynn, Cautin, & Latzman, 2013). The scientific community increasingly acknowledged that a practice cannot be said to be "supported" in the context of a verification framework alone (e.g., identifying successful examples based on more or less anecdotal reports) and that a framework of falsification (e.g., searching for counterexamples or alternative explanations) should also be implemented in the evaluation process. Not surprisingly, the randomized clinical trial (RCT) became the gold standard for establishing an evidence-based psychological treatment. Probably the first large-scale RCTs involving psychotherapy, as compared with pharmacotherapy, were related to cognitive psychotherapy for depression (Rush, Beck, Kovacs, & Hollon, 1977). No matter how useful this approach, it nevertheless ignored, or gave short shrift to, evaluation of the underlying theory of the practice.

It seems fair to say that psychotherapy continues to evolve, as exemplified by recent efforts to identify and assess mechanisms of successful treatment outcomes within and across interventions. Yet, to this day, clinical practice continues to be based largely on personal experiences with a particular therapy, expert consensus, and reports of successful clinical applications (see Lilienfeld, Ritschel, Lynn, Cautin, & Latzman, 2013). Still, in recent years, researchers and clinicians have gradually, and some might say grudgingly,

come to appreciate the value of testing theories and mechanisms associated with diverse interventions.

1.3 A New Evaluation Scheme for Psychotherapy: Efficacy and Mechanisms

The evaluation of treatment mechanisms is particularly important because any number of explanations or variables may account for why a psychotherapy appears to be efficacious or effective, albeit not for the reasons stipulated by the innovators or promoters of the intervention. Causes of spurious therapeutic efficacy or effectiveness include spontaneous recovery, demand characteristics and nonspecific factors, natural fluctuations in symptoms, and a tendency to remember one's pretreatment functioning as worse than it was (see Lilienfeld, Ritschel, Lynn, Cautin, & Latzman, 2014, for a list of 26 such reasons). To address the need to consider treatment mechanisms in extant evaluative frameworks of psychotherapy, David and Montgomery (2011) proposed a scheme that takes into account both the efficacy and the effectiveness of the intervention package as well as its underlying theory regarding mechanism of change (see Table 1.1).

Table 1.1 A new evaluative framework of psychotherapy (reproduced after David and Montgomery, 2011).

Therapeutic Package	Theory		
	Well Supported[a]	Equivocal: No, Preliminary, or Mixed Data[b]	Strong Contradictory Evidence[c]
Well Supported[d]	Category I	Category II	Category V
Equivocal: No, Preliminary, or Mixed Data[b]	Category III	Category IV	Category VII
Strong Contradictory Evidence[c]	Category VI	Category VIII	Category IX

[a]Well-supported theories are defined as those with evidence based on (1) experimental studies (and sometimes additional/adjunctive correlational studies) and/or (2) component analyses, patient–treatment interactions, and/or mediation/moderation analyses in complex clinical trials (CCTs). Thus, the theory can be tested independent of its therapeutic package (e.g., in experimental studies and sometimes their additional/adjunctive correlational studies) and/or during a CCT. "Well supported" within this framework means that a theory has been empirically supported in at least two rigorous studies, by two different investigators or investigating teams.

[b]Equivocal evidence for therapeutic package and/or theory means no data (data not yet collected), preliminary data (there are data collected, be it supporting or contradictory, but it does not fit the minimum standards), or mixed data (there is both supporting and contradictory evidence).

[c]Strong contradictory evidence for therapeutic package and/or theory means that it has not been empirically supported in at least two rigorous studies, by two different investigators or investigating teams.

[d]Well-supported therapeutic packages are defined as those with randomized clinical trial (or equivalent) evidence of their efficacy (absolute, relative, and/or specific) and/or effectiveness. "Well supported" in this framework means that a package has been empirically supported in at least two rigorous studies, by two different investigators or investigating teams.

The darker backgrounds (Categories V–IX) signify pseudoscientifically oriented psychotherapies (POPs); the core of the POPs is Category IX. The lighter backgrounds (Categories I–IV) signify scientifically oriented psychotherapies (SOPs); the core of the SOPs is Category I. Depending on the progress of research, a psychotherapy could move from one category to another.

1.4 What We Aim to Accomplish

Our book presents the most systematic evaluation of psychotherapies for a variety of psychological disorders. The structure relies heavily on the David and Montgomery (2011) framework for evaluating the state of the science of psychotherapy interventions. More specifically, we have engaged eminent experts to evaluate the scientific status of psychotherapy for each disorder presented in the pages that follow. The new framework is used as a springboard to consider both theory (i.e., mechanisms of psychological change) and the therapeutic package. Contributors evaluate therapies in terms of the extent to which interventions and theoretical mechanisms are supported by empirical evidence ranging from empirically well-supported to contradictory evidence. Although the framework uses categories, with well described criteria for placement in each category (e.g., minimum number of positive trials), to describe the empirical status of studies pertinent to different disorders, the chapter authors do consider the entire body of evidence related to the therapies they describe and address the strengths and weaknesses of the research base in doing so (e.g., through the use of the "mixed data" status). This scheme affords researchers, clinicians, patients, and students the opportunity to assess the empirical status of treatments for disorders likely to be encountered in clinical practice and to separate science-based treatments from primarily pseudoscientific interventions.

To facilitate comparisons across disorders and therapies, and to move the field of psychotherapy forward, we invited experts to present (1) a description of the disorder (e.g., diagnostic features, prevalence); (2) a review of empirical support for the intervention and the supporting theory; and (3) implications for research and practice. The chapters encompass adult and child treatments and family and couples interventions. Our aim was to catalogue studies that support or fail to support treatment efficacy and effectiveness and to assess whether the psychological mechanisms presumed to be associated with therapeutic change are, in fact, supported by empirical studies. Typically, the term "efficacy" refers to studies with maximum internal validity (e.g., an RCT with a well-described treatment protocol and highly trained therapists), whereas the term "effectiveness" refers to studies that evaluate how well an intervention works in the real world or everyday practice. Nevertheless, in this volume, the terms are often used interchangeably by the authors so the exact meaning should be determined in each context. Taking into account our classification scheme, which is based on randomized trials, typically the focus is on efficacy studies, without ignoring existing effectiveness studies.

1.5 Conclusions

In conclusion, the authors of the chapters in this book evaluate the evidentiary status of treatments for a specific disorder or condition in terms of a well-delineated framework. By providing an up-to-date snapshot of the field of psychotherapy and pinpointing gaps in our knowledge of the efficacy and effectiveness of diverse interventions, each chapter provides researchers with potential directions for future studies.

Surveys consistently reveal that many clinicians do not embrace empirically supported psychotherapies, despite clear indications of their superiority over interventions

that might be appealing on face yet have little or no scientific standing (Lilienfeld, Ritschel, Lynn, Cautin, & Latzman, 2013). Accordingly, an overarching goal of this book is to tout the promise of empirically based methods and to increase the accessibility of the very best practices available for psychological disorders and conditions (ranging from insomnia to schizophrenia) and broadly promote their dissemination.

Readers will come to appreciate that the empirical support for theory and treatment protocols varies greatly, and that some treatments are considerably more efficacious or effective than others within and across the psychological disorders and conditions reviewed. We hope that our book will serve as an invaluable resource for the broad range of consumers (or potential consumers) of psychological services who wish to make informed choices regarding the most efficacious treatments for their problems in living and the psychological challenges their loved ones face.

References

Anestis, M. D., Anestis, J. C., Zawilinski, L. L., Hopkins, T. A., & Lilienfeld, S. O. (2014). Equine-related treatments for mental disorders lack empirical support: A systematic review of empirical investigations. *Journal of Clinical Psychology*, *70*(12), 1115–1132. doi:10.1002/jclp.22113

Barlow, D. H., Gorman, J. M., Shear, M. K., & Woods, S. W. (2000). Cognitive-behavioral therapy, imipramine, or their combination for panic disorder: A randomized controlled trial. *JAMA*, *283*(19), 2529–2536. doi:10.1001/jama.283.19.2529

Barnett, E. R., Rosenberg, H. J., Rosenberg, S. D., Osofsky, J. D., & Wolford, G. L. (2014). Innovations in practice: Dissemination and implementation of child–parent psychotherapy in rural public health agencies. *Child and Adolescent Mental Health*, *19*(3), 215–218. doi:10.1111/camh.12041

Butler, A. C., Chapman, J. E., Forman, E. M., & Beck, A. T. (2006). The empirical status of cognitive-behavioral therapy: A review of meta-analyses. *Clinical Psychology Review*, *26*(1), 17–31. doi:10.1016/j.cpr.2005.07.003

Cuijpers, P., De Wit, L., Weitz, E., Andersson, G., & Huibers, M. J. (2015). The combination of psychotherapy and pharmacotherapy in the treatment of adult depression: A comprehensive meta-analysis. *Journal of Evidence-Based Psychotherapies*, *15*(2), 147–168.

David, D., & Montgomery, G. (2011). The scientific status of psychotherapies: A new evaluative framework for evidence-based psychosocial interventions. *Clinical Psychology: Science and Practice*, *18*, 88–99. doi:10.1111/j.1468-2850.2011.01239.x

DiGiuseppe, R., David, X., & Venezia, R. (2016). Cognitive theories. In J. C. Norcross, G. R. VandenBos, D. K. Freedheim, & B. O. Olatunji (Eds.), *APA handbook of clinical psychology: Theory and research* (Vol. 2, pp. 154–182). Washington, DC: American Psychological Association.

Dimidjian, S., Hollon, S. D., Dobson, K. S., Schmaling, K. B., Kohlenberg, R. J., Addis, M. E., … Atkins, D. C. (2006). Randomized trial of behavioral activation, cognitive therapy, and antidepressant medication in the acute treatment of adults with major depression. *Journal of Consulting and Clinical Psychology*, *74*(4), 658–670. doi:10.1037/0022-006x.74.4.658

Freiheit, S. R., Vye, C., Swan, R., & Cady, M. (2004). Cognitive-behavioral therapy for anxiety: Is dissemination working? *Behavior Therapist*, *27*(2), 25–32.

Insel, T. R., & Fenton, W. S. (2005). Psychiatric epidemiology: It's not just about counting. *Archives of General Psychiatry*, *62*(6), 590–592.

Karlin, B. E., & Cross, G. (2014). From the laboratory to the therapy room: National dissemination and implementation of evidence-based psychotherapies in the US Department of Veterans Affairs Health Care System. *American Psychologist*, *69*(1), 19–33. doi:10.1037/a0033888

Kazdin, A. E., & Rabbitt, S. M. (2013). Novel models for delivering mental health services and reducing the burdens of mental illness. *Clinical Psychological Science*, *1*(2), 170–191. doi:10.1177/2167702612463566

Kessler, R. C., Berglund, P., Demier, O., Jin, R., Koretz, D., Merikangas, K. R., … Wang, P. S. (2003). The epidemiology of major depressive disorder: Results from the National Comorbidity Survey Replication (NCS-R). *JAMA*, *289*(23), 3095–3105. doi:10.1001/jama.289.23.3095

Kessler, R. C., Soukup, J., Davis, R. B., Foster, D. F., Wilkey, S. A., Van Rompay, M. I., & Eisenberg, D. M. (2001). The use of complementary and alternative therapies to treat anxiety and depression in the United States. *American Journal of Psychiatry*, *158*(2), 289–294. doi:10.1176/appi.ajp.158.2.289

Kuhn, T. S. (1962). *The structure of scientific revolutions*. Chicago, IL: University of Chicago Press.

Lee, C. M., & Hunsley, J. (2015). Evidence-based practice: Separating science from pseudoscience. *Canadian Journal of Psychiatry*, *60*(12), 534–540. doi:10.1177/070674371506001203

Lemmens, L. H. J. M., Arntz, A., Peeters, F. P. M. L., Hollon, S. D., Roefs, A., & Huibers, M. J. H. (2015). Clinical effectiveness of cognitive therapy v. interpersonal psychotherapy for depression: Results of a randomized controlled trial. *Psychological Medicine*, *45*(10), 2095–2110. doi:10.1017/s0033291715000033

Lilienfeld, S. O. (2007). Psychological treatments that cause harm. *Perspectives on Psychological Science*, *2*(1), 53–70. doi:10.1111/j.1745-6916.2007.00029.x

Lilienfeld, S. O., Lynn, S. J., & Lohr, J. (Eds.). (2015). *Science and pseudoscience in clinical psychology* (2nd ed.). New York, NY: Guilford Press.

Lilienfeld, S. O., Lynn, S. J., & Namy, L. (2018). *Psychology: From inquiry to understanding* (4th ed.). New York, NY: Pearson.

Lilienfeld, S. O., Ritschel, L. A., Lynn, S. J., Brown, A. P., Cautin, R. L., & Latzman, R. D. (2013). The research–practice gap: Bridging the schism between eating disorder researchers and practitioners. *International Journal of Eating Disorders*, *46*(5), 386–394. doi:10.1002/eat.22090

Lilienfeld, S. O., Ritschel, L. A., Lynn, S. J., Cautin, R. L., & Latzman, R. D. (2013). Why many clinical psychologists are resistant to evidence-based practice: Root causes and constructive remedies. *Clinical Psychology Review*, *33*(7), 883–900. doi:10.1016/j.cpr.2012.09.008

Lilienfeld, S. O., Ritschel, L. A., Lynn, S. J., Cautin, R. L., & Latzman, R. D. (2014). Why ineffective psychotherapies appear to work: A taxonomy of causes of spurious therapeutic effectiveness. *Perspectives on Psychological Science*, *9*(4), 355–387. doi:10.1177/1745691614535216

Lilienfeld, S. O., Ruscio, J., & Lynn, S. J. (Eds.). (2008). *Navigating the mindfield: A guide to separating science from pseudoscience in mental health*. Amherst, NY: Prometheus Books.

Lynn, S. J., Krackow, E., Loftus, E. F., Locke, T. J., & Lilienfeld, S. O. (2015). The remembrance of things past: Problematic memory recovery techniques in psychotherapy. In S. O. Lilienfeld, S. J. Lynn, & J. Lohr (Eds.), *Science and pseudoscience in clinical psychology* (2nd ed., pp. 205–242). New York, NY: Guilford Press.

Lynn, S. J., & Lilienfeld, S. O. (2017). *Off the rails: Psychotherapy gone wrong and the road to evidence-based treatment*. Unpublished manuscript.

Lynn, S. J., Malakataris, A., Condon, L., Maxwell, R., & Cleere, C. (2012). The treatment of posttraumatic stress disorder: Cognitive hypnotherapy, mindfulness, and acceptance-based approaches. *American Journal of Clinical Hypnosis*, *54*(4), 311–330. doi:10.1080/00029157.2011.645913

Pignotti, M., & Thyer, B. A. (2009). Use of novel unsupported and empirically supported therapies by licensed clinical social workers: An exploratory study. *Social Work Research*, *33*(1), 5–17. doi:10.1093/swr/33.1.5

Romanczyk, R. G., Turner, L. B., Sevlever, M., & Gillis, J. (2015). The status of treatment for autism spectrum disorders: The weak relationship of science to interventions. In S. O. Lilienfeld, S. J. Lynn, & J. M. Lohr (Eds.), *Science and pseudoscience in contemporary clinical psychology* (pp. 431–465). New York, NY: Guilford Press.

Rush, A. J., Beck, A. T., Kovacs, M., & Hollon, S. (1977). Comparative efficacy of cognitive therapy and pharmacotherapy in the treatment of depressed outpatients. *Cognitive Therapy and Research*, *1*(1), 17–37. doi:10.1007/BF01173502

Russell, M., & Silver, S. M. (2007). Training needs for the treatment of combat-related posttraumatic stress disorder: A survey of Department of Defense clinicians. *Traumatology*, *13*, 4–10. doi:10.1177/1534765607305440

Shim, R. S., Baltrus, P., Ye, J., & Rust, G. (2011). Prevalence, treatment, and control of depressive symptoms in the United States: Results from the National Health and Nutrition Examination Survey (NHANES), 2005–2008. *Journal of the American Board of Family Medicine*, *24*(1), 33–38. doi:10.3122/jabfm.2011.01.100121

Sorsdahl, K., Blanco, C., Rae, D. S., Pincus, H., Narrow, W. E., Suliman, S., & Stein, D. J. (2013). Treatment of anxiety disorders by psychiatrists from the American Psychiatric Practice Research Network. *Revista Brasileira de Psiquiatria*, *35*(2), 136–141. doi:10.1590/1516-4446-2012-0978

Steinert, C., Hofmann, M., Kruse, J., & Leichsenring, F. (2014). Relapse rates after psychotherapy for depression: Stable long-term effects? A meta-analysis. *Journal of Affective Disorders*, *168*, 107–118. doi:10.1016/j.jad.2014.06.043

Stewart, M. O., Raffa, S. D., Steele, J. L., Miller, S. A., Clougherty, K. F., Hinrichsen, G. A., & Karlin, B. E. (2014). National dissemination of interpersonal psychotherapy for depression in veterans: Therapist and patient-level outcomes. *Journal of Consulting and Clinical Psychology*, *82*(6), 1201–1206. doi:10.1037/a0037410

Stewart, R. E., & Chambless, D. L. (2009). Cognitive–behavioral therapy for adult anxiety disorders in clinical practice: A meta-analysis of effectiveness studies. *Journal of Consulting and Clinical Psychology*, *77*(4), 595–606. doi:10.1037/a0016032

Thornicroft, G., Chatterji, S., Evans-Lacko, S., Gruber, M., Sampson, N., Aguilar-Gaxiola, S., … Bruffaerts, R. (2017). Undertreatment of people with major depressive disorder in

21 countries. *British Journal of Psychiatry*, *210*(2), 119–124. doi:10.1192/bjp.bp.116.188078

Wang, P. S., Berglund, P., & Kessler, R. C. (2000). Recent care of common mental disorders in the United States. *Journal of General Internal Medicine*, *15*(5), 284–292. doi:10.1046/j.1525-1497.2000.9908044.x

Weitz, E. S., Hollon, S. D., Twisk, J., Van Straten, A., Huibers, M. J., David, D., … Faramarzi, M. (2015). Baseline depression severity as moderator of depression outcomes between cognitive behavioral therapy vs pharmacotherapy: An individual patient data meta-analysis. *JAMA Psychiatry*, *72*(11), 1102–1109. doi:10.1001/jamapsychiatry.2015.1516

Wolitzky-Taylor, K., Zimmermann, M., Arch, J. J., De Guzman, E., & Lagomasino, I. (2015). Has evidence-based psychosocial treatment for anxiety disorders permeated usual care in community mental health settings? *Behaviour Research and Therapy*, *72*, 9–17. doi:10.1016/j.brat.2015.06.010

2

Varieties of Psychotherapy for Major Depressive Disorder in Adults

An Evidence-Based Evaluation

Daniel David,[1] Ioana A. Cristea,[1] and Aaron T. Beck[2]

[1] *Department of Clinical Psychology and Psychotherapy, Babeș-Bolyai University, Cluj-Napoca, Romania*

[2] *Department of Psychiatry, University of Pennsylvania, Philadelphia, USA*

2.1 Description of the Disorder

According to the *Diagnostic and Statistical Manual of Mental Disorders*, major depressive disorder (MDD) is characterized by persistent low mood and/or loss of interest or pleasure in most activities (American Psychiatric Association, 2013). For a person to qualify for a diagnosis, at least five of nine symptoms must be present, including significant changes in appetite or weight loss, insomnia or hypersomnia, fatigue and loss of energy, noticeable physical agitation or slow-down, feelings of worthlessness or excessive guilt, reduced concentration or persistent indecision, and recurrent thoughts of death or suicide (American Psychiatric Association, 2013). Symptoms have to be present for at least 2 weeks, for most of the day.

According to projections based on the World Health Organization Global Burden of Disease study, by 2030, depression is estimated to be the leading cause of the global burden of disease (Mathers & Loncar, 2006). MDD is considered a leading cause of disability, and in the Global Burden of Disease 2010 study it ranked second after low back pain in terms of years lived with disability, with depression explaining 8.2% of all years lived with disability (Ferrari et al., 2013). Also, MDD is a recurrent, frequently chronic condition, with lifetime prevalence higher than 15%. Researchers have estimated the annual prevalence of MDD in the adult population to be around 7% in the United States (Kessler, Chiu, Demler, & Walters, 2005) and 5% in Western Europe (Paykel, Brugha, & Fryers, 2005). More than 30 million people in Europe alone are affected by MDD, and the associated annual costs are estimated at €92 billion (Olesen et al., 2012). For the United States, the annual costs were estimated at $210.5 billion in 2010 (Greenberg, Fournier, Sisitsky, Pike, & Kessler, 2015). Depression also carries huge societal and economic costs, with estimates of workplace-related costs of $50 billion each year in the United States alone (Greenberg et al., 2003). Antidepressant medication is often effective, yet only

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

50% to 70% of patients respond to a first antidepressant treatment (Berrettini, 2002; Gartlehner et al., 2011; Kornstein & Schneider, 2001). Response rates to psychotherapy (i.e., cognitive–behavioral therapy) are also around 60% for moderate-to-severe MDD (DeRubeis et al., 2005). More than 40% of patients do not or only partially respond to treatment and less than one-third are completely recovered after treatment (Hollon et al., 2002). Spontaneous remission is rather low, with only one in three patients achieving remission (Trivedi et al., 2006). Furthermore, relapse rates are estimated to be 40% after 2 years (Boland & Keller, 2009) and up to 85% within 15 years of recovery from an initial episode (Mueller et al., 1999). Chronic forms of MDD account for approximately 25–30% of all diagnosed cases (Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993) and are associated with higher functional impairment and disability than nonchronic MDD (Gilmer et al., 2005; Satyanarayana, Enns, Cox, & Sareen, 2009). Modeling studies have shown that pharmacological and psychological treatments together can reduce the disease burden of depression by only about 33% (Andrews, Sanderson, Corry, & Lapsley, 2000).

2.2 Classification of Psychotherapies According to David and Montgomery's (2011) Evaluative Framework

David and Montgomery (2011) argued that any classification framework of psychotherapy (i.e., psychological treatments) should be based on two components: (1) the efficacy/effectiveness of the therapeutic package and (2) the empirical support for the theory underlying the clinical protocol. The combination of these two criteria results in the nine categories regarding the empirical state of psychotherapies for a given clinical condition (e.g., MDD) (see Table 2.1). The "therapeutic package" is understood as the clinical protocol for the psychological treatment that is being studied, including everything from clinical case formulation to construction of the therapeutic relationship to suggested intervention techniques. The "theory of change" is understood as relating to the underlying mechanism supporting the clinical protocols and/or to the etiological theory for the clinical condition. Indeed, sometimes the clinical protocols might not be etiologically driven and may instead be pathogenetically and/or symptomatically driven.

In the following, we will analyze the current state of the art of various psychotherapies for MDD, through the lens of David and Montgomery's (2011) psychotherapy classification system. The concepts used for this analysis are thoroughly detailed in David and Montgomery (2011) and Wampold (2001). Briefly, "absolute efficacy" refers to the question of whether the therapeutic package is better than a control condition, which can be no treatment (better than no treatment [BNT]) or a waitlist (better than waitlist [BWL]). "Relative efficacy" refers to the question of whether the therapeutic package is equivalent to or better than another standard psychological intervention (equivalent to standard treatment [EST] or better than standard treatment [BST]). Finally, "specific efficacy" involves meeting two criteria simultaneously. First, the therapeutic package must be (1) significantly better than pill, better than medical placebo, or better than psychological placebo and/or (2) equivalent to or better than standard psychological treatments (EST or BST). Second, the underlying theory for the mechanisms of change

Table 2.1 A graphical depiction of the classification of psychotherapies for MDD using David and Montgomery's (2011) evaluative framework.

	Theory		
Therapeutic Package	**Well Supported**	**Equivocal: No, Preliminary, or Mixed Data**	**Strong Contradictory Evidence**
Well Supported	Category I: Evidence-based psychotherapies CT	Category II: Intervention-driven psychotherapies IPT; BT (BA, PST)	Category V: Good-intervention- and bad-theory-driven psychotherapies None
Equivocal: No, Preliminary, or Mixed Data	Category III: Theory-driven psychotherapies CNT; REBT	Category IV: Investigational psychotherapies STPP; third-wave CBT; BCT	Category VII: Bad-theory-driven psychotherapies None
Strong Contradictory Evidence	Category VI: Good-theory- and bad-intervention-driven psychotherapies None	Category VIII: Bad-intervention-driven psychotherapies GT	Category IX: Bad-theory- and bad-intervention-driven psychotherapies None

Therapeutic package: the clinical protocol for the psychological intervention considered in the analysis. Theory: the underlying mechanism supporting the clinical protocols. BA: behavior activation; BCT: behavioral couple therapy; BT: behavior therapy; CBT: cognitive–behavioral therapy; CNT: concreteness training; CT: cognitive therapy; GT: grief therapy; IPT: interpersonal therapy; PST: problem-solving therapy; REBT: rational emotive behavior therapy; STPP: short-term psychodynamic psychotherapy.

the package is supposed to impact must be empirically supported by experimental studies (and sometimes additional or adjunctive correlational studies), component analysis, patient-by-treatment interactions, and/or analysis of mediation or moderation.

Based on David and Montgomery (2011) (see Table 2.1), "well supported" (i.e., strong supporting evidence) means that a psychotherapy has been empirically supported in at least two rigorous studies, by two different, independent investigators or investigating teams. "Strong contradictory evidence" means that it has been empirically invalidated in at least two rigorous studies, by two different, independent investigators or investigating teams. "Equivocal evidence" means no data (data not yet collected), preliminary data (there are collected data, be they supporting or contradictory, but they do not fit the minimum standards), or mixed data (there is both supporting and contradictory evidence).

2.2.1 Category I: Evidence-Based Psychotherapies

Category I includes psychological treatments for which there is strong supporting evidence both for the efficacy/effectiveness of the clinical protocol and for the theory of change underlying the clinical protocol. In the following, we present the evidence-based psychological treatments for MDD. For each therapy, we predominantly rely on evidence from recent meta-analyses and provide estimations of effect sizes (ESs). Since ES indicators are often difficult to interpret from a clinical point of view, when appropriate, we also include the more intuitive estimated number needed to treat (NNT), indicating the

number of patients who have to be treated with the intervention so as to achieve one additional positive outcome over a comparator (Altman, 1998).

2.2.1.1 Cognitive therapy

Cognitive therapy for depression (A. T. Beck, Rush, Shaw, & Emery, 1979; J. S. Beck, 1995) is based on the notion that depression is maintained by negatively biased information-processing and dysfunctional beliefs that affect current behavior and functioning. Cognitive restructuring, the transformation of dysfunctional cognitions into more adaptive ones, is believed to be at the core of CT (Clark & Beck, 2010). According to a conceptual review (A. T. Beck & Dozois, 2011), the theory behind CT relies on three main propositions: (1) the *access* hypothesis (with appropriate preparation, individuals can become aware of the content and process of their thinking); (2) the *mediation* hypothesis (the way in which individuals interpret and evaluate events influences their emotional and behavioral responses); and (3) the *change* hypothesis (individuals can become better adapted to the circumstances they face by modifying their thinking and behaviors).

CT uses a wide array of techniques to teach patients to monitor and record their thoughts, with the purpose of recognizing the mediating role they play in creating emotional outcomes. Patients are then taught to evaluate the utility and validity of these thoughts, and to test them in behavioral experiments with the ultimate goal of changing them from dysfunctional to more adaptive and useful. Frequently, therapy uses a psychoeducational approach, teaching patients adaptive coping, problem-solving, and, most importantly, cognitive restructuring skills. CT also emphasizes homework assignments and out-of-session activities, which are aimed at helping patients directly test the utility of the changes proposed in therapy.

CT is listed as having strong research support by the American Psychological Association (APA) Division of Clinical Psychology and is also listed by the National Institute for Health and Care Excellence (NICE; previously the National Institute for Health and Clinical Excellence) as a high-intensity intervention recommended for persistent subthreshold depressive symptoms and mild to moderate depression (NICE, 2009). According to the David and Montgomery classification, we argue CT belongs in Category I (evidence-based psychotherapies) for MDD.

In terms of its treatment package, we will refer to meta-analyses (Barth et al., 2013; Braun, Gregor, & Tran, 2013; Cuijpers, Berking et al., 2013; Cuijpers, Cristea, Karyotaki, Reijnders, & Huibers, 2016; Cuijpers, Hollon et al., 2013; Cuijpers et al., 2014) regarding the outcomes of CT for MDD:

2.2.1.1.1 Absolute efficacy

- In one meta-analysis (Cuijpers, Berking et al., 2013), CT was found to be more efficacious than waitlist comparisons (BWL) in 55 randomized controlled trials (RCTs), with a mean ES (Hedges' *g*) of 0.83, corresponding to an NNT of 2.26. Also, CT was more efficacious than usual care in 26 RCTs, with a mean ES of $g = 0.59$, corresponding to an NNT of 3.09. A network meta-analysis (Barth et al., 2013), considering both direct and indirect comparisons, reported similar values: $g = 0.78$ in contrast with waitlist control and, $g = 0.45$ in contrast with usual care. Finally, the most recent meta-analysis (Cuijpers, Cristea et al., 2016), using more restrictive criteria that only included RCTs in which participants met the criteria for MDD according to a

structured diagnostic interview, also confirmed the absolute efficacy for CT as both BWL (28 RCTs, $g = 0.98$, NNT = 2.85), as well as in comparison with usual care (30 RCTs, $g = 0.60$, NNT = 4.99).

2.2.1.1.2 Relative efficacy

- With regard to comparisons with psychological placebo, CT was found to be better than placebo and better than both psychological and pill placebo taken together in two meta-analyses: $g = 0.51$, corresponding to an NNT of 3.55, for 13 RCTs of direct comparisons (Cuijpers, Berking et al., 2013), and $g = 0.45$ in a network meta-analysis considering both direct and indirect comparisons (Barth et al., 2013). Regarding the comparison with pill placebo, the results have been more mixed, but overall they favor CT. Traditional aggregate data meta-analyses found CT to be better, with effects ranging from $g = 0.33$ (Cuijpers et al., 2014) to $g = 0.55$ (Cuijpers, Cristea et al., 2016), although the number of trials contributing to this effect was small (five in each case). Conversely, a recent individual patient data meta-analysis (Furukawa et al., 2017), which obtained all individual-level data (509 participants) from all five trials comparing CT with pill placebo, found a significant effect for the Hamilton Rating Scale for Depression (M. Hamilton, 1960), $g = 0.22$, but not for the Beck Depression Inventory (A. T. Beck, Steer, & Brown, 1996), $g = 0.05$.
- With regard to other standard treatments for depression (antidepressant medication, interpersonal psychotherapy, behavioral activation), CT was found to be equally efficacious (EST). Recent meta-analyses that have approached this issue found the following *nonsignificant* between-groups differences:
  - For CT compared with interpersonal psychotherapy: $g = 0$ for eight RCTs of direct comparisons (Cuijpers, Donker, Weissman, Ravitz, & Cristea, 2016); $g = 0.14$ considering both direct and indirect comparisons (Barth et al., 2013); and $g = 0.03$ for patient-rated outcomes and $g = 0.00$ for clinician-rated outcomes, again considering both direct and indirect comparisons (Braun et al., 2013).
  - For CT compared with behavioral therapy or behavioral activation: $g = -0.02$ for eight RCTs of direct comparisons (Cuijpers, Berking et al., 2013); $g = -0.02$ considering both direct and indirect comparisons (Barth et al., 2013); and $g = -0.08$ for patient-rated outcomes and $g = 0.10$ for clinician-rated outcomes, again considering both direct and indirect comparisons (Braun et al., 2013).
  - For CT compared with antidepressant medication, an individual patient data meta-analysis (Weitz et al., 2015), which recovered data from 16 of the 24 conducted trials and analyzed a total of 1,700 participants, found no significant differences between CT and antidepressant medication on the clinically relevant outcomes of response or remission, or on the Beck Depression Inventory. The meta-analysis did, however, find a significant difference on scores for the Hamilton Rating Scale for Depression.

2.2.1.1.3 Specific efficacy

- The first criterion, involving superiority to placebo or equivalence to other standard therapies, is met (see previous point about relative efficacy).
- The second criterion, regarding the empirical support for the underlying theory for specific mechanisms of change in the case of the therapeutic package, is met.

We argue that, following the criteria of David and Montgomery (2011) regarding the validation of the theory of change of a psychotherapy, there is empirical support for

the mechanisms of change postulated by the theory of CT, more precisely in the following:

- *Experimental studies*: Cognitive variables implicated in the cognitive model of depression prospectively predicted depressive outcomes (Abela & D'Alessandro, 2002; for a review see Scher, Ingram, & Segal, 2005). Moreover, a review (Scher et al., 2005) of the empirical literature on both priming and longitudinal studies on the cognitive model of depression supported the diathesis-stress hypothesis, the idea that depressive schemas only affect information-processing and cognition *when* they become activated. Importantly, this review also concluded that evidence supported the cognitive reactivity hypothesis, in that, across age groups, individuals demonstrated greater levels of depressive symptoms as a product of the activation of dysfunctional cognitions. These effects held for both the full diagnosis of depressive disorders and depressive symptoms.
- *Complex controlled trials with mediation analyses and/or patient-treatment interactions*: Changes in cognitive variables postulated by the cognitive model of depression mediated changes in depression outcomes (Quilty, McBride, & Bagby, 2008; Strunk, Brotman, & DeRubeis, 2010; Tang, DeRubeis, Beberman, & Pham, 2005; for a review see Garratt, Ingram, Rand, & Sawalani, 2007). Another review (Hundt, Mignogna, Underhill, & Cully, 2013) also found evidence for the meditational role of the frequency of use and quality of cognitive–behavioral therapy (CBT) skills. Moreover, a longitudinal study (Beevers & Miller, 2005) following patients for a year after 6 months of treatment demonstrated that the association between negative cognitions related to depression in the cognitive model of the disorder (e.g., hopelessness, worthlessness) and depression severity was weaker for individuals receiving CT in combination with medication as compared to those receiving medication alone or the combination with family therapy and medication. This effect was stronger for patients who remitted at the end of treatment.
- *Complex controlled trials with moderation analysis*: Changes in dysfunctional attitudes, a key cognitive construct in the cognitive model of depression, moderated treatment outcomes (i.e., predicted treatment response) (K. E. Hamilton & Dobson, 2002; Jarrett, Vittengl, Doyle, & Clark, 2007; Segal et al., 2006).

However, a potential challenge to our analysis could come from at least two reviews (Kazdin, 2007; Longmore & Worrell, 2007), which have questioned the evidence for change in cognitions as mediating treatment outcomes for depression. A recent meta-analysis (Cristea et al., 2015) provided both support for and challenge to the function of dysfunctional cognitions as mechanisms of change. It reported a moderate effect of CT for dysfunctional thoughts at posttest ($g = 0.50$), which was strongly associated with the effects of CT for depression outcomes. However, it also found no differences in the effects of dysfunctional thoughts between CT and other psychotherapies (except when outcomes were measured by the Dysfunctional Attitudes Scale) and pharmacotherapy. These findings can be construed either as confirming the primacy of cognitive change in symptom change, irrespective of how it is attained, or as supporting the notion that dysfunctional thoughts might simply be another symptom that changes subsequent to treatment. While an in-depth discussion of the complex relationship between cognitive change procedures, change in cognitive mechanisms, and symptom change is beyond the scope of this chapter (for an extensive review, see Lorenzo-Luaces, German, &

DeRubeis, 2015), we would argue that, in the David and Montgomery (2011) framework, especially taking into account the experimental and/or correlational studies, the evidence fits better the *well-supported* than the *mixed data* criterion.

2.2.2 Category II: Intervention-Driven Psychotherapies

Category II includes psychological treatments for which there is strong supporting evidence for the efficacy/effectiveness of the clinical protocol. However, with regard to the theory of change underlying the clinical protocol, the results are equivocal (i.e., *missing*, *preliminary*, or *mixed*). We present in the following the intervention-driven psychotherapies for MDD.

2.2.2.1 Interpersonal psychotherapy

Although based on a psychoanalytic foundation, interpersonal psychotherapy (IPT) also incorporates attachment theory and focuses on interpersonal relationships (Klerman, Weissman, Rounsaville, & Chevron, 1984). According to the theory of IPT, depression is caused by multiple factors, but the focus is placed on explaining and treating it within an interpersonal context. Therapy is focused on current interpersonal difficulties in four main areas: (1) complicated grief, (2) interpersonal disputes, (3) role transitions, and (4) interpersonal deficits.

IPT is listed as having strong research support by the APA Division of Clinical Psychology and is also listed by NICE as a high-intensity intervention recommended for persistent subthreshold depressive symptoms and mild to moderate depression (NICE, 2009). According to David and Montgomery's (2011) classification, we argue that IPT belongs in Category II (intervention-driven psychotherapies) for MDD.

In terms of its treatment package, we will refer to recent meta-analyses (Barth et al., 2013; Braun et al., 2013; Cuijpers, Donker et al., 2016) regarding the efficacy of IPT for major depression.

2.2.2.1.1 Absolute efficacy

- Researchers have found IPT to be more efficacious than a control (usual care, waitlist, other) condition in 31 RCTs, with a mean ES (Cohen's d) of 0.60, corresponding to an NNT of 3 (Cuijpers, Donker et al., 2016). More precisely, IPT was more efficient than usual care in 17 RCTs, with a mean ES (Hedges' g) of 0.59 (Cuijpers, Donker et al., 2016). An earlier network meta-analysis, using evidence from both direct and indirect comparisons, found a very similar effect in the comparison between IPT and usual care, $g = 0.59$ (Barth et al., 2013), enhancing confidence in the robustness of this estimate. This meta-analysis also found a large ES of IPT over waitlist controls, $g = 0.92$.

2.2.2.1.2 Relative efficacy

- With regard to one of the standard treatments for depression (CBT), IPT was shown to be as efficacious (EST). The two psychotherapies were directly compared in eight RCTs with a nonsignificant $d = 0$ for between-group comparisons (Cuijpers, Donker et al., 2016). An earlier network meta-analysis, relying on evidence from both direct and indirect comparisons, reported a nonsignificant g of 0.03 for patient-rated outcomes and a nonsignificant $g = 0.00$ for clinician-rated outcomes (Braun et al.,

2013). Finally, another network meta-analysis did not find differences between IPT and CBT, yielding a nonsignificant $g = 0.14$ (Barth et al., 2013).
- With regard to another standard treatment for depression (antidepressant medication), IPT was also shown to be as efficient (EST). IPT and pharmacotherapy were directly compared in 16 RCTs, which yielded a nonsignificant $d = -0.13$ for between-group comparisons (Cuijpers, Donker et al., 2016).

2.2.2.1.3 Specific efficacy

- The first criterion, involving the equivalence of the treatment to other standard therapies (EST), is met (see previous point about relative efficacy).
- The second criterion, regarding the empirical support for the underlying theory for specific mechanisms of change in the case of the therapeutic package, is not met.

More specifically, a review (Lipsitz & Markowitz, 2013) regarding mechanisms of change in IPT underlines the fact that researchers have so far devoted very little effort to exploring change mechanisms. We attribute this lack of knowledge about what makes IPT efficacious to a pragmatic focus among IPT practitioners and researchers on what benefits the patients, rather than a focus on the genesis of these effects or an integrative view of therapeutic change. Indeed, IPT has emphasized a multifaceted approach, appealing to a number of complementary and interdependent causal factors, both common to other approaches and specific to IPT, rendering it difficult to reduce treatment efficacy to the by-product of one or a few change mechanisms.

Perhaps a quote from the aforementioned review poignantly encapsulates the problem of mechanisms of change in IPT: "How does resolving the interpersonal problem alleviate psychiatric symptoms? To date, IPT has not sufficiently elaborated mechanisms to account for this change" (Lipsitz & Markowitz, 2013, p. 1139).

Lipsitz and Markowitz (2013) proposed and argued theoretically and with some circumstantial (albeit limited) empirical evidence that four mechanisms might account for how resolving interpersonal problems could directly impact symptom change: (1) enhancing social support, (2) decreasing interpersonal stress, (3) facilitating emotional processing, and (4) improving interpersonal skills. However, the authors note that none of these proposed mechanisms have been tested to date in component analyses or as mediators of treatment response in IPT trials. We would qualify this as *no data* for the theory behind the IPT package for depression.

2.2.2.2 Behavioral therapies

According to a review by Hollon and Ponniah (2010), behavioral therapy (BT) includes contextual approaches based on functional analyses (i.e., contingency management and behavioral activation), as well as social skills training, self-control therapy, problem-solving therapy, and behavioral marital therapy. A central tenet of BTs is the notion that depression is a disorder resulting from problematic behavior–environment relationships, more precisely from low levels of positive reinforcement and high aversive control, most likely due to contextual problems (e.g., in the individual's environment, whether social or private) or to a lack of certain skills. Based on early social learning theories of depression (Lewinsohn, 1974), these therapies start off from the observation that depressed individuals withdraw from the environment and that this avoidance exacerbates depressed mood by depriving them of opportunities for positive

reinforcement. As such, therapy is focused on increasing the frequency and quality of pleasant activities, developing one's social skills and sense of mastery, and decreasing aversive consequences received for behaviors or rather lack thereof.

Several types of BT, namely behavioral activation (BA) and problem-solving therapy (PST), have generated particular interest. BA involves systematically increasing activation using exercises to bring the patient in contact with sources of positive reinforcement, identify inhibitors of activation, and teach problem-solving skills. PST involves a structured process to develop a person's problem-resolution strategies and coping skills to manage problematic situations (e.g., stressful experiences).

The APA Division of Clinical Psychology lists BT (in particular BA) as having strong research support. However, according to NICE, while BA is recommended for persistent subthreshold depressive symptoms and mild to moderate depression, it is also noted that the evidence for it is less robust than for IPT or CBT (NICE, 2009). According to David and Montgomery's (2011) classification, we argue that BT belongs in Category II (intervention-driven psychotherapies) for MDD.

In terms of BT treatment packages, we will refer to recent meta-analyses (Barth et al., 2013; Braun et al., 2013; Cuijpers, Van Straten, Andersson, & Van Oppen, 2008; Cuijpers, Van Straten, & Warmerdam, 2007b; Ekers et al., 2014) regarding the efficacy of BA and PST for major depression.

2.2.2.2.1 Absolute efficacy

- BA was documented to be more efficient than a control (waitlist, other) condition in 25 RCTs, with a mean ES (Cohen's *d*) of 0.74, corresponding to an NNT of 2.5. More precisely, BA was BWL in 20 RCTs, with a mean ES (Hedges' *g*) of 0.87, and better than usual care in six trials: $g = 0.78$ (Ekers et al., 2014). An earlier network meta-analysis (Barth et al., 2013), considering both direct and indirect comparisons, reported that BA was more efficacious than waitlist, usual care, and placebo, with mean ESs of 0.80, 0.47, and 0.46, respectively.
- PST was found to be more efficient than a control (waitlist, treatment as usual [TAU], pill placebo, psychological placebo) condition in 10 RCTs, with a mean ES (Cohen's *d*) of 0.83 (Cuijpers, Van Straten, & Warmerdam, 2007a). A network meta-analysis (Barth et al., 2013) of both direct and indirect comparisons also found PST to be more efficient than waitlist, usual care, and placebo, with mean ESs (Hedges' *g*) of 0.74, 0.41, and 0.41, respectively.

2.2.2.2.2 Relative efficacy

- For BA, research has shown it to be as efficacious for depression (EST) with regard to one of the standard treatments for depression (CBT). According to one meta-analysis, BA and CBT were directly compared in eight RCTs with a nonsignificant $d = 0.02$ for between-group comparisons (Cuijpers, Berking et al., 2013). A network meta-analysis (Braun et al., 2013) relying on evidence from both direct and indirect comparisons reported nonsignificant differences between BA and CBT on patient-rated ($g = -0.08$) and clinician-rated outcomes ($g = 0.10$). Another network meta-analysis also reported nonsignificant differences between BA and CBT, $g = 0.02$ (Barth et al., 2013; Richards et al., 2016). Also, relevant for this criterion, one of the most recent and largest RCTs for MDD conducted found BA to be noninferior (i.e., to have no lesser effect) to CBT.

- Also for BA, one meta-analysis (Ekers et al., 2014) found it to be more efficacious than antidepressant medication, $g = 0.42$. We note the confidence interval for the ES is extremely close to 0, and this result is only based on four trials, so it is most likely not very robust.
- For PST, with regard to standard psychotherapy for depression (CBT, interpersonal therapy, other behavioral therapy), the studies are too few and too heterogeneous to permit conclusions regarding relative efficacy (*preliminary data*) (Cuijpers et al., 2007b). A network meta-analysis using evidence from both direct and indirect comparisons did not find differences between PST and CBT ($g = -0.04$) and respectively PST and BA ($g = -0.06$). However, the number of direct comparisons contributing to this effect was too small (four RCTs for CBT and two RCTs for BA) to safely conclude PST is as efficacious as CBT and BA.

2.2.2.2.3 Specific efficacy for behavioral therapy

- The first criterion, involving the equivalence to other standard therapies (EST), is met for the comparison with CBT (see previous point about relative efficacy).
- The second criterion, regarding the empirical support for the underlying theory of specific mechanisms of change in the case of the therapeutic package, is not met.
- Two component analyses, conducted by the same group, showed that the BA component of a cognitive–behavioral protocol that included both CT and BA was as efficacious on its own as the whole CBT package, both during the full treatment package (Jacobson et al., 1996) and in preventing subsequent relapse at follow-up (Gortner, Gollan, Dobson, & Jacobson, 1998). Nevertheless, these analyses were conducted on the same sample, though at different time points, and in the same research group led by Jacobson.
- We were not able to find other studies investigating mechanisms of change in BA interventions, either in other dismantling analyses or in mediation analyses. As such, we argue that there is only *preliminary data* for the theory of change behind BA.

2.2.2.2.4 Specific efficacy for problem-solving therapy

- The first criterion, involving the equivalence to other standard therapies or better than pill or psychological placebo, is not met, as there is only *preliminary or mixed data* for between-group comparisons (see relative efficacy). For instance, for placebo, Barth et al. (2013) found only three RCTs directly comparing PST with a placebo, resulting in a nonsignificant *g* of −0.23.
- The second criterion, regarding the empirical support for the underlying theory of specific mechanisms of change in the case of the therapeutic package, is not met. We have not been able to find any study investing the mechanisms of change for PST, either in component analyses or in meditational analyses in clinical trials. We would qualify this as *no data* for the theory behind PST.

2.2.3 Category III: Theory-Driven Psychotherapies

Category III includes psychological treatments for which there is strong supporting evidence for the etiological theory for depression. However, the results are equivocal (i.e., *missing*, *preliminary*, or *mixed*) in terms of the efficacy/effectiveness of the existing clinical protocols. We present in the following the theory-driven psychotherapies for MDD.

2.2.3.1 Cognitive bias modification: Concreteness training

Concreteness training (CNT) is a guided self-help intervention consisting of repeated sessions in which participants receive explicit instructions to actively engage in generating concrete representations when imagining emotional events, such as focusing on specific details of an event, what makes it special, and the process regarding how it happened (Watkins, Baeyens, & Read, 2009). The intervention includes a face-to-face component as well as a number of daily home practice sessions, using written or audio-recorded exercises.

Regarding *efficacy*, CNT could be classified as supported by *preliminary data*. Only one RCT (Watkins et al., 2012) showed that CNT added to TAU was more efficacious than TAU alone (*absolute efficacy*) and as efficacious as TAU plus relaxation training (*relative efficacy*).

Regarding *theory*, CNT stemmed from a well-supported theory, as it was developed to target two cognitive processes implicated in depression: rumination and overgeneralization (see Watkins & Moberly, 2009). Experimental studies have indicated that both of these processes predict depressive symptoms prospectively:

2.2.3.1.1 Rumination

- *Experimental studies*: Rumination predicted depressive symptoms prospectively (Ciesla & Roberts, 2007; Nolen-Hoeksema, 2000; Nolen-Hoeksema, Morrow, & Fredrickson, 1993; also see Watkins, 2008, for an extensive review of the evidence).
- *Cross-sectional studies*: Rumination is associated with depressive symptoms in adults (see Watkins, 2008, for a review of the evidence).

2.2.3.1.2 Overgeneralization

- *Experimental studies*: Overgeneralization predicted depressive symptoms prospectively (Carver, 1998; Dykman, 1996; Edelman, Ahrens, & Haaga, 1994) and the abstract, overgeneral bias mediated the effect of failure on subsequent negative affect (Brown & Dutton, 1995; Kernis, Brockner, & Frankel, 1989; Wenzlaff & Grozier, 1988).
- We also note that CNT was shown to modify the cognitive processes it was developed to target in the context of both a proof of concept and a phase II RCT (Watkins et al., 2009, 2012). Nevertheless, these studies were both conducted by the same research group.

2.2.3.2 Rational emotive behavioral therapy

Rational emotive behavioral therapy (REBT) is a variant of CBT developed by Albert Ellis (1994). Like CBT, REBT is a short-term therapy focused on helping clients change aspects of their thinking that contribute to emotional and behavioral problems. Distinct aspects of REBT are the relatively greater focus on unconditional self-acceptance and efforts to reduce demanding beliefs (David, Szentagotai, Lupu, & Cosman, 2008). The APA Division of Clinical Psychology lists REBT as having modest research support. REBT is also included in the updated NICE guidelines for depression (NICE, 2009).

Regarding *efficacy*, REBT could be classified as supported by *preliminary data*. Only one RCT (David et al., 2008) found that REBT was as efficient as two standard treatments: CT and pharmacotherapy (*relative efficacy*). A pilot RCT (Macaskill & Macaskill, 1996) also found the combination of REBT and pharmacotherapy to be more efficacious

than pharmacotherapy alone for depressed patients with high levels of cognitive dysfunction.

Regarding the *theory of change*, four correlational studies conducted by three different groups support the relationship between irrational beliefs, particularly demandingness, and negative mood in depressed individuals (Solomon, Arnow, Gotlib, & Wind, 2003; Solomon, Haaga, Brody, Kirk, & Friedman, 1998; Szentagotai, David, Lupu, & Cosman, 2008; Taghavi, Goodarzi, Kazemi, & Ghorbani, 2006).

2.2.4 Category IV: Investigational Psychotherapies

Category IV includes psychological treatments for which there is neither strong supporting nor contradictory evidence for the efficacy/effectiveness of the clinical protocol, nor for the theory of change underlying the clinical protocol. Thus, the state of the evidence for both effectiveness and mechanisms is equivocal (i.e., *missing*, *preliminary*, or *mixed*). We present in the following the intervention-driven psychotherapies for MDD.

2.2.4.1 Short-term psychodynamic psychotherapy

Short-term psychodynamic psychotherapy (STPP) is generally focused on increasing patients' awareness and insight regarding problematic patterns and core relational themes connected to depression. Although this approach is used in clinical trials, assessing the efficacy of STPP is somewhat complicated by the fact that not all of these psychodynamic interventions use treatment manuals, and sometimes they are not specifically addressed to treat depression. Common focuses cited in STPP (Society of Clinical Psychology, 2017) include how past experiences influence (1) current functioning, (2) affect and the expression of emotion, (3) the therapeutic relationship, (4) the facilitation of insight, (5) avoidance of uncomfortable topics, and (6) the identification of core conflictual relationship themes.

The APA Division of Clinical Psychology lists STPP as having modest research support. According to NICE guidelines, STPP should only be considered for people with mild to moderate depression or persistent subthreshold depressive symptoms who decline the recommended high-intensity psychological interventions or antidepressants. Moreover, discussing the uncertainty of effectiveness of STTP with the patient is also recommended (NICE, 2009). According to David and Montgomery's classification (2011), we argue that STPP belongs in Category IV (investigational psychotherapies) for MDD.

In terms of its treatment package, we will refer to reviews and meta-analyses (Abbass et al., 2014; Barth et al., 2013; Driessen et al., 2015) regarding the efficacy of STPP for major depression.

2.2.4.1.1 Absolute efficacy

- An updated Cochrane Review (Abbass et al., 2014) found STPP to be more efficacious than a control conditions (waitlist, TAU, other) for short-term depression outcomes in patients with depressive disorders in five RCTs with a mean ES (Cohen's d) of 0.47. A meta-analysis (Driessen et al., 2015) found STPP to be more efficacious than control (waitlist, TAU, placebo) in 10 trials, with a pooled ES of 0.60, corresponding to an NNT of 2.99. However, a network meta-analysis (Barth et al., 2013) found psychodynamic therapy to be no better than waitlist ($d = -1.46$ ns) or usual care ($d = -0.32$ ns)

in direct comparisons. The meta-analysis did, however, find STPP to be more efficacious than waitlist ($d = 0.72$) and usual care ($d = 0.39$) when indirect comparisons were also considered.

2.2.4.1.2 Relative efficacy

- Meta-analytical data regarding the specific comparison between STPP and CBT is scarce. A network meta-analysis (Barth et al., 2013) using evidence from both direct and indirect comparisons found no differences between STPP and CBT. However, in one recent meta-analysis (Driessen et al., 2015), STPP was found to be *less* efficacious than other psychotherapies taken together (a category that included both CBT and other types of psychotherapy) in 15 trials, with an ES of $d = -0.25$. Unfortunately, the authors did not conduct a subgroup analysis examining only the comparison between STPP and CBT. In the same meta-analysis, no differences were found between STPP and antidepressant medication, but this result was based on only four RCTs and might not be very robust.

2.2.4.1.3 Specific efficacy

- Combining these findings, we categorize the specific efficacy of STPP as *preliminary data* or *mixed data*.

Regarding support for the underlying psychodynamic therapy, the results are *mixed*. In one study, Coleman, Cole, and Wuest (2010) did not find evidence that two hypothesized psychoanalytic change mechanisms (early and mature defenses) predicted symptom change among depressed individuals receiving TAU as opposed to no treatment. In another study, Gibbons et al. (2009) reported that a change mechanism considered to be psychoanalytic (improvement in self-understanding) predicted changes in depression across both psychodynamic treatment and CBT. Nevertheless, when other predictors were considered (i.e., change in compensatory cognitive skills and change in self-concept), this mechanism was no longer significantly related to outcomes. Consequently, we would qualify STPP as *mixed data*. In line with Hollon and Ponniah (2010), we believe that even if there is no compelling evidence so far regarding the specific efficacy of STPP, and reduced and mixed evidence regarding its mechanisms of change, it would be premature to discard it as not efficacious.

2.2.4.2 "Third-wave" cognitive–behavioral psychotherapies

"Third-wave" cognitive–behavioral psychotherapies deem cognitive change to be in no way essential to producing therapeutic change. Instead, they focus on different processes and employ a less didactic and a more experiential approach to clients' beliefs. The processes include constructs such as acceptance, experiential avoidance or psychological flexibility, defusion, and values clarification (Hayes, Luoma, Bond, Masuda, & Lillis, 2006).

Two Cochrane collaborative systematic reviews examined the efficacy of third-wave cognitive–behavioral interventions on depressed patients, when compared with TAU and when compared with other psychological therapies (Churchill et al., 2013; Hunot et al., 2013). Noting that there is still some disagreement regarding which therapies are considered "third wave" (see Hayes, Masuda, Bissett, Luoma, & Guerrero, 2004; Hofmann, Sawyer, & Fang, 2010), the reviewers included the following third-wave

therapies: acceptance and commitment therapy (ACT) (Hayes et al., 2004), compassionate mind training (Gilbert, 2005), functional analytic psychotherapy (Kohlenberg & Tsai, 1991), metacognitive therapy (Wells, 2008), mindfulness-based CT (Teasdale, Segal, & Williams, 1995), dialectical behavior therapy (Linehan, 1993), and the expanded model of BA (Martell, Addis, & Jacobson, 2001). We note that the APA Division of Clinical Psychology also includes one of these therapies (ACT) as having modest research support for depression.

2.2.4.2.1 Absolute efficacy

- Third-wave therapies were more efficacious than a control condition (waitlist, usual care, no treatment) in three RCTs, yielding a risk ratio of 0.51 (Churchill et al., 2013). However, the same principal author (Zettle & Hayes, 1986; Zettle & Rains, 1989) conducted two of these RCTs.

2.2.4.2.2 Relative efficacy

- Third-wave therapies were not different from other psychological treatments (mostly various forms of CT) in the three RCTs that compared them (risk ratio = 1.14) (Hunot et al., 2013).

Based on these considerations, we would categorize the efficacy of third-wave cognitive–behavioral therapies as *preliminary data*.

Regarding the underlying theory supporting these interventions, data remain scarce and *mixed*. We were able to find an empirical investigation of mechanisms of change only for ACT. One study (Zettle, Rains, & Hayes, 2011), which reanalyzed the data of a previous RCT, offered partial evidence of mechanisms of change by showing that posttreatment levels of cognitive defusion (a hypothesized process of change in which participants do not "identify with" or "fuse with" their negative thoughts) mediated the effect of ACT at follow-up. Another study (Forman et al., 2012), using a mixed sample of patients referred for anxiety and depression, reported mixed results, finding evidence of mediation in the ACT treatment arm for some hypothesized ACT processing of change (utilization of psychological acceptance strategies) but also for some CT mechanisms of change (decreases in dysfunctional thinking). It remains unclear whether these mechanisms are intercorrelated or whether one is a consequence of the other. Accordingly, it is impossible to ascertain whether the effects of ACT on depressive outcomes are produced via its postulated mechanisms of change or rather reflect changes in processes more akin to the postulated mechanisms of other therapies (e.g., CT). Consequently, we would code the evidence for the mechanisms of change of third-wave cognitive–behavioral therapies as *preliminary data* and *mixed data*.

2.2.4.3 Behavioral couple therapy

Behavioral couple therapy (BCT) is an intervention for couples with relationship distress and a depressed partner. Its goal is to simultaneously decrease depression and improve relationship functioning. The therapy addresses couple distress and focuses on fostering closeness and support by increasing caring behaviors as well as decreasing conflict.

The APA Division of Clinical Psychology lists BCT as having modest support. According to NICE guidelines, BCT is recommended for mild to moderate depression and

persistent subthreshold depressive symptoms for people for whom the relationship with a regular partner may contribute to the symptoms or in which involving the partner is considered potentially beneficial therapeutically (NICE, 2009).

Evidence for the efficacy of BCT for depression is mixed. Two narrative reviews (Gupta, Coyne, & Beach, 2003; Hollon & Ponniah, 2010) examined BCT trials and concluded that the small number of studies, mixed outcomes, and various methodological problems (e.g., small samples, types of patients) preclude drawing firm conclusions regarding the efficacy of BCT for depression, although the intervention seems to be efficacious for marital distress. We would qualify BCT as supported by *preliminary data* and *mixed data*. Regarding theory of change, we were not able to find any evidence regarding mechanisms by which BCT reduces depressive symptoms, and as such qualified it as *no data*.

2.2.5 Categories V, VI, VII, VIII, and IX

These categories refer to:

- good-intervention- and bad-theory-driven psychotherapies (Category V);
- good-theory- and bad-intervention-driven psychotherapies (Category VI);
- bad-theory-driven psychotherapies (Category VII);
- bad-intervention-driven psychotherapies (Category VIII);
- bad-theory- and bad-intervention-driven psychotherapies (Category IX).

We were not able to identify any psychotherapies in these five categories (except, possibly, for grief therapy). We note that each of these categories requires strong contradictory evidence for either the therapeutic package or the theory, more precisely invalidation in at least two rigorous trials, conducted by at least two independent investigating teams. While a number of psychotherapies probably have no or even harmful effects, they do not seem to make it outside "the file drawer" and into scientific journals. In fact, some researchers (Lilienfeld, 2007) have argued that the search for possibly harmful therapies should be prioritized *over* the search for empirically supported ones.

In addition, we note that some interventions (e.g., dance movement therapy, tai chi) are based on theories and constructs that are difficult or impossible to measure and test (i.e., not falsifiable). However, such treatments are not consistent with our focus—namely, psychotherapies based on psychological theories and interventions.

As a potential example of a psychotherapy with strong contradictory evidence for MDD, we mention grief therapy, even when it does not specifically address patients with depression and targets normal bereavement reactions. A meta-analysis (Neimeyer, 2000) found that 38% of clients receiving grief therapy would have achieved superior end-state functioning if they had been assigned to a no-treatment condition. Moreover, the percentage of deteriorated clients (i.e., who exhibited worse functioning following therapy than at its outset) approached 50% among individuals who experienced "normal" as opposed to "traumatic" grief reactions. We believe these findings qualify grief therapy as having strong contradictory evidence for its therapeutic package (strong contradictory evidence). Nevertheless, it is possible grief therapy could produce better results in treating conditions other than depression.

2.3 Conclusions and Discussion

2.3.1 Implications for Research

One important implication of our analysis is that research regarding the efficacy/effectiveness of psychotherapeutic packages is very advanced, at least in the case of MDD. We identified several recent meta-analyses for the psychotherapies assessed, with many of these therapies enjoying a large evidence base of RCTs. Nevertheless, whereas many researchers have conducted RCTs, most of the reviews and meta-analyses pointed to a less than optimal *quality* of these studies (Cuijpers, 2017; Cuijpers, Cristea et al., 2016; Cuijpers, Smit, Bohlmeijer, Hollon, & Andersson, 2010; Cuijpers, Van Straten, Bohlmeijer, Hollon, & Andersson, 2010). Our analysis did not focus specifically on the quality of the studies supporting a therapeutic intervention, but we believe this is an important aspect to consider in evaluating therapies for depression. Particularly given that we have established that some psychotherapies are clearly efficacious in RCTs, the field could move to evaluating them in better quality trials, actively controlling possible sources of bias, such as those identified in the Cochrane Risk of Bias assessment tool (Higgins et al., 2011).

If we have strong evidence for the efficacy of a few psychotherapeutic interventions for MDD, the same cannot be said about associated theories and mechanisms of change. In fact, we were only able to identify one psychotherapy that would warrant placement in Category I of David and Montgomery's (2011) classification framework, namely CT. Nevertheless, even in this case, although studies and reviews support mechanisms of change constructs that are postulated by the cognitive theory of depression, reviews and meta-analyses also report negative evidence (Cristea et al., 2015; Kazdin, 2007; Longmore & Worrell, 2007). More and better quality research is thus also needed in the case of CT, even if it merits inclusion as a Category I intervention.

In a seminal article, Kazdin (2007) offered a list of reasons for studying mechanisms of change in psychotherapy. For example, given the plethora of current treatments, understanding what makes a certain intervention work can bring "order and parsimony" (p. 4) to the status quo of multiple interventions existing for the same disorder. We believe this argument is very germane to the case of MDD. As we noted, conclusive evidence for mechanisms of change is not available for many psychotherapies. In fact, one of the important psychotherapies for MDD (i.e., IPT), with proven efficacy in at least four meta-analyses of RCTs, literally has *no* study or analysis evaluating its mechanisms of change (Lipsitz & Markowitz, 2013). Only preliminary evidence for postulated mechanisms of change exists for behavior therapy, another psychotherapy with confirmed efficacy based on recent meta-analyses.

We believe, in line with Kazdin (2007) and other researchers in the field, that it is imperative that this state of the science of psychotherapy changes, and that researchers accord importance to evaluating mechanisms of change and identifying specific mediators of treatment response, particularly for the therapies that have been proven to be efficacious and are recognized as such by the current clinical guidelines. Developing RCTs, such as Lemmens et al. (2011), represent promising initiatives to address these deficiencies in our knowledge base.

Interestingly, we were able to identify only two psychotherapies in Category III (i.e., theory-driven psychotherapies). One of these promising interventions (CNT)

represents a rare, prototypical case of starting from theoretical constructs validated in experimental studies and developing an intervention to target them, following all the necessary phases: proof of principle (Watkins & Moberly, 2009), proof of concept (Watkins et al., 2009), and phase II testing in a RCT of depressed individuals (Watkins et al., 2012). More evidence from other, independent research groups is needed to assess the efficacy of the intervention, but CNT has the potential to represent a successful case of a theory-driven, top-down psychotherapy.

On the other side of David and Montgomery's (2011) framework, our analysis revealed a paucity of knowledge and studies regarding therapies and/or their theories with strong contradictory evidence (i.e., having been invalidated in rigorous studies). Other researchers have also raised alarm over potentially harmful psychotherapies (i.e., Categories VI, VIII, and IX in our framework), underscoring that their search should be prioritized *over* the search for evidence-based treatments: "Our field should therefore instead prioritize its efforts toward pinpointing treatments that are demonstrably harmful or ineffective and disseminating this information to current practitioners, students in training, potential clients, managed-care organizations, and the media" (Lilienfeld, 2007, p. 63).

We noted that some therapies (e.g., dance movement therapy, tai chi) are based on theories and constructs that are difficult or *impossible* to measure and test (i.e., not falsifiable). We suggest that it might be worthwhile to extend Categories V, VII, VIII, and IX to include *nonfalsifiable* therapies, at least until researchers associated with such interventions develop measurable, testable constructs (i.e., similar to what happened in psychoanalysis). Moreover, as mentioned previously, most of these "therapies" are not consistent with our focus—namely, psychotherapies (i.e., psychological treatments) that are based on psychological theories and interventions.

2.3.2 Implications for Practice

Our evaluation of psychotherapies based on David and Montgomery's (2011) framework found strong evidence for a number of psychotherapies for MDD in line with current clinical recommendations and guidelines (e.g., APA Division of Clinical Psychology, NICE, the Cochrane Collaboration). At the same time, we found no evidence or at best preliminary evidence for the postulated mechanisms of change pertinent to most of these therapies, despite their proven efficacy. We believe this finding holds important practice implications.

First, as Kazdin (2007) cogently argued, until we understand the processes that drive psychotherapeutic change, we will have difficulty optimizing interventions, as we lack the knowledge to understand what is critical in treatment and how it drives effects on outcomes of interest. Moreover, a lack of understanding of what makes psychotherapies work and what does not will make transitions from controlled clinical settings to real-world settings more challenging (Kazdin, 2007). Finally, understanding key processes in psychotherapies that instigate positive effects can help identify moderators of treatment response and help therapists select patients most suitable for specific interventions (Kazdin, 2007), thereby building more personalized, tailored interventions for patient subgroups. These crucial efforts are particularly relevant for the therapy of depression, which is often a chronic, heterogeneous condition, with moderate rates of response to therapy (i.e., around 60%).

The lack of convincing evidence for mechanisms that underlie successful therapy packages also holds ethical implications for psychotherapy practice. Should patients be informed that they are receiving a therapy that works but that the scientific community has not to date established by what mechanisms? The current clinical guidelines and accompanying patient materials are typically focused on efficacy; however, in actual therapy, the *theory* or the mechanisms that a given psychotherapy purportedly impacts closely inform case formulation, an essential process in psychotherapy (Macneil, Hasty, Conus, & Berk, 2012). It might be the case that providing information about the lack of evidence regarding therapeutic mechanisms could hamper the efficacy or effectiveness of a given psychotherapy. Still, withholding such information might be unethical and risk a breach of confidence in the therapeutic relationship. Consequently, it is important to underline the pressing need for serious discussions within the field about ethical issues such as those outlined previously.

Another important implication of our analysis is that the field of psychotherapy research lacks studies that seek to *invalidate* therapeutic interventions and theories. The reasons for this circumstance are likely complex, but the current publishing standards, favoring the publication of positive results and inhibiting the publication of negative findings, probably rank among the most important reasons. From a clinician's vantage point, this leads to a frustrating situation, as the "market" is flooded with a wealth of interventions for every mental health problem, and the practitioner effectively lacks the evidence necessary to dismiss certain interventions convincingly. More generally, the lack of strong evidence invalidating psychotherapies and/or their underlying theories helps perpetuate misinformation and outright fraud in offering treatment. In line with Lilienfeld (2007), we believe that focusing on invalidating therapeutic interventions and theories has important practical ramifications, given that it is easier to "proscribe than to prescribe clinical practices" (p. 63).

Our list of psychotherapies is certainly not exhaustive, and more psychotherapies for MDD exist and could be considered in this analysis. Nevertheless, it is possible to advance a number of tentative conclusions, as follows:

- There are strong data for the efficacy of at least three psychotherapy packages for MDD (i.e., BA, CT, and interpersonal therapy); however, empirical support for the theory of change exists only in the case of CT.
- Research on the mechanisms of change of psychotherapies lags far behind research on their efficacy, and this also holds true for psychotherapies with strong evidence for their efficacy.
- Rigorous development of a top-down, theory-driven psychotherapy for depression is a rare undertaking; we were able to find just one such example for depression (i.e., CNT).
- We only detailed three types of psychotherapies, presented in clinical guidelines, for Category IV (investigational psychotherapy); however, we note that most psychotherapeutic interventions for MDD would probably warrant placement in this category. Placing diverse psychotherapies in this broad category might hinder distinguishing therapies with high potential to become empirically validated from therapies with no such potential.
- Research on invalidating psychotherapies and/or their theories is scarce, precluding identifying interventions with strong contradictory evidence.

- David and Montgomery's (2011) evaluative framework does not encompass nonfalsifiable theories of change. Such a category could be added or folded into a category or criterion that represents strong contradictory evidence for therapies based on constructs that are not readily operationalized.
- Generally, the field of psychotherapy for depression has a *good* standing with regard to evidence-based psychotherapies and a *less than optimal* standing with regard to evaluating evidence-based theories of change.

References

Abbass, A. A., Kisely, S. R., Town, J. M., Leichsenring, F., Driessen, E., De Maat, S., … Crowe, E. (2014). Short-term psychodynamic psychotherapies for common mental disorders. *Cochrane Database of Systematic Reviews*, *7*, CD004687. doi:10.1002/14651858.CD004687.pub4

Abela, J. R. Z., & D'Alessandro, D. U. (2002). Beck's cognitive theory of depression: A test of the diathesis-stress and causal mediation components. *British Journal of Clinical Psychology*, *41*(Pt. 2), 111–128. doi:10.1348/014466502163912

Altman, D. G. (1998). Confidence intervals for the number needed to treat. *BMJ*, *317*(7168), 1309–1312. doi:10.1136/bmj.317.7168.1309

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.

Andrews, G., Sanderson, K., Corry, J., & Lapsley, H. M. (2000). Using epidemiological data to model efficiency in reducing the burden of depression. *Journal of Mental Health Policy and Economics*, *3*(4), 175–186. doi:10.1002/mhp.96

Barth, J., Munder, T., Gerger, H., Nüesch, E., Trelle, S., Znoj, H., … Cuijpers, P. (2013). Comparative efficacy of seven psychotherapeutic interventions for patients with depression: A network meta-analysis. *PLOS Medicine*, *10*(5), e1001454. doi:10.1371/journal.pmed.1001454

Beck, A. T., & Dozois, D. J. A. (2011). Cognitive therapy: Current status and future directions. *Annual Review of Medicine*, *62*, 397–409. doi:10.1146/annurev-med-052209-100032

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York, NY: Guilford Press.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *BDI-II, Beck Depression Inventory: Manual*. San Antonio, TX: Psychological Corporation.

Beck, J. S. (1995). *Cognitive therapy: Basics and beyond*. New York, NY: Guilford Press.

Beevers, C. G., & Miller, I. W. (2005). Unlinking negative cognition and symptoms of depression: Evidence of a specific treatment effect for cognitive therapy. *Journal of Consulting and Clinical Psychology*, *73*(1), 68–77. doi:10.1037/0022-006X.73.1.68

Berrettini, W. (2002). Psychiatric pharmacogenetics: A developing science. *Neuropsychopharmacology*, *26*(1), 128–129. doi:10.1016/S0893-133X(01)00377-3

Boland, R., & Keller, M. (2009). Course and outcome of depression. In I. H. Gotlib & C. L. Hammen, *Handbook of depression* (2nd ed., pp. 23–43). New York, NY: Guilford Press.

Braun, S. R., Gregor, B., & Tran, U. S. (2013). Comparing bona fide psychotherapies of depression in adults with two meta-analytical approaches. *PLOS One*, *8*(6), e68135. doi:10.1371/journal.pone.0068135

Brown, J. D., & Dutton, K. A. (1995). The thrill of victory, the complexity of defeat: Self-esteem and people's emotional reactions to success and failure. *Journal of Personality and Social Psychology*, *68*(4), 712–722. doi:10.1037//0022-3514.68.4.712

Carver, C. S. (1998). Generalization, adverse events, and development of depressive symptoms. *Journal of Personality*, *66*(4), 607–619. doi:10.1111/1467-6494.00026

Churchill, R., Moore, T. H. M., Furukawa, T. A., Caldwell, D. M., Davies, P., Jones, H., … Hunot, V. (2013). "Third wave" cognitive and behavioural therapies versus treatment as usual for depression. *Cochrane Database of Systematic Reviews*, *10*, CD008705. doi:10.1002/14651858.CD008705.pub2

Ciesla, J. A., & Roberts, J. E. (2007). Rumination, negative cognition, and their interactive effects on depressed mood. *Emotion*, *7*(3), 555–565. doi:10.1037/1528-3542.7.3.555

Clark, D. A., & Beck, A. T. (2010). Cognitive theory and therapy of anxiety and depression: Convergence with neurobiological findings. *Trends in Cognitive Sciences*, *14*(9), 418–424. doi:10.1016/j.tics.2010.06.007

Coleman, D., Cole, D., & Wuest, L. (2010). Cognitive and psychodynamic mechanisms of change in treated and untreated depression. *Journal of Clinical Psychology*, *66*(3), 215–228. doi:10.1002/jclp.20645

Cristea, I. A., Huibers, M. J. H., David, D., Hollon, S. D., Andersson, G., & Cuijpers, P. (2015). The effects of cognitive behavior therapy for adult depression on dysfunctional thinking: A meta-analysis. *Clinical Psychology Review*, *42*, 62–71. doi:10.1016/j.cpr.2015.08.003

Cuijpers, P. (2017). Four decades of outcome research on psychotherapies for adult depression: An overview of a series of meta-analyses. *Canadian Psychology/Psychologie Canadienne*, *58*(1), 7–19. doi:10.1037/cap0000096

Cuijpers, P., Berking, M., Andersson, G., Quigley, L., Kleiboer, A., & Dobson, K. S. (2013). A meta-analysis of cognitive-behavioural therapy for adult depression, alone and in comparison with other treatments. *Canadian Journal of Psychiatry/Revue Canadienne de Psychiatrie*, *58*(7), 376–385. doi:10.1177/070674371305800702

Cuijpers, P., Cristea, I. A., Karyotaki, E., Reijnders, M., & Huibers, M. J. H. (2016). How effective are cognitive behavior therapies for major depression and anxiety disorders? A meta-analytic update of the evidence. *World Psychiatry*, *15*(3), 245–258. doi:10.1002/wps.20346

Cuijpers, P., Donker, T., Weissman, M. M., Ravitz, P., & Cristea, I. A. (2016). Interpersonal psychotherapy for mental health problems: A comprehensive meta-analysis. *American Journal of Psychiatry*, *173*(7), 680–687. doi:10.1176/appi.ajp.2015.15091141

Cuijpers, P., Hollon, S. D., Van Straten, A., Bockting, C., Berking, M., & Andersson, G. (2013). Does cognitive behaviour therapy have an enduring effect that is superior to keeping patients on continuation pharmacotherapy? A meta-analysis. *BMJ Open*, *3*(4). doi:10.1136/bmjopen-2012-002542

Cuijpers, P., Smit, F., Bohlmeijer, E., Hollon, S. D., & Andersson, G. (2010). Efficacy of cognitive-behavioural therapy and other psychological treatments for adult depression: Meta-analytic study of publication bias. *British Journal of Psychiatry*, *196*(3), 173–178. doi:10.1192/bjp.bp.109.066001

Cuijpers, P., Turner, E. H., Mohr, D. C., Hofmann, S. G., Andersson, G., Berking, M., & Coyne, J. (2014). Comparison of psychotherapies for adult depression to pill placebo control groups: A meta-analysis. *Psychological Medicine*, *44*(4), 685–695. doi:10.1017/S0033291713000457

Cuijpers, P., Van Straten, A., Andersson, G., & Van Oppen, P. (2008). Psychotherapy for depression in adults: A meta-analysis of comparative outcome studies. *Journal of Consulting and Clinical Psychology*, *76*(6), 909–922. doi:10.1037/a0013075

Cuijpers, P., Van Straten, A., Bohlmeijer, E., Hollon, S. D., & Andersson, G. (2010). The effects of psychotherapy for adult depression are overestimated: A meta-analysis of study quality and effect size. *Psychological Medicine*, *40*(2), 211–223. doi:10.1017/S0033291709006114

Cuijpers, P., Van Straten, A., & Warmerdam, L. (2007a). Behavioral activation treatments of depression: A meta-analysis. *Clinical Psychology Review*, *27*(3), 318–326. doi:10.1016/j.cpr.2006.11.001

Cuijpers, P., Van Straten, A., & Warmerdam, L. (2007b). Problem solving therapies for depression: A meta-analysis. *European Psychiatry*, *22*(1), 9–15. doi:10.1016/j.eurpsy.2006.11.001

David, D., & Montgomery, G. H. (2011). The scientific status of psychotherapies: A new evaluative framework for evidence-based psychosocial interventions. *Clinical Psychology: Science and Practice*, *18*(2), 89–99. doi:10.1111/j.1468-2850.2011.01239.x

David, D., Szentagotai, A., Lupu, V., & Cosman, D. (2008). Rational emotive behavior therapy, cognitive therapy, and medication in the treatment of major depressive disorder: A randomized clinical trial, posttreatment outcomes, and six-month follow-up. *Journal of Clinical Psychology*, *64*(6), 728–746. doi:10.1002/jclp.20487

DeRubeis, R. J., Hollon, S. D., Amsterdam, J. D., Shelton, R. C., Young, P. R., Salomon, R. M., … Gallop, R. (2005). Cognitive therapy vs medications in the treatment of moderate to severe depression. *Archives of General Psychiatry*, *62*(4), 409–416. doi:10.1001/archpsyc.62.4.409

Driessen, E., Hegelmaier, L. M., Abbass, A. A., Barber, J. P., Dekker, J. J. M., Van, H. L., … Cuijpers, P. (2015). The efficacy of short-term psychodynamic psychotherapy for depression: A meta-analysis update. *Clinical Psychology Review*, *42*, 1–15. doi:10.1016/j.cpr.2015.07.004

Dykman, B. M. (1996). Negative self-evaluations among dysphoric college students: A difference in degree or kind? *Cognitive Therapy and Research*, *20*(5), 445–464. doi:10.1007/BF02227907

Edelman, R. E., Ahrens, A. H., & Haaga, D. A. F. (1994). Inferences about the self, attributions, and overgeneralization as predictors of recovery from dysphoria. *Cognitive Therapy and Research*, *18*(6), 551–566. doi:10.1007/BF02355668

Ekers, D., Webster, L., Straten, A. V., Cuijpers, P., Richards, D., & Gilbody, S. (2014). Behavioural activation for depression: An update of meta-analysis of effectiveness and sub group analysis. *PLOS One*, *9*(6), e100100. doi:10.1371/journal.pone.0100100

Ellis, A. (1994). *Reason and emotion in psychotherapy* (Rev. ed.). Secaucus, NJ: Birch Lane Press.

Ferrari, A. J., Charlson, F. J., Norman, R. E., Patten, S. B., Freedman, G., Murray, C. J. L., … Whiteford, H. A. (2013). Burden of depressive disorders by country, sex, age, and year: Findings from the Global Burden of Disease Study 2010. *PLOS Medicine*, *10*(11), e1001547. doi:10.1371/journal.pmed.1001547

Forman, E. M., Chapman, J. E., Herbert, J. D., Goetter, E. M., Yuen, E. K., & Moitra, E. (2012). Using session-by-session measurement to compare mechanisms of action for acceptance and commitment therapy and cognitive therapy. *Behavior Therapy*, *43*(2), 341–354. doi:10.1016/j.beth.2011.07.004

Furukawa, T. A., Weitz, E. S., Tanaka, S., Hollon, S. D., Hofmann, S. G., Andersson, G., … Cuijpers, P. (2017). Initial severity of depression and efficacy of cognitive–behavioural therapy: Individual-participant data meta-analysis of pill-placebo-controlled trials. *British Journal of Psychiatry*, *210*(3), 190–196. doi:10.1192/bjp.bp.116.187773

Garratt, G., Ingram, R. E., Rand, K. L., & Sawalani, G. (2007). Cognitive processes in cognitive therapy: Evaluation of the mechanisms of change in the treatment of depression. *Clinical Psychology: Science and Practice*, *14*(3), 224–239. doi:10.1111/j.1468-2850.2007.00081.x

Gartlehner, G., Hansen, R. A., Morgan, L. C., Thaler, K., Lux, L. J., Van Noord, M., … Lohr, K. N. (2011). *Second-generation antidepressants in the pharmacologic treatment of adult depression: An update of the 2007 Comparative Effectiveness Review*. Rockville, MD: Agency for Healthcare Research and Quality. Retrieved September 20, 2017, from http://www.ncbi.nlm.nih.gov/books/NBK83442

Gibbons, M. B. C., Crits-Christoph, P., Barber, J. P., Wiltsey Stirman, S., Gallop, R., Goldstein, L. A., … Ring-Kurtz, S. (2009). Unique and common mechanisms of change across cognitive and dynamic psychotherapies. *Journal of Consulting and Clinical Psychology*, *77*(5), 801–813. doi:10.1037/a0016596

Gilbert, P. (2005). *Compassion: Conceptualisations, research and use in psychotherapy*. Hove, UK: Routledge.

Gilmer, W. S., Trivedi, M. H., Rush, A. J., Wisniewski, S. R., Luther, J., Howland, R. H., … Alpert, J. (2005). Factors associated with chronic depressive episodes: A preliminary report from the STAR-D Project. *Acta Psychiatrica Scandinavica*, *112*(6), 425–433. doi:10.1111/j.1600-0447.2005.00633.x

Gortner, E. T., Gollan, J. K., Dobson, K. S., & Jacobson, N. S. (1998). Cognitive-behavioral treatment for depression: Relapse prevention. *Journal of Consulting and Clinical Psychology*, *66*(2), 377–384. doi:10.1037//0022-006x.66.2.377

Greenberg, P. E., Fournier, A.-A., Sisitsky, T., Pike, C. T., & Kessler, R. C. (2015). The economic burden of adults with major depressive disorder in the United States (2005 and 2010). *Journal of Clinical Psychiatry*, *76*(2), 155–162. doi:10.4088/JCP.14m09298

Greenberg, P. E., Kessler, R. C., Birnbaum, H. G., Leong, S. A., Lowe, S. W., Berglund, P. A., & Corey-Lisle, P. K. (2003). The economic burden of depression in the United States: How did it change between 1990 and 2000? *Journal of Clinical Psychiatry*, *64*(12), 1465–1475. doi:10.4088/jcp.v64n1211

Gupta, M., Coyne, J. C., & Beach, S. R. H. (2003). Couples treatment for major depression: Critique of the literature and suggestions for some different directions. *Journal of Family Therapy*, *25*(4), 317–346. doi:10.1111/1467-6427.00253

Hamilton, K. E., & Dobson, K. S. (2002). Cognitive therapy of depression: Pretreatment patient predictors of outcome. *Clinical Psychology Review*, *22*(6), 875–893. doi:10.1016/s0272-7358(02)00106-x

Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery, and Psychiatry*, *23*, 56–62. doi:10.1136/jnnp.23.1.56

Hayes, S. C., Luoma, J. B., Bond, F. W., Masuda, A., & Lillis, J. (2006). Acceptance and commitment therapy: Model, processes and outcomes. *Behaviour Research and Therapy*, *44*(1), 1–25. doi:10.1016/j.brat.2005.06.006

Hayes, S. C., Masuda, A., Bissett, R., Luoma, J., & Guerrero, L. F. (2004). DBT, FAP, & ACT: How empirically oriented are the new behavior therapy technologies? *Behavior Therapy*, *35*(1), 35–54. doi:10.1016/S0005-7894(04)80003-0

Higgins, J. P. T., Altman, D. G., Gotzsche, P. C., Juni, P., Moher, D., Oxman, A. D., … Cochrane Statistical Methods Group. (2011). The Cochrane Collaboration's tool for assessing risk of bias in randomised trials. *BMJ*, *343*, d5928–d5928. doi:10.1136/bmj.d5928

Hofmann, S. G., Sawyer, A. T., & Fang, A. (2010). The empirical status of the "new wave" of cognitive behavioral therapy. *Psychiatric Clinics of North America*, *33*(3), 701–710. doi:10.1016/j.psc.2010.04.006

Hollon, S. D., Muñoz, R. F., Barlow, D. H., Beardslee, W. R., Bell, C. C., Bernal, G., … Sommers, D. (2002). Psychosocial intervention development for the prevention and treatment of depression: Promoting innovation and increasing access. *Biological Psychiatry*, *52*(6), 610–630. doi:10.1016/s0006-3223(02)01384-7

Hollon, S. D., & Ponniah, K. (2010). A review of empirically supported psychological therapies for mood disorders in adults. *Depression and Anxiety*, *27*(10), 891–932. doi:10.1002/da.20741

Hundt, N. E., Mignogna, J., Underhill, C., & Cully, J. A. (2013). The relationship between use of CBT skills and depression treatment outcome: A theoretical and methodological review of the literature. *Behavior Therapy*, *44*(1), 12–26. doi:10.1016/j.beth.2012.10.001

Hunot, V., Moore, T. H. M., Caldwell, D. M., Furukawa, T. A., Davies, P., Jones, H., … Churchill, R. (2013). "Third wave" cognitive and behavioural therapies versus other psychological therapies for depression. *Cochrane Database of Systematic Reviews*, *10*, CD008704. doi:10.1002/14651858.CD008704.pub2

Jacobson, N. S., Dobson, K. S., Truax, P. A., Addis, M. E., Koerner, K., Gollan, J. K., … Prince, S. E. (1996). A component analysis of cognitive-behavioral treatment for depression. *Journal of Consulting and Clinical Psychology*, *64*(2), 295–304. doi:10.1037//0022-006x.64.2.295

Jarrett, R. B., Vittengl, J. R., Doyle, K., & Clark, L. A. (2007). Changes in cognitive content during and following cognitive therapy for recurrent depression: Substantial and enduring, but not predictive of change in depressive symptoms. *Journal of Consulting and Clinical Psychology*, *75*(3), 432–446. doi:10.1037/0022-006X.75.3.432

Kazdin, A. E. (2007). Mediators and mechanisms of change in psychotherapy research. *Annual Review of Clinical Psychology*, *3*, 1–27. doi:10.1146/annurev.clinpsy.3.022806.091432

Kernis, M. H., Brockner, J., & Frankel, B. S. (1989). Self-esteem and reactions to failure: The mediating role of overgeneralization. *Journal of Personality and Social Psychology*, *57*(4), 707–714. doi:10.1037/0022-3514.57.4.707

Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of twelve-month DSM-IV disorders in the National Comorbidity Survey Replication (NCS-R). *Archives of General Psychiatry*, *62*(6), 617–627. doi:10.1001/archpsyc.62.6.617

Kessler, R. C., McGonagle, K. A., Swartz, M., Blazer, D. G., & Nelson, C. B. (1993). Sex and depression in the National Comorbidity Survey. I: Lifetime prevalence, chronicity and recurrence. *Journal of Affective Disorders*, *29*(2/3), 85–96. doi:10.1016/0165-0327(93)90026-g

Klerman, J. L., Weissman, M. M., Rounsaville, B. J., & Chevron, E. S. (1984). *Interpersonal psychotherapy of depression*. New York, NY: Basic Books.

Kohlenberg, R. J., & Tsai, M. (1991). *Functional analytic psychotherapy: A guide for creating intense and curative therapeutic relationships*. New York, NY: Plenum Press.

Kornstein, S. G., & Schneider, R. K. (2001). Clinical features of treatment-resistant depression. *Journal of Clinical Psychiatry*, *62*(Suppl. 16), 18–25.

Lemmens, L. H. J. M., Arntz, A., Peeters, F. P. M. L., Hollon, S. D., Roefs, A., & Huibers, M. J. H. (2011). Effectiveness, relapse prevention and mechanisms of change of cognitive therapy vs. interpersonal therapy for depression: Study protocol for a randomised controlled trial. *Trials*, *12*, 150. doi:10.1186/1745-6215-12-150

Lewinsohn, P. M. (1974). Clinical and theoretical aspects of depression. In K. S. Calhoun, H. E. Adams, & K. M. Mitchell, *Innovative treatment methods of psychopathology* (pp. 63–120). New York, NY: John Wiley & Sons.

Lilienfeld, S. O. (2007). Psychological treatments that cause harm. *Perspectives on Psychological Science*, *2*(1), 53–70. doi:10.1111/j.1745-6916.2007.00029.x

Linehan, M. M. (1993). *Cognitive-behavioral treatment of borderline personality disorder*. New York, NY: Guilford Press.

Lipsitz, J. D., & Markowitz, J. C. (2013). Mechanisms of change in interpersonal therapy (IPT). *Clinical Psychology Review*, *33*(8), 1134–1147. doi:10.1016/j.cpr.2013.09.002

Longmore, R. J., & Worrell, M. (2007). Do we need to challenge thoughts in cognitive behavior therapy? *Clinical Psychology Review*, *27*(2), 173–187. doi:10.1016/j.cpr.2006.08.001

Lorenzo-Luaces, L., German, R. E., & DeRubeis, R. J. (2015). It's complicated: The relation between cognitive change procedures, cognitive change, and symptom change in cognitive therapy for depression. *Clinical Psychology Review*, *41*, 3–15. doi:10.1016/j.cpr.2014.12.003

Macaskill, D. N. D., & Macaskill, A. (1996). Rational-emotive therapy plus pharmacotherapy versus pharmacotherapy alone in the treatment of high cognitive dysfunction depression. *Cognitive Therapy and Research*, *20*(6), 575–592. doi:10.1007/BF02227962

Macneil, C. A., Hasty, M. K., Conus, P., & Berk, M. (2012). Is diagnosis enough to guide interventions in mental health? Using case formulation in clinical practice. *BMC Medicine*, *10*, 111. doi:10.1186/1741-7015-10-111

Martell, C. R., Addis, M. E., & Jacobson, N. S. (2001). *Depression in context: Strategies for guided action*. New York, NY: Norton.

Mathers, C. D., & Loncar, D. (2006). Projections of global mortality and burden of disease from 2002 to 2030. *PLOS Medicine*, *3*(11), e442. doi:10.1371/journal.pmed.0030442

Mueller, T. I., Leon, A. C., Keller, M. B., Solomon, D. A., Endicott, J., Coryell, W., … Maser, J. D. (1999). Recurrence after recovery from major depressive disorder during 15 years of observational follow-up. *American Journal of Psychiatry*, *156*(7), 1000–1006.

National Institute for Health and Clinical Excellence. (2009). *Treatment and management of depression in adults, including adults with a chronic physical health problem*. London, UK: National Institute for Health and Clinical Excellence.

Neimeyer, R. A. (2000). Searching for the meaning of meaning: Grief therapy and the process of reconstruction. *Death Studies*, *24*(6), 541–558. doi:10.1080/07481180050121480

Nolen-Hoeksema, S. (2000). The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *Journal of Abnormal Psychology*, *109*(3), 504–511. doi:10.1037//0021-843x.109.3.504

Nolen-Hoeksema, S., Morrow, J., & Fredrickson, B. L. (1993). Response styles and the duration of episodes of depressed mood. *Journal of Abnormal Psychology*, *102*(1), 20–28. doi:10.1037//0021-843x.102.1.20

Olesen, J., Gustavsson, A., Svensson, M., Wittchen, H.-U., Jönsson, B., CDBE2010 Study Group, & European Brain Council. (2012). The economic cost of brain disorders in Europe. *European Journal of Neurology*, *19*(1), 155–162. doi:10.1111/j.1468-1331.2011.03590.x

Paykel, E. S., Brugha, T., & Fryers, T. (2005). Size and burden of depressive disorders in Europe. *European Neuropsychopharmacology*, *15*(4), 411–423. doi:10.1016/j.euroneuro.2005.04.008

Quilty, L. C., McBride, C., & Bagby, R. M. (2008). Evidence for the cognitive mediational model of cognitive behavioural therapy for depression. *Psychological Medicine*, *38*(11), 1531–1541. doi:10.1017/S0033291708003772

Richards, D. A., Ekers, D., McMillan, D., Taylor, R. S., Byford, S., Warren, F. C., … Finning, K. (2016). Cost and outcome of Behavioural Activation versus Cognitive Behavioural Therapy for Depression (COBRA): A randomised, controlled, non-inferiority trial. *Lancet*, *388*(10047), 871–880. doi:10.1016/S0140-6736(16)31140-0

Satyanarayana, S., Enns, M. W., Cox, B. J., & Sareen, J. (2009). Prevalence and correlates of chronic depression in the Canadian Community Health Survey: Mental health and well-being. *Canadian Journal of Psychiatry/Revue Canadienne de Psychiatrie*, *54*(6), 389–398. doi:10.1177/070674370905400606

Scher, C. D., Ingram, R. E., & Segal, Z. V. (2005). Cognitive reactivity and vulnerability: Empirical evaluation of construct activation and cognitive diatheses in unipolar depression. *Clinical Psychology Review*, *25*(4), 487–510. doi:10.1016/j.cpr.2005.01.005

Segal, Z. V., Kennedy, S., Gemar, M., Hood, K., Pedersen, R., & Buis, T. (2006). Cognitive reactivity to sad mood provocation and the prediction of depressive relapse. *Archives of General Psychiatry*, *63*(7), 749–755. doi:10.1001/archpsyc.63.7.749

Society of Clinical Psychology. (2017). Short-term psychodynamic therapy for depression. Retrieved September 25, 2017, from https://www.div12.org/psychological-treatments/treatments/short-term-psychodynamic-therapy-for-depression

Solomon, A., Arnow, B. A., Gotlib, I. H., & Wind, B. (2003). Individualized measurement of irrational beliefs in remitted depressives. *Journal of Clinical Psychology*, *59*(4), 439–455. doi:10.1002/jclp.10081

Solomon, A., Haaga, D. A., Brody, C., Kirk, L., & Friedman, D. G. (1998). Priming irrational beliefs in recovered-depressed people. *Journal of Abnormal Psychology*, *107*(3), 440–449. doi:10.1037//0021-843x.107.3.440

Strunk, D. R., Brotman, M. A., & DeRubeis, R. J. (2010). The process of change in cognitive therapy for depression: Predictors of early inter-session symptom gains. *Behaviour Research and Therapy*, *48*(7), 599–606. doi:10.1016/j.brat.2010.03.011

Szentagotai, A., David, D., Lupu, V., & Cosman, D. (2008). Rational emotive behavior therapy versus cognitive therapy versus pharmacotherapy in the treatment of major depressive disorder: Mechanisms of change analysis. *Psychotherapy (Chicago, Ill.)*, *45*(4), 523–538. doi:10.1037/a0014332

Taghavi, M. R., Goodarzi, M. A., Kazemi, H., & Ghorbani, M. (2006). Irrational beliefs in major depression and generalized anxiety disorders in an Iranian sample: A preliminary study. *Perceptual and Motor Skills*, *102*(1), 187–196. doi:10.2466/pms.102.1.187-196

Tang, T. Z., DeRubeis, R. J., Beberman, R., & Pham, T. (2005). Cognitive changes, critical sessions, and sudden gains in cognitive-behavioral therapy for depression. *Journal of Consulting and Clinical Psychology*, *73*(1), 168–172. doi:10.1037/0022-006X.73.1.168

Teasdale, J. D., Segal, Z., & Williams, J. M. (1995). How does cognitive therapy prevent depressive relapse and why should attentional control (mindfulness) training help? *Behaviour Research and Therapy*, *33*(1), 25–39. doi:10.1016/0005-7967(94)e0011-7

Trivedi, M. H., Rush, A. J., Wisniewski, S. R., Nierenberg, A. A., Warden, D., Ritz, L., … STAR*D Study Team. (2006). Evaluation of outcomes with citalopram for depression using measurement-based care in STAR*D: Implications for clinical practice. *American Journal of Psychiatry*, *163*(1), 28–40. doi:10.1176/appi.ajp.163.1.28

Wampold, B. E. (2001). *The great psychotherapy debate: Models, methods, and findings*. Mahwah, NJ: Lawrence Erlbaum Associates.

Watkins, E. R. (2008). Constructive and unconstructive repetitive thought. *Psychological Bulletin*, *134*(2), 163–206. doi:10.1037/0033-2909.134.2.163

Watkins, E. R., Baeyens, C. B., & Read, R. (2009). Concreteness training reduces dysphoria: Proof-of-principle for repeated cognitive bias modification in depression. *Journal of Abnormal Psychology*, *118*(1), 55–64. doi:10.1037/a0013642

Watkins, E. R., & Moberly, N. J. (2009). Concreteness training reduces dysphoria: A pilot proof-of-principle study. *Behaviour Research and Therapy*, *47*(1), 48–53. doi:10.1016/j.brat.2008.10.014

Watkins, E. R., Taylor, R. S., Byng, R., Baeyens, C., Read, R., Pearson, K., & Watson, L. (2012). Guided self-help concreteness training as an intervention for major depression in primary care: A Phase II randomized controlled trial. *Psychological Medicine*, *42*(7), 1359–1371. doi:10.1017/S0033291711002480

Weitz, E. S., Hollon, S. D., Twisk, J., Van Straten, A., Huibers, M. J. H., David, D., … Cuijpers, P. (2015). Baseline depression severity as moderator of depression outcomes between cognitive behavioral therapy vs pharmacotherapy: An individual patient data meta-analysis. *JAMA Psychiatry*, *72*(11), 1102–1109. doi:10.1001/jamapsychiatry.2015.1516

Wells, A. (2008). *Metacognitive therapy for depression and anxiety*. New York, NY: Guilford Press.

Wenzlaff, R. M., & Grozier, S. A. (1988). Depression and the magnification of failure. *Journal of Abnormal Psychology*, *97*(1), 90–93. doi:10.1037//0021-843x.97.1.90

Zettle, R. D., & Hayes, S. C. (1986). Dysfunctional control by client verbal behavior: The context of reason-giving. *Analysis of Verbal Behavior*, *4*, 30–38. doi:10.1007/bf03392813

Zettle, R. D., & Rains, J. C. (1989). Group cognitive and contextual therapies in treatment of depression. *Journal of Clinical Psychology*, *45*(3), 436–445. doi:10.1002/1097-4679(198905)45:3<436::aid-jclp2270450314>3.0.co;2-l

Zettle, R. D., Rains, J. C., & Hayes, S. C. (2011). Processes of change in acceptance and commitment therapy and cognitive therapy for depression: A mediation reanalysis of Zettle and Rains. *Behavior Modification*, *35*(3), 265–283. doi:10.1177/0145445511398344

3

Evidence-Based Psychological Interventions for Bipolar Disorder

Aurora Szentágotai-Tătar and Daniel David
Department of Clinical Psychology and Psychotherapy, Babeş-Bolyai University, Cluj-Napoca, Romania

3.1 Overview of Bipolar Disorder

Mania and melancholia are two of the earliest described human disorders, present in the works of the Greek physicians of the classical period (Marneros & Angst, 2000). However, the idea that bipolar disorder (BD) is an entity in its own right was first proposed much later by Jean-Pierre Falret, who in 1851 described a mental disorder that he called "folie circulaire," characterized by cycles of depression and mania with symptom-free intervals (Marneros & Angst, 2000). Important contributions to the conceptualization of the disorder were subsequently made by Emil Kraepelin, who coined the term "manic depressive insanity," and by Karl Kleist, who distinguished between "unipolar" and "bipolar" affective disorders. The pioneering research of Angst (1966) and Perris (1966) confirmed that unipolar depression and BD could be differentiated in terms of clinical presentation, genetics, evolution, and response to treatment (Marneros & Angst, 2000).

BD is conceptualized today as a cyclical disorder, involving periods of profound disruption of mood and behavior due to manic or depressive symptoms, and periods of recovery or improved function (National Institute for Health and Care Excellence [NICE], 2014). Mania (or hypomania) is the key feature of the disorder. According to the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013), manic episodes are characterized by an abnormal and persistent elevated, expansive, or irritable mood, and abnormally and persistently increased goal-directed activity. Several other symptoms producing significant functional impairment may be present, such as inflated self-esteem and grandiosity, decreased need for sleep, the urge to talk incessantly, flight of ideas and racing thoughts, distractibility, psychomotor agitation, and increased involvement in activities with a high potential for negative consequences. The same symptoms can occur in hypomania, but they are less severe and cause less functional impairment (American Psychiatric Association, 2013).

Current classifications recognize a spectrum of BDs, ranging from severe to milder mood alterations (NICE, 2014). In the DSM-5 a distinction is made between bipolar I disorder (BD-I; characterized by full-blown manic episodes, commonly interspersed with major depressive episodes), bipolar II disorder (BD-II; major depressive episodes

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

and hypomanic episodes), and cyclothymia (recurrent hypomanic symptoms that do not meet criteria for a hypomanic episode, and depressive symptoms that do not meet criteria for a major depressive episode).

Although mania and hypomania are the defining characteristics of BD, longitudinal studies show that depressive symptoms are more common than manic symptoms, and that patients with BD spend a substantial proportion of time with subsyndromal or syndromal depressive symptoms (Judd et al., 2002, 2003). Major depressive episodes in BD are similar to those in major depressive disorder. According to the DSM-5 (American Psychiatric Association, 2013), they are characterized by depressed mood and loss of interest in activities, accompanied by weight loss or gain, sleep problems, psychomotor agitation or retardation, fatigue and lack of energy, difficulties concentrating, feelings of worthlessness, excessive guilt, and suicidal thoughts or actions.

A recent large-scale study involving over 60,000 adults from 11 countries reported lifetime prevalences of 0.6% for BD-I, 0.4% for BD-II, and 1.4% for subthreshold BD (Merikangas et al., 2011). In 2010, BD ranked 18th of all health conditions in years lived with disability (Vos et al., 2012), accounting for 7% of years lived with disability due to mental disorders (Whiteford et al., 2013). The high burden of the disorder is due to its early onset, with the first episode usually occurring before the age of 25, and to its chronicity across the lifespan (Kroon et al., 2013; Merikangas et al., 2007). BD is also a leading cause of mortality due to suicide (Høyer, Olesen, & Mortensen, 2004). In addition, diabetes and cardiovascular problems are more prevalent in patients with BD than in the general population, as medication used to treat BD hastens the development of metabolic syndrome and obesity (Fiedorowicz, Palagummi, Forman-Hoffman, Miller, & Haynes, 2008).

Several pharmacological agents are available for the treatment of BD (Fountoulakis et al., 2012), and pharmacological interventions are the primary tool in the management of the disorder. Nevertheless, despite appropriate treatment, a high percentage of patients experience relapses, residual affective symptoms, mood cycling, and significant impairment in psychosocial functioning (Angst, Gamma, Sellaro, Lavori, & Zhang, 2003; Fagiolini et al., 2005).

The past two decades have witnessed an increased acceptance of stress-vulnerability models that highlight the interplay among psychological, social, and biological factors in the onset of and maintenance of severe mental disorders (Scott, 2006). An interest has thus appeared in the development and evaluation of psychological treatments as an adjunct to medication for patients with BD (Scott, 2006). The main psychological treatments currently employed for BD are cognitive–behavioral therapy (CBT), family interventions, interpersonal and social rhythm therapy (IPTSR), individual and group psychoeducation, and multicomponent interventions (Geddes & Miklowitz, 2013; Lam, Jones, & Hayward, 2010; NICE, 2014). Although they are based on different assumptions and use different methods and treatment strategies, these interventions have common objectives, such as educating the patient about the disorder, helping the patient identify and manage early signs of recurrence, increasing treatment adherence, enhancing ability to cope with stressors, stabilizing sleep–wake patterns and daily routines, reducing substance misuse, and improving social relations (Geddes & Miklowitz, 2013). This chapter reviews psychotherapeutic treatments for BD and evidence that supports them based on the framework for evaluating evidence-based psychosocial interventions proposed by David and Montgomery (2011).

3.2 Evidence-Based Interventions for Bipolar Disorder

David and Montgomery (2011) propose a framework for evaluating the scientific status of psychological interventions based on the idea that two aspects to be taken into account when assessing these interventions are evidence concerning the treatment package and evidence concerning the underlying theory. The latter refers to the mechanisms of change (i.e., psychological factors involved in the disorder) targeted by the treatment package (David & Montgomery, 2011). The combination of these two factors results in nine categories of psychological treatments in terms of evidence (see Table 3.1).

Psychosocial interventions for BD were evaluated according to the framework proposed by David and Montgomery (2011), resulting in the classification shown in Table 3.1. For each intervention, treatment package efficacy and evidence regarding theory were derived from clinical guidelines, meta-analyses and systematic reviews, clinical trials, and studies identified through a search of several databases (e.g., PsycINFO, PubMed).

According to David and Montgomery (2011; see also Wampold, 2001), treatment package efficacy can be evaluated as *absolute efficacy* (comparisons with control conditions, either no-treatment or waitlist), *relative efficacy* (comparisons with other evidence-based psychological interventions), or *specific efficacy* (treatment and underlying theory are empirically supported). In the case of BD, pharmacological intervention is the primary tool in the management of the disorder, and psychological treatments are delivered and evaluated as an adjuvant to medication. Despite high rates of medication nonadherence (e.g., Scott & Pope, 2002), there are no studies of structured

Table 3.1 The status of psychological interventions for bipolar disorder based on David and Montgomery's (2011) framework.

	Theory		
Therapeutic Package	**Well Supported**	**Equivocal: No, Preliminary, or Mixed Data**	**Strong Contradictory Evidence**
Well Supported	Category I: Evidence-based psychotherapies None	Category II: Intervention-driven psychotherapies None	Category V: Good-intervention- and bad-theory-driven psychotherapies None
Equivocal: No, Preliminary, or Mixed Data	Category III: Theory-driven psychotherapies None	Category IV: Investigational psychotherapies CBT; individual and family psychoeducation; FFT; IPTSR	Category VII: Bad-theory-driven psychotherapies None
Strong Contradictory Evidence	Category VI: Good-theory- and bad-intervention-driven psychotherapies None	Category VIII: Bad-intervention-driven psychotherapies None	Category IX: Bad-theory- and bad-intervention-driven psychotherapies None

CBT: cognitive–behavioral therapy; FFT: family-focused therapy; IPTSR: interpersonal and social rhythm therapy.

psychological interventions comparing psychotherapy to no-treatment control groups (NICE, 2014). Therefore, the absolute efficacy of psychological interventions cannot be determined in this case.

3.2.1 Category I: Evidence-Based Psychotherapies

This category includes psychological interventions that have a validated treatment package, derived from a validated theory (David & Montgomery, 2011). If we consider effects on the overall clinical presentation of BD, we believe that there are no psychological treatments that currently can be unequivocally included in this category.

3.2.2 Category II: Intervention-Driven Psychotherapies

Interventions in this category are characterized by an evidence-based treatment package but an insufficiently investigated (i.e., no data, preliminary data) or supported (i.e., mixed data) theory of change (David & Montgomery, 2011). Based on the analysis of the literature, and considering effects on the overall clinical presentation of the disorder, no psychological intervention can be unequivocally included in this category.

3.2.3 Category III: Theory-Driven Psychotherapies

This category refers to psychological interventions that are based on a well-supported theory of the disorder but an insufficiently supported treatment package (David & Montgomery, 2011). We did not identify any interventions in this category.

3.2.4 Category IV: Investigational Psychotherapies

Category IV psychotherapies are characterized by insufficiently supported theory and an insufficiently supported treatment package (David & Montgomery, 2011). Insufficient support can be the product of no data on the theory and treatment, preliminary data only, or mixed data. Based on an analysis of the literature, we believe the main psychological interventions for BD warrant inclusion in this category. However, the interventions differ in terms of the strength of the evidence supporting their inclusion in this category. Thus, some interventions have been investigated in several large-scale clinical trials, whereas the data with respect to others are of lower quality. Also, some interventions propose both a theory of change and a treatment package (e.g., CBT), while others are mostly treatment driven, with no specific theory of the disorder.

3.2.4.1 Cognitive–behavioral therapy

Cognitive–behavioral models of BD adapt the cognitive therapy model of unipolar depression (Beck, Rush, Shaw, & Emery, 1979; Power, 2005). They are diathesis–stress models that propose that individuals with BD hold dysfunctional schemas and beliefs that can be triggered by positive and negative life events and influence mood and behavior. Several cognitive–behavioral accounts of BD have been proposed, focusing on different types of dysfunctional beliefs (Power, 2005). Newman, Leahy, Beck, Reilly-Harrington, and Gyulai (2002) have argued that dysfunctional schemas in BD change

polarity under the influence of mood states and life events (e.g., a lovability-related schema can shift from one extreme, with the person feeling completely unlovable, to the other, with the person feeling everybody loves him or her). Lam et al. (2010) proposed a model that emphasizes the role of disrupted circadian rhythms (Healy & Williams, 1989), a poorly regulated behavioral activation system (Depue, Krauss, & Spoont, 1987), and dysfunctional cognitions related to goal attainment and achievement (Lam, Wright, & Smith, 2004). Mansell and colleagues (Mansell, Morrison, Reid, Lowens, & Tai, 2007; Mansell et al., 2014) suggest that people with BD experience difficulties because they try to control their moods in an extreme way, which negatively affects their ability to achieve their broader goals in life.

CBT for patients with BD is focused on education about the illness based on the diathesis–stress model, reducing barriers to treatment adherence, teaching cognitive and behavioral skills to cope with prodromes, stabilizing daily routines and sleep patterns, changing maladaptive beliefs, identifying triggers for mood episodes, and dealing with long-term vulnerabilities to relapse (Reinares, Sánchez-Moreno, & Fountoulakis, 2014).

Evidence on the efficacy of CBT in BD comes mainly from studies involving patients not in an acute phase of the disorder. Nevertheless, there is also evidence on the efficacy of CBT for patients in an acute depressive episode (e.g., Miklowitz, Otto, Frank, Reilly-Harrington, Kogan et al., 2007; Miklowitz, Otto, Frank, Reilly-Harrington, Wisniewski et al., 2007). In terms of outcomes, many CBT studies are relapse prevention studies (Lam et al., 2010), but researchers have also reported data on symptom severity (e.g., depression and mania severity), symptom duration (e.g., days spent in manic or depressive episode), treatment cost and adherence, quality of life, and quality of social functioning (e.g., Costa, Cheniaux, Rangé, Versiani, & Nardi, 2012; Lam et al., 2003; Parikh et al., 2012). Based on existing evidence, the NICE guideline on the assessment and management of BD (NICE, 2014) recommends CBT for managing depression in people with BD in primary and secondary care and for the long-term management of the disorder.

According to David and Montgomery (2011), *absolute efficacy* is determined by comparing CBT to a no-treatment control group or to a waitlist condition. Considering that, despite high treatment nonadherence, there are no studies that examine the effects of CBT in BD in the absence of medication, absolute efficacy cannot be evaluated.

Relative efficacy is determined by comparing CBT with standard treatment (David & Montgomery, 2011). Taking into account that psychotherapy is typically offered as an adjunct to medication in patients with BD, there is no psychological treatment that can be considered standard treatment in this case. Thus, in order to establish relative efficacy, we examined studies that compared CBT plus treatment as usual (TAU) to TAU alone, as well as studies that compared CBT plus TAU to another form of psychotherapy plus TAU. In these studies, in addition to medication, TAU could also involve more or less intensive clinical case management. Data on the efficacy of CBT in BD come from several independent research groups.

In a meta-analysis published in 2010, Szentágotai and David summarized the results of 12 studies comparing CBT to TAU in terms of BD symptoms, relapse prevention, treatment adherence, treatment costs, quality of life and social adjustment, and cognitive–behavioral mechanisms (e.g., dysfunctional cognitions, coping skills). At posttreatment,

low to medium effect sizes in favor of CBT were found in terms of symptoms (Cohen's $d = -0.44$; 95% CI −0.59 to −0.22), cognitive–behavioral mechanisms ($d = -0.49$; 95% CI −0.72 to −0.25), quality of life and social adjustment ($d = 0.36$; 95% CI −0.54 to −0.18), and treatment adherence ($d = -0.53$; CI −0.71 to −0.35). No effect of CBT was found on relapse and treatment costs. The beneficial effects of CBT on symptoms and mechanisms remained significant at follow-ups at 6 months, 12 months, and over 12 months. The effect on treatment adherence remained significant at 6-month follow-up. No effects at follow-up were found on quality of life and social adjustment, treatment adherence, treatment costs, or relapse. Another small-scale meta-analysis (Lynch, Laws, & McKenna, 2010) reported similar findings in terms of relapse prevention.

Although the Szentágotai and David (2009) meta-analysis did not differentiate between types of bipolar symptoms, these were analyzed separately in two subsequent meta-analyses (Gregory, 2010a). CBT had a small yet significant effect on depressive symptoms (Cohen's $d = -0.29$) (Gregory, 2010a) and a small but nonsignificant effect on manic symptoms (Gregory, 2010b) compared to TAU. Benefits of CBT over TAU were also reported by more recent studies not included in the above meta-analyses, such as Costa et al. (2012) (depressive symptoms, frequency and duration of mood episodes, quality of life) and Jones et al. (2015) (self-reported recovery, increased time to any mood relapse).

Thus, existing studies on treatment packages favor CBT plus TAU compared to TAU alone (i.e., better than standard treatment [BST]) in managing depressive symptoms and dysfunctional cognitions and behaviors in patients with BD. Superior effects of CBT added to TAU on quality of life, social adjustment, and treatment adherence are also supported by preliminary data, but the effects seem to be shorter lived (Szentágotai & David, 2009). The data on relapse prevention do not favor CBT, although the evidence in the literature is mixed, with some studies showing significant benefits of CBT added to TAU over TAU (e.g., Lam et al., 2003) and others not (Scott et al., 2006). Moreover, post hoc analyses in the large-scale study of Scott et al. (2006) showed that adjunctive CBT was significantly more effective than TAU in patients with fewer than 12 previous episodes. Further studies are therefore needed to establish the effects of CBT on relapse prevention in BD and to determine the characteristics of patients who would benefit most from CBT.

Some studies compared CBT to active control groups that received other psychological interventions. A study on patients in an acute depressive episode that compared CBT, IPTSR, and family-focused therapy (FFT) to brief psychoeducation reported that all three intensive interventions were better than the brief intervention in terms of recovery rates and time to recovery over 12 months (Miklowitz, Otto, Frank, Reilly-Harrington, Wisniewski et al., 2007) and in terms of increasing overall and relationship functioning and life satisfaction over 9 months (Miklowitz, Otto, Frank, Reilly-Harrington, Kogan et al., 2007), with no differences found among the three intensive treatments. Zaretsky, Lancee, Miller, Harris, and Parikh (2008) found that individual psychoeducation followed by CBT compared to psychoeducation was more effective in reducing depressed mood and antidepressant use over a year; no differences between the two interventions were evident in terms of hospitalization rates, medication adherence, and psychosocial functioning. Parikh and colleagues (2012) found no difference between a 20-session CBT intervention and a 6-session group psychoeducation intervention in symptom burden and likelihood of relapse.

Therefore, evidence on the efficacy of CBT compared to other psychological interventions supports both equivalence between treatments and superiority of CBT. More studies are needed to establish the outcomes that CBT is likely to be most efficient for, particularly given that outcomes measured by existing studies are quite heterogeneous.

In terms of *specific efficacy*, CBT added to TAU is superior to TAU alone in managing depressive symptoms (Gregory, 2010a; Szentágotai & David, 2010) and CBT is as effective or more effective than other psychological interventions for depression in patients with BD. The first criterion of specific efficacy is thus met for depressive symptoms, whereas data on other outcomes (i.e., recovery, treatment adherence, social adjustment) are encouraging but mixed (i.e., mixed data) or insufficient (i.e., no data).

Specific efficacy also reflects the extent to which the theory of change on which the intervention package is based has been validated. Theory-of-change validation relies on experimental studies, component analyses, patient-by-treatment interactions, and mediation/moderation analyses in complex clinical trials (David & Montgomery, 2011). To our knowledge, no such data are available for CBT theories of BD. There are, however, studies linking dysfunctional beliefs to both depression and mania in BD.

Research based on questionnaires shows that people with BD endorse a variety of dysfunctional beliefs (both negative and positive), and studies have demonstrated this pattern across measures of how participants regard themselves, life events, and their accomplishments (for a review see Johnson & Tran, 2007). The level of cognitive dysfunction among participants is related to the number of previous episodes (Scott, Stanton, Garland & Ferrier, 2000) and predicts depressive and manic symptoms (Goldberg, Gerstein, Wenze, Welker, & Beck, 2008; Reilly-Harrington, Alloy, Fresco, & Whitehouse, 1999). Moreover, patients who receive CBT exhibit a decrease in dysfunctional attitudes (Ball, Mitchell, Corry, Skillecorn, Smith, & Mahli, 2006; Lam et al., 2003). Zaretsky et al. (2008) found that changes in dysfunctional attitudes explained 10% of variance in depression levels over the course of a year. Thus, based on existing evidence, we can conclude that there are preliminary data that support CBT models of BD.

3.2.4.2 Mindfulness-based cognitive therapy

Mindfulness-based cognitive therapy (MBCT) was developed as a group therapy program to prevent relapse in major depression (Segal, Williams, & Teasdale, 2001). It integrates elements of CBT for depression (Beck et al., 1979) with elements of mindfulness-based stress reduction (Kabat-Zinn, 1990). Unlike "classic" CBT, MBCT does not emphasize changing the content of thoughts but changing awareness of and relationship to thoughts, feelings, and sensations (Segal et al., 2001).

Given the encouraging data for major depression, several uncontrolled studies have recently examined MBCT with BD patients (e.g., Deckersbach et al., 2012; Miklowitz et al., 2009, 2015; Murray et al., 2015; Weber et al., 2010), some with promising results.

However, the two randomized clinical trials (RCTs) conducted so far (Perich, Manicavasagar, Mitchell, Ball, & Hazdi-Pavlovic, 2013; Williams et al., 2008) have yielded limited support in favor of MBCT. One study (Williams et al., 2008) reported a reduction of depressive symptoms in the MBCT group compared with a waitlist group, but this study included a mix of euthymic, major depressive disorder, and BD patients, and the number of BD participants was very low. Perich et al. (2013) found no impact of MBCT in time to recurrence, total number of recurrences over 12 months, or mood symptom severity in a study including 95 euthymic participants randomized to a MBCT

plus TAU or a TAU group. There is some evidence that MBCT could be useful in reducing anxiety levels in BD patients (Perich et al., 2013; Williams et al., 2008). We believe these preliminary, mixed results are insufficient to establish the *relative efficacy* or *specific efficacy* of MBCT for patients with BD.

3.2.4.3 Psychoeducation

This section reviews psychoeducation delivered to the patient in individual or group formats; family psychoeducation is discussed in Section 3.2.4.4. Psychoeducation refers to interventions that teach patients and their families about the illness with the aim of improving their outcomes (Bond & Anderson, 2015). It is delivered in a group or individual format, and ranges from brief interventions, providing mainly information about the illness and treatment, to more complex ones, covering a variety of aspects of BD. A recent meta-analysis on psychoeducation for BD (Bond & Anderson, 2015) indicates that the number of sessions ranges from 2 to 21, and treatment is characterized by various degrees of personalization. Four major overlapping areas of information provided include (1) causes, symptoms, and course of the illness; (2) treatment; (3) relapse patterns, early symptoms, relapse triggers (such as drug and alcohol use), life and interpersonal stress, and circadian and social rhythms; and (4) strategies to minimize relapse risks (Bond & Anderson, 2015).

Bond and Anderson's (2015) meta-analysis included 16 RCTs and focused on the efficacy of psychoeducation for relapse prevention. All studies involved patients not in an acute phase of the disorder; control conditions were represented by TAU, placebo control (i.e., matched number of meetings that did not involve psychoeducation but where patients did receive medication), and active control (which compared psychoeducation to another psychotherapeutic treatment). Psychoeducation was better than TAU and placebo control in preventing any type of relapse (OR = 1.98; 95% CI 1.09 to 3.58) and manic/hypomanic relapses (OR = 1.68; 95% CI 0.99 to 2.85), but not depressive relapses. Group intervention was superior to individual treatment and, in the case of depressive symptoms, longer interventions were more effective (Bond & Anderson, 2015).

Due to the limited number of studies in this meta-analysis, effect sizes were not computed for the impact of psychoeducation compared with other active interventions. Nevertheless, one study comparing a 20-session individual CBT intervention to 6 sessions of group psychoeducation (Parikh et al., 2012) found no differences between the two in relapse rates. Another study, comparing individual psychoeducation to FFT, found no differences in likelihood of first relapse, but FFT was associated with a reduced likelihood of rehospitalization and fewer mood relapses over 2 years (Rea et al., 2003).

The effects of psychoeducation on mood symptom severity have also been investigated in several studies, yielding mixed data. Two studies comparing group psychoeducation to TAU and group psychoeducation to pharmacotherapy plus placebo found no effects of psychoeducation on manic and depressive symptoms (Castle et al., 2010; de Barros Pellegrinelli et al., 2012). Improvements in mood symptoms in patients receiving psychoeducation compared with TAU were reported in a small-scale study by Doğan and Sabancioğullari (2003) and by Javadpour, Hedayati, Dehbozorgi, and Azizi (2013). Two studies comparing psychoeducation to CBT found no differences in manic and depressive symptom severity (Parikh et al., 2012; Zaretsky et al., 2008), although, in one study, CBT was associated with fewer days of depressed mood over the course of 1 year (Zaretsky et al., 2008).

The data on quality of life and functioning are also mixed, with some studies reporting beneficial effects of psychoeducation compared with TAU (e.g., Doğan & Sabancioğullari, 2003; Javadpour et al., 2013; Lobban et al., 2010; Perry, Tarrier, Morriss, McCarthy, & Limb, 1999) and others not (e.g., de Barros Pellegrinelli et al., 2012). A study comparing psychoeducation with CBT found similar benefits of the two interventions on psychosocial functioning (Zaretsky et al., 2008).

Finally, several studies have reported benefits of psychoeducation compared with TAU on medication knowledge (e.g., Doğan & Sabancioğullari, 2003; Peet & Harvey, 1991), attitudes to medication (Peet & Harvey, 1991), and adherence, expressed both in serum lithium levels (Colom et al., 2003; Doğan & Sabancioğullari, 2003) and taking medication (Doğan & Sabancioğullari, 2003; Eker & Harkin, 2012; Javadpour et al., 2013; Peet & Harvey, 1991). In addition, psychoeducation was comparable to CBT (Zaretsky et al., 2008) and FFT (Rea et al., 2003) in terms of treatment adherence.

The heterogeneity of psychoeducation in which treatment duration and intervention content are concerned makes it difficult to draw clear conclusions on the *relative efficacy* of this approach. Studies seem to support the superiority of psychoeducation added to TAU compared with TAU alone (i.e., BST) in preventing manic relapses, but not depressive relapses. Data on mood symptom severity are preliminary and mixed; more well-controlled studies are needed to establish the efficacy of psychoeducation in reducing manic and depressive symptoms in BD. Data are also preliminary and mixed in terms of effects on quality of life. More consistent effects of psychoeducation have been found on treatment adherence, with most studies reporting benefits over TAU (BST). These conclusions are also supported by the few studies that compare psychoeducation added to TAU with other psychological interventions plus TAU (e.g., CBT, FFT). These studies report equal efficacy on most outcomes, although CBT and FFT are more effective in addressing symptom severity (Bond & Anderson, 2015; Rea et al., 2003; Zaretsky et al., 2008).

The first criterion of *specific efficacy* is related to treatment package results. As already shown, there is evidence that psychoeducation added to TAU is superior to TAU alone in terms of preventing manic relapses and treatment adherence (Bond & Anderson, 2015), and is equivalent to other psychological interventions with respect to these outcomes (Parikh et al., 2012; Rea et al., 2003; Zaretsky et al., 2008). Support for effects on quality of life and functioning and for mood symptom severity is preliminary and more inconsistent, with studies reporting both positive and negative results (mixed data) (Bond & Anderson, 2015). Psychoeducation does not seem to have an impact on preventing depressive relapses (Bond & Anderson, 2015).

In terms of the second criterion of *specific efficacy*, to our knowledge, no specific mechanisms of change of psychoeducation have been tested to date. Bond and Anderson (2015) propose that such mechanisms could involve a variety of candidates, ranging from attitudinal change to changes in coping strategies and lifestyle.

3.2.4.4 Family interventions

Family interventions for BD target the family, not only the patient, and usually include elements of psychoeducation, communication and problem-solving skills, and support for caregivers (Miziou et al., 2015). Although a 2007 Cochrane Review concluded that it was not possible to make a clear case for the use of family interventions as an adjunctive treatment to medication in BD (Justo, Soares, & Calil, 2007), based on more recent data,

NICE's 2014 guideline recommends family interventions for the long-term management of BD in adults. Family interventions have been offered to patients with BD in various formats; here we review evidence for FFT and family psychoeducation, as these have been assessed in clinical trials.

3.2.4.4.1 Family-focused therapy

Employing a stress-vulnerability framework, FFT for BD attempts to balance protective and risk factors in the social and family environment (Morris, Miklowitz, & Waxmonsky, 2007). FFT is based on the observation that a hostile and critical family environment (i.e., a high *expressed-emotion* (EE) environment) leads to an increased likelihood of relapse in BD (Geddes & Miklowitz, 2013). FFT is offered as an adjunct to pharmacotherapy and typically involves the patient and family members (e.g., parent, spouse) in up to 21 therapy sessions over a 9-month period (Geddes & Miklowitz, 2013). Therapy has three phases, beginning with psychoeducation, followed by communications skills training and problem-solving skills training (Geddes & Miklowitz, 2013). The main goals of FFT are to assist patients and their relatives in (1) integrating the experiences associated with the episodes of BD, (2) accepting the idea of vulnerability to future episodes, (3) accepting the idea of dependency on mood-stabilizing medication, (4) distinguishing between the patient's personality and his/her BD, (5) recognizing and coping with stressful life events that trigger new episodes, and (6) reestablishing functional relationships following an episode (Miklowitz, 2008).

The *absolute efficacy* of an FFT treatment package cannot be determined in the absence of studies comparing FFT with a no-treatment control group. Most studies on FFT for BD have compared groups receiving FFT as an adjunct to medication with groups receiving medication and another form of psychosocial intervention of variable complexity (e.g., brief psychoeducation, long-term psychoeducation, health education). *Relative efficacy* evaluation relies on these studies.

According to the NICE (2014) guideline on BD, at posttreatment, FFT has a medium effect compared with control on depressive symptoms (SMD −0.40; 95% CI −0.80 to 0.00). At follow-up there is little difference between FFT and TAU on depressive symptoms (SMD −0.10; 95% CI −0.56 to 0.36) and little evidence of an effect on relapse (RR 0.67; 95% CI 0.34 to 1.30), but FFT reduces the risk of hospitalization (RR 0.24; 95% CI 0.08 to 0.74). The guideline classifies the quality of evidence supporting these effects as low (NICE, 2014).

Miklowitz, Simoneau et al. (2000) and Miklowitz, George, Jeffrey, Richards, Simoneau, and Suddath (2003) reported that patients who received FFT following an illness episode, compared with patients who received a crisis management intervention, experienced greater benefits (expressed in fewer relapses, better medication adherence, more rapid recovery from depression, and less severe depressive symptoms) over a 1- to 2-year follow-up. Miklowitz, Otto, Frank, Reilly-Harrington, Wisniewski et al. (2007) also reported shorter time to recovery and higher recovery rates in depressed patients receiving FFT compared with patients receiving three sessions of collaborative care. A study on adolescents with BD who received FFT following a mood episode, compared with adolescents who received three family sessions focused on relapse prevention, found that FFT was associated with more rapid recovery from depression and less severe symptoms of depression over 2 years; no differences in time to relapse were found (Miklowitz et al., 2008). More recently, Miklowitz et al. (2014) found no differences in

adolescents with BD between FFT plus pharmacotherapy and brief psychoeducation plus pharmacotherapy on time to recovery or severity of mood symptoms over 2 years.

To conclude, preliminary evidence indicates that FFT added to pharmacotherapy is more effective than brief psychoeducation plus pharmacotherapy in terms of time to recovery from depressive episodes, severity of depressive symptoms, and number of rehospitalizations, at least in adults with BD. Data on manic symptoms are scarce and inconclusive. Data for adolescents are mixed, and more studies are needed to answer the question of whether FFT is developmentally attuned to the needs of adolescents (Miklowitz et al., 2014).

In addition to studies comparing FFT with brief interventions, some studies compare FFT with active control groups (e.g., FFT, CBT, IPTSR) receiving long-term psychological treatment. Miklowitz and colleagues (Miklowitz, Otto, Frank, Reilly-Harrington, Kogan et al., 2007; Miklowitz, Otto, Frank, Reilly-Harrington, Wisniewski et al., 2007; see Section 3.2.4.1 on CBT *relative efficacy*) found no differences between FFT, CBT, and IPTSR with respect to various outcomes, whereas all interventions were more effective than brief psychoeducation. Another study, which compared a 9-month FFT intervention to a 9-month individually focused intervention, found that FFT was associated with a reduced likelihood of rehospitalization and fewer mood relapses over 2 years (Rea et al., 2003). Thus, evidence supports both the equivalence of FFT and the superiority of FFT across comparisons with other interventions.

The first component of *specific efficacy* is related to intervention (i.e., treatment package) results. Preliminary evidence indicates that FFT added to medication is superior to both brief psychoeducation (Miklowitz et al., 2000, 2003, 2008) and individual long-term psychoeducation (Rea et al., 2003), and equivalent to other psychological treatments (e.g., CBT, IPTSR) (Miklowitz, Otto, Frank, Reilly-Harrington, Kogan et al., 2007; Miklowitz, Otto, Frank, Reilly-Harrington, Wisniewski et al., 2007). These effects are most convincing in the case of depression.

Although FFT does not propose a theory regarding the mechanisms that lead to the onset of BD, it does maintain that hostile and critical family interactions influence the course of the disorder (Morris et al., 2007) and that higher EE environments predict relapse and worse functioning in patients (Miklowitz, 2008). The relapse hypothesis was only weakly supported in a treatment by EE analysis (Miklowitz et al., 2000) and was not supported in a moderation study (Kim & Miklowitz, 2004). However, these studies did find preliminary evidence for higher levels of mania and depression in patients with high EE relatives over a period of 2 years, and for a better response to treatment in patients with families high in EE.

3.2.4.4.2 Family psychoeducation

Although FFT includes a psychoeducation component, the intervention emphasizes mitigating criticism and hostility in the family (Geddes & Miklowitz, 2013). Psychoeducation for the families of patients with BD has also been used in formats that do not focus on EE.

In some cases, family psychoeducation involves both patients and family members (e.g., Clarkin, Carpenter; Hull, Wilner, & Glick, 1998; D'Souza, Piskulic, & Sundram, 2010; Fiorillo et al., 2015; Miller, Solomon, Ryan, & Keitner, 2004), whereas in others it is delivered to family members only (e.g., Bordbar, Soltanifar, & Talaei, 2009; Madigan et al., 2012; Perlick et al., 2010; Reinares et al., 2008). Psychoeducation has been offered

as a multifamily group intervention, reuniting members of several families (e.g., Miller et al., 2004) or addressing members of one family (e.g., Clarkin et al., 1998). Most studies have been conducted on euthymic patients, but trials also include a mix of euthymic and acute patients (Perlick et al., 2010) and patients in an acute episode (Miller et al., 2004). Although research has shown significant beneficial effects of family psychoeducation for caregivers of patients with BD (e.g., Perlick et al., 2010; Reinares et al., 2004), we only focus on evidence related to patient benefits.

Based on seven RCTs, the NICE guideline for BD concludes that low-quality evidence supports a medium effect of family psychoeducation over TAU on depressive symptoms at posttreatment (SMD −0.73; 95% CI 1.35 to −0.10). At follow-up, the effect on depressive symptoms is not significant (SMD −0.15; 95% CI −0.69 to 0.39). Studies indicate a reduced risk of any relapse and of manic relapses, whereas the effect on depressive relapses is inconclusive (NICE, 2014).

Some of the family psychoeducation studies that only involved family members used no-intervention control groups and can be discussed from the point of view of *absolute efficacy*. This criterion is met based on two studies, one showing that manic patients whose family members receive psychoeducation are less likely to relapse at 12 months (Bordbar et al., 2009) and the other showing that euthymic patients whose caregivers are offered psychoeducation are less likely to experience a manic/hypomanic relapse and have longer symptom-free intervals (Reinares et al., 2008). Further studies are needed to confirm these results. *Relative efficacy* of family psychoeducation for family members is only supported by preliminary data. One study that compared CBT-based family psychoeducation to health education found lower levels of depressive symptoms in patients whose family members were in the CBT-based psychoeducation group (Perlick et al., 2010).

Most trials involving patients in addition to caregivers have compared family psychoeducation plus TAU with TAU (i.e., medication and case management). Benefits of psychoeducation were found with regard to relapse probability and time to relapse in euthymic patients (D'Souza et al., 2010), with regard to overall functioning (Clarkin et al., 1998), and with regard to social functioning (Fiorillo et al., 2015). One study (Miller et al., 2004) compared pharmacotherapy plus family psychoeducation with pharmacotherapy plus another family intervention and pharmacotherapy alone. Neither adjunctive family psychoeducation nor adjunctive family therapy improved recovery rates and time to recovery from a current mood episode. Thus, family psychoeducation involving the patient has some advantages over TAU (i.e., BST), but data supporting this conclusion are scarce and need to be replicated.

To conclude, the overall data supporting both *absolute* and *relative efficacy* of family psychoeducation are preliminary, mixed, and limited in terms of number of studies and outcomes affected by the intervention. As already shown, some studies support the benefits of psychoeducation in reducing depressive symptoms (Perlick et al., 2010), preventing relapse (Bordbar et al., 2009; D'Souza et al., 2010; Reinares et al., 2008), and increasing functioning and quality of life (Clarkin et al., 1998; Fiorillo et al., 2015; Madigan et al., 2012). Other studies, however, report no effects of psychoeducation on symptoms (Clarkin et al., 1998), on recovery from an acute episode (Miller et al., 2004), on time to recovery (Miller et al., 2004), or on functioning (Madigan et al., 2012).

The first component of *specific efficacy* refers to treatment package efficacy and has been discussed already. There are no data available on the second component, validity

of the theory of change, as family psychoeducation programs do not rely on a specific theory of the disorder. Nevertheless, as in the case of FFT, evidence indicates that family psychoeducation may be better suited to families with high levels of impairment (Miller et al., 2004).

3.2.4.5 Interpersonal and social rhythm therapy

Developed as a treatment for unipolar depression (Klerman, Weissman, Rounsaville, & Chevron, 1984), interpersonal psychotherapy (IPT) is based on the idea that depression occurs in a psychosocial context and that stressful interpersonal events may contribute to the onset of the disorder in people with a biological vulnerability (Frank, Swartz, & Kupfer, 2000). IPTSR adapts IPT to patients with BD by incorporating data on the relationship between disrupted circadian rhythms and bipolar episodes and data that link social rhythm disruptions and BD (Frank et al., 2000).

In addressing BD symptoms, IPTSR focuses on (1) the link between mood and life events; (2) the importance of maintaining regular daily rhythms; (3) the identification and management of potential rhythm dysregulation, particularly in the interpersonal domain; (4) the facilitation of mourning the lost healthy self; and (5) the management of affective symptoms (Frank et al., 2000). Based on three clinical trials, the NICE guideline on BD concludes that "there is no evidence that IPTSR is superior to no intervention or to other interventions" (NICE, 2014, p. 262).

Researchers have analyzed the *relative efficacy* of the IPTSR treatment package in four clinical trials conducted on patients in an acute phase of BD. A large-scale clinical trial ($N = 175$) randomized acutely ill patients to IPTSR or equally intensive clinical management (Frank et al., 2005). After acute stabilization, patients were randomly assigned again to IPTSR or intensive clinical management, and treatment continued over a 2-year maintenance phase. Although no differences were observed between treatment conditions in time to stabilization, the group that received IPTSR in the acute phase had longer time to recurrence and better vocational functioning in the maintenance phase. Another study (Miklowitz, Otto, Frank, Reilly-Harrington, Kogan et al., 2007; Miklowitz, Otto, Frank, Reilly-Harrington, Wisniewski et al., 2007; see Section 3.2.4.1) with patients experiencing a depressive episode found that IPTSR is as effective as CBT and FFT, and more effective than brief psychoeducation, in terms of (1) recovery rates and time to recovery over 12 months and (2) increasing overall and relationship functioning and life satisfaction over 9 months. In a pilot study comparing unmedicated BD-II individuals receiving IPTSR ($N = 14$) with individuals receiving quetiapine ($N = 11$), Swartz, Frank, and Cheng (2012) found that IPTSR was as effective as quetiapine in reducing depressive and hypomanic symptoms and overall symptom severity over 12 weeks. Recently, Inder et al. (2015) compared IPTSR with another intensive psychological intervention (i.e., specialist supportive care) in young adults with BD ($N = 100$) and found that the two interventions were equally effective in improving depressive and manic symptoms and in increasing social functioning over 52 weeks postintervention.

To conclude, in terms of *relative efficacy*, there is evidence, based on one study (Swartz et al., 2012), that IPTSR is as effective as standard treatment (i.e., medication; equivalent to other standard therapies) in reducing symptom severity. Nevertheless, considering the small sample size, this evidence can be considered at best preliminary. Also, there is preliminary evidence that IPTSR in as effective as other intensive psychological interventions in reducing symptom severity, facilitating recovery, and increasing overall

functioning (Miklowitz, Otto, Frank, Reilly-Harrington, Kogan et al., 2007; Miklowitz, Otto, Frank, Reilly-Harrington, Wisniewski et al., 2007). Additionally, IPTSR could be useful in preventing relapses in patients with BD (Frank et al., 2005). These conclusions are based on a small number of studies, and they can best be regarded as provisional; additional well-controlled trials are needed to clearly establish the utility of IPTSR in BD.

The studies reviewed herein lend preliminary support to the first criterion of *specific efficacy* of IPTSR, in which symptom severity, recovery, and overall functioning are concerned. Nevertheless, due to the small number of studies, this conclusion is only provisional. In terms of theory of change, in a recent review on the mechanisms of change of IPT, Lipsitz and Markowitz (2013) proposed enhancing social support, decreasing interpersonal stress, processing emotions, and improving interpersonal skills as possible mechanisms of change regarding the effects of IPT in depression, while acknowledging that studies supporting these mechanisms are lacking.

To the mechanisms of change invoked by IPT in depression, IPTSR adds the stabilization of daily rhythms and the management of potential precipitants of rhythm dysregulation as factors that moderate the risk of BD symptoms (Frank et al., 2000). Findings from one clinical trial (Frank et al., 2005) suggest that regulating daily routines mediates the effect of acute-phase IPTSR on recurrence in BD. To our knowledge, studies have not tested other mediators of treatment response to IPTSR. Thus, whereas there is preliminary support for the regulation of daily rhythms, there are no data related to other mechanisms of change.

3.2.5 Categories V, VI, VII, VIII, and IX

Included in Categories V–IX are:

- good-intervention- and bad-theory-driven psychotherapies (Category V);
- good-theory- and bad-intervention-driven psychotherapies (Category VI);
- bad-theory-driven psychotherapies (Category VII);
- bad-intervention-driven psychotherapies (Category VIII);
- bad-theory- and bad-intervention-driven psychotherapies (Category IX).

We did not identify psychological interventions for BD that could be included in these categories.

3.3 Conclusions and Future Directions

Despite appropriate pharmacological treatment, a high percentage of patients with BD continue to experience relapses, residual affective symptoms, mood cycling, and significant impairment in psychosocial functioning. Accordingly, over the past two decades, interest has burgeoned in developing and validating psychological interventions as adjuncts to medication for patients with BD. In this chapter, we have reviewed evidence supporting the main psychotherapeutic approaches to BD: CBT, FFT, IPTSR, and various forms of psychoeducation.

Although these interventions are based on differing assumptions and may employ different methods and treatment structures (Geddes & Miklowitz, 2013), they overlap in terms of their objectives, which usually include educating the patient about the disorder,

helping the patient identify and manage early signs of recurrence, increasing treatment adherence, enhancing ability to cope with stressors, stabilizing sleep–wake patterns and daily routines, reducing substance misuse, and improving social relations (Geddes & Miklowitz, 2013). Indeed, researchers have suggested that psychoeducation is the active ingredient in most psychotherapies for BD, but this hypothesis has yet to be validated (Geddes & Miklowitz, 2013).

Overall, psychotherapy seems to be an effective adjunct to medication or TAU in stabilizing mood episodes and preventing relapses in patients with BD. Two meta-analyses that examined the efficacy of psychotherapy in preventing relapses in BD have found delayed relapses, reduction in relapse rates, and increased social functioning in patients receiving various forms of psychotherapy compared to TAU (Lam, Burbeck, Wright, & Pilling, 2009; Scott, Colom, & Vieta, 2007). Nevertheless, although the number of clinical trials evaluating the efficacy of various psychological treatments for BD has increased in recent years, evidence concerning each treatment, considered individually, is either mixed or too preliminary to support firm conclusions. Moreover, according to the most recent NICE guideline on BD, the quality of evidence ranges from very low to moderate at best, with most studies reporting low- or very-low-quality evidence (NICE, 2014). More large-scale, well-designed clinical trials are needed to clearly establish the benefits of psychological interventions in BD. As seen in this chapter, different psychological interventions may be more effective in affecting different manifestations of the disorder. It is therefore important that future studies focus on determining which type of intervention is most effective for which type of patient (Lam et al., 2009; Scott et al., 2007). Comparisons among psychotherapies for BD are made difficult by the high variability in study criteria and treatment outcomes; therefore, research aims should also include more consistency in criteria for treatment success and standardization of outcomes.

As far as mechanisms of change are concerned, our analysis concurs with previous reviews in concluding that the state of the research is far from ideal and that, despite the expansion of research during the past two decades, true advances in understanding the disorder have been few (Geddes & Miklowitz, 2013). The theories proposed by various psychotherapeutic approaches are generally diathesis–stress models, in which stressful life events interact with biological and psychological predispositions, engendering the illness in vulnerable individuals (Power, 2005). Nevertheless, such theories have been criticized for lacking a detailed account of the complex clinical picture of BD (Power, 2005). Furthermore, studies have yet to test the mechanisms of change proposed by different psychotherapies, as evidence regarding mechanisms is virtually nonexistent. In some cases (e.g., psychoeducation), no cogent theory of change has been proposed to explain the effects of the intervention. We fully agree with Geddes and Miklowitz (2013) that "the next generation of psychotherapy studies will need to systematically assess mediators in the pathways from treatments to changes in illness course" and that "the identification of mechanisms of change might lead to the development of briefer treatments with more durable effects" (p. 1679).

Given the considerations we have noted, it is fair to argue that no psychological intervention for BD currently fits the criteria for Category I (i.e., evidence-based), Category II (i.e., intervention-driven), or Category III (i.e., theory-driven) treatments in David and Montgomery's (2011) framework. We therefore included all of them in Category IV (i.e., investigational). Nevertheless, it is important to mention that the interventions in this category are not equivalent in status. More specifically, whereas some (e.g., CBT,

FFT) are more homogenous in terms of treatment structure and methods and have been assessed in large-scale clinical trials conducted by independent research groups, other interventions are more heterogeneous (e.g., psychoeducation) or have been tested only in a limited number of studies (e.g., IPTSR). Also, some interventions are based on a treatment package and a theory of change (e.g., CBT), whereas others are mostly treatment-driven, with no specific theory of the disorder that motivates the intervention.

In addition to the psychological treatments included in this chapter, collaborative care programs for patients with BD are also available. These programs are usually multifaceted, involving changes to service delivery interventions (Bond & Anderson, 2015). Although investigators have reported encouraging results following these interventions (see Bauer et al., 2006; Kessing et al., 2013; Simon et al., 2005; Simon, Ludman, Bauer, Unutzer, & Operskalski, 2006; Van de Voort et al., 2011), we chose not to include them in our analysis, as their multicomponent nature would not allow a clear evaluation based on the framework used in the chapter.

An overview of the literature shows that studies that assess the efficacy of various psychological treatments for youth with bipolar spectrum disorders (BPSDs) are considerably fewer than corresponding studies that assess treatments for adults. Although BPSDs were once thought to be primarily disorders of adulthood, evidence now shows that prevalence rates of BPSDs among children and adolescents are comparable to estimates reported among adults (Van Meter, Moreira, & Youngstrom, 2011). BPSDs in youth are a serious public health concern as they are associated with considerable morbidity and mortality (Fristad & MacPherson, 2014).

As in the case of adult BD, practice guidelines recommend both pharmacotherapy and psychological approaches in the treatment of youth with BPSDs (see Kowatch, Fristad, Findling, & Post, 2009; NICE, 2014). CBT, family interventions, psychoeducation, and IPT are specifically mentioned by the guidelines. Additionally, the latest NICE guideline recommends manualized psychological interventions as the first line of treatment for depressive episodes in children and young people with BD, and that psychological interventions should be offered before pharmacological interventions are considered (NICE, 2014). Structured individual or family interventions are recommended for the long-term management of BD in youth (NICE, 2014).

A review on the efficacy of psychological interventions for children and adolescents with BPSDs (Fristad & MacPherson, 2014), which analyzed existing studies based on the guidelines proposed by the Task Force on the Promotion and Dissemination of Psychological Procedures (e.g., Chambless & Hollon, 1998), concludes that currently there are no *well-established treatments* or *treatments of questionable efficacy*. Family interventions were evaluated as *probably efficacious*, CBT as *possibly efficacious*, and other treatments such as IPTSR and dialectical behavior therapy were judged as *experimental*.

In terms of David and Montgomery's framework (2011), encouraging (albeit preliminary and/or mixed) data are available for the treatment package of FFT (Miklowitz et al., 2008, 2011, 2013, 2014), multifamily psychoeducational psychotherapy (Fristad, Goldberg-Arnold, & Gavazzi, 2002, 2003; Fristad, Goldberg-Arnold, & Leffler, 2011; Fristad, Verducci, Walters, & Young, 2009; MacPherson, Leffler, & Fristad, 2014), and CBT (Feeny, Danielson, Schwartz, Youngstrom, & Findling, 2006; Pavuluri et al., 2004; West et al., 2009, 2014). To our knowledge, only one study has examined mechanisms of change in the treatment of pediatric BD (MacPherson, Weinstein, Henry, & West, 2016). The authors found that the superior effects (reported in West et al., 2014) of child- and

family-focused CBT compared with TAU in terms of improvement in mania, depression, and global functioning were mediated by parenting skills and coping, family flexibility, and family positive reframing.

Finally, a few recent studies have explored the possibility of offering psychological interventions in web-based formats or via the phone. Some of these interventions combine treatment delivered in a traditional format with technology-delivered content (Depp et al., 2015; Smith et al., 2011), whereas others rely on web-based and/or telephone-based delivery only (Proudfoot et al., 2012; Todd, Jones, Hart, & Lobban, 2014). Initial data support the feasibility and acceptability of these interventions, which are worth further exploration, as they could be associated with benefits such as increased accessibility to psychological treatments and reduced costs and stigma.

References

Angst, J. (1966). *Zur Ätiologie und Nosologie endogener depressiver Psychosen: Eine genetische, soziologische und klinische Studie* [On the etiology and nosology of endogenous depressive psychoses: A genetic, sociological and clinical study]. Berlin: Springer.

Angst, J., Gamma, A., Sellaro, R., Lavori, P. W., & Zhang, H. P. (2003). Recurrence of bipolar disorders and major depression: A life-long perspective. *European Archives of Psychiatry and Clinical Neuroscience*, *253*(5), 236–240. doi:10.1007/s00406-003-0437-2

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.

Ball, J. R., Mitchell, P. B., Corry, J. C., Skillecorn, A., & Malhi, G. S. (2006). A randomized controlled trial of cognitive therapy for bipolar disorder: Focus on long-term change. *Journal of Clinical Psychiatry*, *67*(2), 277–286. doi:10.4088/jcp.v67n0215

Bauer, M. S., McBride, L., Williford, W. O., Glick, H., Kinosian, B., Altshuler, L., … Coauthors for the Cooperative Studies Program 430 Study Team. (2006). Collaborative care for bipolar disorder: Part II. Impact on clinical outcome, function, and costs. *Psychiatric Services*, *57*(7), 937–945. doi:10.1176/appi.ps.57.7.937

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). Cognitive therapy of depression. New York, NY: Guilford Press.

Bond, K., & Anderson, I. M. (2015). Psychoeducation for relapse prevention in bipolar disorder: A systematic review of efficacy in randomized controlled trials. *Bipolar Disorders*, *17*(4), 349–362. doi:10.1111/bdi.12287

Bordbar, M. R. F., Soltanifar, A., & Talaei, A. (2009). Short-term family-focused psycho-educational program for bipolar mood disorder in Mashhad. *Iranian Journal of Medical Sciences*, *34*(2), 104–109.

Castle, D., White, C., Chamberlain, J., Berk, M., Berk, L., Lauder, S., … Gilbert, M. (2010). Group-based psychosocial intervention for bipolar disorder: Randomised controlled trial. *British Journal of Psychiatry*, *196*(5), 383–388. doi:10.1192/bjp.bp.108.058263

Chambless, D. L., & Hollon, S. D. (1998). Defining empirically supported therapies. *Journal of Consulting and Clinical Psychology*, *66*(1), 7–18.

Clarkin, J. F., Carpenter, D., Hull, J., Wilner, P., & Glick, I. (1998). Effects of psychoeducational intervention for married patients with bipolar disorder and their spouses. *Psychiatric Services*, *49*(4), 531–533. doi:10.1176/ps.49.4.531

Colom, F., Vieta, E., Martinez-Aran, A., Reinares, M., Goikolea, J. M., Benabarre, A., … Corominas, J. (2003). A randomized trial on the efficacy of group psychoeducation in the prophylaxis of recurrences in bipolar patients whose disease is in remission. *Archives of General Psychiatry*, *60*(4), 402–407. doi:10.1001/archpsyc.60.4.402

Costa, R. T., Cheniaux, E., Rangé, B. P., Versiani, M., & Nardi, A. E. (2012). Group cognitive behavior therapy for bipolar disorder can improve the quality of life. *Brazilian Journal of Medical and Biological Research*, *45*(9), 862–868. doi:10.1590/S0100-879X2012007500109

David, D., & Montgomery, G. H. (2011). The scientific status of psychotherapies: A new evaluative framework for evidence-based psychosocial interventions. *Clinical Psychology: Science and Practice*, *18*(2), 89–99. doi:10.1111/j.1468-2850.2011.01239.x

de Barros Pellegrinelli, K. D., de O Costa, L. F., Silval, K. I. D., Dias, V. V., Roso, M. C., Bandeira, M., … Moreno, R. A. (2013). Efficacy of psychoeducation on symptomatic and functional recovery in bipolar disorder. *Acta Psychiatrica Scandinavica*, *127*(2), 153–158. doi:10.1111/acps.12007

Deckersbach, T., Holzel, B. K., Eisner, L. R., Stange, J. P., Peckham, A. D., Dougherty, D. D., … Nierenberg, A. A. (2012). Mindfulness-based cognitive therapy for nonremitted patients with bipolar disorder. *CNS Neuroscience & Therapeutics*, *18*(2), 133–141. doi:10.1111/j.1755-5949.2011.00236.x

Depp, C., Ceglowski, J., Wang, V. C., Yaghouti, F., Mausbach, B. T., Thompson, W. K., & Granholm, E. L. (2015). Augmenting psychoeducation with a mobile intervention for bipolar disorder: A randomized controlled trial. *Bipolar Disorders*, *17*, 22–23.

Depue, R. A., Krauss, S. P., & Spoont, M. R. (1987). A two-dimensional threshold model of seasonal bipolar affective disorder. In A. O. D. Magnusson & A. Ohman (Eds.), *Psychopathology: An interactionist perspective* (pp. 95–123). New York, NY: Academic Press.

Doğan, S., & Sabancioğullari, S. (2003). The effects of patient education in lithium therapy on quality of life and compliance. *Archives of Psychiatric Nursing*, *17*(6), 270–275. doi:10.1053/S0883-7417(03)00129-8

D'Souza, R., Piskulic, D., & Sundram, S. (2010). A brief dyadic group based psychoeducation program improves relapse rates in recently remitted bipolar disorder: A pilot randomised controlled trial. *Journal of Affective Disorders*, *120*(1/3), 272–276. doi:10.1016/j.jad.2009.03.018

Eker, F., & Harkin, S. (2012). Effectiveness of six-week psychoeducation program on adherence of patients with bipolar affective disorder. *Journal of Affective Disorders*, *138*(3), 409–416. doi:10.1016/j.jad.2012.01.004

Fagiolini, A., Kupfer, D. J., Masalehdan, A., Scott, J. A., Houck, P. R., & Frank, E. (2005). Functional impairment in the remission phase of bipolar disorder. *Bipolar Disorders*, *7*(3), 281–285. doi:10.1111/j.1399-5618.2005.00207.x

Feeny, N. C., Danielson, C. K., Schwartz, L., Youngstrom, E. A., & Findling, R. L. (2006). Cognitive-behavioral therapy for bipolar disorders in adolescents: A pilot study. *Bipolar Disorders*, *8*(5), 508–515. doi:10.1111/j.1399-5618.2006.00358.x

Fiedorowicz, J. G., Palagummi, N. M., Forman-Hoffman, V. L., Miller, D. D., & Haynes, W. G. (2008). Elevated prevalence of obesity, metabolic syndrome, and cardiovascular risk factors in bipolar disorder. *Annals of Clinical Psychiatry*, *20*(3), 131–137. doi:10.1080/10401230802177722

Fiorillo, A., Del Vecchio, V., Luciano, M., Sampogna, G., De Rosa, C., Malangone, C., … Maj, M. (2015). Efficacy of psychoeducational family intervention for bipolar I disorder:

A controlled, multicentric, real-world study. *Journal of Affective Disorders*, *172*, 291–299. doi:10.1016/j.jad.2014.10.021

Fountoulakis, K. N., Kasper, S., Andreassen, O., Blier, P., Okasha, A., Severus, E., … Vieta, E. (2012). Efficacy of pharmacotherapy in bipolar disorder: A report by the WPA section on pharmacopsychiatry. *European Archives of Psychiatry and Clinical Neuroscience*, *262*, S1–S48. doi:10.1007/s00406-012-0323-x

Frank, E., Kupfer, D. J., Thase, M. E., Mallinger, A. G., Swartz, H. A., Fagiolini, A. M., … Monk, T. (2005). Two-year outcomes for interpersonal and social rhythm therapy in individuals with bipolar I disorder. *Archives of General Psychiatry*, *62*(9), 996–1004. doi:10.1001/archpsyc.62.9.996

Frank, E., Swartz, H. A., & Kupfer, D. J. (2000). Interpersonal and social rhythm therapy: Managing the chaos of bipolar disorder. *Biological Psychiatry*, *48*(6), 593–604. doi:10.1016/S0006-3223(00)00969-0

Fristad, M. A., Goldberg-Arnold, J. S., & Gavazzi, S. M. (2002). Multifamily psychoeducation groups (MFPG) for families of children with bipolar disorder. *Bipolar Disorders*, *4*(4), 254–262. doi:10.1034/j.1399-5618.2002.09073.x

Fristad, M. A., Goldberg-Arnold, J. S., & Gavazzi, S. M. (2003). Multi-family psychoeducation groups in the treatment of children with mood disorders. *Journal of Marital and Family Therapy*, *29*(4), 491–504. doi:10.1111/j.1752-0606.2003.tb01691.x

Fristad, M. A., Goldberg-Arnold, J. S., & Leffler, J. M. (2011). *Psychotherapy for children with bipolar and depressive disorders*. New York, NY: Guilford Press.

Fristad, M. A., & MacPherson, H. A. (2014). Evidence-based psychosocial treatments for child and adolescent bipolar spectrum disorders. *Journal of Clinical Child & Adolescent Psychology*, *43*(3), 339–355. doi:10.1080/15374416.2013.822309

Fristad, M. A., Verducci, J. S., Walters, K., & Young, M. E. (2009). Impact of multifamily psychoeducational psychotherapy in treating children aged 8 to 12 years with mood disorders. *Archives of General Psychiatry*, *66*(9), 1013–1021. doi:10.1001/archgenpsychiatry.2009.112

Geddes, J. R., & Miklowitz, D. J. (2013). Treatment of bipolar disorder. *Lancet*, *381*(9878), 1672–1682. doi:10.1016/s0140-6736(13)60857-0

Goldberg, J. F., Gerstein, R. K., Wenze, S. J., Welker, T. M., & Beck, A. T. (2008). Dysfunctional attitudes and cognitive schemas in bipolar manic and unipolar depressed outpatients: Implications for cognitively based psychotherapeutics. *Journal of Nervous and Mental Disease*, *196*(3), 207–210. doi:10.1097/NMD.0b013e3181663015

Gregory, V. L. (2010a). Cognitive-behavioral therapy for depression in bipolar disorder: A meta-analysis. *Journal of Evidence-Based Social Work*, *7*(4), 269–279. doi:10.1080/15433710903176088

Gregory, V. L. (2010b). Cognitive-behavioral therapy for mania: A meta-analysis of randomized controlled trials. *Social Work in Mental Health*, *6*(8), 483–494. doi:10.1080/15332981003744388

Healy, D., & Williams, J. M. (1989). Moods, misattributions and mania: An interaction of biological and psychological factors in the pathogenesis of mania. *Psychiatric Developments*, *7*(1), 49–70.

Høyer, E. H., Olesen, A. V., & Mortensen, P. B. (2004). Suicide risk in patients hospitalised because of an affective disorder: A follow-up study, 1973–1993. *Journal of Affective Disorders*, *78*(3), 209–217. doi:10.1016/S0165-0327(02)00311-7

Inder, M. L., Crowe, M. T., Luty, S. E., Carter, J. D., Moor, S., Frampton, C. M., & Joyce, P. R. (2015). Randomized, controlled trial of Interpersonal and Social Rhythm Therapy for

young people with bipolar disorder. *Bipolar Disorders*, *17*(2), 128–138. doi:10.1111/bdi.12273

Javadpour, A., Hedayati., A., Dehbozorgi, G. R., & Azizi, A. (2013). The impact of a simple psycho-education program on quality of life, rate of relapse and medication adherence in bipolar disorder patients. *Asian Journal of Psychiatry*, *6*, 208–213. doi:10.1016/j.ajp.2012.12.005

Johnson, S., & Tran, T. (2007). Bipolar disorder: What can psychotherapists learn from the cognitive research? *Journal of Clinical Psychology*, *63*(5), 425–432. doi:10.1002/jclp.20361

Jones, S. H., Smith, G., Mulligan, L. D., Lobban, F., Law, H., Dunn, G., … Morrison, A. P. (2015). Recovery-focused cognitive behavioural therapy for recent-onset bipolar disorder: Randomised controlled pilot trial. *British Journal of Psychiatry*, *206*(1), 58–66. doi:10.1192/bjp.bp.113.141259

Judd, L. L., Akiskal, H. S., Schettler, P. J., Coryell, W., Endicott, J., Maser, J. D., … Keller, M. B. (2003). A prospective investigation of the natural history of the long-term weekly symptomatic status of bipolar II disorder. *Archives of General Psychiatry*, *60*(3), 261–269. doi:10.1001/archpsyc.60.3.261

Judd, L. L., Akiskal, H. S., Schettler, P. J., Endicott, J., Maser, J., Solomon, D. A., … Keller, M. B. (2002). The long-term natural history of the weekly symptomatic status of bipolar I disorder. *Archives of General Psychiatry*, *59*(6), 530–537. doi:10.1001/archpsyc.59.6.530

Justo, L. P., Soares, B. G., & Calil, H. M. (2007). Family interventions for bipolar disorder. *Cochrane Database of Systematic Reviews*, *4*, CD005167. doi:10.1002/14651858.CD005167.pub2

Kabat-Zinn, J. (1990). *Full catastrophe living: The program of the stress reduction clinic at the University of Massachusetts Medical Center*. New York, NY: Delta.

Kessing, L. V., Hansen, H. V., Hvenegaard, A., Christensen, E. M., Dam, H., Gluud, C., … Disor, E. I. A. (2013). Treatment in a specialised out-patient mood disorder clinic v. standard out-patient treatment in the early course of bipolar disorder: Randomised clinical trial. *British Journal of Psychiatry*, *202*(3), 212–219. doi:10.1192/bjp.bp.112.113548

Kim, E. Y., & Miklowitz, D. J. (2004). Expressed emotion as a predictor of outcome among bipolar patients undergoing family therapy. *Journal of Affective Disorders*, *82*(3), 343–352. doi:10.1016/j.jad.2004.02.004

Klerman, G. L., Weissman, M. M., Rounsaville, B. J., & Chevron, E. S. (1984). *Interpersonal psychotherapy of depression*. New York, NY: Basic Books.

Kowatch, R. A., Fristad, M. A., Findling, R. L., & Post, R. M. (2009). *Clinical manual for management of bipolar disorder in children and adolescents*. Washington, DC: American Psychiatric Association.

Kroon, J. S., Wohlfarth, T. D., Dieleman, J., Sutterland, A. L., Storosum, J. G., Denys, D., … Sturkenboom, M. C. (2013). Incidence rates and risk factors of bipolar disorder in the general population: A population-based cohort study. *Bipolar Disorders*, *15*(3), 306–313. doi:10.1111/bdi.12058

Lam, D., Wright, K., & Smith, N. (2004). Dysfunctional assumptions in bipolar disorder. *Journal of Affective Disorders*, *79*(1/3), 193–199. doi:10.1016/S0165-0327(02)00462-7

Lam, D. H., Burbeck, R., Wright, K., & Pilling, S. (2009). Psychological therapies in bipolar disorder: The effect of illness history on relapse prevention—A systematic review. *Bipolar Disorders*, *11*(5), 474–482. doi:10.1111/j.1399-5618.2009.00724.x

Lam, D. H., Jones, S. H., & Hayward, P. (2010). *Cognitive therapy for bipolar disorder: A therapist's guide to concepts, methods and practice*. Chichester, UK: Wiley-Blackwell.

Lam, D. H., Watkins, E. R., Hayward, P., Bright, J., Wright, K., Kerr, N., … Sham, P. (2003). A randomized controlled study of cognitive therapy for relapse prevention for bipolar affective disorder: Outcome of the first year. *Archives of General Psychiatry*, *60*(2), 145–152. doi:10.1001/archpsyc.60.2.145

Lipsitz, J. D., & Markowitz, J. C. (2013). Mechanisms of change in interpersonal therapy (IPT). *Clinical Psychology Review*, *33*(8), 1134–1147. doi:10.1016/j.cpr.2013.09.002

Lobban, F., Taylor, L., Chandler, C., Tyler, E., Kinderman, P., Kolamunnage-Dona, R., … Morriss, R. K. (2010). Enhanced relapse prevention for bipolar disorder by community mental health teams: Cluster feasibility randomised trial. *British Journal of Psychiatry*, *196*(1), 59–63. doi:10.1192/bjp.bp.109.065524

Lynch, D., Laws, K. R., & McKenna, P. J. (2010). Cognitive behavioural therapy for major psychiatric disorder: Does it really work? A meta-analytical review of well-controlled trials. *Psychological Medicine*, *40*(1), 9–24. doi:10.1017/S003329170900590X

MacPherson, H. A., Leffler, J. M., & Fristad, M. A. (2014). Implementation of multi-family psychoeducational psychotherapy for childhood mood disorders in an outpatient community setting. *Journal of Marital and Family Therapy*, *40*(2), 193–211. doi:10.1111/jmft.12013

MacPherson, H. A., Weinstein, S. M., Henry, D. B., & West, A. E. (2016). Mediators in the randomized trial of child and family-focused cognitive-behavioral therapy for pediatric bipolar disorder. *Behaviour Research and Therapy*, *85*, 60–71. doi:10.1016/j.brat.2016.08.014

Madigan, K., Egan, P., Brennan, D., Hill, S., Maguire, B., Horgan, F., … O'Callaghan, E. (2012). A randomised controlled trial of carer-focussed multi-family group psychoeducation in bipolar disorder. *European Psychiatry*, *27*(4), 281–284. doi:10.1016/j.eurpsy.2010.12.008

Mansell, W., Morrison, A. P., Reid, G., Lowens, I., & Tai, S. (2007). The interpretation of, and responses to, changes in internal states: An integrative cognitive model of mood swings and bipolar disorders. *Behavioural and Cognitive Psychotherapy*, *35*(5), 515–539. doi:10.1017/S1352465807003827

Mansell, W., Tai, S., Clark, A., Akgonul, S., Dunn, G., Davies, L., … Morrison, A. P. (2014). A novel cognitive behaviour therapy for bipolar disorders (Think Effectively About Mood Swings or TEAMS): Study protocol for a randomized controlled trial. *Trials*, *15*. doi:10.1186/1745-6215-15-405

Marneros, A., & Angst, J. (2000). Bipolar disorders: Roots and evolution. In A. Marneros & J. Angst (Eds.), *Bipolar disorders 100 years after manic-depressive insanity* (pp. 1–35). New York, NY: Kluwer Academic.

Merikangas, K. R., Akiskal, H. S., Angst, J., Greenberg, P. E., Hirschfeld, R. M., Petukhova, M., & Kessler, R. C. (2007). Lifetime and 12-month prevalence of bipolar spectrum disorder in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *64*(5), 543–552. doi:10.1001/archpsyc.64.5.543

Merikangas, K. R., Jin, R., He, J. P., Kessler, R. C., Lee, S., Sampson, N. A., … Zarkov, Z. (2011). Prevalence and correlates of bipolar spectrum disorder in the World Mental Health Survey Initiative. *Archives of General Psychiatry*, *68*(3), 241–251. doi:10.1001/archgenpsychiatry.2011.12

Miklowitz, D. J. (2008). *Bipolar disorder: A family-focused treatment approach*. New York, NY: Guilford Press.

Miklowitz, D. J., Alatiq, Y., Goodwin, G. M., Geddes, J. R., Fennell, M. J. V., Dimidjian, S., … Williams, J. M. G. (2009). A pilot study of mindfulness-based cognitive therapy for bipolar disorder. *International Journal of Cognitive Therapy*, *2*(4), 373–382.

Miklowitz, D. J., Axelson, D. A., Birmaher, B., George, E. L., Taylor, D. O., Schneck, C. D., … Brent, D. A. (2008). Family-focused treatment for adolescents with bipolar disorder: Results of a 2-year randomized trial. *Archives of General Psychiatry*, *65*(9), 1053–1061. doi:10.1001/archpsyc.65.9.1053

Miklowitz, D. J., Chang, K. D., Taylor, D. O., George, E. L., Singh, M. K., Schneck, C. D., … Garber, J. (2011). Early psychosocial intervention for youth at risk for bipolar I or II disorder: A one-year treatment development trial. *Bipolar Disorders*, *13*(1), 67–75. doi:10.1111/j.1399-5618.2011.00890.x

Miklowitz, D. J., George, E. L., Richards, J. A., Simoneau, T. L., & Suddath, R. L. (2003). A randomized study of family-focused psychoeducation and pharmacotherapy in the outpatient management of bipolar disorder. *Archives of General Psychiatry*, *60*(9), 904–912. doi:10.1001/archpsyc.60.9.904

Miklowitz, D. J., Otto, M. W., Frank, E., Reilly-Harrington, N. A., Kogan, J. N., Sachs, G. S., … Wisniewski, S. R. (2007). Intensive psychosocial intervention enhances functioning in patients with bipolar depression: Results from a 9-month randomized controlled trial. *American Journal of Psychiatry*, *164*(9), 1340–1347. doi:10.1176/appi.apj.2007.07020311

Miklowitz, D. J., Otto, M. W., Frank, E., Reilly-Harrington, N. A., Wisniewski, S. R., Kogan, J. N., … Sachs, G. S. (2007). Psychosocial treatments for bipolar depression: A 1-year randomized trial from the systematic treatment enhancement program. *Archives of General Psychiatry*, *64*(4), 419–427. doi:10.1001/archpsyc.64.4.419

Miklowitz, D. J., Schneck, C. D., George, E. L., Taylor, D. O., Sugar, C. A., Birmaher, B., … Axelson, D. A. (2014). Pharmacotherapy and Family-focused treatment for adolescents with bipolar I and II disorders: A 2-year randomized trial. *American Journal of Psychiatry*, *171*(6), 658–667. doi:10.1176/appi.ajp.2014.13081130

Miklowitz, D. J., Schneck, C. D., Singh, M. K., Taylor, D. O., George, E. L., Cosgrove, V. E., … Chang, K. K. D. (2013). Early intervention for symptomatic youth at risk for bipolar disorder: A randomized trial of family-focused therapy. *Journal of the American Academy of Child & Adolescent Psychiatry*, *52*(2), 121–131. doi:10.1016/j.jaac.2012.10.007

Miklowitz, D. J., Semple, R. J., Hauser, M., Elkun, D., Weintraub, M. J., & Dimidjian, S. (2015). Mindfulness-based cognitive therapy for perinatal women with depression or bipolar spectrum disorder. *Cognitive Therapy and Research*, *39*(5), 590–600. doi:10.1007/s10608-015-9681-9

Miklowitz, D. J., Simoneau, T. L., George, E. L., Richards, J. A., Kalbag, A., Sachs-Ericsson, N., & Suddath, R. (2000). Family-focused treatment of bipolar disorder: 1-year effects of a psychoeducational program in conjunction with pharmacotherapy. *Biological Psychiatry*, *48*(6), 582–592. doi:10.1016/S0006-3223(00)00931-8

Miller, I. W., Solomon, D. A., Ryan, C. E., & Keitner, G. I. (2004). Does adjunctive family therapy enhance recovery from bipolar I mood episodes? *Journal of Affective Disorders*, *82*(3), 431–436. doi:10.1016/j.jad.2004.01.010

Miziou, S., Tsitsipa, E., Moysidou, S., Karavelas, V., Dimelis, D., Polyzoidou, V., & Fountoulakis, K. N. (2015). Psychosocial treatment and interventions for bipolar

disorder: A systematic review. *Annals of General Psychiatry*, *14*. doi:10.1186/s12991-015-0057-z

Morris, C. D., Miklowitz, D. J., & Waxmonsky, J. A. (2007). Family-focused treatment for bipolar disorder in adults and youth. *Journal of Clinical Psychology*, *63*(5), 433–445. doi:10.1002/jclp.20359

Murray, G., Leitan, N. D., Berk, M., Thomas, N., Michalak, E., Berk, L., … Kyrios, M. (2015). Online mindfulness-based intervention for late-stage bipolar disorder: Pilot evidence for feasibility and effectiveness. *Journal of Affective Disorders*, *178*, 46–51. doi:10.1016/j.jad.2015.02.024

National Institute for Health and Care Excellence. (2014). *The NICE guideline on the assessment and management of bipolar disorder in adults, children and young people in primary and secondary care*. London, UK: National Institute for Health and Care Excellence.

Newman, C. F., Leahy, R. L., Beck, A. T., Reilly-Harrington, N., & Gyulai, L. (2002). *Bipolar disorder: A cognitive therapy approach*. Washington, DC: American Psychological Association.

Parikh, S. V., Zaretsky, A., Beaulieu, S., Yatham, L. N., Young, L. T., Patelis-Siotis, I., … Streiner, D. L. (2012). A randomized controlled trial of psychoeducation or cognitive-behavioral therapy in bipolar disorder: A Canadian Network for Mood and Anxiety Treatments (CANMAT) study. *Journal of Clinical Psychiatry*, *73*(6), 803–810. doi:10.4088/Jcp.11m07343

Pavuluri, M. N., Graczyk, P. A., Henry, D. B., Carbray, J. A., Heidenreich, J., & Miklowitz, D. J. (2004). Child- and family-focused cognitive-behavioral therapy for pediatric bipolar disorder: Development and preliminary results. *Journal of the American Academy of Child & Adolescent Psychiatry*, *43*(5), 528–537. doi:10.1097/01.chi.0000116743.71662.f8

Peet, M., & Harvey, N. S. (1991). Lithium maintenance. 1. A standard education-program for patients. *British Journal of Psychiatry*, *158*, 197–200. doi:10.1192/bjp.158.2.197

Perich, T., Manicavasagar, V., Mitchell, P. B., Ball, J. R., & Hadzi-Pavlovic, D. (2013). A randomized controlled trial of mindfulness-based cognitive therapy for bipolar disorder. *Acta Psychiatrica Scandinavica*, *127*(5), 333–343. doi:10.1111/acps.12033

Perlick, D. A., Miklowitz, D. J., Lopez, N., Chou, J., Kalvin, C., Adzhiashvili, V., & Aronson, A. (2010). Family-focused treatment for caregivers of patients with bipolar disorder. *Bipolar Disorders*, *12*(6), 627–637. doi:10.1111/j.1399-5618.2010.00852.x

Perris, C. (1966). A study of bipolar (manic-depressive) and unipolar recurrent depressive psychoses: Introduction. *Acta Psychiatrica Scandinavica*, *194*(Suppl.), 9–14.

Perry, A., Tarrier, N., Morriss, R., McCarthy, E., & Limb, K. (1999). Randomised controlled trial of efficacy of teaching patients with bipolar disorder to identify early symptoms of relapse and obtain treatment. *BMJ*, *318*(7177), 149–153. doi:10.1136/bmj.318.7177.149

Power, M. J. (2005). Psychological approaches to bipolar disorders: A theoretical critique. *Clinical Psychology Review*, *25*(8), 1101–1122. doi:10.1016/j.cpr.2005.06.008

Proudfoot, J., Parker, G., Manicavasagar, V., Hadzi-Pavlovic, D., Whitton, A., Nicholas, J., … Burckhardt, R. (2012). Effects of adjunctive peer support on perceptions of illness control and understanding in an online psychoeducation program for bipolar disorder: A randomised controlled trial. *Journal of Affective Disorders*, *142*(1/3), 98–105. doi:10.1016/j.jad.2012.04.007

Rea, M. M., Tompson, M. C., Miklowitz, D. J., Goldstein, M. J., Hwang, S., & Mintz, J. (2003). Family-focused treatment versus individual treatment for bipolar disorder:

Results of a randomized clinical trial. *Journal of Consulting and Clinical Psychology*, *71*(3), 482–492. doi:10.1037/0022-006X.71.3.482

Reilly-Harrington, N. A., Alloy, L. B., Fresco, D. M., & Whitehouse, W. G. (1999). Cognitive styles and life events interact to predict bipolar and unipolar symptomatology. *Journal of Abnormal Psychology*, *108*(4), 567–578. doi:10.1037//0021-843x.108.4.567

Reinares, M., Colom, F., Sánchez-Moreno, J., Torrent, C., Martinez-Aran, A., Comes, M., … Vieta, E. (2008). Impact of caregiver group psychoeducation on the course and outcome of bipolar patients in remission: A randomized controlled trial. *Bipolar Disorders*, *10*(4), 511–519. doi:10.1111/j.1399-5618.2008.00588.x

Reinares, M., Sánchez-Moreno, J., & Fountoulakis, K. N. (2014). Psychosocial interventions in bipolar disorder: What, for whom, and when. *Journal of Affective Disorders*, *156*, 46–55. doi:10.1016/j.jad.2013.12.017

Reinares, M., Vieta, E., Colom, F., Martinez-Aran, A., Torrent, C., Comes, M., … Sanchez-Moreno, J. (2004). Impact of a psychoeducational family intervention on caregivers of stabilized bipolar patients. *Psychotherapy and Psychosomatics*, *73*(5), 312–319. doi:10.1159/000078848

Scott, J. (2006). Psychotherapy for bipolar disorders: Efficacy and effectiveness. *Journal of Psychopharmacology*, *20*(2), 46–50. doi:10.1177/1359786806063078

Scott, J., Colom, F., & Vieta, E. (2007). A meta-analysis of relapse rates with adjunctive psychological therapies compared to usual psychiatric treatment for bipolar disorders. *International Journal of Neuropsychopharmacology*, *10*(1), 123–129. doi:10.1017/S1461145706006900

Scott, J., Paykel, E., Morriss, R., Bentall, R., Kinderman, P., Johnson, T., … Hayhurst, H. (2006). Cognitive-behavioural therapy for severe and recurrent bipolar disorders: Randomised controlled trial. *British Journal of Psychiatry*, *188*, 313–320. doi:10.1192/bjp.188.4.313

Scott, J., & Pope, M. (2002). Self-reported adherence to treatment with mood stabilizers, plasma levels, and psychiatric hospitalization. *American Journal of Psychiatry*, *159*(11), 1927–1929. doi:10.1176/appi.ajp.159.11.1927

Scott, J., Stanton, B., Garland, A., & Ferrier, I. N. (2000). Cognitive vulnerability in patients with bipolar disorder. *Psychological Medicine*, *30*(2), 467–472. doi:10.1017/S0033291799008879

Segal, Z. W., Williams, J. M. G., & Teasdale, J. D. (2001). *Mindfulness-based cognitive therapy for depression*. New York, NY: Guilford Press.

Simon, G. E., Ludman, E. J., Bauer, M. S., Unutzer, J., & Operskalski, B. (2006). Long-term effectiveness and cost of a systematic care program for bipolar disorder. *Archives of General Psychiatry*, *63*(5), 500–508. doi:10.1001/archpsyc.63.5.500

Simon, G. E., Ludman, E. J., Unutzer, J., Bauer, M. S., Operskalski, B., & Rutter, C. (2005). Randomized trial of a population-based care program for people with bipolar disorder. *Psychological Medicine*, *35*(1), 13–24. doi:10.1017/S0033291704002624

Smith, D. J., Griffiths, E., Poole, R., di Florio, A., Barnes, E., Kelly, M. J., … Simpson, S. (2011). Beating bipolar: Exploratory trial of a novel internet-based psychoeducational treatment for bipolar disorder. *Bipolar Disorders*, *13*(5/6), 571–577. doi:10.1111/j.1399-5618.2011.00949.x

Swartz, H. A., Frank, E., & Cheng, Y. (2012). A randomized pilot study of psychotherapy and quetiapine for the acute treatment of bipolar II depression. *Bipolar Disorders*, *14*(2), 211–216. doi:10.1111/j.1399-5618.2012.00988.x

Szentágotai, A., & David, D. (2010). The efficacy of cognitive-behavioral therapy in bipolar disorder: A quantitative meta-analysis. *Journal of Clinical Psychiatry*, *71*(1), 66–72. doi:10.4088/Jcp.08r04559yel

Todd, N. J., Jones, S. H., Hart, A., & Lobban, F. A. (2014). A web-based self-management intervention for bipolar disorder "Living with Bipolar": A feasibility randomised controlled trial. *Journal of Affective Disorders*, *169*, 21–29. doi:10.1016/j.jad.2014.07.027

Van der Voort, T. Y. G., Van Meijel, B., Goossens, P. J. J., Renes, J., Beekman, A. T. F., & Kupka, R. W. (2011). Collaborative care for patients with bipolar disorder: A randomised controlled trial. *BMC Psychiatry*, *11*, 393–400. doi:10.1186/1471-244x-11-133

Van Meter, A. R., Moreira, A. L. R., & Youngstrom, E. A. (2011). Meta-analysis of epidemiologic studies of pediatric bipolar disorder. *Journal of Clinical Psychiatry*, *72*(9), 1250–1256. doi:10.4088/Jcp.10m06290

Vos, T., Flaxman, A. D., Naghavi, M., Lozano, R., Michaud, C., Ezzati, M., … Murray, C. J. L. (2012). Years lived with disability (YLDs) for 1160 sequelae of 289 diseases and injuries 1990–2010: A systematic analysis for the Global Burden of Disease Study 2010. *Lancet*, *380*(9859), 2163–2196.

Wampold, B. E. (2001). *The great psychotherapy debate: Models, methods, and findings*. Mahwah, NJ: Lawrence Erlbaum Associates.

Weber, B., Jermann, F., Gex-Fabry, M., Nallet, A., Bondolfi, G., & Aubry, J. M. (2010). Mindfulness-based cognitive therapy for bipolar disorder: A feasibility trial. *European Psychiatry*, *25*(6), 334–337. doi:10.1016/j.eurpsy.2010.03.007

West, A. E., Jacobs, R. H., Westerholm, R., Lee, A., Carbray, J., Heidenreich, J., & Pavuluri, M. N. (2009). Child and family-focused cognitive-behavioral therapy for pediatric bipolar disorder: Pilot study of group treatment format. *Journal of the Canadian Academy of Child & Adolescent Psychiatry*, *18*(3), 239–246.

West, A. E., Weinstein, S. M., Peters, A. T., Katz, A. C., Henry, D. B., Cruz, R. A., & Pavuluri, M. N. (2014). Child- and family-focused cognitive-behavioral therapy for pediatric bipolar disorder: A randomized clinical trial. *Journal of the American Academy of Child & Adolescent Psychiatry*, *53*(11), 1168–1178. doi:10.1016/j.jaac.2014.08.013

Whiteford, H. A., Degenhardt, L., Rehm, J., Baxter, A. J., Ferrari, A. J., Erskine, H. E., … Vos, T. (2013). Global burden of disease attributable to mental and substance use disorders: Findings from the Global Burden of Disease Study 2010. *Lancet*, *382*(9904), 1575–1586. doi:10.1016/S0140-6736(13)61611-6

Williams, J. M. G., Alatiq, Y., Crane, C., Barnhofer, T., Fennell, M. J. V., Duggan, D. S., … Goodwin, G. M. (2008). Mindfulness-based cognitive therapy (MBCT) in bipolar disorder: Preliminary evaluation of immediate effects on between-episode functioning. *Journal of Affective Disorders*, *107*(1/3), 275–279. doi:10.1016/j.jad.2007.08.022

Zaretsky, A., Lancee, W., Miller, C., Harris, A., & Parikh, S. V. (2008). Is cognitive-behavioural therapy more effective than psychoeducation in bipolar disorder? *Canadian Journal of Psychiatry/Revue Canadienne de Psychiatrie*, *53*(7), 441–448. doi:10.1177/070674370805300709

4

The Treatment of Panic Disorder and Phobias

Barbara Depreeuw,[1] Leigh A. Andrews,[2] Sharon Eldar,[1] and Stefan G. Hofmann[1]
[1] *Department of Psychological and Brain Sciences, Boston University, Boston, USA*
[2] *Department of Psychological and Brain Sciences, University of Delaware, Newark, USA*

Epidemiological studies suggest that a vast number of US adults currently suffer from an anxiety disorder, with a 12-month prevalence rate of 22.2% and a lifetime prevalence rate of 41.7% (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012). Anxiety disorders are the most commonly diagnosed mental disorders and frequently occur in the presence of other disorders, including substance use and mood disorders (Grant et al., 2004). They are associated with significant distress and functional impairment in social, occupational, and health-related domains, and result in considerable economic and societal costs each year (Barrera & Norton, 2009; Lépine, 2002).

This chapter discusses the defining features and conceptualization of panic disorder, social anxiety disorder, and specific phobia from the perspective of their many proposed treatments. We utilize the nine-category scheme discussed earlier in this book (see Chapter 1), in which treatments are separated based on their efficacy and theoretical support.

4.1 Panic Disorder

4.1.1 Defining Features

Panic disorder (PD) is defined by repeated and unexpected panic attacks, which are defined as discrete periods of intense fear and discomfort that develop abruptly, peak within a few minutes, and contain at least 4 out of a total of 13 cognitive and physical symptoms (American Psychiatric Association, 2013). Panic attacks are characterized by palpitations or an accelerated heart rate, sweating, trembling, shortness of breath, feelings of choking, chest pain, nausea, dizziness or lightheadedness, temperature disruption, numbness or tingling sensations, feelings of unreality or depersonalization, fear of "losing one's mind," and/or fear of imminent death. To meet the criteria for PD, individuals must also experience persistent concern or fear related to having another panic attack, worry about the consequences or implications of their panic attacks, or make significant behavioral changes related to the attacks (e.g., avoidance of unfamiliar situations undertaken to avoid triggering future attacks) for at least 1 month.

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

Panic attacks are not specific to PD and can occur in many different situations and disorders. For example, people suffering from social anxiety disorder may experience a panic attack when confronted with a stressful situation, such as a job interview. Exposure to clowns may trigger a panic attack for someone with coulrophobia, and the noise of a helicopter could trigger a panic attack in someone with posttraumatic stress disorder (PTSD) acquired in a warzone. People with PD, however, experience panic attacks that occur unexpectedly and without any clear triggers. In addition to being either unexpected or expected, panic attacks can be classified as either situationally bound (e.g., panic attacks that only occur in response to a certain cue) or situationally predisposed (e.g., panic attacks that are more likely to occur in the presence of certain cues but lack a one-to-one relationship) (Barlow, 2002). Perhaps the best example of an unexpected panic attack is a nocturnal one, which wakes the individual up in the middle of the night (Craske et al., 2002). This is an occurrence that is clearly without any kind of obvious trigger and is extremely distressing for the individual. However, Meuret et al. (2011) have suggested that even unexpected panic attacks can be predicted through physiological monitoring up to 45 minutes before the individual is aware of the panic attack, calling into question the concept of an uncued or unexpected panic attack.

Epidemiological studies report a 12-month prevalence rate of 2.4% and a lifetime prevalence rate of 6.8% in the United States (Kessler et al., 2012). People with PD report an average age of onset of 23 years, with a strong gender bias of roughly twice as many women as men (Eaton, Kessler, Wittchen, & Magee, 1994; Kessler et al., 2012). In the absence of treatment, PD follows a chronic course and is associated with high rates of relapse even when symptoms do spontaneously remit for a period of time.

4.1.2 Treatments

4.1.2.1 Category I: Therapeutically and theoretically well supported

4.1.2.1.1 Cognitive–behavioral therapy

PD has been widely studied and cognitive–behavioral treatments for it have a solid base of empirical support. In addition to having acute efficacy, cognitive–behavioral therapy (CBT) has a long-term effect, and people maintain their treatment gains even after 2 years (McHugh, Smits, & Otto, 2009). Cognitive–behavioral models for PD combine three key components: a fear of bodily sensations, cognitive distortions or catastrophic misinterpretations, and avoidance behavior (Clark 1999; Clark & Beck, 2010; Craske & Barlow, 2006; Otto & Pollack, 2009). CBT treatment for PD focuses on combating these three key components conjointly and separately. Over the course of treatment, the relationships between maladaptive thought processes, avoidance behaviors, and anxious feelings are weakened, often with a significant reduction of PD to the point of remittance.

Fear of bodily sensations. Individuals with PD who notice hyperarousal or autonomic activation in their body (e.g., an increase in heart rate or shortness of breath) are usually alarmed or intensely fearful. These changes in bodily sensations are seen as a sign that "something is wrong with my body" that must be dangerous or harmful. For example, the palpitations and shortness of breath triggered by exercising or the hot flush and the tightness of the chest brought on by taking a hot shower are sensations that are similar in feel to the onset of a panic attack. These "symptoms" are typically interpreted as a

sign of a health-related malady such as a heart attack or stroke, terrifying the individual with PD. An intense fear of physical sensations, which often become accompanied by feelings of anxiety, fear, and panic, is typical of people with PD (McNally, 2002).

Catastrophic misinterpretations and cognitive bias. People with PD are hypervigilant to the detection of the interoceptive cues mentioned above and their attention is biased toward these changes, although they do not have more actual physiological responses than those without PD (Beck, Stanley, Averill, Baldwin, & Deagle, 1992; Clark & Beck, 2010; Ehlers & Breuer, 1992). The fear they feel is accompanied by thoughts such as "I am losing control, I am choking, I am dying"—catastrophic misinterpretations that elicit more fear, which can trigger more intense bodily sensations. These cognitive biases reinforce the perceived salience of "threat-relevant" cues and are therefore, according to the cognitive–behavioral model, an important factor in this vicious cycle of high anxiety and panic (Clark 1999; Otto & Pollack, 2009). Thus, the development and maintenance of PD are influenced by several cognitive factors (automatic associations in memory, interference effects, and interpretational biases) that all uniquely predict panic symptoms.

Avoidance. Avoidance is an important component in the CBT conceptualization of PD. Having a first (unexpected) panic attack can be a highly aversive experience; subsequently, people with PD fear experiencing fear and panic attacks. Avoidance behavior is then corollary. There are four kinds of avoidance behavior for PD: interoceptive avoidance, agoraphobic avoidance, distraction, and safety behaviors (Barlow, 2002). *Interoceptive avoidance* is related to the fear of bodily sensations. It is characterized as the avoidance of any activities that elicit sensations similar to a panic attack. Commonly avoided activities are drinking caffeine, running, and having sex. *Agoraphobic avoidance* is a consequence of the fear of having a panic attack or related symptoms in a setting from which it would be difficult or embarrassing to escape, such as public spaces, public transportation, bridges, elevators, theaters, grocery stores, restaurants, and crowds. *Distraction* as an avoidance behavior can sometimes be difficult to identify, because it is a very subtle method of avoidance. It is, however, very important to eliminate it, because it can undermine treatment if untargeted. Examples of distraction include turning on the radio, forcing oneself to think of something else, trying to read, pinching oneself, or engaging in a conversation instead of experiencing anxiety. *Safety behaviors* are the last type of PD avoidance. Safety behaviors serve an important function for people with PD, as engaging in them allows people to feel more secure if a panic attack occurs and also helps them to try to prevent the onset of a panic attack (Salkovskis, Clark, Hackmann, Wells, & Gelder, 1999). This behavior is often very reinforcing, because as people get more comfortable moving around they become less limited, but it is also deeply impairing, because people become totally dependent on these "safe" cues. Safety behaviors are key to the persistence of anxiety disorders. They provide supportive evidence for inaccurate catastrophic beliefs about what happens in the event of panic (Salkovskis, Clark, & Gelder, 1996). They also prevent people from learning corrective information about what would happen in the presence of unfiltered anxiety (i.e., if they did not engage in the safety behaviors). Common safety behaviors are carrying medication or water bottles, bringing along a spouse or another "safe" person, and having a cell phone on one's

person. Safety behaviors can become day-to-day routines for a person with PD to the extent that it is often difficult for them to be identified without the help of a therapist.

CBT for PD is a learning-based approach (McHugh et al., 2009) that aims to help people to relearn a feeling of safety despite anxiety or panic symptoms. This implies a combination of psychoeducation regarding the nature of anxiety and the CBT conceptualization, self-monitoring of anxiety symptoms and their cues, cognitive restructuring of misinterpretations, and the key component of effective treatment: exposure to the feared stimuli and decent relapse prevention.

Repeated and systematic exposure to feared stimuli is the most important aspect of the treatment for anxiety disorders (Barlow, 2002). It is based on the well-established theory of fear extinction learning (Vervliet, Craske, & Hermans, 2013). The fear of anxiety symptoms and panic attacks can be addressed with interoceptive exposure to physical sensations, although exposure in vivo is often needed as well. This is the repeated and systematic real-life exposure to agoraphobic or panic-producing situations. During these exposures, it is very important that people do not engage in safety behaviors (e.g., carrying medication, a bottle of water, etc.), emotional avoidance strategies (e.g., "I don't want to feel this"), or distraction (e.g., thinking of something else, humming). As patients are exposed to fearful situations, the therapist encourages them to allow their feelings of anxiety to occur and not to try to avoid them.

4.1.2.2 Category II: Therapeutically well supported and theoretically equivocal

We found no evidence in the literature of treatments for PD that were therapeutically well supported but theoretically equivocal.

4.1.2.3 Category III: Therapeutically equivocal and theoretically well supported

4.1.2.3.1 Physical exercise

People who exercise regularly have fewer anxiety symptoms and are less likely to meet diagnostic criteria for PD and agoraphobia, in comparison to people who do not exercise regularly (DeBoer, Powers, Utschig, Otto, & Smits, 2012). Physical exercise for PD has a strong theoretical base but currently lacks sufficient evidence of effectiveness to be considered a first-line treatment for PD (Otto & Smits, 2011). Multiple neurotransmitter, neuromodulator, and psychological mechanisms of action have received support for physical exercise as an anxiolytic (DeBoer et al., 2012). Physical exercise seems to operate on both the psychological and physiological levels. Exercising is a stressor for the body and exposing the body to exercise, which also improves fitness, results in a system that is more resilient toward other stressors. Intensive training also decreases blood pressure and heart rate, and reduces physiological arousal and psychological reactions to various stressors (Throne, Bartholomew, Craig, & Farrar, 2000). Another view of exercise as a treatment is based on its release of neurotransmitters, such as serotonin and endorphins. Serotonin, which plays a role in causing and maintaining depressive and anxious disorders, is the primary target of selective serotonin reuptake inhibitors, which increase the persistence of serotonin in the synapse through blocking reuptake. Exercising increases serotonin levels by roughly the same amount, so the protective function of physical exercise could also be explained as a natural antidepressant (Dey, Singh, &

Dey, 1992; Wilson & Marsden, 1996). High-intensity exercise, in particular, is an effective way to reduce fear of physical anxiety symptoms, one of the prime components of PD (Otto & Smits, 2011). People with PD try to avoid changes in bodily sensations due to their fear of triggering a panic attack, so as symptoms worsen they begin to exercise less and less because they are afraid that any increase in heart rate will trigger a catastrophe. When people begin physical exercise again, this forces them to return to a more active behavioral pattern and gives them a chance to learn that these situations are safer and more rewarding than expected. As in traditional CBT exposure, fear extinction learning can occur, which is the scientific and empirically well-supported model behind the well-known efficacy of exposure treatment (Vervliet et al., 2013). Physical exercise can thus allow people to break the vicious cycle of high anxiety and high avoidance behavior.

There is strong evidence that physical exercise is useful for PD (for a review see DeBoer et al., 2012). A randomized clinical trial (RCT) comparing aerobic exercise, placebos, and medication in people with PD found that exercise was significantly more effective than the placebo condition but that it was less effective than treatment with medication (Broocks et al., 1998). Another study found that exercise or exercise in conjunction with cognitive restructuring both substantially decreased anxiety sensitivity (McNally, 2002). Recently, however, an RCT compared CBT with physical exercise for PD and showed that, although both conditions reduced panic symptoms, there were better results for CBT after treatment and at the 12-month follow-up (Hovland et al., 2013).

4.1.2.3.2 Mindfulness

Mindfulness refers to any process that leads to a centered mental state primarily characterized by a nonjudgmental awareness of the present (including sensations, thoughts, bodily states, consciousness, and one's environment) and encouragement of openness, curiosity, and acceptance (Allen, Blashki, & Gullone, 2006; Bishop et al., 2004; Kabat-Zinn, 2003). The two main components of mindfulness can more simply be separated into self-regulation of attention and an open and accepting orientation toward the present. The theoretical concept behind mindfulness is that these attitudes allow people to counter the negative effects of stressors and avoid excessive worrying about the future or ruminating on the past. It is also suggested that this reflective, rather than reflexive, stance allows people to stop their avoidance behaviors more easily and begin to experience unwanted internal states in an unaltered manner. A number of meta-analyses have shown them to be effective in treating depression and anxiety disorders (Hofmann, Sawyer, Witt, & Oh, 2010; Khoury et al., 2013). Thus, for PD, mindfulness would encourage someone to experience a panic attack without attempting to control it or worrying that it might turn out to be a heart attack. As with physical exercise, however, there is less evidence relevant to PD specifically. Some studies have suggested that group mindfulness training can help to maintain reductions of symptoms in patients with PD and PD with agoraphobia and provide improvements for up to 3 years (Kabat-Zinn et al., 1992; Miller, Fletcher, & Kabat-Zinn, 1995). Mindfulness has also been suggested as an aid to pharmacotherapy in treating PD, as it can allow participants to capitalize on the medically driven reduction in symptoms more than an active control (Kim et al., 2010). Though mindfulness' relevance to treating anxiety disorders is quite clear, until more

RCTs specifically focusing on its role in PD are conducted it will be impossible to determine its efficacy as a PD treatment, rather than a treatment useful for reducing anxious symptoms throughout the anxiety disorders more generally.

4.1.2.3.3 Respiratory training with biofeedback

A third technique for treating PD with a strong theoretical backing and promising therapeutic results is respiratory training with biofeedback. Hypocapnia, or lower than normal levels of carbon dioxide, has been noted as a common biomarker for PD (Hegel & Ferguson, 1997; Papp, Klein, & Gorman, 1993). Hypocapnia can produce a range of unpleasant symptoms common to PD, such as lightheadedness, feelings of suffocation, and severe distress. The rebreathing training is thus designed to help patients learn to readjust their own carbon dioxide levels. Most early rebreathing made no use of actual physiological measures, simply proposing that improved breathing would solve any hypocapnic issues. More recently, Meuret, Rosenfield, Seidel, Bhaskara, and Hofmann (2010) introduced capnometry-assisted respiratory training for PD, which relies on actual measures of PCO_2 (partial pressure of carbon dioxide) and biofeedback. People first learn to control their breathing through training with the portable monitor measuring their carbon dioxide. Later, through practice and a better understanding of their own physiological state, they can manage their breathing without assistance. This is hypothesized to reduce PD symptoms by (1) reducing the frequency and intensity of the physical distress to which those with PD are so sensitive and (2) giving people a greater sense of control over themselves, which can lead to reduced fear of physical sensations. Although this is somewhat contradictory to newer CBT approaches, in which accepting a lack of control is important (Craske et al., 2014), the two mechanisms have also shown great efficacy in concert, suggesting that both pathways are appropriate means through which to treat PD.

There is strong evidence that respiratory training is useful for PD. Meuret and colleagues have conducted two RCTs comparing capnometric assisted respiratory training to a waitlist control and CBT respectively in people with PD; they found that the training was significantly more effective than the placebo condition, and comparable to CBT (Meuret et al., 2010; Meuret, Wilhelm, Ritz, & Roth, 2008). As both of these RCTs have come out of the same lab, however, respiratory training cannot yet be described as therapeutically broadly supported.

4.1.2.3.4 Applied relaxation

The last technique we describe in Category III for PD is applied relaxation. Probably the most common technique is progressive muscle relaxation (Jacobson, 1938). People learn to tense and relax their muscles at set intervals and to gain more familiarity and control in the process of relaxation. In the 1970s, it was proposed as an addition to behavioral treatments (Öst, 1986). Over the course of progressive muscle relaxation training, people learn first how to rapidly relax their muscles after tensing them, then to relax without tensing, then to associate muscular release with the feeling of relaxing, then how to walk and talk and move around while relaxing muscles, and finally to do so in phobic situations. Theoretically, applied relaxation rests on improving the feeling of control that people with PD lack, as their new applied relaxation techniques allow them to respond differently to panic attacks. It also teaches them to be more

in touch with their own physiological sensations and less afraid of panic attacks and arousal (Öst, 1986).

There is more mixed evidence that applied relaxation is useful for PD. A number of RCTs have been run comparing applied relaxation and cognitive therapy, waitlist, or active control that initially found that applied relaxation was as effective or significantly more effective than controls or cognitive therapy in reducing symptoms of PD posttreatment and at 3- and 6-month follow-up (Öst, 1988; Öst & Westling, 1995; Öst, Westling, & Hellström, 1993). When Clark et al. (1994) compared applied relaxation to imipramine and CBT, they found that CBT was superior to applied relaxation at 3 months, 6 months, and 15 months, while imipramine was superior at 3 months and 6 months, although many imipramine patients relapsed between 6 and 15 months after treatment. Additionally, a slightly more recent study, also from outside the Öst lab, found that CBT was superior to applied relaxation in reducing panic frequency and symptom severity (Arntz & Van den Hout, 1996). Trying to relax during panic sensations is often very difficult and can be used as an avoidance behavior if people do not want to feel fear symptoms. This is contradictory to newer CBT approaches, in which accepting a lack of control is more important. Although applied relaxation does have some therapeutic and theoretical support in treating PD, it is not quite a gold-standard treatment. More research could be valuably targeted at determining whether or not it would be a useful additive to traditional CBT.

4.1.2.4 Category IV: Therapeutically and theoretically equivocal

4.1.2.4.1 Panic-focused psychodynamic psychotherapy

Panic-focused psychodynamic psychotherapy (PFPP) for PD has been examined in a few RCTs. Its theoretical base is equivocal, however, and its therapeutic effect controversial.

The theoretical psychodynamic formulation suggests that certain individuals have, from birth, a fearful temperament and a predisposition to anxiety. This leads to a tendency to develop a fearful dependency on others, which causes them to feel narcissistic humiliation, as their parents are presumed to have had a maladaptive parenting style (Shear, Cooper, Knijnik, Busch, & Shapiro, 1993). In response to perceived rejection or simple unavailability, the child becomes angry at their key attachment figures, a response that is amplified by the innate narcissistic injury of dependence. This anger is experienced as a dangerous sensation triggering a further need for security, and a vicious cycle of fearful dependency and anger can occur. The vicious cycle continues in adulthood whenever the individual experiences or perceives a threat to the integrity of important attachment relationships (Busch & Milrod, 2009).

Based on this theoretical formulation, Busch, Milrod, and Singer (1999) developed the PFPP, which is a specific manualized form of time-limited psychoanalytic psychotherapy, delivered twice a week for a duration of 12 weeks (24 sessions in total). The PFPP manual divides treatment into three phases, including understanding the stressors surrounding the panic attacks; understanding the patient's feelings and fantasies about relationships at present, in the past, and in the transference relationship; and working on expressing anger in a less overwhelming way.

PFPP has been studied in three RCTs, all of them conducted with the same treatment manual and with therapists who were trained by the same research team. It was shown that PFPP reduced panic severity more significantly than applied relaxation in PD

patients with comorbid personality disorders (Milrod, Leon, Barber, Markowitz, & Graf, 2007), and when compared to CBT both treatments showed the same effectiveness at 6-month follow-up (Beutel et al., 2013). In the most recent two-site RCT (Milrod et al., 2015), PFPP was compared to CBT and applied relaxation and the findings were controversial. PFPP had the same effect as the other two treatments at one site but not the other, making the findings difficult to interpret.

4.1.2.4.2 Client-centered therapy

Client-centered therapy (CCT) is a nondirective talk therapy that aims to encourage the personal growth of the patient (Rogers, 1951). According to this approach, the therapist's attitude is based on three principles: empathy, genuineness, and an unconditional positive regard toward the patient. This attitude encourages patients' full disclosure and is predicated on the belief that individuals need only the guiding hand of the therapist in order to find answers that were always within themselves. CCT with PD patients has been studied only in one RCT, which compared CCT alone to CCT with behavioral exposure and behavioral exposure (Teusch & Böhme, 1999). Behavioral exposure, either alone or combined with CCT, significantly supported patients' determination to expose themselves to phobic situations more than did CCT alone, and they showed greater symptom reduction as well. But there were no differences in improvement at one-year follow-up. Hence, CCT has the potential to stand in as a treatment for panic, but more research is needed.

4.1.2.5 Category V: Therapeutically well supported and theoretically contradictory

No treatments were found for this category.

4.1.2.6 Category VI: Therapeutically contradictory and theoretically well supported

No treatments were found for this category.

4.1.2.7 Category VII: Therapeutically equivocal and theoretically contradictory

No treatments were found for this category.

4.1.2.8 Category VIII: Therapeutically contradictory and theoretically equivocal

4.1.2.8.1 Eye movement desensitization and reprocessing

Eye movement desensitization and reprocessing, a successful treatment for PTSD, consists of having individuals recall anxiety-producing memories or traumas while undergoing specific eye movements or rhythmic sensations (Cusack et al., 2016). For panic, it has not been shown to be a successful treatment (for more detail see Goldstein, de Beurs, Chambless, & Wilson, 2000).

4.1.2.8.2 Emotion-focused therapy

Emotion-focused therapy is a type of talk therapy where emotions play a central role (Greenberg, 2004). Only one RCT has examined its role in PD and it was no more effective than a placebo (Shear, Houck, Greeno, & Masters, 2001).

4.1.2.9 Category IX: Therapeutically and theoretically contradictory

4.1.2.9.1 Hypnosis

Although hypnosis is popular as an alternative therapy, as a therapy treatment for PD it is not recommended (Kessler et al., 2001; Van Dyck & Spinhoven, 1997).

4.2 Social Anxiety Disorder

4.2.1 Defining Features

Social phobia (also known as social anxiety disorder [SAD]) is a chronic disorder characterized by excessive anxiety and fear of the negative social evaluations of others in one or more social or performance situations (American Psychiatric Association, 2013). Individuals with SAD often understand that these fears are unreasonable and excessive. They typically avoid a range of social situations due to their fear of negative, and infrequently positive, social evaluation (Weeks, Heimberg, Rodebaugh, & Norton, 2008). SAD is also often characterized by increased awareness of physiological symptoms (blushing, sweating, etc.) of anxiety as well as fear of the social consequences of this arousal (Anderson & Hope, 2009). If these situations cannot be avoided, patients with SAD endure a great deal of anxiety and distress. Some classical examples of social situations individuals with SAD fear and avoid include initiating and maintaining conversations, interacting with authority figures, dating, and performance situations that require talking, writing, or eating in front of others. When individuals fear most or all social situations, they are diagnosed with the generalized type of SAD. A common subtype is often referred to as "performance-only" subtype and is limited to only one particular type of social situation (typically public speaking or other forms of performance) (Kessler, Stein, & Berglund, 1998). Most performance-only SAD involves substantial impairment in the individual's professional life. The generalized subtype of SAD has been shown to be associated with greater symptom severity and impairment (Hofmann, Heinrichs, & Moscovitch, 2004).

Epidemiological studies from the United States report a lifetime prevalence rate of 12.1–13% and a 12-month prevalence of 7.1–7.4%, making SAD the second most commonly diagnosed anxiety disorder following specific phobia (Kessler, Berglund et al., 2005; Kessler, Chiu, Demler, Merikangas, & Walters, 2005; Kessler et al., 2012). The average age of onset for SAD is the earliest (15–17 years) of any of the anxiety disorders in the National Comorbidity Survey, other than specific phobia. Those afflicted with SAD frequently describe experiencing extreme shyness, anxiety-regarding negative evaluations, school refusal, or some form of separation anxiety as children. In contrast to the many other anxiety disorders that are more prevalent in women, the prevalence rates for SAD are roughly equivalent across the sexes. In the absence of any intervention or treatment, SAD follows a chronic and unremitting course and leaves the individual substantially impaired across both interpersonal and occupational domains, which can often result in increased time taken off work or school as well as decreased productivity levels (Katzelnick et al., 2001; Stein & Kean, 2000; Stein, McQuaid, Laffaye, & McCahill, 1999). SAD is additionally associated with high rates of psychiatric comorbidity, with estimates ranging from 56.9% in community samples (Magee, Eaton, Wittchen, McGonagle, & Kessler, 1996) to 83% in clinical samples (Goisman, Goldenberg, Vasile, & Keller, 1995). SAD is an undertreated disorder, despite the severe functional impairment

it produces. Generally, the more social fears one has, the more severe the manifestation of social phobia, although this is not always the case (Ruscio et al., 2008). It is extremely important for practitioners to learn to recognize SAD and help people seek treatment for this debilitating disorder.

4.2.2 Treatments

4.2.2.1 Category I: Therapeutically and theoretically well supported

4.2.2.1.1 Cognitive–behavioral therapy

The most effective psychotherapy for SAD is, as for PD, CBT. Over the years there has been a lot of research conducted to determine its efficacy (Hofmann & Smits, 2008). The underlying theory and the therapeutic package itself are well supported (Hofmann & Otto, 2009). As in PD, the CBT approach assumes that maladaptive cognitions contribute to the maintenance of the disorder and that correcting the maladaptive thoughts associated with these emotional issues can lessen symptoms, which is why the conceptualization is discussed below.

CBT models. There are several well-supported CBT models of the onset and the maintenance of SAD. These models all agree that people with SAD have apprehensions about the self in interactions with any audience, which leads to heightened self-directed attention and subsequent avoidance behaviors as a result of perceived negative evaluations about the self. The maintenance of SAD is manifested in a feedback loop: perceived negative evaluations about performance or audience response drive the patient with social anxiety to avoid social situations, which can feed the idea that one is socially inept or inexperienced and confirms the social anxiety (Clark & Wells, 1995; Hofmann, 2008; Rapee & Heimberg, 1997).

Avoidance behavior. Individuals with SAD engage in a variety of avoidance or safety behaviors, such as avoiding big crowds, making eye contact, or entering evaluative social or performance situations. Some of these avoidance behaviors are overt and obvious (e.g., refusing to do a public speech, canceling a job interview, leaving a party extremely early); others are subtle (e.g., avoiding attending a party alone, remaining silent, trying to control the sound of the voice or maintaining tight control over the body, being overly apologetic) and hard to detect (e.g., only attending parties at which alcohol is served, being aloof or cold in interpersonal interactions). Often avoidance behavior becomes such a habit that people stop noticing it at all. As in other anxiety disorders, avoidance behavior is key to the maintenance of SAD. Avoidance behaviors are very rewarding in the short term, as they temporarily reduce anxiety or prevent anxiety from increasing. In the long term, of course, these behaviors increase anxiety. They reinforce social anxiety by teaching the body that the threat was real and they prevent people from testing out the feared outcome and dealing with the real consequences of engaging in feared situations (Clark & Wells, 1995; Hofmann, 2008; Rapee & Heimberg, 1997).

Selective attention toward the self, cognitive biases, and information-processing. Research on cognitive and information-processing biases in people with SAD suggests that

their attention is preferentially directed to social-threat-relevant information and environmental cues that indicate negative evaluation. There is both hypervigilance and a selective attention bias toward perceived social failures and toward aspects of the self that are not necessarily useful for performing a task, such as one's arousal (blushing or sweating), emotions (feeling the anxiety), private self (performance itself), or public self (perception of others) (Mulkens, De Jong, & Bögels, 1997). Individuals with SAD also tend to be more self-critical and judgmental than they are of other people, and they judge themselves more harshly than others do (Heinrichs, Hoffman, & Hofmann, 2001; Hofmann, 2007; Rapee & Heimberg, 1997; Williams, Watts, MacLeod, & Mathews, 1997). This selective attention makes the social anxiety worse. Increased social anxiety and its accompanying avoidance behavior can impair a performance. Whether or not the performance was objectively good or bad, people with SAD tend to ruminate about their performance afterwards. This postevent processing can cause experiences that were initially positive or pleasant to become viewed as negative and unpleasant over time (Hofmann, 2007; Hofmann & Otto, 2008).

CBT treatment. CBT for SAD consists of psychoeducation, cognitive restructuring, attention modification, and exposure exercises. Individuals with SAD often believe that social anxiety or intense shyness is a fundamental part of them and cannot be changed through treatment. Psychoeducation prepares patients to actively engage in the treatment process by addressing these misconceptions and explaining the rationale behind exposure exercises before an individual gets involved in exposure itself. People with SAD often overestimate the likelihood of negative outcomes in a social situation and the costs of these negative outcomes. Cognitive restructuring identifies and then challenges maladaptive thoughts about the probability and the cost of negative evaluation, negative attribution bias, and poor self-concept (Hofmann & Otto, 2008). Exposing patients to feared social or performance situations is also important in CBT. During these exposure exercises, patients are prevented from engaging in avoidance behaviors and must rely on acceptance in order to cope with their feelings of anxiety. In vivo exposures, or exposures that involve patients directly confronting the feared situation, can allow patients to test assumptions about the likelihood and cost of a negative outcome in a certain social situation. Video feedback can provide information about the patient's poor self-concept and their (often false) beliefs about their own performance (Warnock-Parkes et al., 2016). Often, patients learn that their feared outcome is highly unlikely and that even when it occurs it will not result in catastrophic consequences.

Task concentration training. Task concentration training (TCT) can provide a valuable addition to exposure exercises (Bögels, 2006). TCT is a therapy technique that is based on cognitive models of SAD, as already stated, where self-focused attention is a key component in the maintenance of SAD (Clark & Wells, 1995; Daly, Vangelisti, & Lawrence, 1989; Hope, Gansler, & Heimberg, 1989; Wells & Matthews, 1994). TCT is meant to redirect people's attention outward, to the (social) task at hand or neutral aspects of the environment (e.g., furnishings), instead of toward their bodily symptoms or their debilitating and maladaptive thoughts. This can reduce awareness of bodily symptoms and provide corrective information when people are doing exposure exercises. Patients can observe how their level of anxiety changes in response to modifying the focus of their attention, and learn how it is possible to stay present in any social situation, despite

anxiety. The supplemental effect of TCT is particularly strong for erythrophobes (those with a fear of blushing) and has been strongly supported in a number of trials.

4.2.2.2 Category II: Therapeutically well supported and theoretically equivocal

4.2.2.2.1 Social skills training

Social skills training (SST) was a very popular topic of research in the 1970s and 1980s but has received far less attention in the past few decades despite its efficacy as a stand-alone treatment (Mersch, Emmelkamp, Bögels, & Van der Sleen, 1989; Stravynski, Marks, & Yule, 1982; Wlazlo, Schroeder–Hartwig, Hand, Kaiser, & Münchau, 1990). The basic theory behind SST is that many sufferers of SAD are less socially skilled than the typical adult. This fundamental assumption underpinning SST is quite controversial as many studies have either supported or contradicted this hypothesis (Bögels & Voncken, 2008; Herbert et al., 2005). Regardless of whether those with social phobia are less socially skilled, however, several studies have shown that they are far less able to employ their social skills in anxiety-provoking situations and are often judged by independent raters to be less likeable (Alden & Bieling, 1998). In SST the focus is on acquiring (or better employing) adequate social skills, such as keeping eye contact; starting, maintaining, and ending conversations; and giving and receiving a compliment. Not doing such things is a common avoidance behavior for people with SAD. SST can also extend to more complex skills, such as asserting oneself, refusing requests, or expressing annoyance. People are trained through psychoeducation, modeling, post hoc analyses of situations and role-playing, and feedback sessions. The goal is to learn how to cope better and respond differently to anxiety, and often trainees receive homework, in order to practice at home and in real situations. Criticism of the theory behind SST claims that it is hard to determine whether the decrease in anxiety after SST is because of the huge amount of exposure that proper adherence generates or due to an increase in efficacy in social situations (Herbert et al., 2005).

More recently, Herbert and colleagues (2005) combined SST with cognitive–behavioral group therapy and found the combination to be more effective than CBT alone. A meta-analysis of SAD treatment efficacy (reviewed by Rodebaugh, Holaway, & Heimberg, 2004) has also shown that SST is as effective as other cognitive–behavioral treatments, such as exposure, cognitive restructuring, or a combination of the two, leaving SST's status as a stand-alone treatment and its mechanism still in doubt (Turner, Beidel, & Cooley-Quille, 1995; Turner, Beidel, Cooley, Woody, & Messer, 1994; Van Dam-Baggen & Kraaimaat, 2000). According to a more recent meta-analysis done by Acarturk, Cuijpers, Van Straten, and de Graaf (2009), it is impossible to draw definite conclusions about the additional effect of each of these techniques, because not enough studies have examined these techniques separately.

4.2.2.3 Category III: Therapeutically equivocal and theoretically well supported

4.2.2.3.1 Attention bias modification

Attention bias modification (ABM) for SAD is a computer-based treatment with a strong theoretical base and controversial efficacy results. ABM was derived from the cognitive theories of psychopathology that emphasize attentional bias for threat as implicated in the maintenance and/or etiology of anxiety (Heeren, Mogoaşe, Philippot, & McNally, 2015). The clinical purpose of ABM is to reduce attention bias toward these negative stimuli via attention training in order to reduce anxiety severity and symptoms

(MacLeod & Mathews, 2012). The most common ABM procedure is a modification of the visual dot-probe task (MacLeod, Rutherford, Campbell, Ebsworthy, & Holker, 2002).

In the classical dot-probe task (MacLeod, Mathews, & Tata, 1986), two stimuli, one threat-related and one benign (positive or neutral), are shown in each trial and their offset is then followed by a target probe, which appears with equal probabilities at either location. Participants have to respond as fast as possible to the probe. Response latencies provide a "snapshot" of the distribution of participants' attention, with faster responses typical for probes presented at the attended, relative to the unattended, location. Attention bias toward threat is inferred when participants respond faster to probes replacing threat-related rather than benign stimuli. Threat-related attentional bias on the dot-probe task has been found in anxious individuals but not in nonanxious subjects (for a review see Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & Van Izendoorn, 2007).

Attention biases toward threat can be retrained toward nonthreatening cues. Hence, attention bias change is expected to have a tight association with reduction in anxiety symptoms. Success in changing attention bias is expected to have a moderating role on treatment effects (Linetzky, Pergamin-Hight, Pine, & Bar-Haim, 2015). Actually treating SAD with ABM, however, has been more ambiguous. While several studies did find that, relative to a control condition, ABM reduced symptoms in people with SAD (e.g., Amir, Weber, Beard, Bomyea, & Taylor, 2008; Amir et al., 2009; Heeren, Reese, McNally, & Philippot, 2012; Li, Tan, Qian, & Liu, 2008; Schmidt, Richey, Buckner, & Timpano, 2009), other studies did not (e.g., Boettcher, Berger, & Renneberg, 2012; Boettcher et al., 2013; Carlbring et al., 2012; Heeren, Lievens, & Philippot, 2011; Julian, Beard, Schmidt, Powers, & Smits, 2012; McNally, Enock, Tsai, & Tousian, 2013). In a recent meta-analysis (Heeren et al., 2015), ABM was found to have a significant but small effect on self-reported social anxiety symptoms and on speech challenges at posttest. The mechanism of modifying attention bias, however, had only a very small effect on these variables. Future investigations should seek to elucidate the exact mechanisms by which ABM may affect levels of social anxiety in patients with SAD.

4.2.2.4 Category IV: Therapeutically and theoretically equivocal

4.2.2.4.1 Interpersonal therapy

In interpersonal therapy (IPT), symptoms of social phobia are viewed as part of a more general interpersonal impairment. SAD patients use self-protective behaviors, such as withdrawal, that help them reduce their anxiety. However, these behaviors elicit negative reactions from others and consolidate the social role insecurity and negative self-evaluation (Alden & Taylor, 2004). In turn, hypervigilant self-observation avoidance of social activities strengthens role insecurity and sense of defectiveness, and thus serves to maintain the disorder (Lipsitz, Markowitz, Cherry, & Fyer, 1999). IPT seeks to ameliorate symptoms by specifically targeting interpersonal problems. IPT is a time-limited psychotherapy that focuses not on cognitions or behavioral tasks but on feelings in interpersonal situations (Klerman & Weissman, 1993; Weissman, Markowitz, & Klerman, 2000). IPT sessions avoid psychodynamic interpretations of fears but do have less structured, more informal, more affect-focused, and more emotionally charged sessions than CBT. Both CBT and IPT do seek affectively charged cognitions, however, and believe that sessions should engage emotions and challenge their patients. The model has been empirically supported in depression research, but there are equivocal results in research

on SAD. CBT has generally had better outcome effects than IPT in RCTs (Borge et al., 2008; Lipsitz et al., 2008).

4.2.2.4.2 Mindfulness-based stress reduction

Mindfulness-based stress reduction (MBSR) involves a nonjudgmental, nonreactive, moment-to-moment awareness of mental states and experiences. According to Koszycki, Benger, Shlik, and Bradwejn (2007), there are numerous reasons why MBSR may be beneficial for SAD. First, it can help patients shift their focus from self-critical cognitions toward the external social situation. Second, mindfulness practices can help reduce the uncomfortable physiological symptoms (e.g., blushing, trembling, sweating) that are such an issue for socially anxious individuals. Finally, MBSR can be delivered outside psychiatric settings, as it can be self-taught or taught by health care professionals and educators from a broad range of disciplines. This can attract patients who would otherwise be embarrassed to receive conventional mental health care.

Although MBSR has the potential to stand as a clinical intervention for SAD, there have been only four studies that have examined its effect and just two RCTs that compared MBSR to group CBT. Generally, greater reductions in anxiety levels were found with the CBT group, but significant improvements were found from pre- to posttreatment with the mindfulness interventions in both studies (Faucher, Koszycki, Bradwejn, Merali, & Bielajew, 2016). Thus, studies have revealed some preliminary evidence that mindfulness training can reduce social anxiety, but its efficacy is unclear and more research is needed (Goldin, Ramel, & Gross, 2009).

4.2.2.4.3 Psychodynamic treatment

There have also been a few psychodynamic therapies proposed for SAD, though unfortunately these do not rely on a well-researched model or theory. The two main research groups that have studied psychodynamic therapy with SAD—led by Knijnik (Knijnik, Blanco et al., 2008; Knijnik, Hauck, Mombach, de Almeida, & Eizirik, 2008; Knijnik, Kapczinski, Chachamovich, Margis, & Eizirik, 2004) and Leichsenring (Leichsenring et al., 2013)—each created manuals that were based on other, more general, psychodynamic treatments. Broadly, the assumption underlining these treatments is that avoidance of social situations contributes to avoidance of the conscious experience of having sexual or aggressive wishes. In addition, the clinical work with SAD patients reveals internalized representations of relationships with parents, caretakers, or siblings, who shame, criticize, ridicule, humiliate, abandon, and embarrass them. These perceptions are established early in life and later repeatedly projected onto random persons in their environment, who are then avoided for fear of criticism and rejection (Knijnik, Blanco et al., 2008).

Knijnik and colleagues (Knijnik et al., 2004; Knijnik, Blanco et al., 2008) published two RCTs that studied the effect of a psychodynamic group treatment (PGT) on SAD. These two studies did not use exactly the same protocol, but both included 12 sessions for each treatment group, in which interpretation of possible symptom–conflict relationships was discussed. In the first study, the researchers found that PGT reduced social anxiety more than a control group. In their second RCT, they compared PGT plus clonazepam (psychopharmacological treatment) with clonazepam alone. Results revealed that PGT plus clonazepam was more effective than clonazepam alone. Leichsenring et al. (2013) also developed a manual-guided form of psychodynamic therapy

that was specifically adapted to treat SAD. This model encompasses interventions that were designed to relate the symptoms of SAD to the patient's underlying conflict-based relationship theme in order to reduce the patient's symptoms. This treatment effectiveness was compared to CBT in a RCT using 25 individual sessions in each treatment (Leichsenring et al., 2013). Both CBT and psychodynamic therapy proved to be superior to a waitlist control with regard to remission and response.

4.2.2.4.4 Imagery rescripting

Imagery rescripting (ImRs) was originally developed to treat people with traumatic (childhood) memories (Arntz & Weerman, 1999) and demonstrated strong results for PTSD (Arntz, 2012). ImRs is now successfully applied to a variety of disorders, in particular SAD (for an overview and meta-analysis see Morina, Lancee, & Arntz, 2017). ImRs is often combined with other treatments (mainly CBT), but there is evidence that it can be used as a stand-alone treatment for SAD (Lee & Kwon, 2013; Reimer & Moscovitch, 2015). In one RCT, ImRs was compared to cognitive restructuring (but not exposure) and shown to be as effective in reducing trait social anxiety (Norton & Abbot, 2016).

Emotional memories typically present in the form of an image. Moreover, in SAD, negative imagery of the self, built upon unpleasant past autobiographical memories of social judgment or embarrassment, are central to the maintenance cycle of SAD (Hirsch, Meynen, & Clark, 2004). Imagination provides access to the emotions that are associated with these images. Additionally, ImRs aims to change the meaning of the emotional memory, by asking the patient to imagine the situation with the (often disturbing) images linked to the memory. Next, the patient is instructed to describe and imagine a desired outcome of the situation as vividly as possible. The patient is encouraged to imagine a change in narrative of the emotional memory, resulting in a fundamental shift in cognitive processing similar to that which follows CBT (Wheatley & Hackmann, 2011).

ImRs is a relatively new technique and still lacks a clear theoretical framework. There are two memory-focused hypotheses about the underlying mechanisms. Brewin and colleagues assume that rescripting an emotional memory creates an alternative memory trace that can compete with the original memory (Brewin, 2006; Stopa & Jenkins, 2007). On the other hand, Arntz and colleagues hypothesize that ImRs changes the original memory and can be viewed as a form of reconsolidation of the emotional memory (Arntz, 2012; Arntz, Sofi, & Van Breukelen, 2013; Arntz & Weertman, 1999; Dibbets, Poort, & Arntz, 2011). In conclusion, ImRs has the potential to be a powerful and auspicious therapy technique for SAD as a stand-alone treatment or in combination with CBT. Further clinical research is needed to further establish its therapeutic effects, and more fundamental research is needed to shed light on the theoretical framework and underlying mechanisms.

4.2.2.5 Category V: Therapeutically well supported and theoretically contradictory

No treatments were found for this category.

4.2.2.6 Category VI: Therapeutically contradictory and theoretically well supported

No treatments were found for this category.

4.2.2.7 Category VII: Therapeutically equivocal and theoretically contradictory

No treatments were found for this category.

4.2.2.8 Category VIII: Therapeutically contradictory and theoretically equivocal

No treatments were found for this category.

4.2.2.9 Category IX: Therapeutically and theoretically contradictory

No treatments were found for this category.

4.3 Specific Phobia

4.3.1 Defining Features

Specific phobia requires an individual to present an intense and persistent fear in the presence or anticipation of a specific situation or object (e.g., dogs, airplanes, injections, spiders) (American Psychiatric Association, 2013). When people with specific phobia encounter their phobia, they experience an immediate fear response disproportionate to the stimulus. This frequently manifests as either situationally predisposed panic attacks (i.e., panic attacks that are more likely to occur in the presence of certain cues but lack a one-to-one relationship) or situationally bound panic attacks (i.e., panic attacks that only occur in response to a certain cue). The anxiety and avoidance associated with the phobic object or situation result in significant impairment in day-to-day activities, occupational or academic functioning, and relationships.

People with specific phobia typically understand that their fear is excessive and unreasonable but experience such intense anxiety and distress when confronted with the stimulus that it frequently drives them to avoid these feared situations entirely. Many individuals with specific phobia also experience anxiety over embarrassing themselves in front of others. Symptoms of specific phobia also share many similarities with other anxiety disorders, which can result in issues at diagnosis (Stinson et al., 2007). Specific phobia is differentiated by the focus of the fear: Specific phobia is characterized by fears of a specific situation and PD is more characterized by a fear of the actual panic attack (Barlow, 2002). The emphasis in specific phobia thus lies on the stimulus and not the fear symptoms.

The DSM-5 recognizes five types of specific phobia, including animal type (e.g., fears of snakes, spiders, insects, dogs), natural environment type (e.g., fear of stormy weather, heights, being close to water), blood–injection–injury type (e.g., fear of needles, seeing blood, surgical procedures), situational type (e.g., fear of small places, elevators, airplanes), and other types (e.g., fear of vomiting or choking, loud noises) (American Psychiatric Association, 2013). It is important for the diagnostician to distinguish specific phobia from other anxiety disorders that also involve both avoidance and panic attacks, as the treatments focus on different aspects of the anxiety.

According to epidemiological studies from the United States, specific phobia has a lifetime prevalence rate of 18.4% and a 12-month prevalence rate of 12.1%, making specific phobia the most common of the anxiety disorders (Kessler et al., 2012). Women report a greater number of phobias, higher fear ratings, and more animal and situational phobias than men, and are about twice as likely overall to suffer from specific phobia (Fredrikson, Annas, Fischer, & Wik, 1996). Specific phobia is a chronic disorder, and symptomatic episodes tend to last continuously for an average of 20 years in the absence of treatment or intervention (Stinson et al., 2007). The onset of specific phobia typically occurs in childhood or adolescence, with an average onset ranging from 7 years

of age to 17 depending on the sample (Kessler, Berglund et al., 2005; Kessler et al., 2012). Despite the early age of onset, most people with specific phobia do not receive treatment until middle adulthood (average age of first treatment = 31 years) and only 8% of those who seek treatment report receiving treatment specifically for specific phobia (Stinson et al., 2007). People often do not seek out treatment, because if the situation can be avoided, then the anxiety can be alleviated. If people with a specific phobia cannot avoid the phobic situation, however, there is often interference with their work and leisure activities and it impacts their quality of life (Choy, Fyer, & Lipsitz, 2007).

4.3.2 Treatments

4.3.2.1 Category I: Therapeutically and theoretically well supported

4.3.2.1.1 Cognitive–behavioral therapy

CBT for specific phobia is based on the well-established theory of fear acquisition and on various well-known cognitive biases that characterize specific phobia. It is the only effective well-studied treatment for specific phobia.

Fear acquisition models. It has been assumed that a specific phobia develops when an association is learned between a neutral stimulus (e.g., a dog) and a stimulus that elicits distress or pain (e.g., a dog attack) (Mowrer, 1939; Rachman, 1991). Henceforth, this stimulus will elicit fear reactions and becomes, in Pavlovian terms, a conditioned stimulus. However, this classical conditioning model cannot explain why many individuals with specific phobia cannot recall a traumatic experience at the onset of their phobia or why some people develop phobias after simply hearing about or observing a traumatic conditioning experience (Field, 2006; Rachman, 1991). An additional model suggests that we are predisposed to developing phobias toward certain stimuli that are aversive for evolutionary reasons or through information pathways. The neo-conditioning model emphasizes the primacy of fear acquisition through information pathways (e.g., a child learns that snakes are harmful) and vicarious learning (e.g., a child observes her father responding fearfully to a snake) (Rachman, 1991). The neo-conditioning model has been supported through animal research showing that even rhesus monkeys display observational learning and can acquire a fear of snakes only by observing a video in which other monkeys respond fearfully to snakes (Mineka, Davidson, Cook, & Keir, 1984). However, this model does not adequately touch on the learning that can occur through nonassociative pathways. Some have suggested that there are a small number of innate fears (e.g., objects that have historically posed a threat to survival) that do not require any form of associative or vicarious learning to develop (Öhman & Mineka, 2001). There is a great deal of support for nonassociative fear acquisition in humans and animals (Cook & Mineka, 1990; Menzies & Parker, 2001). When evaluating empirical support for the conceptualization of specific phobia, it is important to note that the majority of research has been conducted with animal models, which may not translate directly to humans (Vervliet & Raes, 2013). On the other hand, the models do draw on behavioral animal research that is unavailable in other disorders (e.g., PD or SAD).

Cognitive biases and information-processing. Higher-order thought processes are important in fear acquisition and the development of specific phobia. The most important processes are harm expectancy, perception of controllability, and predictability

(Hofmann, 2008). In specific phobia, individuals learn an association between the phobic stimulus and an aversive experience (e.g., being appalled by a spider and experiencing an intense fear reaction), which results in their conclusion that the phobic stimulus is a direct source of danger or a signal of threat. Because of the harm expectation ("spiders will startle me and I will feel the fear"), people with a specific phobia will avoid the phobic stimulus and feel distress and anxiety if exposed to the phobic stimulus; consequently, the association between the phobic stimulus and fear is reinforced. The unpredictability of the onset of a feared situation or object and the lack of control the patient has over the aversive event both play a causal role in the genesis of anxiety (Barlow, 2002). For individuals with specific phobia, maladaptive cognitions concerning their inability to predict when they will encounter the phobic stimulus result in maladaptive coping: a hypervigilance to threat detection and a tendency to constantly scan their environment for their particular phobic stimulus. Individuals with specific phobia display a strong attentional bias toward their feared stimulus. In both animals and humans, there are certain stimuli that are preferentially activated and particularly susceptible to fear acquisition due to preparedness, or an innate biological predisposition toward fear (Mineka & Zinbarg, 2006). Accordingly, attentional biases seem to be more common in people with specific phobias of spiders, snakes, or heights. Attentional biases are often expressed in persistent scanning of the environment for the presence of the phobic stimulus, and result in frequent misinterpretation of a neutral object as the phobic stimulus and subsequent onset of a panic attack or fear response despite the lack of "legitimate" stimuli (MacLeod et al., 2002).

Specific phobia treatment components. Effective treatment of specific phobia focuses primarily on the extinction of the learned association between the phobic stimulus and the conditioned fear response. Exposure to the phobic stimulus is key, though it can be useful to start with a decent psychoeducation about the nature of anxiety and avoidance behavior as a fear response. It is often a significant relief for patients to learn about the nature of specific phobia as well as to understand how their avoidance behaviors are reinforcing their fear and anxiety. Cognitive restructuring can be a useful tool to address dysfunctional beliefs about the phobic stimulus, by helping the patient to evaluate the accuracy or true probability of each situation, though this step is often redundant, as many patients with specific phobia understand that their fears are irrational and exaggerated. Exposure is the critical component of treatment for specific phobia. Before beginning exposure exercises, the therapist must identify the phobic stimuli and situations that trigger the phobic's fear response. Exposure treatment can be done gradually, beginning with the least fear-provoking and gradually progressing through a fear hierarchy, but doing so is not necessary to obtain decent extinction (Craske et al., 2014). During exposure it is very important that the patient does not engage in any safety or avoidance behaviors, as this will prevent fear from being truly extinguished and will instead teach patients that their safety cues are what prevents the anxiety. Behavioral exposures are the most effective treatment when compared to placebo or active alternative psychotherapeutic approaches (Choy et al., 2007; Wolitzky-Taylor, Horowitz, Powers, & Telch, 2008). In a meta-analysis of 33 randomized treatment studies of specific phobia, placebo treatments proved superior to inactive control conditions, suggesting that people with specific phobia may be responsive to placebo treatments (Wolitzky-Taylor et al., 2008). It is noteworthy that behavioral exposures with direct

contact with the phobic stimulus produced the most robust effect size at posttreatment. However, in vivo exposures were not significantly better at follow-up than imaginal exposures, in which patients are asked to imagine that they are in the phobic situation, or from virtual reality exposures, in which patients are exposed to a virtual environment containing the feared stimulus. Treatment gains achieved during in vivo exposures are generally maintained for at least 1 year but are also associated with high rates of attrition and low ratings of treatment acceptability (Choy et al., 2007). The attrition rates and low treatment acceptability underpin the increasing popularity of imaginal and virtual reality exposures.

4.3.2.2 Category II: Therapeutically well supported and theoretically equivocal

No treatments were found for this category.

4.3.2.3 Category III: Therapeutically equivocal and theoretically well supported

No treatments were found for this category.

4.3.2.4 Category IV: Therapeutically and theoretically equivocal

No treatments were found for this category.

4.3.2.5 Category V: Therapeutically well supported and theoretically contradictory

No treatments were found for this category.

4.3.2.6 Category VI: Therapeutically contradictory and theoretically well supported

No treatments were found for this category.

4.3.2.7 Category VII: Therapeutically equivocal and theoretically contradictory

No treatments were found for this category.

4.3.2.8 Category VIII: Therapeutically contradictory and theoretically equivocal

No treatments were found for this category.

4.3.2.9 Category IX: Therapeutically and theoretically contradictory

No treatments were found for this category.

4.4 Implications for Research

CBT is the most effective treatment for PD, SAD, and specific phobia, and it is also the most thoroughly researched. However, not all patients respond well to this treatment and research on the long-term effects is inconsistent. Fear can easily be extinguished, but often there is a spontaneous return of fear (Vervliet et al., 2013). Thus, it is highly important to continue to study ways to enhance the (long-term) effects of CBT, as well as its mechanisms. Additionally, since there are a number of patients who do not respond well to CBT for anxiety disorders, it might be useful to study the effectiveness of other treatments in order to find alternatives or adjunctive treatments for CBT.

One promising direction is to add cognitive enhancers such as yohimbine or d-cycloserine, or beta-blockers, such as propranolol, to exposure procedures (Guastella

et al., 2008; Hofmann et al., 2006; Soeter & Kindt, 2015). Another interesting path is the combination of CBT with elements or techniques from other treatments, such as the physical exercises and mindfulness techniques in PD treatment or the mindfulness and attention modification training in SAD treatment. All these techniques have solid theoretical backgrounds, but not enough research has been done to consider these treatments as solid stand-alone treatments. It would thus be useful to research these treatments as stand-alone treatments and in combination with CBT. It is also possible that elements of these treatments share mechanisms with the CBT approach. For example, physical exercises can be similar to some of the interoceptive exposure exercises, as are mindfulness techniques that help patients to experience panic symptoms without trying to control or judge them; therefore, this experience is a type of exposure to the panic sensations. Social skills training may be effective in treating SAD because it improves people's social skills or because practicing these skills is a type of repeated exposure exercise.

Some treatments we described as Category IV in this chapter could potentially be useful, but there are not enough RCTs available and the theory is not solidly based. For these treatments, more research is necessary to either consolidate the potential beneficial effects or reject these treatments entirely.

4.5 Implications for Practice

CBT is the first-line treatment for PD and phobias. This treatment is based on theoretical models and clear concepts and has shown its merits over decades of research. Although exposure is key in CBT for PD and phobias, not all therapists who claim to do CBT use exposure exercises. Lack of training or a fear of patients' fear can often be the origin of this avoidance.

Exposure, however, is more than just facing fears. Patients must understand the rationale of the treatment: why they have to expose themselves to the feared stimuli and the great importance of ending their avoidance or safety behaviors. Forcing people to face feared situations without proper preparation can elicit a new aversive learning experience, which increases the chance that patients will drop out of what is their best treatment option. CBT therapists have to find a good balance between encouraging and motivating the patient to do the exposure exercises, and giving support and validation to the debilitating and intense fear responses that these exposure exercises elicit. All therapists who would like to treat anxiety disorders need to have received decent (practical and theoretical) training in CBT before attempting it on their own.

In some cases patients will refuse to do exposures. Therapists should work with this resistance and invest time in psychoeducation and cognitive restructuring before engaging in exposures. Nonetheless, therapists should be familiar with the other psychotherapies that are present in this chapter so that, if needed, they can offer their patients other types of treatments while emphasizing that they either are less well researched or have been found to be less effective.

4.6 Conclusions

There are a number of treatments available to treat people who suffer from fear and anxiety, and CBT is definitively the first-line treatment for PD, SAD, and SP. Its outcomes

are well studied and show good effect sizes (Deacon & Abramowitz, 2004). For the three disorders we described above, CBT has solid theoretical underpinning. A more detailed description of the CBT techniques presented in this chapter can be found in Hofmann (2011). There is room for improvement, since CBT is not effective for all patients. Therefore, the combination of CBT with other treatments based on well-researched theories is promising and should continue to be studied.

References

Acarturk, C., Cuijpers, P., Van Straten, A., & de Graaf, R. (2009). Psychological treatment of social anxiety disorder: A meta-analysis. *Psychological Medicine*, *39*(2), 241–254. doi:10.1017/S0033291708003590

Alden, L. E., & Bieling, P. (1998). Interpersonal consequences of the pursuit of safety. *Behaviour Research and Therapy*, *36*(1), 53–64. doi:10.1016/S0005-7967(97)00072-7

Alden, L. E., & Taylor, C. T. (2004). Interpersonal processes in social phobia. *Clinical Psychology Review*, *24*(7), 857–882. doi:10.1016/j.cpr.2004.07.006

Allen, N. B., Blashki, G., & Gullone, E. (2006). Mindfulness-based psychotherapies: A review of conceptual foundations, empirical evidence and practical considerations. *Australian & New Zealand Journal of Psychiatry*, *40*(4), 285–294. doi:10.1111/j.1440-1614.2006.01794

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.

Amir, N., Beard, C., Taylor, C. T., Klumpp, H., Elias, J., Burns, M., & Chen, X. (2009). Attention training in individuals with generalized social phobia: A randomized controlled trial. *Journal of Consulting and Clinical Psychology*, *77*(5), 961–973. doi:10.1037/a0016685

Amir, N., Weber, G., Beard, C., Bomyea, J., & Taylor, C. T. (2008). The effect of a single-session attention modification program on response to a public-speaking challenge in socially anxious individuals. *Journal of Abnormal Psychology*, *117*(4), 860–868. doi:10.1037/a0013445

Anderson, E. R., & Hope, D. A. (2009). The relationship among social phobia, objective and perceived physiological reactivity, and anxiety sensitivity in an adolescent population. *Journal of Anxiety Disorders*, *23*(1), 18–26. doi:10.1016/j.janxdis.2008.03.011

Arntz, A. (2012). Imagery rescripting as a therapeutic technique: Review of clinical trials, basic studies, and research agenda. *Journal of Experimental Psychopathology*, *3*(2), 189–208. doi:10.5127/jep.024211

Arntz, A., Sofi, D., & Van Breukelen, G. (2013). Imagery rescripting as treatment for complicated PTSD in refugees: A multiple baseline case series study. *Behaviour Research and Therapy*, *51*(6), 274–283. doi:10.1016/j.brat.2013.02.009

Arntz, A., & Van den Hout, M. (1996). Psychological treatments of panic disorder without agoraphobia: Cognitive therapy versus applied relaxation. *Behaviour Research and Therapy*, *34*(2), 113–121. doi:10.1016/0005-7967(95)00061-5

Arntz, A., & Weertman, A. (1999). Treatment of childhood memories: Theory and practice. *Behaviour Research and Therapy*, *37*(8), 715–740. doi:10.1016/S0005-7967(98)00173-9

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & Van Izendoorn, M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: A meta-analytic study. *Psychological Bulletin*, *133*(1), 1–24. doi:10.1037/0033-2909.133.1.1

Barlow, D. H. (2002). *Anxiety and its disorders: The nature and treatment of anxiety and panic* (2nd ed.). New York, NY: Guilford Press.

Barrera, T. L., & Norton, P. J. (2009). Quality of life impairment in generalized anxiety disorder, social phobia, and panic disorder. *Journal of Anxiety Disorders*, *23*(8), 1086–1090. doi:10.1016/j.janxdis.2009.07.011

Beck, J. G., Stanley, M. A., Averill, P. M., Baldwin, L. E., & Deagle, E. A. (1992). Attention and memory for threat in panic disorder. *Behaviour Research and Therapy*, *30*(6), 619–629. doi:10.1016/0005-7967(92)90007-4

Beutel, M. E., Scheurich, V., Knebel, A., Michal, M., Wiltink, J., Graf-Morgenstern, M., & Subic-Wrana, C. (2013). Implementing panic-focused psychodynamic psychotherapy into clinical practice. *Canadian Journal of Psychiatry/La Revue Canadienne de Psychiatrie*, *58*(6), 326–334. doi:10.1177/070674371305800604

Bishop, S. R., Lau, M., Shapiro, S., Carlson, L., Anderson, N. D., Carmody, J., & Devins, G. (2004). Mindfulness: A proposed operational definition. *Clinical Psychology: Science and Practice*, *11*(3), 230–241. doi:10.1093/clipsy.bph077

Brewin, C. R. (2006). Understanding cognitive behaviour therapy: A retrieval competition account. *Behaviour Research and Therapy*, *44*(6), 765–784. doi:10.1016/j.brat.2006.02.005

Boettcher, J., Berger, T., & Renneberg, B. (2012). Internet-based attention training for social anxiety: A randomized controlled trial. *Cognitive Therapy and Research*, *36*(5), 522–536. doi:10.1007/s10608-011-9374-y

Boettcher, J., Leek, L., Matson, L., Holmes, E. A., Browning, M., MacLeod, C., … Carlbring, P. (2013). Internet-based attention bias modification for social anxiety: A randomised controlled comparison of training towards negative and training towards positive cues. *PLOS One*, *8*(9). doi:10.1371/journal.pone.0071760

Bögels, S. M. (2006). Task concentration training versus applied relaxation, in combination with cognitive therapy, for social phobia patients with fear of blushing, trembling, and sweating. *Behaviour Research and Therapy*, *44*(8), 1199–1210. doi:10.1016/j.brat.2005.08.010

Bögels, S. M., & Voncken, M. (2008). Social skills training versus cognitive therapy for social anxiety disorder characterized by fear of blushing, trembling, or sweating. *International Journal of Cognitive Therapy*, *1*(2), 138–150. doi:10.1521/ijct.2008.1.2.138

Borge, F., Hoffart, A., Sexton, H., Clark, D. M., Markowitz, J. C., & McManus, F. (2008). Residential cognitive therapy versus residential interpersonal therapy for social phobia: A randomized clinical trial. *Journal of Anxiety Disorders*, *22*(6), 991–1010. doi:10.1016/j.janxdis.2007.10.002

Broocks, A., Bandelow, B., Pekrun, G., George, A., Meyer, T., Bartmann, U., & Rüther, E. (1998). Comparison of aerobic exercise, clomipramine, and placebo in the treatment of panic disorder. *American Journal of Psychiatry*, *155*(5), 603–609. doi:10.1176/ajp.155.5.603

Busch, F. N., & Milrod, B. L. (2009). Psychodynamic treatment of panic disorder. In R. A. Levy, J. S. Ablon, R. A. Levy, & J. S. Ablon (Eds.), *Handbook of evidence-based psychodynamic psychotherapy: Bridging the gap between science and practice* (pp. 29–44). Totowa, NJ: Humana Press.

Busch, F. N., Milrod, B. L., & Singer, M. B. (1999). Theory and technique in psychodynamic treatment of panic disorder. *Journal of Psychotherapy Practice and Research*, *8*(3), 234–242.

Carlbring, P., Apelstrand, M., Sehlin, H., Amir, N., Rousseau, A., Hofmann, S. G., & Andersson, G. (2012). Internet-delivered attention bias modification training in individuals with social anxiety disorder: A double blind randomized controlled trial. *BMC Psychiatry*, *12*, 66. doi:10.1186/1471-244X-12-66

Choy, Y., Fyer, A. J., & Lipsitz, J. D. (2007). Treatment of specific phobia in adults. *Clinical Psychology Review*, *27*(3), 266–286. doi:10.1016/j.cpr.2006.10.002

Clark, D. A., & Beck, A. T. (2010). Cognitive theory and therapy of anxiety and depression: Convergence with neurobiological findings. *Trends in Cognitive Sciences*, *14*(9), 418–424. doi:10.1016/j.tics.2010.06.007

Clark, D. M. (1999). Anxiety disorders: Why they persist and how to treat them. *Behaviour Research and Therapy*, *37*, S5–S27. doi:10.1016/S0005-7967(99)00048-0

Clark, D. M., Salkovskis, P. M., Hackmann, A., Middleton, H., Anastasiades, P., & Gelder, M. (1994). A comparison of cognitive therapy, applied relaxation and imipramine in the treatment of panic disorder. *British Journal of Psychiatry*, *164*(6), 759–769. doi:10.1192/bjp.164.6.759

Clark, D. M., & Wells, A. (1995). A cognitive model of social phobia. In R. G. Heimberg, M. R. Liebowitz, D. A. Hope, & F. R. Schneier (Eds.), *Social phobia: Diagnosis, assessment, and treatment* (pp. 69–93). New York, NY: Guilford Press.

Cook, M., & Mineka, S. (1990). Selective associations in the observational conditioning of fear in rhesus monkeys. *Journal of Experimental Psychology: Animal Behavior Processes*, *16*(4), 372–389. doi:10.1037/0097-7403.16.4.372

Craske, M. G., & Barlow, D. H. (2006). *Mastery of your anxiety and worry: Workbook*. Oxford, UK: Oxford University Press.

Craske, M. G., Lang, A. J., Mystkowski, J. L., Zucker, B. G., Bystritsky, A., & Yan-Go, F. (2002). Does nocturnal panic represent a more severe form of panic disorder? *Journal of Nervous and Mental Disease*, *190*(9), 611–618. doi:10.1097/00005053-200209000-00006

Craske, M. G., Niles, A. N., Burklund, L. J., Wolitzky-Taylor, K. B., Vilardaga, J. P., Arch, J. J., … Lieberman, M. D. (2014). Randomized controlled trial of cognitive behavioral therapy and acceptance and commitment therapy for social phobia: Outcomes and moderators. *Journal of Consulting and Clinical Psychology*, *82*(6), 1034–1048. doi:10.1037/a0037212

Cusack, K., Jonas, D. E., Forneris, C. A., Wines, C., Sonis, J., Middleton, J. C., & Gaynes, B. N. (2016). Psychological treatments for adults with posttraumatic stress disorder: A systematic review and meta-analysis. *Clinical Psychology Review*, *43*, 128–141. doi:10.1016/j.cpr.2015.10.003

Daly, J. A., Vangelisti, A. L., & Lawrence, S. G. (1989). Self-focused attention and public speaking anxiety. *Personality and Individual Differences*, *10*(8), 903–913. doi:10.1016/0191-8869(89)90025-1

Deacon, B. J., & Abramowitz, J. S. (2004). Cognitive and behavioral treatments for anxiety disorders: A review of meta-analytic findings. *Journal of Clinical Psychology*, *60*(4), 429–441. doi:10.1002/jclp.10255

DeBoer, L. B., Powers, M. B., Utschig, A. C., Otto, M. W., & Smits, J. J. (2012). Exploring exercise as an avenue for the treatment of anxiety disorders. *Expert Review of Neurotherapeutics*, *12*(8), 1011–1022. doi:10.1586/ern.12.73

Dey, S., Singh, R. H., & Dey, P. K. (1992). Exercise training: Significance of regional alterations in serotonin metabolism of rat brain in relation to antidepressant effect of exercise. *Physiology & Behavior*, *52*(6), 1095–1099. doi:10.1016/0031-9384(92)90465-E

Dibbets, P., Poort, H., & Arntz, A. (2012). Adding imagery rescripting during extinction leads to less ABA renewal. *Journal of Behavior Therapy and Experimental Psychiatry*, *43*(1), 614–624. doi:10.1016/j.jbtep.2011.08.006

Eaton, W. W., Kessler, R. C., Wittchen, H. U., & Magee, W. J. (1994). Panic and panic disorder in the United States. *American Journal of Psychiatry*, *151*, 413–420. doi:10.1176/ajp.151.3.413

Ehlers, A., & Breuer, P. (1992). Increased cardiac awareness in panic disorder. *Journal of Abnormal Psychology*, *101*(3), 371–382. doi:10.1037/0021-843X.101.3.371

Faucher, J., Koszycki, D., Bradwejn, J., Merali, Z., & Bielajew, C. (2016). Effects of CBT versus MBSR treatment on social stress reactions in social anxiety disorder. *Mindfulness*, *7*(2), 514–526. doi:10.1007/s12671-015-0486-4

Field, A. P. (2006). Is conditioning a useful framework for understanding the development and treatment of phobias? *Clinical Psychology Review*, *26*(7), 857–875. doi:10.1016/j.cpr.2005.05.010

Fredrikson, M., Annas, P., Fischer, H., & Wik, G. (1996). Gender and age differences in the prevalence of specific fears and phobias. *Behaviour Research and Therapy*, *34*(1), 33–39. doi:10.1016/0005-7967(95)00048-3

Goisman, R. M., Goldenberg, I., Vasile, R. G., & Keller, M. B. (1995). Comorbidity of anxiety disorders in a multicenter anxiety study. *Comprehensive Psychiatry*, *36*(4), 303–311. doi:10.1016/S0010-440X(95)90076-4

Goldin, P., Ramel, W., & Gross, J. (2009). Mindfulness meditation training and self-referential processing in social anxiety disorder: Behavioral and neural effects. *Journal of Cognitive Psychotherapy*, *23*(3), 242–257. doi:10.1891/0889-8391.23.3.242

Goldstein, A. J., de Beurs, E., Chambless, D. L., & Wilson, K. A. (2000). EMDR for panic disorder with agoraphobia: Comparison with waiting list and credible attention-placebo control conditions. *Journal of Consulting and Clinical Psychology*, *68*(6), 947–956. doi:10.1037/0022-006X.68.6.947

Grant, B. F., Stinson, F. S., Dawson, D. A., Chou, S. P., Dufour, M. C., Compton, W., … Kaplan, K. (2004). Prevalence and co-occurrence of substance use disorders and independent mood and anxiety disorders: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Archives of General Psychiatry*, *61*(8), 807–816. doi:10.1001/archpsyc.61.8.807

Greenberg, L. S. (2004). Emotion-focused therapy. *Clinical Psychology & Psychotherapy*, *11*(1), 3–16. doi:10.1002/cpp.388

Guastella, A. J., Richardson, R., Lovibond, P. F., Rapee, R. M., Gaston, J. E., Mitchell, P., & Dadds, M. R. (2008). A randomized controlled trial of d-cycloserine enhancement of exposure therapy for social anxiety disorder. *Biological Psychiatry*, *63*(6), 544–549. doi:10.1016/j.biopsych.2007.11.011

Heeren, A., Lievens, L., & Philippot, P. (2011). How does attention training work in social phobia: Disengagement from threat or re-engagement to non-threat? *Journal of Anxiety Disorders*, *25*(8), 1108–1115. doi:10.1016/j.janxdis.2011.08.001

Heeren, A., Mogoaşe, C., Philippot, P., & McNally, R. J. (2015). Attention bias modification for social anxiety: A systematic review and meta-analysis. *Clinical Psychology Review*, *40*, 76–90. doi:10.1016/j.cpr.2015.06.001

Heeren, A., Reese, H. E., McNally, R. J., & Philippot, P. (2012). Attention training toward and away from threat in social phobia: Effects on subjective, behavioral, and

physiological measures of anxiety. *Behaviour Research and Therapy*, *50*(1), 30–39. doi:10.1016/j.brat.2011.10.005

Hegel, M. T., & Ferguson, R. J. (1997). Psychophysiological assessment of respiratory function in panic disorder: Evidence for a hyperventilation subtype. *Psychosomatic Medicine*, *59*(3), 224–230. doi:10.1097/00006842-199705000-00003

Heinrichs, N., Hoffman, E. C., & Hofmann, S. G. (2001). Cognitive-behavioral treatment for social phobia in Parkinson's disease: A single-case study. *Cognitive and Behavioral Practice*, *8*(4), 328–335. doi:10.1016/S1077-7229(01)80005-5

Herbert, J. D., Gaudiano, B. A., Rheingold, A. A., Myers, V. H., Dalrymple, K., & Nolan, E. M. (2005). Social skills training augments the effectiveness of cognitive behavioral group therapy for social anxiety disorder. *Behavior Therapy*, *36*(2), 125–138. doi:10.1016/S0005-7894(05)80061-9

Hirsch, C., Meynen, T., & Clark, D. (2004). Negative self-imagery in social anxiety contaminates social interactions. *Memory*, *12*(4), 496–506. doi:10.1080/09658210444000106

Hofmann, S. G. (2007). Cognitive factors that maintain social anxiety disorder: A comprehensive model and its treatment implications. *Cognitive Behaviour Therapy*, *36*(4), 193–209. doi:10.1080/16506070701421313

Hofmann, S. G. (2008). Social anxiety disorder: Neglected no more. *PsycCRITIQUES*, *53*(30). doi:10.1037/a0012556

Hofmann, S. G. (2011). *An introduction to modern CBT: Psychological solutions to mental health problems*. Malden, MA: Wiley-Blackwell.

Hofmann, S. G., Heinrichs, N., & Moscovitch, D. A. (2004). The nature and expression of social phobia: Toward a new classification. *Clinical Psychology Review*, *24*(7), 769–797. doi:10.1016/j.cpr.2004.07.004

Hofmann, S. G., Meuret, A. E., Smits, J. J., Simon, N. M., Pollack, M. H., Eisenmenger, K., … Otto, M. W. (2006). Augmentation of exposure therapy with d-cycloserine for social anxiety disorder. *Archives of General Psychiatry*, *63*(3), 298–304. doi:10.1001/archpsyc.63.3.298

Hofmann, S. G., & Otto, M. W. (2008). *Cognitive-behavior therapy for social anxiety disorder: Evidence-based and disorder-specific treatment techniques*. New York, NY: Routledge.

Hofmann, S. G., Sawyer, A. T., Witt, A. A., & Oh, D. (2010). The effect of mindfulness-based therapy on anxiety and depression: A meta-analytic review. *Journal of Consulting and Clinical Psychology*, *78*(2), 169–183. doi:10.1037/a0018555

Hofmann, S. G., & Smits, J. J. (2008). Cognitive-behavioral therapy for adult anxiety disorders: A meta-analysis of randomized placebo-controlled trials. *Journal of Clinical Psychiatry*, *69*(4), 621–632. doi:10.4088/JCP.v69n0415

Hope, D. A., Gansler, D. A., & Heimberg, R. G. (1989). Attentional focus and causal attributions in social phobia: Implications from social psychology. *Clinical Psychology Review*, *9*(1), 49–60. doi:10.1016/0272-7358(89)90046-9

Hovland, A., Nordhus, I. H., Sjøbø, T., Gjestad, B. A., Birknes, B., Martinsen, E. W., & Pallesen, S. (2013). Comparing physical exercise in groups to group cognitive behaviour therapy for the treatment of panic disorder in a randomized controlled trial. *Behavioural and Cognitive Psychotherapy*, *41*(4), 408–432. doi:10.1017/s1352465812000446

Jacobson, E. (1938). *Progressive relaxation* (2nd ed.). Chicago, IL: University Chicago Press.

Julian, K., Beard, C., Schmidt, N. B., Powers, M. B., & Smits, J. J. (2012). Attention training to reduce attention bias and social stressor reactivity: An attempt to replicate and extend previous findings. *Behaviour Research and Therapy*, *50*(5), 350–358. doi:10.1016/j.brat.2012.02.015

Kabat-Zinn, J. (2003). Mindfulness-based interventions in context: Past, present, and future. *Clinical Psychology: Science and Practice*, *10*(2), 144–156. doi:10.1093/clipsy/bpg016

Kabat-Zinn, J., Massion, A. O., Kristeller, J., Peterson, L. G., Fletcher, K. E., Pbert, L., … Santorelli, S. F. (1992). Effectiveness of a meditation-based stress reduction program in the treatment of anxiety disorders. *American Journal of Psychiatry*, *149*, 936–943. doi:10.1176/ajp.149.7.936

Katzelnick, D. J., Kobak, K. A., DeLeire, T., Henk, H. J., Greist, J. H., Davidson, J. T., … Helstad, C. P. (2001). Impact of generalized social anxiety disorder in managed care. *American Journal of Psychiatry*, *158*(12), 1999–2007. doi:10.1176/appi.ajp.158.12.1999

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*(6), 593–602. doi:10.1001/archpsyc.62.6.593

Kessler, R. C., Chiu, W. T., Demler, O., Merikangas, K. R., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*(6), 617–627. doi:10.1001/archpsyc.62.6.617

Kessler, R. C., Petukhova, M., Sampson, N. A., Zaslavsky, A. M., & Wittchen, H. U. (2012). Twelve-month and lifetime prevalence and lifetime morbid risk of anxiety and mood disorders in the United States. *International Journal of Methods in Psychiatric Research*, *21*(3), 169–184. doi:10.1002/mpr.1359

Kessler, R. C., Soukup, J., Davis, R. B., Foster, D. F., Wilkey, S. A., Van Rompay, M. I., & Eisenberg, D. M. (2001). The use of complementary and alternative therapies to treat anxiety and depression in the United States. *American Journal of Psychiatry*, *158*(2), 289–294. doi:10.1176/appi.ajp.158.2.289

Kessler, R. C., Stein, M. B., & Berglund, P. (1998). Social phobia subtypes in the National Comorbidity Survey. *American Journal of Psychiatry*, *155*(5), 613–619. doi:10.1176/ajp.155.5.613

Khoury, B., Lecomte, T., Fortin, G., Masse, M., Therien, P., Bouchard, V., … Hofmann, S. G. (2013). Mindfulness-based therapy: A comprehensive meta-analysis. *Clinical Psychology Review*, *33*(6), 763–771. doi:10.1016/j.cpr.2013.05.005

Kim, B., Lee, S., Kim, Y. W., Choi, T. K., Yook, K., Suh, S. Y., & Yook, K. (2010). Effectiveness of a mindfulness-based cognitive therapy program as an adjunct to pharmacotherapy in patients with panic disorder. *Journal of Anxiety Disorders*, *24*(6), 590–595. doi:10.1016/j.janxdis.2010.03.019

Klerman, G. L., & Weissman, M. M. (1993). *New applications of interpersonal psychotherapy*. Washington, DC: American Psychiatric Press.

Knijnik, D. Z., Blanco, C., Salum, G. A., Moraes, C. U., Mombach, C., Almeida, E., & Eizirik, C. L. (2008). A pilot study of clonazepam versus psychodynamic group therapy plus clonazepam in the treatment of generalized social anxiety disorder. *European Psychiatry*, *23*(8), 567–574. doi:10.1016/j.eurpsy.2008.05.004

Knijnik, D. Z., Hauck, S., Mombach, C. K., de Almeida, E. A., & Eizirik, C. L. (2008). Research in analytically oriented group psychotherapy: A tool for the evaluation of adherence to the technique in studies of effectiveness. *International Journal of Psychoanalysis*, *89*(6), 1222–1224. doi:10.1111/j.1745-8315.2008.00095.x

Knijnik, D. Z., Kapczinski, F., Chachamovich, E., Margis, R., & Eizirik, C. L. (2004). Psicoterapia psicodinâmica em grupo para fobia social generalizada [Psychodynamic group treatment for generalized social phobia]. *Revista Brasileira de Psiquiatria*, *26*(2), 77–81. doi:10.1590/S1516-44462004000200003

Koszycki, D., Benger, M., Shlik, J., & Bradwejn, J. (2007). Randomized trial of a meditation-based stress reduction program and cognitive behavior therapy in generalized social anxiety disorder. *Behaviour Research and Therapy*, *45*(10), 2518–2526. doi:10.1016/j.brat.2007.04.011

Lee, S. W., & Kwon, J. H. (2013). The efficacy of imagery rescripting (IR) for social phobia: A randomized controlled trial. *Journal of Behavior Therapy and Experimental Psychiatry*, *44*(4), 351–360. doi:10.1016/j.jbtep.2013.03.001

Leichsenring, F., Salzer, S., Beutel, M. E., Herpertz, S., Hiller, W., Hoyer, J., … Leibing, E. (2013). Psychodynamic therapy and cognitive-behavioral therapy in social anxiety disorder: A multicenter randomized controlled trial. *American Journal of Psychiatry*, *170*(7), 759–767.

Lépine, J. P. (2002). The epidemiology of anxiety disorders: Prevalence and societal costs. *Journal of Clinical Psychiatry*, *63*(14), 4–8.

Li, S., Tan, J., Qian, M., & Liu, X. (2008). Continual training of attentional bias in social anxiety. *Behaviour Research and Therapy*, *46*(8), 905–912. doi:10.1016/j.brat.2008.04.005

Linetzky, M., Pergamin-Hight, L., Pine, D. S., & Bar-Haim, Y. (2015). Quantitative evaluation of the clinical efficacy of attention bias modification treatment for anxiety disorders. *Depression and Anxiety*, *32*(6), 383–391. doi:10.1002/da.22344

Lipsitz, J. D., Gur, M., Vermes, D., Petkova, E., Cheng, J., Miller, N., … Fyer, A. J. (2008). A randomized trial of interpersonal therapy versus supportive therapy for social anxiety disorder. *Depression and Anxiety*, *25*(6), 542–553. doi:10.1002/da.20364

Lipsitz, J. D., Markowitz, J. C., Cherry, S., & Fyer, A. J. (1999). Open trial of interpersonal psychotherapy for the treatment of social phobia. *American Journal of Psychiatry*, *156*(11), 1814–1816.

MacLeod, C., & Mathews, A. (2012). Cognitive bias modification approaches to anxiety. *Annual Review of Clinical Psychology*, *8*, 189–217. doi:10.1146/annurev-clinpsy-032511-143052

MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology*, *95*(1), 15–20. doi:10.1037/0021-843X.95.1.15

MacLeod, C., Rutherford, E., Campbell, L., Ebsworthy, G., & Holker, L. (2002). Selective attention and emotional vulnerability: Assessing the causal basis of their association through the experimental manipulation of attentional bias. *Journal of Abnormal Psychology*, *111*(1), 107–123. doi:10.1037/0021-843X.111.1.107

Magee, W. J., Eaton, W. W., Wittchen, H., McGonagle, K. A., & Kessler, R. C. (1996). Agoraphobia, simple phobia, and social phobia in the National Comorbidity Survey. *Archives of General Psychiatry*, *53*(2), 159–168. doi:10.1001/archpsyc.1996.01830020077009

McHugh, R. K., Smits, J. A., & Otto, M. W. (2009). Empirically supported treatments for panic disorder. *Psychiatric Clinics of North America*, *32*(3), 593–610. doi:10.1016/j.psc.2009.05.005

McNally, R. J. (2002). Anxiety sensitivity and panic disorder. *Biological Psychiatry*, *52*(10), 938–946. doi:10.1016/S0006-3223(02)01475-0

McNally, R. J., Enock, P. M., Tsai, C., & Tousian, M. (2013). Attention bias modification for reducing speech anxiety. *Behaviour Research and Therapy*, *51*(12), 882–888. doi:10.1016/j.brat.2013.10.001

Menzies, R. G., & Parker, L. (2001). The origins of height fear: An evaluation of neoconditioning explanations. *Behaviour Research and Therapy*, *39*(2), 185–199. doi:10.1016/S0005-7967(99)00177-1

Mersch, P. P., Emmelkamp, P. M., Bögels, S. M., & Van der Sleen, J. (1989). Social phobia: Individual response patterns and the effects of behavioral and cognitive interventions. *Behaviour Research and Therapy*, *27*(4), 421–434. doi:10.1016/0005-7967(89)90013-2

Meuret, A. E., Rosenfield, D., Seidel, A., Bhaskara, L., & Hofmann, S. G. (2010). Respiratory and cognitive mediators of treatment change in panic disorder: Evidence for intervention specificity. *Journal of Consulting and Clinical Psychology*, *78*(5), 691–704. doi:10.1037/a0019552

Meuret, A. E., Rosenfield, D., Wilhelm, F. H., Zhou, E., Conrad, A., Ritz, T., & Roth, W. T. (2011). Do unexpected panic attacks occur spontaneously? *Biological Psychiatry*, *70*(10), 985–991. doi:10.1016/j.biopsych.2011.05.027

Meuret, A. E., Wilhelm, F. H., Ritz, T., & Roth, W. T. (2008). Feedback of end-tidal pCO_2 as a therapeutic approach for panic disorder. *Journal of Psychiatric Research*, *42*(7), 560–568. doi:10.1016/j.jpsychires.2007.06.005

Miller, J. J., Fletcher, K., & Kabat-Zinn, J. (1995). Three-year follow-up and clinical implications of a mindfulness meditation-based stress reduction intervention in the treatment of anxiety disorders. *General Hospital Psychiatry*, *17*(3), 192–200. doi:10.1016/0163-8343(95)00025-M

Mineka, S., & Zinbarg, R. (2006). A contemporary learning theory perspective on the etiology of anxiety disorders: It's not what you thought it was. *American Psychologist*, *61*(1), 10–26. doi:10.1037/0003-066X.61.1.10

Milrod, B., Chambless, D. L., Gallop, R., Busch, F. N., Schwalberg, M., McCarthy, K. S., … Barber, J. P. (2015). Psychotherapies for panic disorder: A tale of two sites. *Journal of Clinical Psychiatry*, *77*(7), 927–935. doi:10.4088/JCP.14m09507

Milrod, B. L., Leon, A. C., Barber, J. P., Markowitz, J. C., & Graf, E. (2007). Do comorbid personality disorders moderate panic-focused psychotherapy? An exploratory examination of the American Psychiatric Association practice guideline. *Journal of Clinical Psychiatry*, *68*(6), 885–891. doi:10.4088/JCP.v68n0610

Mineka, S., Davidson, M., Cook, M., & Keir, R. (1984). Observational conditioning of snake fear in rhesus monkeys. *Journal of Abnormal Psychology*, *93*(4), 355–372. doi:10.1037/0021-843X.93.4.355

Morina, N., Lancee, J., & Arntz, A. (2017). Imagery rescripting as a clinical intervention for aversive memories: A meta-analysis. *Journal of Behavior Therapy and Experimental Psychiatry*, *55*, 6–15. doi:10.1016/j.jbtep.2016.11.003

Mowrer, O. H. (1939). A stimulus-response analysis of anxiety and its role as a reinforcing agent. *Psychological Review*, *46*(6), 553–565. doi:10.1037/h0054288

Mulkens, S., De Jong, P. J., & Bögels, S. M. (1997). High blushing propensity: Fearful preoccupation or facial coloration? *Personality and Individual Differences*, *22*(6), 817–824. doi:10.1016/S0191-8869(97)00008-1

Norton, A. R., & Abbott, M. J. (2016). The efficacy of imagery rescripting compared to cognitive restructuring for social anxiety disorder. *Journal of Anxiety Disorders*, *40*, 18–28. doi:10.1016/j.janxdis.2016.03.009

Öhman, A., & Mineka, S. (2001). Fears, phobias, and preparedness: Toward an evolved module of fear and fear learning. *Psychological Review*, *108*(3), 483–522. doi:10.1037/0033-295X.108.3.483

Öst, L. (1986). Antidepressiva farmaka i behandling av agorafobi och panikstörning: En evaluerande översikt [Antidepressants and the treatment of agoraphobia and panic disorders: An evaluative overview]. *Nordisk Psykiatrisk Tidsskrift*, *40*(6), 429–434. doi:10.3109/08039488609096506

Öst, L. (1988). Applied relaxation in the treatment of panic disorder. *Scandinavian Journal of Behaviour Therapy*, *17*(2), 111–124. doi:10.1080/16506078809456266

Öst, L., & Westling, B. E. (1995). Applied relaxation vs. cognitive behavior therapy in the treatment of panic disorder. *Behaviour Research and Therapy*, *33*(2), 145–158. doi:10.1016/0005-7967(94)E0026-F

Öst, L., Westling, B. E., & Hellström, K. (1993). Applied relaxation, exposure in vivo and cognitive methods in the treatment of panic disorder with agoraphobia. *Behaviour Research and Therapy*, *31*(4), 383–394. doi:10.1016/0005-7967(93)90095-C

Otto, M. W., & Pollack, M. H. (2009). *Stopping anxiety medication: Therapist guide*. New York, NY: Oxford University Press.

Otto, M. W., & Smits, J. J. (2011). *Exercise for mood and anxiety: Proven strategies for overcoming depression and enhancing well-being*. New York, NY: Oxford University Press.

Papp, L. A., Klein, D. F., & Gorman, J. M. (1993). Carbon dioxide hypersensitivity, hyperventilation, and panic disorder. *American Journal of Psychiatry*, *150*(8), 1149–1157. doi:10.1176/ajp.150.8.1149

Rachman, S. (1991). Neo-conditioning and the classical theory of fear acquisition. *Clinical Psychology Review*, *11*(2), 155–173. doi:10.1016/0272-7358(91)90093-A

Rapee, R. M., & Heimberg, R. G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy*, *35*(8), 741–756. doi:10.1016/S0005-7967(97)00022-3

Reimer, S. G., & Moscovitch, D. A. (2015). The impact of imagery rescripting on memory appraisals and core beliefs in social anxiety disorder. *Behaviour Research and Therapy*, *75*, 48–59. doi:10.1016/j.brat.2015.10.007

Rodebaugh, T. L., Holaway, R. M., & Heimberg, R. G. (2004). The treatment of social anxiety disorder. *Clinical Psychology Review*, *24*(7), 883–908. doi:10.1016/j.cpr.2004.07.007

Rogers, C. (1951). *Client-centered therapy*. Oxford, UK: Houghton Mifflin.

Ruscio, A. M., Brown, T. A., Chiu, W. T., Sareen, J., Stein, M. B., & Kessler, R. C. (2008). Social fears and social phobia in the USA: Results from the National Comorbidity Survey Replication. *Psychological Medicine*, *38*(1), 15–28. doi:10.1017/S0033291707001699

Salkovskis, P. M., Clark, D. M., & Gelder, M. G. (1996). Cognition-behaviour links in the persistence of panic. *Behaviour Research and Therapy*, *34*(5), 453–458. doi:10.1016/0005-7967(95)00083-6

Salkovskis, P. M., Clark, D. M., Hackmann, A., Wells, A., & Gelder, M. G. (1999). An experimental investigation of the role of safety-seeking behaviours in the maintenance of panic disorder with agoraphobia. *Behaviour Research and Therapy*, *37*(6), 559–574. doi:10.1016/S0005-7967(98)00153-3

Schmidt, N. B., Richey, J. A., Buckner, J. D., & Timpano, K. R. (2009). Attention training for generalized social anxiety disorder. *Journal of Abnormal Psychology*, *118*(1), 5–14. doi:10.1037/a0013643

Shear, M. K., Cooper, A. M., Knijnik, G. L., Busch, F. N., & Shapiro, T. (1993). A psychodynamic model of panic disorder. *American Journal of Psychiatry*, *150*(6), 859–866. doi:10.1176/ajp.150.6.859

Shear, M. K., Houck, P., Greeno, C., & Masters, S. (2001). Emotion-focused psychotherapy for patients with panic disorder. *American Journal of Psychiatry*, *158*(12), 1993–1998. doi:10.1176/appi.ajp.158.12.1993

Soeter, M., & Kindt, M. (2015). Retrieval cues that trigger reconsolidation of associative fear memory are not necessarily an exact replica of the original learning experience. *Frontiers in Behavioral Neuroscience*, *9*(122). doi:10.3389/fnbeh.2015.00122

Stein, M. B., & Kean, Y. M. (2000). Disability and quality of life in social phobia: Epidemiologic findings. *American Journal of Psychiatry*, *157*(10), 1606–1613. doi: 10.1176/appi.ajp.157.10.1606

Stein, M. B., McQuaid, J. R., Laffaye, C., & McCahill, M. E. (1999). Social phobia in the primary care medical setting. *Journal of Family Practice*, *48*(7), 514–519.

Stinson, F. S., Dawson, D. A., Chou, S. P., Smith, S., Goldstein, R. B., Ruan, W. J., & Grant, B. F. (2007). The epidemiology of DSM-IV specific phobia in the USA: Results from the national epidemiologic survey on alcohol and related conditions. *Psychological Medicine*, *37*(7), 1047–1059. doi:10.1017/S0033291707000086

Stopa, L., & Jenkins, A. (2007). Images of the self in social anxiety: Effects on the retrieval of autobiographical memories. *Journal of Behavior Therapy and Experimental Psychiatry*, *38*(4), 459–473. doi:10.1016/j.jbtep.2007.08.006

Stravynski, A., Marks, I., & Yule, W. (1982). Social skills problems in neurotic outpatients: Social skills training with and without cognitive modification. *Archives of General Psychiatry*, *39*(12), 1378–1385. doi:10.1001/archpsyc.1982.04290120014003

Teusch, L., & Böhme, H. (1999). Is the exposure principle really crucial in agoraphobia? The influence of client-centered "nonprescriptive" treatment on exposure. *Psychotherapy Research*, *9*(1), 115–123. doi:10.1093/ptr/9.1.115

Throne, L. C., Bartholomew, J. B., Craig, J., & Farrar, R. P. (2000). Stress reactivity in fire fighters: An exercise intervention. *International Journal of Stress Management*, *7*(4), 235–246. doi:10.1023/A:1009574428627

Turner, S. M., Beidel, D. C., Cooley, M. R., Woody, S. R., & Messer, S. C. (1994). A multicomponent behavioral treatment for social phobia: Social effectiveness therapy. *Behaviour Research and Therapy*, *32*(4), 381–390. doi:10.1016/0005-7967(94)90001-9

Turner, S. M., Beidel, D. C., & Cooley-Quille, M. R. (1995). Two-year follow-up of social phobics treated with social effectiveness therapy. *Behaviour Research and Therapy*, *33*(5), 553–555. doi:10.1016/0005-7967(94)00086-Y

Van Dam-Baggen, R., & Kraaimaat, F. (2000). Group social skills training or cognitive group therapy as the clinical treatment of choice for generalized social phobia? *Journal of Anxiety Disorders*, *14*(5), 437–451. doi:10.1016/S0887-6185(00)00038-4

Van Dyck, R., & Spinhoven, P. (1997). Does preference for treatment matter? A study of exposure in vivo with or without hypnosis in the treatment of panic disorder with agoraphobia. *Behavior Modification*, *21*(2), 172–186. doi:10.1177/01454455970212003

Vervliet, B., Craske, M. G., & Hermans, D. (2013). Fear extinction and relapse: State of the art. *Annual Review of Clinical Psychology*, *9*, 215–248. doi:10.1146/annurev-clinpsy-050212-185542

Vervliet, B., & Raes, F. (2013). Criteria of validity in experimental psychopathology: Application to models of anxiety and depression. *Psychological Medicine*, *43*(11), 2241–2244. doi:10.1017/S0033291712002267

Warnock-Parkes, E., Wild, J., Stott, R., Grey, N., Ehlers, A., & Clark, D. M. (2016). Seeing is believing: Using video feedback in cognitive therapy for social anxiety disorder. *Cognitive and Behavioral Practice*, *24*(2), 245–255. doi:10.1016/j.cbpra.2016.03.007

Weeks, J. W., Heimberg, R. G., Rodebaugh, T. L., & Norton, P. J. (2008). Exploring the relationship between fear of positive evaluation and social anxiety. *Journal of Anxiety Disorders*, *22*(3), 386–400. doi:10.1016/j.janxdis.2007.04.009

Weissman, M. M., Markowitz, J. C., & Klerman, G. L. (2000). *Comprehensive guide to interpersonal psychotherapy*. New York, NY: Basic Books.

Wells, A., & Matthews, G. (1994). Self-consciousness and cognitive failures as predictors of coping in stressful episodes. *Cognition and Emotion*, *8*(3), 279–295. doi:10.1080/02699939408408942

Wheatley, J., & Hackmann, A. (2011). Using imagery rescripting to treat major depression: Theory and practice. *Cognitive and Behavioral Practice*, *18*(4), 444–453. doi:10.1016/j.cbpra.2010.06.004

Williams, J. M., Watts, F. N., MacLeod, C., & Mathews, A. (1997). *Cognitive psychology and emotional disorder*. Chichester, UK: John Wiley & Sons.

Wilson, W. M., & Marsden, C. A. (1996). In vivo measurement of extracellular serotonin in the ventral hippocampus during treadmill running. *Behavioural Pharmacology*, *7*(1), 101–104. doi:10.1097/00008877-199601000-00011

Wlazlo, Z., Schroeder-Hartwig, K., Hand, I., Kaiser, G., & Münchau, N. (1990). Exposure in vivo vs. social skills training for social phobia: Long-term outcome and differential effects. *Behaviour Research and Therapy*, *28*(3), 181–193. doi:10.1016/0005-7967(90)90001-Y

Wolitzky-Taylor, K. B., Horowitz, J. D., Powers, M. B., & Telch, M. J. (2008). Psychological approaches in the treatment of specific phobias: A meta-analysis. *Clinical Psychology Review*, *28*(6), 1021–1037. doi:10.1016/j.cpr.2008.02.007

5

The Psychological Treatment of Generalized Anxiety Disorder

Hanna McCabe-Bennett,[1] Katie Fracalanza,[2] and Martin M. Antony[1]
[1] *Department of Psychology, Ryerson University, Toronto, Canada*
[2] *Department of Psychiatry and Behavioral Sciences, Stanford University, Stanford, USA*

5.1 Features and Prevalence of Generalized Anxiety Disorder

The cardinal feature of generalized anxiety disorder (GAD) is excessive and uncontrollable worry about several life domains, lasting at least 6 months (American Psychiatric Association, 2013). GAD is also marked by at least three associated symptoms, such as feeling keyed up or on edge, difficulty sleeping, fatigue, muscle tension, poor concentration, and irritability (American Psychiatric Association, 2013). GAD is a common and debilitating disorder. The lifetime prevalence of GAD is 5.7% in the United States (Kessler, Berglund, Demler, Jin, & Walters, 2005), and it is associated with lost work productivity, impaired social functioning, and low quality of life (for a review see Hoffman, Dukes, & Wittchen, 2008). Further, individuals with GAD are at increased risk of other psychological disorders (Hoffman et al., 2008) and of physical illnesses, such as chronic migraine, gastrointestinal problems, and cardiac disorders (Härter, Conway, & Merikangas, 2003). Given this, it is unsurprising that GAD is associated with substantial health care costs (e.g., Hoffman et al., 2008). Unfortunately, GAD is a chronic condition, typically unremitting if untreated (e.g., Bruce et al., 2005), and, although disorder-specific treatments lead to recovery for many individuals (approximately 60–70%), a substantial proportion of people still do not attain high end-state functioning with treatment (Hanrahan, Field, Jones, & Davey, 2013).

Given the impairment associated with GAD, there is a need to treat those with pathological worry using evidence-based treatment strategies, and resources that summarize such approaches are needed. The present chapter reviews the evidence for current GAD treatments and their theories, and classifies them according to David and Montgomery's (2011) new evaluative framework for evidence-based psychotherapies (see Table 5.1). This rubric classifies psychotherapies into nine categories, each associated with a different degree of empirical support. The therapies described in the present chapter fall into Categories I through IV. Psychotherapies that have a well-supported treatment package *and* empirically supported theory are considered Category I; psychotherapies with a well-supported treatment package and preliminary theoretical support are classified as

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

Table 5.1 Organization of the theories and treatments reviewed in this chapter by category according to the framework of David and Montgomery (2011).

Theory and Treatment	Category
Intolerance of uncertainty model of GAD and intolerance of uncertainty therapy	I
Metacognitive model of GAD and metacognitive therapy	I
Acceptance-based theory and acceptance-based behavior therapy for GAD	I
Applied relaxation	II
Transdiagnostic model	II
Emotion dysregulation model of GAD and emotion regulation therapy	III
Other mindfulness interventions for GAD	III
Cognitive/contrast avoidance theory of GAD and interpersonal/emotional processing therapy	III
Psychodynamic model and treatments	IV

To the authors' knowledge, there is no available information and there are no protocols that fit Categories V–IX.

Category II; psychotherapies with preliminary research supporting the treatment package and strong theoretical backing fall under Category III; and psychotherapies with preliminary research on the treatment package and theory comprise Category IV. Categories V through IX include psychotherapies that have contradictory evidence in terms of their treatment protocol or theory. There are currently no available theories or protocols regarding the treatment of GAD that would be classified in Categories V through IX. The present chapter concludes with a discussion of future research directions and clinical implications.

5.2 Review of Theory and Evidence Supporting GAD Psychotherapies

5.2.1 Category I

5.2.1.1 Intolerance of uncertainty model of GAD and intolerance of uncertainty therapy

Dugas and colleagues posit that intolerance of uncertainty (IU)—a set of negative beliefs about uncertainty and its implications—is the central cognitive process that underpins excessive worry (Dugas, Gagnon, Ladouceur, & Freeston, 1998). IU is theorized to contribute to uncontrollable worry by making even the slightest chance of a negative event feel unacceptable and upsetting. In other words, high IU leads individuals to interpret unknown outcomes as threatening. Elevated IU is also thought to contribute to GAD indirectly through its connection to other worry-related cognitive processes, including positive beliefs about worry, negative problem orientation, and cognitive avoidance (Dugas et al., 1998). The IU model suggests that positive beliefs about worry prompt an individual to engage in worry more frequently. In addition, a negative attitude toward problems and problem-solving is theorized to interfere with resolving day-to-day difficulties, leading to persistent problems that fuel worry. Finally, the IU model purports

that feeling overwhelmed by uncertainty, an inevitable part of daily life, may lead individuals to engage in cognitive avoidance strategies. Paradoxically, however, suppressing threatening mental imagery may interfere with emotional processing, maintaining anxiety in individuals with GAD (Sibrava & Borkovec, 2006).

Consistent with the IU model of GAD, research has shown that IU, positive beliefs about worry, negative problem orientation, and cognitive avoidance are associated with GAD symptoms, and differentiate people with GAD from nonclinical controls (e.g., Dugas et al., 1998, 2007). In line with the theorized primary role of IU over other cognitive factors in Dugas and colleagues' model of GAD, IU has been found to be more strongly related to GAD severity than other model components (Dugas et al., 1998); however, this is also true of negative problem orientation (Dugas et al., 2007). In addition, one study found that, of the model components, only IU was elevated in individuals with GAD compared to individuals with other anxiety disorders (Ladouceur et al., 1999); however, other studies have found that IU is equally elevated across the anxiety disorders (e.g., Carleton et al., 2012). Despite the possible lack of specificity of IU to GAD, there is ample evidence of a connection between IU and worry. Indeed, IU is more strongly related to worry than are other anxiety-related cognitive processes, such as perfectionism (Buhr & Dugas, 2006) and causal uncertainty (i.e., social/interpersonal uncertainty; Kusec, Tallon, & Koerner, 2016). In addition, studies have shown that people with high IU interpret ambiguous situations as more worrisome than individuals with low IU (Koerner & Dugas, 2008). It also seems that IU and worry change together. When IU is experimentally manipulated, worry levels change in the corresponding direction (e.g., Rosen & Knäuper, 2009). Consistent with this, studies have shown that change in IU and acute cognitive avoidance significantly correspond with improvement in GAD symptoms following imaginal exposure (Fracalanza, Koerner, & Antony, 2014). Further, studies have shown that changes in IU may precede changes in worry over the course of imaginal exposure (Goldman, Dugas, Sexton, & Gervais, 2007) as well as over the course of individual and group cognitive–behavioral therapy (CBT) for GAD (Bomyea et al., 2015; Dugas & Ladouceur, 2000; Torbit & Laposa, 2016). In sum, the evidence to date suggests that IU may be a risk factor for GAD (Koerner & Dugas, 2008).

Dugas and colleagues developed a cognitive–behavioral treatment for GAD based directly on their IU theory, and the treatment is sometimes called intolerance of uncertainty therapy (IUT; Dugas & Robichaud, 2007). IUT begins with psychoeducation, which involves learning about the IU model of worry. Next clients learn to recognize behaviors that they use to avoid or control uncertainty in their lives—for example, avoiding trying new things or overpreparing for everyday events. Clients plan to gradually seek out uncertainty-inducing situations in order to improve their tolerance for uncertainty. Following this, positive beliefs about worry are identified and the accuracy of these beliefs is challenged by evaluating the evidence for and against these beliefs. Next, clients are trained in problem-solving skills and practice applying problem-solving to their current difficulties. Finally, cognitive avoidance is targeted through the use of imaginal exposure, which involves audio-recording a narrative of a client's worst fear(s) coming true and then the client repeatedly listening to the recording in order to "emotionally process" their core fear(s).

Several studies have evaluated the effectiveness of IUT for GAD. First, Ladouceur and colleagues (2000) compared IUT to a waitlist condition. They found that IUT resulted in large, significant improvement in GAD symptoms, depression, and IU, and these

gains were maintained at 6- and 12-month follow-ups. Next, Dugas and colleagues (2003) compared group IUT against a waitlist condition and found similar results. Group IUT led to large, significant improvement in GAD symptoms, depression, IU, and social adjustment at posttreatment. In the IUT group, improvements in depression and social adjustment were maintained at 2-year follow-up, and worry and IU continued to improve from posttreatment to follow-up (Dugas et al., 2003). More recently, Dugas and colleagues (2010) compared IUT to applied relaxation (AR) and a waitlist condition. The authors found that both IUT and applied relaxation led to large, significant improvement in GAD symptoms compared to waitlist, although IUT also led to improvement in global clinical functioning and continued improvement in GAD symptoms from posttreatment to 2-year follow-up, whereas AR did not lead to such effects. Finally, Van der Heiden, Muris, and Van der Molen (2012) compared IUT to another CBT called metacognitive therapy and a waitlist condition. The authors found that both active treatments were effective in producing large, significant improvement in GAD symptoms compared to the waitlist control, and gains were maintained at 6-month follow-up. However, this study also showed that, compared to IUT, metacognitive therapy led to greater reductions in GAD symptoms at posttreatment and 6-month follow-up. Thus, although one study suggests that another disorder-specific CBT produced superior results than IUT, on the whole the evidence suggests that IUT is an effective treatment for GAD. Based on the available evidence, we conclude that IU theory and the corresponding treatment have considerable empirical support, making this a Category I treatment.

5.2.1.2 Metacognitive model of GAD and metacognitive therapy

The metacognitive model theorizes that beliefs about worry are central to the development and maintenance of GAD (see Wells, 2004). The metacognitive model of GAD distinguishes between two forms of worry: type 1 worry, which is worry about the occurrence of negative outcomes in individuals' external world (e.g., being fired, losing a loved one), and type 2 worry, which refers to worry about worry itself, or metaworry (e.g., worry is uncontrollable, worry is dangerous). Wells' theory also proposes that most people hold positive metabeliefs; however, these beliefs can lead a person to more frequently select worry as a strategy to cope with perceived threat. Such an overreliance on worry to cope may lead to negative beliefs about worry (i.e., type 2 worry), and negative metacognitions are thought to be the key maintenance factor in GAD (Wells, 2004). Negative beliefs about worry are thought to lead a person to engage in maladaptive avoidance behaviors and thought control strategies that paradoxically confirm beliefs about the uncontrollability and dangerousness of worry. For example, suppressing thoughts may lead to unintended rebound effects, seemingly confirming the uncontrollability of worry.

In line with the tenets of metacognitive theory, research has demonstrated that, although individuals with GAD, clinical controls, and nonclinical controls all endorse positive beliefs about worry, stronger positive metabeliefs are significantly associated with worry proneness and trait anxiety (Cartwright-Hatton & Wells, 1997). Several studies have produced results consistent with Wells' supposition that negative beliefs about worry have a robust relationship with GAD. Negative beliefs about worry predicted pathological worry over and above positive beliefs about worry (e.g., Penney, Mazmanian, & Rudanycz, 2013) and worry severity (e.g., Nuevo, Montorio, & Borkovec, 2004). Negative metacognitive beliefs also predicted frequency of daily worry over and above IU in a nonclinical sample (Thielsch, Andor, & Ehring, 2015). Further, baseline

negative metacognitions predicted the presence of GAD after 12 to 15 weeks, even after accounting for baseline GAD symptoms and trait anxiety (Nassif, 1999). In addition, Ruscio and Borkovec (2004) found that the severity of negative beliefs about worry differentiated individuals with high worry from individuals with GAD. Studies have also shown that the strength of negative metacognitions discriminates individuals with GAD from individuals with somatic anxiety and nonanxious individuals (Davis & Valentiner, 2000) and that reductions in negative metacognitive beliefs may reduce emotional distress via reductions in worry (McEvoy, Erceg-Hurn, Anderson, Campbell, & Nathan, 2015). In line with this, individuals with GAD have been found to endorse stronger negative beliefs about worry than individuals with panic disorder, social anxiety disorder (SAD), or no anxiety disorder (Wells & Carter, 2001); however, individuals with obsessive–compulsive disorder and GAD have been found to report comparable levels of negative beliefs about worry (Cartwright-Hatton & Wells, 1997).

There are also data supporting the notion that thought suppression is linked to worry. In a nonclinical sample, de Bruin, Muris, and Rassin (2007) found that the tendency to suppress thoughts was a unique predictor of dispositional worry. Consistent with this, when attempting to suppress worrisome thoughts, individuals with GAD reported an increase in the worries that they were trying to suppress compared to when they were attempting to suppress neutral thoughts (Becker, Rinck, Roth, & Margraf, 1998). Similarly, Iijima and Tanno (2012) found evidence of a link between failed thought suppression and an increase in worry, but only in people with high versus low worry. Despite this evidence, McLean and Broomfield (2007) found contradictory evidence suggesting that using thought suppression led to improved beliefs about the controllability of worry. Overall, some studies suggest a link between thought suppression and worry, although additional research is needed to clarify under what circumstances thought suppression leads to worry and the association of these factors to negative beliefs about worry.

Metacognitive therapy (MCT; Wells, 2009) is a form of CBT based directly on the metacognitive model of GAD. First, clients are socialized to the metacognitive model of GAD, and then metacognitive beliefs are targeted. This is done using traditional CBT strategies, such as Socratic questioning and examining the evidence supporting or refuting metacognitive beliefs. More specific to MCT, clients directly test the belief that worry is uncontrollable using worry postponement, a technique that involves delaying worry until a predetermined time each day. In addition, beliefs about the dangerousness of worry are addressed through psychoeducation and the use of behavioral experiments wherein the client deliberately tries to cause harm to him/herself by worrying so as to learn that this is impossible. In MCT, positive beliefs about worry are challenged with worry modulation experiments (i.e., purposely increasing worry to test whether this increases positive outcomes) and the mismatch strategy (i.e., comparing the accuracy of outcomes one predicts when worrying to actual outcomes). Finally, clients are taught to use ways of responding to worry triggers other than worry, such as letting go of thoughts and considering possible positive outcomes.

MCT for GAD has been evaluated in several studies to date. The first open trial showed that MCT led to large, significant improvements in worry, anxiety, and depression from pre- to posttreatment, and these gains were maintained in most people (90%) at 6- and 12-month follow-ups (Wells & King, 2006). Next, Wells and colleagues (2010) tested MCT against AR and found that MCT resulted in significantly larger improvements in worry, anxiety, and metacognitions compared to AR, and these gains were

maintained at 6- and 12-month follow-ups. As discussed in Section 5.2.1.1, MCT has been compared to IUT and a waitlist condition, and the results suggest that MCT was highly effective in improving GAD symptoms, not only compared to waitlist but also compared to IUT (Van der Heiden et al., 2012). Finally, Van der Heiden, Melchior, and de Stigter (2013) evaluated the effects of group MCT and found that it produced large improvements in worry, trait anxiety, and negative beliefs about worry from pre- to post-treatment. Similar outcomes have been reported for a condensed seven-session version of group MCT (McEvoy, Erceg-Hurn, Anderson, Campbell, Swan et al., 2015). Given the research to date, the metacognitive theory and treatment for GAD are well supported, warranting a Category I classification.

5.2.1.3 Acceptance-based theory and acceptance-based behavior therapy for GAD

Roemer and Orsillo (2002) were the first to propose integrating acceptance-based strategies with change-based strategies in the treatment of GAD. This was inspired by Hayes' model of experiential avoidance and rule-governed behavior (e.g., Hayes, Strosahl, & Wilson, 1999), which posits that attempts to control or avoid frightening objects or situations (i.e., by worrying) in turn strengthen one's fear of these stimuli. Drawing on this, Roemer and Orsillo advocate for the use of mindfulness and other acceptance-based strategies in GAD treatment, to help people let go of unhelpful control strategies and experiential avoidance. Mindfulness is a strategy used to help individuals bring their attention to the present and respond to present-moment experiences thoughtfully, intentionally, and nonjudgmentally. Mindful attention to the present moment is incompatible with automatically avoiding uncomfortable internal experiences; thus, in theory it should reduce experiential avoidance and, consequently, reduce worry and anxiety. Roemer and Orsillo also propose that greater engagement with goal-directed behavior (i.e., a change strategy) should help individuals with GAD to focus on positive long-term goals in order to make better decisions in the present moment.

Roemer and Orsillo (2002) recommend mindfulness based on its adoption and success in the treatment of other difficulties, such as borderline personality disorder, wherein mindfulness is one acceptance-based technique that is used in combination with other change-based strategies (Linehan, 1994). Martin (1997) describes mindfulness as a common factor across various therapeutic orientations that involves developing alternative perspectives and becoming less attached to habitual ways of responding. Further, it may be a helpful strategy in improving cognitive flexibility in individuals with GAD (Lee & Orsillo, 2014). Mindfulness has also been suggested as a means of targeting cognitive anxiety through relaxation and stress reduction. Studies have shown that experiential acceptance leads to lowered anxiety and avoidance compared to attempts at experiential control (e.g., Campbell-Sills, Barlow, Brown, & Hofmann, 2006) and that individuals with GAD tend to have lower levels of mindfulness and more difficulty with emotion regulation than nonanxious individuals (Roemer et al., 2009). Mindfulness has been shown to produce greater increases in emotional comprehension as well as somatic and autonomic regulation compared to progressive muscle relaxation in high worriers (Delgado et al., 2010). With respect to the role of mindful action, Michelson, Lee, Orsillo, and Roemer (2011) found that treatment-seeking individuals with GAD reported less behavior that was consistent with their personal values than nonanxious controls, and that valued action was negatively correlated with experiential avoidance and distress about emotions and positively correlated with quality of life.

Based on evidence supporting the emotion dysregulation model of GAD (reviewed in Section 5.2.3.1) and research on the usefulness of acceptance for GAD, Roemer and Orsillo (2002) developed acceptance-based behavioral therapy (ABBT). ABBT integrates techniques from CBT (e.g., Borkovec & Roemer, 1994), acceptance and commitment therapy (e.g., Hayes et al., 1999), dialectical behavior therapy (e.g., Linehan, 1994), and mindfulness (e.g., Hahn, 1976). Roemer and Orsillo (2007) delineate three guiding principles in the development of their treatment: "(a) Expanding present-moment awareness, (b) encouraging acceptance (i.e., a willingness to have one's internal responses in order to participate in meaningful experiences) rather than judgment and avoidance of internal experiences, and (c) promoting action in areas of importance to the individual" (p. 74). Their treatment protocol includes psychoeducation about the function of worry and emotions; mindfulness, early cue detection, and monitoring; relaxation and mindfulness techniques (e.g., progressive muscle relaxation, diaphragmatic breathing); and mindful action (Roemer & Orsillo, 2005).

An initial case series that assessed group ABBT for GAD indicated that three of four patients were considered treatment responders on the majority of outcome measures, including clinician-rated symptom severity and participant-rated worrying, anxiety, depression, and acceptance (Orsillo, Roemer, & Barlow, 2003). Two of four were considered to have high end-state functioning after treatment. In the first open trial of ABBT for GAD, Roemer and Orsillo (2007) reported significant medium to large reductions in overall symptom severity, worry, anxiety, depression, and stress, as well as reductions in experiential avoidance and fear of emotions at posttreatment. A large improvement in quality of life was also observed. All effects were maintained at 3-month follow-up; 50% of the sample was considered recovered and to have high end-state functioning. In a later randomized controlled trial (RCT), GAD symptom severity, worry, stress, depression, experiential avoidance, and fear of emotions all improved significantly in individuals who received ABBT compared to a waitlist condition (Roemer, Orsillo, & Salters-Pedneault, 2008). Additionally, 75% of the treatment group, compared to 8.3% of the waitlist group, were considered responders and met criteria for high end-state functioning at posttreatment. Gains were largely maintained at 3- and 9-month follow-ups. In a further analysis of data from this trial by Hayes, Orsillo, and Roemer (2010), improvements in acceptance and valued action—proposed mechanisms of change in ABBT—were shown to be predictors of treatment response and quality of life posttreatment. Interestingly, increases in decentering, another possible mechanism, are also associated with symptom reduction in GAD (Hayes-Skelton, Calloway, Roemer, & Orsillo, 2015); however, decentering improves to the same degree in both ABBT and AR, suggesting that this mechanism may not be specific to ABBT.

Another trial of ABBT compared to a waitlist control showed that it led to improvements in emotion regulation, fear of emotional responses, IU, and perceived control over anxiety, with gains maintained at 3- and 9-month follow-up (Treanor, Erisman, Salters-Pedneault, Roemer, & Orsillo, 2011). A comparison of ABBT to AR showed that both treatments led to similar gains in clinician-rated GAD severity, anxiety, and decreased number of diagnoses, as well as self-reported worry, tension, anxiety, depression, and quality of life (Hayes-Skelton, Roemer, & Orsillo, 2013). A recent trial of internet-delivered ABBT resulted in improved GAD symptoms with large effect sizes, moderate reductions in depressive symptoms, but no changes in quality of life (Dahlin et al., 2016). Other acceptance-based treatment programs also demonstrate promising

outcomes. For example, a 6-week acceptance and commitment therapy (ACT) group showed similar improvements to a 6-week CBT group in worry, depression, and quality of life; however, the CBT group showed continued improvements following treatment termination whereas the ACT group did not (Avdagic, Morrissey, & Boschen, 2014). We classify ABBT as a Category I psychotherapy based on empirical study of the theoretical usefulness of acceptance-based strategies in GAD (e.g., the emotion dysregulation model of GAD; Mennin, Turk, Heimberg, & Carmin, 2004) and the research support for ABBT protocols.

5.2.2 Category II

5.2.2.1 Applied relaxation

Applied relaxation (AR) for the treatment of GAD was adopted based on the notion that GAD appears to be lacking in external anxiety cues and is therefore more difficult to treat using exposure strategies than are other anxiety disorders (Borkovec et al., 1987). This led researchers to implement AR as a way of targeting potential internal anxiety cues based on the understanding that anxiety is a state of cognitive and somatic arousal. In addition, given that muscle tension is a symptom of GAD, AR is thought to be helpful because it directly targets this aspect of the disorder. AR typically consists of early anxiety cue detection, practicing focusing on feelings of relaxation in the moment, self-control desensitization, and progressive muscle relaxation.

A number of studies have compared the efficacy of AR plus nondirective "talk therapy" with AR plus cognitive therapy or CBT and have found AR to be as efficacious as cognitive therapy and CBT, with all treatments consistently showing similar improvements on measures of overall symptom severity, worry, cognitive and somatic anxiety, and depression at posttreatment and follow-up (Arntz, 2003; Borkovec et al., 1987; Borkovec & Costello, 1993; Öst & Breitholtz, 2000). Some studies suggest that, compared to individuals treated with AR, individuals treated with CBT were more likely to be responders or achieve high end-state functioning; however, AR has shown superior effects to CBT on some outcomes, such as reduced daily anxiety (Borkovec & Costello, 1993). In a meta-analysis conducted by Siev and Chambless (2007), cognitive therapy and AR were shown to be equally effective treatments for GAD. However, more recent studies have shown that disorder-specific CBTs may have some advantages over AR. Dugas et al. (2010) found that an IUT and AR both led to similar improvements on GAD-related outcomes, but only the CBT group showed continued improvement over time. A study of metacognitive therapy showed that it was significantly superior to AR with respect to recovery rates and worry at posttreatment, 6-month follow-ups, and 12-month follow-ups (Wells et al., 2010).

Researchers have begun to investigate potential mechanisms of change in AR. For example, Borkovec et al. (1987) found that relaxation-induced anxiety was associated with poorer outcomes on anxiety and depression measures, suggesting that an inability to relax is closely tied to anxious symptomatology and, as such, reducing physical tension may improve other anxiety symptoms. Conrad, Isaac, and Roth (2008) found that relaxation training did not decrease muscle tension or autonomic activation in individuals with GAD, which was surprising at the time given that the focus of relaxation training is to reduce muscle tension. This finding may be explained by the presence of other possible mediators of change. For example, there appears to be a bidirectional

relationship between improvement in worry and improvement in somatic anxiety that occurs over the course of CBT and AR (e.g., Donegan & Dugas, 2012). In addition, based on their study using a case-series design, Hayes-Skelton, Usmani, Lee, Roemer, and Orsillo (2012) suggested mindfulness, decentering, and acceptance may be possible mechanisms of change in AR. Considering the literature to date, we classify AR as a Category II psychotherapy given that the theory supporting AR is sparse and yet that, despite this, AR is an effective approach to improving GAD symptoms.

5.2.2.2 Transdiagnostic model

Based on suggestions of shared psychopathology and comorbidity among anxiety and depressive disorders, and specifically GAD (Brown, Campbell, Lehman, Grisham, & Mancill, 2001), transdiagnostic approaches that emphasize similarities rather than differences between diagnoses have been gaining in popularity among researchers and clinicians (e.g., Barlow, Allen, Choate, 2004; Norton, 2006). Generally, transdiagnostic treatments have been shown to be superior to waitlist control groups, and they have similar effect sizes as efficacy and effectiveness studies of disorder-specific protocols (McEvoy, Nathan, & Norton, 2009). In a recent RCT that compared the effects of Norton and Hope's (2002) transdiagnostic treatment to relaxation training for people with GAD, no significant differences in session-by-session anxiety were found (Norton, 2012a). Similarly, inferiority analyses revealed no differences between treatment conditions on clinician-rated symptom severity or self-reported depression, but results were inconclusive for self-reported GAD symptoms. Norton and Barrera (2012) conducted a RCT to test the comparative efficacy of diagnosis-specific group CBT for SAD, panic disorder, and GAD versus Norton's (2012b) transdiagnostic anxiety treatment protocol. Noninferiority analyses revealed that Norton's transdiagnostic protocol was not inferior to Dugas and Robichard's (2007) GAD protocol with respect to session-by-session improvement in reduction in GAD and depression symptoms and clinician-rated overall symptom severity from pre- to posttreatment.

In addition, there has been growing interest in therapist-assisted or self-help transdiagnostic treatments delivered online as a way of increasing access to care and easing the demand on clinicians. Newby, Williams, and Andrews (2014) found that an online CBT treatment for mixed GAD and depression significantly reduced repetitive negative thinking, a feature of both GAD and depression (i.e., worry and rumination, respectively), at posttreatment and 3-month follow-up. Open trials and RCTs of Titov and colleagues' therapist-assisted internet-based cognitive–behavioral treatment (ICBT) for anxiety and depressive disorders show that this approach produces significant symptom improvement with medium to large effects at posttreatment and follow-up (e.g., Titov et al., 2011). Titov et al.'s ICBT protocol has also been shown to be helpful for individuals with multiple comorbidities, specifically in producing significant improvement in GAD and depression symptoms (Johnston, Titov, Andrews, Dear, & Spence, 2013). Two RCTs comparing a transdiagnostic internet-based guided self-help program with guided disorder-specific online programs for GAD found no significant differences between the transdiagnostic treatment compared to disorder-specific treatments at posttreatment and 6-month follow-up (Berger, Boettcher, & Caspar, 2014), or between the self-guided and therapist-guided programs (Dear et al., 2015). However, limitations of the online approach may include high dropout and low adherence to the programs (Newby, Mewton, Williams, & Andrews, 2014). Overall, we classify transdiagnostic treatment for

GAD as Category II given that the protocol is effective in improving GAD symptomatology, despite limited theoretical inquiry to date into possible mechanisms of change.

5.2.3 Category III

5.2.3.1 Emotion dysregulation model of GAD and emotion regulation therapy

Mennin and colleagues' (2004) emotion dysregulation theory proposes that individuals with GAD have four interconnected emotion regulation deficits that contribute to and maintain pathological worry. Mennin et al. also suggest that excessive worry leads to further emotion regulation difficulties, creating a feedback loop that maintains GAD. The first tenet of the emotion dysregulation model is that individuals with GAD experience emotions more intensely than the average person and that they react more quickly to perceived threats in their environment and express emotions more often than others. The second tenet is that people with GAD have difficulty identifying and understanding their emotions, making it more challenging for them to use information conveyed by their emotions in an adaptive way. The third tenet is that individuals with GAD tend to become overwhelmed by emotions due to difficulties with heighted emotional intensity and poor emotional understanding, which then leads them to react negatively to their emotional experiences. The fourth tenet is that these difficulties with emotion regulation are all proposed to result in the use of maladaptive coping strategies to modulate emotions (i.e., cognitive control strategies such as worry; Mennin et al., 2004).

Several studies support the proposal that individuals with GAD experience negative emotions more strongly than others, including nonanxious individuals and individuals with SAD or depression (e.g., Decker, Turk, Hess, & Murray, 2008; Mennin, Holaway, Fresco, Moore, & Heimberg, 2007; Turk, Heimberg, Luterek, Mennin, & Fresco, 2005). In addition, there is some support for the notion that people with GAD have greater difficulty understanding their emotions than do healthy controls (Mennin et al., 2007), although this result is inconsistent with those from other studies (Decker et al., 2008). In fact, Mennin, McLaughlin, and Flanagan (2009) found that deficits in understanding emotions may be more strongly related to SAD than to GAD.

Data from several studies demonstrate that individuals with GAD experience more negative reactions to their emotions than do nonanxious individuals and individuals with SAD (e.g., Turk et al., 2005). Indeed, Roemer, Salters, Raffa, and Orsillo (2005) found that negative reactions to emotions successfully classified individuals with and without GAD. On the other hand, some studies have not found differences in negative reactivity to emotions between people with GAD, SAD, or depression (Mennin et al., 2007). With respect to ineffective regulation strategies, Mennin and colleagues (2007) found that the use of maladaptive emotion management significantly predicted GAD, but not SAD or depression. Relatedly, Decker et al. (2008) found that people with GAD were more likely to use emotion regulation strategies (e.g., distraction, hiding/masking emotions, situation selection) than nonanxious controls, although the degree to which such strategies are effective in the long term is unclear. A later study by Aldao and Mennin (2012) showed that, when GAD analogues were instructed to use an adaptive emotion regulation strategy (e.g., cognitive reappraisal), they were able to regulate their emotions to the same extent as nonanxious individuals. Thus, it may be a lack of awareness of effective emotion regulation strategies and not an inability to use such strategies that differentiates people with GAD from others and contributes to

worry. Finally, McLaughlin, Mennin, and Farach (2007) found that individuals with GAD experienced more intense negative emotions in response to an emotion trigger after worrying than they did after relaxing or neutral thinking. This finding suggests that worry may indeed contribute to further emotion dysregulation, in line with the proposed bidirectional relationship between difficulty regulating emotions and worry.

Emotion regulation therapy (ERT; Mennin & Fresco, 2009) was developed to address the vulnerabilities suggested by the emotion dysregulation theory of GAD. Although the techniques used in ERT are broadly aimed at improving emotional awareness and regulation, the strategies do not clearly map onto the four proposed emotional difficulties outlined in the emotion dysregulation model. ERT begins with psychoeducation, in which clients are taught to identify and acknowledge their emotions through self-monitoring. Clients build emotion regulation skills by engaging in mindfulness and progressive muscle relaxation. They also practice accepting emotions, decentering from emotions, and reframing unhelpful cognitions, including negative thoughts about emotional experiences. Clients practice these emotion regulation skills systematically and employ them spontaneously "in the moment" in response to intense emotions. Following this, clients begin experiential exposure with the goal of eliciting and fully experiencing emotions. Experiential exposure is promoted through (1) reducing the use of strategies aimed at controlling emotional experience (e.g., avoidance, reassurance-seeking); (2) systematically engaging in values-consistent behaviors outside of session and feeling associated emotions (e.g., pursuing challenging goals); or (3) imaginal exposure to feared outcomes or potentially rewarding situations with possible negative outcomes (e.g., going on a date). Experiential exposure is also facilitated by two-chair dialogue, borrowed from emotion-focused therapy, which involves exploring competing motivations for action (i.e., need for security vs. need for reward).

The effects of ERT have only recently been tested. The first open trial of ERT showed that it led to large, significant improvement in GAD symptoms, trait anxiety, depression, and quality of life, and these gains remained at 9-month follow-up (Mennin & Fresco, 2011). Similarly, a recent RCT showed that ERT produced significant medium to large improvements in GAD symptoms, trait anxiety, depression, quality of life, and role functioning compared to a modified attention control condition, and these gains were maintained at 9-month follow-up (Mennin, Fresco, Hiemberg, & Ciesla, 2012). In addition, an open trial for individuals with comorbid GAD and depression demonstrated large effect sizes for GAD and depression severity ratings and self-reported anxiety at posttreatment, 3-month follow-up, and 9-month follow-up (Mennin, Fresco, Ritter, & Heimberg, 2015). In sum, the emotion dysregulation theory of GAD has substantial empirical support, and the early stages of testing ERT suggest that this approach can be helpful for people with GAD. However, results from only one open trial of ERT have been disseminated in peer-reviewed scientific journals, the rest having been presented only at scientific conferences. In addition, they have not been replicated by independent research teams. As such, this psychotherapy is classified as Category III.

5.2.3.2 Other mindfulness interventions for GAD

Given the move toward including mindfulness strategies in the treatment of GAD as outlined in Section 5.2.1.3, the efficacy of mindfulness-based cognitive therapy (MBCT) and mindfulness-based stress reduction (MBSR) has also been examined. Mindful meditation and developing a present-focused, nonjudgmental acceptance of experience

are central components of both MBCT and MBSR, which are delivered in group format. MBCT for GAD has been shown to decrease anxiety, worry, and depression with gains largely maintained at 6-week and 3-month follow-up in two open trials (Craigie, Rees, Marsh, & Nathan, 2008; Evans et al., 2008). In addition, results from two RCTs have shown MBCT to be efficacious at reducing anxiety symptoms when used as an adjunct to pharmacotherapy (Kim et al., 2009; Lee et al., 2007). MBSR produced significantly greater reductions in anxiety and stress reactivity, and significantly improved positive self-statements, compared to stress management education (Hoge et al., 2013). MBSR also reduced hormonal and immunological stress markers (Hoge et al., 2017). Although MBSR has been shown to elicit improvements in anxiety and depression with effect sizes ranging from small to large, mediation analyses have failed to demonstrate that increased mindfulness is a significant mediator of these effects (Vollestad, Sivertsen, & Neilsen, 2011). Rather, changes in decentering but not in mindfulness have been reported as significant mediators of symptom improvement in MBSR (Hoge et al., 2015). Furthermore, although MBCT appears to be superior to treatment as usual for GAD, it is not superior to psychoeducation in its effect on worry symptoms (Wong et al., 2016). In sum, we classify other mindfulness interventions for GAD as Category III given the evidence supporting the use of mindfulness approaches for GAD reviewed elsewhere in this chapter and preliminary evidence that mindfulness may be a helpful strategy in and of itself. However, there is a need for further investigation of mindfulness-based treatment in the absence of change strategies compared to other disorder-specific treatments for GAD.

5.2.3.3 Cognitive/contrast avoidance theories of GAD and interpersonal/emotional processing therapy

5.2.3.3.1 Cognitive avoidance theory

The cognitive avoidance theory of worry, which stems from research on the maladaptive effects of thought suppression (Roemer & Borkovec, 1994), states that worry is an avoidance strategy that maintains anxiety and promotes future worry episodes. According to this theory, worry is used to mentally prepare for possible future threats while simultaneously avoiding certain aspects of anxiety in the moment. This theory suggests that, by engaging in worry in response to threat cues, individuals may avoid clear mental images of the scenarios that they fear and may reduce somatic anxious arousal. The avoidance of threatening imagery and associated emotional arousal may maintain worry by preventing full emotional processing of fears. The cognitive avoidance model also posits that individuals with GAD hold positive beliefs about worry that preserve the occurrence of worry. This theory suggests several potential origins of pathological worry, including IU (reviewed in Section 5.2.1.1), insecure attachments in childhood, and interpersonal problems in adulthood (Sibrava & Borkovec, 2006).

Basic research on anxiety and worry supports several tenets of this theory. Typical GAD worries are verbal–linguistic rather than imagery-based (e.g., Hirsch, Hayes, Mathews, Perman, & Borkovec, 2012) and are more abstract than concrete in nature (Stöber, Tepperwien, & Staak, 2000). Both the verbal and abstract qualities of worry may allow people to dampen emotional arousal when considering future threats. Indeed, thoughts of feared scenarios are associated with less physiological arousal than are mental images of the same scenarios (Vrana, Cuthbert, & Lang, 1986). Research has also shown that worriers tend to avoid aversive imagery to a greater extent than do

nonworriers (Laguna, Ham, Hope, & Bell, 2004), presumably to avoid anxious arousal. In addition, compared to worrying in words, worrying in images has been found to have a stronger relationship with attentional biases to threat and increased negative intrusions (Williams, Mathews, & Hirsch, 2014), which may contribute to a sense of worry being uncontrollable (Stokes & Hirsch, 2010). The abstract quality of worry has similarly been shown to mitigate anxiety in the moment but may serve to increase anxiety in the longer term (Behar et al., 2012). Further, there is evidence that, when individuals consider their worst-case scenarios in detail and evoke associated negative feelings, worry and acute cognitive avoidance improve (e.g., Fracalanza et al., 2014). Research has also shown that worriers hold positive beliefs about the function of worry—for example, that worry is a helpful problem-solving strategy or that worry reduces the likelihood that negative events will occur (Freeston, Rhéaume, Letarte, Dugas, & Ladouceur, 1994). Stefan and David (2013) found that, although high worriers tend to endorse positive beliefs about worry, their predictions about the benefits of worrying do not materialize.

With respect to childhood attachment and adult interpersonal difficulties, some researchers have identified several problematic interpersonal subtypes within GAD (e.g., overly nurturant, intrusive, socially avoidant, and nonassertive; Salzer, Pincus, Winkelbach, Leichsenring, & Leibing, 2011), whereas others have found that individuals with GAD do not have greater interpersonal problems compared to people with posttraumatic stress disorder, other anxiety disorders, or depression (Uhmann, Beesdo-Baum, Becker, & Hoyer, 2010). Eng and Heimberg (2006) found that an analogue GAD sample reported elevated interpersonal problems but that these reports were not corroborated by their friends. This suggests that, although individuals with GAD may worry more about interpersonal situations, they may not actually have social difficulties. Eng and Heimberg also found that a GAD analogue sample reported less secure attachment to parents than a control sample. Indeed, poor self-reported maternal attachment in childhood predicts GAD status (Cassidy, Lichtenstein-Phelps, Sibrava, Thomas, & Borkovec, 2009). However, in a 5-year longitudinal study of adolescent GAD, worry was a significant predictor of perceived negative parental behavior, whereas perceived parental behavior did not predict future worry (Hale, Klimstra, Branje, Wijsbroek, & Meeus, 2013). This finding suggests that childhood attachment may not be any worse for individuals who develop GAD but that GAD worries may influence the perception of parental attachment or play a role in the onset of negative parental behaviors. Overall, research to date supports several but not all aspects of the cognitive avoidance theory of worry.

5.2.3.3.2 Contrast avoidance theory

The original *cognitive* avoidance theory has since been revised in the *contrast* avoidance model of GAD to address those areas of the former theory that have questionable support (Newman & Llera, 2011). For example, worry may not actually serve to help people avoid negative emotions; rather, some studies have shown that worry is associated with increased arousal and negative emotionality (see Brosschot, Gerin, & Thayer, 2006). As such, the contrast avoidance theory suggests that worry may be used to avoid unpleasant *shifts* in emotions rather than unpleasant emotions in and of themselves. This theory suggests that individuals with GAD prefer to endure a chronic state of negative emotion rather than risk experiencing a shift from a positive or neutral state to a negative one. Several studies have shown that individuals with GAD with higher baseline

physiological anxious arousal demonstrate attenuated increases in anxious arousal in response to stressful cues compared to those with lower baseline arousal (Fisher, Granger, & Newman, 2010; Fisher & Newman, 2013). In an experiment that tested this new model, Llera and Newman (2010) found that worry allowed participants to avoid feeling increases in negative emotion rather than avoiding experiencing negative emotion altogether. A later study by Llera and Newman (2014) similarly found that individuals with GAD prefer to remain in a negative emotion state rather than experiencing negative emotion shifts, whereas nonanxious individuals prefer the opposite. Llera and Newman also found that participants with GAD reported worrying as being a more helpful strategy than did their nonanxious counterparts. Furthermore, they showed that individuals in a worry condition experienced a greater decrease in negative emotion compared to those in other conditions following exposure to a positive stimulus (i.e., a humorous film clip). These findings have been bolstered by examining the relationships between worry and experience of negative emotional contrasts in a naturalistic prospective study. Crouch, Lewis, Erickson, and Newman (2017) found that higher baseline GAD symptoms predicted greater emotional contrasts during negative events in daily living. In addition, higher daily worry moderated the relationship between emotional contrasts and negative emotion, suggesting that worry does indeed have a buffering effect to negative emotional shifts. In sum, the contrast avoidance model suggests that individuals with GAD may worry not to avoid negative emotions but, instead, to avoid experiencing such emotions unexpectedly, and this appears to be supported by empirical evidence.

5.2.3.3.3 Interpersonal/emotional processing therapy

Newman, Castonguay, Borkovec, and Molnar (2004) outline an integrative psychotherapy called interpersonal/emotional processing (I/EP) therapy based on the original cognitive avoidance model of worry. Using I/EP, the therapeutic hour is extended to 2 standard therapy hours that include 55 consecutive minutes of CBT and 55 consecutive minutes of I/EP. Newman et al.'s integrative psychotherapy targets four difficulties: (1) current relational styles, (2) the origins of relational difficulties, (3) relationship problems that arise in the therapeutic relationship, and (4) emotional avoidance. The CBT portion of the treatment protocol includes psychoeducation about anxiety, cognitive restructuring, and AR training. This CBT treatment has previously been found to be efficacious in treating GAD (Borkovec & Costello, 1993; Borkovec, Newman, Pincus, & Lytle, 2002).

The I/EP portion of this treatment approach, which is modeled after Safran and Segal's (1990) model of interpersonal schema therapy, includes psychoeducation about the negative effects of avoiding emotions and social situations (Newman et al., 2004). Specific I/EP techniques include exploring past and current relationships and specific interpersonal difficulties, and teaching clients alternative strategies for dealing with problematic interpersonal patterns using social skills training and role-plays. Therapists are also instructed to monitor the ways in which the client's interpersonal behaviors influence their own responses, to resist impulses to respond negatively to these behaviors, and to discuss these issues with the client. This serves to illuminate how the client's behavior impacts the behavior of those around him or her. Emotional deepening is proposed to occur when therapists make note of emotional markers (e.g., change in voice quality, tears) and ask clients to experience the current emotion. This can be done using experiential techniques such as a two-chair exercise, an empty-chair exercise, or systematic

evocative unfolding. Homework is integrated into both the CBT and the I/EP portions of the treatment.

In an open trial of this integrated protocol, 83.3% of participants were considered responders at the end of treatment, falling to 58.8% at 6-month follow-up but increasing to 76.5% at 1-year follow-up (Newman, Castonguay, Borkovec, Fisher, & Nordberg, 2008). Responder status was defined as a 20% change in scores from pre- to posttreatment on at least four of six primary outcome measures: clinician-rated GAD severity and overall anxiety, and participant-rated daily anxiety, trait anxiety, fear of relaxation, and worry. This is in comparison with 66.7% of participants in a CBT plus supportive listening condition who were responders at posttreatment, which fell to 33.3% at 6-month follow-up and remained at this level at 1-year follow-up. Effect sizes on combined severity and anxiety ratings for CBT plus I/EP were 3.15 at posttreatment and 2.97 at 1-year follow-up. The results of this open trial show promise for the inclusion of the I/EP component. However, in a follow-up RCT, no significant differences were found between CBT plus I/EP and CBT plus supportive listening at posttreatment or 2-year follow-up, with an effect size of 1.86 showing a nonsignificant decrease in GAD symptoms in both conditions (Newman et al., 2011). Participants in both treatments showed significant improvements over time. Diagnostic criteria for GAD were no longer met at 2-year follow-up for 75% of those in the CBT plus I/EP condition and 63.6% of those in the CBT plus supportive listening condition. These findings call into question the utility of adding an I/EP component to existing CBT protocols and open the door for other treatments that may differentially target the problematic avoidance in GAD.

Driven by the failure of the integrative treatment protocol to increase response rates in patients with GAD, Newman and Llera (2011) suggested several adaptations to I/EP treatment based on their updated contrast avoidance theory. They propose using graduated exposure techniques to address feared and avoided outcomes (rather than anxious triggers) to promote reduction of anxiety in response to emotional shifts. They also propose adopting a focus on distress tolerance as opposed to distress diminishment, as it seems that inability to tolerate increases in negative emotions may be underlying the need to worry perpetually. However, no treatment protocol has yet been developed or studied based on this revised model.

To summarize, there is empirical support for several tenets of the cognitive avoidance theory of worry, including the notions that worry is verbal–linguistic in nature, that people with GAD may avoid considering scenarios that they fear clearly, and that individuals with GAD have positive beliefs about worry. The data also support extensions of this theory in that worry does seem to maintain a negative emotional state in individuals with GAD, preventing sudden shifts in emotion. Additional research is needed to clarify the proposed extensions to I/EP therapy based on the contrast avoidance theory and to examine its comparability to other disorder-specific CBTs. For these reasons, I/EP is classified as a Category III psychotherapy for GAD, though it is expected to show promise as an approach to treating GAD.

5.2.4 Category IV

5.2.4.1 Psychodynamic model and treatments

The supportive–expressive (SE) psychodynamic model of GAD draws on the interpersonal and attachment concerns suspected to be present in GAD (reviewed in

Section 5.2.3.3.1) as well as research suggesting that individuals with GAD report traumatic experiences to a greater extent than do nonanxious individuals (Borkovec, 1994). Perhaps most relevant to the psychodynamic model of GAD is the finding that, even though on average people with GAD are more likely to have experienced traumatic events than nonanxious individuals, those with GAD actually worry less about such traumas than their nonanxious counterparts (Borkovec, 1994). According to psychodynamic theorists, this suggests that worrying may be a defense mechanism used to protect the individual from emotionally disturbing material (Crits-Christoph, 2002; Crits-Christoph, Connolly, Azarian, Crits-Christoph, & Shappell, 1996). This is consistent with previous research on the avoidance function of worry (see Section 5.2.1.1, Section 5.2.1.2, and Section 5.2.1.3).

Crits-Christoph and colleagues (1995) developed a 16-session SE manual specifically for GAD based on Luborsky's (1984) general SE model. SE treatment for GAD produced significant improvements in anxiety, depression, worry, and interpersonal problems in an open trial (Crits-Christoph et al., 1996). Leichsenring et al. (2009) conducted an RCT comparing a 30-session modified version of SE with a 30-session CBT treatment. They found that both groups experienced significant improvements in anxiety, worry, depression, and interpersonal problems at posttreatment and 6-month follow-up. However, the CBT group showed significantly greater improvements in trait anxiety, worry, and depression. These patterns were maintained at 12-month follow-up (Salzer, Winkelbach, Leweke, Leibing, & Leichsenring, 2011). These results indicate that, although both treatments appear to be efficacious, CBT produced superior results.

The presence of major depressive disorder or of certain personality disorder comorbidities[1] is associated with worse outcomes in SE psychotherapy, and more severe interpersonal problems (specifically interpersonal styles characterized as overly nurturing) predict worse outcomes (Crits-Christoph et al., 2004). Crits-Christoph et al. (2004) showed that positive expectations about psychotherapy were related to better treatment outcomes. These authors also examined which of the following factors were most predictive of posttreatment worry: comorbid personality or depressive disorders, an overly nurturing interpersonal style, or expectancies for treatment; they found that an overly nurturing personality style was the only significant unique predictor. The findings from this study provide some insight into who might benefit from SE psychotherapy. Although SE psychotherapy has been shown to be superior to supportive therapy in reducing GAD symptoms, it has not been shown to improve overly nurturant interpersonal problems (Crits-Christoph, Gibbons, Narducci, Schamberger, & Gallop, 2005), a proposed maintenance factor in this model.

Ferrero et al. (2007) conducted a study comparing the effects of 10–15 sessions of brief Adlerian psychodynamic psychotherapy (B-APP) to medication treatment and to combined medication plus B-APP. They found that the B-APP, medication, and combined treatment groups all showed significantly improved anxiety, depression, overall severity and functioning at posttreatment, 6-month follow-up, and 1-year follow-up. The effects for all three groups were medium to large at posttreatment and large at 6-month and 1-year follow-ups. In addition, there were no significant differences between the groups in terms of remission rates (63–78%) at 1-year follow-up. It is also of note that 28.7% of participants in Ferrero et al.'s study had a comorbid personality disorder[2] and that individuals with a comorbid personality disorder fared worse if they were in the medication-only group compared to the B-APP or combined treatment groups.

5.3 Implications for Research

One priority area in GAD research is the need for independent research groups to replicate current findings. For the most part, each psychotherapy for GAD is tested by a single research team. Although these programs of research provide compelling evidence for the respective models and treatments, their findings must be replicated in order for the wide adoption of their proposed psychotherapies to be confidently undertaken. Research on specific mechanisms of change within each treatment should also be a focus of future research. Furthermore, it remains unclear whether certain treatments are more effective for possible GAD subtypes. For example, do all individuals with GAD experience emotion dysregulation, or is this more pronounced for a subset of the clinical population? If this phenomenon occurs in only a subset of individuals, then perhaps emotion regulation skills would benefit those individuals more than they would other individuals for whom emotion dysregulation is less of a problem. In other words, more research is required to determine which treatments work best for whom. Tailoring the type of treatment to client-specific personality or behavioral presentations may improve outcomes in this population. In addition, the promising findings thus far regarding online interventions for GAD suggest that this line of research should be further pursued, as it may provide an opportunity for larger number of individuals with GAD to receive evidence-based assistance.

5.4 Implications for Practice

Although more research is needed, the available evidence on GAD treatments reviewed in this chapter suggests that some treatments and associated theories have more thorough support than others. For example, psychodynamic treatments appear to be less efficacious than CBT-based treatments for this population. In general, although the Category I psychotherapies for GAD appear to be highly efficacious, studies on these treatments require replication by independent research teams. Given the paucity of empirical inquiry about which evidence-based treatments are best suited to particular clients, clinicians may wish to select from among the treatments classified as Category I in this chapter based on their own case conceptualization of the most salient maintenance factors (e.g., IU, metacognitive beliefs, emotional avoidance) for a particular client.

5.5 Conclusions

Despite the vast amount of research that has been conducted to date on various models of and treatments for GAD, it remains a difficult diagnosis to treat effectively, and there remains room for improvement with respect to treatment outcomes for this population. The treatments described in the present chapter are intended to provide a starting point for clinicians and researchers with respect to available psychotherapies that may be effective for people with GAD. However, additional research is required to provide clearer guidelines on how to optimally assist individuals who suffer with unrelenting worry.

Notes

1 In Crits-Christoph et al.'s (2004) study, participants were excluded if diagnostic criteria were met for schizoid, schizotypal, paranoid, or borderline personality disorder.
2 Ferrero et al. (2007) specify that these participants met diagnostic criteria for cluster B personality disorders ($n = 5$) or cluster C personality disorders ($n = 20$) based on DSM-IV-TR classifications (American Psychiatric Association, 2000).

References

Aldao, A., & Mennin, D. S. (2012). Paradoxical cardiovascular effects of implementing adaptive emotion regulation strategies in generalized anxiety disorder. *Behaviour Research and Therapy*, *50*, 122–130. doi:10.1016/j.brat.2011.12.004

Arntz, A. (2003). Cognitive therapy versus applied relaxation as treatment of generalized anxiety disorder. *Behaviour Research and Therapy*, *41*, 633–646. doi:10.1016/s0005-7967(02)00045-1

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed. TR). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.

Avdagic, E., Morrissey, S. A., & Boschen, M. J. (2014). A randomised controlled trial of acceptance and commitment therapy and cognitive-behaviour therapy for generalised anxiety disorder. *Behaviour Change*, *31*, 110–130. doi:10.1017/bec.2014.5

Barlow, D. H., Allen, L. B., & Choate, M. L. (2004). Toward a unified treatment for emotional disorders. *Behavior Therapy*, *35*, 205–230. doi:10.1016/s0005-7894(04)80036-4

Becker, E. S., Rinck, M., Roth, W. T., & Margraf, J. (1998). Don't worry and beware of white bears: Thought suppression in anxiety patients. *Journal of Anxiety Disorders*, *12*, 39–55. doi:10.1016/s0887-6185(97)00048-0

Behar, E., McGowan, S. K., McLaughlin, K. A., Borkovec, T. D., Goldwin, M., & Bjorkquist, O. (2012). Concreteness of positive, negative, and neutral repetitive thinking about the future. *Behavior Therapy*, *43*, 300–312. doi:10.1016/j.beth.2011.07.002

Berger, T., Boettcher, J., & Caspar, F. (2014). Internet-based guided self-help for several anxiety disorders: A randomized controlled trial comparing a tailored with a standardized disorder-specific approach. *Psychotherapy*, *51*, 207–219. doi:10.1037/a0032527

Bomyea, J., Ramsawh, H., Ball, T. M., Taylor, C. T., Paulus, M. P., Lang, A. J., & Stein, M. B. (2015). Intolerance of uncertainty as a mediator of reductions in worry in a cognitive behavioral treatment program for generalized anxiety disorder. *Journal of Anxiety Disorders*, *33*, 90–94. doi:10.1016/j.janxdis.2015.05.004

Borkovec, T. D. (1994). The nature, functions, and origins of worry. In G. C. L. Davey & F. Tallis (Eds.), *Worrying: Perspectives on theory, assessment, and treatment* (pp. 5–33). Chichester, UK: John Wiley & Sons.

Borkovec, T. D., & Costello, E. (1993). Efficacy of applied relaxation and cognitive-behavioral therapy in the treatment of generalized anxiety disorder. *Journal of Consulting and Clinical Psychology*, *61*, 611–619. doi:10.1037//0022-006x.61.4.611

Borkovec, T. D., Mathews, A. M., Chambers, A., Ebrahimi, S., Lytle, R., & Nelson, R. (1987). The effects of relaxation training with cognitive or nondirective therapy and the role of relaxation-induced anxiety in the treatment of generalized anxiety. *Journal of Consulting and Clinical Psychology*, *55*, 883–888. doi:10.1037//0022-006x.55.6.883

Borkovec, T. D., Newman, M. G., Pincus, A. L., & Lytle, R. (2002). A component analysis of cognitive-behavioral therapy for generalized anxiety disorder and the role of interpersonal problems. *Journal of Consulting and Clinical Psychology*, *70*, 288–298. doi:10.1037//0022-006x.70.2.288

Borkovec, T. D., & Roemer, L. (1994). Generalized anxiety disorder. In R. T. Ammerman & M. Hersen (Eds.), *Handbook of prescriptive for adults* (pp. 261–281). New York, NY: Plenum Press.

Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research*, *60*, 113–124. 0.1016/j.jpsychores.2005.06.074

Brown, T. A., Campbell, L. A., Lehman, C. L., Grisham, J. R., & Mancill, R. B. (2001). Current and lifetime comorbidity of the DSM-IV anxiety and mood disorders in a large clinical sample. *Journal of Abnormal Psychology*, *110*, 585–599. doi:10.1037//0021-843x.110.4.585

Bruce, S. E., Yonkers, K. A., Otto, M. W., Eisen, J. L., Weisberg, R. B., Pagano, M., … Keller, M. B. (2005). Influence of psychiatric comorbidity on recovery and recurrence in generalized anxiety disorder, social phobia, and panic disorder: A 12-year prospective study. *American Journal of Psychiatry*, *162*, 1179–1187. doi:10.1176/appi.ajp.162.6.1179

Buhr, K., & Dugas, M. J. (2006). Investigating the construct validity of intolerance of uncertainty and its unique relationship with worry. *Journal of Anxiety Disorders*, *20*, 222–236. doi:10.1016/j.janxdis.2004.12.004

Campbell-Sills, L., Barlow, D. H., Brown, T. A., & Hofmann, S. G. (2006). Effects of suppression and acceptance on emotional responses of individuals with anxiety and mood disorders. *Behaviour Research and Therapy*, *44*, 1251–1263. doi:10.1016/j.brat.2005.10.001

Carleton, R. N., Mulvogue, M. K., Thibodeau, M. A., McCabe, R. E., Antony, M. M., & Asmundson, G. J. G. (2012). Increasingly certain about uncertainty: Intolerance of uncertainty across anxiety and depression. *Journal of Anxiety Disorders*, *26*, 468–479. doi:10.1016/j.janxdis.2012.01.011

Cartwright-Hatton, S., & Wells, A. (1997). Beliefs about worry and intrusions: The meta-cognitions questionnaire and its correlates. *Journal of Anxiety Disorders*, *11*, 279–296. doi:10.1016/s0887-6185(97)00011-x

Cassidy, J., Lichtenstein-Phelps, J., Sibrava, N. J., Thomas, C. L., Jr., & Borkovec, T. D. (2009). Generalized anxiety disorder: Connections with self-reported attachment. *Behavior Therapy*, *40*, 23–38. doi:10.1016/j.beth.2007.12.004

Conrad, A., Isaac, L., & Roth, W. T. (2008). The psychophysiology of generalized anxiety disorder: 2. Effects of applied relaxation. *Psychophysiology*, *45*, 377–388. doi:10.1111/j.1469-8986.2007.00644.x

Craigie, M. A., Rees, C. S., Marsh, A., & Nathan, P. (2008). Mindfulness-based cognitive therapy for generalized anxiety disorder: A preliminary evaluation. *Behavioural and Cognitive Psychotherapy*, *36*, 553–568. doi:10.1017/s135246580800458x

Crits-Christoph, P. (2002). Psychodynamic-interpersonal treatment of generalized anxiety disorder. *Clinical Psychology: Science and Practice*, *9*, 81–84. doi:10.1093/clipsy/9.1.81

Crits-Christoph, P., Connolly, M. B., Azarian, K., Crits-Christoph, K., & Shappell, S. (1996). An open trial of brief supportive-expressive psychotherapy in the treatment of generalized anxiety disorder. *Psychotherapy*, *33*, 418–430. doi:10.1037/0033-3204.33.3.418

Crits-Christoph, P., Crits-Christoph, K., Wolf-Palacio, D., Fichter, R., & Rudick, D. (1995). Supportive-expressive dynamic psychotherapy for generalized anxiety disorder. In J. P. Barger & P. Crits-Christoph (Eds.), *Dynamic therapies for psychiatric disorders: Axis I* (pp. 43–83). New York, NY: Basic Books.

Crits-Christoph, P., Gibbons, M. B. C., Losardo, D., Narducci, J., Schamberger, M., & Gallop, R. (2004). Who benefits from brief psychodynamic therapy for generalized anxiety disorder? *Canadian Journal of Psychoanalysis*, *12*, 301–324.

Crits-Christoph, P., Gibbons, M. B. C., Narducci, J., Schamberger, M., & Gallop, R. (2005). Interpersonal problems and the outcome of interpersonally oriented psychodynamic treatment of GAD. *Psychotherapy*, *42*, 211–224. 0.1037/0033-3204.42.2.211

Crouch, T. A., Lewis, J. A., Erickson, T. M., & Newman, M. G. (2017). Prospective investigation of the contrast avoidance model of generalized anxiety and worry. *Behavior Therapy*, *48*(4), 544–556. doi:10.1016/j.beth.2016.10.001

Dahlin, M., Andersson, G., Magnusson, K., Johansson, T., Sjögren, J., Håkansson, A., … Carlbring, P. (2016). Internet-delivered acceptance-based behaviour therapy for generalized anxiety disorder: A randomized controlled trial. *Behaviour Research and Therapy*, *77*, 86–95. doi:10.1016/j.brat.2015.12.007

David, D., & Montgomery, G. H. (2011). The scientific status of psychotherapies: A new evaluative framework for evidence-based psychosocial interventions. *Clinical Psychology: Science and Practice*, *18*(2), 89–99. doi:10.1111/j.1468-2850.2011.01239.x

Davis, R. N., & Valentiner, D. P. (2000). Does meta-cognitive theory enhance our understanding of pathological worry and anxiety? *Personality and Individual Differences*, *29*, 513–526. doi:10.1016/s0191-8869(99)00211-1

Dear, B. F., Staples, L. G., Terides, M. D., Karin, E., Zou, J., Johnston, L., … Titov, N. (2015). Transdiagnostic versus disorder-specific and clinician-guided versus self-guided internet-delivered treatment for generalized anxiety disorder and comorbid disorders: A randomized controlled trial. *Journal of Anxiety Disorders*, *36*, 63–77. doi:10.1016/j.janxdis.2015.09.003

de Bruin, G. O., Muris, P., & Rassin, E. (2007). Are there specific meta-cognitions associated with vulnerability to symptoms of worry and obsessional thoughts? *Personality and Individual Differences*, *42*, 689–699. doi:10.1016/j.paid.2006.08.015

Decker, M. L., Turk, C. L., Hess, B., & Murray, C. E. (2008). Emotion regulation among individuals classified with and without generalized anxiety disorder. *Journal of Anxiety Disorders*, *22*, 485–494. doi:10.1016/j.janxdis.2007.04.002

Delgado, L. C., Guerra, P., Perakakis, P., Vera, M. N., del Paso, G. R., & Vila, J. (2010). Treating chronic worry: Psychological and physiological effects of a training programme based on mindfulness. *Behaviour Research and Therapy*, *48*, 873–882. doi:10.1016/j.brat.2010.05.012

Donegan, E., & Dugas, M. J. (2012). Generalized anxiety disorder: A comparison of symptom change in adults receiving cognitive-behavioral therapy or applied relaxation. *Journal of Consulting and Clinical Psychology*, *80*, 490–496. doi:10.1037/a0028132

Dugas, M. J., Brillon, P., Savard, P., Turcotte, J., Gaudet, A., Ladouceur, R., … Gervais, N. J. (2010). A randomized clinical trial of cognitive-behavioral therapy and applied relaxation

for adults with generalized anxiety disorder. *Behavior Therapy*, *41*, 46–58. doi:10.1016/j.beth.2008.12.004

Dugas, M. J., Gagnon, F., Ladouceur, R., & Freeston, M. H. (1998). Generalized anxiety disorder: A preliminary test of a conceptual model. *Behaviour Research and Therapy*, *36*, 215–226. doi:10.1016/s0005-7967(97)00070-3

Dugas, M. J., & Ladouceur, R. (2000). Treatment of GAD: Targeting intolerance of uncertainty in two types of worry. *Behavior Modification*, *24*, 635–657. doi:10.1177/0145445500245002

Dugas, M. J., Ladouceur, R., Léger, E., Freeston, M. H., Langolis, F., Provencher, M. D., & Boisvert, J. (2003). Group cognitive-behavioral therapy for generalized anxiety disorder: Treatment outcome and long-term follow-up. *Journal of Consulting and Clinical Psychology*, *71*, 821–825. doi:10.1037/0022-006x.71.4.821

Dugas, M. J., & Robichaud, M. (2007). *Cognitive-behavioral treatment for generalized anxiety disorder: From science to practice*. New York, NY: Routledge.

Dugas, M. J., Savard, P., Gaudet, A., Turcotte, J., Laugesen, N., Robichaud, M., … Koerner, N. (2007). Can the components of a cognitive model predict the severity of generalized anxiety disorder? *Behavior Therapy*, *38*, 169–178. doi:10.1016/j.beth.2006.07.002

Eng, W., & Heimberg, R. G. (2006). Interpersonal correlates of generalized anxiety disorder: Self versus other perception. *Journal of Anxiety Disorders*, *20*, 380–387. doi:10.1016/j.janxdis.2005.02.005

Evans, S., Ferrando, S., Findler, M., Stowell, C., Smart, C., & Haglin, D. (2008). Mindfulness-based cognitive therapy for generalized anxiety disorder. *Journal of Anxiety Disorders*, *22*, 716–721. doi:10.1016/j.janxdis.2007.07.005

Ferrero, A., Pierò, A., Fassina, S., Massola, T., Lanteri, A., Daga, G. A., & Fassino, S. (2007). A 12-month comparison of brief psychodynamic psychotherapy and pharmacotherapy treatment in subjects with generalised anxiety disorders in a community setting. *European Psychiatry*, *22*, 530–539. doi:10.1016/j.eurpsy.2007.07.004

Fisher, A. J., Granger, D. A., & Newman, M. G. (2010). Sympathetic arousal moderates self-reported physiological arousal symptoms at baseline and physiological flexibility in response to a stressor in generalized anxiety disorder. *Biological Psychology*, *83*, 191–200. doi:10.1016/j.biopsycho.2009.12.007

Fisher, A. J., & Newman, M. G. (2013). Heart rate and autonomic response to stress after experimental induction of worry versus relaxation in healthy, high-worry, and generalized anxiety disorder individuals. *Biological Psychology*, *93*, 65–74. doi:10.1016/j.biopsycho.2013.01.012

Fracalanza, K., Koerner, N., & Antony, M. M. (2014). Testing a procedural variant of written imaginal exposure for generalized anxiety disorder. *Journal of Anxiety Disorders*, *28*, 559–569. doi:10.1016/j.janxdis.2014.05.011

Freeston, M. H., Rhéaume, J., Letarte, H., Dugas, M. J., & Ladouceur, R. (1994). Why do people worry? *Personality and Individual Differences*, *17*, 791–802. doi:10.1016/0191-8869(94)90048-5

Goldman, N., Dugas, M. J., Sexton, K. A., & Gervais, N. J. (2007). The impact of written exposure on worry: A preliminary investigation. *Behavior Modification*, *31*, 512–538. doi:10.1177/0145445506298651

Hahn, T. N. (1976). *The miracle of mindfulness: A manual for meditation*. Boston, MA: Beacon Press.

Hale, W. W., III., Klimstra, T. A., Branje, S. J. T., Wijsbroek, S. A. M., & Meeus, W. H. J. (2013). Is adolescent generalized anxiety disorder a magnet for negative parental interpersonal behaviors? *Depression and Anxiety*, *30*, 849–856. doi:10.1002/da.22065

Hanrahan, F., Field, A. P., Jones, F. W., & Davey, G. C. L. (2013). A meta-analysis of cognitive therapy for worry in generalized anxiety disorder. *Clinical Psychology Review*, *33*, 120–132. doi:10.1016/j.cpr.2012.10.008

Härter, M. C., Conway, K. P., & Merikangas, K. R. (2003). Associations between anxiety disorders and physical illness. *European Archives of Psychiatry and Clinical Neuroscience*, *253*, 313–320. doi:10.1007/s00406-003-0449-y

Hayes, S. A., Orsillo, S. M., & Roemer, L. (2010). Changes in proposed mechanisms of action during an acceptance-based behavior therapy for generalized anxiety disorder. *Behaviour Research and Therapy*, *48*, 238–245. doi:10.1016/j.brat.2009.11.006

Hayes, S. C., Strosahl, K., & Wilson, K. G. (1999). *Acceptance and commitment therapy: An experiential approach to behavior change*. New York, NY: Guilford Press.

Hayes-Skelton, S. A., Calloway, A., Roemer, L., & Orsillo, S. M. (2015). Decentering as a potential common mechanism across two therapies for generalized anxiety disorder. *Journal of Consulting and Clinical Psychology*, *83*, 395–404. doi:10.1037/a0038305

Hayes-Skelton, S. A., Roemer, L., & Orsillo, S. M. (2013). A randomized clinical trial comparing an acceptance-based behavior therapy to applied relaxation for generalized anxiety disorder. *Journal of Consulting and Clinical Psychology*, *81*, 761–773. doi:10.1037/a0032871

Hayes-Skelton, S. A., Usmani, A., Lee, J. K., Roemer, L., & Orsillo, S. M. (2012). A fresh look at potential mechanisms of change in applied relaxation for generalized anxiety disorder: A case series. *Cognitive and Behavioral Practice*, *19*, 451–462. doi:10.1016/j.cbpra.2011.12.005

Hirsch, C. R., Hayes, S., Mathews, A., Perman, G., & Borkovec, T. (2012). The extent and nature of imagery during worry and positive thinking in generalized anxiety disorder. *Journal of Abnormal Psychology*, *121*, 238–243. doi:10.1037/a0024947

Hoffman, D. L., Dukes, E. M., & Wittchen, H. (2008). Human and economic burden of generalized anxiety disorder. *Depression and Anxiety*, *25*, 72–90. doi:10.1002/da.20257

Hoge, E. A., Bui, E., Goetter, E., Robinaugh, D. J., Ojserkis, R. A., Fresco, D. M., & Simon, N. M. (2015). Change in decentering mediates improvement in anxiety in mindfulness-based stress reduction for generalized anxiety disorder. *Cognitive Therapy Research*, *39*, 228–235. doi:10.1007/s10608-014-9646-4

Hoge, E. A., Bui, E., Marques, L., Metcalf, C. A., Morris, L. K., Robinaugh, D. J., … Simon, N. M. (2013). Randomized controlled trial of mindfulness meditation for generalized anxiety disorder: Effects on anxiety and stress reactivity. *Journal of Clinical Psychiatry*, *74*, 786–792. doi:10.4088/jcp.12m08083

Hoge, E. A., Bui, E., Palitx, S. A., Schwarz, N. R., Owens, M. E., Johnston, J. M., … Simon, N. M. (2017). The effect of mindfulness meditation training on biological acute stress responses in generalized anxiety disorder. *Psychiatry Research.* doi:10.1016/j.psychres.2017.01.006

Iijima, Y., & Tanno, Y. (2012). The rebound effect in the unsuccessful suppression of worrisome thoughts. *Personality and Individual Differences*, *53*, 347–350. doi:10.1016/j.paid.2012.03.023

Johnston, L., Titov, N., Andrews, G., Dear, B. F., & Spence, J. (2013). Comorbidity and internet-delivered transdiagnostic cognitive behavioural therapy for anxiety disorders. *Cognitive Behaviour Therapy*, *42*, 180–192. doi:10.1080/16506073.2012.753108

Kessler, R. C., Berglund, P. A., Demler, O., Jin, R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication (NCS-R). *Archives of General Psychiatry*, *62*, 593–602. doi:10.1001/archpsyc.62.6.593

Kim, Y. W., Lee, S.-H., Choi, T. K., Suh, S. Y., Kim, B., Kim, C. M., … Yook, H.-K. (2009). Effectiveness of mindfulness-based cognitive therapy as an adjuvant to pharmacotherapy in patients with panic disorder or generalized anxiety disorder. *Depression and Anxiety*, *26*, 601–606. doi:10.1002/da.20552

Koerner, N., & Dugas, M. J. (2008). An investigation of appraisals in individuals vulnerable to excessive worry: The role of intolerance of uncertainty. *Cognitive Therapy and Research*, *32*, 619–638. doi:10.1007/s10608-007-9125-2

Kusec, A., Tallon, K., & Koerner, N. (2016). Intolerance of uncertainty, causal uncertainty, causal importance, self-concept clarity and their relations to generalized anxiety disorder. *Cognitive Behaviour Therapy*, *45*, 307–323. doi:10.1080/16506073.2016.1171391

Ladouceur, R., Dugas, M. J., Freeston, M. H., Léger, E., Gagnon, F., & Thibodeau, N. (2000). Efficacy of a cognitive–behavioral treatment for generalized anxiety disorder: Evaluation in a controlled clinical trial. *Journal of Consulting and Clinical Psychology*, *68*, 957–964. doi:10.1037//0022-006x.68.6.957

Ladouceur, R., Dugas, M. J., Freeston, M. H., Rhéaume, J., Blais, F., Boisvert, J., … Thibodeau, N. (1999). Specificity of generalized anxiety disorder symptoms and processes. *Behavior Therapy*, *30*, 191–207. doi:10.1016/s0005-7894(99)80003-3

Laguna, L. B., Ham, L. S., Hope, D. A., & Bell, C. (2004). Chronic worry as avoidance of arousal. *Cognitive Therapy and Research*, *28*, 269–281. doi:10.1023/b:cotr.0000021545.79764.69

Lee, J. K., & Orsillo, S. M. (2014). Investigating cognitive flexibility as a potential mechanism of mindfulness in generalized anxiety disorder. *Journal of Behavior Therapy and Experimental Psychiatry*, *45*, 208–216. doi:10.1016/j.jbtep.2013.10.008

Lee, S. H., Ahn, S. C., Lee, Y. J., Choi, T. K., Yook, K. H., & Suh, S. Y. (2007). Effectiveness of a meditation-based stress management program as an adjunct to pharmacotherapy in patients with anxiety disorder. *Journal of Psychosomatic Research*, *62*(2), 189–195. doi:10.1016/j.jpsychores.2006.09.009

Leichsenring, F., Salzer, S., Jaeger, U., Kächele, H., Kreische, R., Leweke, F., … Leibing, E. (2009). Short-term psychodynamic psychotherapy and cognitive-behavioral therapy in generalized anxiety disorder: A randomized, controlled trial. *American Journal of Psychiatry*, *166*, 875–881. doi:10.1176/appi.ajp.2009.09030441

Linehan, M. M. (1994). Acceptance and change: The central dialectic in psychotherapy. In S. C. Hayes, N. S. Jacobson, V. M. Follette, & M. J. Dougher (Eds.), *Acceptance and change: Content and context in psychotherapy* (pp. 73–86). Reno, NV: Context Press.

Llera, S. J., & Newman, M. G. (2010). Effects of worry on physiological and subjective reactivity to emotional stimuli in generalized anxiety disorder and nonanxious control participants. *Emotion*, *10*, 640–650. doi:10.1037/a0019351

Llera, S. J., & Newman, M. G. (2014). Rethinking the role of worry in generalized anxiety disorder: Evidence supporting a model of emotional contrast avoidance. *Behavior Therapy*, *45*, 283–299. doi:10.1016/j.beth.2013.12.011

Luborsky, L. (1984). *Principles of psychoanalytic psychotherapy: A manual for supportive-expressive treatment*. New York, NY: Basic Books.

Martin, J. R. (1997). Mindfulness: A proposed common factor. *Journal of Psychotherapy Integration*, *7*, 291–312. doi:10.1023/b:jopi.0000010885.18025.bc

McEvoy, P. M., Erceg-Hurn, D. M., Anderson, R. A., Campbell, B. N. C., & Nathan, P. R. (2015). Mechanisms of change during group metacognitive therapy for repetitive negative thinking in primary and non-primary generalized anxiety disorder. *Journal of Anxiety Disorders*, *35*, 19–26. doi:10.1016/j.janxdis.2015.07.003

McEvoy, P. M., Erceg-Hurn, D. M., Anderson, R. A., Campbell, B. N. C., Swan, A., Saulsman, L. M., … Nathan, P. R. (2015). Group metacognitive therapy for repetitive negative thinking in primary and non-primary generalized anxiety disorder: An effectiveness trial. *Journal of Affective Disorders*, *175*, 124–132. doi:10.1016/j.jad.2014.12.046

McEvoy, P. M., Nathan, P., & Norton, P. J. (2009). Efficacy of transdiagnostic treatments: A review of published outcome studies and future research directions. *Journal of Cognitive Psychotherapy*, *23*, 20–33. doi:10.1891/0889-8391.23.1.20

McLean, A., & Broomfield, N. M. (2007). How does thought suppression impact upon beliefs about uncontrollability of worry? *Behaviour Research and Therapy*, *45*, 2938–2949. doi:10.1016/j.brat.2007.08.005

McLaughlin, K. A., Mennin, D. S., & Farach, F. J. (2007). The contributory role of worry in emotion generation and dysregulation in generalized anxiety disorder. *Behaviour Research and Therapy*, *45*, 1735–1752. doi:10.1016/j.brat.2006.12.004

Mennin, D. S., & Fresco, D. M. (2009). Emotion regulation as an integrative framework for understanding and treating psychopathology. In A. M. Kring & D. S. Sloan (Eds.), *Emotion regulation and psychopathology* (pp. 356–379). New York, NY: Guilford Press.

Mennin, D. S., & Fresco, D. M. (2011, November). Emotion regulation therapy for complex and refractory presentations of anxiety and depression. Paper presented at the meeting of the Association for Behavioral and Cognitive Therapies, Toronto, ON.

Mennin, D. S., Fresco, D. M., Heimberg, R. G., & Ciesla, J. (2012, April). Randomized control trial of emotion regulation therapy for generalized anxiety disorder and comorbid depression. Paper presented at the meeting of the Anxiety Disorders Association of America, Arlington, VA.

Mennin, D. S., Fresco, D. M., Ritter, M., & Heimberg, R. G. (2015). An open trial of emotion regulation therapy for generalized anxiety disorder and coocurring depression. *Depression and Anxiety*, *32*, 614–623. doi:10.1002/da.22377

Mennin, D. S., Holaway, R. M., Fresco, D. M., Moore, M. T., & Heimberg, R. G. (2007). Delineating components of emotion and its dysregulation in anxiety and mood psychopathology. *Behavior Therapy*, *38*, 284–302. doi:10.1016/j.beth.2006.09.001

Mennin, D. S., McLaughlin, K. A., & Flanagan, T. J. (2009). Emotion regulation deficits in generalized anxiety disorder, social anxiety disorder, and their co-occurrence. *Journal of Anxiety Disorders*, *23*, 866–871. doi:10.1016/j.janxdis.2009.04.006

Mennin, D. S., Turk, C. L., Heimberg, R. G., & Carmin, C. (2004). Regulation of emotion in generalized anxiety disorder. In M. A. Reinecke & D. A. Clark (Eds.), *Cognitive therapy over the lifespan: Theory, research, and practice* (pp. 60–89). Hoboken, NJ: John Wiley & Sons.

Michelson, S. E., Lee, J. K., Orsillo, S. M., & Roemer, L. (2011). The role of values-consistent behavior in generalized anxiety disorder. *Depression and Anxiety*, *28*, 358–366. doi:10.1002/da.20793

Nassif, Y. (1999). Predictors of pathological worry (Unpublished master's thesis). University of Manchester, Manchester, UK.

Newby, J. M., Mewton, L., Williams, A. D., & Andrews, G. (2014). Effectiveness of transdiagnostic internet cognitive behavioural treatment for mixed anxiety and

depression in primary care. *Journal of Affective Disorders*, *165*, 45–52. doi:10.1016/j.jad.2014.04.037

Newby, J. M., Williams, A. D., & Andrews, G. (2014). Reductions in negative repetitive thinking and metacognitive beliefs during transdiagnostic internet cognitive behavioural therapy (iCBT) for mixed anxiety and depression. *Behaviour Research and Therapy*, *59*, 52–60. doi:10.1016/j.brat.2014.05.009

Newman, M. G., Castonguay, L. G., Borkovec, T. D., Fisher, A. J., Boswell, J. F., Szkodny, L. E., & Nordberg, S. S. (2011). A randomized controlled trial of cognitive-behavioral therapy for generalized anxiety disorder with integrated techniques from emotion-focused and interpersonal therapies. *Journal of Consulting and Clinical Psychology*, *79*, 171–181. doi:10.1037/a0022489

Newman, M. G., Castonguay, L. G., Borkovec, T. D., Fisher, A. J., & Nordberg, S. S. (2008). An open trial of integrative therapy for generalized anxiety disorder. *Psychotherapy*, *45*, 135–147.

Newman, M. G., Castonguay, L. G., Borkovec, T. D., & Molnar, C. (2004). Integrative psychotherapy. In R. G. Heimberg, C. L. Turk, & D. S. Mennin (Eds.), *Generalized anxiety disorder: Advances in research and practice* (pp. 320–350). New York, NY: Guilford Press.

Newman, M. G., & Llera, S. J. (2011). A novel theory of experiential avoidance in generalized anxiety disorder: A review and synthesis of research supporting a contrast avoidance model of worry. *Clinical Psychology Review*, *31*, 371–382. doi:10.1016/j.cpr.2011.01.008

Norton, P. J. (2006). Toward a clinically-oriented model of anxiety disorders. *Cognitive Behaviour Therapy*, *35*, 88–105. doi:10.1080/16506070500441561

Norton, P. J. (2012a). A randomized clinical trial of transdiagnostic cognitive-behavioral treatments for anxiety disorder by comparison to relaxation training. *Behavior Therapy*, *43*, 506–517. doi:10.1016/j.beth.2010.08.011

Norton, P. J. (2012b). *Group cognitive-behavioral therapy of anxiety: A transdiagnostic treatment manual*. New York, NY: Guilford Press.

Norton, P. J., & Barrera, T. L. (2012). Transdiagnostic versus diagnosis-specific CBT for anxiety disorders: A preliminary randomized controlled noninferiority trial. *Depression and Anxiety*, *29*, 874–882. doi:10.1002/da.21974

Norton, P. J., & Hope, D. A. (2002). Anxiety treatment program: Therapist manual. Unpublished treatment manual.

Nuevo, R., Montorio, I., & Borkovec, T. D. (2004). A test of the role of metaworry in the prediction of worry severity in an elderly sample. *Journal of Behavior Therapy and Experimental Psychiatry*, *35*, 209–218. doi:10.1016/j.jbtep.2004.03.002

Orsillo, S. M., Roemer, L., & Barlow, D. H. (2003). Integrating acceptance and mindfulness into existing cognitive-behavioral treatment for GAD: A case study. *Cognitive and Behavioral Practice*, *10*, 222–230. doi:10.1016/s1077-7229(03)80034-2

Öst, L.-G., & Breitholtz, E. (2000). Applied relaxation vs. cognitive therapy in the treatment of generalized anxiety disorder. *Behaviour Research and Therapy*, *38*, 777–790. doi:10.1016/s0005-7967(99)00095-9

Penney, A. M., Mazmanian, D., & Rudanycz, C. (2013). Comparing positive and negative beliefs about worry in predicting generalized anxiety disorder symptoms. *Canadian Journal of Behavioural Science*, *45*, 34–41. doi:10.1037/a0027623

Roemer, L., & Borkovec, T. D. (1994). Effects of suppressing thoughts about emotional material. *Journal of Abnormal Psychology*, *103*, 467–474. doi:10.1037//0021-843x.103.3.467

Roemer, L., Lee, J. K., Salters-Pedneault, K., Erisman, S. M., Orsillo, S. M., & Mennin, D. S. (2009). Mindfulness and emotion regulation difficulties in generalized anxiety disorder: Preliminary evidence for independent and overlapping contributions. *Behavior Therapy*, *40*, 142–154. doi:10.1016/j.beth.2008.04.001

Roemer, L., & Orsillo, S. M. (2002). Expanding our conceptualization of and treatment for generalized anxiety disorder: Integrating mindfulness/acceptance-based approaches with existing cognitive-behavioral models. *Clinical Psychology: Science and Practice*, *9*, 54–68. doi:10.1093/clipsy/9.1.54

Roemer, L., & Orsillo, S. M. (2005). An acceptance-based behavior therapy for generalized anxiety disorder. In S. M. Orsillo & L. Roemer (Eds.), *Acceptance and mindfulness-based approaches to anxiety: Conceptualization and treatment* (pp. 213–240). New York, NY: Springer.

Roemer, L., & Orsillo, S. M. (2007). An open trial of an acceptance-based behavior therapy for generalized anxiety disorder. *Behavior Therapy*, *38*, 72–85. doi:10.1016/j.beth.2006.04.004

Roemer, L., Orsillo, S. M., & Salters-Pedneault, K. (2008). Efficacy of an acceptance-based behavior therapy for generalized anxiety disorder: Evaluation in a randomized controlled trial. *Journal of Consulting and Clinical Psychology*, *76*, 1083–1089.

Roemer, L., Salters, K., Raffa, S. D., & Orsillo, S. M. (2005). Fear and avoidance of internal experiences in GAD: Preliminary tests of a conceptual model. *Cognitive Therapy and Research*, *29*, 71–88.

Rosen, N. O., & Knäuper, B. (2009). A little uncertainty goes a long way: State and trait differences in uncertainty interact to increase information seeking but also increase worry. *Health Communication*, *24*, 228–238. doi:10.1037/a0012720

Ruscio, A. M., & Borkovec, T. D. (2004). Experience and appraisal of worry among high worriers with and without generalized anxiety disorder. *Behaviour Research and Therapy*, *42*, 1469–1482. doi:10.1016/j.brat.2003.10.007

Safran, J. D., & Segal, Z. V. (1990). *Interpersonal process in cognitive therapy*. Lanham, MD: Jason Aronson.

Salzer, S., Pincus, A. L., Winkelbach, C., Leichsenring, F., & Leibing, E. (2011). Interpersonal subtypes and change of interpersonal problems in the treatment of patients with generalized anxiety disorder: A pilot study. *Psychotherapy*, *48*, 304–310. doi:10.1037/a0022013

Salzer, S., Winkelbach, C., Leweke, F., Leibing, E., & Leichsenring, F. (2011). Long-term effects of short-term psychodynamic psychotherapy and cognitive-behavioural therapy in generalized anxiety disorder: 12-month follow-up. *Canadian Journal of Psychiatry/La Revue Canadienne de Psychiatrie*, *56*, 503–508. doi:10.1177/070674371105600809

Sibrava, N. J., & Borkovec, T. D. (2006). The cognitive avoidance theory of worry. In G. C. L. Davey & A. Wells (Eds.), *Worry and its psychological disorders: Theory, assessment and treatment* (pp. 239–256). Hoboken, NJ: John Wiley & Sons.

Siev, J., & Chambless, D. L. (2007). Specificity of treatment effects: Cognitive therapy and relaxation for generalized anxiety and panic disorders. *Journal of Consulting and Clinical Psychology*, *75*, 513–522. doi:10.1037/0022-006x.75.4.513

Stefan, S., & David, D. (2013). The functions of worry in uncontrollable situations: Perceived effects of worry and the feeling of being in control. *Erdélyi Pszichológiai Szemle*, *14*, 3–17.

Stöber, J., Tepperwien, S., & Staak, M. (2000). Worrying leads to reduced concreteness of problem elaborations: Evidence for the avoidance theory of worry. *Anxiety, Stress, and Coping*, *13*, 217–227. doi:10.1080/10615800008415725

Stokes, C., & Hirsch, C. R. (2010). Engaging in imagery versus verbal processing of worry: Impact on negative intrusions in high worriers. *Behaviour Research and Therapy*, *48*, 418–423. doi:10.1016/j.brat.2009.12.011

Thielsch, C., Andor, T., & Ehring, T. (2015). Do metacognitions and intolerance of uncertainty predict worry in everyday life? An ecological momentary assessment study. *Behavior Therapy*, *46*, 532–543. doi:10.1016/j.beth.2015.05.001

Titov, N., Dear, B. F., Schwencke, G., Andrews, G., Johnston, L., Craske, M. G., & McEvoy, P. (2011). Transdiagnostic internet treatment for anxiety and depression: A randomised controlled trial. *Behaviour Research and Therapy*, *49*, 441–452. doi:10.1016/j.brat.2011.03.007

Torbit, L., & Laposa, J. M. (2016). Group CBT for GAD: The role of change in intolerance of uncertainty in treatment outcomes. *International Journal of Cognitive Therapy*, *9*, 356–368. doi:10.1521/ijct_2016_09_17

Treanor, M., Erisman, S. M., Salters-Pedneault, K., Roemer, L., & Orsillo, S. M. (2011). Acceptance-based behavioral therapy for GAD: Effects on outcomes from three theoretical models. *Depression and Anxiety*, *28*, 127–136. doi:10.1002/da.20766

Turk, C. L., Heimberg, R. G., Luterek, J. A., Mennin, D. S., & Fresco, D. M. (2005). Emotion dysregulation in generalized anxiety disorder: A comparison with social anxiety disorder. *Cognitive Therapy and Research*, *29*, 89–106. doi:10.1007/s10608-005-1651-1

Uhmann, S., Beesdo-Baum, K., Becker, E. S., & Hoyer, J. (2010). Specificity of interpersonal problems in generalized anxiety disorder versus other anxiety disorders and depression. *Journal of Nervous and Mental Disease*, *198*, 846–851. doi:10.1097/nmd.0b013e3181f98063

Van der Heiden, C., Melchior, K., & de Stigter, E. (2013). The effectiveness of group metacognitive therapy for generalised anxiety disorder: A pilot study. *Journal of Contemporary Psychotherapy*, *43*, 151–157. doi:10.1007/s10879-013-9235-y

Van der Heiden, C., Muris, P., & Van der Molen, H. T. (2012). Randomized controlled trial on the effectiveness of metacognitive therapy and intolerance-of-uncertainty therapy for generalized anxiety disorder. *Behaviour Research and Therapy*, *50*, 100–109. doi:10.1016/j.brat.2011.12.005

Vollestad, J., Sivertsen, B., & Neilsen, G. H. (2011). Mindfulness-based stress reduction for patients with anxiety disorders: Evaluation in a randomized controlled trial. *Behaviour Research and Therapy*, *49*, 281–288. doi:10.1016/j.brat.2011.01.007

Vrana, S. R., Cuthbert, B. N., & Lang, P. J. (1986). Fear imagery and text processing. *Psychophysiology*, *23*, 247–253. doi:10.1111/j.1469-8986.1986.tb00626.x

Wells, A. (2004). A cognitive model of GAD: Metacognitions and pathological worry. In R. G. Heimberg, C. L. Turk, & D. S. Mennin (Eds.), *Generalized anxiety disorder: Advances in research and practice* (pp. 164–186). New York, NY: Guilford Press.

Wells, A. (2009). *Metacognitive therapy for anxiety and depression*. New York, NY: Guilford Press.

Wells, A., & Carter, K. (2001). Further tests of a cognitive model of generalized anxiety disorder: Metacognitions and worry in GAD, panic disorder, social phobia, depression, and nonpatients. *Behavior Therapy*, *32*, 85–102. doi:10.1016/s0005-7894(01)80045-9

Wells, A., & King, P. (2006). Metacognitive therapy for generalized anxiety disorder: An open trial. *Journal of Behavior Therapy and Experimental Psychiatry*, *37*, 206–212. doi:10.1016/j.jbtep.2005.07.002

Wells, A., Welford, M., King, P., Papageorgiou, C., Wisely, J., & Mendel, E. (2010). A pilot randomized trial of metacognitive therapy vs applied relaxation in the treatment of adults with generalized anxiety disorder. *Behaviour Research and Therapy*, *48*, 429–434. doi:10.1016/j.brat.2009.11.013

Williams, M. C., Mathews, A., & Hirsch, C. R. (2014). Verbal worry facilitates attention to threat in high-worriers. *Journal of Behavior Therapy and Experimental Psychiatry*, *45*, 8–14. doi:10.1016/j.jbtep.2013.05.006

Wong, S. Y. S., Yip, B. H. K., Mak, W. W. S., Mercer, S., Cheung, E. Y. L., Ling, C. Y. M. L., … Ma, H. S. W. (2016). Mindfulness-based cognitive therapy v. group psychoeducation for people with generalised anxiety disorder: Randomised controlled trial. *British Journal of Psychiatry*, *209*, 68–75.

6

The Treatment of Obsessive–Compulsive Disorder

Lindsey M. Collins, Laura B. Bragdon, and Meredith E. Coles
Department of Psychology, Binghamton University, Binghamton, USA

Obsessive–compulsive disorder (OCD) is characterized by the presence of obsessions and/or compulsions that consume substantial time or are associated with significant impairment in functioning or distress (American Psychiatric Association, 2013). Cross-national research suggests that the lifetime prevalence of OCD ranges from 1.1% to 1.8% (Weissman, 1998). OCD is associated with a host of negative consequences for individuals with OCD and the people around them. Individuals with OCD often experience substantial disruptions in mood and functioning (Eisen et al., 2006; Fineberg, Fourie, Gale, & Sivakumaran, 2005). Further, they frequently report impaired quality of life due to their symptoms (Koran, Thienemann, & Davenport, 1996), high levels of marital discord (Riggs, Hiss, & Foa, 1992), and frequent family conflict (Calvocoressi, Lewis, Harris, & Trufan, 1995). Indeed, the substantial burdens of OCD are reflected by the fact that nearly two-thirds of individuals with the disorder report suicidal ideation and over one quarter report at least one past suicide attempt (Torres et al., 2006). At a societal level, OCD is associated with increased health care costs (Simon, Ormel, VonKorff, & Barlow, 1995) and the World Health Organization (Murray & Lopez, 1996) has characterized OCD as one of the top 10 most disabling of all psychiatric and medical conditions in the industrialized world. In addition, over 20% of clients with OCD are unemployed (DuPont et al., 1996). Finally, the negative impact of OCD is heightened by the fact that it is one of the most chronic forms of psychopathology, rarely remitting without intervention (Eisen & Rasmussen, 2002; Eisen et al., 1999; Pinto, Mancebo, Eisen, Pagano, & Rasmussen, 2006).

The specific symptom content in individuals with OCD varies greatly but often falls within several broad domains. For example, many individuals with OCD are concerned about contamination and fear becoming ill or dying as a result of contact with dirt or germs. These fears are often then associated with extreme efforts to avoid contamination or to keep things clean, such as showering many times a day, insisting that family members change into clean clothes before entering the house, or having stringent rules regarding who can touch various objects in the home. Other common areas of concern in OCD include fears of doing or saying something blasphemous, and fears of accidentally or intentionally harming others. Methods of avoiding or neutralizing fears in OCD

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

can include checking, counting, ordering, and arranging objects, or performing actions in a ritualistic manner.

Despite the diversity of the specific symptoms found in OCD, there are shared core features. Obsessions are often characterized as intrusions: intrusive, unwanted thoughts that recur and are associated with substantial anxiety or distress. These intrusions can typically be manifested in a variety of forms, including images, thoughts, or urges. Given the distress that accompanies these intrusions, individuals with OCD often attempt to suppress or dismiss the intrusions by performing compulsions. Compulsions are purposeful behaviors that are typically intended to reduce anxiety and prevent negative outcomes expressed within the obsessive thought. However, there is increasing recognition that, in some cases, compulsions may not be associated with feared consequences but may be driven by sensations that things are "not just right" or are incomplete (Pietrefesa & Coles, 2008). Just as the form of obsessions can vary, so can compulsions. Compulsions can be behaviors performed in a particular sequence or a set number of times, or they can be thoughts or images that are brought into one's mind to prevent something bad from happening.

Recognizing the negative impact of OCD, there have been significant strides in understanding the nature and negative consequences of the disorder. As recently as the 1970s, OCD was thought to be rare, chronic, and intractable (Price, Rasmussen, & Eisen, 1999). However, empirical studies have demonstrated that OCD is far more common than originally thought (Diefenbach, Abramowitz, Norberg, & Tolin, 2007; Foa & Goldstein, 1978; Franklin & Foa, 2002; Moritz et al., 2005). A great deal of effort has been devoted to establishing theories on the nature of symptoms, which has facilitated the development of treatments for OCD. Most recently, following the emergence of efficacious OCD treatments, researchers have developed theories focusing on specific treatment targets (e.g., attentional control) in order to enhance symptom reduction and improve functioning. Many of these treatment approaches have been supported with empirical data; however, the amount of evidence showing efficacy and/or confirming the role of the hypothesized mechanisms by which the treatments are proposed to exert their effect has varied.

In this chapter we utilize the approach developed by David and Montgomery (2011) to evaluate currently available forms of psychotherapy for OCD. This approach evaluates psychotherapy research in two domains: efficacy and theory. The relative efficacy and absolute efficacy of many treatments have already been established with outcome studies, but understanding the theories underlying change is also a crucial part of evaluating a treatment.

Ideally, interventions should be grounded in a theory regarding the mechanisms that maintain the condition or symptoms being targeted (Kazdin, 2009). To best assess the strength of a theory, we followed the requirements outlined by Kazdin (2007, 2009) for demonstrating mediators and mechanisms of change. For example, we considered a variable to be a mediator only if temporal precedence was established through measurement at multiple time points (more than just pre–post). Therefore, we did not consider cross-sectional data. Additionally, in order to establish mediation, it is necessary to use appropriate statistical techniques, such as structural equation modeling or multiple regression (Kazdin, 2007). Therefore, due to our stringent criteria, we excluded even rigorous studies if they used a more traditional cross-sectional approach to mediation (e.g., Barron & Kenny, 1986). We begin our examination of therapeutic approaches with exposure and ritual prevention (ERP).[1]

6.1 Exposure and Ritual Prevention

Exposure therapy for OCD is based on the model of emotional processing originally proposed by Foa and Kozak (1986). In their model, Foa and Kozak distinguished between normal and maladaptive fear structures and proposed that activating maladaptive fear structures and facilitating emotional processing of this information would produce symptom reductions. In other words, they proposed that in vivo, imaginal, and interoceptive exposures could create opportunities to work through activation of the fear structure and subsequent emotional processing (Foa & Kozak, 1986). Based on this model, clients are encouraged to complete exposure trials in which they willingly confront feared situations, thereby triggering anxiety and activating the fear network. The assumption is that the fear of the situation is not realistic and that this exposure allows the individual to experience within-session habituation to the feared stimulus and to disconfirm his or her feared outcome (Foa & Kozak, 1986). This new information and experience are negatively reinforcing and gradually shift beliefs about the probability of threat, facilitating confrontation of additional feared situations. Foa and Kozak (1986) proposed that both within-session and between-session habituation (i.e., decreases in anxiety level within one session and from one session to the next) are necessary for emotional processing and fear reductions. Finally, ritual prevention eliminates the negative reinforcement that is experienced from doing compulsions and provides an opportunity for new learning.

Several revisions and updates have been made to the Foa and Kozak (1986) model since its first publication. In the most recent iteration, Foa, Huppert, and Cahill (2006) note changes and additions that have grown out of 20 years of additional data and clinical observations. One revision that has received a great deal of attention is an increased emphasis on new learning. In their original writing, Foa and Kozak (1986) proposed that repeated, purposeful confrontation with the feared stimulus would weaken erroneous associations and acquire new associations. Over time, it has become increasingly clear that the exposure process does not wipe out prior learning but instead creates new learning that competes with existing information (Bouton, 2000; Rescorla, 2001). The practical implication of this insight is that an exposure can activate either the original fear or a new association. Practicing and strengthening the new association in as many contexts as possible may help to mitigate the possibility of the return of fear across contexts and has been increasingly emphasized since the mid-2000s. Other changes have been the relative deemphasis on the importance of habituation within session and the increase in the perceived importance of extinction between sessions. Although the majority of data supporting the shift has come from other forms of anxiety, not OCD, we are not aware of a reason that it would not also be applicable to OCD. Finally, the proposed mechanism underlying exposure therapy is not habituation per se but the modification of the relevant erroneous associations through disconfirming information.

6.1.1 Support for Treatment

The efficacy of ERP for OCD has been well studied. These studies have ranged from small case studies to modest open trials and up to very large randomized clinical trials (RCTs). Further, researchers have compared ERP with placebo and other treatments, both alone and in combination with other interventions. Investigations have examined

ERP delivered in individual and group formats, in person, via the telephone, via the internet and video conferencing, and with session frequency ranging from daily to weekly and beyond. Across all of these studies and variations, researchers have repeatedly documented the ability of ERP to reduce symptoms, thereby supporting this treatment's efficacy. Given the large number of studies testing ERP, a detailed review of each is beyond the scope of this chapter. Nevertheless, we can reference several thorough meta-analyses and qualitative review papers to augment this chapter (see, e.g., Olatunji, Davis, Powers, & Smits, 2013; Öst, Havnen, Hansen, & Kvale, 2015). In order to provide a sense of the literature, we will focus our attention on recent trials that have utilized strong methods. These trials tend to have compared ERP to other active treatments, as ample data already existed showing ERP to be superior to comparison conditions.

One of the largest RCTs tested the efficacy of ERP compared to clomipramine and to the combination of ERP and clomipramine (Foa et al., 2005). In this trial, 149 individuals with OCD were randomized to one of the three active conditions or placebo, and 87 completed the trial. ERP was delivered in an intensive format for 4 weeks followed by 8 weekly maintenance sessions. Clomipramine was delivered for 12 weeks. At week 12, all active treatments were better than placebo. The addition of clomipramine to ERP was not found to improve outcomes significantly. Of clients completing ERP alone and ERP plus clomipramine, 86% and 79% were classified as responders, respectively.

In addition to comparing ERP with pharmacotherapy, researchers have compared it with several other forms of psychotherapy. For example, ERP has frequently been compared with cognitive therapy (CT; Cottraux, Note et al., 2001; Emmelkamp & Beens, 1991; McLean et al., 2001; Van Oppen & Haan, 1995). With regard to whether ERP is superior to CT, both the strength of the studies addressing this issue and the results are mixed. Several studies showed ERP to be superior to CT, a few failed to find differences, and only one that we are aware of showed ERP to be less effective than CT (for a review see Eddy, Dutra, Bradley, & Westen, 2004). Regardless of the relative strength of ERP versus CT, in this section we are primarily interested in the efficacy of ERP.

In an RCT comparing ERP to CT, McLean and colleagues (2001) randomized 76 clients. Two cotherapists delivered therapy in groups of 6 to 8 clients, meeting with them once per week (for 2.5 hours) for 12 weeks. Both ERP and CT were associated with significantly larger reductions in clinician-rated OCD severity than the control group. Further, when taking group differences in medication use into account, ERP was associated with significantly larger OCD symptom reductions than CT at the end of treatment. At the 3-month follow-up, 45% of the ERP clients met stringent criteria for being classified as "recovered" (decrease in Yale–Brown Obsessive Compulsive Scale [Y-BOCS] score $\geq$ 6 points and final score <12), and this rate was significantly higher than the 16% found for CT. Finally, ERP continued to be associated with higher rates of recovery than CT, even when differential rates of drop-out and treatment refusal were factored in.

In a recent randomized trial, Simpson et al. (2013) examined the ability of ERP to further reduce OCD symptoms after partial response to serotonin reuptake inhibitors (SRIs). Specifically, if clients continued to experience moderate or severe OCD symptoms after 12 or more weeks on an SRI, they were then randomized to the addition of either ERP (two sessions per week) or risperidone. With respect to symptom change, 80% of the 40 clients who completed ERP reported decreases in their Y-BOCS scores of at least 25%. At posttreatment, 43% of the clients treated with ERP were classified as having minimal symptoms (Y-BOCS less than or equal to 12).

6.1.2 Support for Theory

Despite the dominance of exposure therapy in the treatment of OCD, a dearth of research examines mechanisms hypothesized to underlie treatment. Of the few studies that discuss mechanisms, the large majority are either not sufficiently designed or analyzed to test mediation. For example, at least five studies have examined both changes in cognitions and symptoms from treatment (Cottraux, Yao et al., 2001; Emmelkamp & Beens, 1991; Kozak, Foa, & Steketee, 1988; Overton & Menzies, 2005; Solem, Håland, Vogel, Hansen, & Wells, 2009). Nevertheless, these studies did not include a suitable prospective design or sufficient statistical analyses to model change over time. Therefore, we are unable to conclude whether changes in cognitions or beliefs produce changes in symptoms.

Two studies have used contemporary analytic methods to test mechanisms underlying exposure therapy for OCD. Olatunji, Rosenfield et al. (2013) reanalyzed the data from Cottraux, Note et al. (2001) to test whether changes in interpretations reflecting responsibility or in avoidance predicted subsequent symptom change better than vice versa (symptom change predicting changes in avoidance or responsibility beliefs). The findings regarding potential mechanisms of change were not as clear as hypothesized, as is often the case. The results of the mediation analyses (collapsing the ERP and CT groups) did not show significant effects of responsibility or avoidance for predicting subsequent changes in clinician-rated OCD severity over time. Further, contrary to the prediction, decreases in OCD symptoms were found to predict later decreases in behavioral avoidance, which was the opposite of the original hypothesis. A more recent study by Su, Carpenter, Zandberg, Simpson, and Foa (2016) examined cognitive mediation of change from ERP. Results using multilevel modeling failed to find support for cognitive mediation (changes in cognitions did not predict subsequent changes in symptoms). Further, there was not support for changes in symptoms predicting subsequent changes in beliefs. More work is needed to delineate the specific effects of ERP.

6.1.3 Summary

Due to the strong evidence for the efficacy of ERP for OCD, but the lack of sufficient evidence for the mechanism of action, we classified this treatment within the scientifically oriented psychotherapies, specifically Category II of David and Montgomery's (2011) framework: intervention-driven psychotherapies. The core features of intervention-driven psychotherapies are a body of empirical support for their efficacy but limited empirical information on the underlying theory or mechanisms. Indeed, various investigative teams (see Cottraux, Note et al., 2001; Emmelkamp & Beens, 1991; Foa et al., 2005; McLean et al., 2001; Simpson et al., 2013) have consistently found support for the efficacy of ERP for the treatment of OCD in rigorous randomized and controlled trials. However, there are surprisingly few studies that have investigated the mechanisms of ERP for OCD (Olatunji, Rosenfield et al., 2013; Su et al., 2016). Furthermore, the two papers that have examined potential cognitive mediation of changes in OCD symptoms have not found support (Olatunji, Rosenfield et al., 2013; Su et al., 2016). Given the information available, the treatment is well supported, but more research is needed in order to determine the validity of the proposed theory.

6.2 Cognitive Therapy

While a small group of experts was focusing on the role of behavior in psychopathology, a separate, yet equally novel, movement was afoot examining cognitions. A. T. Beck and colleagues first developed the cognitive model as a theory of depression. Beck hypothesized that an individual's core beliefs about him/herself influence interpretations of daily events, and that certain types of interpretations can contribute to the maintenance of depressive symptoms (Beck, 1963). For example, an individual whose core belief is "I'm worthless" will interpret small mistakes as reflective of his or her incompetence. Researchers therefore developed therapy based on this model to identify potential distortions within individuals' automatic thoughts. Automatic thoughts are the minute-to-minute thoughts that come to mind throughout the day and are often influenced by our core beliefs. For example, someone with a core belief that they are worthless may think, "The audience isn't looking at me because I'm boring" or "They can tell I'm really nervous and think I'm not qualified." Potentially unhelpful portions of these thoughts are addressed in therapy by using Socratic dialogue (cognitive restructuring), empirically testing the validity of thoughts (cognitive experiments), and identifying themes in behaviors and cognitions. A focus on the present and on the processing of conscious experiences initially differentiated CT from psychoanalytic therapies. Later on, cognitive models of anxiety were developed; they posited that beliefs regarding heightened probability of harm lead individuals to misinterpret ambiguous stimuli and to attempt to avoid threat (Beck, Emery, & Greenberg, 1985).

Over time, cognitive theory was applied to other conditions, including OCD. Theorists argue that, in OCD, unwanted intrusive thoughts are misinterpreted as dangerous (e.g., thinking of something bad happening makes it more likely to happen), leading to increased state anxiety and a subsequent urge to neutralize the feeling with another thought or action (Rachman, 1993, 1997; Salkovskis, 1985, 1989). These misinterpretations and faulty appraisals are thought to reflect core OCD belief domains (responsibility and probability of harm, importance and control of thoughts, intolerance of uncertainty, and perfectionism; Obsessive Compulsive Cognitions Working Group, 2001; Rachman, 1993, 1997; Salkovskis, 1985). For example, the intrusive thought "I hit someone with my car" might make individuals with OCD more anxious because they experience difficulty tolerating the uncertainty of the situation and feel more responsible for the wellbeing of others. CT for OCD was designed to help clients challenge their beliefs by targeting maladaptive interpretations of their thoughts (stimuli) and developing more balanced ways of viewing their intrusions (i.e., "I might have hit them, but it is unlikely"; Van Oppen & Arntz, 1994). In addition, CT therapists aim to help individuals directly challenge and alter their beliefs about responsibility, overestimation of threat, importance and control of thoughts, intolerance of uncertainty, and perfectionism. Behavioral experiments are sometimes used to test the dysfunctional thoughts related to these beliefs. In sum, cognitive techniques for OCD are proposed to help clients challenge and modify their OC beliefs, thereby reducing their distress in response to intrusions and subsequent desire to perform compulsions.

6.2.1 Support for Treatment

Many studies have examined the efficacy of CT for OCD, and at least eight are RCTs (Belloch, Cabedo, & Carrio, 2008; Cottraux, Note et al., 2001; Emmelkamp & Beens,

1991; Emmelkamp, Visser, & Hoekstra, 1988; McLean et al., 2001; Van Oppen & Haan, 1995; Vos, Huibers, & Arntz, 2012; Wilhelm et al., 2009). Overall, findings demonstrate that CT is efficacious for OCD, showing greater symptom reductions than waitlist and similar outcomes to ERP. Given that efforts to develop CT followed the development of ERP, studies testing CT often examined its efficacy relative to ERP instead of waitlist. However, CT has been shown to be superior to waitlist in reducing OCD symptoms (Van Balkom et al., 1998; Wilhelm et al., 2009).

Wilhelm et al. (2009) investigated the absolute efficacy of CT compared with a waitlist comparison condition. In this RCT, the researchers randomly assigned 29 clients either to 22 sessions of modular CT (Wilhelm & Steketee, 2006) or to a 12-week waitlist period. After 12 weeks, the CT group showed significant improvements in OCD symptoms (Y-BOCS) from pre- to posttreatment and significantly greater improvements compared with the waitlist group. From pretreatment to posttreatment (22 sessions), clients experienced significant reductions in OCD symptoms and depressive symptoms (per the Beck Depression Inventory II). According to the clinician global improvement scale within the Anxiety Disorders Interview Schedule, a clinician rating of global improvement (Guy, 1976), compared to before treatment, 81% of treatment completers and 63% of the entire sample were classified as "much improved" or "very much improved" at posttreatment. From posttreatment to 3-month follow up, Y-BOCS and levels of OCD-related beliefs remained stable.

Although researchers have demonstrated the absolute effectiveness of CT, a greater number of studies have focused on the relative efficacy of CT compared to ERP. One of the first RCTs of CT for OCD compared 16 sessions of CT with self-controlled exposure in 71 clients (Van Oppen & Haan, 1995). Seven clients dropped out in each treatment, but significant improvements were observed in both treatment groups. No significant differences in symptom improvement were found between CT and ERP; however, the effect of treatment was slightly larger in the CT group (CT: Cohen's $d = 1.43$; ERP: $d = 1.03$). At posttreatment, 50% of clients in the CT group demonstrated a score of 12 or lower on the Y-BOCS, deeming them "recovered," compared with 28% of the clients in the self-ERP condition.

Since this trial, many other rigorous RCTs have further demonstrated the efficacy of CT compared with ERP. McLean et al. (2001; see Section 6.1) found that CT and ERP groups both reported significantly lower Y-BOCS total scores at posttreatment than the waitlist group and that the groups did not differ significantly from each other at posttreatment and follow-up. Belloch et al. (2008; $N = 29$) found that the majority of clients in the CT condition (81.25%) achieved "recovered" status at posttreatment, meaning that their Y-BOCS scores decreased by at least six points, and they demonstrated a Y-BOCS score of less than 12 at posttreatment. Further, at a 1-year follow-up, symptom reduction was maintained and even improved further ($d = 4.03$ from pretreatment) for the CT condition. The researchers also found that symptom reduction in the CT and ERP groups did not differ significantly. Nevertheless, the effect size for the CT ($d = 3.68$) group was slightly higher than for the ERP group ($d = 2.72$).

6.2.2 Support for Theory

Proponents of CT for OCD posit that CT produces symptom reduction by reducing obsessive–compulsive beliefs (Wilhelm & Steketee, 2006). Many studies have found that obsessive–compulsive beliefs are significantly lower after treatment (Belloch et al.,

2008; Belloch, Cabedo, Carrió, & Larsson, 2010; Cottraux, Note et al., 2001; Emmelkamp & Beens, 1991; Emmelkamp et al., 1988; McLean et al., 2001; Van Oppen & de Haan, 1995; Vos et al., 2012). Other studies have found significant correlations between changes in obsessive–compulsive beliefs and obsessive–compulsive symptoms from pre- to posttreatment (Abramowitz, Nelson, Rygwall, & Khandker, 2007; Emmelkamp et al., 1988; Olatunji, Rosenfield et al., 2013). Although these study designs provide preliminary data supporting the theory that a reduction in obsessive–compulsive beliefs is the mechanism by which CT improves OC symptoms, only two studies have assessed this directly with a longitudinal mediation analysis.

Olatunji, Rosenfield et al. (2013; see Section 6.1) used multilevel modeling to reevaluate the results of an RCT conducted by Cottraux, Note and colleagues (2001). Olatunji, Davis et al. (2013) found that responsibility beliefs did not significantly precede or predict later changes in Y-BOCS scores. Although this finding does not support the proposed mechanism of CT, it should be considered that the mediation analyses were conducted on the combined sample of individuals in both the CT and ERP sections. Although ERP might also reduce symptoms through a reduction in OC beliefs, the combined sample might have conversely diluted a mediation effect present only in the CT condition.

Wilhelm, Berman, Keshaviah, Schwartz, and Steketee (2015) recently investigated the relationship between obsessive beliefs and OCD symptom severity (Y-BOCS total) at multiple time points during an open trial and waitlist controlled trial of CT for OCD ($N = 39$; see also Wilhelm et al., 2005, 2009). Wilhelm et al. (2015) used time-lagged random effect regressions to predict subsequent Y-BOCS scores from obsessive beliefs. They found that the perfectionism and certainty subscale score significantly predicted prospective change in Y-BOCS total score in the next session (2.1 greater decrease; $d = 0.77$) but that responsibility/threat and importance/control of thoughts did not.

6.2.3 Summary

Due to the strong evidence for the efficacy of CT for OCD but the lack of sufficient evidence for the mechanism of action, we classified this treatment within Category II (intervention-driven psychotherapies). Various investigative teams have repeatedly supported the efficacy of CT for OCD in rigorous RCTs (Belloch et al., 2008; Cottraux, Note et al., 2001; Emmelkamp & Beens, 1991; Emmelkamp et al., 1988; McLean et al., 2001; Van Oppen & Haan, 1995; Vos et al., 2012; Wilhelm et al., 2009). Nevertheless, we could only identify two studies investigating the mechanisms of CT for OCD (Olatunji, Rosenfield et al., 2013; Wilhelm et al., 2015). Although Olatunji, Rosenfield et al. (2013) found a small effect of obsessive beliefs on symptom change in the expected direction, the results were not significant. Wilhelm and colleagues (2015) found that perfectionism and certainty significantly mediated prospective symptom change but failed to find the effect for responsibility/threat or for importance/control of thoughts. Although the results from these studies suggest that CT might work by way of modifying obsessive beliefs, the results were not strong enough (especially in Olatunji, Rosenfield et al., 2013) to confidently validate the theory for CT. Given the information available, the treatment is well supported, but more research is needed in order to determine the validity of the proposed theory.

6.3 Cognitive–Behavioral Therapy

Cognitive–behavioral models of psychopathology emphasize the strong bidirectional interplay between thoughts and behaviors. Indeed, this overlap is often emphasized in therapy, with clinicians helping clients become more aware of the impact of their interpretations of events on their subsequent behavior and vice versa. Clients are taught that behavioral exercises can be used to alter beliefs and cognitive interventions can be used to alter behavior. This approach is helpful in that it increases the number of strategies that can be used to reduce OCD symptoms. However, this close relationship can make it very difficult to categorize a treatment as "behavioral," "cognitive," or "cognitive–behavioral." In this section, we discuss interventions that are reported to contain elements from both approaches. These studies are few, as there has been much more interest in comparing distinct components than in testing their combination.

6.3.1 Support for Treatment

Our review yielded several studies that tested the efficacy of cognitive–behavioral therapy (CBT) for OCD using RCT designs. These studies compared CBT to ERP, pharmacotherapy, and waitlist. Compared with waitlist, researchers have found that CBT is superior for reducing OCD symptom severity (Freeston et al., 1997; O'Connor, Todorov, Robillard, Borgeat, & Brault, 1999; Vogel, Stiles, & Gunnar, 2004) and, on average, clients who receive CBT are rated as only having mild symptoms at the end of treatment. Comparing CBT with ERP alone, Whittal, Thordarson, and McLean (2005; $N = 83$) found that, after 12 weeks, clients in the CBT group experienced significant declines in symptoms with a comparable magnitude of change to ERP. Further, the proportion of clients in the CBT group classified as "recovered" (decrease in Y-BOCS ≥ 6 points and score ≥ 11) at posttreatment did not differ significantly from the ERP group (67% and 59%, respectively). The reductions in OCD severity were maintained at 3-month follow-up, with 76% of the clients in the CBT group labeled as recovered. Two RCTs have examined the efficacy of 12 weeks of CBT delivered in a group format (Cordioli et al., 2003; Sousa, Isolan, Oliveira, Manfro, & Cordioli, 2006). Both studies found support for CBT reducing OCD symptoms. First, Cordioli and colleagues (2003) found that CBT was associated with significant reductions in clinician-rated OCD compared with waitlist. Specifically, 69.6% of clients in the CBT group experienced at least a 35% reduction in their OCD severity, as rated by a clinician, compared with 4% in the waitlist group. The second study, by Sousa et al. (2006), found similar results when CBT was compared with medication, with individuals in the CBT group having significantly greater reductions than those in the sertraline group (44% vs. 28% reduction, respectively). Further, the researchers found that approximately one-third of clients in the CBT group met criteria for remission (Y-BOCS ≤ 8) at the end of treatment, compared to only one client (4%) in the sertraline group.

6.3.2 Support for Theory

Turning to empirical support for the hypothesized mechanisms underlying the treatment, we did not find studies that tested the mechanisms of the combined approach directly. However, CBT can be thought of as a combination of the effects of cognitive

and behavioral techniques. Therefore, support for the mechanisms underlying ERP and CT is also applicable (see, e.g., Olatunji, Rosenfield et al., 2013).

6.3.3 Summary

Due to the strong evidence for the efficacy of CBT for OCD but the insufficient amount of evidence for the proposed mechanisms of change, we classified this treatment within Category II (intervention-driven psychotherapies). The efficacy of CBT for OCD in both individual and group formats has been supported by at least three RCTs by different investigative teams (Cordioli et al., 2003; Sousa et al., 2006; Whittal et al., 2005). However, we could not identify any studies investigating the mechanisms of CBT, and we found only two studies investigating the mechanisms of CT or ERP for OCD (Olatunji, Rosenfield et al., 2013; Wilhelm et al., 2015). Although the results from these studies suggest that CBT might work by way of modifying obsessive beliefs and reducing avoidance/responsibility, the results were not strong enough (especially in Olatunji, Rosenfield et al., 2013) to confidently validate the theory for a combination of cognitive and behavioral techniques. Given the information available, the treatment is well supported, but more research is needed in order to determine the validity of the proposed theory.

6.4 Acceptance and Commitment Therapy

Acceptance and commitment therapy (ACT) was developed in the 1980s and 1990s, and extended prior cognitive and behavioral theories and techniques in a new direction. Building from these prior theories, ACT took a slightly different perspective on psychopathology and how to treat it, becoming part of what can be known as the "third wave" of empirically based psychotherapies. ACT is unique with regard to *how* cognitions and behaviors are addressed and the goals in doing so (Bluett, Homan, Morrison, Levin, & Twohig, 2014; Twohig, Hayes, & Masuda, 2006; Twohig, 2009). ACT techniques help clients decrease their struggles with anxiety and increase their focus on meaningful activities, regardless of their OCD symptoms. For example, therapists often guide clients toward discussions of their long-term priorities (e.g., having a family, giving back to their community) and what they view as core life values (e.g., honesty, compassion), and away from discussing symptoms and their negative impact. Concepts and skills are taught through experiential exercises, discussion, metaphors, and analogies. Concepts covered in session are practiced between sessions.

Two OCD-relevant beliefs often addressed in ACT are viewing negative inner experiences as dangerous and viewing them as needing to be diminished or controlled. ACT therapists use six primary processes to increase psychological flexibility: (1) acceptance, (2) defusion, (3) self as context, (4) values, (5) being present, and (6) committed action (Twohig, Hayes, Plumb, Pruitt, & Collins, 2011). Treatment of OCD using an ACT framework will often begin with targeting acceptance: learning to not try to control inner experiences (e.g., obsessions). Defusion focuses on altering the way that thoughts are experienced and reducing their perceived importance. For example, individuals with OCD often interpret words, sentences, and thoughts very literally, or as representing truth (e.g., "Thinking of my loved one being in a car accident makes them

more likely to be in an accident"). Defusion exercises are used to remedy this. Defusion exercises include watching thoughts float by, repeating certain words or phrases over and over, and pretending that thoughts are passengers on a bus. Self as context refers to one's unchanging sense of self, and values are the guiding principles that one cares most deeply about. Being present means focusing on what one is experiencing right now instead of directing attention to what could go wrong in the future. Finally, committed action allows the therapist and client to integrate behavioral techniques that seem to be potentially beneficial. In treating OCD with ACT, the therapist varies the amount of time spent on each of these six processes based on the client's needs, and many of these techniques are conveyed through metaphor (see Dehlin, Morrison, & Twohig, 2013).

6.4.1 Support for Treatment

Our review of the literature on ACT for OCD revealed three studies with built-in control conditions. Two studies used baseline periods during which treatment was not delivered (Dehlin et al., 2013; Twohig et al., 2006) and the other was an RCT (Twohig et al., 2011). All three studies provided support for the efficacy of eight sessions of ACT in reducing self-reported and/or clinician-rated OCD. In an initial sample of four individuals, Twohig et al. (2006) were the first to demonstrate the efficacy of ACT for OCD. Using a multiple baseline design, they found that none of the clients showed symptom reductions during a baseline period, but all four showed large reductions when treatment was introduced. Specifically, in eight sessions, the ACT intervention produced clinically significant reductions in compulsions, and improvements were maintained at a 3-month follow-up. Further, their findings also showed decreases in experiential avoidance, the believability of obsessions, and the perceived need to respond to obsessions. Also using a multiple baseline design, Dehlin et al. (2013) found initial support for an eight-session ACT intervention for OCD focused on scrupulosity. Results based on five adults with scrupulosity concerns showed that ACT was associated with substantial reductions in the frequency of compulsions (74% reduction at posttreatment and 80% reduction at follow-up). Further, clinician-rated OCD scores decreased over 50% from pre- to posttreament and pretreatment to follow-up. Finally, consistent with the primary targets in ACT, treatment was also associated with decreases in avoidance activities.

In the largest study to date of ACT for OCD, Twohig and colleagues (2011) randomized 79 adults with OCD to either eight sessions of ACT or progressive relaxation training (PRT). ACT was found to produce greater reductions in OCD symptoms than PRT at posttreatment and follow-up. Further, a higher proportion of participants in the ACT group (23 of 41) met criteria for a clinically significant change in OCD symptoms at posttreatment compared to the PRT group (7 of 38). Finally, in addition to changes in symptoms, members of the ACT group had significantly greater improvements in psychological flexibility, reductions in thought control, and reductions in thought–action fusion (the belief that thinking of doing something is equivalent to actually doing it) than members of the PRT group.

6.4.2 Support for Theory

We were only able to identify one study that directly tested the hypothesized mechanisms of ACT and their subsequent impact on symptom changes (Twohig, Vilardaga,

Levin, & Hayes, 2015). In ACT, the clinician is generally working to help his or her clients to increase their psychological flexibility. A variety of techniques are used to help clients decrease their struggles with anxiety and increase their focus on meaningful activities. Within their trial comparing ACT with PRT, Twohig and colleagues (2015) also tested whether changes in psychological flexibility predicted changes in OCD symptoms. Examining the weekly process and outcomes, they found that the process measures predicted outcomes at 1 through 4 weeks later. Further, the process measures predicted subsequent changes in outcome better than outcomes predicted changes in the process measures. In addition, Twohig and colleagues tested the extent to which posttreatment process scores predicted changes in symptoms from pretreatment to follow-up. These analyses showed that changes in psychological flexibility mediated symptom change from pre- to posttreatment. In contrast, changes in thought control and thought–action fusion were not found to mediate symptom changes. In summary, despite there being only one study testing mediation in ACT for OCD, this study and the analyses conducted were strong methodologically and provided support for the primary construct (psychological flexibility) as a mediator of treatment effects.

6.4.3 Summary

Due to the strong evidence for the efficacy of ACT for OCD but an insufficient number of rigorous studies testing the hypothesized mechanisms, we classified this treatment within Category II (intervention-driven psychotherapies). The efficacy of ACT for OCD has been supported by two multiple baseline trials and one RCT (Dehlin et al., 2013; Twohig et al., 2006, 2011). We could only identify one study investigating whether the mechanisms of ACT preceded and predicted outcomes (Twohig et al., 2015). The results of this study provide strong evidence that psychological flexibility mediates symptom change in ACT for OCD. Nevertheless, one study does not provide sufficient evidence to confidently validate the theory. Given the information available, the treatment is well supported, but more research is needed in order to determine the validity of the proposed theory.

6.5 Metacognitive Therapy

Metacognitive theory and the therapy derived from it are similar to CT and ACT in that they are designed to provide the client with an alternative perspective on his or her thoughts. However, these approaches differ in many important ways. For example, in metacognitive therapy (MCT), the emphasis is not on the content of these thoughts (e.g., cognitive distortions) per se, as is the case with CT, but on clients' thoughts about these thoughts. In other words, MCT focuses on thinking about thinking.

Wells and Matthews presented the first metacognitive model of OCD in 1994, and it has undergone several updates since that time (Wells, 2000, 2013). These authors explained how thoughts about thoughts, and the techniques used to try to regulate thoughts, could be important in OCD. They distinguished between thoughts regarding the meaning of intrusions and thoughts regarding responding (or not responding) to an intrusion and the implications of that decision. Individuals with OCD often believe their thoughts to be important or have meaning. For example, as mentioned in Section 6.4, many clients with OCD demonstrate thought–action fusion. Specifically, they believe

that having a thought about something is equivalent to it actually happening, or that having a thought about something increases the likelihood that it will happen.

MCT addresses these thoughts about the importance of the original intrusion and helps the client develop an adaptive plan for responding to obsessions. Another important difference from CT is that metacognitive theory emphasizes dysfunctional internal criteria that clients use to guide their rituals. Whereas CT focuses on core beliefs that drive interpretations of individual events, MCT focuses on clients' beliefs about their cognitive processes. For example, clients will use an internal state or sensation as a marker for when to stop performing compulsions, which can result in hypervigilance to both internal and external cues signaling that an internal state has been adequately achieved or that threat is still present. Clients are also frequently hypervigilant regarding the occurrence of intrusions themselves, a process referred to as "heightened cognitive self-consciousness." MCT emphasizes clients' beliefs about their cognitive processes, such as their perceived ability to remember things or sustain attention.

6.5.1 Support for Treatment

There is mounting support for the use of MCT in the treatment of OCD. Since the mid-2000s there have been both open and controlled trials examining MCT. Several open trials have found support for MCT for OCD using case studies (Fisher & Wells, 2008; Fitt & Rees, 2012; Rees & Van Koesveld, 2008). These studies have demonstrated that MCT is associated with reductions in OCD symptom severity and changes in metacognitive beliefs such as the need for control. In addition, at least two studies of MCT for OCD have used more empirically rigorous methods. First, Moritz, Jelinek, Hauschildt, and Naber (2010) tested the efficacy of a metacognitive intervention delivered in a self-help format. Participants were randomly assigned to either a waitlist or 4 weeks of MCT. After 4 weeks, participants in the MCT condition showed greater improvement in OCD symptoms overall and obsessions, in comparison with waitlist participants. Examination of specific symptom subtypes via a self-report measure showed that the reductions in OCD symptoms were driven largely by reductions in obsessing, and that significant changes were not found for ordering, checking, or neutralizing symptoms. Using a similar intervention to Moritz et al. (2010) but delivered in person, Andouz, Dolatshahi, Moshtagh, and Dadkhah (2012) used a multiple baseline design to examine the efficacy of MCT for clients with OCD characterized by obsessions only. In this study, clients initially completed a baseline, which was then followed by 14 weeks of treatment. All participants showed significant symptom improvement on both clinician-rated and self-reported measures of OCD once treatment was introduced, compared with stable symptom levels during baseline. There was also moderate support for the maintenance of gains 3 months posttreatment. In conclusion, both studies are methodologically strong and provide support for the efficacy of MCT for reducing symptoms of OCD. Nevertheless, additional studies with strong designs, with multiple indicators of change, and from a variety of research groups are needed to increase confidence in stating that MCT is empirically supported for OCD.

6.5.2 Support for Theory

Despite the clear theoretical rationale of MCT, few studies have tested the mechanisms underlying the treatment. Our review revealed one study that directly tested the

hypothesis that changing metacognitive beliefs will produce changes in OCD symptoms. Specifically, Myers and Wells (2013) used an experimental design to test whether inducing metacognitive beliefs regarding the need to control thoughts would increase OCD symptoms. Their findings showed a significant interaction between OCD symptom levels (low/high) with condition (experimental/control) and that individuals with heightened OCD symptoms may have been more reactive to the manipulation (inducing beliefs regarding the need to control thoughts) than individuals with low OCD symptoms. Within the high symptom group (but not the low symptom group), individuals in the experimental condition reported spending significantly more time thinking about the topic they were primed to avoid compared with the control group. Further supporting this theory, Fisher and Wells (2005) showed that a 5-minute exposure exercise framed within a metacognitive explanation produced reductions in metacognitive beliefs, anxiety, and urges to respond to intrusions. However, the authors did not test whether the changes in metacognition predicted subsequent change in OCD symptoms.

6.5.3 Summary

Based on the empirical support for MCT for OCD and the hypothesized mechanisms underlying it, we classified this treatment under Category II (intervention-driven psychotherapies). The efficacy of MCT for OCD has been supported by many open trials, one study using a multiple baseline design, and one RCT (Andouz et al., 2012; Fisher & Wells, 2008; Fitt & Rees, 2012; Moritz et al., 2010; Rees & van Koesveld, 2008). The available data suggest that MCT may be particularly helpful for obsessions. Nevertheless, we could only identify one study investigating the direct link between metacognitive beliefs (need to control thoughts) and OCD symptoms (Myers & Wells, 2013). The results of this study provide strong evidence of a causal relationship between metacognitive beliefs and obsessive–compulsive symptoms, but more research is needed in order to confidently document a relationship between MCT, metacognitive beliefs, and symptom reduction.

6.6 Attention Bias Modification Training

Williams, Watts, MacLeod, and Mathews (1988, 1997) proposed that a better understanding of the basic cognitive processes underlying emotional processing and psychopathology would be beneficial in refining our models of anxiety and depression and developing interventions to address these symptoms. Building from the field of cognitive psychology, this approach also cut across Beck's cognitive (schema) theory and semantic network models (Bower, 1981; Foa et al., 1986) and made distinctions between multiple levels of cognition. Specifically, Williams et al. (1988) proposed that conscious and nonconscious processing of information should be considered. Applying this model, both theory and data led to the hypothesis that anxiety primarily impacts early information-processing (initial attention) whereas depression exerts a greater influence on later processing (e.g., memory; Williams et al., 1988). With regard to anxiety specifically, Williams et al. (1988) proposed that anxious individuals preferentially allocate attention to threatening information, increasing their anxious arousal. To measure these rapid cognitive processes believed to be associated with anxiety and mood, researchers

utilized highly controlled and intricate computerized paradigms originally designed for studies in cognitive psychology.

For many years, the emotional Stroop task was the main paradigm used to assess attention biases in anxiety and depression (Gotlib & McCann, 1984; Mathews & MacLeod, 1985). This task modified the original Stroop task (Stroop, 1935) and measures the time required for participants to name the ink color in which both negative- and positive-valence words are printed. Response times are believed to indicate how long it takes a participant to ignore the meaning of the word in order to name the color of the text. Individuals with anxiety are often slower to name the color of negative words than positive or neutral words, indicating an attention bias toward negative stimuli (i.e., it is hard for them to ignore the meaning). Researchers have also used the dot-probe paradigm extensively to measure attentional biases and to modify attention (MacLeod & Mathews, 1988; MacLeod, Mathews, & Tata, 1986). This paradigm measures attention based on the premise that participants will detect a probe faster when it appears where they were already looking compared to when it appears in a different location on the screen. Faster detection of probes presented in the location previously occupied by threatening stimuli (vs. neutral stimuli) is interpreted as an attentional bias to threat (MacLeod et al., 1986).

Many studies have supported a link between anxiety and attention bias to threat (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & Van IJzendoorn, 2007; Mogg & Bradley, 1998). Nevertheless, other studies have failed to replicate this bias or have found the opposite (Koster, Crombez, Van Damme, Verschuere, & De Houwer, 2005; Yiend & Mathews, 2001). In order to modify this bias, existing experimental paradigms were redesigned to no longer measure but to subtly alter attentional biases to threat. Beginning with MacLeod, Rutherford, Campbell, Ebsworthy, and Holker (2002), researchers began to explore the efficacy of modifying these computerized tasks to subtly alter attention and investigated the effects this would have on participants' emotional states (Amir, Najmi, & Morrison, 2009; Hazen, Vasey, & Schmidt, 2009; MacLeod, Soong, Rutherford, & Campbell, 2007). First, researchers hypothesized that attention could be manipulated by strategically altering the proportion of trials in which the probe followed threatening versus neutral stimuli, an approach referred to as attention bias modification training (ABMT). Second, researchers hypothesized that training with preference toward neutral versus threatening stimuli would decrease or increase stress reactivity, respectively. MacLeod et al. (2002) provided support for both theories and showed that training with the probe always following the same stimulus type led to changes in response time to the probe and reactivity to stress. This research was the first to demonstrate that altering the parameters of an evaluative task can be used to advantage as an intervention. Building from this work, other investigators found that ABMT, in which probes were biased to follow the nonthreatening stimuli (neutral or positive), significantly reduced anxiety and lessened reactivity to stress for clinically anxious participants, with small to medium effects (Hakamata et al., 2010; Mogoaşe, 2014).

Given that OCD was considered to be an anxiety disorder for many years and that data suggest it may also be associated with attention bias to threat, researchers have recently explored the utility of ABMT for OCD (Amir, Kuckertz, Najmi, & Conley, 2015; Amir et al., 2009; Cisler & Olatunji, 2010; Muller & Roberts, 2005; Najmi & Amir, 2010; Tallis, 1997). Studies examining ABMT for OCD have primarily used a modified dot-probe task in which ideographic threat words related to the individual's main feared outcome

(e.g., contamination, harm to others) and neutral words are presented, followed by a probe that always replaces the neutral word on the screen (Amir et al., 2015; Najmi & Amir, 2010). The individual is instructed to identify the position of the probe as quickly as possible, thereby "training" them to attend to the neutral words.

6.6.1 Support for Treatment

We were only able to identify one study in which ABMT was examined in a clinical sample of individuals with OCD (Amir et al., 2015). In this study, 22 treatment-seeking individuals diagnosed with OCD received a novel treatment that included self-directed ERP and cognitive bias modification (including ABMT and interpretation bias modification). Five blocks of treatment (ABMT, psychoeducation, control, attention control training, working memory training, and interpretation bias modification) were administered in counterbalanced order. Self-reported levels of OCD symptom levels after ABM were not significantly different but were smaller than before ABM ($d = 0.14$). Also, the change in self-reported OCD symptoms from pre- to posttreatment in the ABMT condition was not significantly different from the change in the control condition. These findings are therefore inconclusive with regard to the efficacy of ABMT for OCD.

6.6.2 Support for Theory

Proponents of ABMT contend that the training reduces stress reactivity by reducing an attentional bias to threat. Despite the numerous studies on ABMT, we only found one study that employed mediational analyses to investigate the mechanisms of ABMT for OCD. Najmi and Amir (2010) investigated the relation between ABMT, change in attention bias to threat, and subsequent behavioral approach to threatening stimuli in individuals with subclinical obsessive–compulsive-related fears of contamination. In this study, 52 undergraduates high in contamination fears were randomly assigned to either an ABMT or to an attentional control condition (in which the probe appeared equally behind threatening and neutral words). At posttraining, the ABMT group demonstrated a significantly smaller attention bias toward threat and completed significantly more steps on a behavioral approach to contamination test than the attentional control condition group. Change in attention bias from pre- to posttreatment significantly mediated the relationship between experimental group and number of steps completed on the behavioral approach to contamination test. Although this study provides preliminary support for the theorized mechanism of ABMT (reduction of attention bias to threat), the experimental design did not allow the investigators to determine whether the change in attentional biases both preceded and predicted outcome.

6.6.3 Summary

Due to the lack of rigorous studies conducted and only preliminary evidence for the efficacy and mechanisms of action in ABMT for OCD, this treatment was classified within Category IV (investigational psychotherapies). Such interventions do not have sufficient support for their efficacy or underlying hypothesized mechanisms, frequently due to them being in the early stages of development. We could only identify one rigorous clinical trial of ABMT for OCD (Amir et al., 2015), and the results from this trial did not strongly support the efficacy of ABMT. Similarly, we could only identify one study

investigating the mechanisms of ABMT for OCD (Najmi & Amir, 2010). Although this study suggests that ABMT effectively modifies attentional biases, the experiment and statistical analyses were not implemented in a way to rigorously test for causal relations between the variables. Given the available evidence, more research is needed to determine both the efficacy of the treatment and the validity of the proposed theory.

6.7 Interpretation Bias Modification Training

Interpretation bias modification training (IBMT) is designed to implicitly modify interpretations through repeatedly inducing neutral or positive interpretation–situation pairings. IBMT differs from cognitive, metacognitive, and acceptance therapies in that the client is not taught conscious skills that they can use to assess interpretations but rather is subtly "trained" to favor more helpful and adaptive interpretations. Williams and colleagues (1988) proposed that, in addition to attention biases, individuals with anxiety have implicit interpretation biases. Specifically, they posited that anxious individuals tend to interpret ambiguous stimuli as threatening, thereby increasing the salience of relevant cues in their environment. According to their model, individuals with anxiety will preferentially process negatively valenced information, which leads them to interpret relevant stimuli as more threatening. Numerous studies using both self-reported and behavioral indices of interpretation (response latency, sentence completion) have supported such negative interpretation biases in anxious individuals (see review by Mathews & MacLeod, 2005). Interpretation biases have been found in many forms of anxiety, leading researchers to propose that interpretation biases are likely to contribute to the development and maintenance of OCD. Specifically, it is suggested that individuals with OCD negatively interpret their thoughts (stimuli), which transforms normal intrusive thoughts into obsessions (Rachman, 1997).

Mathews and Mackintosh (2000) were the first to demonstrate that interpretations could be manipulated with an "ambiguous scenario training" paradigm. In this design, participants are presented with ambiguous scenarios for which they must fill in the final word (e.g., "If I talk, they will think I'm ____"). Participants are presented with two word fragments (positive and negative) from which to choose, only one of which fits conceptually in the sentence (e.g., sm_rt vs. f_t). After correctly completing the sentence, participants are given a comprehension question that they are instructed to answer in line with the prior interpretation, sometimes expanding beyond the specific example (e.g., "Will they dislike you?"). Using this paradigm, Mathews and Mackintosh (2000) successfully induced positive and negative interpretations that produced increases and decreases in state anxiety, respectively. Building on this study, similar IBMT paradigms have been effective in influencing state mood and anxiety (Menne-Lothmann et al., 2014). Given the research indicating that interpretations are also important in the maintenance of OCD, investigators have begun to assess the efficacy of IBMT for OCD.

6.7.1 Support for Treatment

Like attention bias modification, IBMT for OCD was born out of existing paradigms of IBMT for anxiety. The few studies that have assessed IBMT for OCD have used ambiguous scenario paradigms similar to that of Mathews and Mackintosh (2000),

either with scenarios designed according to the OCD belief domains (Obsessive Compulsive Cognitions Working Group, 2001; Steketee et al., 2003) or with ideographically chosen scenario–word pairings. Our literature searches revealed one rigorous controlled trial using IBMT for OCD. Amir et al., 2015 (reviewed in Section 6.6) compared the efficacy of self-directed ERP and cognitive bias modification (including ABMT and IBMT). Scores on the Obsessive–Compulsive Inventory–Revised significantly decreased from pre- to post-IBMT ($d = 0.39$), and these reductions were significantly greater than the reductions in the control condition ($d = 0.48$).

6.7.2 Support for Theory

Proponents of IBMT for OCD contend that the training reduces interpretation biases to threat, which subsequently reduces urges to neutralize intrusive thoughts and OC-related beliefs. A few studies have assessed the effect of IBMT on these variables in undergraduate and community samples, but they did not test for mediation (Clerkin & Teachman, 2011; Grisham, Becker, Williams, Whitton, & Makkar, 2014; Williams & Grisham, 2013). We could only find one study that examined whether change in interpretation bias predicted prospective change in obsessive–compulsive beliefs.

Specifically, Beadel, Smyth, and Teachman (2014) investigated the trajectories of change in interpretation biases, psychophysiological arousal, obsessive–compulsive beliefs, and symptoms during a course of IBMT toward positive versus negative interpretations. This study assessed changes in 75 undergraduates high in obsessive–compulsive symptoms. The results demonstrated that IBMT produced a gradual decrease in negative interpretations and effectively reduced obsessive–compulsive beliefs in the positive condition, but not the negative condition. Moreover, changes in interpretation bias predicted obsessive–compulsive beliefs only in the positive condition, but changes in obsessive–compulsive beliefs did not significantly predict reactivity to an obsessive–compulsive stressor.

6.7.3 Summary

Due to the lack of rigorous studies conducted and the existence of only preliminary evidence for the efficacy and mechanisms of action in IBMT for OCD, we classified this treatment within Category IV (investigational psychotherapies). We could only identify one rigorous clinical trial of IBMT for OCD in an adult sample (Amir et al., 2015), but the results strongly supported the efficacy of IBMT. Similarly, we could only identify one study investigating the mechanisms of IBMT for OCD (Beadel et al., 2014). Although the results from the study suggest that IBMT changes obsessive–compulsive beliefs by way of interpretation biases, changes in interpretation biases were not shown to predict subsequent reactivity to obsessive–compulsive stressors. Given the available evidence, more research is needed in order to determine both the efficacy of the treatment and the validity of the proposed theory.

6.8 Eye Movement Desensitization and Reprocessing Therapy

Researchers have primarily examined eye movement desensitization and reprocessing therapy (EMDR) as an intervention for posttraumatic stress disorder. Beginning with

work by Francine Shapiro in the 1980s, EMDR quickly received a great deal of attention, and over time it was explored for the treatment of other anxiety disorders and psychological conditions. EMDR is purported to draw from several theoretical perspectives, including cognitive–behavioral and information-processing theories, and behavioral, psychodynamic, interactional, and body-based approaches (Shapiro & Maxfield, 2002) and is based on the adaptive information-processing model (Shapiro & Maxfield, 2002). The primary tenet of the adaptive information-processing model is that it typically processes new information adaptively and stores this new information in memory networks. Further, over time, as new experiences occur, the memory networks are linked. Problems can manifest when new distressing experiences are not processed fully and are linked to existing memories. EMDR draws from data showing that eye movements and other dual-attention stimuli enhance information-processing (Shapiro, 2001). There is significant overlap in the methods used in EMDR and ERP, including rapport-building, testing thoughts, and eliciting fear and associated emotions and interpretations. Nevertheless, when fear is elicited in EMDR, clients are also asked to engage in a task that requires dual attention, such as tapping or moving the eyes from side to side for at least 15 seconds. In EMDR, these techniques are proposed to target shifting affect and beliefs by developing connections between the memories and more adaptive information.

6.8.1 Support for Treatment

We were only able to identify one study that tested the efficacy of EMDR for treating OCD (Nazari, Momeni, Jariani, & Tarrahi, 2011). In that study, 90 individuals with OCD were randomly assigned to either EMDR or the medication citalopram. Thirty clients in each group completed the study through the follow-up assessment and were therefore included in the analyses. Supporting the efficacy of the therapy, the EMDR group was found to have significant reductions in clinician-rated OCD severity from pre- to post-treatment. Further, these symptom reductions were found to be significantly greater for the EMDR group than the citalopram group.

6.8.2 Support for Theory

Researchers have linked horizontal saccadic eye movements to many components of memory. For example, they have shown that eye movements are associated with increased recall of both recent and childhood memories, recall of larger numbers of neutral words, and decreased distress associated with autobiographical memories (Samara, Elzinga, Slagter, & Nieuwenhuis, 2011). A study of female college students tested whether horizontal saccadic movements, like those used in EMDR, would improve the connectivity between the two hemispheres of the brain (Samara et al., 2011). There was some evidence that the eye movements were associated with recalling more emotional words compared with the control procedure. Nevertheless, there was not support for the hypothesis that the eye movements would improve functional connectivity (Samara et al., 2011). Furthermore, we are not aware of any studies that have evaluated whether improvement in information-processing mediates the relationship between eye movements and symptom reduction in individuals with OCD.

6.8.3 Summary

Due to the lack of rigorous studies conducted on the efficacy and theory of EMDR, we classified this treatment within Category IV (investigational psychotherapies). We could only identify one rigorous clinical trial of EMDR for OCD (Nazari et al., 2011), and one study testing the hypothesized mechanisms. Further, as reviewed, the one study that examined mechanisms did not find support and was conducted in a college sample. Clearly, more work is needed before EMDR can be recommended as an empirically supported treatment for OCD.

6.9 Implications for Research

This review and analysis of psychotherapy for OCD provides a detailed assessment of the current status of these treatments and is helpful in identifying implications for future research and clinical work. First, this review reiterates the conclusion that strongly supported treatments for OCD exist. Indeed, five of the interventions were categorized as intervention-driven psychotherapies (Category II). For each of these five approaches, there is strong support for the therapeutic package but less support for the hypothesized theories. Second, when systematically reviewing the literature using the criteria set out by David and Montgomery (2011), the limitations of existing studies became more apparent. In most cases, it was not the case that studies had tested mediation and failed to find evidence for it; instead, it was primarily the case that very few studies truly test mediation. It is apparent that treatments have been developed based on clinical judgment and outcomes such as symptom reduction. Now that there is considerable support for the efficacy of these treatments, more research is needed to understand the mechanisms by which they work. Further, recent advances in statistical methodologies available for assessing mediation (i.e., path analysis and multilevel modeling) will greatly facilitate these efforts. Experts agree that additional research on the mechanisms of treatment for OCD should be a top priority for the field. Further questions that should be explored concern the best standardized definitions of response, remission, and treatment types to use; broader treatment outcomes (i.e., quality of life, functioning); dose and frequency of therapy; the effectiveness of the therapies in different environments; and the decision rules for seeking and providing empirically supported treatments.

Further evaluation of the mechanisms of change in the treatment of OCD is important for several reasons. First, a greater understanding of the mediators of change might help clinicians decide which treatments to use for specific cases (Kazdin, 2007). Such understanding is especially relevant for OCD treatment, because symptom presentation is so heterogeneous. Studying mechanisms might help us identify the components of treatment that work optimally for individuals with specific symptom types (e.g., checking vs. washing, harm avoidant vs. incompleteness driven).

Second, understanding mechanisms may also help in optimizing treatment response. If we can identify the most beneficial components of a therapy that produces relevant change, we can utilize that understanding to make treatments even more effective and more efficient, possibly eliminating aspects of a treatment that do not engender significant improvement.

Third, we need to understand how treatments work in order to improve our ability to generalize treatment response from controlled trials to interventions provided by community practitioners (Kazdin, 2007). Specifically, the study of mechanisms could help us elucidate which conditions and components should be maintained when transferred from research to applied settings.

Finally, understanding the "active" components of treatments could potentially reveal that techniques currently described using different terminology overlap considerably. Many of the interventions we reviewed have been shown to be efficacious, but the mechanisms of change of these interventions may be more similar than different. If we were to identify commonalities across treatments, we might be able to combine or collapse treatments, which might facilitate dissemination and understanding of empirically supported options. This approach may be especially needed in OCD treatment, because, according to our review, many treatments have empirical support.

After reviewing the literature, a host of questions on mechanisms surfaced. First, does exposure therapy produce symptom reduction by activating the fear network and gradual habituation to the stimulus, or by disconfirming expectations (new learning), or via both mechanisms? Foa and Kozak (1986) initially hypothesized that exposure therapy works by activating the fear network and habituation. As we have reviewed, some studies have found support for this theory (e.g., Kozak et al., 1988). Nevertheless, surprisingly few studies have measured these hypothesized mechanisms at all. More recently, Craske et al. (2008) proposed that exposure does not work through habituation but through a disconfirmation of expected outcomes, or "inhibitory learning." Studies have also demonstrated support for this theory (e.g., Baker, Mystkowski, & Culver, 2010), but none have tested for true mediation and few have examined this theory in relation to OCD treatment.

Second, does exposure also produce symptom reduction by changing cognitions? This possibility might reflect a similar process to the "new learning" that Craske and others hypothesized. Exposure may essentially test the validity of thoughts, create inhibitory learning, and alter expectations for the future. Indeed, exposure is very similar to the "behavioral experiments" in pure cognitive therapy, and studies have demonstrated that cognitive change occurs in ERP (Overton & Menzies, 2005; Solem et al., 2009). However, a recent study by Su et al. (2016) did not find support for changes in cognitions predicting subsequent changes in symptoms during the course of ERP. More research on these treatments and their proposed mechanisms is needed.

Third, does ABMT primarily work by modifying attentional biases, or does it work by way of a different mechanism? In ABMT, clients are essentially "exposed" to their feared outcomes in addition to the proposed "active" component of attention retraining. Furthermore, researchers have found that the attention bias in OCD attenuates, which might suggest a habituation to the stimuli presented in the task (Amir et al., 2009). Future studies should assess whether the proposed mechanism of ABMT does exist or whether mechanisms more closely resembling habituation, inhibitory learning, or cognitive control are at play.

Fourth, are the core components of EMDR and exposure therapy essentially the same? Indeed, EMDR is designed to help clients habituate to their feared stimuli similarly to exposure, but researchers continue to debate whether differences in presentation of stimuli and added saccadic eye movements meaningfully differentiate the two treatments (for a review see Perkins & Rouanzoin, 2002). Nevertheless, research

on the theory of EMDR, especially the eye movement component, is limited. Future studies should aim to clarify the role and benefit of EMDR beyond exposure and assess possible mechanism differences across these treatments. Furthermore, little research has been conducted on EMDR for OCD, so more studies are definitely needed in order to assess the efficacy and theory of EMDR for OCD. These and other questions about the theory of OCD treatments can best be answered by testing for mediation. Future studies implementing path analysis and multilevel modeling will be able to assess whether proposed mechanisms precede and predict outcome and whether they are differentially present in different treatments for OCD.

In addition to studying mechanisms, an important next step is to clarify the optimal dose (number of sessions) and frequency (how often sessions are held) of these treatments. Identifying and communicating the optimal dose of a particular treatment can help bridge the gap between clinical trials and routine clinical practice, thereby improving the generalizability of interventions. Studies of psychotherapy in general have found that more sessions are related to greater symptom reduction (Orlinsky, Grawe, & Parks, 1994). With regard to OCD in particular, we were only able to find studies examining dose and frequency for ERP and CT. These studies showed that, although cognitive and behavioral interventions for OCD had used a wide range of sessions (from 5 to 22 sessions), number of sessions did not significantly correlate with effect size (Olatunji, Davis et al., 2013). For ERP more specifically, similar effect sizes have been found in clinical trials using different session frequencies (i.e., daily, twice a week, once a week; Abramowitz, Foa, & Franklin, 2003; Öst et al., 2015). The research to date suggests that dose and frequency do not significantly influence ERP treatment outcome; however, more studies utilizing newer methods (i.e., dose–response curves) could further clarify these findings. The lack of research on the optimal number and frequency of sessions for other treatments (including CT) should also be addressed in future studies.

A long-standing tradition is to emphasize *reductions* in clients' symptoms as the primary outcome measure, or measure of success in treatment. This information is clearly important. Nevertheless, we propose that it will be beneficial to also consider clients' symptom severity (diagnostic status) and quality of life. Studies on treatment efficacy currently use variable methods for determining client improvement (i.e., "response" vs. "remission"), and even differing cut-offs for the same outcome labels. These inconsistencies influence interpretation of findings, making comparisons across studies difficult and clouding our ability to judge the efficacy of interventions (Simpson, Huppert, Petkova, Foa, & Liebowitz, 2006; Simpson & Franklin, 2005). Utilizing signal detection analyses, Tolin, Abramowitz, and Diefenbach (2005) identified that a 30% reduction in the Y-BOCS was optimal for treatment response and a 40–50% reduction was optimal for predicting mild illness (i.e., remission) at posttreatment. As we move forward, researchers should consider the symptom reduction they expect to see if treatments are truly effective, and should determine a consensus cut-off based on this. Future researchers should also consider the optimal goal of treatment beyond symptoms and include constructs such as quality of life, ability to work, and suicidal ideation. Studies measuring constructs such as quality of life in OCD treatment are limited, with most RCTs focusing on symptom reduction as their primary outcome. Although it is easy to assume that functioning across domains improves when OCD symptoms have reduced, this may not be the case. Several studies provide evidence that CBT significantly improves quality of life in individuals with OCD, but the effect is small (Cordioli et al., 2003; Diefenbach

et al., 2007; Hofmann, Wu, Boettcher, & Sturm, 2014). This limited evidence suggests that CBT improves quality of life in OCD, but more studies are needed to fully support this notion and to incorporate other indices of functioning. Further, we are only aware of such studies focusing on CBT and encourage individuals studying other treatments for OCD to also evaluate changes in life functioning following treatment. In conclusion, it may be beneficial to develop standard expectations for treatment response in terms of both symptom severity and functioning.

6.10 Implications for Practice

In addition to considering the implications for research, it is also worthwhile to consider the clinical implications of the findings reviewed. Nevertheless, the classifications of treatments presented herein represent the current state of affairs and will change over time. As more studies are conducted, the classifications may change based on the findings. For example, one of the primary limitations of several approaches was a lack of studies that have tested the theory. This chapter, other scientific articles, and even National Institute of Mental Health grant requests call for more work to test the theoretically hypothesized mechanisms thought to underlie these interventions, and new findings will further inform our efforts to understand what are the best forms of treatment for OCD and which components of the treatments have the strongest effects.

Based on the current state of affairs, ERP, CT, CBT, ACT, and MCT were all classified in Category II (intervention-driven psychotherapies). The findings of this review are exciting, given that OCD was considered intractable less than 50 years ago. In contrast, there are now several good options for reducing OCD symptoms. One caveat is that the bulk of support for these interventions comes from assessing symptoms at the end of treatment, with much less data available over follow-up. Therefore, clinicians may want to consider building in ways to assess the durability of their clients' gains. For example, all clients could be scheduled for a standard follow-up session at a duration of the clinician's choosing (e.g., 1 month, 6 months). This practice would allow the clinician to evaluate whether additional intervention is needed and would also provide data on the durability of gains.

There has been a long-standing interest in identifying which treatments are most effective for which clients (see, e.g., Paul, 1969). However, related to the historically limited options for treating OCD, researchers have not yet addressed these questions with respect to OCD treatment in particular. Now that the number of viable treatments has grown, questions of which treatments will be best for which clients are becoming more prominent and warrant increased attention.

Some initial considerations may suggest relevant areas for study. For example, Abramowitz and Schwartz (2003) present an extensive list of potential factors to consider in selecting which treatment to use. These include factors related to symptom presentation, demographic factors, and environmental and social factors. Given the heterogeneity of symptoms in OCD, symptom subtypes may also play an important role in selecting a treatment (see Sookman, Abramowitz, Calamari, Wilhelm, & McKay, 2005). Further, characteristics such as age, level of education, and cognitive level may impact clients' abilities to engage in certain interventions or impact side-effect profiles from medications. More pragmatic factors, such as availability of providers

with adequate training in a given technique, will also influence which clients get which treatments. In addition, client preference is likely to play a role, as client engagement is one of the most potent predictors of treatment outcomes (Kazantzis & Lampropoulos, 2002; Kozak & Foa, 1997; Krupnick & Sotsky, 1996; Ledley, Marx, & Heimberg, 2011; Vogel, Hansen, Stiles, & Götestam, 2006). As we move forward, developing plans for collecting systematic data on these topics will be beneficial.

6.11 Conclusions

Using the criteria set forth by David and Montgomery (2011), we reviewed and categorized therapeutic interventions for OCD. This was an exciting task in that it emphasized how much progress has been made in treating OCD over a relatively short period of time. In addition to several pharmacological options, there are now several types of therapy with support. Interestingly, none of the therapies meet the highest criteria (Category I: evidence-based psychotherapies). This was largely due to a lack of sufficient studies adequately studying mediation and treatment mechanisms. As noted at the beginning of this chapter, there have been major advances in statistical methods for evaluating mediation. Embracing these sophisticated methods will promote significant advances in our understanding of which treatment components are pivotal and which components are superfluous. Modifications based on these findings could make treatments more powerful and/or reduce the number of sessions needed. Given the numerous treatment options, as noted, learning how to match specific treatments to particular clients could further enhance clinical outcomes.

To truly reduce the burdens of OCD, the development of efficacious treatments alone is not sufficient. The benefits of these interventions are only realized when clients receive them. Currently, only a small portion of individuals with OCD receive treatment (Coles & Coleman, 2010; Mayerovitch et al., 2003; Torres et al., 2007). Efforts to disseminate treatment to community mental health centers and to train more clinicians are an important step in helping more people access treatment. Efforts to reduce stigma regarding seeking help for mental health problems may also prove useful in encouraging more people to access treatment. Finally, increasing public recognition of OCD is also important and warrants additional attention. Efforts to increase treatment-seeking will allow more people to benefit from the treatments reviewed herein and contribute to improving the lives of individuals with OCD.

Note

1 In developing cognitive–behavioral therapy for OCD, the creators originally referred to the treatment as "exposure and response prevention." However, it was later recognized that the term "exposure and ritual prevention" would more accurately reflect the treatment. Therefore, we use the label "exposure and ritual prevention" herein.

References

Abramowitz, J. S., Foa, E. B., & Franklin, M. E. (2003). Exposure and ritual prevention for obsessive-compulsive disorder: Effects of intensive versus twice-weekly sessions. *Journal*

of Consulting and Clinical Psychology, *71*(2), 394–398. doi:10.1037/0022-006x.71.2.394

Abramowitz, J. S., Nelson, C. A., Rygwall, R., & Khandker, M. (2007). The cognitive mediation of obsessive-compulsive symptoms: A longitudinal study. *Journal of Anxiety Disorders*, *21*(1), 91–104. doi:10.1016/j.janxdis.2006.05.003

Abramowitz, J. S., & Schwartz, S. A. (2003). Treatments for obsessive-compulsive disorder: Deciding what method for whom. *Brief Treatment*, *3*(2), 261–274. doi:10.1093/brief-treatment/mhg019

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.

Amir, N., Kuckertz, J. M., Najmi, S., & Conley, S. L. (2015). Preliminary evidence for the enhancement of self-conducted exposures for OCD using cognitive bias modification. *Cognitive Therapy and Research*, *39*, 424–440. doi:10.1007/s10608-015-9675-7

Amir, N., Najmi, S., & Morrison, A. S. (2009). Attenuation of attention bias in obsessive-compulsive disorder. *Behaviour Research and Therapy*, *47*(2), 153–157. doi:10.1016/j.brat.2008.10.020

Andouz, Z., Dolatshahi, B., Moshtagh, N., & Dadkhah, A. (2012). The efficacy of metacognitive therapy on patients suffering from pure obsession. *Iranian Journal of Psychiatry*, *7*(1), 11–21.

Baker, A., Mystkowski, J., & Culver, N. (2010). Does habituation matter? Emotional processing theory and exposure therapy for acrophobia. *Behaviour Research and Therapy*, *48*(11), 1139–1143. doi:10.1016/j.brat.2010.07.009

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & Van IJzendoorn, M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: A meta-analytic study. *Psychological Bulletin*, *133*(1), 1–24. doi:10.1037/0033-2909.133.1.1

Barron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, *51*, 1173–1183. doi:10.1037//0022-3514.51.6.1173

Beadel, J. R., Smyth, F. L., & Teachman, B. A. (2014). Change processes during cognitive bias modification for obsessive compulsive beliefs. *Cognitive Therapy and Research*, *38*(2), 103–119. doi:10.1007/s10608-013-9576-6

Beck, A. (1963). Thinking and depression: I. Idiosyncratic content and cognitive distortions. *Archives of General Psychiatry*, *9*(4), 324. doi:10.1001/archpsyc.1963.01720160014002

Beck, A., Emery, G., & Greenberg, R. (1985). *Anxiety disorders and phobias: A cognitive approach*. New York: Basic Books.

Belloch, A., Cabedo, E., & Carrio, C. (2008). Cognitive versus behaviour therapy in the individual treatment of obsessive-compulsive disorder: Changes in cognitions and clinically significant outcomes at post-treatment and one-year follow-up. *Behavioural and Cognitive Psychotherapy*, *36*(5), 521–540. doi:10.1017/s1352465808004451

Belloch, A., Cabedo, E., Carrió, C., & Larsson, C. (2010). Cognitive therapy for autogenous and reactive obsessions: Clinical and cognitive outcomes at post-treatment and 1-year follow-up. *Journal of Anxiety Disorders*, *24*(6), 573–580. doi:10.1016/j.janxdis.2010.03.017

Bluett, E. J., Homan, K. J., Morrison, K. L., Levin, M. E., & Twohig, M. P. (2014). Acceptance and commitment therapy for anxiety and OCD spectrum disorders: An empirical review. *Journal of Anxiety Disorders*, *28*(6), 612–624. doi:10.1016/j.janxdis.2014.06.008

Bouton, M. (2000). A learning theory perspective on lapse, relapse, and the maintenance of behavior change. *Health Psychology*, *19*(1S), 57–63. doi:10.1037//0278-6133.19.suppl1.57

Bower, G. H. (1981). Mood and memory. *American Psychologist*, *36*(2), 129–148. doi:10.1037/0003-066X.36.2.129

Calvocoressi, L., Lewis, B., Harris, M., & Trufan, S. J. (1995). Family accommodation in obsessive-compulsive disorder. *American Journal of Psychiatry*, *152*(3), 441–443. doi:10.1176/ajp.152.3.441

Cisler, J., & Olatunji, B. (2010). Components of attentional biases in contamination fear: Evidence for difficulty in disengagement. *Behaviour Research and Therapy*, *48*(1), 74–78. doi:10.1016/j.brat.2009.09.003

Clerkin, E. M., & Teachman, B. A. (2011). Training interpretation biases among individuals with symptoms of obsessive compulsive disorder. *Journal of Behavior Therapy and Experimental Psychiatry*, *42*(3), 337–343. doi:10.1016/j.jbtep.2011.01.003

Coles, M. E., & Coleman, S. L. (2010). Barriers to treatment seeking for anxiety disorders: Initial data on the role of mental health literacy. *Depression and Anxiety*, *27*(1), 63–71. doi:10.1002/da.20620

Cordioli, A. V., Heldt, E., Bochi, D. B., Margis, R., de Sousa, M. B., Tonello, J. F., … Kapczinski, F. (2003). Cognitive-behavioral group therapy in obsessive-compulsive disorder: A randomized clinical trial. *Psychotherapy and Psychosomatics*, *72*(4), 211–216. doi:10.1159/000070785

Cottraux, J., Note, I., Yao, S. N., Lafont, S., Note, B., Mollard, E., … Dartigues, J.-F. (2001). A randomized controlled trial of cognitive therapy versus intensive behavior therapy in obsessive compulsive disorder. *Psychotherapy and Psychosomatics*, *70*(6), 288–297. doi:10.1159/000056269

Cottraux, J., Yao, S. N., Lafont, S., Mollard, E., Bouvard, M., Sauteraud, A., … Dartigues, J.-F. (2001). A randomized controlled trial of cognitive therapy versus intensive behavior therapy in obsessive compulsive disorder. *Psychotherapy and Psychosomatics*, *70*(6), 288–297.

Craske, M., Kircanski, K., Zelikowsky, M., Mystkowski, J., Chowdhury, N., & Baker, A. (2008). Optimizing inhibitory learning during exposure therapy. *Behaviour Research and Therapy*, *46*(1), 5–27. doi:10.1016/j.brat.2007.10.003

David, D., & Montgomery, G. (2011). The scientific status of psychotherapies: A new evaluative framework for evidence-based psychosocial interventions. *Clinical Psychology: Science and Practice*, *18*(2), 89–99. doi:10.1111/j.1468-2850.2011.01239.x

Dehlin, J. P., Morrison, K. L., & Twohig, M. P. (2013). Acceptance and commitment therapy as a treatment for scrupulosity in obsessive compulsive disorder. *Behavior Modification*, *37*(3), 409–430. doi:10.1177/0145445512475134

Diefenbach, G. J., Abramowitz, J. S., Norberg, M. M., & Tolin, D. F. (2007). Changes in quality of life following cognitive-behavioral therapy for obsessive-compulsive disorder. *Behaviour Research and Therapy*, *45*(12), 3060–3068. doi:10.1016/j.brat.2007.04.014

DuPont, R. L., Rice, D. P., Miller, L. S., Shiraki, S. S., Rowland, C. R., & Harwood, H. J. (1996). Economic costs of anxiety disorders. *Anxiety*, *2*, 167–172. doi:10.1002/(sici)1522-7154(1996)2:4<167::aid-anxi2>3.0.co;2-l

Eddy, K. T., Dutra, L., Bradley, R., & Westen, D. (2004). A multidimensional meta-analysis of psychotherapy and pharmacotherapy for obsessive-compulsive disorder. *Clinical Psychology Review*, *24*(8), 1011–1030. doi:10.1016/j.cpr.2004.08.004

Eisen, J. L., Goodman, W. K., Keller, M. B., Warshaw, M. G., DeMarco, L. M., Luce, D. D., & Rasmussen, S. A. (1999). Patterns of remission and relapse in obsessive-compulsive disorder: A 2-year prospective study. *Journal of Clinical Psychiatry*, *60*(5), 346–351. doi:10.4088/jcp.v60n0514

Eisen, J. L., Mancebo, M. A., Pinto, A., Coles, M. E., Pagano, M. E., Stout, R., & Rasmussen, S. A. (2006). Impact of obsessive-compulsive disorder on quality of life. *Comprehensive Psychiatry*, *47*(4), 270–275. doi:10.1016/j.comppsych.2005.11.006

Eisen, J. L., & Rasmussen, S. A. (2002). Phenomenology of obsessive compulsive disorder. In D. J. Stein & E. Hollander (Eds.), *Textbook of anxiety disorders* (pp. 261–286). Washington, DC: American Psychiatric Press.

Emmelkamp, P., & Beens, H. (1991). Cognitive therapy with obsessive compulsive disorder: A comparative evaluation. *Behaviour Research and Therapy*, *29*(3), 293–300. doi:10.1016/0005-7967(91)90120-r

Emmelkamp, P., Visser, S., & Hoekstra, R. (1988). Cognitive therapy vs exposure in vivo in the treatment of obsessive-compulsives. *Cognitive Therapy and Research*, *12*(1), 103–114. doi:10.1007/bf01172784

Fineberg, N. A., Fourie, H., Gale, T. M., & Sivakumaran, T. (2005). Comorbid depression in obsessive compulsive disorder (OCD): Symptomatic differences to major depressive disorder. *Journal of Affective Disorders*, *87*(2/3), 327–330. doi:10.1016/j.jad.2005.04.004

Fisher, P. L., & Wells, A. (2005). Experimental modification of beliefs in obsessive-compulsive disorder: A test of the metacognitive model. *Behaviour Research and Therapy*, *43*(6), 821–829. doi:10.1016/j.brat.2004.09.002

Fisher, P. L., & Wells, A. (2008). Metacognitive therapy for obsessive–compulsive disorder: A case series. *Journal of Behavior Therapy and Experimental Psychiatry*, *39*(2), 117–132. doi:10.1016/j.jbtep.2006.12.001

Fitt, S., & Rees, C. (2012). Metacognitive therapy for obsessive compulsive disorder by videoconference: A preliminary study. *Behaviour Change*, *29*(4), 213–229. doi:10.1017/bec.2012.21

Foa, E. B., & Goldstein, A. J. (1978). Continuous exposure and complete response prevention in the treatment of obsessive-compulsive neurosis. *Behavior Therapy*, *9*(5), 821–829. doi:10.1016/S0005-7894(78)80013-6

Foa, E. B., Huppert, J. D., & Cahill, S. P. (2006). Emotional processing theory: An update. In B. O. Rothbaum (Ed.), *Pathological anxiety: Emotional processing in etiology and treatment* (pp. 3–24). New York, NY: Guilford Press.

Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin*, *99*(1), 20–35. doi:10.1037/0033-2909.99.1.20

Foa, E. B., Kozak, M. J., Barlow, D., Cuthbert, B., Foa, U., Hallam, D., … Persons, J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin*, *99*(1), 20–35. doi:10.1037//0033-2909.99.1.20

Foa, E. B., Liebowitz, M. R., Kozak, M. J., Davies, S., Campeas, R., Franklin, M. E., … Schmidt, A. B. (2005). Randomized, placebo-controlled trial of exposure and ritual prevention, clomipramine, and their combination in the treatment of obsessive-compulsive disorder. *American Journal of Psychiatry*, *162*(1), 151–161.

Franklin, M. E., & Foa, E. B. (2002). Cognitive behavioral treatments for obsessive compulsive disorder. In P. E. Nathan & J. M. Gorman (Eds.), *A guide to treatments that work* (2nd ed., pp. 367–386). London, UK: Oxford University Press.

Freeston, M. H., Ladouceur, R., Gagnon, F., Thibodeau, N., Rhéaume, J., Letarte, H., & Bujold, A. (1997). Cognitive-behavioral treatment of obsessive thoughts: A controlled study. *Journal of Consulting and Clinical Psychology*, *65*(3), 405–413. doi:10.1037//0022-006x.65.3.405

Gotlib, I., & McCann, C. (1984). Construct accessibility and depression: An examination of cognitive and affective factors. *Journal of Personality and Social Psychology*, *47*(2), 427–439. doi:10.1037//0022-3514.47.2.427

Grisham, J. R., Becker, L., Williams, A. D., Whitton, A. E., & Makkar, S. R. (2014). Using cognitive bias modification to deflate responsibility in compulsive checkers. *Cognitive Therapy and Research*, *38*(5), 505–517. doi:10.1007/s10608-014-9621-0

Guy, W. (1976). *ECDEU assessment manual for psychopharmacology* (Rev. ed.). Rockville, MD: National Institute of Mental Health.

Hakamata, Y., Lissek, S., Bar-Haim, Y., Britton, J. C., Fox, N. A., Leibenluft, E., … Pine, D. S. (2010). Attention bias modification treatment: A meta-analysis toward the establishment of novel treatment for anxiety. *Biological Psychiatry*, *68*(11), 982–990. doi:10.1016/j.biopsych.2010.07.021

Hazen, R. A., Vasey, M. W., & Schmidt, N. B. (2009). Attentional retraining: A randomized clinical trial for pathological worry. *Journal of Psychiatric Research*, *43*(6), 627–633. doi:10.1016/j.jpsychires.2008.07.004

Hofmann, S. G., Wu, J. Q., Boettcher, H., & Sturm, J. (2014). Effect of pharmacotherapy for anxiety disorders on quality of life: A meta-analysis. *Journal of Consulting and Clinical Psychology*, *23*(3), 1141–1153. doi:10.1007/s11136-013-0573-8

Kazantzis, N., & Lampropoulos, G. (2002). Reflecting on homework in psychotherapy: What can we conclude from research and experience? *Journal of Clinical Psychology*, *58*(5), 577–585. doi:10.1002/jclp.10034

Kazdin, A. (2007). Mediators and mechanisms of change in psychotherapy research. *Annual Review of Clinical Psychology*, *3*, 1–27. doi:10.1146/annurev.clinpsy.3.022806.091432

Kazdin, A. (2009). Understanding how and why psychotherapy leads to change. *Psychotherapy Research*, *19*(4-5), 418–428. doi:10.1080/10503300802448899

Koran, L. M., Thienemann, M. L., & Davenport, R. (1996). Quality of life for patients with obsessive-compulsive disorder. *American Journal of Psychiatry*, *153*(6), 783–788. doi:10.1176/ajp.153.6.783

Koster, E., Crombez, G., Van Damme, S., Verschuere, B., & De Houwer, J. (2005). Signals for threat modulate attentional capture and holding: Fear-conditioning and extinction during the exogenous cueing task. *Cognition and Emotion*, *19*(5), 771–780. doi:10.1080/02699930441000418

Kozak, M. J., & Foa, E. B. (1997). Treatment planning: Understanding and persuasion. In *Mastery of obsessive-compulsive disorder: A cognitive-behavioral approach* (pp. 43–57). New York, NY: Oxford University Press.

Kozak, M. J., Foa, E. B., & Steketee, G. (1988). Process and outcome of exposure treatment with obsessive-compulsives: Psychophysiological indicators of emotional processing. *Behavior Therapy*, *19*, 157–169. doi:10.1016/s0005-7894(88)80039-x

Krupnick, J., & Sotsky, S. (1996). The role of the therapeutic alliance in psychotherapy and pharmacotherapy outcome: Findings in the National Institute of Mental Health Treatment of Depression. *Journal of Consulting and Clinical Psychology*, *64*(3), 532–539. doi:10.1037//0022-006x.64.3.532

Ledley, D., Marx, B., & Heimberg, R. (2011). Introducing cognitive-behavioral process. In *Making cognitive-behavioral therapy work: Clinical process for new practitioners* (pp. 1–20). New York, NY: Guilford.

MacLeod, C., & Mathews, A. (1988). Anxiety and the allocation of attention to threat. *Quarterly Journal of Experimental Psychology*, *40*(4), 653–670. doi:10.1080/14640748808402292

MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology*, *95*(1), 15–20. doi:10.1037/0021-843X.95.1.15

MacLeod, C., Rutherford, E., Campbell, L., Ebsworthy, G., & Holker, L. (2002). Selective attention and emotional vulnerability: Assessing the causal basis of their association through the experimental manipulation of attentional bias. *Journal of Abnormal Psychology*, *111*(1), 107–123. doi:10.1037//0021-843x.111.1.107

MacLeod, C., Soong, L., Rutherford, E., & Campbell, L. (2007). Internet-delivered assessment and manipulation of anxiety-linked attentional bias: Validation of a free-access attentional probe software package. *Behavior Research Methods*, *39*, 533–538.

Mathews, A., & Mackintosh, B. (2000). Induced emotional interpretation bias and anxiety. *Journal of Abnormal Psychology*, *109*(4), 602–615. doi:10.1037//0021-843x.109.4.602

Mathews, A., & MacLeod, C. (1985). Selective processing of threat cues in anxiety states. *Behaviour Research and Therapy*, *23*(5), 563–569. doi:10.1016/0005-7967(85)90104-4

Mathews, A., & MacLeod, C. (2005). Cognitive vulnerability to emotional disorders. *Annual Review of Clinical Psychology*, *1*(1), 167–195. doi:10.1146/annurev.clinpsy.1.102803.143916

Mayerovitch, J. I., du Fort, G. G., Kakuma, R., Bland, R. C., Newman, S. C., & Pinard, G. (2003). Treatment seeking for obsessive-compulsive disorder: Role of obsessive-compulsive disorder symptoms and comorbid psychiatric diagnoses. *Comprehensive Psychiatry*, *44*(2), 162–168. doi:10.1053/comp.2003.50005

McLean, P. D., Whittal, M. L., Thordarson, D. S., Taylor, S., Söchting, I., Koch, W. J., … Anderson, K. W. (2001). Cognitive versus behavior therapy in the group treatment of obsessive-compulsive disorder. *Journal of Consulting and Clinical Psychology*, *69*(2), 205–214. doi:10.1037/0022-006X.69.2.205

Menne-Lothmann, C., Viechtbauer, W., Hohn, P., Kasanova, Z., Haller, S., Drukker, M., … Lau, J. (2014). How to boost positive interpretations? A meta-analysis of the effectiveness of cognitive bias modification for interpretation. *PLoS ONE*, *9*(6), e100925. doi:10.1371/journal.pone.0100925

Mogg, K., & Bradley, B. P. (1998). A cognitive-motivational analysis of anxiety. *Behaviour Research and Therapy*, *36*(9), 809–848. doi:10.1016/s0005-7967(98)00063-1

Mogoaşe, C. (2014). Clinical efficacy of attentional bias modification procedures: An updated meta-analysis. *Journal of Clinical Psychology*, *70*(12), 1133–1157. doi:10.1002/jclp.22081

Moritz, S., Jelinek, L., Hauschildt, M., & Naber, D. (2010). How to treat the untreated: Effectiveness of a self-help metacognitive training program (myMCT) for obsessive-compulsive disorder. *Dialogues in Clinical Neuroscience*, *12*(2), 209–220.

Moritz, S., Rufer, M., Fricke, S., Karow, A., Morfeld, M., Jelinek, L., & Jacobsen, D. (2005). Quality of life in obsessive-compulsive disorder before and after treatment. *Comprehensive Psychiatry*, *46*(6), 453–459. doi:10.1016/j.comppsych.2005.04.002

Muller, J., & Roberts, J. E. (2005). Memory and attention in obsessive-compulsive disorder: A review. *Journal of Anxiety Disorders*, *19*(1), 1–28. doi:10.1016/j.janxdis.2003.12.001

Murray, C., & Lopez, A. (1996). Evidence-based health policy: Lessons from the Global Burden of Disease Study. *Science*, *274*(5288), 740–743. doi:10.1126/science.274.5288.740

Myers, S. G., & Wells, A. (2013). An experimental manipulation of metacognition: A test of the metacognitive model of obsessive-compulsive symptoms. *Behaviour Research and Therapy*, *51*(4/5), 177–184. doi:10.1016/j.brat.2013.01.007

Najmi, S., & Amir, N. (2010). The effect of attention training on a behavioral test of contamination fears in individuals with subclinical obsessive-compulsive symptoms. *Journal of Abnormal Psychology*, *119*(1), 136–142. doi:10.1037/a0017549

Nazari, H., Momeni, N., Jariani, M., & Tarrahi, M. J. (2011). Comparison of eye movement desensitization and reprocessing with citalopram in treatment of obsessive–compulsive disorder. *International Journal of Psychiatry in Clinical Practice*, *15*(4), 270–274. doi:10.3109/13651501.2011.590210

O'Connor, K., Todorov, C., Robillard, S., Borgeat, F., & Brault, M. (1999). Cognitive-behaviour therapy and medication in the treatment of obsessive-compulsive disorder: A controlled study. *Canadian Journal of Psychiatry/Revue Canadienne de Psychiatrie*, *44*(1), 64–71.

Obsessive Compulsive Cognitions Working Group. (2001). Development and initial validation of the Obsessive Beliefs Questionnaire and the Interpretation of Intrusions Inventory. *Behaviour Research and Therapy*, *39*(8), 987–1006. doi:10.1016/s0005-7967(00)00085-1

Olatunji, B., Davis, M., Powers, M., & Smits, J. (2013). Cognitive-behavioral therapy for obsessive-compulsive disorder: A meta-analysis of treatment outcome and moderators. *Journal of Psychiatric Research*, *47*(1), 33–41.

Olatunji, B. O., Rosenfield, D., Tart, C. D., Cottraux, J., Powers, M. B., & Smits, J. A. J. (2013). Behavioral versus cognitive treatment of obsessive-compulsive disorder: An examination of outcome and mediators of change. *Journal of Consulting and Clinical Psychology*, *81*(3), 415–428. doi:10.1037/a0031865

Orlinsky, D., Grawe, K., & Parks, B. (1994). Process and outcome in psychotherapy: Noch einmal. In A. Bergin & J. S. Garfield (Eds.), *Handbook of psychotherapy and behaviour change* (4th ed., pp. 270–378). New York, NY: John Wiley & Sons.

Öst, L., Havnen, A., Hansen, B., & Kvale, G. (2015). Cognitive behavioral treatments of obsessive–compulsive disorder: A systematic review and meta-analysis of studies published 1993–2014. *Clinical Psychology Review*, *40*, 156–169. doi:10.1016/j.cpr.2015.06.003

Overton, S. M., & Menzies, R. G. (2005). Cognitive change during treatment of compulsive checking. *Behaviour Change*, *22*(3), 172–184. doi:10.1375/bech.2005.22.3.172

Paul, G. (1969). Behavior modification research: Design and tactics. In C. M. Franks (Ed.), *Behavior therapy: Appraisal and status* (pp. 29–62). New York, NY: McGraw-Hill.

Perkins, B. R., & Rouanzoin, C. C. (2002). A critical evaluation of current views regarding eye movement desensitization and reprocessing (EMDR): Clarifying points of confusion. *Journal of Clinical Psychology*, *58*(1), 77–97. doi:10.1002/jclp.1130

Pietrefesa, A. S., & Coles, M. E. (2008). Moving beyond an exclusive focus on harm avoidance in obsessive compulsive disorder: Considering the role of incompleteness. *Behavior Therapy*, *39*(3), 224–231. doi:10.1016/j.beth.2007.08.004

Pinto, A., Mancebo, M. C., Eisen, J. L., Pagano, M. E., & Rasmussen, S. A. (2006). The Brown Longitudinal Obsessive Compulsive Study: Clinical features and symptoms of the sample at intake. *Journal of Clinical Psychiatry*, *67*(05), 703–711. doi:10.4088/jcp.v67n0503

Price, L. H., Rasmussen, S. A., & Eisen, E. J. (1999). The natural history of obsessive-compulsive disorder. *Archives of General Psychiatry*, *56*(2), 131–132.

Rachman, S. (1993). Obsessions, responsibility and guilt. *Behaviour Research and Therapy*, *31*(2), 149–154. doi:10.1016/0005-7967(93)90066-4

Rachman, S. (1997). A cognitive theory of obsessions. *Behaviour Research and Therapy*, *35*(9), 793–802. doi:10.1016/S0005-7967(97)00040-5

Rees, C. S., & Van Koesveld, K. E. (2008). An open trial of group metacognitive therapy for obsessive-compulsive disorder. *Journal of Behavior Therapy and Experimental Psychiatry*, *39*(4), 451–458. doi:10.1016/j.jbtep.2007.11.004

Rescorla, R. (2001). Experimental extinction. In R. Mowrer & S. Klein (Eds.), *Handbook of contemporary learning theories* (pp. 119–154). Mahwah, NJ: Lawrence Erlbaum Associates.

Riggs, D. S., Hiss, H., & Foa, E. B. (1992). Marital distress and the treatment of obsessive compulsive disorder. *Behavior Therapy*, *23*(4), 585–597. doi:10.1016/S0005-7894(05)80223-0

Salkovskis, P. (1985). Obsessional-compulsive problems: A cognitive-behavioural analysis. *Behaviour Research and Therapy*, *23*(5), 571–583. doi:10.1016/0005-7967(85)90105-6

Salkovskis, P. M. (1989). Cognitive-behavioural factors and the persistence of intrusive thoughts in obsessional problems. *Behaviour Research and Therapy*, *27*(6), 677–682. doi:10.1016/0005-7967(89)90152-6

Samara, Z., Elzinga, B. M., Slagter, H. A., & Nieuwenhuis, S. (2011). Do horizontal saccadic eye movements increase interhemispheric coherence? Investigation of a hypothesized neural mechanism underlying EMDR. *Frontiers in Psychiatry*, *2*, 4. doi:10.3389/fpsyt.2011.00004

Shapiro, F., & Maxfield, L. (2002). Eye movement desensitization and reprocessing (EMDR): Information processing in the treatment of trauma. *Journal of Clinical Psychology*, *58*(8), 933–946. doi:10.1002/jclp.10068

Shapiro, K. (2001). *The limits of attention: Temporal constraints in human information processing*. Oxford, UK: Oxford University Press.

Simon, G., Ormel, J., VonKorff, M., & Barlow, W. (1995). Health care costs associated with depressive and anxiety disorders in primary care. *American Journal of Psychiatry*, *152*(3), 352–357. doi:10.1176/ajp.152.3.352

Simpson, H. B., Foa, E. B., Liebowitz, M. R., Huppert, J. D., Cahill, S., Maher, M. J., … Campeas, R. (2013). Cognitive-behavioral therapy vs risperidone for augmenting serotonin reuptake inhibitors in obsessive-compulsive disorder. *JAMA Psychiatry*, *70*(11), 1190. doi:10.1001/jamapsychiatry.2013.1932

Simpson, H. B., & Franklin, M. (2005). Standard criteria for relapse are needed in obsessive–compulsive disorder. *Depression and Anxiety*, *21*(1), 1–8. doi:10.1002/da.20052

Simpson, H. B., Huppert, J. D., Petkova, E., Foa, E. B., & Liebowitz, M. R. (2006). Response versus remission in obsessive-compulsive disorder. *Journal of Clinical Psychiatry*, *67*(02), 269–276. doi:10.4088/jcp.v67n0214

Solem, S., Håland, Å. T., Vogel, P. A., Hansen, B., & Wells, A. (2009). Change in metacognitions predicts outcome in obsessive–compulsive disorder patients undergoing

treatment with exposure and response prevention. *Behaviour Research and Therapy*, *47*(4), 301–307. doi:10.1016/j.brat.2009.01.003

Sookman, D., Abramowitz, J. S., Calamari, J. E., Wilhelm, S., & McKay, D. (2005). Subtypes of obsessive-compulsive disorder: Implications for specialized cognitive behavior therapy. *Behavior Therapy*, *36*(4), 393–400. doi:10.1016/S0005-7894(05)80121-2

Sousa, M. B., Isolan, L. R., Oliveira, R. R., Manfro, G. G., & Cordioli, A. V. (2006). A randomized clinical trial of cognitive-behavioral group therapy and sertraline in the treatment of obsessive-compulsive disorder. *Journal of Clinical Psychiatry*, *67*(7), 1133–1139. doi:10.4088/JCP.v67n0717

Steketee, G., Frost, R., Bhar, S., Bouvard, M., Calamari, J., Carmin, C., … Forrester, E. (2003). Psychometric validation of the Obsessive Beliefs Questionnaire and the Interpretation of Intrusions Inventory: Part I. *Behaviour Research and Therapy*, *41*(8), 863–878. doi:10.1016/s0005-7967(02)00099-2

Stroop, J. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology*, *18*(6), 643–662. doi:10.1037/h0054651

Su, Y. J., Carpenter, J. K., Zandberg, L. J., Simpson, H. B., & Foa, E. B. (2016). Cognitive mediation of symptom change in exposure and response prevention for obsessive-compulsive disorder. *Behavior Therapy*, *47*(4), 474–486. doi:10.1016/j.beth.2016.03.003

Tallis, F. (1997). The neuropsychology of obsessive-compulsive disorder: A review and consideration of clinical implications. *British Journal of Clinical Psychology*, *36*(1), 3–20. doi:10.1111/j.2044-8260.1997.tb01226.x

Tolin, D. F., Abramowitz, J. S., & Diefenbach, G. J. (2005). Defining response in clinical trials for obsessive-compulsive disorder: A signal detection analysis of the Yale–Brown Obsessive Compulsive Scale. *Journal of Clinical Psychiatry*, *66*(12), 1549–1557. doi:10.4088/jcp.v66n1209

Torres, A. R., Moran, P., Bebbington, P., Brugha, T., Bhugra, D., Coid, J. W., … Prince, M. (2006). Obsessive-compulsive disorder and personality disorder: Evidence from the British National Survey of Psychiatric Morbidity 2000. *Social Psychiatry and Psychiatric Epidemiology*, *41*(11), 862–867. doi:10.1007/s00127-006-0118-3

Torres, A. R., Prince, M. J., Bebbington, P. E., Bhugra, D. K., Brugha, T. S., Farrell, M., … Singleton, N. (2007). Treatment seeking by individuals with obsessive-compulsive disorder from the British Psychiatric Morbidity Survey of 2000. *Psychiatric Services (Washington, DC)*, *58*(7), 977–982. doi:10.1176/appi.ps.58.7.977

Twohig, M. P. (2009). The application of acceptance and commitment therapy to obsessive-compulsive disorder. *Cognitive and Behavioral Practice*, *16*(1), 18–28. doi:10.1016/j.cbpra.2008.02.008

Twohig, M. P., Hayes, S. C., & Masuda, A. (2006). Increasing willingness to experience obsessions: Acceptance and commitment therapy as a treatment for obsessive-compulsive disorder. *Behavior Therapy*, *37*(1), 3–13. doi:10.1016/j.beth.2005.02.001

Twohig, M. P., Hayes, S. C., Plumb, J. C., Pruitt, L. D., & Collins, A. B. (2011). A randomized clinical trial of acceptance and commitment therapy vs. progressive relaxation training for obsessive compulsive disorder. *Journal of Consulting and Clinical Psychology*, *78*(5), 705–716. doi:10.1037/a0020508.A

Twohig, M. P., Vilardaga, J. C. P., Levin, M. E., & Hayes, S. C. (2015). Changes in psychological flexibility during acceptance and commitment therapy for obsessive

compulsive disorder. *Journal of Contextual Behavioral Science*, *4*(3), 196–202. doi:10.1016/j.jcbs.2015.07.001

Van Balkom, A. J., de Haan, E., Van Oppen, P., Spinhoven, P., Hoogduin, K. A., & Van Dyck, R. (1998). Cognitive and behavioral therapies alone versus in combination with fluvoxamine in the treatment of obsessive compulsive disorder. *Journal of Nervous and Mental Disease*, *186*(8), 492–499. doi:10.1097/00005053-199808000-00007

Van Oppen, P., & Arntz, A. (1994). Cognitive therapy for obsessive-compulsive disorder. *Behaviour Research and Therapy*, *32*(1), 79–87. doi:10.1016/0005-7967(94)90086-8

Van Oppen, P., & de Haan, E. (1995). Cognitive therapy and exposure in vivo in the treatment of obsessive compulsive disorder. *Behaviour Research and Therapy*, *33*(4), 379–390.

Vogel, P., Hansen, B., Stiles, T., & Götestam, K. (2006). Treatment motivation, treatment expectancy, and helping alliance as predictors of outcome in cognitive behavioral treatment of OCD. *Journal of Behavior Therapy and Experimental Psychiatry*, *37*(3), 247–255. doi:10.1016/j.jbtep.2005.12.001

Vogel, P. A., Stiles, T. C., & Gunnar, K. G. (2004). Adding Cognitive therapy elements to exposure therapy for obsessive compulsive disorder: A controlled study. *Behavioural and Cognitive Psychotherapy*, *32*(3), 275–290. doi:10.1017/S1352465804001353

Vos, S. P. F., Huibers, M. J. H., & Arntz, A. (2012). Experimental investigation of targeting responsibility versus danger in cognitive therapy of obsessive-compulsive disorder. *Depression and Anxiety*, *29*(7), 629–637. doi:10.1002/da.21915

Weissman, M. (1998). Cross-national epidemiology of obsessive-compulsive disorder. *CNS Spectrums*, *3*(Suppl. 1), 6–9. doi:10.1017/S1092852900007136

Wells, A. (2000). *Emotional disorders and metacognition: Innovative cognitive therapy*. Chichester, UK: John Wiley & Sons.

Wells, A. (2013). *Cognitive therapy of anxiety disorders: A practice manual and conceptual guide*. Chichester, UK: John Wiley & Sons.

Wells, A., & Matthews, G. (1994). *Attention and emotion: A clinical perspective*. Hove, UK: Lawrence Erlbaum Associates.

Whittal, M. L., Thordarson, D. S., & McLean, P. D. (2005). Treatment of obsessive–compulsive disorder: Cognitive behavior therapy vs. exposure and response prevention. *Behaviour Research and Therapy*, *43*(12), 1559–1576. doi:10.1016/j.brat.2004.11.012

Wilhelm, S., Berman, N. C., Keshaviah, A., Schwartz, R. A., & Steketee, G. (2015). Mechanisms of change in cognitive therapy for obsessive compulsive disorder: Role of maladaptive beliefs and schemas. *Behaviour Research and Therapy*, *65*, 5–10. doi:10.1016/j.brat.2014.12.006

Wilhelm, S., & Steketee, G. (2006). *Cognitive therapy for obsessive compulsive disorder: A guide for professionals*. Oakland, CA: New Harbinger Publications.

Wilhelm, S., Steketee, G., Fama, J. M., Buhlmann, U., Teachman, B. A., & Golan, E. (2009). Modular cognitive therapy for obsessive-compulsive disorder: A wait-list controlled trial. *Journal of Cognitive Psychotherapy*, *23*(4), 294–305.

Wilhelm, S., Steketee, G., Reilly-Harrington, N. A., Deckersbach, T., Buhlmann, U., & Baer, L. (2005). Effectiveness of cognitive therapy for obsessive-compulsive disorder: An open trial. *Journal of Cognitive Psychotherapy*, *19*(2), 173-179. doi:10.1891/jcop.19.2.173.66792

Williams, A. D., & Grisham, J. R. (2013). Cognitive bias modification (CBM) of obsessive compulsive beliefs. *BMC Psychiatry*, *13*(1), 256. doi:10.1186/1471-244X-13-256

Williams, J. M. G., Watts, F. N., MacLeod, C., & Mathews, A. (1988). *Cognitive psychology and emotional disorders* (1st ed.). Chichester, UK: John Wiley & Sons.

Williams, J. M. G., Watts, F. N., MacLeod, C., & Mathews, A. (1997). *Cognitive psychology and emotional disorders* (2nd ed.). Chichester, UK: John Wiley & Sons.

Yiend, J., & Mathews, A. (2001). Anxiety and attention to threatening pictures. *Quarterly Journal of Experimental Psychology*, *54*(3), 665–681. doi:10.1080/02724980042000462

7

Evidence-Based Practice for Posttraumatic Stress Disorder

B. Christopher Frueh,[1] Anouk L. Grubaugh,[2] Alok Madan,[3] Sandra M. Neer,[4] Jon D. Elhai,[5] and Deborah C. Beidel[4]

[1] *Department of Psychology, University of Hawaii, Hilo, Hawaii*
[2] *Department of Psychiatry, Medical University of South Carolina, Charleston, USA*
[3] *Department of Psychiatry, Baylor College of Medicine, Houston, USA*
[4] *Department of Psychology, University of Central Florida, Orlando, USA*
[5] *Department of Psychology, University of Toledo, Toledo, USA*

7.1 Overview

Since posttraumatic stress disorder (PTSD) was first added to the diagnostic lexicon of psychiatry in 1980, a wide range of interventions have been developed or adapted to treat the core symptoms of the disorder, as well as frequently co-occurring symptoms (e.g., sleep impairment, family discord) and disorders (e.g., major depression). These interventions have included variations on traditional psychotherapies (e.g., cognitive, behavioral, psychodynamic, humanistic), psychiatric medications, neuromodulation and stimulation interventions, new psychotherapies ("energy" therapies), and use of a wide range of complementary and alternative medicine approaches (e.g., yoga, acupuncture, equine therapy). We will consider the evidence for psychotherapies using the framework suggested by David and Montgomery (2011), which considers both the extent of support for the treatment package and the mechanisms hypothesized to mediate or moderate treatment outcomes. According to this classification framework, treatments range across nine categories that span well-validated theory and well-validated treatment package (Category I) to bad-theory- and bad-intervention-driven psychotherapy (Category IX).

In addition, we will briefly review the evidence for other PTSD interventions so as to provide a comprehensive summary of treatment options for the disorder. Because there are virtually no rigorous empirical data on complementary and alternative medicine approaches, we will not review these interventions, although we note their use has become widespread in recent years.

PTSD is now included in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) in a section titled "Trauma and Stress-Related Disorders," which also contains acute stress disorder and the adjustment disorders (American Psychiatric Association, 2013). Both PTSD and acute stress disorder were previously classified under the "anxiety disorders" in the DSM-IV, while the adjustment disorders were

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

classified as a residual diagnostic category (American Psychiatric Association, 1994). PTSD is a severe psychiatric disorder resulting from a history of exposure to a traumatic event (Criterion A) that results in a minimum threshold of symptoms across four symptom clusters: intrusion, avoidance, negative alterations in cognitions and mood, and alterations in arousal and reactivity (Criteria B through E). Additional criteria relate to duration of symptoms (Criterion F), functioning (Criterion G), and differential diagnosis due to a substance or other co-occurring condition (Criterion H).

It is well accepted that many different events may lead to the onset of PTSD, including sexual assault, combat, natural disasters, motor vehicle accidents, and witnessing horrific events experienced by others. More specifically for Criterion A, an event associated with PTSD must include actual or threatened death, serious injury, or sexual violation resulting from one or more of the following scenarios:

- directly experiencing the traumatic event;
- witnessing the traumatic event in person;
- experiencing the actual or threatened death of a close family member or friend that is either violent or accidental;
- directly experiencing repeated and extreme exposure to aversive details of the event (i.e., the type of exposure frequently encountered by police officers and first responders).

To meet criteria for PTSD, an individual must also report symptoms from each of the four symptom clusters. *Intrusion symptoms* (Criterion B) include repetitive, involuntary, and intrusive memories of the event; traumatic nightmares; dissociative reactions (i.e., flashbacks) along a broad continuum; intense prolonged distress after exposure to reminders of the trauma; and heightened physiological reactivity to reminders of the trauma. *Avoidance symptoms* (Criterion C) include avoidance of trauma-related thoughts and/or feelings, and avoidance of people, places, activities, and so forth that cue distressing thoughts or feelings about the traumatic event. *Negative alterations in cognitions and mood symptoms* (Criterion D) include a persistent and distorted sense of self or the world; blame of self or others; persistent trauma-related emotions, such as anger, guilt, and/or shame; feeling estranged or detached from others; marked lack of interest in pretrauma activities; restricted range of affect; and difficulty or inability remembering important parts of the traumatic event. Finally, *alterations in arousal and reactivity symptoms* (Criterion E) include irritability and aggressiveness, self-destructive or reckless behaviors, sleep difficulties, hypervigilance, marked startle response, concentration difficulties, and sleep disturbance.

An individual must endorse at least one symptom from Criterion B, one symptom from Criterion C, two symptoms from Criterion D, and two symptoms from Criterion E, and the symptoms endorsed in Critera B through E must persist for 1 month or longer (Criterion F). The symptoms must also be accompanied by significant distress or impairment in social, occupational, or other important life domains (Criterion G), and symptoms cannot be better explained by another medical or psychiatric illness (Criterion H).

The DSM-5 includes two additional specifiers or associated features that can be added to a PTSD diagnosis: "with dissociated symptoms" and "with delayed expression." The dissociated-symptoms specifier includes either *depersonalization* (i.e., experience of being an outside observer to one's experience or feeling detached from oneself) or *derealization* (i.e., experience of unreality or distorted reality) in response to trauma-related

cues. The delayed-onset specifier includes an onset of symptoms that can occur immediately after the trauma but that may not meet full criteria for PTSD until at least 6 months after the trauma.

Although the PTSD criteria in the DSM-5 are similar to what they were in the DSM-IV, there are some slight differences. In addition to the inclusion of specifiers for depersonalization and derealization, the DSM-5 provides greater specification regarding which events constitute a traumatic event (i.e., which events constitute a Criterion A event) and excludes the need for an individual to have experienced intense fear, helplessness, or horror at the time of the trauma due to its lack of predictive utility. Furthermore, the avoidance/numbing symptom cluster in the DSM-IV is divided into two clusters in the DSM-5: *avoidance* and *negative alterations in cognitions and mood*. The latter of these clusters retains most of the DSM-IV numbing symptoms but also includes a wider range of unpleasant emotional reactions. Finally, Criterion E, alterations in arousal and reactivity, retains the majority of the DSM-IV arousal symptoms but also includes symptoms regarding aggressive or reckless behavior.

7.1.1 Clinical Features

The clinical expression of PTSD can vary significantly in terms of severity. Although the diagnosis is categorical, there is evidence of a dimensional structure to PTSD (Broman-Fulks et al., 2006; Forbes, Haslam, Williams, & Creamer, 2005). One implication of this dimensional structure is that subthreshold symptoms of PTSD may cause significant distress and functional impairment. One study found that veterans with subthreshold PTSD underutilize mental health care, despite increased psychiatric comorbidity and impairment relative to veterans without PTSD (Grubaugh et al., 2005). Other studies have found an association between subthreshold PTSD and elevated levels of anger and hostility, physical health functioning, and an increased likelihood of hopelessness and suicidal ideation relative to those without PTSD (Jakupcak et al., 2011). Among civilians, subthreshold PTSD has likewise been associated with levels of impairment and suicidality that are equivalent to those of individuals with full PTSD (Zlotnick, Franklin, & Zimmerman, 2002).

The clinical syndrome of PTSD is associated with social maladjustment, poor quality of life, medical comorbidity, and general symptom severity and illness burden. Social and emotional problems include social avoidance, guilt, anger, unemployment, impulsive or violent behavior, relationship difficulties, family discord, and employment instability (Frueh, Turner, Beidel, & Cahill, 2001). Even after accounting for other factors (such as demographics, severity of trauma exposure, physical injury, depression, and substance abuse), PTSD contributes 8% of the variance in physical health and 6% of the variance in mental health aggregate scores (Asnaani, Reddy, & Shea, 2014). When compared with veterans with depression alone, veterans with PTSD and depression were significantly more emotionally distressed, had more frequent mental health specialty visits, had more total outpatient visits, and had higher outpatient mental health care costs (Chan, Cheadle, Reiber, Unutzer, & Chaney, 2009).

Suicide risk is elevated among individuals with PTSD (Panagioti, Gooding, & Tarrier, 2009; Jakupcak et al., 2011), and particular types of trauma, such as childhood abuse, military sexual trauma, and combat, may be more strongly associated with suicidality than other types of trauma (Afifi et al., 2008; Kimerling, Gima, Smith, Street, &

Frayne, 2007). Additionally, increased risk of suicidality is uniquely associated with PTSD (Sareen, Houlahan, Cox, & Asmundson, 2005; Sareen et al., 2007). That is, this association is not solely accounted for by the presence of other psychiatric conditions commonly found with PTSD. Of course, an increased risk of suicidality is present in a number of other psychiatric conditions (e.g., depression, substance abuse, eating disorders) to a comparable or greater degree than that found in PTSD (Nock, Hwang, Sampson, & Kessler, 2010).

The societal and economic costs of PTSD are extremely high. In one study of combat veterans considering all deployed service personnel at that time, the estimated cost was more than $6.2 billion during the first 2 years postdeployment, with a majority of the costs due to lost work productivity (Tanielian & Jaycox, 2008; see also Gates et al., 2012). Health care burden is also high. People with PTSD have a higher number of missed work days. The median public health care cost for PTSD is in the range of $8,000 per year per veteran, and veterans with PTSD have a higher utilization of the overall health care system when compared to other veterans (Asnaani et al., 2014; Tuerk et al., 2013). The appropriate use of evidence-based practices, administered as soon as possible after return from deployment (Gates et al., 2012), may offer the best hope for alleviating PTSD and preventing subsequent occupational, social, and familial impairments.

7.1.2 Diagnostic Considerations

Comorbidity is a concern in the diagnosis and treatment of PTSD. Large nationally representative samples have found that PTSD is significantly correlated with the majority of mood and anxiety disorders, as well as alcohol use disorders (National Comorbidity Survey–Replication [NCS–R]: Kessler, Chiu, Demler, & Walters, 2005; National Comorbidity Survey: Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Data from the NCS–R found that approximately half of those who met the criteria for PTSD also met the criteria for *at least* three additional psychiatric diagnoses (Kessler et al., 1995). Although there is some degree of symptom overlap between PTSD and other psychiatric diagnoses (e.g., sleep and concentration difficulties and diminished interest in activities are common to both depression and PTSD), this overlap does not account for the high rate of comorbidity (Elhai, Grubaugh, Kashdan, & Frueh, 2008). When comorbid with mood disorders, PTSD is more likely to be primary, whereas it is more likely to be secondary when comorbid with anxiety disorders (Kessler et al., 1995). Importantly, PTSD and comorbid diagnoses may change over time within a given individual. A study of trauma survivors found that half of those who reported PTSD only at 3-month follow-up reported depression only at 12-month follow-up; likewise, half of those with depression only at 3-month follow-up reported PTSD only at 12-month follow-up (O'Donnell, Creamer, & Pattison, 2004).

7.1.3 Epidemiology

In the US general population, the 12-month and lifetime prevalences of PTSD are 3.5% and 6.8%, respectively (Kessler, Burglund et al., 2005; Kessler, Chiu et al., 2005). The point prevalence of PTSD among US combat veterans is estimated to be approximately 8%, depending on the characteristics of the sample and the measurement strategies used (Richardson, Frueh, & Acierno, 2010; Smith et al., 2008). There are different conditional probabilities of developing PTSD by trauma type. For example, interpersonal violence,

such as combat exposure or physical/sexual assault, is more often associated with PTSD than other types of trauma. Despite this variability, the symptom expression of PTSD remains fairly consistent regardless of the type of trauma experienced.

7.1.4 Psychological and Biological Assessment

A number of diagnostic measures can be used to assess PTSD. The Clinician-Administered PTSD Scale (CAPS; Weathers, Keane, & Davidson, 2001) is the most common interviewer-based instrument for PTSD and possesses robust psychometric properties. The CAPS includes a detailed assessment of each traumatic event, frequency and severity ratings for each symptom, and overall distress and impairment ratings. Several CAPS scoring algorithms have demonstrated good diagnostic utility (Weathers, Ruscio, & Keane, 1999). Other interview measures include the PTSD Symptom Scale–Interview (Foa & Tolin, 2000) and the Structured Interview for PTSD (Davidson, Smith, & Kudler, 1989). Additionally, the Structured Clinical Interview for DSM-IV (First, Spitzer, Gibbon, & Williams, 1996) and the Anxiety Disorders Interview Schedule (Brown, Di Nardo, & Barlow, 1994) contain modules for diagnosing the disorder.

Self-report questionnaires may also be used to assess PTSD and to measure symptom change over time. Commonly used measures include the PTSD Checklist (Blanchard, Jones-Alexander, Buckely, & Forneris, 1996), the PTSD Symptom Scale–Self Report (Foa, Riggs, Dancu, & Rothbaum, 1993), and the Posttraumatic Diagnostic Scale (Foa, Cashman, Jaycox, & Perry, 1997). An extensive list of measures used to assess PTSD is available from the National Center for PTSD (http://www.ptsd.va.gov). Most of these measures have recently been revised to reflect changes to the diagnostic criteria of PTSD in the DSM-5, and therefore the revised versions have far less research on their psychometric properties.

Aside from interview and self-report measures of PTSD, several physiological variables have been found to distinguish current PTSD from lifetime PTSD and the absence of PTSD. These include an increased resting heart rate, an increased response to non-trauma-related stressors, and increased heart rate, skin conductance, and diastolic blood pressure in response to trauma cues (Pole, 2007). However, the diagnostic utility of these physiological variables is limited in that they tend to be less accurate in predicting PTSD than interview-based and self-report assessments.

7.2 Etiological Pathways and Causal Mechanisms

A number of etiological pathways and causal mechanisms have been implicated in the development of PTSD. These include genetic factors, brain structure and neurochemical abnormalities, pre- and posttrauma life events, cognitive appraisals and attentional biases, and sociodemographic variables such as gender. Some of these variables have implications for treatment, though as a field we are only just beginning to understand them.

7.2.1 Behavioral and Molecular Genetics

Among US Vietnam War veterans, the risk of developing PTSD has been explained by a genetic factor common to alcohol use and PTSD, a genetic factor associated with

PTSD but not with alcohol use, and unique environmental effects (Xian et al., 2000). Yet another twin study of Vietnam-era veterans found that genetic factors that accounted for the relationship between combat exposure and PTSD also accounted for the relationship between combat exposure and alcohol use (McLeod et al., 2001). Genetic factors contributed more to the relationship between combat exposure and PTSD than environmental factors did, whereas genetic and environmental factors contributed equally to the relationship between combat exposure and alcohol use. Interestingly, genetic factors that account for the presence of PTSD may also influence exposure to certain types of traumatic events. Concordance of interpersonal violence and PTSD is higher among monozygotic twins compared with dizygotic twins, whereas other types of trauma (e.g., natural disasters, motor vehicle accidents) are not accounted for by genetic factors (Stein, Jang, Taylor, Vernon, & Livesley, 2002).

In terms of specific genetic markers, researchers have associated the 5-HTTLPR polymorphism with an increased risk of developing PTSD in specific groups of trauma survivors, including hurricane survivors with a high degree of exposure (Kilpatrick et al., 2003) and individuals reporting a traumatic event in childhood as well as adulthood (Xie et al., 2009). A similar interaction has been reported for variants of polymorphisms in the FK506 binding protein 5 (FKBP5) gene, which is involved in regulating the intracellular effects of cortisol. Individuals with these variants who reported severe child abuse were found to be at increased risk for developing PTSD after experiencing a traumatic event in adulthood (Binder et al., 2008; Xie et al., 2009). This gene was underexpressed among survivors of the September 11, 2001, attacks on the World Trade Center who developed PTSD compared with those who did not (Yehuda et al., 2009). There is also evidence for candidate genes in other systems (e.g., the dopamine system), but findings have been limited or inconsistent (Broekman, Olff, & Boer, 2007; Koenen, 2007; Nugent, Amstadter, & Koenen, 2008).

7.2.2 Neuroanatomy and Neurobiology

Several brain structures have been implicated in PTSD, including the amygdala, the medial prefrontal cortex, and the hippocampus. First, PTSD is associated with increased activation in the amygdala in response to trauma-related stimuli (Francati, Vermetten, & Bremner, 2007). This increased activity is thought to represent the neural substrates of exaggerated fear acquisition and expression, and may explain the salience of trauma memories in people with PTSD (Rauch, Shin, & Phelps, 2006). Hyperactivity in the amygdala is not unique to PTSD. Increased activity in response to disorder-related stimuli has also been noted in specific phobia and social anxiety disorder (Etkin & Wagner, 2007; Shin & Liberzon, 2010). Second, PTSD is associated with deficient functioning in the medial prefrontal cortex (Francati et al., 2007; Shin & Liberzon, 2010). This deficiency is thought to underlie inadequate top-down modulation of the amygdala (Rauch et al., 2006). Moreover, the medial prefrontal cortex is thought to regulate processes that are important for habituation and extinction of fear responses, including emotional appraisal (Liberzon & Sripada, 2008). Third, PTSD is associated with abnormalities in the hippocampus. These abnormalities may underlie difficulties contextualizing memories, such as recognizing that certain feared situations are actually safe (Liberzon & Spirada, 2008; Rauch et al., 2006).

One meta-analysis concluded that increased PTSD severity is associated with decreased volume of the hippocampus as well as decreased volume in the amygdala

and the anterior cingulate, a structure in the medial prefrontal cortex (Karl et al., 2006). Decreased hippocampal volume likely represents a risk factor for developing PTSD, as opposed to a neurobiological effect of trauma (McNally, 2003). Consistent with this, hippocampal volume does not change over time following trauma exposure (Bonne et al., 2001). Moreover, a study of veteran twin pairs discordant for combat exposure and PTSD found that PTSD severity among affected twins was negatively correlated with not only their own hippocampal volume but also that of their nonexposed twin (Gilbertson et al., 2002).

The neurochemical underpinnings of PTSD likely involve catecholamines (epinephrine, norepinephrine, and dopamine) and cortisol (a hormone involved in the neuroendocrine response to stress) as well as a variety of other neurotransmitters (Yehuda, 2006). PTSD may also be characterized by disturbance of the hypothalamic–pituitary–adrenal axis, arising primarily from hypersensitivity of glucocorticoid (i.e., cortisol) receptors (Yehuda et al., 2009). This may represent a risk factor, although the research findings are not yet clearly integrated into a cohesive model that can explain or predict.

7.2.3 Learning, Modeling, and Life Events

Although we recognize that traumatic life events contribute to PTSD, what is not clear is whether trauma exposure and PTSD share a dose–response relationship. That is, the data do not provide a clear understanding of how frequency and/or intensity of trauma correspond with symptom severity or prevalence rates of the disorder. Rates of PTSD vary based on the type of traumatic event, with interpersonal violence (assaultive violence and sexual assault) associated with the highest rates (Breslau et al., 1998; Norris, 1992). Rates of PTSD among Vietnam-era veterans roughly correspond to degree of combat exposure (Dohrenwend et al., 2006). However, PTSD severity has not been found to correspond to severity of exposure in other trauma samples, such as motor vehicle accident survivors and political prisoners (Başoğlu et al., 1994; Schnyder, Moergeli, Klaghofer, & Buddeberg, 2001). Thus, the dose–response relationship between trauma exposure and PTSD may be nonlinear. That is, after a certain degree of trauma exposure, symptom exacerbation may reach a plateau (McNally, 2003).

PTSD may also be related to degree of stress and/or trauma exposure prior to the precipitating traumatic event. Exposure to childhood physical or sexual abuse is associated with an increased risk of future trauma exposure as well as the development of PTSD in response to those subsequent traumas (Koenen, Moffitt, Poulton, Martin, & Caspi, 2007). Meta-analyses have also identified other pretrauma risk factors for PTSD, such as level of prior psychological adjustment and the presence of a previous personal or family history of psychiatric illness. Posttrauma risk factors also include a lack of social support and additional life stressors (Brewin, Andrews, & Valentine, 2000; Ozer, Best, Lipsey, & Weiss, 2008).

7.2.4 Cognitive Influences

Cognitive influences of PTSD include maladaptive beliefs about the meaning of the traumatic event after it is experienced (e.g., self-blame, guilt). Consistent with this, cognitive reprocessing therapy emphasizes the importance of identifying and revising maladaptive beliefs about the trauma and promoting a more balanced integration of cognitions

toward the traumatic event (Resick & Schnicke, 1993). Other possible cognitive mechanisms of PTSD include attentional or memory-related biases toward threat-related stimuli or trauma-related material, which may specifically reflect a cognitive vulnerability to developing PTSD (Brewin & Homes, 2003; Weber, 2008). PTSD is also influenced by the perceived seriousness of the threat, which in turn may be influenced by cognitive variables such as autobiographical memory and understanding of the context within which events occurred (Ehlers & Clark, 2000).

7.2.5 Sex and Racial/Ethnic Considerations

Epidemiological surveys suggest that women are more likely to report sexual assault or child molestation, and men are more likely to report physical assault, combat exposure, or being threatened or attacked with a weapon (Cyr et al., 2017). Prevalence studies of PTSD further indicate that women are more likely to develop PTSD relative to men (at a 2:1 ratio) given exposure to a traumatic event (Breslau, 2009). That is, women have a higher conditional risk of developing PTSD relative to men.

Findings regarding the interplay among trauma exposure, PTSD, and race/ethnicity are often inconsistent (Pole, Gone, & Kulkarni, 2008). Overall, however, most studies have found comparable rates of PTSD between African Americans and Caucasians. The few studies that have found significant racial/ethnic differences report higher base rates of PTSD among African Americans relative to Caucasians that largely disappear once severity of trauma exposure is controlled for. The most consistent findings regarding PTSD and race/ethnicity pertain to Hispanics. Relative to non-Hispanic Caucasians, Hispanics often have higher rates of PTSD in both community and clinical samples (Pole et al., 2008). Cultural context may influence some aspects of PTSD, but the disorder generally presents as a coherent group of symptoms across cultures.

7.2.6 Course, Prognosis, and Treatment

The symptoms of PTSD may begin immediately following or some time after a traumatic event, and they may persist for years after the index trauma. The diagnostic specifier "with delayed expression" allows for a diagnosis of PTSD when all of the criteria for the disorder are not met for 6 months or longer after the traumatic event. Although "delayed onset" of PTSD has been noted in some studies, these cases are most likely due to an exacerbation of prior symptoms over time. Supporting this view, a review on the topic found that delayed-onset PTSD in the complete absence of prior symptoms was extremely rare (Andrews, Brewin, Philpott, & Stewart, 2007), a conclusion that other empirical studies have since supported (Frueh, Grubaugh, Yeager & Magruder, 2009).

Consistent with cognitive–behavioral treatments' theoretical underpinnings, clinical practice guidelines generally recommend cognitive–behavioral interventions as the most effective treatment approach for PTSD (Department of Veterans Affairs & Department of Defense [DVA & DoD], 2010; Foa, Keane, & Friedman, 2009; Institute of Medicine & the National Research Council [IOM & NRC], 2007; National Institute for Clinical Excellence [NICE], 2005). Treatments that fall under this umbrella typically include elements of psychoeducation, stress reduction, exposure to trauma-related cues and memories, and cognitive restructuring, with the latter two components being considered the "active ingredients" for PTSD symptom reduction.

Although a number of interventions emphasize exposure and/or cognitive restructuring, the empirical data weigh heavily in favor of two specific treatment approaches for adults and children with PTSD: exposure therapy (Foa, Hembree, & Rothbaum, 2007) and cognitive therapy (Resick & Schnicke, 1993). According to the framework of David and Mongomery (2011), each of these interventions should be classified as evidence-based psychotherapies (Category I). Substantial empirical evidence supports both the intervention package and the mechanisms (i.e., habituation for exposure therapy and cognitive changes in cognitive therapy) hypothesized to mediate or moderate treatment gains. The focus in exposure therapy is on habituation to feared images and situations, whereas the focus of cognitive therapy is on modification of maladaptive trauma-related beliefs (e.g., denial or self-blame). That said, cognitive therapy often includes exposure exercises and exposure therapy often includes elements of cognitive restructuring. Thus, the exact mechanisms of action in these two general forms of therapy are not neatly disentangled in most clinical trials. Moreover, adding cognitive restructuring to exposure does not appear to increase its efficacy (Foa et al., 2005), nor does adding writing exposure exercises to cognitive therapy (Resick et al., 2008), suggesting that the therapies are efficacious in both their combined and component forms.

Treatment reviews suggest the average patient receiving exposure or cognitive therapy fares better than 86% to 90% of patients who are assigned to a control group (i.e., who do not receive what is considered an active treatment) (Bradley, Greene, Russ, Dutra, & Westen, 2005; Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010). Despite the overall efficacy of these interventions, 18% to 35% of individuals with PTSD who complete treatment retain the diagnosis at follow-up, and civilians show dramatically greater improvement than military veterans (Bradley et al., 2005). Disability incentives to remain ill have been posited as one possible reason why veterans evidence less clinical improvement than civilians (Frueh, Grubaugh, Elhai, & Buckley, 2007; Gros, Price, Yuen, & Acierno, 2013; McNally & Frueh, 2013), as have other characteristics unique to veteran populations (e.g., nature of combat trauma). Clearly there is a sociopolitical aspect to the modern experience of military veterans that sets it apart from prior eras (e.g., in the United States, the US Civil War; Frueh & Smith, 2012); the United States, for example, is currently facing a national epidemic of veteran maladjustment that is without historical precedent (McNally & Frueh, 2013). Another concern is that treatment dropout rates hover around 30% across clinical populations (Cloitre, 2009).

Reflecting neurobiological models of the disorder, pharmacological treatments for PTSD act primarily on the neurotransmitters associated with fear and anxiety, which include serotonin, norepinephrine, GABA, and dopamine. Selective serotonin reuptake inhibitors (SSRIs) are generally considered the pharmacological treatment of choice for PTSD (DVA & DoD, 2010; Stein, Ipser, & McAnda, 2009), and this class of drugs includes the only two medications that are currently approved by the US Food and Drug Administration for the treatment of PTSD: sertraline (Zoloft) and paroxetine (Paxil). Although there is some support for the efficacy of psychotropic medications for the treatment of PTSD, not all practice guidelines support their use. After a review of 37 PTSD pharmacotherapy trials, the Institute of Medicine determined that there was insufficient evidence in support of any psychotropic medications for PTSD, including SSRIs (IOM & NRC, 2007). Additionally, psychotropic medications do not typically alleviate all of the symptoms associated with this disorder, and it is generally

recommended that patients take medications in conjunction with a psychotherapy specifically developed to treat PTSD.

7.3 Evidence-Based Practices

7.3.1 Psychosocial Treatments for PTSD

As already noted, clinical practice guidelines generally recommend cognitive–behavioral interventions as the most effective treatment approach for PTSD (DVA & DoD, 2010; Foa et al., 2009; IOM & NRC, 2007; NICE, 2005). Although a number of interventions emphasize exposure and/or cognitive restructuring, the empirical data weigh most heavily in favor of two specific treatments: exposure therapy (an exposure-based intervention; Foa et al., 2007) and cognitive processing therapy (CPT; predominantly a cognitive restructuring intervention but includes elements of exposure; Resick & Schnicke, 1993). The imbalance in the literature emphasizing exposure therapy and CPT over other similar treatments is probably not based on their superiority per se but rather the greater adoption of these specific interventions by individual providers and health care agencies, such as the Veterans Health Administration (VHA) in the United States (Rosen, Ruzek, & Karlin, 2017).

Partly driving the successful dissemination of these treatments is the fact that both exposure therapy and CPT are available as manualized treatments. The second driving force is the availability of training workshops, which often include follow-up consultations, for both CPT and exposure therapy throughout the United States (one reason is that they have been funded by the VHA since 2007). These factors have influenced the adoption and spread of exposure therapy and CPT relative to similar treatments. In 2007, the VHA initiated a broad roll-out of exposure therapy and CPT training to VHA providers nationwide. This ongoing effort includes extensive training workshops for VHA providers, support materials and supervision, and formal processes to certify clinicians and trainers. Reflecting the broad scope of this effort, as of December of 2011, over 4,000 VHA clinicians had been trained in exposure therapy or CPT.

7.3.1.1 Exposure therapy

The consensus statement on PTSD by the International Consensus Group on Depression and Anxiety deemed exposure therapy to be the most efficacious psychotherapy for PTSD (Ballenger et al., 2000; Foa, 2000). The National Institute for Health and Care Excellence (previously the National Institute for Clinical Excellence, 2005) and the Institute of Medicine (IOM & NRC, 2007) reaffirmed this conclusion. Exposure therapy is an effective and accepted intervention for a range of anxiety disorders and is theoretically appropriate for treating acute symptoms of PTSD. However, while there is strong evidence for the effectiveness of exposure therapy for the treatment of PTSD in civilians, there is a lack of corresponding evidence to support the treatment of PTSD in combat veterans (Bradley et al., 2005; Frueh et al., 2007; IOM & NRC, 2007). Moreover, exposure therapy may not address the "negative" symptoms of PTSD (e.g., social withdrawal, interpersonal difficulties, occupational maladjustment, emotional numbing), depression, or feelings of guilt, and it may not improve emotional regulation, such as

anger management (Frueh, Turner, & Beidel, 1995). This is because exposure is specifically focused on anxiety and fear reduction, and hence does not address other clinical features of PTSD. Therefore, it is possible that, for treatment to be fully successful, it must address not only the problem of maladaptive anxiety and fear but also the marked social impairment and emotional dysregulation characteristic of PTSD. According to the framework of David and Mongomery (2011), exposure therapy for PTSD should be classified as an evidence-based psychotherapy (Category I).

7.3.1.1.1 Exposure-based interventions

Exposure-based interventions broadly involve exposing patients to feared stimuli (i.e., objects, places, thoughts) within a safe environment in order to overcome or "extinguish" their fear reaction and can be traced back to Pavlovian fear conditioning models of animal behavior. Within this framework, extinction occurs when a previously learned response to a cue (e.g., fear response to food that is paired with an electric shock) is reduced when the cue (food) is presented in the absence of the previously paired aversive stimulus (shock). Though subtle differences may exist in the application of different types of exposure protocols for PTSD, the general underlying principles of these treatments involve repeatedly exposing an individual to feared traumatic memories and associated feelings in the absence of a negative outcome. This process ultimately results in a decrease in the patient's fear response to trauma-related cues and, as a result, a decrease in the avoidance behaviors that maintain PTSD.

When an individual encounters a feared object, event, or situation, the typical response is to become anxious and then to try to escape from the situation. Escape may temporarily eliminate fear, but it can lead to a pattern of further anxiety and avoidance. The anxious response can be eliminated through the process of exposure to the feared object, experience, or situation. On one level, explaining exposure therapy is easy. If you are afraid of a dog, how do you get over that fear? You have to be around a dog. Exposure therapy requires the individual to be placed in the situation that creates anxiety and to remain there until the emotional distress elicited has been extinguished. So, if a person were afraid of a dog and they were asked to be in a room with a dog, they would remain in contact with the dog until they were no longer anxious. This is called within-session habituation. On a neurological level, learning is occurring and the brain is establishing new neural connections about the situation (Davis, Ressler, Rothbaum, & Richardson, 2006)—"just because I am in contact with a dog does not mean that I will get bitten." Although not targeted directly, unprompted cognitive change during exposure therapy may be a secondary mechanism that facilitates recovery during exposure sessions and there are some data supporting this (Zalta et al., 2014).

When an exposure session is repeated over a number of days, these neural networks are strengthened to the point that they are stronger than the old fear network. On an emotional level, repeating the exposure over time results in diminishment of the initial anxiety response and a shorter time to return to a nonanxious state, until even immediate contact with the object or event no longer produces an anxious response. This is called between-session habituation. Put simply, exposure therapies help patients realize that thoughts and feelings about their traumatic memory are different from the trauma itself, are not inherently dangerous, and can be tolerated.

7.3.1.1.2 Prolonged Exposure

Prolonged Exposure (PE) is a specific brand of exposure therapy developed by Foa and colleagues that has generated the most empirical support of treatments within this area of treatment (Foa et al., 2007). A recent meta-analysis of 13 randomized controlled trials (RCTs) for PE revealed a combined effect size of 1.08, suggesting that the average patient receiving PE fares better than 86% of patients who are assigned to a control group (i.e., do not receive what is considered an active treatment) (Powers et al., 2010). Another meta-analysis found a mean effect size of 1.26 for a range of exposure therapies relative to a control group, suggesting that the average patient receiving an exposure-based intervention fares better than 90% of patients who are assigned to a control group (Bradley et al., 2005).

Similar to exposure therapies more broadly, the theoretical model of PE is based on conditioning principles: trauma-related cues have become conditioned stimuli that are avoided. Exposure treatment requires that an individual be exposed to feared elements of the traumatic memory and habituate to them through extinction principles. As habituation occurs, anxiety decreases. PE is also based on emotional processing theory, which suggests that traumatic events are incompletely and inaccurately encoded in memory as "fear networks." Gradual exposure to corrective information through the confrontation of traumatic stimuli within a safe, therapeutic environment results in a competing and antithetical memory structure that inhibits the conditioned fear response and leads to a more organized, less distressing, and less intrusive traumatic memory.

Like the majority of exposure-based interventions, PE relies on two primary therapeutic tools to accomplish extinction: imaginal exposure and in vivo exposure. During imaginal exposure, patients "revisit" the index event mentally, providing a detailed verbal account of the traumatic memory that includes sensory information, thoughts, feelings, and reactions experienced during the trauma (Foa et al., 2007). While recounting their traumatic memory in vivid detail, patients are instructed to verbalize subjective units of distress (SUDS) every 5 to 7 minutes on a scale from 0 to 100 or 0 to 10. These SUDS ratings are then used to guide the pace and intensity of treatment in order to provide the patient with a therapeutic level of in-session anxiety (i.e., a level that is challenging yet not overwhelming) as well as provide patients with an ongoing awareness of how their anxiety will decrease over time if they tolerate the initial discomfort associated with confronting distressing memories (i.e., habituation/extinction).

Similar to the principles of imaginal exposure, for in vivo activities outside of session, patients confront feared objects, experiences, and situations that remind them of the trauma but that are not dangerous. Patients work off a list previously constructed with the help of the therapist, and patients are encouraged to start with an item that elicits a moderate level of anxiety (i.e., SUDS of 40), to monitor their SUDS, and to remain in the moment until they note a decrease in their distress. Particularly anxious or avoidant patients may need to start with items that elicit less distress (i.e., SUDS of 25) in order to ensure they have some success with the exercise. Patients' awareness of how their distress decreases as they confront anxiety-provoking stimuli and memories provides positive reinforcement for engaging in treatment. Treatment with PE typically ranges from nine to twelve 90-minute sessions. The first two sessions include information-gathering and psychoeducation, with later sessions focusing on imaginal exposure and monitoring of in vivo homework assignments. According to the framework of David and

Mongomery (2011), PE for PTSD should be classified as an evidence-based psychotherapy (Category I).

7.3.1.1.3 Trauma management therapy

Trauma management therapy (TMT) is a multicomponent treatment incorporating exposure therapy and designed to specifically address the multiple problems associated with the clinical syndrome of PTSD (Frueh, Turner, & Beidel, 1996; Turner, Frueh, & Beidel, 2005). TMT was developed for use with combat veterans with PTSD who were reporting reduced anxiety after exposure therapy but were not showing improved social and emotional functioning, in part because it was perceived that they lacked social and emotional regulation skills. In addition, they were reporting a high frequency of physical and verbal rage episodes, and feelings of depression and guilt. Thus, TMT was developed to address all of these features. The treatment contains two general components. The first component is individual exposure therapy, which addresses the unique features of each veteran's trauma. The second component is social and emotional rehabilitation, which uses a skills training format and is conducted in small groups. It includes interpersonal skills training, anger and emotion management training, and more recently behavioral activation (Lejuez, Hopko, Acierno, Daughters, & Pagoto, 2011) for depression.

TMT is not merely a combination of exposure and traditional social skills training. Rather, it includes content areas designed to remedy specific difficulties seen in veterans with chronic PTSD, and it represents a model that may be adapted for civilians with the disorder. The intervention involves identifying specific areas of functioning (e.g., occupational, family, social) in which the patient would like to make changes and examining the values held within those areas. This is followed by identifying and planning daily activities that help one to live in accordance with the values identified as important. Accomplishing activities that are closely linked to core values results in more positive and enjoyable experiences.

Initial research with this multicomponent program demonstrates promise. A RCT showed that TMT resulted in significant improvement in primary PTSD symptoms after exposure therapy, with improved social and emotional functioning only after the other components of TMT were completed (Beidel, Frueh, Uhde, Wong, & Mentrikoski, 2011). An adapted variation of this treatment model has also shown promise with civilians suffering from comorbid PTSD and schizophrenia (Frueh, Grubaugh, Cusack et al., 2009). Additional research with the program is underway, including a current large RCT, and recent data suggest this program can be effectively implemented as an intensive outpatient program that condenses TMT from a 3-month into a 3-week treatment program (Beidel, Frueh, Neer, & Lejuez, 2017). According to the framework of David and Mongomery (2011), TMT for PTSD should be classified as an evidence-based psychotherapy (Category I).

7.3.1.1.4 Virtual reality

Virtual reality (VR) technology shows promise as an augmentation approach to exposure therapy to enhance the extinction response mechanism. Typically, exposure therapy is conducted using either imaginal or in vivo methods. In vivo means that the individual actually comes into physical contact with the fearful object/event. Many specific phobias

(dogs, heights, spiders) can be treated effectively with in vivo exposure therapy. However, there are other situations and events that are not reproducible in real life (such as the events associated with military combat trauma) and, in those cases, many clinicians conduct imaginal therapy. Together, the clinician and patient construct a scene based on the traumatic event. The individual imagines this scene until such time as distress is eliminated. Imaginal therapy has long been the mainstay form of behavior therapy for complicated anxiety disorders such as PTSD when actual recreation of the trauma is not possible.

VR as a means of augmenting exposure therapy has recently been introduced into many treatment settings (Rothbaum, Hodges, Ready, Graap, & Alarcon, 2001). VR is useful when in vivo exposure to traumatic events precipitating the onset of PTSD is not possible (e.g., car bombings cannot be re-created) and patients are unable to or do not want to reimagine the traumatic events. In other words, exposure using a VR environment overcomes a significant hurdle for many individuals with PTSD: an inability to engage in imagery of sufficient detail and affective magnitude to re-create essential aspects of the traumatic event. A basic tenet of any form of exposure therapy is the need to engage as many of the senses as possible. Thus, VR, with its ability to simultaneously present visual, auditory, olfactory, and tactile cues, offers the promise of optimizing exposure therapy. A recent RCT found VR exposure therapy was efficacious for treating deployment-related PTSD among active-duty military personnel, although it was not found to be superior to imaginal exposure therapy (Reger et al., 2016). According to the framework of David and Mongomery (2011), VR-assisted exposure therapy for PTSD should be classified as an evidence-based psychotherapy (Category I).

7.3.1.2 Cognitive therapy

Not everyone responds to or is willing to attempt exposure therapy, and one possible explanation is that some participants have an overfixation on the meaning of the trauma (e.g., betrayal, moral transgressions) and this overfixation interferes with the cognitive demands needed for habituation and the success of exposure therapy. Cognitive interventions for PTSD are based on cognitive therapy principles that can be traced to the work of Aaron Beck and Albert Ellis. These interventions are based on the assumption that thoughts precede mood and that negative thoughts and beliefs are at the core of negative mood states. The goal of most cognitive therapies is to identify the negative and distorted beliefs that patients have and replace them with positive thoughts that are believed to be more balanced and healthy. With PTSD the focus of treatment is on identifying and targeting the negative cognitions that drive the core features of PTSD as well as other maladaptive cognitions common to trauma victims, such as feelings of guilt, shame, and self-blame.

There are very few RCTs of cognitive therapy that do not include elements of exposure therapy. Bradley and colleagues (2005) separated cognitive behavior therapies with and without an exposure component and found comparable results in terms of their efficacy. Cognitive–behavioral therapies without exposure yielded a mean effect size of 1.26 relative to a control group, suggesting that the average patient receiving a non-exposure-based intervention fares better than 90% of patients who are assigned to a control group, and these results are comparable to those reported in Section 7.3.1.1 for exposure therapies (Bradley et al., 2005).

7.3.1.2.1 Cognitive processing therapy

Cognitive processing therapy (CPT) is a manualized cognitive therapy for PTSD with the most empirical support relative to other treatments in this category. It attempts to promote recovery from PTSD by examining the meaning of the traumatic event (Resick & Schnicke, 1993). Although classified here based on its emphasis on cognitive restructuring over exposure, CPT typically includes exposure through a written narrative component and is classified by some as an exposure-based intervention (e.g., IOM & NRC, 2007). Direct comparisons between the cognitive and exposure therapies have failed to find significant differences across conditions (Resick, Nishith, Weaver, Astin, & Feuer, 2002), and thus it may be difficult to completely disentangle the exact mechanisms of action.

CPT is based on information-processing theory, which emphasizes the importance of how individuals encode and recall information. This process largely involves schemata (similar to cognitive therapies more broadly), which are frameworks for organizing and interpreting information. Schemata are generally useful in that they save us time (i.e., they serve as cognitive shortcuts), but they can also lead to errors if relevant information is not processed properly. In the context of a sexual assault, a woman will try to process the assault given her existing schemata regarding sexual assault more broadly and her beliefs about herself. To the degree to which these conflict, she will have difficulty integrating the event into her belief system. This discrepancy is perhaps best highlighted by the "just-world" belief, which is a central tenet of CPT and emerged from the social psychology literature.

The just-world belief is the common human tendency to believe that good things happen to good people and bad things happen to bad people. This tendency is largely driven by the need for humans to feel more in control of random events, thereby decreasing their sense of vulnerability. A sexual assault is generally discrepant with the just-world belief. Thus, when a woman is assaulted, she must struggle with integrating this event with her preexisting views of herself and her understanding of the world around her. In some cases, it may be easier for a woman to blame herself for the assault rather than to believe that she lives in a world where random acts of violence occur. Conversely, a woman may decide that the world is a dangerous place and avoid places or people that remind her of the trauma or social intimacy more broadly. Either view would be considered extreme and maladaptive and subsequently targeted in treatment.

CPT, like any cognitive therapy approach, emphasizes the importance of identifying and revising maladaptive beliefs about the trauma and promoting a more balanced integration of the traumatic event. Treatment typically consists of twelve 50-minute sessions. The initial session includes education about PTSD and a rationale for the treatment. Patients are then instructed to write an impact statement prior to the next session on the meaning of the traumatic experience, including the effects of the experience on their beliefs about themselves, others, and the world more broadly. They are also instructed to consider themes such as trust, safety, and competency when writing their statement.

Subsequent sessions (3 through 7) focus on identifying maladaptive thoughts (stuck points), increasing the patient's awareness of their thoughts and feelings, and aiding their processing of the traumatic event. Processing the traumatic event typically involves writing and reading a detailed account of their worst traumatic memory and Socratic questioning designed to help patients challenge their faulty thinking patterns.

Thus, through a series of questions, the therapist encourages patients to essentially "weigh the evidence" and reevaluate their maladaptive thoughts. This process, if successful, leads patients to a more balanced view of themselves, others, and the world. Sessions 8 through 12 follow a similar strategy of Socratic questioning but focus more specifically on the specific themes of safety, trust, power and control, esteem, and intimacy issues.

The efficacy of CPT is well supported via RCTs conducted with females with PTSD related to sexual assault experiences. The intervention appears to achieve significant symptom reduction by targeting maladaptive cognitions (Gallagher & Resick, 2012; Resick, Williams, Suvak, Monson, & Gradus, 2012). Repeated altering of maladaptive cognitions and the integration of the traumatic event into the patient's self-schema are theorized to reduce the secondary emotions (e.g., depression, guilt, self-blame) and intrusive recollections associated with PTSD. The different underlying mechanisms of change have been suggested to promote increased recovery in their respective domains. In one retrospective data analysis, CPT produced a decrease in PTSD symptoms in civilian sexual assault victims, as mediated by greater changes in hopelessness. Exposure therapy produced a similar PTSD symptom decrease as mediated by habituation, independent of cognitive changes in hopelessness (Gallagher & Resick, 2012).

Implementation of CPT in military populations has occurred more recently and, although the extant literature is not as large as for exposure therapy (Chard, Schumm, Owens, & Cottingham, 2010; Forbes et al., 2012; Morland et al., 2014; Suris, Link-Malcolm, Chard, Ahn, & North, 2013), it does suggest that CPT is efficacious for veterans with PTSD. However, it appears to be significantly more efficacious when delivered in an individual rather than a group format (Resick et al., 2017). According to the framework of David and Mongomery (2011), CPT for PTSD should be classified as an evidence-based psychotherapy (Category I).

7.3.1.2.2 Stress inoculation training

Stress inoculation training (SIT) is another form of cognitive–behavioral therapy for PTSD. As the name suggests, SIT was developed to help "inoculate" patients from their PTSD anxiety and reactivity by providing them with anxiety management skills training prior to exposure. SIT places particular emphasis on anxiety management techniques such as systematic desensitization (progressive muscle relaxation) and breathing retraining (teaching patients to breathe deeply from the diaphragm). However, it also includes cognitive techniques (i.e., positive thinking and reframing techniques, and thought-stopping) and exposure. After patients master the necessary anxiety management skills, they confront feared stimuli from a hierarchical list of fear-inducing items starting with the least feared item and working up the list. Although considered an efficacious treatment approach for PTSD in some treatment guideline reports (DVA & DoD, 2010; Foa et al., 2009), SIT and other anxiety management approaches have not experienced the widespread clinical evaluation or dissemination of the other psychosocial interventions. More often, elements of anxiety management approaches are integrated into other cognitive–behavioral packages. Because of this, there are relatively few recent treatment outcome studies using SIT or similar approaches. Thus, according to David and Montgomery's (2011) framework, SIT should be classified as a theory-driven psychotherapy (Category III).

7.3.1.2.3 Eye movement desensitization and reprocessing

Eye movement desensitization and reprocessing (EMDR), sometimes classified separately, is discussed here as another variant of cognitive–behavioral interventions for PTSD. EMDR was developed in the late 1980s from the personal observations of Francine Shapiro and was subsequently manualized in 1995 (Shapiro, 1995). Shapiro noticed that rapid back-and-forth eye movements had a desensitizing effect on her own unpleasant or distressing thoughts. In further observing that lateral eye movements in isolation did not yield therapeutic effects, she then integrated lateral eye movements (and subsequently other variations of bilateral audio or visual stimulation) into a more comprehensive treatment package for PTSD that includes elements of exposure and cognitive restructuring.

EMDR is a somewhat controversial treatment in that its proponents have historically argued that lateral eye movements facilitate the cognitive processing of traumatic memories via their effect on dampening the distress associated with a traumatic memory, while other researchers simply view EMDR as a variant of exposure therapy. Consistent with the latter view, a meta-analysis and other subsequent studies suggest that the lateral eye movements in EMDR do not offer any additional treatment benefits over exposure alone (Davidson & Parker, 2001), and there is no credible theoretical reason to expect that they would.

Currently, the EMDR Institute offers a more tempered view of lateral eye movements, asserting that the success of EMDR is due to its many treatment components, all of which contribute somewhat equally to treatment efficacy. Regardless of whether EMDR is considered a unique treatment approach or whether lateral eye movements are essential to the efficacy of EMDR, a number of practice guidelines support its use in the treatment of PTSD. The average effect sizes for EMDR relative to control or nonspecific therapies are Cohen's $d = 1.25$ and $d = 0.75$, respectively (Bradley et al., 2005). These numbers suggest that the average patient receiving EMDR fares better than 90% of patients who are assigned to a control group and 79% of those who are assigned to a nonspecific therapy group. These findings suggest that EMDR is similar in efficacy to other cognitive–behavioral interventions, although there is no scientifically valid theory or empirical data to support the saccadic eye movement aspect of this intervention. Thus, according to David and Montgomery's (2011) framework, EMDR should be classified a good-intervention- and bad-theory-driven psychotherapy (Category V).

7.3.1.2.4 Energy therapies

The so-called energy therapies, popular with many master's level and new-age therapists, often identified as "Thought Field Therapy," "the Tapping Method," "Emotional Freedom Techniques," "acupoint stimulation," "Neuro Emotional Technique," and many other variations, have no scientifically valid theory or mechanism of action (e.g., there is absolutely no scientific evidence to support the notion of body "meridians") or rigorously controlled empirical data to support their efficacy. Whatever response their practitioners have noted is impossible to separate from a simple placebo response. Proponents of these interventions often claim something to the effect of: "Absence of efficacy data does not mean these interventions do not work." To this antiscientific perspective, we respond: "Given there are so many psychotherapies for PTSD with strong efficacy data, it cannot be justified to use treatments for which there are no meaningful efficacy data."

According to David and Montgomery's (2011) framework, energy therapies should be classified as pseudoscientifically oriented, or bad-theory- and bad-intervention-driven psychotherapies (Category IX).

7.3.1.3 Summary of psychotherapy for PTSD

The bottom line with exposure therapy and cognitive therapy is that they are both indicated for the treatment of PTSD based on strong theoretical models and controlled empirical support. Multiple RCTs and meta-analyses have provided evidence that exposure and cognitive therapy have relatively equivalent beneficial outcomes (e.g., Foa & Rauch, 2004; Moser, Cahill, & Foa, 2010), including some beneficial change in both cognitive and physiological arousal dimensions of PTSD (Foa & Rauch, 2004; Gallagher & Resick, 2012; Resick et al., 2012; Zalta et al., 2014). That said, despite their overall positive effects, these two widely accepted approaches are not efficacious for all people with PTSD.

Treatment effect sizes for PTSD are moderate, treatment is associated with high dropout rates, and treatment is minimally beneficial or ineffective for a portion of patients (Bradley et al., 2005; Roberts, Kitchiner, Kenardy, & Bisson, 2009; Rothbaum et al., 2014). The dropout rate in civilian PTSD (e.g., motor vehicle accidents, nonsexual physical trauma) is approximately 20% (Bryant et al., 2007; Imel, Laska, Jakupcak, & Simpson, 2013) and higher among military populations (averaging 27% but ranging from 17% to 52%; Gros, Yoder, Tuerk, Lozano, Acierno, 2011; Reger et al., 2011; Tuerk et al., 2011). Among civilians, dropout data show individual characteristics associated with dropout include catastrophic cognitions and higher behavioral avoidance (Bryant et al., 2007).

As noted, although the two theoretically different approaches may impact overall treatment outcome, they may not do so directly and to an extent *sufficient to produce an overall positive treatment outcome*. For military personnel, for example, whose clinical presentation of PTSD has a strong moral injury component, simply treating that individual with exposure therapy and hoping that secondary change in cognitions will occur may be therapeutically naive. Directly addressing the cognitive element may be necessary to produce beneficial therapeutic change. However, efforts to combine interventions that directly target both the physiological/habituation process and the cognitive restructuring process have not resulted in improved treatment efficacy (Foa & Rauch, 2004; Moser et al., 2010).

EMDR likewise has generated sufficient evidence of efficacy by most guidelines, but studies have failed to demonstrate that the saccadic eye movements unique to this intervention are necessary. In this regard, EMDR represents another variant of exposure therapy that, similarly to PE, has been successfully disseminated and marketed. Although some providers advocate for one approach over the other based on personal preference, there are no conclusive data to suggest one is superior to the other. In fact, most meta-analyses suggest that cognitive–behavioral interventions for PTSD are comparable in efficacy and all likely benefit, in part, from the effects of nonspecific factors (Powers et al., 2010; Seidler & Wagner, 2006).

Finally, while there is a good deal of research examining clinical outcomes from a variety of psychosocial interventions for PTSD, there is far less extant research examining the theorized mechanisms of these interventions. This is true generally for interventions across the range of mood and anxiety disorders. For example, while targeting

threat reappraisal via cognitive therapy is related to clinical symptom improvement in anxiety disorders, there are few studies that meet the criteria necessary to demonstrate that it causes symptom improvement and that it is not a proxy for other variables (e.g., nonspecific factors; Smits, Julian, Rosenfield, & Powers, 2012).

7.3.2 Psychopharmacological Therapy for PTSD

Medications for treating PTSD act primarily on the neurotransmitters associated with fear and anxiety, which include serotonin, norepinephrine, GABA, and dopamine. SSRIs are considered the pharmacological treatment of choice for PTSD and in the United States include the only two medications that are approved by the Food and Drug Administration for the treatment of PTSD: sertraline (Zoloft) and paroxetine (Paxil). SSRIs primarily affect the neurotransmitter serotonin, which is implicated in regulating mood, anxiety, and sleep. Consistent with this, studies generally support these drugs' efficacy in decreasing the anxiety and depression that are typically associated with PTSD (Stein et al., 2009). SSRIs take about 6 to 8 weeks to reach maximum effectiveness, and relapse of PTSD symptoms is less likely if the medication is taken for at least 1 year. As with depression and other anxiety disorders, some patients respond sufficiently to an initial dosing recommendation for an SSRI, while others will need changes in dosing or additional care by a mental health provider. Although there is some support for their efficacy, not all practice guidelines support the use of psychotropic medications for the treatment of PTSD. For example, after a review of 37 PTSD pharmacotherapy trials, the Institute of Medicine concluded there was insufficient evidence in support of *any* psychotropic medications for PTSD, including SSRIs (IOM & NRC, 2007).

7.3.2.1 Neuromodulation and neurostimulation for PTSD

Given that dysfunctional neurocircuitry is likely implicated in the etiology and expression of PTSD symptoms (Sheynin & Liberzon, 2016), brain stimulation technologies have been posited as potential novel therapeutic agents to directly target these underlying cortical structures and pathways, especially among more treatment-refractory cases. The evidence in support of these technologies, however, is in its relative infancy. Repetitive transcranial magnetic stimulation (TMS), which uses pulsed magnetic fields delivered via the scalp to noninvasively modulate neuronal activity, has been the most widely studied approach (Clark, Cole, Winter, Williams, & Grammer, 2015). Seminal efforts among adults with PTSD have targeted myriad brain structures with varying device parameters and have yielded promising findings (Hedges' *g* effect sizes ranged from 0.83 to 3.6; Karsen, Watts, & Holtzheimer, 2014), especially when targeting the right dorsolateral prefrontal cortex (Clark et al., 2015). Deep brain stimulation (DBS) is a more invasive brain stimulation technology than TMS and has also been suggested as a possible treatment option. This approach requires the placement of electrodes directly into the brain to stimulate structures of interest. Preclinical, laboratory studies have stimulated multiple brain structures to study the effects on conditioned fear, extinction, and anxiety; targeted structures include the basolateral amygdala, ventral striatum, hippocampus, and prefrontal cortex (Reznikov & Hamani, 2017). Findings from this body of work suggest that stimulation may facilitate fear extinction and improve anxiety-like behavior in rodents (Reznikov & Hamani, 2017). There has been a proposal for an RCT of DBS to treat PTSD in adults (Koek et al., 2014); those data, however, are still pending.

On the other end of the spectrum, noninvasive technologies, such as transcranial direct current stimulation, where very mild electrical current (e.g., 2 mA) is delivered via sponge electrodes placed on the scalp, also show promise in the laboratory in improving psychophysiological characteristics typical of PTSD (Herrmann, Beier, Simons, & Polak, 2016). Though early efforts are promising, no brain stimulation technology has enough evidence to support its use in routine clinical practice, and its role in the treatment of PTSD is likely to evolve considerably over the coming decades, including being used as an adjunctive therapy to potentiate the effect of existing evidence-based treatments.

7.3.2.2 Variability in treatment response

There is considerable variation in treatment response across different patient populations. Typically, women have responded better to PTSD treatments than men, although gender differences are likely better explained by the nature of the traumas experienced by men and women in these studies. That is, male samples have predominantly consisted of men with combat-related PTSD and female samples have predominantly consisted of female sexual assault survivors. It could be that combat-related PTSD is more resistant to treatment than other types of trauma experience, and in the United States the disability policies and incentives of the VHA may discourage treatment success (McNally & Frueh, 2013).

7.3.3 Interventions for PTSD and Comorbid Disorders

Patients with comorbid psychiatric disorders have typically been excluded from PTSD clinical trials for the sake of increased internal validity. As a result, despite the high overlap between PTSD and some psychiatric disorders, treatment approaches for dual disorders remain underdeveloped and there are insufficient data to classify these treatments as frontline interventions for PTSD. There are also limited data to inform the optimal sequence of care with regard to the treatment of PTSD and comorbid conditions (i.e., whether treatment should be integrated and both disorders targeted simultaneously or whether it is better to treat one disorder first and then the other)—though recent clinical wisdom leans toward an integrated treatment approach.

7.3.3.1 Substance abuse

Substance abuse is a common and highly comorbid problem with PTSD. An intervention known as Seeking Safety (SS) is an integrated treatment approach for PTSD and substance use disorders and is the most researched dual-diagnosis PTSD intervention. In guidelines from the International Society for Traumatic Stress Studies on the treatment of PTSD and comorbid conditions, SS was the only integrated treatment for comorbidity to receive a Level A rating (Foa et al., 2009). Level A ratings are based on the Agency for Health Care Policy and Research guidelines and represent the highest possible grade on the scale.

SS (Najavits, Gallop, & Weiss, 2006; Najavits, Weiss, Shaw, & Muenz, 1998) is a present-focused cognitive–behavioral therapy developed to target trauma/PTSD and comorbid substance use disorder that includes cognitive, behavioral, interpersonal, and case management components. Thus, there is no clearly articulated mechanism of action for its effects. It is a flexible treatment designed to be delivered in both individual and group format and has been used in both inpatient and outpatient settings

with a range of substance-abusing patient populations, including prisoners. SS consists of 25 topics that can be covered in any order. Representative topics include "Coping With Triggers" (a behavioral topic where patients are encouraged to fight their triggers), "Recovery Thinking" (a cognitive topic where patients are encouraged to explore the role of honesty in their lives and role-play with other members), "Asking for Help" (an interpersonal topic where patients learn how to ask for and receive help), and "Getting Others to Support Your Recovery" (an interpersonal topic that encourages patients to identify people in their lives who are positive influences on their recovery and to explore ways to solicit support from these individuals). Across topics, the overarching goal of SS is to promote patients' sense of safety in their thoughts, behaviors, and feelings. It has demonstrated modest efficacy with a range of challenging populations, including adolescent girls (Najavits et al., 2006), incarcerated males (Wolff, Huening, Shi, Frueh, Hoover, & McHugo, 2015) and females (Zlotnick, Johnson, & Najavits, 2009), and homeless female veterans (Desai, Harpaz-Rotem, Najavits, & Rosenheck, 2008). However, the mechanisms for its effects are not known (Boden et al., 2012). Thus, according to David and Montgomery's (2011) framework, SS should be classified as an investigational psychotherapy (Category IV).

7.3.3.2 Severe mental illness

Severe mental illness (SMI) represents another category with special treatment needs. Patients with PTSD who also suffer from schizophrenia, bipolar disorder, or severe major depression have long been excluded from clinical trials aimed at PTSD, and yet these patients with SMI are known to have very high levels of PTSD (Grubaugh, Zinzow, Paul, Egede, & Frueh, 2011). Thus, we know very little about how to best treat PTSD in the SMI population, but preliminary data are encouraging that exposure and cognitive therapies are effective for this population (Frueh, Grubaugh, Cusack et al., 2009; Grubaugh et al., 2011, 2016).

Altogether, it is a bit surprising that more progress has not been made in the treatment of more complicated patient populations such as those with comorbid psychiatric conditions. However, there has been a growing emphasis since the mid-2000s on expanding clinical research to more representative patient populations and practice settings—that is, a growing emphasis on external validity versus internal validity and the importance of developing treatments that will disseminate effectively outside of the research lab.

7.3.4 Telemedicine for PTSD

Access to evidence-based care is lacking for many people with PTSD (as well as all other psychiatric disorders) who live in rural areas or areas lacking in specialty mental health care providers. Telemedicine—typically live and synchronous videoconferencing technology—offers a viable means of delivering this care to those living in remote or rural areas, lacking in transportation, or experiencing ambulatory problems such as many elderly people do. This is an atheoretical service delivery mode. The use of telemedicine has grown rapidly since the mid-2000s to provide mental health care services, including direct patient psychotherapy, assessment and evaluation, medication management, case management, supportive counseling, psychoeducation, and professional supervision and training. High levels of satisfaction and acceptance with telemedicine have consistently been found among patients across a variety of clinical

populations and for a broad range of services (Richardson, Frueh, Grubaugh, Egede, & Elhai, 2009). Moreover, the clinical effectiveness of telemedicine is supported by RCTs that demonstrate the clinical efficacy (as well as lack of adverse events) for military veterans with PTSD (Acierno et al., 2016; Frueh, Monnier, Yim et al., 2007; Morland et al., 2010, 2014, 2015). Data also show that patients experience good therapeutic alliance (Greene et al., 2010) and therapists demonstrate strong fidelity to manualized cognitive–behavioral interventions when delivering care via telemedicine (Frueh, Monnier, Grubaugh et al., 2007).

7.4 Conclusions

PTSD is widely accepted as a severe psychiatric disorder, associated with significant distress and functional impairment for individual sufferers and great expense and lost productivity for society. It is also a relatively "new" disorder in that it was not formally recognized in the DSM nosology until 1980. The intervening years have seen a dramatic growth in research on mechanisms, biomarkers, psychotherapies, and other treatments for the disorder, yet we still have much to learn about it. At this point, variations on exposure therapy and cognitive therapy appear to be the primary efficacious psychotherapies for treating PTSD. Both of these approaches have theoretically sound mechanisms of action.

References

Acierno, R., Gros, D. F., Ruggiero, K. J., Hernandez-Tejada, M. B. A., Knapp, R. G., Lejuez, C. W., … Tuerk, P. W. (2016). Behavioral activation and therapeutic exposure for posttraumatic stress disorder: A non-inferiority trial of treatment delivered in person vs home-based telehealth. *Depression and Anxiety*, *33*, 415–423. doi:10.1002/da.22476

Afifi, T. O., Enns, M. W., Cox, B. J., Asmundson, G. J. G., Stein, M. B., & Sareen, J. (2008). Population attributable fractions of psychiatric disorders and suicide ideation and attempts associated with adverse childhood experiences. *American Journal of Public Health*, *98*, 946–952. doi:10.2105/ajph.2007.120253

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.

Andrews, B., Brewin, C. R., Philpott, R., & Stewart, L. (2007). Delayed-onset posttraumatic stress disorder: A systematic review of the evidence. *American Journal of Psychiatry*, *164*, 1319–1326. doi:10.1176/appi.ajp.2007.06091491

Asnaani, A., Reddy, M. K., & Shea, M. T. (2014). The impact of PTSD symptoms on physical and mental health functioning in returning veterans. *Journal of Anxiety Disorders*, *28*, 310–317. doi:10.1016/j.janxdis.2014.01.005

Ballenger, J. C., Davidson, J. R., Lecrubier, Y., Nutt, D. J., Foa, E. B., Kessler, R. C., … Shalev, A. Y. (2000). Consensus statement on posttraumatic stress disorder from the International Consensus Group on Depression and Anxiety. *Journal of Clinical Psychiatry*, *61*(Suppl. 5), 60–66.

Başoğlu, M., Paker, M., Paker, O., Ozmen, E., Marks, I., Incesu, D., … Sarimurat, N. (1994). Psychological effects of torture: A comparison of tortured with nontortured political activists in Turkey. *American Journal of Psychiatry*, *151*, 76–81. doi:10.1176/ajp.151.1.76

Beidel, D. C., Frueh, B. C., Neer, S., & Lejuez, C. (2017). The efficacy of trauma management therapy: A controlled pilot investigation of a three-week intensive outpatient program for combat-related PTSD. *Journal of Anxiety Disorders*, *50*, 23–32. doi:10.1016/j.janxdis.2017.05.001

Beidel, D. C., Frueh, B. C., Uhde, T. W., Wong, N., & Mentrikoski, J. (2011). Multicomponent behavioral treatment for chronic combat-related posttraumatic stress disorder: A randomized controlled trial. *Journal of Anxiety Disorders*, *25*, 224–231. doi:10.1016/j.janxdis.2010.09.006

Binder, E. B., Bradley, R. G., Liu, W., Epstein, M. P., Deveau, T. C., Mercer, K. B., … Ressler, K. J. (2008). Association of FKBP5 polymorphisms and childhood abuse with risk of posttraumatic stress disorder symptoms in adults. *JAMA*, *299*, 1291–1305. doi:10.1001/jama.299.11.1291

Blanchard, E., Jones-Alexander, J., Buckely, T. C., & Forneris, C. A. (1996). Psychometric properties of the PTSD Checklist (PCL). *Behaviour Research and Therapy*, *34*, 669–673. doi:10.1016/0005-7967(96)00033-2

Boden, M. T., Kimerling, R., Jacobs-Lentz, J., Bowman, D., Weaver, C., Carney, D., … Trafton, J. A. (2012). Seeking safety treatment for male veterans with a substance use disorder and post-traumatic stress disorder symptomatology. *Addiction*, *107*, 578–586. doi:10.1111/j.1360-0443.2011.03658.x

Bonne, O., Brandes, D., Gilboa, A., Gomori, J. M., Shenton, M. E., Pitman, R. K., & Shalev, A. Y. (2001). Longitudinal MRI study of hippocampal volume in trauma survivors with PTSD. *American Journal of Psychiatry*, *158*, 1248–1251. doi:10.1176/appi.ajp.158.8.1248

Bradley, R., Greene, J., Russ, E., Dutra, L., & Westen, D. (2005). A multidimensional meta-analysis of psychotherapy for PTSD. *American Journal of Psychiatry*, *162*, 214–227. doi:10.1176/appi.ajp.162.2.214

Breslau, N. (2009). The epidemiology of trauma, PTSD, & other posttrauma disorders. *Trauma, Violence, & Abuse*, *10*, 198–210. doi:10.1177/1524838009334448

Breslau, N., Kessler, R. C., Chilcoat, H. D., Schultz, L. R., Davis, G. C., & Andreski, P. (1998). Trauma and posttraumatic stress disorder in the community: The 1996 Detroit Area Survey of Trauma. *Archives of General Psychiatry*, *55*, 626–632. doi:10.1001/archpsyc.55.7.626

Brewin, C. R., Andrews, B., & Valentine, J. D. (2000). Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *Journal of Consulting and Clinical Psychology*, *68*, 748–766. doi:10.1037//0022-006x.68.5.748

Brewin, C. R., & Holmes, E. A. (2003). Psychological theories of posttraumatic stress disorder. *Clinical Psychology Review*, *23*, 339–376. doi:10.1016/s0272-7358(03)00033-3

Broekman, B. F. P., Olff, M., & Boer, F. (2007). The genetic background to PTSD. *Neuroscience & Biobehavioral Reviews*, *31*, 348–362. doi:10.1016/j.neubiorev.2006.10.001

Broman-Fulks, J. J., Ruggiero, K. J., Green, B. A., Kilpatrick, D. G., Danielson, C. K., Resnick, H. S., & Saunders, B. E. (2006). Taxometric investigation of PTSD: Data from two nationally representative samples. *Behavior Therapy*, *37*, 364–380. doi:10.1016/j.beth.2006.02.006

Brown, T. A., DiNardo, P. A., & Barlow, D. H. (1994). *Anxiety Disorders Interview Schedule for DSM-IV*. Albany, NY: Graywind.

Bryant, R. A., Salmon, K., Sinclair, E., & Davidson, P. A. (2007). A prospective study of appraisals in childhood posttraumatic stress disorder. *Behaviour Research and Therapy*, *45*, 2502–2507. doi:10.1016/j.brat.2007.04.009

Chan, D., Cheadle, A. D., Reiber, G., Unutzer, J., & Chaney, E. F. (2009). Health care utilization and its costs for depressed veterans with and without comorbid PTSD symptoms. *Psychiatric Services*, *60*, 1612–1617. doi:10.1176/ps.2009.60.12.1612

Chard, K. M., Schumm, J. A., Owens, G. P., & Cottingham, S. M. (2010). A comparison of OEF and OIF veterans and Vietnam veterans receiving cognitive processing therapy. *Journal of Traumatic Stress*, *23*, 25–32. doi:10.1002/jts.20500

Clark, C., Cole, J., Winter, C., Williams, K., & Grammer, G. (2015). A review of transcranial magnetic stimulation as a treatment for post-traumatic stress disorder. *Current Psychiatry Reports*, *17*, 83.

Cloitre, M. (2009). Effective psychotherapies for posttraumatic stress disorder: A review and critique. *CNS Spectrums*, *14*(Suppl. 1), 32–43.

Cyr, K., Chamberland, C., Clement, M. E., Wemmers, J. A., Collin-Vezina, D., Lessard, G., … Damant, D. (2017). The impact of lifetime victimization and polyvictimization on adolescents in Quebec: Mental health symptoms and gender differences. *Violence and Victims*, *32*, 3–21. doi:10.1891/0886-6708.vv-d-14-00020

David, D., & Montgomery, G. H. (2011). The scientific status of psychotherapies: A new evaluative framework for evidence-based psychosocial interventions. *Clinical Psychology: Science and Practice*, *18*, 89–99. doi:10.1111/j.1468-2850.2011.01239.x

Davidson, J. R. T., Smith, R. D., & Kudler, H. S. (1989). Validity and reliability of the DSM-III criteria for posttraumatic stress disorder: Experience with a structured interview. *Journal of Nervous and Mental Disease*, *177*, 336–341. doi:10.1097/00005053-198906000-00003

Davidson, P. R., & Parker, K. C. (2001). Eye movement desensitization and reprocessing (EMDR): A meta-analysis. *Journal of Consulting and Clinical Psychology*, *69*, 305–316. doi:10.1037//0022-006x.69.2.305

Davis, M., Ressler, K., Rothbaum, B. O., & Richardson, R. (2006). Effects of D-cycloserine on extinction: Translation from preclinical to clinical work. *Biological Psychiatry*, *60*, 369–375. doi:10.1016/j.biopsych.2006.03.084

Department of Veterans Affairs & Department of Defense. (2010). *VA/DoD clinical practice guideline for management of post-traumatic stress*. Retrieved October 3, 2017, from https://www.healthquality.va.gov/guidelines/MH/ptsd/cpg_PTSD-full-201011612.PDF

Desai, R. A., Harpaz-Rotem, I., Najavits, L. M., & Rosenheck, R. A. (2008). Impact of the Seeking Safety program on clinical outcomes among homeless female veterans with psychiatric disorders. *Psychiatric Services*, *59*, 996–1003. doi:10.1176/appi.ps.59.9.996

Dohrenwend, B. P., Turner, J. B., Turse, N. A., Adams, B. G., Koenen, K. C., & Marshall, R. (2006). The psychological risks of Vietnam for US veterans: A revisit with new data and methods. *Science*, *313*, 979–982. doi:10.1126/science.1128944

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy*, *38*, 319–345. doi:10.1016/s0005-7967(99)00123-0

Elhai, J. D., Grubaugh, A. L., Kashdan, T. B., & Frueh, B. C. (2008). Empirical examination of a proposed refinement to DSM-IV posttraumatic stress disorder symptom criteria using the National Comorbidity Survey Replication data. *Journal of Clinical Psychiatry*, *69*, 597–602. doi:10.4088/jcp.v69n0411

Etkin, A., & Wagner, T. D. (2007). Functional neuroimaging of anxiety: A meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. *American Journal of Psychiatry*, *164*, 1476–1488. doi:10.1176/appi.ajp.2007.07030504

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. (1996). *Structured Clinical Interview for the DSM-IV Axis I disorders*. Washington, DC: American Psychiatric Press.

Foa, E. B. (2000). Psychosocial treatment of posttraumatic stress disorder. *Journal of Clinical Psychiatry*, *61*(Suppl. 5), 49–51.

Foa, E. B., Cashman, L., Jaycox, L., & Perry, K. (1997). The validation of a self-report measure of posttraumatic stress disorder: The Posttraumatic Diagnostic Scale. *Psychological Assessment*, *9*, 445–451. doi:10.1037//1040-3590.9.4.445

Foa, E. B., Hembree, E. A., Cahill, S. P., Rauch, S. A., Riggs, D. S., Feeny, N. C., & Yadin, E. (2005). Randomized trial of prolonged exposure for posttraumatic stress disorder with and without cognitive restructuring: Outcome at academic and community clinics. *Journal of Consulting and Clinical Psychology*, *73*, 953–964. doi:10.1037/0022-006x.73.5.953

Foa, E. B., Hembree, E. A., & Rothbaum, B. O. (2007). *Prolonged exposure therapy for PTSD: Therapist guide*. New York, NY: Oxford University Press.

Foa, E. B., Keane, T. M., & Friedman, M. J. (2009). *Effective treatments for PTSD: Practice guidelines from the International Society for Traumatic Stress Studies*. New York, NY: Guilford Press.

Foa, E. B., & Rauch, S. A. (2004). Cognitive changes during prolonged exposure versus prolonged exposure plus cognitive restructuring in female assault survivors with posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, *72*, 879–884. doi:10.1037/0022-006x.72.5.879

Foa, E. B., Riggs, D. S., Dancu, C. V., & Rothbaum, B. O. (1993). Reliability and validity of a brief instrument for assessing post-traumatic stress disorder. *Journal of Traumatic Stress*, *6*, 459–473. doi:10.1007/bf00974317

Foa, E. B., & Tolin, D. F. (2000). Comparison of the PTSD Symptom Scale–Interview Version and the Clinician-Administered PTSD Scale. *Journal of Traumatic Stress*, *13*, 181–191. doi:10.1023/a:1007781909213

Forbes, D., Haslam, N., Williams, B. J., & Creamer, M. (2005). Testing the latent structure of posttraumatic stress disorder: A taxometric study of combat veterans. *Journal of Traumatic Stress*, *18*, 647–656. doi:10.1002/jts.20073

Forbes, D., Lloyd, D., Nixon, R. D., Elliott, P., Varker, T., Perry, D., … Creamer, M. (2012). A multisite randomized controlled effectiveness trial of cognitive processing therapy for military-related posttraumatic stress disorder. *Journal of Anxiety Disorders*, *26*, 442–452.

Francati, V., Vermetten, E., & Bremner, J. D. (2007). Functional neuroimaging studies in posttraumatic stress disorder: Review of current methods and findings. *Depression and Anxiety*, *24*, 202–218. doi:10.1002/da.20208

Frueh, B. C., Grubaugh, A. L., Acierno, R., Elhai, J. D., Cain, G., & Magruder, K. M. (2007). Age differences in posttraumatic stress disorder, psychiatric disorders, and healthcare service use among veterans in Veterans Affairs primary care clinics. *American Journal of Geriatric Psychiatry*, *15*, 660–672. doi:10.1097/01.jgp.0000260855.42209.31

Frueh, B. C., Grubaugh, A. L., Cusack, K. J., Kimble, M. O., Elhai, J. D., & Knapp, R. G. (2009). Exposure-based cognitive-behavioral treatment of PTSD in adults with schizophrenia or schizoaffective disorder: A pilot study. *Journal of Anxiety Disorders*, *23*, 665–675. doi:10.1016/j.janxdis.2009.02.005

Frueh, B. C., Grubaugh, A. L., Elhai, J. D., & Buckley, T. C. (2007). US Department of Veterans Affairs disability policies for PTSD: Administrative trends and implications for treatment, rehabilitation, and research. *American Journal of Public Health*, *97*, 2143–2145. doi:10.2105/ajph.2007.115436

Frueh, B. C., Grubaugh, A. L., Yeager, D. E., & Magruder, K. M. (2009). Delayed-onset posttraumatic stress disorder among veterans in primary care clinics. *British Journal of Psychiatry*, *194*, 515–520. doi:10.1192/bjp.bp.108.054700

Frueh, B. C., Monnier, J., Grubaugh, A. L., Elhai, J. D., Yim, E., & Knapp, R. (2007). Therapist adherence and competence with manualized cognitive-behavioral therapy for PTSD delivered via videoconferencing technology. *Behavior Modification*, *31*, 856–866. doi:10.1177/0145445507302125

Frueh, B. C., Monnier, J., Yim, E., Grubaugh, A. L., Hamner, M. B., & Knapp, R. G. (2007). A randomized trial of telepsychiatry for post-traumatic stress disorder. *Journal of Telemedicine and Telecare*, *13*, 142–147.

Frueh, B. C., & Smith, J. A. (2012). Suicide, alcoholism, and psychiatric illness among Union forces during the US Civil War. *Journal of Anxiety Disorders*, *26*, 769–775. doi:10.1016/j.janxdis.2012.06.006

Frueh, B. C., Turner, S. M., & Beidel, D. C. (1995). Exposure therapy for combat-related PTSD: A critical review. *Clinical Psychology Review*, *15*, 799–817. doi:10.1016/0272-7358(95)00049-6

Frueh, B. C., Turner, S. M., & Beidel, D. C. (1996). Trauma management therapy: A preliminary evaluation of a multicomponent behavioral treatment for chronic combat-related PTSD. *Behaviour Research and Therapy*, *34*, 533–543. doi:10.1016/0005-7967(96)00020-4

Frueh, B. C., Turner, S. M., Beidel, D. C., & Cahill, S. P. (2001). Assessment of social functioning in combat veterans with PTSD. *Aggression and Violent Behavior*, *6*, 79–90. doi:10.1016/s1359-1789(99)00012-9

Gallagher, M. W., & Resick, P. A. (2012). Mechanisms of change in cognitive processing therapy and prolonged exposure therapy for PTSD: Preliminary evidence for the differential effects of hopelessness and habituation. *Cognitive Therapy and Research*, *36*(6). doi:10.1007/s10608-011-9423-6

Gates, M. A., Holowka, D. W., Vasterline, J. J., Keane, T. M., Marx, B. P., & Rosen, R. C. (2012). Posttraumatic stress disorder in veterans and military personnel: Epidemiology, screening, and case recognition. *Psychological Services*, *9*, 361–382. doi:10.1037/a0027649

Gilbertson, M. W., Shenton, M. E., Ciszewski, A., Kasai, K., Lasko, N. B., Orr, S. P., & Pitman, R. K. (2002). Smaller hippocampal volume predicts pathologic vulnerability to psychological trauma. *Nature Neuroscience*, *5*, 1242–1247. doi:10.1038/nn958

Greene, C. J., Morland, L. A., Macdonald, A., Frueh, B. C., Grubbs, K. M., & Rosen, C. (2010). How does telemental health affect the group therapy process? Results from a randomized noninferiority trial. *Journal of Consulting and Clinical Psychology*, *78*, 746–750. doi:10.1037/a0020158

Gros, D. R., Price, M., Yuen, E. K., & Acierno, R. (2013). Predictors of completion of exposure therapy in OEF/OIF veterans with posttraumatic stress disorder. *Depression and Anxiety*, *30*, 1107–1113. doi:10.1002/da.22207

Gros, D. F., Yoder, M., Tuerk, P. W., Lozano, B. E., & Acierno, R. (2011). Exposure therapy for PTSD delivered to veterans via telehealth: Predictors of treatment completion and outcome and comparison to treatment delivered in person. *Behavior Therapy*, *42*, 276–283. doi:10.1016/j.beth.2010.07.005

Grubaugh, A. L., Clapp, J. D., Frueh, B. C., Tuerk, P. W., Knapp, R. G., & Egede, L. E. (2016). Open trial of exposure therapy for PTSD among patients with severe and persistent mental illness. *Behavior Research and Therapy*, *78*, 1–12. doi:10.1016/j.brat.2015.12.006

Grubaugh, A. L., Magruder, K. M., Waldrop, A. E., Elhai, J. D., Knapp, R. G., & Frueh, B. C. (2005). Subthreshold PTSD in primary care: Prevalence, psychiatric disorders, healthcare use, and functional status. *Journal of Nervous and Mental Disease*, *193*, 658–664. doi:10.1097/01.nmd.0000180740.02644.ab

Grubaugh, A. L., Zinzow, H. M., Paul, L., Egede, L. E., & Frueh, B. C. (2011). Trauma exposure and posttraumatic stress disorder in adults with severe mental illness: A critical review. *Clinical Psychology Review*, *31*, 883–899. doi:10.1016/j.cpr.2011.04.003

Herrmann, M. J., Beier, J. S., Simons, B., & Polak, T. (2016). Transcranial direct current stimulation (tDCS) of the right inferior frontal gyrus attenuates skin conductance responses to unpredictable threat conditions. *Frontiers Human Neuroscience*, *10*, 352.

Imel, Z. E., Laska, K., Jakupcak, M., & Simpson, T. L. (2013). Meta-analysis of dropout in treatments for posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, *81*, 394–404. doi:10.1037/a0031474

Institute of Medicine & the National Research Council. (2007). *Treatment of PTSD: An assessment of the evidence*. Washington, DC: National Academies Press.

Jakupcak, M., Hoerster, K. D., Varra, A., Vannoy, S., Felker, B., & Hunt, S. (2011). Hopelessness and suicidal ideation in Iraq and Afghanistan war veterans reporting subthreshold and threshold posttraumatic stress disorder. *Journal of Nervous and Mental Disease*, *199*, 272–275. doi:10.1097/nmd.0b013e3182124604

Karl, A., Schaefer, M., Malta, L. S., Dörfel, D., Rohleder, N., & Werner, A. (2006). A meta-analysis of structural brain abnormalities in PTSD. *Neuroscience & Biobehavioral Reviews*, *30*, 1004–1031. doi:10.1016/j.neubiorev.2006.03.004

Karsen, E. F., Watts, B. V., & Holtzheimer, P. E. (2014). Review of the effectiveness of transcranial magnetic stimulation for post-traumatic stress disorder. *Brain Stimulation*, *7*, 151–157. doi:10.1016/j.brs.2013.10.006

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Survey Replication. *Archives of General Psychiatry*, *62*, 593–602. doi:10.1001/archpsyc.62.6.593

Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*, 617–627. doi:10.1001/archpsyc.62.6.617

Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. B. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry*, *52*, 1048–1060. doi:10.1001/archpsyc.1995.03950240066012

Kilpatrick, D. G., Ruggiero, K. J., Acierno, R., Saunders, B. E., Resnick, H. S., & Best, C. L. (2003). Violence and risk of PTSD, major depression, substance abuse/dependence, and comorbidity: Results from the National Survey of Adolescents. *Journal of Consulting and Clinical Psychology*, *71*, 692–700. doi:10.1037/0022-006x.71.4.692

Kimerling, R., Gima, K., Smith, M. W., Street, A., & Frayne, S. (2007). The Veterans Health Administration and military sexual trauma. *American Journal of Public Health*, *97*, 2160–2166. doi:10.2105/ajph.2006.092999

Koek, R. J., Langevin, J. P., Krahl, S. E., Kosoyan, H. J., Schwartz, H. N., Chen, J. W., … Sultzer, D. (2014). Deep brain stimulation of the basolateral amygdala for treatment-refractory combat post-traumatic stress disorder (PTSD): Study protocol for a pilot randomized controlled trial with blinded, staggered onset of stimulation. *Trials*, *15*, 356. doi:10.1186/1745-6215-15-356

Koenen, K. C. (2007). Genetics of posttraumatic stress disorder: Review and recommendations for future studies. *Journal of Traumatic Stress*, *20*, 737–750. doi:10.1002/jts.20205

Koenen, K. C., Moffitt, T. E., Poulton, R., Martin, J., & Caspi, A. (2007). Early childhood factors associated with the development of post-traumatic stress disorder: Results from a longitudinal birth cohort. *Psychological Medicine*, *37*, 181–192. doi:10.1017/s0033291706009019

Lejuez, C. W., Hopko, D. R., Acierno, R., Daughters, S. B., & Pagoto, S. L. (2011). Ten year revision of the brief behavioral activation treatment for depression: Revised treatment manual. *Behavior Modification*, *35*, 111–161. doi:10.1177/0145445510390929

Liberzon, I., & Sripada, C. S. (2008). The functional neuroanatomy of PTSD: A critical review. *Progress in Brain Research*, *167*, 151–169. doi:10.1016/s0079-6123(07)67011-3

McLeod, D. S., Koenen, K. C., Meyer, J. M., Lyons, M. J., Eisen, S., True, W., & Goldberg, J. (2001). Genetic and environmental influences on the relationship among combat exposure, posttraumatic stress disorder symptoms, and alcohol use. *Journal of Traumatic Stress*, *14*, 259–275. doi:10.1023/a:1011157800050

McNally, R. J. (2003). Progress and controversy in the study of posttraumatic stress disorder. *Annual Review of Psychology*, *54*, 229–252. doi:10.1146/annurev.psych.54.101601.145112

McNally, R. J., & Frueh, B. C. (2013). Why are Iraq and Afghanistan war veterans seeking PTSD disability compensation at unprecedented rates? *Journal of Anxiety Disorder*, *27*, 520–526. doi:10.1016/j.janxdis.2013.07.002

Morland, L. A., Greene, C. J., Rosen, C., Foy, D., Reilly, P., Shore, J., He, Q., & Frueh, B. C. (2010). Telemedicine anger management with a rural PTSD population: A randomized noninferiority trial. *Journal of Clinical Psychiatry*, *71*, 855–863. doi:10.4088/jcp.09m05604blu

Morland, L. A., Mackintosh, M. A., Greene, C. J., Rosen, C., Chard, K. M., Resick, P., & Frueh, B. C. (2014). Cognitive processing therapy for posttraumatic stress disorder delivered to rural veterans via telemental health: A randomized noninferiority clinical trial. *Journal of Clinical Psychiatry*, *75*, 470–476. doi:10.4088/jcp.13m08842

Morland, L. A., Mackintosh, M. A., Rosen, C. S., Willis, E., Resick, P., Chard, K., & Frueh, B. C. (2015). Telemedicine vs. in-person delivery of cognitive processing therapy for women with posttraumatic stress disorder: A randomized non-inferiority trial. *Depression and Anxiety*, *32*, 811–820. doi:10.1002/da.22397

Moser, J. S., Cahill, S. P., & Foa, E. B. (2010). Evidence for poorer outcome in patients with severe negative trauma-related cognitions receiving prolonged exposure plus cognitive restructuring: Implications for treatment matching in posttraumatic stress disorder. *Journal of Nervous and Mental Disease*, *198*, 72–75. doi:10.1097/nmd.0b013e3181c81fac

Najavits, L. M., Gallop, R. J., & Weiss, R. D. (2006). Seeking Safety therapy for adolescent girls with PTSD and substance use disorder: A randomized controlled trial. *Journal of Behavioral Health Services & Research*, *33*, 453–463. doi:10.1007/s11414-006-9034-2

Najavits, L. M., Weiss, R. D., Shaw, S. R., & Muenz, L. R. (1998). "Seeking Safety": Outcome of a new cognitive-behavioral psychotherapy for women with posttraumatic stress disorder and substance dependence. *Journal of Traumatic Stress Disorder*, *11*, 437–456. doi:10.1023/a:1024496427434

National Institute for Clinical Excellence. (2005). *Clinical guideline 26: PTSD—The management of PTSD in adults & children in primary & secondary care*. London: National Collaborating Centre for Mental Health.

Nock, M. K., Hwang, I., Sampson, N. A., & Kessler, R. C. (2010). Mental disorders, comorbidity and suicidal behavior: Results from the National Comorbidity Survey Replication. *Molecular Psychiatry*, *15*, 868–876. doi:10.1038/mp.2009.29

Norris, F. H. (1992). Epidemiology of trauma: Frequency and impact of different potentially traumatic events on different demographic groups. *Journal of Consulting and Clinical Psychology*, *60*, 409–418. doi:10.1037//0022-006x.60.3.409

Nugent, N. R., Amstadter, A. B., & Koenen, K. C. (2008). Genetics of posttraumatic stress disorder: Informing clinical conceptualizations and promoting future research. *American Journal of Medical Genetics Part C: Seminars in Medical Genetics*, *148C*, 127–132. doi:10.1002/ajmg.c.30169

O'Donnell, M. L., Creamer, M., & Pattison, P. (2004). Posttraumatic stress disorder and depression following trauma: Understanding comorbidity. *American Journal of Psychiatry*, *161*, 1390–1396. doi:10.1176/appi.ajp.161.8.1390

Ozer, E. J., Best, S. R., Lipsey, T. L., & Weiss, D. S. (2008). Predictors of posttraumatic stress disorder and symptoms in adults: A meta-analysis. *Psychological Bulletin*, *129*(1), 52–73. doi:10.1037//0033-2909.129.1.52

Panagioti, M., Gooding, P., & Tarrier, N. (2009). Posttraumatic stress disorder and suicidal behavior: A narrative review. *Clinical Psychology Review*, *29*, 471–482. doi:10.1016/j.cpr.2009.05.001

Pole, N. (2007). The psychophysiology of posttraumatic stress disorder: A meta-analysis. *Psychological Bulletin*, *133*, 725–746. doi:10.1037/0033-2909.133.5.725

Pole, N., Gone, J. P., & Kulkarni, M. (2008). Posttraumatic stress disorder among ethnoracial minorities in the United States. *Clinical Psychology: Science and Practice*, *51*, 35–61. doi:10.1111/j.1468-2850.2008.00109.x

Powers, M. B., Halpern, J. M., Ferenschak, M. P., Gillihan, S. J., & Foa, E. B. (2010). A meta-analytic review of prolonged exposure for posttraumatic stress disorder. *Clinical Psychology Review*, *30*, 635–641. doi:10.1016/j.cpr.2010.04.007

Rauch, S. L., Shin, L. M., & Phelps, E. A. (2006). Neurocircuitry models of posttraumatic stress disorder and extinction: Human neuroimaging research—past, present, and future. *Biological Psychiatry*, *60*, 376–382. doi:10.1016/j.biopsych.2006.06.004

Reger, G. M., Holloway, K. M., Candy, C., Rothbaum, B. O., Difede, J., Rizzo, A. A., & Gahm, G. A. (2011). Effectiveness of virtual reality exposure therapy for active duty soldiers in a military mental health clinic. *Journal of Traumatic Stress*, *24*, 93–96. doi:10.1002/jts.20574

Reger, G. M., Koenen-Woods, P., Zetocha, K., Smolenski, D. J., Holloway, K. M., Rothbaum, B. O., … Gahm, G. A. (2016). Randomized controlled trial of prolonged exposure using imaginal exposure vs. virtual reality exposure in active duty soldiers with deployment-related posttraumatic stress disorder (PTSD). *Journal of Consulting and Clinical Psychology*, *84*, 946–959. doi:10.1037/ccp0000134

Resick, P. A., Galovski, T. E., O'Brien Uhlmansiek, M., Scher, C. D., Clum, G. A., & Young-Xu, Y. (2008). A randomized clinical trial to dismantle components of cognitive processing therapy for posttraumatic stress disorder in female victims of interpersonal violence. *Journal of Consulting and Clinical Psychology*, *76*, 243–258. doi:10.1037/0022-006x.76.2.243

Resick, P. A., Nishith, P., Weaver, T. L., Astin, M. C., & Feuer, C. A. (2002). A comparison of cognitive-processing therapy with prolonged exposure and a waiting condition for the treatment of chronic posttraumatic stress disorder in female rape victims. *Journal of Consulting and Clinical Psychology*, *70*, 867–879. doi:10.1037//0022-006x.70.4.867

Resick, P. A., & Schnicke, M. K. (1993). *Cognitive processing therapy for rape victims: A treatment manual*. Newbury Park, CA: Sage.

Resick, P. A., Wachen, J. S., Dondanville, K. A., Pruiksma, K. E., Yarvis, J. S., Peterson, A. L., … the Strong Star Consortium. (2017). Effect of group vs individual cognitive processing therapy in active-duty military seeking treatment for posttraumatic stress disorder: A randomized clinical trial. *JAMA Psychiatry*, *74*, 28–36. doi:10.1001/jamapsychiatry.2016.2729

Resick, P. A., Williams, L. F., Suvak, M. K., Monson, C. M., & Gradus, J. L. (2012). Long-term outcomes of cognitive-behavioral treatments for posttraumatic stress disorder among female rape survivors. *Journal of Consulting and Clinical Psychology*, *80*, 201–210. doi:10.1037/a0026602

Reznikov, R., & Hamani, C. (2017). Posttraumatic stress disorder: Perspectives for the use of deep brain stimulation. *Neuromodulation*, *20*, 7–14. doi:10.1111/ner.12551

Richardson, L. K., Frueh, B. C., & Acierno, R. (2010). Prevalence estimates of combat-related posttraumatic stress disorder: A critical review. *Australian & New Zealand Journal of Psychiatry*, *44*, 4–19.

Richardson, L. K., Frueh, B. C., Grubaugh, A. L., Egede, L., & Elhai, J. D. (2009). Current directions in videoconferencing tele-mental health research. *Clinical Psychology: Science and Practice*, *16*, 323–338. doi:10.1111/j.1468-2850.2009.01170.x

Roberts, N. P., Kitchiner, N. J., Kenardy, J., & Bisson, J. A. (2009). Systematic review and meta-analysis of multiple-session early interventions following traumatic events. *American Journal of Psychiatry*, *166*, 293–301. doi:10.1176/appi.ajp.2008.08040590

Rosen, R. C., Ruzek, J. I., & Karlin, B. E. (2017). Evidence-based training in the era of evidence-based practice: Challenges and opportunities for training of PTSD providers. *Behavior Research and Therapy*, *88*, 37–48. doi:10.1016/j.brat.2016.07.009

Rothbaum, B. O., Hodges, L. F., Ready, D., Graap, K., & Alarcon, R. D. (2001). Virtual reality exposure therapy for Vietnam veterans with posttraumatic stress disorder. *Journal of Clinical Psychiatry*, *62*, 617–622. doi:10.4088/jcp.v62n0808

Rothbaum, B. O., Price, M., Jovanovic, T., Norrholm, S. D., Gerardi, M. M., Dunlop, B., … Ressler, K. J. (2014). A randomized, double-blind evaluation of d-cycloserine or alprazolam combined with virtual reality exposure therapy for posttraumatic stress disorder in Iraq and Afghanistan war veterans. *American Journal of Psychiatry*, *171*, 640–648. doi:10.1176/appi.ajp.2014.13121625

Sareen, J., Cox, B. J., Stein, M. B., Afifi, T. O., Fleet, C., & Asmundson, G. J. G. (2007). Physical and mental comorbidity, disability, and suicidal behavior associated with posttraumatic stress disorder in a large community sample. *Psychosomatic Medicine*, *69*, 242–248. doi:10.1097/psy.0b013e31803146d8

Sareen, J., Houlahan, T., Cox, B., & Asmundson, G. J. G. (2005). Anxiety disorders associated with suicidal ideation and suicide attempts in the National Comorbidity Survey. *Journal of Nervous and Mental Disease*, *193*, 450–454. doi:10.1097/01.nmd.0000168263.89652.6b

Schnyder, U., Moergeli, H., Klaghofer, R., & Buddeberg, C. (2001). Incidence and prediction of posttraumatic stress disorder symptoms in severely injured accident victims. *American Journal of Psychiatry*, *158*, 594–599. doi:10.1176/appi.ajp.158.4.594

Seidler, G. H., & Wagner, F. E. (2006). Comparing the efficacy of EMDR and trauma-focused cognitive-behavioral therapy in the treatment of PTSD: A meta-analytic study. *Psychological Medicine*, *36*, 1515–1522. doi:10.1017/s0033291706007963

Shapiro, F. (1995). *Eye movement desensitization and reprocessing: Basic principles, protocols, and procedures*. New York, NY: Guilford Press.

Sheynin, J., & Liberzon, I. (2016). Circuit dysregulation and circuit-based treatments in posttraumatic stress disorder. *Neuroscience Letters*, *649*, 133–138. doi:10.1016/j.neulet.2016.11.014

Shin, L. M., & Liberzon, I. (2010). The neurocircuitry of fear, stress, and anxiety disorders. *Neuropsychopharmacology Reviews*, *35*, 169–191. doi:10.1038/npp.2009.83

Smith, T. C., Ryan, M. A. K., Wingard, D. L., Slymen, D. J., Sallis, J. F., & Kritz-Silverstein, D. (2008). New onset and persistent symptoms of post-traumatic stress disorder self-reported after deployment and combat exposures: Prospective population based US military cohort study. *BMJ*, *336*, 366–371. doi:10.1136/bmj.39430.638241.ae

Smits, J. A. J., Julian, K., Rosenfield, D., & Powers, M. B. (2012). Threat reappraisal as a mediator of symptom change in cognitive-behavioral treatment of anxiety disorders: A systematic review. *Journal of Consulting and Clinical Psychology*, *80*, 624–635. doi:10.1037/a0028957

Stein, D. J., Ipser, J., & McAnda, N. (2009). Pharmacotherapy of posttraumatic stress disorder: A review of meta-analyses and treatment guidelines. *CNS Spectrums*, *1*(Suppl. 1), 25–31.

Stein, M. B., Jang, K. L., Taylor, S., Vernon, P. A., & Livesley, W. J. (2002). Genetic and environmental influences on trauma exposure and posttraumatic stress disorder symptoms: A twin study. *American Journal of Psychiatry*, *159*, 1675–1681. doi:10.1176/appi.ajp.159.10.1675

Suris, A., Link-Malcolm, J., Chard, K., Ahn, C., & North, C. (2013). A randomized clinical trial of cognitive processing therapy for veterans with PTSD related to military sexual trauma. *Journal of Traumatic Stress*, *26*, 28–37. doi:10.1002/jts.21765

Tanielian, T., & Jaycox, L. H. (2008). *Invisible wounds of war: Psychological and cognitive injuries, their consequences, and services to assist recovery*. Santa Monica, CA: Rand Center for Military Health Policy Research.

Tuerk, P. W., Wangelin, B., Rauch, S. A., Dismuke, C. E., Yoder, M., Myrick, H., … Acierno, R. (2013). Health service utilization before and after evidence-based treatment for PTSD. *Psychological Services*, *10*, 401–409. doi:10.1037/a0030549

Tuerk, P. W., Yoder, M., Grubaugh, A., Myrick, H., Hamner, M., & Acierno, R. (2011). Prolonged exposure therapy for combat-related posttraumatic stress disorder: An examination of treatment effectiveness for veterans of the wars in Afghanistan and Iraq. *Journal of Anxiety Disorders*, *25*, 397–403. doi:10.1016/j.janxdis.2010.11.002

Turner, S. M., Frueh, B. C., & Beidel, D. C. (2005). Multicomponent behavioral treatment for chronic combat-related posttraumatic stress disorder: Trauma Management Therapy. *Behavior Modification*, *29*, 39–69. doi:10.1177/0145445504270872

Weathers, F., Ruscio, A., & Keane, T. M. (1999). Psychometric properties of the nine scoring rules for the Clinician Administered PTSD Scale. *Psychological Assessment*, *11*, 124–133. doi:10.1037//1040-3590.11.2.124

Weathers, F. W., Keane, T. M., & Davidson, J. R. T. (2001). Clinician-Administered PTSD Scale: A review of the first ten years of research. *Depression and Anxiety*, *13*, 132–156. doi:10.1002/da.1029

Weber, D. L. (2008). Information processing bias in post-traumatic stress disorder. *Open Neuroimaging Journal*, *2*, 29–51. doi:10.2174/1874440000802010029

Wolff, N., Huening, J., Shi, J., Frueh, B. C., Hoover, D. R., & McHugo, G. (2015). Implementation and effectiveness of integrated trauma and addiction treatment for incarcerated men. *Journal of Anxiety Disorders*, *30C*, 66–80. doi:10.1016/j.janxdis.2014.10.009

Xian, H., Chantarujikapong, S., Scherrer, J. F., Eisen, S. A., Lyons, M. J., Goldberg, J., … True, W. R. (2000). Genetic and environmental influences on posttraumatic stress disorder, alcohol and drug dependence in twin pairs. *Drug and Alcohol Dependence*, *61*, 95–102. doi:10.1016/s0376-8716(00)00127-7

Xie, P., Kranzler, H. R., Poling, J., Stein, M. B., Anton, R. F., Brady, K., … Gelernter, J. (2009). Interactive effect of stressful life events and the serotonin transporter 5-HTTLPR genotype on posttraumatic stress disorder diagnosis in two independent populations. *Archives of General Psychiatry*, *66*, 1201–1209. doi:10.1001/archgenpsychiatry.2009.153

Yehuda, R. (2006). Advances in understanding neuroendocrine alterations in PTSD and their therapeutic implications. *Annals of the New York Academy of Sciences*, *1071*, 137–166. doi:10.1196/annals.1364.012

Yehuda, R., Cai, G., Golier, J. A., Sarapas, C., Galea, S., Ising, M., … Buxbaum, J. D. (2009). Gene expression patterns associated with posttraumatic stress disorder following exposure to the World Trade Center attacks. *Biological Psychiatry*, *66*, 708–711. doi:10.1016/j.biopsych.2009.02.034

Zalta, A. K., Gillihan, S. J., Fisher, A. J., Mintz, J., McLean, C. P., Yehuda, R., & Foa, E. B. (2014). Change in negative cognitions associated with PTSD predicts symptom reduction in prolonged exposure. *Journal of Consulting and Clinical Psychology*, *82*, 171–175. doi:10.1037/a0034735

Zlotnick, C., Franklin, C. L., & Zimmerman, M. (2002). Does "subthreshold" posttraumatic stress disorder have any clinical relevance? *Comprehensive Psychiatry*, *43*, 413–419. doi:10.1053/comp.2002.35900

Zlotnick, C., Johnson, J., & Najavits, L. M. (2009). Randomized controlled pilot study of cognitive-behavioral therapy in a sample of incarcerated women with substance use disorder and PTSD. *Behavioral Therapy*, *40*, 325–336. doi:10.1016/j.beth.2008.09.004

8

Evidence-Based Psychological Interventions for Eating Disorders

Diana M. Cândea, Daniel David, and Aurora Szentágotai-Tătar
Department of Clinical Psychology and Psychotherapy, Babeş-Bolyai University, Cluj-Napoca, Romania

8.1 Description of the Disorders

According to the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013), eating disorders are characterized by severe disturbance in eating behaviors, such as (1) persistent restriction of food intake leading to significantly low body weight relative to the person's developmental stage along with an intense fear of gaining weight or becoming fat (anorexic symptoms); (2) recurrent episodes of binge eating and a sense of lack of control over eating during the episode (binge eating symptoms); and (3) ill-suited methods to prevent weight gain (e.g., bulimic symptoms such as purging, excessive exercise, or laxative or diuretic misuse; American Psychiatric Association, 2013).

Despite the increasing number of clinical investigations aimed at developing more efficacious treatments or improving existing ones, the incidence and prevalence of eating disorders have not diminished (Currin, Schmidt, Treasure, & Jick, 2005). Epidemiological data from Western European countries indicate an estimated lifetime prevalence for anorexia nervosa, bulimia nervosa, and binge eating disorder of 0.48%, 0.51%, and 1.12%, respectively, with higher rates among females for all eating disorders (3–8 times higher; Preti, Rocchi, Sisti, Camboni, & Miotto, 2011). The incidence of these disorders is estimated at 8 cases per 100,000 persons per year for anorexia and 12 cases per 100,000 persons for bulimia (Hoek & Van Hoeken, 2003).

Eating disorders are disabling conditions, associated with high levels of comorbid psychopathology (Blinder, Cumella, & Sanathara, 2006; Braun, Sunday, & Halmi, 1994), medical complications (Dickstein, Franco, Rome, & Auron, 2014), increased role impairment (Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011), and high suicide rates, with anorexia evidencing the highest mortality rate of all mental disorders (Arcelus, Mitchell, Wales, & Nielsen, 2011). Eating disorders are also associated with a substantial economic burden in terms of both lost productivity costs and health care costs; in the case of anorexia, some studies indicate that the mean annual treatment cost is significantly higher than the mean cost for schizophrenia (Striegel-Moore, Leslie, Petrill, Garvin, & Rosenheck, 2000).

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

The National Institute for Health and Care Excellence (previously the National Institute for Clinical Excellence [NICE], 2004) and the American Psychological Association (APA; APA Presidential Task Force on Evidence-Based Practice, 2006) list several psychological interventions as efficacious treatments for eating disorders. Nevertheless, in the case of anorexia, 20% of patients develop a chronic condition and 5% die from the disorder (Steinhausen, 2002); in the case of bulimia, between 9% and 20% of patients still meet diagnostic criteria after completing treatment (Collings & King, 1994; Keel & Mitchell, 1997).

8.2 David and Montgomery's (2011) Evaluative Framework

In this chapter, we review psychological interventions for eating disorders based on David and Montgomery's (2011) framework, which evaluates interventions in terms of two components: (1) the efficacy/effectiveness of the clinical protocol and (2) the empirical support for the theory underlying the clinical protocol. The combination of the two criteria yields nine categories, reflecting the state of psychotherapy for a given condition. We consider interventions separately for each eating disorder: anorexia nervosa, binge eating disorder, and bulimia nervosa (see Table 8.1, Table 8.2, and Table 8.3). For each intervention, we derived evidence regarding treatment package efficacy and the underlying theory from clinical guidelines (e.g., NICE and APA guidelines), meta-analyses, systematic reviews of clinical trials, and individual clinical trials and experimental or correlational studies, identified through a search on the PubMed and Google Scholar databases. We express the efficacy of the clinical protocol in terms of *absolute efficacy* (comparisons with waitlist or no-treatment control group), *relative efficacy* (comparisons with other standard treatments), and *specific efficacy* (therapeutic package and the underlying theory for specific mechanisms are empirically supported). To prove *specific efficacy*, the therapeutic package must be (1) significantly better than placebo and/or (2) equivalent to active/standard psychological therapies or significantly better than other active/standard psychotherapies.

8.3 Classification of Psychological Interventions for Anorexia Nervosa

8.3.1 Category I: Evidence-Based Psychotherapies

Category I includes psychological interventions for which both the efficacy/effectiveness of the clinical protocol and the theory of change underlying the clinical protocol are well supported. None of the identified and evaluated therapies can be included in this category.

8.3.2 Category II: Intervention-Driven Psychotherapies

Psychological treatments included in this category are those for which there is strong supporting evidence for the efficacy/effectiveness of the clinical protocol but findings

Table 8.1 A graphical representation of the classification of psychological interventions for anorexia nervosa using David and Montgomery's (2011) evaluative framework.

	Theory		
Therapeutic Package	**Well Supported**	**Equivocal: No, Preliminary, or Mixed Data**	**Strong Contradictory Evidence**
Well Supported	Category I: Evidence-based psychotherapies None	Category II: Intervention-driven psychotherapies None	Category V: Good-intervention- and bad-theory-driven psychotherapies None
Equivocal: No, Preliminary, or Mixed Data	Category III: Theory-driven psychotherapies None	Category IV: Investigational psychotherapies BT; CAT; CBT; EOIT; FBT; FPT; IPT; MANTRA; NC	Category VII: Bad-theory-driven psychotherapies None
Strong Contradictory Evidence	Category VI: Good-theory- and bad-intervention-driven psychotherapies None	Category VIII: Bad-intervention-driven psychotherapies None	Category IX: Bad-theory- and bad-intervention-driven psychotherapies None

BT: behavior therapy; CAT: cognitive analytical therapy; CBT: cognitive–behavioral therapy; EOIT: ego-oriented individual therapy; FBT: family-based therapy; FPT: focal psychodynamic therapy; IPT: interpersonal therapy; MANTRA: Maudsley model of anorexia nervosa treatment for adults; NC: nutritional counseling.

concerning the theory of change underlying the clinical protocol are equivocal (i.e., no data, preliminary, or mixed). We did not identify any therapies appropriate for this category.

8.3.3 Category III: Theory-Driven Psychotherapies

Category III includes treatments with strong supporting evidence for the etiological theory of the disorder (i.e., anorexia) but with equivocal results regarding the efficacy/effectiveness of the clinical protocol. We did not identify any psychological interventions appropriate for this category.

8.3.4 Category IV: Investigational Psychotherapies

Category IV includes psychological treatments for which the evidence for both components (efficacy/effectiveness of the clinical protocol and the theory of change) is equivocal (i.e., where there is neither strong supporting nor contradictory evidence). Interventions appropriate for this category are presented below.

8.3.4.1 Family-based therapy

Family-based therapy (FBT) is an outpatient intervention, usually used for adolescents, designed to restore weight. The focus of FBT is not on what causes anorexia, as it does

not maintain that family dynamics plays a causal role in the pathogenesis of the disorder (Lock, Agras, Bryson, & Kraemer, 2005). FBT is focused on behavioral recovery, achieved by empowering family members to support the recovery of the adolescent. The treatment consists of three phases (Lock, Le Grange, Agras, & Dare, 2001). During the first phase, the main aims are refeeding and weight gain by teaching families how to manage the eating behavior and physical activity of the adolescent. In this phase, the responsibility for the eating pattern of the adolescent is placed in the hands of the parents, while the autonomy of the adolescent in other domains is preserved. The second phase addresses the psychological factors that interfere with refeeding and teaches problem-solving strategies within the family, while the adolescent gradually begins to take control of his/her own eating. The final phase focuses on more general family and psychological issues to increase adolescent autonomy and establish proper family boundaries.

Another family treatment is behavioral family systems therapy (BFST), which is similar to FBT in a number of respects. BFST addresses cognitive distortions and issues within the family structure following three stages of treatment, which are highly similar to those in FBT (Ball & Mitchell, 2004; Robin, Siegel, & Moye, 1995). Given their similarity, the two therapies are considered together in this chapter. Although other forms of family treatments are also available (e.g., structural family therapy; Minuchin, Rosman, & Baker, 1978), we only discuss interventions for which sufficient data exist to be evaluated according to David and Montgomery's (2011) classification system.

FBT is listed as having strong research support by the APA (APA Presidential Task Force on Evidence-Based Practice, 2006). NICE states that "family interventions that directly address the eating disorder should be offered to children and adolescents with anorexia nervosa" (2004, p. 90). Based on the data synthesized in a recent meta-analysis (Fisher, Hetrick, & Rushford, 2010) and on several randomized controlled trials (RCTs), we argue that FBT for anorexia belongs in Category IV of David and Montgomery's (2011) classification: investigational psychotherapies.

8.3.4.1.1 Absolute efficacy

We did not identify any study that compared FBT with a no-treatment or a waitlist control group. As no studies document FBT's absolute efficacy, relative efficacy should be interpreted with caution (for a discussion see Temple & Ellenberg, 2000). This observation applies to all treatment studies lacking a no-treatment or waitlist control group.

8.3.4.1.2 Relative efficacy

One recent meta-analysis (Fisher et al., 2010) reported a significant increase in postintervention remission rates of individuals treated with FBT compared with individuals in the standard care and treatment as usual groups (two RCTs; RR 3.83 95%; CI 1.60 to 9.13). Also, a small-scale RCT (Ball & Mitchell, 2004; $N = 25$) that compared FBT and cognitive–behavioral therapy (CBT) found no differences between groups at posttreatment or follow-up. At the end of the treatment, 60% of the total sample ($N = 15$) could be categorized as "good/intermediate" outcomes, with the gains maintained at follow-up (Ball & Mitchell, 2004).

8.3.4.1.3 Specific efficacy

The first criterion, regarding equivalence to other standard therapies (*equivalent to standard treatment*), is met (see Section 8.3.4.1.2). The second criterion, referring to the empirical support for the theory underlying the treatment package (i.e., specific mechanisms of change), is not met. FBT does not propose a theory regarding the mechanisms that lead to the onset of anorexia; however, family dysfunction is considered to play a role in the course and maintenance of the disorder (Holtom-Viesel & Allan, 2014). One study shows that the extent of family dysfunction is negatively associated with anorexia nervosa severity, and clinical improvement is not associated with improvement in family functioning (Gowers & North, 1999). Another study shows that healthy family functioning predicts good short-term treatment outcomes (North, Gowers, & Byram, 1997). The current status of FBT, from the point of view of the theory allied with the treatment, can be thus classified as having *preliminary and mixed data*.

8.3.4.2 Cognitive–behavioral therapy

CBT for anorexia nervosa is based on the notion that shape- and weight-related concerns engender dietary restriction and other extreme methods of weight control that maintain anorexic symptoms (Fairburn, Shafran, & Cooper, 1999). CBT for anorexia employs behavioral strategies that include establishing a regular eating pattern, reintroducing forbidden foods, and eliminating frequent body-checking or avoidance, while simultaneously addressing problematic thinking patterns and cognitive aspects of the disorder, such as motivation for change and perfectionism.

The APA Presidential Task Force on Evidence-Based Practice (2006) lists CBT for anorexia as having modest research support for posthospitalization relapse prevention and a controversial status for acute weight gain. NICE (2004) lists CBT as one of the therapies to be considered in the psychological treatment of anorexia and further notes that limited or insufficient evidence exists regarding its efficacy. We argue that, following the criteria of David and Montgomery (2011), CBT for anorexia can be classified as an investigational psychotherapy.

From the point of view of treatment package *efficacy*, the evidence for CBT can be classified as *preliminary data*. We did not identify any study that investigated its *absolute efficacy*. Regarding *relative efficacy*, one RCT (McIntosh et al., 2005) found that CBT was equivalent to a control group in which participants received a treatment that combined clinical management and supportive psychotherapy (nonspecific supportive clinical management), developed to mimic the kind of outpatient treatment that can be offered to individuals with anorexia in usual clinical practice. Similar results were reported by another RCT comparing CBT with optimized treatment as usual (Zipfel et al., 2014). CBT was also compared with a specialist supportive clinical management intervention in an RCT that found the two treatments to be equivalent at posttreatment but that patients in the CBT group had lower levels of eating disorder symptoms at 12-month follow-up (Touyz et al., 2013). Another RCT compared a control group (i.e., half-hour meetings with a psychiatrist focused on weight monitoring and restoration) with a CBT intervention and a group that received a behavioral intervention (Channon, De Silva, Hemsley, & Perkins, 1989). However, the CBT and the behavioral intervention were merged when compared with the control group; thus, the specific effect of CBT could not be isolated. Another study that compared CBT with FBT found no

differences between the two treatments (*equivalent to standard treatment*; Ball & Mitchell, 2004).

In terms of the mechanisms of change proposed by CBT, several correlational studies show that assumptions and negative beliefs related to eating disorders are associated with symptoms of anorexia (Cooper & Turner, 2000; Turner & Cooper, 2002). For example, in a pre–post treatment study, reduction in weight and shape concerns was associated with weight gain at the end of treatment and at 3-year follow-up (Ricca et al., 2010). To our knowledge, no experimental studies or controlled trials reported mediation or moderation analyses in investigating the relationship between dysfunctional attitudes and treatment outcomes. Therefore, we evaluate the CBT theory of change in anorexia to be supported by *preliminary data*.

8.3.4.3 Cognitive analytical therapy

Cognitive analytical therapy (CAT) combines brief, focused psychodynamic psychotherapy with elements of cognitive therapy. The therapist assumes a nondirective approach and collaborates with the patient to reformulate the patient's history and problems (Ryle, 1990). In this process, patients analyze the role and place of anorexia in their experience of themselves and their early and current relationships. CAT integrates psychodynamic factors (focused on interpersonal and transference issues) with behavioral ones and aims to help patients manage their emotions and relationships and eliminate the need for anorexia (Treasure et al., 1995). The CAT procedural sequence model maintains that all aim-directed activity is the consequence of several ordered sequences (i.e., aim generation, environmental evaluation, plan formation, action, evaluation of consequences, and remedial procedural revision; Denman, 2001). These procedural sequences are developed and shaped on the basis of experience and are regularly revised if they prove ineffective. Although these procedural sequences are usually effective and adaptive, whenever a person faces emotional problems, some of these sequences will be faulty and will be repeatedly deployed without revision. A second cause of neurotic difficulty is the presence of restrictions in the procedural repertoire, which may be due to reduced environmental opportunities for learning new procedures, deliberate attempts by caregivers to limit them, and difficulties in new emotional learning due to previously learned faulty procedures (Denman, 2001). NICE (2004) lists CAT as a therapy that should be considered in the psychological treatment of anorexia.

Evidence regarding the efficacy of CAT is limited, as *absolute efficacy* has not been established. Only two RCTs investigated its *relative efficiency*. The first one (Treasure et al., 1995) showed that CAT is equivalent to educational behavioral treatment (the authors state that this intervention is similar to treatment as usual). The second RCT (Dare, Eisler, Russell, Treasure, & Dodge, 2001) found no significant differences between CAT and routine treatment or between CAT and family treatment. The clinical protocol can be thus categorized as supported by *preliminary data*.

To our knowledge, no studies have investigated the mechanisms of change proposed by CAT. Therefore, we evaluate the theory as supported by *no data*. We argue that, following the criteria of David and Montgomery (2011), CAT for anorexia can be classified as an investigational psychotherapy.

8.3.4.4 Interpersonal therapy

Interpersonal therapy (IPT) focuses on the interpersonal difficulties in the patient's life. The clinician takes a history of the patient's significant life events, relationships, and eating problems, and investigates the link between these problems and the development and maintenance of the eating disorder (McIntosh et al., 2005). After identifying specific interpersonal problem areas currently affecting the patient (within the four interpersonal problem areas: grief, interpersonal disputes, role transitions, and interpersonal deficits), the therapist encourages the patient to take the lead in facilitating change in addressing interpersonal problems (McIntosh, Bulik, McKenzie, Luty, & Jordan, 2000). The goal of IPT is to help patients identify and alleviate current interpersonal problems.

One study found that IPT is inferior to nonspecific supportive clinical management and equivalent to CBT (*relative efficacy*; McIntosh et al., 2005). Considering that *absolute efficacy* for IPT has not been established, the analysis of *relative efficacy* should be considered with caution, as it could wrongly suggest that IPT is as efficacious as other psychological treatments that have proven to have *absolute efficacy*.

To our knowledge, no studies have investigated the mechanisms of change proposed by IPT. We argue that, following David and Montgomery's (2011) criteria, IPT for anorexia can be classified as an investigational psychotherapy supported by *preliminary and mixed data* for the clinical package and *no data* for the theory.

8.3.4.5 Maudsley model of anorexia nervosa treatment for adults (MANTRA)

The model underpinning the Maudsley model of anorexia nervosa treatment for adults (MANTRA) maintains that anorexia typically appears in people with anxious and/or obsessional traits. Anorexia is maintained by four factors: cognitive rigidity and attention to detail, impairments in the socioemotional domain, beliefs about the utility of anorexia nervosa in the patient's life, and certain actions of parents or partners (Schmidt & Treasure, 2006). The conceptualization and treatment plan are presented using a motivational interviewing style. The treatment is guided by the principles of behavioral change.

One study investigating the efficacy of this treatment reported no differences in outcomes compared to specialist supportive clinical management (*relative efficacy*; Schmidt et al., 2012). The same study showed no treatment effects with respect to cognitive inflexibility, a factor presumed to maintain disordered eating. Based on this limited evidence, we argue that MANTRA is supported by *preliminary data* for both the clinical protocol and the theory, and should be included in Category IV.

8.3.4.6 Ego-oriented individual therapy

Ego-oriented individual therapy (EOIT) aims to increase the patient's self-acceptance and to "decrease ineffectiveness and poor identity formation associated with the pursuit of thinness" (Robin et al., 1999, p. 1484). Therapy sessions are focused on building patients' ego strength, coping skills, and interpersonal issues related to physical, social, and emotional domains, and patients' concerns regarding their eating behavior, weight expectations, and body shape and image. The only study that has compared this treatment with a standard one (FBT) reported that EOIT is inferior in terms of weight gain and rates of restoring menstruation (*relative efficacy*; Robin et al., 1999). Therefore, there

are only *preliminary negative data* regarding its efficacy and *no data* regarding the theory underlying the treatment package.

8.3.4.7 Focal psychodynamic therapy

Focal psychodynamic therapy (FPT) is a short-term psychodynamic therapy that focuses mainly on the therapeutic alliance, the psychological meaning of the symptoms in light of the patient's history, and the association between interpersonal relationships and eating behavior, ego-syntonic beliefs, and self-esteem (Zipfel et al., 2014). One study found FPT superior to routine treatment in terms of decreasing weight gain rates and increasing remission rates (*relative efficiency*; Dare et al., 2001). Researchers also compared FPT with CBT and found no significant differences on the primary outcome measure (i.e., body mass index), although CBT was more effective with respect to decreasing speed of weight gain and improving eating disorder psychopathology (Zipfel et al., 2014). Given the limited evidence, we include FBT in Category IV, with *preliminary and mixed data* for the intervention package and *no data* for the theory, as we were not able to identify any study investigating the theory.

8.3.4.8 Behavioral therapy

The aim of treatment in behavioral therapy (BT) is to build graded hierarchies of feared foods and situations and to follow this up with graded exposure to these items under safe conditions. We identified only one study that compared BT with CBT, and it did not reveal significant differences on any primary outcome measure (*relative efficiency*; Channon et al., 1989). We evaluate BT as supported by *preliminary data* for the clinical protocol and *no data* regarding the theory.

8.3.4.9 Nutritional counseling

Nutritional counseling (NC) is a psychoeducational and supportive intervention that focuses on specific dietary analyses and balanced meal planning. In one study in which this intervention was compared with a standard one, researchers found that criteria for "good outcome" were met by significantly more patients who received CBT (44%) than patients in the NC group (7%; Pike, Walsh, Vitousek, Wilson, & Bauer, 2003). Another clinical study found no differences in body mass index for cognitive therapy versus nutritional/dietary counseling (Serfaty, Turkington, Heap, Ledsham, & Jolley, 1999). Thus, only *preliminary data* support the efficacy of NC. There are *no data* regarding the theory of change, as NC does not rely on a specific theory of the disorder. Nevertheless, a poor diet and meal planning might play a role in the maintenance of the disorder, but we did not identify any study assessing their impact on anorexia nervosa treatment outcome.

8.3.5 Categories V, VI, VII, VIII, and IX

These categories refer to:

- good-intervention- and bad-theory-driven psychotherapies (Category V);
- good-theory- and bad-intervention-driven psychotherapies (Category VI);
- bad-theory-driven psychotherapies (Category VII);
- bad-intervention-driven psychotherapies (Category VIII);
- bad-theory- and bad-intervention-driven psychotherapies (Category IX).

We did not identify any psychotherapeutic interventions in any of these five categories.

8.4 Classification of Psychological Interventions for Binge Eating Disorder

Table 8.2 A graphical representation of the classification of psychological interventions for binge eating disorder using David and Montgomery's (2011) evaluative framework.

	Theory		
Therapeutic Package	**Well Supported**	**Equivocal: No, Preliminary, or Mixed Data**	**Strong Contradictory Evidence**
Well Supported	Category I: Evidence-based psychotherapies CBT	Category II: Intervention-driven psychotherapies None	Category V: Good-intervention- and bad-theory-driven psychotherapies None
Equivocal: No, Preliminary, or Mixed Data	Category III: Theory-driven psychotherapies None	Category IV: Investigational psychotherapies BWC; DBT; IPT	Category VII: Bad-theory-driven psychotherapies None
Strong Contradictory Evidence	Category VI: Good-theory- and bad-intervention-driven psychotherapies None	Category VIII: Bad-intervention-driven psychotherapies None	Category IX: Bad-theory- and bad-intervention-driven psychotherapies None

BWC: behavioral weight control; CBT: cognitive–behavioral therapy; DBT: dialectical behavior therapy; IPT: interpersonal therapy.

8.4.1 Category I: Evidence-Based Psychotherapies

This category includes psychological interventions for binge eating disorder with a validated clinical protocol and validated theoretical framework. Cognitive–behavioral therapy and guided self-help (based on CBT principles) can be included in this category.

8.4.1.1 Cognitive–behavioral therapy for binge eating disorder (CBT-BED)

Cognitive–behavioral therapy for binge eating (CBT-BED) targets the main features of the disorder, namely binge eating and the associated emotional distress and loss of control. Treatment addresses the maladaptive eating patterns that maintain binge eating (Wilfley et al., 1993). The APA Presidential Task Force on Evidence-Based Practice (2006) lists CBT as having strong support, whereas the NICE guideline states that "cognitive behavior therapy for binge eating disorder (CBT-BED), a specifically adapted form of CBT, should be offered to adults with binge eating disorder" (NICE, 2004, p. 161).

Our assessment of treatment package efficacy is based on the NICE (2004) guideline for eating disorders.

8.4.1.1.1 Absolute efficacy

CBT is more efficacious than waitlist (*better than waitlist*) both in terms of remission ($N = 4$; $n = 226$; Random Effects RR = 0.64; 95% CI 0.49 to 0.84; NNT = 3; 95% CI 2 to 7) and in terms of reducing the frequency of binge eating at the end of treatment ($N = 4$; $n = 214$; Random Effects SMD = −1.30; 95% CI −2.13 to −0.48).

8.4.1.1.2 Relative efficacy

CBT was found to be as efficacious (*equivalent to standard treatment*) as other standard treatments (i.e., interpersonal psychotherapy) in reducing binge eating frequency, both at the end of the treatment ($N = 2$; $n = 194$; SMD = −0.07; 95 % CI −0.35 to 0.22) and at follow-up ($N = 1$; $n = 138$; SMD = 0.14; 95% CI −0.19 to 0.48).

8.4.1.1.3 Specific efficacy

The first criterion, referring to equivalence to other standard therapies (*equivalent to standard treatment*), is met with IPT (see Section 8.4.1.1.2). The second criterion, referring to the empirical support for the theory underlying the treatment package, is also met. We argue that there is empirical support for the mechanisms of change proposed by the CBT-BED theory, investigated in the following studies:

- *Experimental studies*. Changes in cognitive factors included in the cognitive model of binge eating (i.e., shape and weight concerns, body image, disturbed attitudes about eating) are associated with changes in binge eating symptoms (Smith, Marcus, & Kaye, 1992; Wolff & Clark, 2001).
- *Complex controlled trials with mediation analyses*. The reduction of weight concerns and changes in eating and shape concerns mediate binge eating symptom reduction at posttreatment (Dingemans, Spinhoven, & Van Furth, 2007).
- *Complex controlled trials with moderation analysis*. Shape- and weight-related concerns and overvaluation at pretreatment and midtreatment moderate treatment outcomes (e.g., predicted treatment nonresponse or binge eating remission at posttreatment; Grilo, Masheb, & Crosby, 2012; Hilbert et al., 2007).

Based on the existing data, we argue that both the treatment package and the underlying theory are validated. CBT is delivered not only in traditional settings (i.e., individual or group) but also in guided or pure self-help formats. Given that a number of studies have specifically investigated the efficacy of these CBT delivery methods, we present below the status of the evidence for CBT offered in these formats.

8.4.1.2 Guided self-help cognitive–behavioral therapy

Guided self-help CBT (GSH-CBT) is designed following the principles of CBT, but a specialist or a facilitator with limited clinical training assists the patient in the process. Assistance is offered face to face or via other means of communication (e.g., telephone, internet) in an individual or group setting. Participants are typically asked to read a book or watch a video while they are guided in following the self-help program. The self-help program consists of a psychoeducation component, which provides the rationale for the self-help program, and the program itself, which involves several additive steps regarding how to change eating habits or associated problems (Carter & Fairburn, 1998).

We evaluate GSH-CBT as having strong evidence regarding its treatment package based on the following data.

8.4.1.2.1 Absolute efficacy

GSH-CBT was found to be more efficacious than waitlist (*better than waitlist*) in reducing the frequency of binge eating behaviors (Carrard et al., 2011; Carter & Fairburn, 1998; Peterson et al., 1998).

8.4.1.2.2 Relative efficacy

GSH-CBT was found to be as effective as IPT (Wilson, Wilfley, Agras, & Bryson, 2010). Also, GSH-CBT was found to be superior to a control comparison condition (self-monitoring without providing a treatment manual) in terms of remission rates (Grilo & Masheb, 2005).

8.4.1.2.3 Specific efficacy

The first criterion, regarding equivalence to other standard therapies (*equivalent to standard treatment*), is met with IPT (see Section 8.4.1.2.2). The second criterion, regarding empirical support for the theory underlying the treatment package, is also met.

Given that GSH-CBT is based on the same theory of the disorder as CBT, the theory allied with the treatment protocol can be considered validated. However, mediation and moderation studies are needed in order to explore whether other mechanisms of change may also be involved in the delivery of CBT in this guided self-help format. We identified only one study (using a sample of 38 patients receiving GSH-CBT) that documented that higher regular eating adherence was associated with lower binge weekly frequency (Zendegui, West, & Zandberg, 2014).

8.4.1.3 Pure self-help cognitive–behavioral therapy

Pure self-help cognitive–behavioral therapy (PSH-CBT) is based on the same principles as GSH-CBT but does not involve professional assistance. Rather, it entails the self-administration of a program that addresses disordered eating. Participants are typically asked to read a book or watch video material, and to do their best to follow the program.

In terms of *absolute efficacy*, PSH-CBT was found to be more efficacious in reducing the frequency of binge eating behavior than a waitlist control (*better than waitlist*) in three RCTs (Carter & Fairburn, 1998; Peterson et al., 1998; Peterson, Mitchell, Crow, Crosby, & Wonderlich, 2009) but not in reducing abstinence rates (Peterson et al., 2009). We did not identify any study comparing PSH-CBT with an evidence-based intervention (e.g., IPT). We argue that only *preliminary and mixed data* support the efficacy of CBT delivered in a PSH format. Given that PSH-CBT is based on the same theory of the disorder as CBT, we evaluate the theory allied with the treatment protocol as validated. However, as noted in Section 8.4.1.2.3, it is important to test whether there are also other mechanisms of change for CBT delivered in this particular format. We did not identify any mediation or moderation studies investigating mechanisms of change for PSH-CBT.

8.4.2 Category II: Intervention-Driven Psychotherapies

This category refers to treatments with a well-validated treatment protocol and equivocal evidence for the theory. We did not identify any psychological treatments that can be included in this category.

8.4.3 Category III: Theory-Driven Psychotherapies

This category includes treatments with a well-validated theory but with equivocal evidence for the clinical protocol. Based on the analysis of the literature, no psychological interventions can be definitively included in this category.

8.4.4 Category IV: Investigational Psychotherapies

This category includes psychological interventions characterized by equivocal evidence for both theory and treatment package. The following interventions are included in this category.

8.4.4.1 Interpersonal psychotherapy

IPT for binge eating disorder follows a similar approach to that described for anorexia, the focus being on interpersonal difficulties in the patient's life. IPT assumes that binge eating occurs as a response to interpersonal problems and consequent negative moods. The APA Presidential Task Force on Evidence-Based Practice (2006) lists IPT as having strong research support, and NICE (2004) recommends IPT for adults with persistent binge eating disorder. Based on the data summarized in the following, IPT qualifies for Category IV (investigational psychotherapies) of David and Montgomery's (2011) classification. Our assessment is based on the NICE (2004) guideline for eating disorders.

8.4.4.1.1 Absolute efficacy

IPT is superior to waitlist (*better than waitlist*) both in terms of remission rate ($N = 1$; $n = 38$; RR = 0.56; 95% CI 0.37 to 0.84; NNT = 3; 95% CI 2 to 5) and mean frequency of binge eating by the end of the treatment ($N = 1$; $n = 38$; SMD = −1.44; 95% CI −2.16 to −0.72).

8.4.4.1.2 Relative efficacy

As shown in Section 8.4.1.1, IPT was found to be as effective as CBT in two RCTs (Wilfley et al., 1993, 2002).

8.4.4.1.3 Specific efficacy

The first criterion, concerning equivalence to other standard therapies (*equivalent to standard treatment*), is met with CBT (see Section 8.4.4.1.2). The second criterion, regarding empirical support for the theory underlying the treatment package, is not met.

We argue that the data related to IPT efficacy are still *preliminary*; only two studies are reported in the literature, and they were conducted by the same team (Wilfley et al., 1993, 2002). Regarding the theory of change, only one study found that the extent of interpersonal problems prior to treatment and at midtreatment moderated treatment outcomes (i.e., predicted treatment nonresponse at posttreatment and at 1-year follow-up; Hilbert et al., 2007). One clinical trial reported significant improvements in interpersonal functioning during the course of treatment (Wilfley et al., 2002). Based on this evidence, we evaluate IPT theory as supported by *preliminary data*.

8.4.4.2 Behavioral weight control

Behavioral weight control (BWC) combines moderate caloric restriction and exercise. The program focuses on establishing a healthy meal pattern by self-monitoring exercise, fat intake, and caloric intake. Based on the available data, we argue that BWC should also

be included in Category IV (investigational psychotherapies). We evaluate the treatment package as having *mixed data*, based on the following evidence.

8.4.4.2.1 Absolute efficacy

Limited evidence regarding the absolute efficacy of BWC is available. One study found that BWC is superior to waitlist (*better than waitlist*) on mean frequency of binge eating by the end of treatment ($N = 1$; $n = 82$; SMD = −0.45; 95% CI −0.89 to −0.01; Reeves et al., 2001).

8.4.4.2.2 Relative efficacy

Studies that compared BWC with other standard treatments for binge eating disorder found BWC to be as effective as CBT in reducing binge eating in the short term in some cases (e.g., Munsch et al., 2007) and less effective in other cases (e.g., Agras et al., 1994; Grilo & Masheb, 2005). Also, BWC is less effective than IPT or CBT in terms of remission at follow-up (Nauta, Hospers, Kok, & Jansen, 2000; Wilson et al., 2010).

8.4.4.2.3 Specific efficacy

The first criterion, equivalence to other standard therapies (*equivalent to standard treatment*), is not met, as the evidence is mixed (see Section 8.4.4.2.2). The second criterion, regarding empirical support for the theory underlying the treatment package, is not met. As BWC does not target specific etiological factors, mechanisms of change directly related to what caused the disorder cannot be evaluated (*no data*). In terms of the maintenance of binge eating disorder, unhealthy meal patterns might be considered a maintaining factor, but to our knowledge no studies have investigated its mediator or moderator role.

8.4.4.3 Dialectical behavior therapy

Dialectical behavior therapy (DBT) aims to eliminate binge eating by ameliorating emotion dysregulation by teaching patients adaptive emotion regulation skills. The assumption underlying of DBT as applied to binge eating disorder is that "individuals with [binge eating disorder] have difficulties regulating negative emotions, and that under conditions of high negative arousal, binge eating rapidly reduces aversive emotional experiences and thus is maintained by negative reinforcement"; in addition, "treatment outcome is mediated by three factors: (a) acquisition and strengthening of adaptive methods of emotion regulation; (b) enhanced ability to tolerate negative emotions; and (c) increased awareness of the longer-term aversive consequences of binge eating and the positive consequences of alternative behaviors" (Telch, Agras, & Linehan, 2000, p. 571).

We argue that DBT should be classified as supported by *preliminary data* for efficacy. Concerning *absolute efficacy*, one study found that DBT is superior to waitlist (*better than waitlist*) in terms of remission at the end of treatment ($N = 1$; $n = 44$; RR = 0.30; 95% CI 0.15 to 0.60; NNT = 2; 95% CI 1 to 3; Telch, Agras, & Linehan, 2001). Another study reports that DBT is superior to a control comparison group (psychological placebo) in terms of abstinence from binge eating at the end of the treatment, but not at follow-up (*relative efficacy*; Safer, Robinson, & Jo, 2010).

We identified several experimental studies that offer *preliminary data* for the theory underlying the treatment package. Two studies indicate that induced negative mood

compared with neutral mood significantly increases loss of control over eating and the occurrence of binge eating in women with binge eating disorder (Agras & Telch, 1998; Telch & Agras, 1996). Another study documented improvements in expectancies for self-regulation of negative moods and a reduction in the urge to eat when participants experienced negative moods after treatment (Telch et al., 2000). Finally, greater improvement in emotion regulation between pre- and posttreatment predicted binge eating abstinence at posttreatment and follow-up in a sample of 60 community men and women with binge eating disorder receiving guided self-help DBT (Wallace, Masson, Safer, & von Ranson, 2014).

8.4.5 Categories V, VI, VII, VIII, and IX

We did not identify any psychotherapeutic treatments that would fit into any of these five categories.

8.5 Classification of Psychological Interventions for Bulimia Nervosa

Table 8.3 A graphical representation of the classification of psychological interventions for bulimia nervosa using David and Montgomery's (2011) evaluative framework.

	Theory		
Therapeutic Package	**Well Supported**	**Equivocal: No, Preliminary, or Mixed Data**	**Strong Contradictory Evidence**
Well Supported	Category I: Evidence-based psychotherapies CBT	Category II: Intervention-driven psychotherapies FSP	Category V: Good-intervention- and bad-theory-driven psychotherapies None
Equivocal: No, Preliminary, or Mixed Data	Category III: Theory-driven psychotherapies None	Category IV: Investigational psychotherapies BT; DBT; DC; FBT; IPT; POSP; PP	Category VII: Bad-theory-driven psychotherapies None
Strong Contradictory Evidence	Category VI: Good-theory- and bad-intervention-driven psychotherapies None	Category VIII: Bad-intervention-driven psychotherapies None	Category IX: Bad-theory- and bad-intervention-driven psychotherapies None

BT: behavior therapy; CBT: cognitive–behavioral therapy; DBT: dialectical behavioral therapy; DC: dietary counseling; FBT: family-based therapy; FSP: focal supportive psychotherapy; IPT: interpersonal therapy; POSP: psychodynamically oriented supportive psychotherapy; PP: psychodynamic psychotherapy.

8.5.1 Category I: Evidence-Based Psychotherapies

8.5.1.1 Cognitive–behavioral therapy

CBT for bulimia nervosa (CBT-BN) is based on the same principles as those described for anorexia and binge eating and directly addresses binge eating, unhealthy

compensatory behaviors, and excessive preoccupation with body shape and weight, which are the core features of this disorder (Loeb et al., 2005). Treatment focuses on the binge–purge cycle and on how it perpetuates symptoms. The APA Division of Clinical Psychology lists CBT-BN as having strong support (American Psychological Association Presidential Task Force on Evidence-Based Practice, 2006) whereas NICE states that CBT-BN "should be offered to most adults with bulimia nervosa" (2004, p. 128). We include CBT-BN in Category I (evidence-based interventions) of David and Montgomery's (2011) framework, with both treatment package and theory of change well validated.

In the following, we summarize the evidence in favor of CBT-BN. In light of this evidence, based on the NICE (2004) guideline for eating disorders, we evaluate CBT-BN as having strong support for the intervention package.

8.5.1.1.1 Absolute efficacy

Strong evidence indicates that CBT is more efficacious that waitlist control (*better than waitlist*), both in terms of remission ($N = 3$; $n = 136$; RR = 0.73; 95% CI 0.61 to 0.88; NNT = 4; 95% CI 3 to 9) and in terms of reducing the frequency of binge eating ($N = 5$; $n = 185$; SMD = −0.75; 95% CI −1.05 to −0.44) and purging symptoms ($N = 6$; $n = 192$; Random Effects SMD = −1.00; 95% CI −1.63 to −0.36) at the end of treatment (NICE, 2004).

8.5.1.1.2 Relative efficacy

One study found CBT to be superior to placebo in terms of cessation of binge–purge episodes (Thackwray, Smith, Bodfish, & Meyers, 1993). There is strong evidence that CBT is more efficacious that IPT in terms of remission of binge eating at the end of treatment ($N = 2$; $n = 270$; RR = 0.77; 95% CI 0.67 to 0.87; NNT = 5; 95% CI 4 to 20; NICE, 2004).

Based on the following evidence, we argue that there is also strong support for the CBT theory. First, experimental studies have found that decreases in body-related dysfunctional beliefs are associated with decreases in weight concern during treatment and that increases in dietary restraint are associated with increases in vomiting frequency (Spangler, Baldwin, & Agras, 2004). In addition, increases in self-efficacy are associated with decreases in binge eating and purging (Wilson & Fairburn, 1993).

Second, a complex controlled trial involving mediation analysis has shown that changes in dietary restraint mediate posttreatment improvement in both binge eating and vomiting (Wilson, Fairburn, Agras, Walsh, & Kraemer, 2002). The same study indicates that the treatment effect on binge eating frequency at follow-up is fully mediated by the change in dietary restraint at week 4.

In the following, we discuss evidence for the delivery of CBT in a guided self-help or pure self-help manner.

8.5.1.2 Guided self-help cognitive–behavioral therapy

As in the case of binge eating disorder, guided self-help programs for bulimia are based on CBT's principles and theory of the disorder. The theory allied with these programs is thus well validated; however, mediation and moderation analyses are needed to clarify

whether specific mechanisms of change related to this method of delivery exist. There is also strong evidence regarding treatment package efficacy.

8.5.1.2.1 Absolute efficacy

Compared with waitlist control, GSH-CBT improves remission rates at the end of treatment (*better than waitlist*; Banasiak, Paxton, & Hay, 2005; Palmer, Birchall, McGrain, & Sullivan, 2002) and at follow-up (Sánchez-Ortiz et al., 2011).

8.5.1.2.2 Relative efficacy

GSH-CBT is equivalent to CBT in terms of remission rates (*equivalent to standard treatment*; Bailer et al., 2004).

8.5.1.2.3 Specific efficiency

The first criterion, referring to equivalence to other standard therapies (*equivalent to standard treatment*), is met with CBT. The second criterion, regarding empirical support for the theory underlying the treatment package, is also met.

8.5.1.3 Pure self-help cognitive–behavioral therapy

We assess the data available so far for PSH-CBT as *preliminary and mixed*. While the theory allied with the programs is well validated, as it reflects the CBT conceptualization of the disorder, studies investigating specific mechanisms of change for this type of delivery format are needed. Evidence for the treatment package is summarized as follows.

8.5.1.3.1 Absolute efficacy

Limited evidence suggests that, compared with waitlist control, PSH-CBT does not improve the remission of binge eating ($N = 2$; $n = 139$; RR = 0.96; 95% CI 0.85 to 1.09) or purging ($N = 2$; $n = 139$; RR = 0.97; 95% CI 0.87 to 1.07) at the end of treatment (NICE, 2004).

Given that absolute efficacy is not established for PSH-CBT, its relative efficiency should be treated with caution.

8.5.1.3.2 Relative efficacy

Limited evidence indicates that there is no difference between PSH-CBT and CBT in terms of mean frequency of binge eating episodes at the end of treatment ($N = 1$; $n = 80$; SMD = 0.03; 95% CI −0.43 to 0.49; NICE, 2004).

8.5.1.3.3 Specific efficacy

The first criterion, referring to equivalence to other standard therapies (*equivalent to standard treatment*), is not met. The second criterion, regarding empirical support for the theory underlying the treatment package, is met.

8.5.2 Category II: Intervention-Driven Psychotherapies

8.5.2.1 Focal supportive psychotherapy

Focal supportive psychotherapy (FSP) is a nondirective therapy that is supportive and educational in orientation. The emphasis is on providing information on bulimia nervosa, self-monitoring, and self-disclosure. No specific theory of the disorder underlies this treatment. In terms of treatment package efficacy, the NICE (2004) guideline for eating disorders lists FSP as a treatment to be considered for bulimia and reports strong evidence for its efficacy.

8.5.2.1.1 Absolute efficacy

FSP reduces the frequency of binge eating ($N = 1$; $n = 50$; SMD = –1.12; 95% CI −1.73 to −0.51) and purging symptoms ($N = 2$; $n = 84$; SMD = –1.43; 95% CI −1.93 to −0.94) at the end of treatment compared with controls (*better than waitlist*; NICE, 2004).

8.5.2.1.2 Relative efficacy

FSP is equivalent to CBT (*equivalent to standard treatment*) in reducing the frequency of binge eating ($N = 3$; $n = 111$; SMD = 0.00; 95% CI −0.37 to 0.38) and purging ($N = 4$; $n = 144$; SMD = –0.13; 95% CI −0.46 to 0.20) at the end of treatment (NICE, 2004).

8.5.2.1.3 Specific efficiency

The first criterion, involving equivalence to other standard therapies (*equivalent to standard treatment*), is met with CBT. The second criterion, regarding empirical support for the theory underlying the treatment package, is not met.

8.5.3 Category III: Theory-Driven Psychotherapies

We did not identify any interventions that could be included in this category.

8.5.4 Category IV: Investigational Psychotherapies

8.5.4.1 Interpersonal therapy

IPT for bulimia is based on the same principles as described for anorexia and binge eating. The APA Presidential Task Force on Evidence-Based Practice (2006) lists IPT as having strong support and NICE recommends that IPT "should be considered as an alternative to CBT, but patients should be informed it takes eight to 12 months to achieve results comparable with CBT" (NICE, 2004, p. 128).

We did not find any study that compared IPT for bulimia with a no-treatment control or waitlist (i.e., *absolute efficacy*). As absolute efficacy for IPT has not yet been established, the analysis of *relative efficacy* should be considered with caution, as it could suggest IPT is as efficacious as other psychological treatments with demonstrated absolute efficacy.

With respect to *relative efficacy*, the NICE guideline for eating disorders (NICE, 2004) cites evidence that IPT is inferior to CBT in terms of binge eating ($N = 2$; $n = 270$; RR = 0.77; 95% CI 0.67 to 0.87; NNT = 5; 95% CI 4 to 20) and purging cessation ($N = 1$; $n = 220$; RR = 0.76; 95% CI 0.67 to 0.86; NNT = 5; 95% CI 4 to 8) at the end of treatment, but it is equally efficacious in reducing the frequency of binge eating at the end of

treatment ($N = 2$; $n = 262$; SMD = –0.24; 95% CI –0.48 to 0.01) and at posttreatment follow-up ($N = 2$; $n = 257$; SMD = –0.04; 95% CI –0.29 to 0.20). Thus, data regarding IPT efficacy are *preliminary and mixed*.

Regarding the theory allied with the treatment package, data from two clinical trials show that changes in social adjustment and interpersonal functioning are associated with changes in treatment outcomes (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000; Fairburn et al., 1991). We consider these data *preliminary* as they come from the same research team.

8.5.4.2 Dialectical behavioral therapy

As in binge eating disorder, the DBT model maintains that emotional dysregulation is the core problem in bulimia, and its symptoms (i.e., binge eating and purging behaviors) are seen as attempts to influence, change, or control painful emotional states. In order to counteract these problems, patients are taught a series of skills to replace these dysfunctional behaviors (Wiser & Telch, 1999).

Data on the efficacy of DBT for bulimia are *preliminary*. One study tested the *absolute efficacy* of DBT for bulimia nervosa and found that DBT is superior to waitlist control (*better than waitlist*) in terms of binge eating or purging remission at the end of treatment ($N = 1$; $n = 31$; RR = 0.75; 95% CI 0.57 to 1.00; NNT = 4; 95% CI 3 to 100; Safer, Telch, & Agras, 2001). We did not find any study that directly tested the theory allied with DBT in bulimia nervosa patients. Nevertheless, the proposed mechanisms are similar to those involved in binge eating disorder. Thus, based on the data regarding binge eating disorder (see Section 8.4), we conclude that *preliminary data* support the DBT model for bulimia nervosa.

8.5.4.3 Behavior therapy

As in anorexia, BT for bulimia is focused on the eating pattern itself and on reestablishing a normal eating pattern, while regaining control over eating and ceasing dieting. Evidence regarding BT's efficacy is limited. We did not find any study investigating the *absolute efficacy* of BT. In terms of *relative efficacy*, BT was found to be as efficacious as CBT in reducing the frequency of binge eating ($N = 3$; $n = 131$; SMD = –0.11; 95% CI –0.45 to 0.24) and purging ($N = 3$; $n = 131$; SMD = 0.08; 95% CI –0.27 to 0.42) at the end of treatment (NICE, 2004). Thus, *preliminary data* support the efficacy of the treatment protocol. We did not find any study that directly addressed the mechanisms of change proposed by BT for bulimia nervosa. However, findings that support CBT's theory, by documenting that changes in dietary restraint mediate posttreatment improvement in binge eating and vomiting (Wilson et al., 2002), also constitute support for BT. Based on these outcomes, we argue that there are *preliminary data* for BT's theory.

8.5.4.4 Family-based therapy

Family-based therapy (FBT) for bulimia is guided by the same principles as the family-based treatment of anorexia. The focus of therapy is not on what caused the bulimia but on what can be done to resolve the disorder; thus we can classify FBT theory as supported by *no data*. Nevertheless, as in the case of anorexia nervosa, family variables can be conceptualized as maintenance factors. *Preliminary data* document that negative family communication is a significant predictor of bulimic symptoms (Crowther, Kichler, Sherwood, & Kuhner, 2002), and family dysfunction is associated with bulimic

symptoms in college females at a low or moderate level (Murray, Waller, & Legg, 2000; Scalf-McIver & Thompons, 1989). Evidence regarding treatment package efficacy is also limited, so *absolute efficacy* has not been established. In terms of comparison with other standard treatments, one study found that a significantly higher proportion of patients who received guided self-help were free of binge eating after 6 months of treatment (i.e., posttreatment) compared with patients who received FBT (Schmidt et al., 2007). The same study showed no differences in binge eating between the two treatments at 12 months and in vomiting at 6 and 12 months. Another study, which compared FBT with supportive psychotherapy, reported that significantly more patients who received FBT were binge-and-purge abstinent both at the end of treatment and at 6-month follow-up (Le Grange, Crosby, Rathouz, & Leventhal, 2007). The evidence in favor of FBT can thus be classified as *preliminary*.

8.5.4.5 Dietary counseling

The aim of dietary/nutritional counseling (DC) is to educate patients about the principles of healthy nutrition and the relationship between dieting and overeating. Patients are taught how to record precise details of their eating behavior while introducing meal planning as a way to establish and maintain a pattern of regular eating (Hsu et al., 2001). DC is not an etiopathogenic treatment but rather a symptomatic one; therefore, the validity of the theory of change can be classified as supported by *no data*. However, meal patterns can be viewed as maintenance factors, and some limited evidence shows that increased consumption of evening meals over the course of treatment, for example, is associated with a significant decrease in binging and purging behavior at follow-up (Ellison et al., 2016). The evidence related to treatment efficacy is also limited. We only found three studies that investigated the efficacy of DC. In one, DC was compared with a control group (support group; *relative efficacy*) and the two groups did not differ in terms of reducing binge or vomiting frequency (Hsu et al., 2001). The same study found that cognitive therapy is more efficacious than DC in reducing bulimic symptoms (measured with the Eating Disorders Inventory; Garner, Olmsted, & Polivy, 1983). Another study found that patients who received DC had significantly higher abstinence rates from binge eating compared with those who received a stress management intervention (a combination of interpersonal and CBT techniques; Laessle et al., 1991). Finally, a third study reported that DC is inferior to CBT-BN in terms of reducing vomiting at 6-month and 18-month follow-up (Sundgot-Borgen, Rosenvinge, Bahr, & Schneider, 2002). Thus, data regarding the efficacy of DC for bulimia are *mixed and preliminary*.

8.5.4.6 Psychodynamically oriented supportive psychotherapy

Psychodynamically oriented supportive psychotherapy (POSP) is a nondirective treatment that emphasizes patient self-exploration and understanding. The aim of the intervention is to facilitate self-disclosure and to explore underlying emotional problems while fostering independence and responsibility for change (Walsh et al., 1997). We identified only two studies that evaluated the efficacy of POSP. The first found that supportive therapy is inferior to CBT both in terms of binge eating cessation and in terms of binge frequency reduction (Walsh et al., 1997). The second study reported that CBT and POSP are equally effective in reducing the frequency of binge eating but that CBT is superior in reducing the frequency of vomiting (*relative efficacy*; Garner et al., 1993). We did not find any study that investigated POPS's mechanisms of change. We evaluate

this intervention as supported by *preliminary and mixed data* regarding its efficacy and as having *no supportive data* for its theory.

8.5.4.7 Psychoanalytic psychotherapy

Psychoanalytic psychotherapy (PP) assumes that bulimic symptoms are rooted in a need to ward off inner-feeling states and desires (Clinton, 2006) and are generated by difficulties in acknowledging and regulating these inner states (Fonagy, Gergely, Jurist, & Target, 2002). The aims of therapy are to increase the patient's ability to tolerate affective experiences and to facilitate insight into the patient's unconscious mechanisms and disavowed experiences (McWilliams, 2004). One study that compared psychoanalytic psychotherapy (2 years of treatment) and CBT (5 months of treatment) found that PP is inferior to CBT in terms of abstinence from binge eating and purging (Poulsen et al., 2014). Given that we were not able to find any study investigating the theory, and that there is only one study investigating *relative efficacy*, we argue that PP can be classified as supported by *no data* for the theory and supported by *preliminary data* for the treatment package.

8.5.5 Categories V, VI, VII, VIII, and IX

We did not identify any psychotherapies that fit into any of these five categories.

8.6 Discussion and Conclusions

The data summarized and evaluated in this chapter indicate that, in the case of eating disorders, particularly anorexia nervosa, research on treatment packages and the underlying theories is still at an early stage. Few well-validated treatments exist and, even in these cases, data are limited. Also, few quantitative reviews synthesize the extant evidence, with some offering only limited information regarding the efficacy of psychological interventions. Specifically, (1) only two reviews provide data on the efficacy of psychological interventions compared with waitlist or treatment as usual (Fisher et al., 2010; Hay, Bacaltchuk, Stefano, & Kashyap, 2009); (2) studies tend to compare a specific intervention (e.g., CBT) with a combination of several other psychotherapeutic interventions, taken together (e.g., Fisher et al., 2010); (3) studies tend to focus on the efficacy of a specific intervention but include patients with different types of eating disorders (Couturier, Kimber, & Szatmari, 2013); and (4) studies tend to compare psychological interventions, taken together, with pharmacotherapy (Vocks et al., 2010). Given these limitations, our analysis relied mostly on individual clinical trials and the NICE (2004) guideline for eating disorders, which includes a synthesis of the data available until 2004.

Based on the data synthesized in this chapter, CBT (including the self-help format) is the only evidence-based intervention for eating disorders. More specifically, its efficacy has been proven for binge eating and bulimia, whereas in the case of anorexia no treatments fulfill David and Montgomery's (2011) criteria for evidence-based interventions. Data reviewed here suggest not only the lack of well-supported theories of eating disorders (which is the case for other disorders as well) but also the fact that there are few psychological interventions for which treatment efficacy has been demonstrated clearly.

Among eating disorders, anorexia most lacks efficacious interventions. In this case, we failed to identify well-validated and intervention-driven therapies. The best available therapy is an investigational one (i.e., FBT), which is effective mainly for adolescents and to a lesser extent for adults. The majority of the psychological interventions for eating disorders are investigational psychotherapies, with only preliminary or no data for the clinical protocol and the theory. Despite the fact that numerous interventions have been developed or adapted for eating disorders, most of them do not have established absolute efficacy, and head-to-head comparisons with standard treatments and placebo interventions are also limited. We found a large number of studies that tested an intervention by comparing it with other treatments that are not the standard.

Evidence related to the theories underpinning different psychological treatments is even more limited. The only validated theory for eating disorders (i.e., binge eating disorder and bulimia nervosa) is the cognitive–behavioral model, and, even in this case, researchers have conducted only a few studies to evaluate mediation or moderation in terms of specific treatment effects. Most psychotherapies offered to patients suffering from eating disorders are not supported by data regarding the theory, whereas only a few are supported by preliminary and positive findings.

8.6.1 Implications for Research

We strongly agree with the research recommendations of NICE (2004) and other reviews (Wilson, Grilo, & Vitousek, 2007) that highlight the need for adequately powered efficacy studies of specific psychological interventions for eating disorders; the need to discern between promising and less promising interventions in the case of anorexia and the need to focus clinical trials on promising interventions, based on the existing data (Agras et al., 2004); and the need for more efficacy studies on the treatment of adults with anorexia nervosa, of adolescents with bulimia nervosa, and of individuals with atypical eating disorders. Moreover, future reviews should also focus on the clinical significance of treatment results (e.g., large, clinically meaningful effect sizes and/or change in diagnostic status) in addition to statistical significance. It is important that analyses such as the one in this chapter also include effect sizes of the differences between various treatments and/or waitlist or treatment as usual. Such analyses would prove useful in terms of ascertaining clinical significance as well as statistical significance.

Theory validation is another important issue for future research as it can inform practice by suggesting new techniques for approaching disordered eating more efficiently. From this point of view, a valuable research direction would be to test theoretical mechanisms hypothesized to moderate or mediate treatment effects, particularly for treatments with demonstrably efficacious clinical packages.

8.6.2 Implications for Practice

The assessment of psychological treatments for eating disorders based on the framework proposed by David and Montgomery (2011) supports the recommendations of existing clinical guidelines that family-based intervention should be the treatment of choice for anorexia nervosa and that CBT should be the first line of intervention for bulimia nervosa and binge eating disorder. However, in the case of anorexia, our analysis

also shows that, although FBT is the recommended treatment, there is not enough evidence to strongly support its efficacy and its underlying theory.

The idea behind evidence-based practice is to offer patients the best available treatments based on research. Nevertheless, CBT (for example) seems to be relatively rarely implemented in routine clinical practice (Haas & Clopton, 2003; Mussell et al., 2000). The lack of opportunities for adequate clinical training or barriers to the dissemination of evidence-based treatments, such as misconceptions about manualized interventions and the relevance of RCTs for practice, could explain this situation (Wilson et al., 2007). We argue that more consistent efforts need to be made to overcome these obstacles.

While offering patients the best available intervention is the first step in efficiently treating them, the next step involves acknowledging the limitations of the treatment, accompanied by attempts to personalize it in order to more accurately target the client's problems. The disappointing findings of treatment research in the case of anorexia are frequently due to the fact that many patients reject treatment, drop out prematurely, or show little improvement in terms of behavioral change in the absence of external contingencies (Wilson et al., 2007). This suggests that addressing patients' attitudes about their symptoms could be an important way of improving treatment efficacy.

To conclude, important advances have been made in the psychological treatment of eating disorders since the 1990s, and evidence-based interventions are available as the first line of treatment for bulimia nervosa and binge eating disorder. However, in order to develop more efficacious treatments for anorexia nervosa and to increase the efficacy of existing ones for other eating disorders, clinicians and researchers should devote increased attention to the understanding of the etiological factors involved in the development of these disorders and of the mechanisms of change associated with psychological interventions.

References

Agras, W. S., Brandt, H. A., Bulik, C. M., Dolan-Sewell, R., Fairburn, C. G., Halmi, K. A., … Le Grange, D. (2004). Report of the National Institutes of Health workshop on overcoming barriers to treatment research in anorexia nervosa. *International Journal of Eating Disorders*, *35*(4), 509–521. doi:10.1002/eat.10261

Agras, W. S., & Telch, C. F. (1998). The effects of caloric deprivation and negative affect on binge eating in obese binge-eating disordered women. *Behavior Therapy*, *29*(3), 491–503. doi:10.1016/S0005-7894(98)80045-2

Agras, W. S., Telch, C. F., Arnow, B., Eldredge, K., Wilfley, D. E., Raeburn, S. D., … Marnell, M. (1994). Weight loss, cognitive-behavioral, and desipramine treatments in binge eating disorder: An additive design. *Behavior Therapy*, *25*(2), 225–238. doi:10.1016/S0005-7894(05)80285-0

Agras, W. S., Walsh, B. T., Fairburn, C. G., Wilson, G. T., & Kraemer, H. C. (2000). A multicenter comparison of cognitive-behavioral therapy and interpersonal psychotherapy for bulimia nervosa. *Archives of General Psychiatry*, *57*(5), 459–466. doi:10.1001/archpsyc.57.5.459

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.

American Psychological Association Presidential Task Force on Evidence-Based Practice. (2006). Evidence-based practice in psychology. *American Psychologist*, *61*, 271–285.

Arcelus, J., Mitchell, A. J., Wales, J., & Nielsen, S. (2011). Mortality rates in patients with anorexia nervosa and other eating disorders: A meta-analysis of 36 studies. *Archives of General Psychiatry*, *68*(7), 724–731. doi:10.1001/archgenpsychiatry.2011.74

Bailer, U., de Zwaan, M., Leisch, F., Strnad, A., Lennkh-Wolfsberg, C., El-Giamal, N., … Kasper, S. (2004). Guided self-help versus cognitive-behavioral group therapy in the treatment of bulimia nervosa. *International Journal of Eating Disorders*, *35*(4), 522–537. doi:10.1002/eat.20003

Ball, J., & Mitchell, P. (2004). A randomized controlled study of cognitive behavior therapy and behavioral family therapy for anorexia nervosa patients. *Eating Disorders*, *12*(4), 303–314. doi:10.1080/10640260490521389

Banasiak, S. J., Paxton, S. J., & Hay, P. (2005). Guided self-help for bulimia nervosa in primary care: A randomized controlled trial. *Psychological Medicine*, *35*(9), 1283–1294. doi:10.1017/S0033291705004769

Blinder, B. J., Cumella, E. J., & Sanathara, V. A. (2006). Psychiatric comorbidities of female inpatients with eating disorders. *Psychosomatic Medicine*, *68*(3), 454–462. doi:10.1097/01.psy.0000221254.77675.f5

Braun, D. L., Sunday, S. R., & Halmi, K. A. (1994). Psychiatric comorbidity in patients with eating disorders. *Psychological Medicine*, *24*(4), 859–867. doi:10.1017/S0033291700028956

Carrard, I., Crépin, C., Rouget, P., Lam, T., Golay, A., & Van der Linden, M. (2011). Randomised controlled trial of a guided self-help treatment on the internet for binge eating disorder. *Behaviour Research and Therapy*, *49*(8), 482–491. doi:10.1016/j.brat.2011.05.004

Carter, J. C., & Fairburn, C. G. (1998). Cognitive–behavioral self-help for binge eating disorder: A controlled effectiveness study. *Journal of Consulting and Clinical Psychology*, *66*(4), 616–623. doi:10.1037/0022-006X.66.4.616

Channon, S., De Silva, P., Hemsley, D., & Perkins, R. (1989). A controlled trial of cognitive-behavioural and behavioural treatment of anorexia nervosa. *Behaviour Research and Therapy*, *27*(5), 529–535. doi:10.1016/0005-7967(89)90087-9

Clinton, D. (2006). Affect regulation, object relations and the central symptoms of eating disorders. *European Eating Disorders Review*, *14*(4), 203–211. doi:10.1002/erv.710

Collings, S., & King, M. (1994). Ten-year follow-up of 50 patients with bulimia nervosa. *British Journal of Psychiatry*, *164*(1), 80–87. doi:10.1192/bjp.164.1.80

Cooper, M., & Turner, H. (2000). Underlying assumptions and core beliefs in anorexia nervosa and dieting. *British Journal of Clinical Psychology*, *39*(2), 215–218. doi:10.1348/014466500163239

Couturier, J., Kimber, M., & Szatmari, P. (2013). Efficacy of family-based treatment for adolescents with eating disorders: A systematic review and meta-analysis. *International Journal of Eating Disorders*, *46*(1), 3–11. doi:10.1002/eat.22042

Crowther, J. H., Kichler, J. C., Sherwood, N. E., & Kuhnert, M. E. (2002). The role of familial factors in bulimia nervosa. *Eating Disorders*, *10*(2), 141–151. doi:10.1080/10640260290081704

Currin, L., Schmidt, U., Treasure, J., & Jick, H. (2005). Time trends in eating disorder incidence. *British Journal of Psychiatry*, *186*(2), 132–135. doi:10.1192/bjp.186.2.132

Dare, C., Eisler, I., Russell, G., Treasure, J., & Dodge, L. (2001). Psychological therapies for adults with anorexia nervosa: Randomised controlled trial of out-patient treatments. *British Journal of Psychiatry*, *178*, 216–221. doi:10.1192/bjp.178.3.216

David, D., & Montgomery, G. H. (2011). The scientific status of psychotherapies: A new evaluative framework for evidence-based psychosocial interventions. *Clinical Psychology: Science and Practice*, *18*(2), 89–99. doi:10.1111/j.1468-2850.2011.01239.x

Denman, C. (2001). Cognitive–analytic therapy. *Advances in Psychiatric Treatment*, *7*(4), 243–252. doi:10.1192/apt.7.4.243

Dickstein, L. P., Franco, K. N., Rome, E. S., & Auron, M. (2014). Recognizing, managing medical consequences of eating disorders in primary care. *Cleveland Clinic Journal of Medicine*, *81*(4), 255–263. doi:10.3949/ccjm.81a.12132

Dingemans, A. E., Spinhoven, P., & Van Furth, E. F. (2007). Predictors and mediators of treatment outcome in patients with binge eating disorder. *Behaviour Research and Therapy*, *45*(11), 2551–2562. doi:10.1016/j.brat.2007.06.003

Ellison, J. M., Simonich, H. K., Wonderlich, S. A., Crosby, R. D., Cao, L., Mitchell, J. E., … Peterson, C. B. (2016). Meal patterning in the treatment of bulimia nervosa. *Eating Behaviors*, *20*, 39–42. doi:10.1016/j.eatbeh.2015.11.008

Fairburn, C. G., Jones, R., Peveler, R. C., Carr, S. J., Solomon, R. A., O'Connor, M. E., … Hope, R. A. (1991). Three psychological treatments for bulimia nervosa: A comparative trial. *Archives of General Psychiatry*, *48*(5), 463–469. doi:10.1001/archpsyc.1991.01810290075014

Fairburn, C. G., Shafran, R., & Cooper, Z. (1999). A cognitive behavioural theory of anorexia nervosa. *Behaviour Research and Therapy*, *37*(1), 1–13. doi:10.1016/S0005-7967(98)00102-8

Fisher, C. A., Hetrick, S. E., & Rushford, N. (2010). Family therapy for anorexia nervosa. *Cochrane Database of Systematic Reviews*, *4*, CD004780. doi:10.1002/14651858.CD004780.pub2

Fonagy, P., Gergely, G., Jurist, E. L., & Target, M. (2002). *Affect regulation, mentalization, and the development of the self* (Vol. 13). New York, NY: Other Press.

Garner, D. M., Olmsted, M. P., & Polivy, J. (1983). The Eating Disorders Inventory: A measure of cognitive behavioral dimensions of anorexia and bulimia. In D. L. Darby (Ed.), *Anorexia nervosa* (pp. 21–40). New York, NY: Alan R. Liss.

Garner, D. M., Rockert, W., Davis, R., Garner, M. V., Olmsted, M. P., & Eagle, M. (1993). Comparison of cognitive-behavioral and supportive-expressive therapy for bulimia nervosa. *American Journal of Psychiatry*, *150*(1), 37–46. doi:10.1176/ajp.150.1.37

Gowers, S., & North, C. (1999). Difficulties in family functioning and adolescent anorexia nervosa. *British Journal of Psychiatry*, *174*(1), 63–66. doi:10.1192/bjp.174.1.63

Grilo, C. M., & Masheb, R. M. (2005). A randomized controlled comparison of guided self-help cognitive behavioral therapy and behavioral weight loss for binge eating disorder. *Behaviour Research and Therapy*, *43*(11), 1509–1525. doi:10.1016/j.brat.2004.11.010

Grilo, C. M., Masheb, R. M., & Crosby, R. D. (2012). Predictors and moderators of response to cognitive behavioral therapy and medication for the treatment of binge eating disorder. *Journal of Consulting and Clinical Psychology*, *80*(5), 897–906. doi:10.1037/a0027001

Haas, H. L., & Clopton, J. R. (2003). Comparing clinical and research treatments for eating disorders. *International Journal of Eating Disorders*, *33*(4), 412–420. doi:10.1002/eat.10156

Hay, P. P. J., Bacaltchuk, J., Stefano, S., & Kashyap, P. (2009). Psychological treatments for bulimia nervosa and binging. *Cochrane Database of Systematic Reviews*, *4*, CD000562. doi:10.1002/14651858.CD000562.pub3

Hilbert, A., Saelens, B. E., Stein, R. I., Mockus, D. S., Welch, R. R., Matt, G. E., & Wilfley, D. E. (2007). Pretreatment and process predictors of outcome in interpersonal and cognitive behavioral psychotherapy for binge eating disorder. *Journal of Consulting and Clinical Psychology*, *75*(4), 645–651. doi:10.1037/0022-006X.75.4.645

Hoek, H. W., & Van Hoeken, D. (2003). Review of the prevalence and incidence of eating disorders. *International Journal of Eating Disorders*, *34*(4), 383–396. doi:10.1002/eat.10222

Holtom-Viesel, A., & Allan, S. (2014). A systematic review of the literature on family functioning across all eating disorder diagnoses in comparison to control families. *Clinical Psychology Review*, *34*(1), 29–43. doi:10.1016/j.cpr.2013.10.005

Hsu, L. K. G., Rand, W., Sullivan, S., Liu, D. W., Mulliken, B., McDonagh, B., & Kaye, W. H. (2001). Cognitive therapy, nutritional therapy and their combination in the treatment of bulimia nervosa. *Psychological Medicine*, *31*(5), 871–879. doi:10.1017/S003329170100410X

Keel, P. K., & Mitchell, J. E. (1997). Outcome in bulimia nervosa. *American Journal of Psychiatry*, *54*(3), 313–321.

Laessle, R. G., Beumont, P. J., Butow, P., Lennerts, W., O'Connor, M., Pirke, K. M., … Waadt, S. (1991). A comparison of nutritional management with stress management in the treatment of bulimia nervosa. *British Journal of Psychiatry*, *159*(2), 250–261. doi:10.1192/bjp.159.2.250

Le Grange, D., Crosby, R. D., Rathouz, P. J., & Leventhal, B. L. (2007). A randomized controlled comparison of family-based treatment and supportive psychotherapy for adolescent bulimia nervosa. *Archives of General Psychiatry*, *64*(9), 1049–1056. doi:10.1001/archpsyc.64.9.1049

Lock, J., Agras, W. S., Bryson, S., & Kraemer, H. C. (2005). A comparison of short- and long-term family therapy for adolescent anorexia nervosa. *Journal of the American Academy of Child & Adolescent Psychiatry*, *44*(7), 632–639. doi:10.1097/01.chi.0000161647.82775.0a

Lock, J., Le Grange, D., Agras, W. S., & Dare, C. (2001). *Treatment manual for anorexia nervosa: A family-based approach*. New York, NY: Guilford Press.

Loeb, K. L., Wilson, G. T., Labouvie, E., Pratt, E. M., Hayaki, J., Walsh, B. T., … Fairburn, C. G. (2005). Therapeutic alliance and treatment adherence in two interventions for bulimia nervosa: A study of process and outcome. *Journal of Consulting and Clinical Psychology*, *73*(6), 1097–1106. doi:10.1037/0022-006X.73.6.1097

McIntosh, V. V., Bulik, C. M., McKenzie, J. M., Luty, S. E., & Jordan, J. (2000). Interpersonal psychotherapy for anorexia nervosa. *International Journal of Eating Disorders*, *27*(2), 125–139. doi:10.1002/(SICI)1098-108X(200003)27:2<125::AID-EAT1>3.0.CO;2-4

McIntosh, V. V. W., Jordan, J., Carter, F. A., Luty, S. E., McKenzie, J. M., Bulik, C. M., … Joyce, P. R. (2005). Three psychotherapies for anorexia nervosa: A randomized, controlled trial. *American Journal of Psychiatry*, *162*(4), 741–747. doi:10.1176/appi.ajp.162.4.741

McWilliams, N. (2004). *Psychoanalytic psychotherapy: A practitioner's guide*. New York, NY: Guilford Press.

Minuchin, S., Rosman, B. L., & Baker, L. (1978). *Psychosomatic families: Anorexia nervosa in context*. Cambridge, MA: Harvard University Press.

Munsch, S., Biedert, E., Meyer, A., Michael, T., Schlup, B., Tuch, A., & Margraf, J. (2007). A randomized comparison of cognitive behavioral therapy and behavioral weight loss treatment for overweight individuals with binge eating disorder. *International Journal of Eating Disorders*, *40*(2), 102–113. doi:10.1002/eat.20350

Murray, C., Waller, G., & Legg, C. (2000). Family dysfunction and bulimic psychopathology: The mediating role of shame. *International Journal of Eating Disorders*, *28*(1), 84–89. doi:10.1002/(SICI)1098-108X(200007)28:1<84::AID-EAT10>3.0.CO;2-R

Mussell, M. P., Crosby, R. D., Crow, S. J., Knopke, A. J., Peterson, C. B., Wonderlich, S. A., & Mitchell, J. E. (2000). Utilization of empirically supported psychotherapy treatments for individuals with eating disorders: A survey of psychologists. *International Journal of Eating Disorders*, *27*(2), 230–237. doi:10.1002/(SICI)1098-108X(200003)27:2<230::AID-EAT11>3.0.CO;2-0

National Institute for Clinical Excellence. (2004). *Eating disorders: Core interventions in the treatment and management of anorexia nervosa, bulimia nervosa and related eating disorders*. Leicester, UK: British Psychological Society/Royal College of Psychiatrists.

Nauta, H., Hospers, H., Kok, G., & Jansen, A. (2000). A comparison between a cognitive and a behavioral treatment for obese binge eaters and obese non-binge eaters. *Behavior Therapy*, *31*(3), 441–461. doi:10.1016/S0005-7894(00)80024-6

North, C., Gowers, S., & Byram, V. (1997). Family functioning and life events in the outcome of adolescent anorexia nervosa. *British Journal of Psychiatry*, *171*(6), 545–549. doi:10.1192/bjp.171.6.545

Palmer, R. L., Birchall, H., McGrain, L., & Sullivan, V. (2002). Self-help for bulimic disorders: A randomised controlled trial comparing minimal guidance with face-to-face or telephone guidance. *British Journal of Psychiatry*, *181*(3), 230–235. doi:10.1192/bjp.181.3.230

Peterson, C. B., Mitchell, J. E., Crow, S. J., Crosby, R. D., & Wonderlich, S. A. (2009). The efficacy of self-help group treatment and therapist-led group treatment for binge eating disorder. *American Journal of Psychiatry*, *166*(12), 1347–1354. doi:10.1176/appi.ajp.2009.09030345

Peterson, C. B., Mitchell, J. E., Engbloom, S., Nugent, S., Mussell, M. P., & Miller, J. P. (1998). Group cognitive-behavioral treatment of binge eating disorder: A comparison of therapist-led versus self-help formats. *International Journal of Eating Disorders*, *24*(2), 125–136. doi:10.1002/(SICI)1098-108X(199809)24:2<125::AID-EAT2>3.0.CO;2-G

Pike, K. M., Walsh, B. T., Vitousek, K., Wilson, G. T., & Bauer, J. (2003). Cognitive behavior therapy in the posthospitalization treatment of anorexia nervosa. *American Journal of Psychiatry*, *160*(11), 2046–2049. doi:10.1176/appi.ajp.160.11.2046

Poulsen, S., Lunn, S., Daniel, S. I. F., Folke, S., Mathiesen, B. B., Katznelson, H., & Fairburn, C. G. (2014). A randomized controlled trial of psychoanalytic psychotherapy or cognitive-behavioral therapy for bulimia nervosa. *American Journal of Psychiatry*, *171*(1), 109–116. doi:10.1176/appi.ajp.2013.12121511

Preti, A., Rocchi, M. B. L., Sisti, D., Camboni, M. V., & Miotto, P. (2011). A comprehensive meta-analysis of the risk of suicide in eating disorders. *Acta Psychiatrica Scandinavica*, *124*(1), 6–17. doi:10.1111/j.1600-0447.2010.01641.x

Reeves, R. S., McPherson, R. S., Nichaman, M. Z., Harrist, R. B., Foreyt, J. P., & Goodrick, G. K. (2001). Nutrient intake of obese female binge eaters. *Journal of the American Dietetic Association*, *101*(2), 209–215. doi:10.1016/S0002-8223(01)00055-4

Ricca, V., Castellini, G., Lo Sauro, C., Mannucci, E., Ravaldi, C., Rotella, F., & Faravelli, C. (2010). Cognitive-behavioral therapy for threshold and subthreshold anorexia nervosa: A

three-year follow-up study. *Psychotherapy and Psychosomatics*, *79*(4), 238–248. doi:10.1159/000315129

Robin, A. L., Siegel, P. T., & Moye, A. (1995). Family versus individual therapy for anorexia: Impact on family conflict. *International Journal of Eating Disorders*, *17*(4), 313–322. doi:10.1002/1098-108X(199505)17:4<313::AID-EAT2260170402>3.0.CO;2-8

Robin, A. L., Siegel, P. T., Moye, A. W., Gilroy, M., Dennis, A. B., & Sikand, A. (1999). A controlled comparison of family versus individual therapy for adolescents with anorexia nervosa. *Journal of the American Academy of Child & Adolescent Psychiatry*, *38*(12), 1482–1489. doi:10.1097/00004583-199912000-00008

Ryle, A. (1990). *Cognitive analytic therapy: Active participation in change*. Chichester, UK: John Wiley & Sons.

Safer, D. L., Robinson, A. H., & Jo, B. (2010). Outcome from a randomized controlled trial of group therapy for binge eating disorder: Comparing dialectical behavior therapy adapted for binge eating to an active comparison group therapy. *Behavior Therapy*, *41*(1), 106–120. doi:10.1016/j.beth.2009.01.006

Safer, D. L., Telch, C. F., & Agras, W. S. (2001). Dialectical behavior therapy for bulimia nervosa. *American Journal of Psychiatry*, *158*(4), 632–634. doi:10.1176/appi.ajp.158.4.632

Sánchez-Ortiz, V. C., Munro, C., Stahl, D., House, J., Startup, H., Treasure, J., … Schmidt, U. (2011). A randomized controlled trial of internet-based cognitive-behavioural therapy for bulimia nervosa or related disorders in a student population. *Psychological Medicine*, *41*(2), 407–417. doi:10.1017/S0033291710000711

Scalf-McIver, L., & Thompson, J. K. (1989). Family correlates of bulimic characteristics in college females. *Journal of Clinical Psychology*, *45*(3), 467–472. doi:10.1002/1097-4679(198905)45:3<467::aid-jclp2270450319>3.0.co;2-0

Schmidt, U., Lee, S., Beecham, J., Perkins, S., Treasure, J., Yi, I., … Eisler, I. (2007). A randomized controlled trial of family therapy and cognitive behavior therapy guided self-care for adolescents with bulimia nervosa and related disorders. *American Journal of Psychiatry*, *164*(4), 591–598. doi:10.1176/appi.ajp.164.4.591

Schmidt, U., Oldershaw, A., Jichi, F., Sternheim, L., Startup, H., McIntosh, V., … Treasure, J. (2012). Out-patient psychological therapies for adults with anorexia nervosa: Randomised controlled trial. *British Journal of Psychiatry*, *201*(5), 392–399. doi:10.1192/bjp.bp.112.112078

Schmidt, U., & Treasure, J. (2006). Anorexia nervosa: Valued and visible—A cognitive-interpersonal maintenance model and its implications for research and practice. *British Journal of Clinical Psychology*, *45*(3), 343–366. doi:10.1348/014466505X53902

Serfaty, M. A., Turkington, D., Heap, M., Ledsham, L., & Jolley, E. (1999). Cognitive therapy versus dietary counselling in the outpatient treatment of anorexia nervosa: Effects of the treatment phase. *European Eating Disorders Review*, *7*(5), 334–350. doi:10.1002/(SICI)1099-0968(199911)7:5<334::AID-ERV311>3.0.CO;2-H

Smith, D. E., Marcus, M. D., & Kaye, W. (1992). Cognitive-behavioral treatment of obese binge eaters. *International Journal of Eating Disorders*, *12*(3), 257–262. doi:10.1002/1098-108X(199211)12:3<257::AID-EAT2260120305>3.0.CO;2-S

Spangler, D. L., Baldwin, S. A., & Agras, W. S. (2004). An examination of the mechanisms of action in cognitive behavioral therapy for bulimia nervosa. *Behavior Therapy*, *35*(3), 537–560. doi:10.1016/S0005-7894(04)80031-5

Steinhausen, H. C. (2002). The outcome of anorexia nervosa in the 20th century. *American Journal of Psychiatry*, *159*(8), 1284–1293. doi:10.1176/appi.ajp.159.8.1284

Striegel-Moore, R. H., Leslie, D., Petrill, S. A., Garvin, V., & Rosenheck, R. A. (2000). One-year use and cost of inpatient and outpatient services among female and male patients with an eating disorder: Evidence from a national database of health insurance claims. *International Journal of Eating Disorders*, *27*(4), 381-389. doi:10.1002/(SICI)1098-108X(200005)27:4<381::AID-EAT2>3.0.CO;2-U

Sundgot-Borgen, J., Rosenvinge, J. H., Bahr, R., & Schneider, L. S. (2002). The effect of exercise, cognitive therapy, and nutritional counseling in treating bulimia nervosa. *Medicine & Science in Sports & Exercise*, *34*(2), 190–195. doi:10.1097/00005768-200202000-00002

Swanson, S. A., Crow, S. J., Le Grange, D., Swendsen, J., & Merikangas, K. R. (2011). Prevalence and correlates of eating disorders in adolescents: Results from the National Comorbidity Survey Replication adolescent supplement. *Archives of General Psychiatry*, *68*(7), 714–723. doi:10.1001/archgenpsychiatry.2011.22

Telch, C. F., & Agras, W. S. (1996). Do emotional states influence binge eating in the obese? *International Journal of Eating Disorders*, *20*(3), 271–279. doi:10.1002/(SICI)1098-108X(199611)20:3<271::AID-EAT6>3.0.CO;2-

Telch, C. F., Agras, W. S., & Linehan, M. M. (2000). Group dialectical behavior therapy for binge-eating disorder: A preliminary, uncontrolled trial. *Behavior Therapy*, *31*(3), 569–582. doi:10.1016/S0005-7894(00)80031-3

Telch, C. F., Agras, W. S., & Linehan, M. M. (2001). Dialectical behavior therapy for binge eating disorder. *Journal of Consulting and Clinical Psychology*, *69*(6), 1061–1065. doi:10.1037/0022-006X.69.6.1061

Temple, R., & Ellenberg, S. S. (2000). Placebo-controlled trials and active-control trials in the evaluation of new treatments: Part 1. Ethical and scientific issues. *Annals of Internal Medicine*, *133*(6), 455–463. doi:10.7326/0003-4819-133-6-200009190-00014

Thackwray, D. E., Smith, M. C., Bodfish, J. W., & Meyers, A. W. (1993). A comparison of behavioral and cognitive-behavioral interventions for bulimia nervosa. *Journal of Consulting and Clinical Psychology*, *61*(4), 639–645. doi:10.1037/0022-006X.61.4.639

Touyz, S., Grange, D. L., Lacey, H., Hay, P., Smith, R., Maguire, S., … Crosby, R. D. (2013). Treating severe and enduring anorexia nervosa: A randomized controlled trial. *Psychological Medicine*, *43*(12), 2501–2511. doi:10.1017/S0033291713000949

Treasure, J., Todd, G., Brolly, M., Tiller, J., Nehmed, A., & Denman, F. (1995). A pilot study of a randomised trial of cognitive analytical therapy vs educational behavioral therapy for adult anorexia nervosa. *Behaviour Research and Therapy*, *33*(4), 363–367. doi:10.1016/0005-7967(94)00070-Z

Turner, H., & Cooper, M. (2002). Cognitions and their origins in women with anorexia nervosa, normal dieters and female controls. *Clinical Psychology & Psychotherapy*, *9*(4), 242–252. doi:10.1002/cpp.317

Vocks, S., Tuschen-Caffier, B., Pietrowsky, R., Rustenbach, S. J., Kersting, A., & Herpertz, S. (2010). Meta-analysis of the effectiveness of psychological and pharmacological treatments for binge eating disorder. *International Journal of Eating Disorders*, *43*(3), 205–217. doi:10.1002/eat.20696

Wallace, L. M., Masson, P. C., Safer, D. L., & von Ranson, K. M. (2014). Change in emotion regulation during the course of treatment predicts binge abstinence in guided self-help dialectical behavior therapy for binge eating disorder. *Journal of Eating Disorders*, *2*(1), 2–35. doi:10.1186/s40337-014-0035-x

Walsh, B. T., Wilson, G. T., Loeb, K. L., Devlin, M. J., Pike, K. M., Roose, S. P., … Waternaux, C. (1997). Medication and psychotherapy in the treatment of bulimia nervosa. *American Journal of Psychiatry*, *154*(4), 523–531. doi:10.1176/ajp.154.4.523

Wilfley, D. E., Stewart, W., Telch, C. F., Rossiter, E. M., Schneider, J. A., Cole, A. G., … Raeburn, S. D. (1993). Group cognitive-behavioral therapy and group interpersonal psychotherapy for the nonpurging bulimic individual: A controlled comparison. *Journal of Consulting and Clinical Psychology*, *61*(2), 296–305. doi:10.1037/0022-006X.61.2.296

Wilfley, D. E., Welch, R. R., Stein, R. I., Spurrell, E. B., Cohen, L. R., Saelens, B. E., … Matt, G. E. (2002). A randomized comparison of group cognitive-behavioral therapy and group interpersonal psychotherapy for the treatment of overweight individuals with binge-eating disorder. *Archives of General Psychiatry*, *59*(8), 713–721. doi:10.1001/archpsyc.59.8.713

Wilson, G., Wilfley, D. E., Agras, W., & Bryson, S. W. (2010). Psychological treatments of binge eating disorder. *Archives of General Psychiatry*, *67*(1), 94–101. doi:10.1001/archgenpsychiatry.2009.170

Wilson, G. T., & Fairburn, C. G. (1993). Cognitive treatments for eating disorders. *Journal of Consulting and Clinical Psychology*, *61*, 261–269. doi:10.1037/0022-006X.61.2.261

Wilson, G. T., Fairburn, C. C., Agras, W. S., Walsh, B. T., & Kraemer, H. (2002). Cognitive-behavioral therapy for bulimia nervosa: Time course and mechanisms of change. *Journal of Consulting and Clinical Psychology*, *70*(2), 267–274. doi:10.1037/0022-006X.70.2.267

Wilson, G. T., Grilo, C. M., & Vitousek, K. M. (2007). Psychological treatment of eating disorders. *American Psychologist*, *62*(3), 199–216. doi:10.1037/0003-066X.62.3.199

Wiser, S., & Telch, C. F. (1999). Dialectical behavior therapy for binge-eating disorder. *Journal of Clinical Psychology*, *55*(6), 755–768. doi:10.1002/(SICI)1097-4679(199906)55:6<755::AID-JCLP8>3.0.CO;2-R

Wolff, G. E., & Clark, M. M. (2001). Changes in eating self-efficacy and body image following cognitive–behavioral group therapy for binge eating disorder: A clinical study. *Eating Behaviors*, *2*(2), 97–104. doi:10.1016/S1471-0153(01)00021-6

Zendegui, E. A., West, J. A., & Zandberg, L. J. (2014). Binge eating frequency and regular eating adherence: The role of eating pattern in cognitive behavioral guided self-help. *Eating Behaviors*, *15*(2), 241–243. doi:10.1016/j.eatbeh.2014.03.002

Zipfel, S., Wild, B., Groß, G., Friederich, H.-C., Teufel, M., Schellberg, D., … ANTOP Study Group. (2014). Focal psychodynamic therapy, cognitive behaviour therapy, and optimised treatment as usual in outpatients with anorexia nervosa (ANTOP study): Randomised controlled trial. *Lancet*, *383*(9912), 127–137. doi:10.1016/S0140-6736(13)61746-8

9

Evidence-Based Treatment for Alcohol Use Disorders

A Review Through the Lens of the Theory × Efficacy Matrix

James MacKillop,[1] Monika Stojek,[2,3] Lauren VanderBroek-Stice,[2] and Max M. Owens[2]

[1] *Department of Psychiatry and Behavioural Neurosciences, DeGroote School of Medicine, McMaster University, Hamilton, Canada; Peter Boris Centre for Addictions Research, St. Joseph's Healthcare Hamilton, Hamilton, Canada*

[2] *Department of Psychology, University of Georgia, Athens, USA*

[3] *Medical College of Georgia, Georgia Regents University, Augusta, USA*

Alcohol use disorders (AUDs) are among the most common psychiatric conditions and are a major cause of distress and harm to those who are afflicted and their loved ones. Treatment for AUDs is commonly available in the United States and the developed world but comprises an exceptionally broad and diverse array of programs, approaches, and techniques. Within this clinical patchwork are approaches of all conceivable provenances: treatments that are direct extensions of basic behavioral science, treatments that grew out of clinical intuition, and treatments that come entirely from the lay community of patients and their families. This is, in part, because of the unique history of AUD treatment, which, unlike other mental health treatments, first took shape outside traditional health care settings and is often still apart from the larger medical enterprise. Historically, the formal scientific study of AUDs and other addictive disorders and, more importantly, the application of science to clinical applications emerged relatively recently. As a result, a clinical science perspective represents a recent and minority viewpoint.

In this context, the broad goal of this chapter is to separate the "wheat" from the "chaff" in characterizing evidence-based treatment for AUDs. To do so, we review a number of psychosocial treatments for AUDs using the theory × efficacy (T×E) matrix proposed by David and Montgomery (2011) as a prism through which to evaluate these approaches. (The matrix is described in detail in Chapter 1, but, in brief, it characterizes the empirical evidence supporting both theory and treatment efficacy according to three levels of strength: well-supported, equivocal, and strong contradictory evidence.) After a concise critique of the theory and evidence behind each approach, the treatments are classified as being in one of the nine matrix categories reflecting the intersection of the two domains. In general, we view this framework as being salutary to the field and offering a more comprehensive perspective on scientifically grounded psychosocial treatments for psychiatric disorders.

To start, the chapter provides an overview of the scientific history, current diagnostic nomenclature, and epidemiology of AUDs. We then review and classify the treatments

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

according to the T×E matrix. Finally, we specifically comment on the implications of the review for future research and clinical practice. Our hope is that, in applying this perspective, we can provide a systematic perspective on evidence-based treatment for AUDs.

9.1 A Brief History of Alcohol Use Disorder Treatment

Although a comprehensive account is beyond the scope of this chapter, AUDs have historically been considered a moral failing, not clinical disorders that fall within the purview of professional treatment (White, 2014). The history of contemporary treatment maps onto changes in the perception of the problem and can be broadly divided into three epochs: a prehistory reflecting a gradual increase in the perception of AUD as disorders, not simply character flaws; the rise of a lay solution, Alcoholics Anonymous; and the development of a formal scientifically grounded clinical enterprise.

The prehistory largely begins in the 18th and 19th centuries with initial insights from Benjamin Rush (Katcher, 1993), one of the progenitors of psychiatry and an early proponent of treating alcohol misuse medically. Indeed, the term "alcoholic" was first coined by a Swedish physician, Magnus Huss, in 1849 to clinically describe an individual experiencing severe consequences of excessive alcohol use (Miller & Hester, 2003). However, formal alcohol treatment was largely not available in the United States during this time period, with the exception of a small number of alcohol-specific facilities. The New York State Inebriate Asylum was the first dedicated hospital for treating alcohol problems and opened in 1864 in Binghamton. However, these inpatient hospitals were the exceptions, rather than the rule, and the treatment strategies employed were largely nonscientific. Nonetheless, they represent a first major milestone in the history of AUD treatment, reflecting the beginning of medical and psychological treatment as opposed to social opprobrium.

The second milestone is probably the best known, the founding of the mutual support group Alcoholics Anonymous (AA) by William Wilson (Bill W.) and Robert Smith (Dr. Bob) in 1935. The premises of AA are widely known, that individuals are powerless to overcome alcohol and should turn to a higher power and their fellow sufferers for salvation, specifically through a 12-step program that provides guidance for sober living. More than 75 years on, AA continues to have literally millions of members and a vast network of groups throughout the world. It remains the most widely known form of treatment for AUDs to the public (Blum, Roman, & Bennett, 1989; Caetano, 1987) and formal treatment programs are often oriented around its teachings, the so-called Minnesota model.

The most recent milestone in the United States is the 1970 passage of the Comprehensive Alcohol Abuse and Alcoholism Prevention, Treatment, and Rehabilitation Act (Pub. L. 91–616), "to provide a comprehensive Federal program for the prevention and treatment of alcohol abuse and alcoholism" (p. 1). This, in turn, gave rise to the National Institute on Alcohol Abuse and Alcoholism, the major funder of basic and clinical alcohol research in the United States. Furthermore, the act included AUDs within the remit of health care more broadly, fundamentally changing the perception of AUDs in the eyes of the law. The contemporary evidence-based treatment enterprise can largely be traced to empirical research and perspectives that emerged following the passage of this act.

9.1.1 Psychiatric Nosology of Alcohol Use Disorders

The diagnosis of an AUD has largely mapped onto these historical trends. The first and second versions of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1952, 1968) categorized alcoholism and other addictions as personality disorders, reflecting the putatively characterological nature of the disorder. In the DSM-III (American Psychiatric Association, 1980, 1987), alcohol and other forms of drug addiction became an independent category, substance use disorders (SUDs). In addition, the DSM-III denoted two AUD diagnoses: alcohol abuse and alcohol dependence. This change was intended to reflect the fact that lower and higher severity syndromes were observed in clinical practice. As a result, the term alcoholism, which was by then both colloquial and generally pejorative, was also dropped. Two AUD diagnoses were used also in the DSM-IV (American Psychiatric Association, 1994) and DSM-IV-TR (American Psychiatric Association, 2000), which was the prevailing diagnostic system until 2013. Alcohol abuse was historically considered a prodromal phase of dependence, as it was purported to represent a less severe variant of an AUD (Hasin et al., 2003). Thus, it could only be diagnosed in the absence of dependence. The other major classification system, the International Classification of Diseases (ICD-9 and ICD-10), has a similar set of criteria for alcohol dependence (World Health Organization [WHO], 1992). Interestingly, however, the ICD does not include the legal and social problems in the criteria for diagnosing the other AUD, and instead labels it harmful alcohol use (Hasin, Stinson, Ogburn, & Grant, 2007; WHO, 1992). The argument for not including these two requirements in the diagnosis of the second syndrome is that legal and social problems may have different meanings in different cultures and may change over time in a society (Hasin et al., 2007; Schuckit, 2009).

One of the major goals of the most recent version of the manual, the DSM-5 (American Psychiatric Association, 2013), was improving the empirical basis for psychiatric diagnosis. While the reliability and validity of the alcohol dependence syndrome have been fairly well established, there has been controversy over the label of alcohol abuse (Schuckit et al., 2008). The problematic issues related to the abuse diagnosis include the lack of a clear conceptual framework, the difficulty of defining a syndrome when recurrent problems are only required in one life area, and low reliability (Hasin et al., 2003; Schuckit et al., 2005, 2008). Thus, in the DSM-5, alcohol dependence and alcohol abuse have been combined into a single diagnosis, and an individual has to exhibit criterion A plus 2 or more of the 11 criteria in section B (Table 9.1). The DSM-5 also specifies three broad categories for level of severity: mild (2–3 symptoms), moderate (4–5 symptoms), and severe (6+ symptoms). The legal problems criterion was removed from the AUD diagnosis, as the prevalence of this symptom was low in epidemiological studies (Saha, Chou, & Grant, 2006) and factor analytic studies indicated that it did not load onto the AUD diagnosis (Kahler & Strong, 2006). Finally, a criterion involving craving was added to the diagnosis. Individuals with AUDs often report strong and persistent desire to use alcohol (e.g., Bohn, Krahn, & Staehler, 1995; Oslin, Cary, Slaymaker, Colleran, & Blow, 2009). Craving has also been associated with in vivo alcohol consumption in the laboratory (MacKillop & Lisman, 2005; MacKillop, Menges, McGeary, & Lisman, 2007; O'Malley, Krishnan-Sarin, Farren, Sinha, & Kreek, 2002) and loads on a unidimensional AUD diagnosis with the other symptoms (Murphy, Stojek, Few, Rothbaum, & MacKillop, 2014).

Table 9.1 DSM-5 alcohol use disorder syndrome. For an individual to receive a diagnosis of an alcohol use disorder, they must meet criteria (A) and have two or more symptoms in (B).

Symptom
A. **A problematic pattern of alcohol use leading to clinically significant impairment or distress.**
B. **Two (or more) of the following occurring within a 12-month period:**
1. Alcohol is often taken in larger amounts or over a longer period than was intended.
2. A persistent desire or unsuccessful effort to cut down or control alcohol use.
3. A great deal of time is spent in activities necessary to obtain alcohol, use alcohol, or recover from its effects.
4. Recurrent alcohol use resulting in a failure to fulfill major role obligations at work, school, or home (e.g., repeated absences or poor work performance related to alcohol use; alcohol-related absences, suspensions, or expulsions from school; neglect of children or household).
5. Continued alcohol use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of alcohol.
6. Important social, occupational, or recreational activities are given up or reduced because of alcohol use.
7. Recurrent alcohol use in situations in which it is physically hazardous (e.g., driving an automobile or operating a machine when impaired by alcohol use).
8. Alcohol use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by alcohol.
9. Tolerance, as defined by either or both of the following:
a. a need for markedly increased amounts of alcohol to achieve intoxication or a desired effect;
b. markedly diminished effect with continued use of the same amount of alcohol.
10. Withdrawal, as manifested by either of the following:
a. the characteristic withdrawal syndrome for alcohol;
b. the same (or a closely related) substance is taken to relieve or avoid withdrawal symptoms.
11. Craving or a strong desire or urge to use alcohol.

9.1.2 Epidemiology of Alcohol Use Disorders

With regard to the scope of AUDs in contemporary society, several large epidemiological studies are highly informative. One of the largest and most systematic was the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC; Grant et al., 2004). In a nationally representative sample of 43,093 respondents, NESARC examined AUD prevalence and the comorbidity of DSM-IV SUDs with nine independent mood and anxiety disorders. Face-to-face diagnostic interviews were conducted on all participants. The National Comorbidity Survey–Replication (NCS–R) was another large epidemiological survey that used the DSM-IV criteria to determine the prevalence and correlates of mental disorders in the United States (Kessler et al., 2005). Finally, the National Survey on Drug Use and Health (NSDUH) collected data on AUD symptoms in a recent iteration (Esser et al., 2014). Prevalence rates from these studies are presented in Figure 9.1.

According to NESARC, the prevalence of lifetime AUDs was just under 20% and the 12-month prevalence was 8.5% (Hasin et al., 2007). In terms of associated characteristics, AUDs were significantly more prevalent among men, individuals of white race, Native Americans, younger and unmarried adults, and respondents with lower

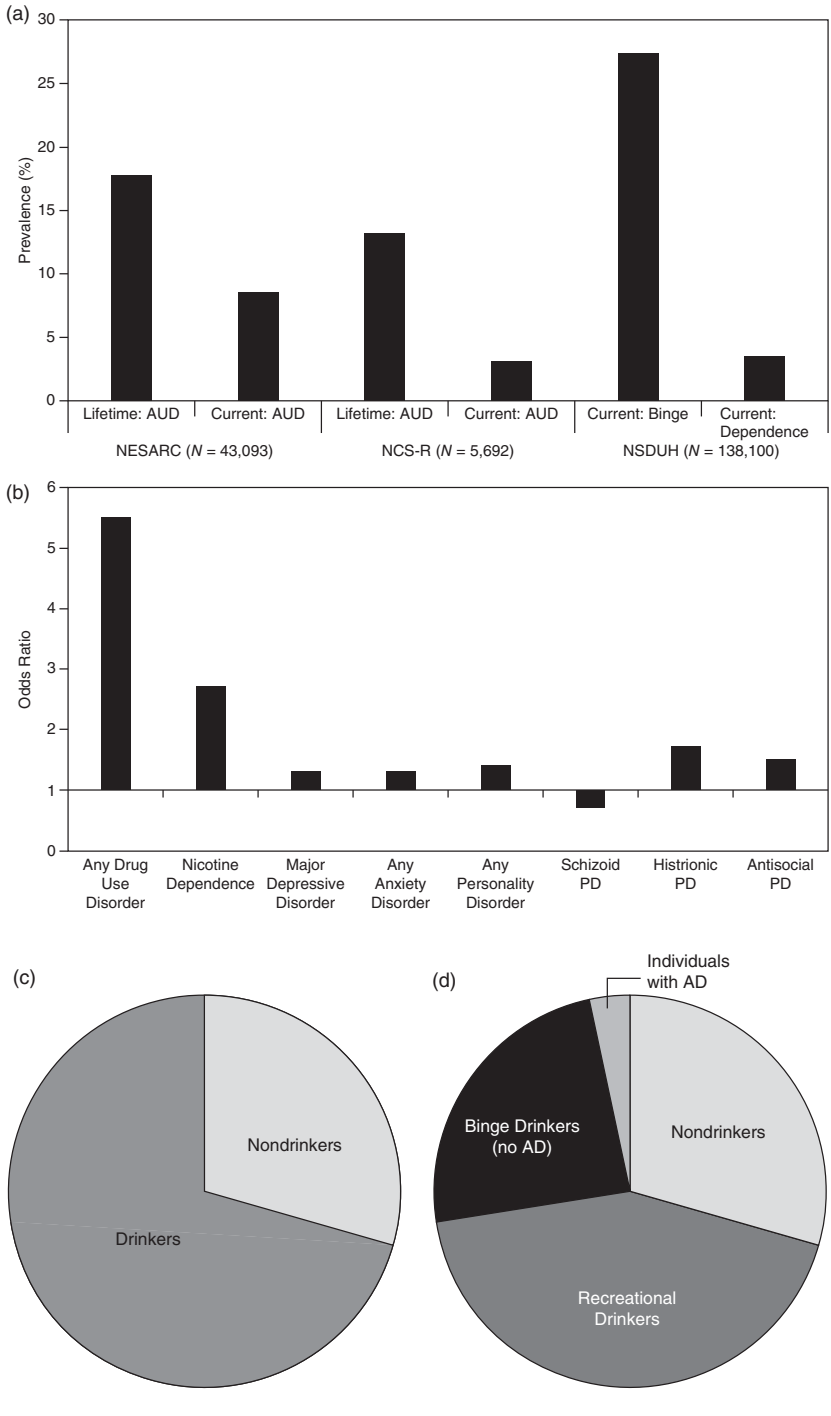

Figure 9.1 Epidemiology of alcohol use disorders (AUDs). Panel (a) depicts the prevalence of AUD, alcohol dependence, or binge drinking in three large epidemiological cohorts. Panel (b) depicts the significant odds-ratios for other comorbid conditions with AUDs from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) survey. Panel (c) depicts the overall proportion of drinkers in the general population from the National Survey on Drug Use and Health (NSDUH) survey. Panel (d) depicts the prevalence of different classes of drinkers within the larger population from the NDSUH survey. AD = alcohol dependence; NCS–R = National Comorbidity Survey–Replication; PD = personality disorder.

income (Hasin et al., 2007). Although somewhat higher, these estimates are comparable to those from the NCS–R (Kessler et al., 2005). The NSDUH data only address current prevalence of alcohol dependence but are similar to the alcohol-dependence-specific NESARC estimate of 3.8%.

An important consideration is that AUDs are also associated with substantial comorbidity with other psychological disorders. In the NESARC data, AUDs were significantly positively associated with other drug use disorders, nicotine dependence, major depressive disorder, anxiety disorders, and antisocial and histrionic personality disorder (Hasin et al., 2007). Interestingly, AUDs were significantly negatively associated with schizoid personality disorder. These relationships included the covariation of sociodemographic characteristics and other psychiatric disorders, for maximum precision. The specific odds-ratios are presented in Figure 9.1 and reveal that the most substantial relationships are with other drug use disorders and nicotine dependence. Individuals with an AUD are approximately five and a half times more likely to have a DSM-IV drug use disorder (abuse or dependence) and almost three times more likely to be nicotine dependent.

Finally, it is worthwhile to consider the group of individuals who are appropriate for some level of psychosocial intervention as extending beyond those who meet criteria for a clinical diagnosis. For example, the NSDUH survey also assessed binge drinking (5+/4+ drinks in an episode for males/females, respectively), which is substantially associated with increased risk for negative consequences from drinking and adverse health consequences (Courtney & Polich, 2009). This revealed that, of the 70.5% of the population that reported drinking in the past year, more than a quarter of respondents reported binge drinking in the past month (Esser et al., 2014). This indicates that a substantial proportion of the population drinks at unhealthy levels, even without necessarily having an AUD per se (Figure 9.1). Indeed, only about 10% of the binge drinkers identified in the NSDUH survey met criteria for alcohol dependence.

9.2 Treatment Reviews

In the following sections, we review a wide variety of treatments that are used to address AUDs. In each case, we follow the same general format for consistency. This comprises an introduction to the treatment, a brief review of its components, a discussion of the empirical support for its theoretical underpinnings, a discussion of the empirical support for its efficacy, and a disposition of the approach in one of the nine categories in the T×E matrix (see Table 9.2).

9.2.1 Reinforcement-Based Treatments

Treatments with foundations in operant learning theory (Skinner, 1938) are among the oldest research-based approaches (Higgins, Heil, & Lussier, 2004). These treatments emphasize reinforcement learning as the prepotent determinant of alcohol and drug use and misuse, and are clinical extensions of this perspective. Specifically, a reinforcement-based approach takes the position that alcohol and other drugs are powerful positive and negative reinforcers and, for individuals with SUDs, drug-reinforcing value remains persistently high and displaces alternative reinforcers in spite of the accumulating negative consequences. Put another way, from this perspective, individuals with AUDs exhibit persistently high sensitivity to the reinforcing value of alcohol and low sensitivity to

Table 9.2 Classification of alcohol use disorder treatments within David and Montgomery's (2011) theory × evidence matrix. (a) Treatment and category classification. (b) Matrix.

(a)

Treatment	Category
Community reinforcement approach	I
Contingency management	I
Motivational interviewing	I
Cognitive–behavioral therapy	I
Behavioral couples therapy	I
Alcoholics Anonymous	II
Confrontational interventions	IX

(b)

Therapeutic Package	Theory		
	Well Supported	Equivocal: No, Preliminary, or Mixed Data	Strong Contradictory Evidence
Well Supported	Category I	Category II	Category V
Equivocal: No, Preliminary, or Mixed Data	Category III	Category IV	Category VII
Strong Contradictory Evidence	Category VI	Category VIII	Category IX

its punishing effects. Two treatments that address AUDs from this perspective are the community reinforcement approach (CRA; Hunt & Azrin, 1973), a comprehensive psychosocial intervention that works with the patient to develop alternative competing sources of reinforcement that are mutually exclusive with drinking, and contingency management (CM; Stitzer & Petry, 2006), a more microcosmic strategy that directly reinforces abstinence from drug use and protreatment behaviors using vouchers and other incentives.

The empirical support for the theory behind the two approaches is fundamentally the same and there is an extensive basic behavioral science literature that supports a reinforcement-based perspective. Early residential studies provided evidence that alcohol consumption could be examined using operant procedures (Mendelson & Mello, 1966; Nathan, Titler, Lowenstein, Solomon, & Rossi, 1970) and subsequently demonstrated that modifications to the reinforcement contingencies, such as increasing response cost or providing access to alternative reinforcers, suppressed drinking behavior as predicted (for a review, see Bigelow, 2001). Parallel findings were also detected for other drugs (e.g., Bickel, Hughes, DeGrandpre, Higgins, & Rizzuto, 1992; Higgins, Bickel, & Hughes, 1994), supporting the generality of an operant perspective. More recently, studies using "purchase tasks" to assess relative reinforcing value have similarly affirmed the sensitivity of alcohol to increases in response cost and found indices of reinforcing value are significantly associated with level of alcohol misuse and

treatment response (Gray & MacKillop, 2014; Murphy, MacKillop, Skidmore, & Pederson, 2009; Murphy & MacKillop, 2006)). Again, parallel findings have been present for other drugs, supporting the generality of the approach (Collins, Vincent, Yu, Liu, & Epstein, 2014; MacKillop et al., 2008). Finally, although not from the traditional behavioral perspective, studies on motives for drinking and alcohol expectancies suggest that alcohol use is largely driven by its positively and negatively reinforcing effects (Darkes, Greenbaum, & Goldman, 2004; Kuntsche, Knibbe, Gmel, & Engels, 2006). Taken together, there is extensive empirical support for an operant learning perspective underlying reinforcement-based treatments for AUDs.

In terms of efficacy, there is also considerable empirical support for both the CRA and CM. In the first case, the CRA recognizes that, for many individuals, the development of an AUD has a recursively negative effect on the reinforcing contingencies in a person's life. In other words, the more the person drinks, and the more problems that accumulate, the fewer the reasons they have to not drink. As relationships fray, jobs are lost, and bills mount, drinking becomes one of the few remaining sources of reinforcement. To address this, the CRA attempts to break the vicious cycle and restore nondrinking sources of reinforcement, including establishing vocational, social, and familial reinforcers in a person's life. The earliest trials of the CRA reported very positive outcomes in contrast to what was then standard inpatient care (Azrin, 1976; Azrin, Sisson, Meyers, & Godley, 1982; Hunt & Azrin, 1973). Subsequently, larger trials provided further evidence of efficacy, including in challenging patient groups, such as adolescents and homeless individuals (Godley, Godley, Dennis, Funk, & Passetti, 2002; Meyers & Miller, 2001; Smith, Meyers, & Delaney, 1998). In addition, the CRA has been found to be effective in treating other SUDs beyond alcohol (Abbott, Weller, Delaney, & Moore, 1998; De Jong, Roozen, Van Rossum, Krabbe, & Kerkhof, 2007; Dennis et al., 2004; Roozen, Kerkhof, & Van den Brink, 2003). Finally, the CRA has also been adapted into a program to provide training from this perspective for families of individuals with AUDs, termed the Community Reinforcement Approach Family Training (CRAFT). Like the original program, CRAFT has been evaluated by a number of trials that support its efficacy for alcohol (Dutcher et al., 2009; Miller, Meyers, & Hiller-Sturmhofel, 1999; Sisson & Azrin, 1986) and other drugs (Kirby, Marlowe, Festinger, Garvey, & La Monaca, 1999; Meyers, Miller, Smith, & Tonigan, 2002; Waldron, Kern-Jones, Turner, Peterson, & Ozechowski, 2007). Considered as a corpus, the CRA has a very strong empirical basis for its positive treatment effects. In turn, given its strong theoretical support also, we would classify it as Category I in terms of its matrix position, reflecting a well-supported theory and well-supported therapeutic package.

With regard to CM, which creates explicit relationships between protreatment behaviors (e.g., attendance, evidence of abstinence) and vouchers, there is a similarly strong empirical literature supporting its efficacy. An early clinical trial found that standard treatment plus CM generated large-magnitude positive effects compared to standard treatment alone (Petry, Martin, Cooney, & Kranzler, 2000). A challenge in applying CM to AUDs is that the most common strategy for verifying abstinence is breath alcohol, which is a state measure with a narrow time window. To address this, recent pilot studies have demonstrated the feasibility of novel strategies for confirming abstinence from drinking, including the urinary biomarker ethyl glucuronide (EtG) and transdermal alcohol monitoring (Barnett, Tidey, Murphy, Swift, & Colby, 2011; McDonell et al., 2012). In the case of EtG, a subsequent study implementing its use in a CM trial found

positive consequences (Dougherty et al., 2014). In addition, a recent trial employed remote alcohol breathalyzing using cellular phones and found positive effects of CM on drinking (Alessi & Petry, 2013). CM has also been applied to a number of other addictive drugs, including cocaine, opioids, and tobacco (Higgins, Wong, Badger, Ogden, & Dantona, 2000; Ledgerwood, Arfken, Petry, & Alessi, 2014; McCaul, Stitzer, Bigelow, & Liebson, 1984), and a meta-analysis of 47 trials supports its effectiveness (Prendergast, Podus, Finney, Greenwell, & Roll, 2006). The application of CM to an array of other health behaviors also speaks to the approach's efficacy more broadly. For example, it has been successfully used to address obesity, HIV risk, and cardiovascular medication compliance (Ghitza, Epstein, & Preston, 2008; John et al., 2011; Volpp, John et al., 2008; Volpp, Loewenstein et al., 2008). Considered together, these findings are highly supportive of the efficacy of CM and, in combination with the strong theoretical basis, we would also classify it as belonging in Category I.

9.2.2 Motivational Interviewing

Many individuals in AUD treatment are ambivalent about changing their drinking. Recognizing this as an inherent aspect of behavior change, motivational interviewing (MI) seeks to help clients resolve conflicting motives toward commitment to change (Miller, 1983; Miller & Rollnick, 2012). As a treatment approach, it is based both on a recognition of the importance of empathy in treatment and intuitions about the social dynamics between a clinician and an ambivalent patient. Two distinct theoretical perspectives effectively coexist within an MI framework. First, MI is influenced by the humanistic client-centered approach and as such it incorporates a supportive and empathic therapeutic style (Rogers, 1959). Second, MI leverages the principles of social psychological theories, in particular, Bem's (1972) self-perception theory and Festinger's (1957) cognitive dissonance theory. These perspective propose that people tend to become more committed to that which they hear themselves defend, and that people strive toward resolving a discrepancy between their beliefs and their actions, respectively. As such, MI utilizes a subtle but nonetheless directive method of resolving ambivalence toward change by exploring clients' own reasons for change, amplifying these reasons, and developing a discrepancy between clients' goals and their current behavior (Miller & Rollnick, 2012; Miller, Zweben, DiClemente, & Rychtarik, 1992). The language of reasons for change is referred to as "change talk" and the therapist's role is to elicit change talk and respond to it with reflective listening and affirmation. In addition, the therapist offers periodic summaries of change talk to consolidate the information that the client has already volunteered.

The core principles of MI comprise empathizing with the client's perspective, developing discrepancies between alcohol use and outcomes the client desires, supporting self-efficacy, and rolling with resistance (Miller & Rollnick, 2012). The overall goal is to resolve clients' ambivalence and strengthen the commitment to change by activating the resources that clients are assumed to already possess (e.g., willingness, hope) in a nonjudgmental and supportive environment (Miller & Rollnick, 2012). Motivational Enhancement Therapy (MET; Miller et al., 1992) is one manualized adaptation of MI that has been widely used. This variation of MI incorporates structured assessment and objective feedback within the MI framework. It was developed from the Drinker's Check-Up (Miller, Sovereign, & Krege, 1988) for Project MATCH, a multisite clinical

trial that tested the effectiveness of different approaches for AUD treatment outcomes (Project MATCH Research Group, 1998a). As MET is an extended version of MI but uses all the MI principles, it will be reviewed here along with MI, as is customary in meta-analyses and literature reviews on the subject.

Although MI integrates the principles of a Rogerian humanistic approach (Rogers, 1959) with principles of cognitive dissonance (Festinger, 1957) and self-perception theories (Bem, 1972), it is not a direct literal extension of these theories. Therefore, MI is probably best understood by evaluating the mechanisms of action in treatment instead of evaluating the empirical evidence supporting the theories themselves. Fortunately, the ingredients of success in MI have been of interest from this treatment's inception and have received a moderate amount of direct study. While there is consistent general support for the efficacy of MI, as will be discussed, it has been associated with considerable variability in effects across studies and therapists (Ball et al., 2007; Hettema, Steele, & Miller, 2005). Thus, studies examining the active components of MI have attempted to disentangle these variable effects by understanding the active ingredients. In addition, there is some research on the moderators of these effects, shedding further light on mechanisms of action.

It appears that elements adopted from both orientations, humanistic and cognitive dissonance/self-perception theory, are indeed instrumental to effecting behavior change. Therapists' empathy and interpersonal skills have been found to be positively related to MI outcomes in several studies (Gaume, Gmel, & Daeppen, 2008; Moyers, Martin, Houck, Christopher, & Tonigan, 2009; Moyers, Miller, & Hendrickson, 2005). Project MATCH, which was the first multisite clinical trial of the efficacy of MI, found that MI was particularly effective for clients with high levels of anger compared to the other two treatments tested (Project MATCH Research Group, 1998b). A recent meta-analysis of 19 studies examined a mediational model of MI intervention that effects change through therapists' behaviors and clients' subsequent responses (Apodaca & Longabaugh, 2009). It indicated that therapists' MI-consistent behaviors, such as affirming and reflecting, increased change talk and decreased resistance on the part of clients (Apodaca & Longabaugh, 2009). Increased change talk and greater experience of discrepancy were in turn related to improved outcomes in treatment (Apodaca & Longabaugh, 2009; Gaume et al., 2008; Moyers et al., 2009). This meta-analysis lends considerable support to a mechanistic relationship between therapists' in-session behaviors specific to MI, clients' response to these behaviors, and treatment outcomes. It also complements the early research indicating that greater resistance was associated with poorer outcomes (Miller, Benefield, & Tonigan, 1993). Based on this evidence, MI's theoretical foundations may be classified as well supported.

In terms of empirical support for the therapeutic package, there are numerous studies supporting the efficacy of MI for AUDs (for a review, see Miller & Rose, 2009). The most extensive randomized controlled trial (RCT) for AUDs that used MI-based treatment was Project MATCH and it examined the efficacy of the four-session MET intervention delivered over 12 weeks, cognitive–behavioral therapy, and 12-step facilitation (Project MATCH Research Group, 1997a, 1997b, 1998a, 1998b). In this case, MET was found to be as effective as the other two treatments in reducing drinking, as measured by drinks per drinking day and percentage of abstinent days (Project MATCH Research Group, 1997a). These findings were corroborated by a similar endeavor in the United Kingdom

that examined over 700 individuals seeking treatment for an AUD (UKATT Research Team, 2005). In this trial, three sessions of MI over 12 weeks were as effective as nine sessions of social behavior and network therapy over the same period of time (UKATT Research Team, 2005). Meta-analyses have also consistently found significant effects of MI in improving drinking outcomes as well as treatment adherence, engagement, and retention (Hettema et al., 2005; Vasilaki, Hosier, & Cox, 2006). These studies concluded that MI is effective as a stand-alone brief treatment and as an add-on at the beginning of another treatment. Additionally, MI has been found to take, on average, 100 minutes less than other treatments to effect change, and the effects of MI appear to be relatively long-lasting (Lundahl, Kunz, Brownell, Tollefson, & Burke, 2010).

Thus, given both support for the theory underlying MI via elucidation of its mechanistic processes and support for the efficacy of the therapeutic package, we classify it as a Category I treatment.

9.2.3 Cognitive–Behavioral Therapy

Cognitive–behavioral therapy (CBT) for AUDs emerged in the 1980s and can be understood as an extension of the broader general cognitive–behavioral movement in psychotherapy and from Bandura's social learning theory (Bandura & McClelland, 1977). Rather than stable personality traits, this perspective emphasizes the role of an individual's learning history and environment in determining behavior. Alcohol use, healthy or unhealthy, is putatively maintained by environmental cues, reinforcement contingencies, expectancies about the effects of alcohol, and an individual's coping capacity. By extension, CBT for AUDs is rooted in the notion that individuals with AUDs lack the necessary coping skills to deal with certain aversive experiences (e.g., stress, sadness, cravings, withdrawal symptoms) and other situations that invite alcohol use (e.g., socializing in general, being around others drinking). Individuals with AUDs are proposed to use alcohol as the dominant coping strategy for managing these antecedents, and the goal of CBT is to develop or enhance alternative coping skills so that the individual is no longer reliant on alcohol. The two major variants that exist in the field are coping skills training (Monti & O'Leary, 1999) and relapse prevention (Witkiewitz & Marlatt, 2004).

Understanding the antecedents of drinking and addressing the coping deficits associated with them is the primary focus of CBT, and its primary components reflect these goals. Treatment typically begins with a functional analysis of the individual's drinking patterns in order to identify the internal and external precipitants, such as people, places, events, and internal experiential states. Coping-focused CBT then proceeds with idiographic coping skills training that is specific to the patient's high-risk situations (typical antecedents of drinking). For example, social skills training focuses on improving patients' interpersonal skills in nondrinking situations. This, in turn, is intended to enhance social relationships, reduce conflict, increase support, and create other positive lifestyle changes (Monti & Rohsenow, 1999). If craving in response to environmental triggers is relevant, coping-focused CBT can use in vivo exposure to alcohol-related cues for eliciting cravings in session as a way to teach craving management skills. Relapse prevention has a greater emphasis on cognitive influences, such as the importance of alcohol expectancies in maintaining drinking and the "abstinence violation effect," a term

used to describe the guilt and perceived loss of control commonly reported after an initial lapse to drinking.

The evidence in support of the theoretical model of CBT is substantial, although not unequivocal. Behavioral research has consistently linked the putative targets of CBT to AUDs. For example, environmental triggers have consistently been associated with dynamic increases in craving (Carter & Tiffany, 1999), and individuals with AUDs exhibit comparatively poorer coping skills (Monti, 2002). More persuasive still, there is increasing evidence that coping skills are the mechanism of positive treatment effects. In a clinical study focusing on drinking behavior after an initial lapse, severity of AUD prior to treatment and improved coping behaviors following treatment were the strongest predictors of treatment outcomes (Witkiewitz & Masyn, 2008). In a large sample of individuals in community residential facilities, alcohol-specific and general coping skills were associated with treatment success (Forys, McKellar, & Moos, 2007). Two recent studies provide additional evidence of CBT's effectiveness. First, in a trial comparing computerized CBT to standard outpatient treatment, the CBT intervention significantly and persistently increased coping skills, and quality of coping skills mediated treatment response (Kiluk, Nich, Babuscio, & Carroll, 2010). The sample in this study was a mixed group of individuals with SUDs, but the findings are nonetheless supportive of the theoretical approach. Second, in a study of individuals receiving CBT for AUD, numerous coping strategies were predictive of drinking outcomes, including 13 urge-specific coping skills and 18 general coping skills (Dolan, Rohsenow, Martin, & Monti, 2013). However, not all research in this area has been supportive. For example, Marlatt's originally proposed relapse taxonomy was not supported in a large multisite study (Kadden, 1996), and the efficacy of in vivo cue exposure as a treatment component has been contentious (Conklin & Tiffany, 2002; Monti & MacKillop, 2007). Additionally, in an investigation comparing group CBT and interactional therapy (Litt, Kadden, Cooney, & Kabela, 2003), coping skills predicted long-term outcome, but, surprisingly, were not differentially increased among the CBT group. Taken together, although the literature is not definitive, it nonetheless substantially supports the classification of the theoretical basis of CBT for AUDs as well supported.

In terms of efficacy, several reviews have been conducted specifically investigating the efficacy of relapse prevention. A meta-analysis of 26 studies including 9,504 total participants found that relapse prevention was effective for SUDs in general and most effective in AUDs (Irvin, Bowers, Dunn, & Wang, 1999). McCrady (2000) conducted a comprehensive review of 62 alcohol treatment studies that compared 13 different psychosocial approaches to treating AUDs; it concluded that relapse prevention is empirically validated based on the traditionally established criteria. More broadly, the most persuasive findings come from a meta-analysis of 53 trials including both CBT and relapse prevention (Magill & Ray, 2009). In this study, CBT was found to be efficacious for both SUDs in general and for AUDs in particular. Interestingly, the CBT effect was significant but modest in effect size when compared to other active treatments, but both significant and large in magnitude when compared to nontreatment control conditions. Thus, it appears that CBT is helpful in general but the amount of benefit is smaller in the context of other active treatments. Taken together, we consider the efficacy of CBT to be well supported. In conjunction with its strong theoretical basis described above, we classify CBT in Category I, being a treatment based on both well-supported theory and efficacy.

9.2.4 Behavioral Couples Therapy

Behavioral couples therapy for AUDs involves the patient's intimate partner in the treatment process in order to build motivation for abstinence and to restructure the interpersonal and environmental contingencies maintaining the patient's alcohol use. The approach is based on the assumption that AUDs are best conceptualized in an interactional context (Klostermann, Kelley, Mignone, Pusateri, & Wills, 2011). For example, an individual's alcohol use might contribute to instability and conflict in the dyad, while dysfunction in the couple's relationship might reinforce and maintain the patient's alcohol use. As such, behavioral couples therapy attempts to achieve positive outcomes by simultaneously targeting problematic alcohol use and relationship dysfunction and distress. Within treatment, alcohol-focused modules aim to promote abstinence, while relationship-focused modules aim to improve relationship functioning.

An important note about behavioral couples therapy is that two major forms exist: Alcohol Behavioral Couples Therapy (ABCT; McCrady & Epstein, 2009) and Behavioral Couples Therapy (BCT) for AUDs (O'Farrell & Fals-Stewart, 2006).[1] The programs use similar relationship-focused interventions but different alcohol use interventions. The relationship-focused interventions are thought to build incentives for abstinence through increased couple satisfaction and enjoyment when the partners spend time together. In terms of alcohol use interventions, ABCT relies on functional analyses to identify triggers, dysfunctional thoughts and emotions, and consequences of the patient's alcohol use. The therapist then helps the couple develop alternative behaviors and teaches the partner how to differentially reinforce drinking and abstinence behaviors. ABCT can be thought of as a specific application of CBT to the intersection of the dyad and alcohol misuse. In contrast, BCT utilizes a daily "trust discussion" during which the patient states his/her intent to remain abstinent that day. The couple is discouraged from discussing the patient's alcohol use at all other times throughout the day. Additionally, BCT recommends that the patient participate in adjunct treatments including taking Antabuse (disulfiram; a medication that interferes with the metabolism of alcohol and causes unpleasant physical sensations when the patient drinks alcohol), self-help groups (e.g., AA), and individual therapy. Table 9.3 reviews the components in the two respective therapeutic packages.

In terms of theoretical support, as mentioned, the fundamental premise of behavioral couple therapy for AUDs is that dysfunctional dyadic processes contribute to and maintain drinking behavior (O'Farrell & Clements, 2012). Based on this conceptualization, BCT is theorized to achieve positive outcomes by improving relationship functioning (O'Farrell & Clements, 2012). To date, three studies have been published that meet David and Montgomery's (2011) guidelines for evaluating a treatment's theoretical basis independent of the therapeutic package. These studies examined the mechanisms of action in BCT by testing treatment moderators and mediators in complex clinical trials. The first study demonstrated that having a supportive significant other (SSO) actively participate in at least one individual AUD treatment session resulted in fewer drinking days and drinking-related problems posttreatment than experienced by patients whose significant other did not participate in treatment (Hunter-Reel, Witkiewitz, & Zweben, 2012). An important caveat to this finding is that SSO attendance predicted better outcomes for some treatment activities but not others. For example, SSO attendance during drink refusal training, but not during communication training, resulted in better treatment outcomes (Hunter-Reel et al., 2012).

Table 9.3 Components of the two major forms of behavioral couples therapy for alcohol use disorder.

	Alcohol Behavioral Couples Therapy (McCrady & Epstein, 2009)	Behavioral Couples Therapy for Alcohol Use Disorders (O'Farrell & Fals-Stewart, 2006)
General		
Duration	Twelve 90-minute sessions (weekly)	Between twelve and twenty 50–60-minute sessions (weekly)
Typical session structure	1. Check-in and review homework 2. Introduce and practice new skills 3. Assign homework and plan for upcoming week	1. Review urges and any problems that occurred since the previous session 2. Review recovery contract and practice trust discussion 3. Review homework 4. Discuss current problems 5. Cover new material 6. Assign homework
Adjunct treatments	None specified	1. Daily Antabuse (disulfiram) medication 2. Self-help groups 3. Individual therapy sessions
Alcohol Use Intervention(s)		
Central component	*Functional analysis* PATIENT is taught to: 1. Identify triggers and high-risk situations, dysfunctional thoughts and emotions, and consequences of drinking 2. Modify triggers, thoughts and feelings, behavior, and consequences of drinking PARTNER is taught to: 1. Identify actions that might trigger the patient to drink 2. Develop alternative behaviors 3. Differentially reinforce drinking and abstinence behaviors	*Recovery contract* • Specifies what each partner will do to manage conflict about the patient's alcohol use, to rebuild trust, and to reward abstinence • Includes a daily "trust discussion" between partners during which the patient states his/her intent to remain abstinent and the partner observes the patient ingest Antabuse (disulfiram)
Other component(s)	Motivational enhancement in early sessions: 1. Therapist provides feedback about the patient's drinking 2. Together, the couple completes a decisional matrix exercise to identify the pros and cons of abstinence	Some session time is used to identify triggers for drinking and to develop coping strategies

Table 9.3 (*Continued*)

	Alcohol Behavioral Couples Therapy (McCrady & Epstein, 2009)	Behavioral Couples Therapy for Alcohol Use Disorders (O'Farrell & Fals-Stewart, 2006)
Couples Intervention(s)		
Reciprocity enhancement	Focuses on increasing positive activities, goodwill, and commitment to the relationship: 1. Each partner is taught to identify positive behavior in the other 2. Partners learn to give positive feedback for positive actions 3. Partners develop shared activities that they both enjoy	Focuses on increasing positive activities, goodwill, and commitment to the relationship: 1. "Catch your partner doing something nice": each partner tells the other partner one positive thing their partner did each day 2. "Caring day": partners plan surprise activities for each other 3. "Shared rewarding activities": partners develop shared activities that they both enjoy
Communication/ problem-solving training	Teaches couples how to resolve conflicts and problems: 1. Basic communication skills (e.g., effective listening and speaking) 2. Advanced skills (e.g., conflict resolution, negotiating for desired changes, and problem-solving)	Same as for Alcohol Behavioral Couples Therapy
Long-Term Planning		
Relapse prevention	The couple problem solves together to develop a prevention and relapse management plan for each partner and for the couple as a whole	• "Continuing recovery plan": specifies which aspects of BCT the couple will continue (e.g., morning trust discussions) • "Action plan": the partners plan out the steps they will take to prevent relapse • The couple may also come in for "checkups" every few months if needed

Other mechanistic findings provide similar support. A second study examined the association between relationship satisfaction and drinking urges on a day-to-day level for women undergoing either Alcohol Behavioral Individual Therapy (ABIT) or ABCT (Owens et al., 2013). The results demonstrated that a participant's average relationship satisfaction level over the course of treatment was related to that participant's drinking urges, and this association was moderated by treatment condition and time in treatment (Owens et al., 2013). Additionally, fewer drinking urges were observed among those with higher relationship satisfaction levels, and patients in the ABCT condition

demonstrated changes in the association between relationship satisfaction and drinking urges during treatment, while those in the ABIT condition did not (Owens et al., 2013). A third RCT examined whether improved couple communication differentially mediated alcohol outcomes in two treatment groups, BCT patients, and individual therapy patients (Walitzer, Dermen, Shyhalla, & Kubiak, 2013). It found that BCT resulted in fewer negative statements made by the couple when discussing conflict than the individual condition, and the relationship between spouse involvement and reductions in negative communication in the dyad was mediated by reductions in heavy drinking frequency. Importantly, results supported the hypothesis that the BCT condition increased problem-solving communication and reduced negative communication in the dyad (Walitzer et al., 2013). Together, these studies lend empirical support to the theorized mechanisms of action in BCT for AUDs by demonstrating that including a significant other in AUD treatment leads to improved relationship functioning and that improved relationship functioning contributes to better drinking outcomes.

Behavioral couples therapy for AUDs also has strong empirical support for the efficacy of the treatment approach. In an early meta-analytic review, Stanton and Shadish (1997) concluded that family-involved interventions result in higher abstinence levels than individual-based interventions. More recently, a meta-analysis evaluated eight RCTs and identified a clear and robust overall advantage of behavioral couples therapy over individual-based alcohol use treatments (Cohen's $d = 0.54$; Powers, Vedel, & Emmelkamp, 2008). BCT also proved superior across all three outcome domains evaluated: frequency of use (Cohen's $d = 0.36$), consequences of use (Cohen's $d = 0.52$), and relationship satisfaction (Cohen's $d = 0.57$; Powers et al., 2008). These findings were corroborated by recent RCTs demonstrating higher rates of abstinence at 1-year follow-up for patients who underwent BCT than for those who underwent individual therapy (McCrady, Epstein, Cook, Jensen, & Hildebrandt, 2009). In a more recent narrative review, Klostermann et al. (2011) argued that partner-involved treatments are the most broadly efficacious alcohol use interventions.

Additionally, BCT has been shown to be effective in secondary outcome domains and with a range of patient types (O'Farrell & Clements, 2012). For example, three studies have demonstrated that BCT is more effective than individual treatment at reducing high levels of interpersonal violence among treatment-seeking alcohol users (Fals-Stewart, Birchler, & Kelley, 2006; Fals-Stewart & Clinton-Sherrod, 2009; Schumm, O'Farrell, Murphy, & Fals-Stewart, 2009). Other studies have demonstrated increased child adjustment for children of alcoholic parents undergoing BCT (Kelley & Fals-Stewart, 2002, 2008; Lam, Fals-Stewart, & Kelley, 2008, 2009). The BCT approach has also been shown to be efficacious at increasing abstinence and relationship adjustment among gay and lesbian alcoholic patients (Fals-Stewart, O'Farrell, & Lam, 2009) and male veterans with comorbid AUD and posttraumatic stress disorder (Rotunda, O'Farrell, Murphy, & Babey, 2008). Additionally, two studies have evidenced preliminary support for behavioral couples therapy in treating dual-AUD couples (McCrady et al., 2009; Rotunda et al., 2008; Schumm, O'Farrell, & Burdzovic, 2012), although both studies were relatively small in size.

Considering this literature together, BCT may be classified as a Category I treatment in the T×E matrix. The theoretical foundation of BCT for treating problematic alcohol use is well supported, as evidenced by multiple studies that have found support for the mechanisms that the theory proposes underlie clinical effects. Furthermore, both

existing therapeutic packages are well supported, as each has been tested in at least two rigorous studies conducted by at least two different investigators or investigating teams.

9.2.5 Alcoholics Anonymous

AA grew out of the tradition of self-help groups that has been present in the United States since the 19th century (Kelly, 2003). From this perspective, AA's original (and contemporary) purpose was to provide a nonjudgmental environment for individuals with alcohol problems to help one another (AA, 1953). The central premise of AA is that following the 12 steps toward individual, interpersonal, and spiritual goals is the path to recovery. While AA is nondenominational, there is a strong spiritual component and individuals are encouraged to accept their powerlessness in the face of drinking and turn to a "higher power." Members are also encouraged to accept an alcoholic identity, assume abstinence as the treatment goal, and attend the meetings regularly (preferably daily during the first 90 days). AA is the largest and most popular mutual support program worldwide with over 2 million members in more than 180 countries (AA, 2017). At the same time, the structure of AA is highly decentralized, with great variability among groups and members and no administrative oversight or quality control. In contrast to AA itself, 12-step facilitation (TSF; Nowinski, Baker, & Carroll, 1992) is a treatment approach that is based on providing a framework to encourage clients to attend AA meetings (Finney, Wilbourne, & Moos, 2007; Project MATCH Research Group, 1997a). As part of TSF, individuals are expected to become involved in 12-step activities (e.g., going to meetings, seeking out a sponsor). From a theoretical perspective, TSF encompasses all the same principles as AA, but a clinician facilitates involvement in AA meetings and helps clients work through the steps of the program (Nowinski et al., 1992; Project MATCH Research Group, 1997a).

Regarding the etiology of alcoholism, the AA approach is atheoretical, meaning that, although the source writings of AA reflect an understanding of the biological, psychological, and social influences on alcoholism, AA does not focus on factors that lead to the initiation of alcohol use (Kurtz, 1979; Miller & Kurtz, 1994). Rather, AA's theoretical foundations speak to agents that lead to recovery. Specifically, an implicit recovery mechanism in the AA model involves members turning to a "higher power" for spiritual support (Miller & Kurtz, 1994). Several studies speak to the empirical validity of this theoretical mechanism. For instance, higher spirituality at baseline has been found to predict higher AA meeting attendance, greater involvement in AA-recommended activities, improved drinking outcomes at 1-year follow-up, and length of sobriety (Connors, Tonigan, & Miller, 1996, 2001; Zemore & Kaskutas, 2004). In one study, baseline spirituality failed to predict drinking outcomes; however, those who reported experiencing a "spiritual awakening" as a result of involvement in AA were more likely to be abstinent at 3-year follow-up (Kaskutas, Bond, & Weisner, 2003). Recently, a small number of studies have directly examined spirituality as a mediator of treatment outcome. A longitudinal study using the Project MATCH dataset found that attending AA was associated with increases in spiritual practices and that the relationship between AA attendance and improved drinking outcomes was partially mediated by increases in spirituality (Kelly, Stout, Magill, Tonigan, & Pagano, 2011). This finding has been replicated in an independent sample (Krentzman, Cranford, & Robinson, 2013). Thus, currently there is some evidence that spirituality is a mechanism of action for AA.

Other mechanisms of action for AA's positive effects have also been examined. One such mechanism is the level of active involvement in AA, which includes sponsoring other members and actively working through the steps of recovery, in addition to attendance rate. Several studies have found that involvement in AA-related activities produced somewhat higher rates of abstinence than CBT via fostering engagement with the group (Humphreys, Huebsch, Finney, & Moos, 1999) and predicted abstinence and lower levels of drinking when drinks were consumed in the first year posttreatment (Montgomery, Miller, & Tonigan, 1995; Pagano, Friend, Tonigan, & Stout, 2004; Tonigan, Miller, & Connors, 2000). Another proposed mechanism of action in AA is altering individuals' social networks (Groh, Jason, & Keys, 2008; Kelly, Magill, & Stout, 2009). Longabaugh, Wirtz, Zweben, and Stout (1998) found that Project MATCH participants with social networks supportive of drinking had better long-term outcomes in TSF than participants with social networks supportive of drinking in the other two conditions (i.e., CBT and MET). Other studies have suggested that social network effects are among the most prominent mechanisms of drinking outcomes in AA and that the effect of AA attendance on treatment outcome is mediated by participants' establishing a nondrinking network (Cooney, Babor, DiClemente, & Del Boca, 2003; Kelly, Hoeppner, Stout, & Pagano, 2012). Most recently, there is evidence that AA may exert positive effects by leading to a reduction in impulsivity (Blonigen, Timko, & Moos, 2013). Thus, there appear to be several plausible mechanisms of action for positive AA effects, not all of which are encompassed by AA's theoretical foundations. As AA is based on a spiritual model, it does not necessarily emphasize active involvement with the group or altering the social network, even if the approach itself does. Therefore, given only a modest literature in support of spirituality as a mechanism of positive effects of AA, in addition to findings that support change mechanisms *not* encompassed by AA's theoretical underpinnings (e.g., social network changes), we consider the support for the theory of AA as equivocal.

Evaluating the efficacy of AA is complicated by a number of factors. As noted previously, AA is a community-based, grassroots, mutual support organization, not a treatment in the conventional sense. It is not delivered by a clinician, attendance is entirely voluntary, and the groups and practices can be highly heterogeneous. As such, AA is not particularly compatible with being studied using RCTs. In studies that have undertaken examination of AA's effects on drinking, the impact of AA is typically measured via a frequency count of AA meetings attendance. In a review of 107 studies that involved AA, the authors found that AA had positive, moderate effects on drinking behavior and psychosocial functioning (Emrick, Tonigan, Montgomery, & Little, 1993). However, a meta-analysis that included only RCTs concluded that the evidence for the efficacy of AA was not supported, although the results were likely confounded by the fact that participants were mandated to seek treatment (e.g., by the judicial system; Kownacki & Shadish, 1999). A recent review also suggests that, considered narrowly, there is insufficient evidence to conclude that AA is an efficacious treatment (Ferri, Amato, & Davoli, 2006).

In individual studies that do not operate as conventional RCTs, however, AA often appears to be associated with improved outcomes. In longitudinal investigations, AA involvement has been found to predict abstinence and improved psychosocial functioning in a number of studies (Cross, Morgan, Mooney, Martin, & Rafter, 1990; Humphreys, Moos, & Finney, 1996; Kelly, Stout, Zywiak, & Schneider, 2006; Pisani, Fawcett, Clark, & McGuire, 1993; Tonigan, Connors, & Miller, 2002). This relationship may be dose-dependent. For example, Montgomery et al. (1995) demonstrated that AA

meeting attendance alone did not predict treatment outcome, but the degree to which patients had become involved in AA did. In fact, a dose–response analysis in another study indicated that even small amounts of participation may be helpful in increasing abstinence, though higher doses may be needed to reduce relapse intensity (Kelly et al., 2006). AA and AA plus formal treatment have also been found to produce lower abstinence rates than formal treatment alone over a 3-year follow-up (Timko, Moos, Finney, & Lesar, 2000). In a naturalistic design in a US Veterans Affairs setting, individuals who were involved in AA were more likely to be abstinent at 1-year follow-up compared to those receiving cognitive–behavioral treatment (Ouimette, Finney, & Moos, 1997). Additionally, continual involvement in AA at 1-year follow-up was associated with better alcohol-related and psychosocial outcomes (Ouimette et al., 1997). Therefore, despite methodological issues inherent in research on AA, participation in AA appears to be associated with improved psychosocial functioning and drinking outcomes, possibly in a dose–response relationship.

There has been more success in systematically evaluating the effect of TSF on drinking outcomes using RCTs, as it is a manualized treatment approach that has all the parameters of other formal treatments. In Project MATCH, TSF was equally as effective as CBT and MET in improving drinking outcomes (Project MATCH Research Group, 1997a). In fact, at 12-month follow-up, the clients in TSF reported higher abstinence than those in CBT and MET (Project MATCH Research Group, 1997a). A study that compared TSF to relapse prevention in individuals with SUDs (28.6% of participants met criteria for alcohol dependence) found that the two treatments produced equivalent outcomes, although they differed in processes involved (Brown, Seraganian, Tremblay, & Annis, 2002). Given that there have been two studies by independent research groups indicating that TSF is at least equivalent to another empirically supported treatment, it should be considered an empirically supported treatment.

Classifying AA in the T×E matrix is challenging. With regard to theory, there is some support for spiritual rehabilitation as the mechanism of recovery, as well as evidence that processes not articulated by AA source documents do play an important role (e.g., social network changes, impulsivity). This suggests that support for the theory of AA is best categorized as equivocal, or, more aptly, incomplete in terms of the processes involved. This should not be considered too pejorative a judgment in light of the fact that the "theory" of AA is over 60 years old and never purported to be a psychological theory of behavior change. With regard to efficacy, considering that AA is sui generis among AUD treatments, the broad corpus of findings suggests that it is well supported as a treatment. To be clear, that is not to say that AA will be categorically helpful for all individuals. Rather, the empirical literature clearly indicates that people who self-select into AA experience benefits and that a formal AA-facilitation treatment modality generates comparable outcomes to other well-supported treatments. Together, we consider AA to be best classified as Category II, a well-supported treatment with an equivocal theoretical basis.

9.2.6 Confrontational Interventions

For the general public, the most likely recommendation to motivate a person to enter treatment is to stage an "intervention," or a structured confrontation of the individual by their family and friends. Developed in the 1970s, the practice is broadly intended to bulldoze through the denial that characterizes the disorder. Confrontational

interventions have several core components: they are arranged secretly, with the intention of surprising the target individual; during the intervention, each person enumerates the adverse consequences the target's drinking has had on them and the specific consequences of not immediately entering treatment; and finally, the target is asked to accept treatment or prepare for the dire consequences previously threatened. Confrontational interventions are very widely known to the general public because of their prominence in popular culture. It was the strategy used to push First Lady Betty Ford to seek treatment, and interventions have been dramatically featured on a large number of television programs. Indeed, a reality television program, *Intervention*, is now in its 15th season of presenting case studies of active addicts experiencing confrontational interventions.

Evaluating the theory and efficacy of confrontational interventions is surprisingly challenging, however. There is relatively little in terms of theory underlying the approach, beyond the original book recommending the practice (Johnson, 1986), and there has been only a modest amount of empirical research on its utility. Nonetheless, given its high visibility and the fact that it is actively being employed in the community, a disposition will be made to the extent possible. With regard to theory, a core principle of confrontational interventions is that individuals with AUDs and other addictive disorders are in denial about their problem; that is, they do not acknowledge that their drinking is a problem. This is not supported in empirical studies of motivation for change among individuals with AUDs and other SUDs. A large body of work on measures of treatment motivation reveals that substantial proportions of individuals with AUDs commonly acknowledge that they consider their drinking or other drug use to be problematic (Maisto, Chung, Cornelius, & Martin, 2003; Maisto et al., 1999; Mitchell, Francis, & Tafrate, 2005). The data from these measures suggest that there is a broad distribution of ambivalence and motivation for change among individuals with AUDs. A second theoretical principle of confrontational interventions is that addiction is also maintained by denial and enabling on the part of the individual's family and friends, both of whom are targets of the intervention. However, this premise is also not supported by empirical findings. From the perspective of mechanistic research, the other method of theory validation, no studies have verified that positive effects of confrontational interventions can be attributed to effects in these domains. In short, the theory supporting confrontational interventions is generally meager, and, where it can be addressed by empirical studies, they are generally not supportive.

With regard to clinical outcome, the findings are also not highly supportive of confrontational interventions. One early study reported high rates of treatment entry following interventions, but with a small sample and with high rates of families being unwilling to undertake the confrontation (Liepman, Nirenberg, & Begin, 1989). A second study retrospectively compared a confrontational intervention to four other strategies and found higher rates of entry in response to the confrontational strategy but also higher rates of relapse (Loneck, Garrett, & Banks, 1996a, 1996b). This suggests that the approach succeeds in strong-arming individuals into treatment but fails at the ultimate goal of treatment success. The most rigorously designed study was an RCT that systematically compared confrontational interventions with CRAFT and an Al-Anon (a program of recovery for people who are affected by another person's drinking) facilitation therapy (Miller, Meyers, & Tonigan, 1999). That study found the CRAFT intervention was superior to both other strategies, and, again, only a small proportion

of families were willing to execute the confrontational intervention. Although this literature is small, it is fairly uniformly not supportive of confrontational interventions. This is particularly the case as the CRAFT approach has been validated in its own right and demonstrated to be superior to confrontational interventions.

Based on the literature on theory and efficacy, we consider confrontational interventions to be unsupported on both fronts, classifying it in Category IX. The theoretical basis for effectively "ganging up" on patients to browbeat them into treatment is weak, and the empirical data in clinical studies are not supportive. As this approach is probably second only to AA in terms of public awareness, this disposition is particularly troubling.

9.3 Implications for Research and Practice

There are a number of implications from the preceding review, both for research on AUD treatment and for clinical practice more broadly. Some are relatively straightforward extensions of the rationales for different classification. For example, AA was determined to fall within Category II because the theoretical foundations require further explication and specific testing. However, there are also important implications for the treatments categorized as well supported in terms of theory and efficacy. In each case, although the literature is generally supportive, we would not assert that these are definitive conclusions. As noted, in most cases there were notable lacunae, whether in aspects of theory testing or in the evaluation of clinical outcomes. In particular, empirical testing of the mechanisms of behavior change that are associated with a given treatment remains a shallow domain in the literature. This is of particular priority for the field because understanding the treatment processes that are most helpful, for a specific treatment and across several well-supported ones, will be critical for optimizing care. In addition, there is increasing interest in using novel neurocognitive assessments to understand treatment prognosis and mechanisms of behavior change (Feldstein Ewing & Chung, 2013). These tools include functional magnetic resonance imaging, diffusion tensor imaging, and magnetoencephalography, among others, and the well-supported treatments highlighted in this chapter represent particularly good candidates for further dissection using these methods.

The further importance of clarifying mechanisms of action is in the service of clarifying the overlap and unique components of the well-supported treatments. Although treatments were reviewed as discrete units, there were shared components in some cases and multiple variants of the same treatment in others. For example, the CRA includes elements of relapse prevention and behavioral couples therapy includes aspects of CBT. Thus, it would be artificially definitive to conclude that we identified six truly discrete treatments that were well supported in terms of efficacy. Instead, there is enough overlap to suggest that AUD treatment is best thought of as a Venn diagram of evidence-based approaches that have varying degrees of overlap. Teasing out the active ingredients underlying positive effects will illuminate the extent to which different approaches are useful because they effectively share common processes and the extent to which they leverage distinct mechanisms.

The implications for practice from the preceding review are starker. Given the evidence that there are a number of treatments that are well supported in terms of theory and efficacy, one clear implication is that these treatments should be widely available to

the general public. The reality is that they are typically only available in a minority of treatment facilities (Carroll, 2014). Furthermore, although not formalized to the extent that they could be evaluated in the current chapter, many practices that have no basis in evidence are actively in widespread use in the treatment community. As highlighted in the recent general-audience book *Inside Rehab: The Surprising Truth About Addiction Treatment—and How to Get Help That Works* (Fletcher, 2013), the "real-world" treatment landscape is a hodgepodge of practices that frequently have a tenuous link to the research literature and can come at exorbitant cost. This would be an entirely unacceptable state of affairs in the treatment of, say, cardiovascular disease or cancer, or virtually all other medical conditions, but it is the reality in AUD and the treatment of other addictive disorders. The converse implication is also true. The practice of confrontational interventions and other dubious treatments that are not supported by either theory or evidence should be explicitly cautioned against in practice guidelines. In short, there is a need for systemic change in the availability of evidence-based treatment.

9.4 Conclusions

What emerges from the perspective of the T×E matrix is a generally auspicious state of affairs for AUD treatments. The majority of the approaches reviewed were classified as having convincing evidence in support of both the theoretical approach and the treatment package. The CRA, CM, MI, CBT, and behavioral couples therapy would all be considered well-supported, evidence-based treatments in the conventional sense. AA requires a more nuanced perspective, which is unsurprising given its unique niche in the clinical landscape, but would also be considered well supported with the attendant considerations. Only one approach was found to entirely fall short, the use of a confrontational intervention to initiate treatment. In this case, we found support for neither the proposed theory nor the efficacy of the approach. Unfortunately, despite this, confrontational interventions continue to be widely implemented and have high levels of public awareness. Among the well-supported treatments, ambiguities remain with regard to the operative mechanisms underlying positive effects and the overlap in active ingredients. Furthermore, there remain major gaps in implementation, as many of the well-supported treatments discussed are scarcely available in the community at large. Continuing to make progress in these two domains is a critical priority for the future.

Acknowledgments

Dr. MacKillop is the holder of the Peter Boris Chair in Addictions Research, which partially supported his contributions.

Note

1 The acronyms ABCT and BCT refer to these specific therapeutic packages, but "behavioral couples therapy" refers to the broad domain of treating AUDs in the context of the dyad.

References

Abbott, P. J., Weller, S. B., Delaney, H. D., & Moore, B. A. (1998). Community reinforcement approach in the treatment of opiate addicts. *American Journal of Drug and Alcohol Abuse*, *24*(1), 17–30. doi:10.3109/00952999809001696

Alcoholics Anonymous. (1953). *Twelve steps and twelve traditions*. New York, NY: Alcoholics Anonymous Publishing.

Alcoholics Anonymous. (2017). Estimates of AA groups and members as of January 2, 2017: Service material from the General Service Office. Retrieved October 16, 2017, from https://www.aa.org/assets/en_US/smf-53_en.pdf

Alessi, S. M., & Petry, N. M. (2013). A randomized study of cellphone technology to reinforce alcohol abstinence in the natural environment. *Addiction*, *108*(5), 900–909. doi:10.1111/add.12093

American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders* (1st ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd rev. ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed. TR).Washington, DC: American Psychiatric Association.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.

Apodaca, T. R., & Longabaugh, R. (2009). Mechanisms of change in motivational interviewing: A review and preliminary evaluation of the evidence. *Addiction*, *104*(5), 705–715. doi:10.1111/j.1360-0443.2009.02527.x

Azrin, N. H. (1976). Improvements in the community-reinforcement approach to alcoholism. *Behavioral Research Therapy*, *14*(5), 339–348. doi:10.1016/0005-7967(76)90021-8

Azrin, N. H., Sisson, R. W., Meyers, R., & Godley, M. (1982). Alcoholism treatment by disulfiram and community reinforcement therapy. *Journal of Behavioral Therapy and Experimental Psychiatry*, *13*(2), 105–112. doi:10.1016/0005-7916(82)90050-7

Ball, S. A., Todd, M., Tennen, H., Armeli, S., Mohr, C., Affleck, G., & Kranzler, H. R. (2007). Brief motivational enhancement and coping skills interventions for heavy drinking. *Addictive Behaviors*, *32*(6), 1105–1118. doi:10.1016/j.addbeh.2006.07.014

Bandura, A., & McClelland, D. (1977). *Social learning theory*. New York, NY: General Learning Press.

Barnett, N. P., Tidey, J., Murphy, J. G., Swift, R., & Colby, S. M. (2011). Contingency management for alcohol use reduction: A pilot study using a transdermal alcohol sensor. *Drug and Alcohol Dependence*, *118*(2), 391–399. doi:10.1016/j.drugalcdep.2011.04.02

Bem, D. J. (1972). Self-perception theory. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (pp. 1–62). New York, NY: Academic Press.

Bickel, W. K., Hughes, J. R., DeGrandpre, R. J., Higgins, S. T., & Rizzuto, P. (1992). Behavioral economics of drug self-administration: IV. The effects of response requirement on the consumption of and interaction between concurrently available coffee and cigarettes. *Psychopharmacology*, *107*(2/3), 211–216. doi:10.1007/bf02245139

Bigelow, G. (2001). An operant behavioral analysis of alcohol abuse and dependence. In N. Heather, T. J. Peters, & T. Stockwell (Eds.), *International handbook of alcohol dependence and problems* (pp. 299–315). Chichester, UK: John Wiley & Sons.

Blonigen, D. M., Timko, C., & Moos, R. H. (2013). Alcoholics Anonymous and reduced impulsivity: A novel mechanism of change. *Substance Abuse*, *34*(1), 4–12. doi:10.1080/08897077.2012.691448

Blum, T. C., Roman, P. M., & Bennett, N. (1989). Public images of alcoholism: Data from a Georgia survey. *Journal of Studies on Alcohol*, *50*(1), 5–14. doi:10.15288/jsa.1989.50.5

Bohn, M. J., Krahn, D. D., & Staehler, B. A. (1995). Development and initial validation of a measure of drinking urges in abstinent alcoholics. *Alcoholism: Clinical and Experimental Research*, *19*(3), 600–606. doi:10.1111/j.1530-0277.1995.tb01554.x

Brown, T., Seraganian, G., Tremblay, P., & Annis, H. (2002). Process and outcome changes with relapse prevention versus 12-step aftercare programs for substance abuses. *Addiction*, *97*(6), 677–689. doi:10.1046/j.1360-0443.2002.00101.x

Caetano, R. (1987). Public opinions about alcoholism and its treatment. *Journal of Studies on Alcohol*, *48*(2), 153–160. doi:10.15288/jsa.1987.48.153

Carroll, K. M. (2014). Lost in translation? Moving contingency management and cognitive behavioral therapy into clinical practice. *Annals of the New York Academy of Sciences*, *1327*, 94–111. doi:10.1111/nyas.12501

Carter, B. L., & Tiffany, S. T. (1999). Meta-analysis of cue-reactivity in addiction research. *Addiction*, *94*(3), 327–340. doi:10.1046/j.1360-0443.1999.9433273.x

Collins, R. L., Vincent, P. C., Yu, J., Liu, L., & Epstein, L. H. (2014). A behavioral economic approach to assessing demand for marijuana. *Experimental and Clinical Psychopharmacology*, *22*(3), 211–221. doi:10.1037/a0035318

Conklin, C. A., & Tiffany, S. T. (2002). Applying extinction research and theory to cue-exposure addiction treatments. *Addiction*, *97*(2), 155–167. doi:10.1046/j.1360-0443.2002.00014.x

Connors, G. J., Tonigan, J. S., & Miller, W. R. (1996). A measure of religious background and behavior for use in behavior change research. *Psychology of Addictive Behaviors*, *10*(2), 90–96. doi:10.1037//0893-164x.10.2.90

Connors, G. J., Tonigan, J. S., & Miller, W. R. (2001). A longitudinal model of intake symptomatology, AA participation and outcome: Retrospective study of the Project MATCH outpatient and aftercare samples. *Journal of Studies on Alcohol and Drugs*, *62*(6), 817–825. doi:10.15288/jsa.2001.62.817

Cooney, N. L., Babor, T. F., DiClemente, C. C., & Del Boca, F. K. (2003). Clinical and scientific implications of Project MATCH. In T. F. Babor & F. K. Del Boca (Eds.), *Treatment matching in alcoholism* (pp. 222–237). New York, NY: Cambridge University Press.

Courtney, K. E., & Polich, J. (2009). Binge drinking in young adults: Data, definitions, and determinants. *Psychological Bulletin*, *135*(1), 142–156. doi:10.1037/a0014414

Cross, G. M., Morgan, C. W., Mooney, A. J., Martin, C. A., & Rafter, J. A. (1990). Alcoholism treatment: A ten-year follow-up study. *Alcoholism: Clinical and Experimental Research*, *14*(2), 169–173. doi:10.1111/j.1530-0277.1990.tb00465.x

Darkes, J., Greenbaum, P. E., & Goldman, M. S. (2004). Alcohol expectancy mediation of biopsychosocial risk: Complex patterns of mediation. *Experimental and Clinical Psychopharmacology*, *12*(1), 27–38. doi:10.1037/1064-1297.12.1.27 2004-10475-007

David, D., & Montgomery, G. H. (2011). The scientific status of psychotherapies: A new evaluative framework for evidence-based psychosocial interventions. *Clinical Psychology: Science and Practice*, *18*(2), 89–99. doi:10.1111/j.1468-2850.2011.01239.x

De Jong, C. A. J., Roozen, H. G., Van Rossum, L. G. M., Krabbe, P. F. M., & Kerkhof, A. J. F. M. (2007). High abstinence rates in heroin addicts by a new comprehensive treatment approach. *American Journal on Addictions*, *16*(2), 124–130. doi:10.1080/10550490601184472

Dennis, M., Godley, S. H., Diamond, G., Tims, F. M., Babor, T., Donaldson, J., … Funk, R. (2004). The Cannabis Youth Treatment (CYT) Study: Main findings from two randomized trials. *Journal of Substance Abuse Treatment*, *27*(3), 197–213. doi:10.1016/j.jsat.2003.09.005

Dolan, S. L., Rohsenow, D. J., Martin, R. A., & Monti, P. M. (2013). Urge-specific and lifestyle coping strategies of alcoholics: Relationships of specific strategies to treatment outcome. *Drug and Alcohol Dependence*, *128*(1/2), 8–14. doi:10.1016/j.drugalcdep.2012.07.010

Dougherty, D. M., Hill-Kapturczak, N., Liang, Y., Karns, T. E., Cates, S. E., Lake, S. L., … Roache, J. D. (2014). Use of continuous transdermal alcohol monitoring during a contingency management procedure to reduce excessive alcohol use. *Drug and Alcohol Dependence*, *142*, 301–306. doi:10.1016/j.drugalcdep.2014.06.039

Dutcher, L. W., Anderson, R., Moore, M., Luna-Anderson, C., Meyers, R., Delaney, H. D., & Smith, J. E. (2009). Community reinforcement and family training (CRAFT): An effectiveness study. *Journal of Behavior Analysis in Health, Sports, Fitness and Medicine*, *2*(1), 80–93.

Emrick, C. D., Tonigan, J. S., Montgomery, H., & Little, L. (1993). Alcoholics Anonymous: What is currently known? In B. S. McCrady & W. R. Miller (Eds.), *Research on Alcoholics Anonymous: Opportunities and alternatives* (pp. 41–76). New Brunswick, NJ: Rutgers Center of Alcohol Studies.

Esser, M. B., Hedden, S. L., Kanny, D., Brewer, R. D., Gfroerer, J. C., & Naimi, T. S. (2014). Prevalence of alcohol dependence among US adult drinkers, 2009–2011. *Preventing Chronic Disease*, *11*, E206. doi:10.5888/pcd11.140329

Fals-Stewart, W., Birchler, G. R., & Kelley, M. L. (2006). Learning sobriety together: A randomized clinical trial examining behavioral couples therapy with alcoholic female patients. *Journal of Consulting and Clinical Psychology*, *74*(3), 579–591. doi:10.1037/0022-006X.74.3.579

Fals-Stewart, W., & Clinton-Sherrod, M. (2009). Treating intimate partner violence among substance-abusing dyads: The effect of couples therapy. *Professional Psychology: Research and Practice*, *40*(3), 257–263. doi:10.1037/a0012708

Fals-Stewart, W., O'Farrell, T. J., & Lam, W. K. K. (2009). Behavioral couple therapy for gay and lesbian couples with alcohol use disorders. *Journal of Substance Abuse Treatment*, *37*(4), 379–387. doi:10.1016/j.jsat.2009.05.001

Feldstein Ewing, S. W., & Chung, T. (2013). Neuroimaging mechanisms of change in psychotherapy for addictive behaviors: Emerging translational approaches that bridge biology and behavior. *Psychology of Addictive Behaviors*, *27*(2), 329–335. doi:10.1037/a0031491

Ferri, M., Amato, L., & Davoli, M. (2006). Alcoholics Anonymous and other 12-step programmes for alcohol dependence. *Cochrane Database of Systematic Reviews*, *3*(2), CD005032. doi:10.1002/14651858.CD005032.pub2

Festinger, L. (1957). *A theory of cognitive dissonance*. Stanford, CA: Stanford University Press.

Finney, J. W., Wilbourne, P. L., & Moos, R. H. (2007). Psychosocial treatments for substance use disorders. In P. E. Nathan & J. M. Gorman (Eds.), *A guide to treatments that work* (pp. 179–202). New York, NY: Oxford University Press.

Fletcher, A. M. (2013). *Inside rehab: The surprising truth about addiction treatment—and how to get help that works*. Harmondsworth, UK: Penguin.

Forys, K., McKellar, J., & Moos, R. (2007). Participation in specific treatment components predicts alcohol-specific and general coping skills. *Addictive Behaviors*, *32*(8), 1669–1680. doi:10.1016/j.addbeh.2006.11.023

Gaume, J., Gmel, G., & Daeppen, J.-B. (2008). Brief alcohol interventions: Do counsellors' and patients' communication characteristics predict change? *Alcohol and Alcoholism*, *43*(1), 62–69. doi:10.1093/alcalc/agm141

Ghitza, U. E., Epstein, D. H., & Preston, K. L. (2008). Contingency management reduces injection-related HIV risk behaviors in heroin and cocaine using outpatients. *Addictive Behaviors*, *33*(4), 593–604. doi:10.1016/j.addbeh.2007.11.009

Godley, M. D., Godley, S. H., Dennis, M. L., Funk, R., & Passetti, L. L. (2002). Preliminary outcomes from the assertive continuing care experiment for adolescents discharged from residential treatment. *Journal of Substance Abuse Treatment*, *23*(1), 21–32. doi:10.1016/s0740-5472(02)00230-1

Grant, B., Stinson, F., Dawson, D. A., Chou, S. P., Dufour, M. C., Compton, W., … Kaplan, K. (2004). Prevalence and co-occurrence of substance use disorders and independent mood and anxiety disorders: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Archives of General Psychiatry*, *61*(8), 807–816. doi:10.1001/archpsyc.61.8.807

Gray, J. C., & MacKillop, J. (2014). Interrelationships among individual differences in alcohol demand, impulsivity, and alcohol misuse. *Psychology of Addictive Behaviors*, *28*(1), 282–287. doi:10.1037/a0032766

Groh, D. R., Jason, L. A., & Keys, C. B. (2008). Social network variables in Alcoholics Anonymous: A literature review. *Clinical Psychology Review*, *28*(3), 430–450. doi:10.1016/j.cpr.2007.07.014

Hasin, D. S., Schuckit, M. A., Martin, C. S., Grant, B. F., Bucholz, K. K., & Helzer, J. E. (2003). The validity of DSM-IV alcohol dependence: What do we know and what do we need to know? *Alcoholism: Clinical and Experimental Research*, *27*(2), 244–252. doi:10.1097/01.ALC.0000060878.61384.ED

Hasin, D. S., Stinson, F. S., Ogburn, E., & Grant, B. F. (2007). Prevalence, correlates, disability, and comorbidity of DSM-IV alcohol abuse and dependence in the United

States: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Archives of General Psychiatry*, *64*(7), 830–842. doi:10.1001/archpsyc.64.7.830

Hettema, J., Steele, J., & Miller, W. R. (2005). Motivational interviewing. *Annual Review of Clinical Psychology*, *1*, 91–111. doi:10.1146/annurev.clinpsy.1.102803.143833

Higgins, S. T., Bickel, W. K., & Hughes, J. R. (1994). Influence of an alternative reinforcer on human cocaine self-administration. *Life Sciences*, *55*(3), 179–187. doi:10.1016/0024-3205(94)00878-7

Higgins, S. T., Heil, S. H., & Lussier, J. P. (2004). Clinical implications of reinforcement as a determinant of substance use disorders. *Annual Review of Psychology*, *55*, 431–461. doi:10.1146/annurev.psych.55.090902.142033

Higgins, S. T., Wong, C. J., Badger, G. J., Ogden, D. E., & Dantona, R. L. (2000). Contingent reinforcement increases cocaine abstinence during outpatient treatment and 1 year of follow-up. *Journal of Consulting and Clinical Psychology*, *68*(1), 64–72. doi:10.1037//0022-006x.68.1.64

Humphreys, K., Huebsch, P. D., Finney, J. W., & Moos, R. H. (1999). A comparative evaluation of substance abuse treatment: V. Substance abuse treatment can enhance the effectiveness of self-help groups. *Alcoholism: Clinical and Experimental Research*, *23*(3), 558–563. doi:10.1111/j.1530-0277.1999.tb04153.x

Humphreys, K., Moos, R. H., & Finney, J. W. (1996). Life domains, Alcoholics Anonymous, and role incumbency in the 3-year course of problem drinking. *Journal of Nervous and Mental Disease*, *184*(8), 475–481. doi:10.1097/00005053-199608000-00004

Hunt, G. M., & Azrin, N. H. (1973). A community-reinforcement approach to alcoholism. *Behavioral Research Therapy*, *11*(1), 91–104. doi:10.1016/0005-7967(73)90072-7

Hunter-Reel, D., Witkiewitz, K., & Zweben, A. (2012). Does session attendance by a supportive significant other predict outcomes in individual treatment for alcohol use disorders? *Alcoholism: Clinical and Experimental Research*, *36*(7), 1237–1243. doi:10.1111/j.1530-0277.2011.01719.x

Irvin, J. E., Bowers, C. A., Dunn, M. E., & Wang, M. C. (1999). Efficacy of relapse prevention: A meta-analytic review. *Journal of Consulting and Clinical Psychology*, *67*(4), 563–570. doi:10.1037//0022-006x.67.4.563

John, L. K., Loewenstein, G., Troxel, A. B., Norton, L., Fassbender, J. E., & Volpp, K. G. (2011). Financial incentives for extended weight loss: A randomized controlled trial. *Journal of General Internal Medicine*, *26*(6), 621–626. doi:10.1007/s11606-010-1628-y

Johnson, V. E. (1986). *Intervention: How to help someone who doesn't want help—A step-by-step guide for families and friends of chemically dependent persons.* Center City, MN: Hazelden.

Kadden, R. M. (1996). Is Marlatt's relapse taxonomy reliable or valid? *Addiction*, *91*(Suppl.), S139–S145. doi:10.1046/j.1360-0443.91.12s1.4.x

Kahler, C. W., & Strong, D. R. (2006). A Rasch model analysis of DSM-IV alcohol abuse and dependence items in the National Epidemiological Survey on Alcohol and Related Conditions. *Alcoholism: Clinical and Experimental Research*, *30*(7), 1165–1175. doi:10.1111/j.1530-0277.2006.00140.x

Kaskutas, L. A., Bond, J., & Weisner, C. (2003). The role of religion, spirituality and Alcoholics Anonymous in sustained sobriety. *Alcoholism Treatment Quarterly*, *21*(1), 1–16. doi:10.1300/J020v21n01_01

Katcher, B. S. (1993). Benjamin Rush's educational campaign against hard drinking. *American Journal of Public Health*, *83*(2), 273–281. doi:10.2105/ajph.83.2.273

Kelley, M. L., & Fals-Stewart, W. (2002). Couples- versus individual-based therapy for alcohol and drug abuse: Effects on children's psychosocial functioning. *Journal of Consulting and Clinical Psychology*, *70*(2), 417–427. doi:10.1037//0022-006x.70.2.417

Kelley, M. L., & Fals-Stewart, W. (2008). Treating paternal drug abuse using Learning Sobriety Together: Effects on adolescents versus children. *Drug and Alcohol Dependence*, *92*(1/3), 228–238. doi:10.1016/j.drugalcdep.2007.08.003

Kelly, J. F. (2003). Self-help for substance-use disorders: History, effectiveness, knowledge gaps, and research opportunities. *Clinical Psychology Review*, *23*(5), 639–663. doi:10.1016/S0272-7358(03)00053-9

Kelly, J. F., Hoeppner, B., Stout, R. L., & Pagano, M. (2012). Determining the relative importance of the mechanisms of behavior change within Alcoholics Anonymous: A multiple mediator analysis. *Addiction*, *107*(2), 289–299. doi:10.1111/j.1360-0443.2011.03593.x

Kelly, J. F., Magill, M., & Stout, R. L. (2009). How do people recover from alcohol dependence? A systematic review of the research on mechanisms of behavior change in Alcoholics Anonymous. *Addiction Research & Theory*, *17*(3), 236–259. doi:10.1080/16066350902770458

Kelly, J. F., Stout, R. L., Magill, M., Tonigan, J. S., & Pagano, M. E. (2011). Spirituality in recovery: A lagged mediational analysis of Alcoholics Anonymous' principal theoretical mechanism of behavior change. *Alcoholism: Clinical and Experimental Research*, *35*(3), 454–463. doi:10.1111/j.1530-0277.2010.01362.x

Kelly, J. F., Stout, R., Zywiak, W., & Schneider, R. (2006). A 3-year study of addiction mutual-help group participation following intensive outpatient treatment. *Alcoholism: Clinical and Experimental Research*, *30*(8), 1381–1392. doi:10.1111/j.1530-0277.2006.00165.x

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*(6), 593–602. doi:10.1001/archpsyc.62.6.593

Kiluk, B. D., Nich, C., Babuscio, T., & Carroll, K. M. (2010). Quality versus quantity: Acquisition of coping skills following computerized cognitive-behavioral therapy for substance use disorders. *Addiction*, *105*(12), 2120–2127. doi:10.1111/j.1360-0443.2010.03076.x

Kirby, K. C., Marlowe, D. B., Festinger, D. S., Garvey, K. A., & La Monaca, V. (1999). Community reinforcement training for family and significant others of drug abusers: A unilateral intervention to increase treatment entry of drug users. *Drug and Alcohol Dependence*, *56*(1), 85–96. doi:10.1016/s0376-8716(99)00022-8

Klostermann, K., Kelley, M. L., Mignone, T., Pusateri, L., & Wills, K. (2011). Behavioral couples therapy for substance abusers: Where do we go from here? *Substance Use & Misuse*, *46*(12), 1502–1509. doi:10.3109/10826084.2011.576447

Kownacki, R. J., & Shadish, W. R. (1999). Does Alcoholics Anonymous work? The results from a meta-analysis of controlled experiments. *Substance Use & Misuse*, *34*(13), 1897–1916. doi:10.3109/10826089909039431

Krentzman, A. R., Cranford, J. A., & Robinson, E. A. R. (2013). Multiple dimensions of spirituality in recovery: A lagged mediational analysis of Alcoholics Anonymous'

principal theoretical mechanism of behavior change. *Substance Abuse*, *34*(1), 20–32. doi:10.1080/08897077.2012.691449

Kuntsche, E., Knibbe, R., Gmel, G., & Engels, R. (2006). Who drinks and why? A review of socio-demographic, personality, and contextual issues behind the drinking motives in young people. *Addictive Behaviors*, *31*(10), 1844–1857. doi:10.1016/j.addbeh.2005.12.028

Kurtz, E. (1979). *Not God: A history of Alcoholics Anonymous*. Center City, MN: Hazelden.

Lam, W. K., Fals-Stewart, W., & Kelley, M. L. (2008). Effects of parent skills training with behavioral couples therapy for alcoholism on children: A randomized clinical pilot trial. *Addictive Behaviors*, *33*(8), 1076–1080. doi:10.1016/j.addbeh.2008.04.002

Lam, W. K. K., Fals-Stewart, W., & Kelley, M. L. (2009). Parent training with behavioral couples therapy for fathers' alcohol abuse: Effects on substance use, parental relationship, parenting, and CPS involvement. *Child Maltreatment*, *14*(3), 243–254. doi:10.1177/1077559509334091

Ledgerwood, D. M., Arfken, C. L., Petry, N. M., & Alessi, S. M. (2014). Prize contingency management for smoking cessation: A randomized trial. *Drug and Alcohol Dependence*, *140*, 208–212. doi:10.1016/j.drugalcdep.2014.03.032

Liepman, M. R., Nirenberg, T. D., & Begin, A. M. (1989). Evaluation of a program designed to help family and significant others to motivate resistant alcoholics into recovery. *American Journal of Drug and Alcohol Abuse*, *15*(2), 209–221. doi:10.3109/00952998909092721

Litt, M. D., Kadden, R. M., Cooney, N. L., & Kabela, E. (2003). Coping skills and treatment outcomes in cognitive-behavioral and interactional group therapy for alcoholism. *Journal of Consulting and Clinical Psychology*, *71*(1), 118–128. doi:10.1037//0022-006x.71.1.118

Loneck, B., Garrett, J. A., & Banks, S. M. (1996a). A comparison of the Johnson Intervention with four other methods of referral to outpatient treatment. *American Journal of Drug and Alcohol Abuse*, *22*(2), 233–246. doi:10.3109/00952999609001656

Loneck, B., Garrett, J. A., & Banks, S. M. (1996b). The Johnson Intervention and relapse during outpatient treatment. *American Journal of Drug and Alcohol Abuse*, *22*(3), 363–375. doi:10.3109/00952999609001665

Longabaugh, R., Wirtz, P. W., Zweben, A., & Stout, R. L. (1998). Network support for drinking, Alcoholics Anonymous and long-term matching effects. *Addiction*, *93*(9), 1313–1333. doi:10.1046/j.1360-0443.1998.93913133.x

Lundahl, B. W., Kunz, C., Brownell, C., Tollefson, D., & Burke, B. L. (2010). A meta-analysis of motivational interviewing: Twenty-five years of empirical studies. *Research on Social Work Practice*, *20*(2), 137–160. doi:10.1177/1049731509347850

MacKillop, J., & Lisman, S. A. (2005). Reactivity to alcohol cues: Isolating the role of perceived availability. *Experimental and Clinical Psychopharmacology*, *13*(3), 229–237. doi:10.1037/1064-1297.13.3.229

MacKillop, J., Menges, D. P., McGeary, J. E., & Lisman, S. A. (2007). Effects of craving and DRD4 VNTR genotype on the relative value of alcohol: An initial human laboratory study. *Behavioral and Brain Functions*, *3*, 11. doi:10.1186/1744-9081-3-11

MacKillop, J., Murphy, J. G., Ray, L. A., Eisenberg, D. T., Lisman, S. A., Lum, J. K., & Wilson, D. S. (2008). Further validation of a cigarette purchase task for assessing the relative reinforcing efficacy of nicotine in college smokers. *Experimental and Clinical Psychopharmacology*, *16*(1), 57–65. doi:10.1037/1064-1297.16.1.57

Magill, M., & Ray, L. (2009). Cognitive-behavioral treatment with adult alcohol and illicit drug users: A meta-analysis of randomized controlled trials. *Journal of Studies on Alcohol and Drugs*, *70*, 516–527. doi:10.15288/jsad.2009.70.516

Maisto, S. A., Chung, T. A., Cornelius, J. R., & Martin, C. S. (2003). Factor structure of the SOCRATES in a clinical sample of adolescents. *Psychology of Addictive Behaviors*, *17*(2), 98–107. doi:10.1037/0893-164x.17.2.98

Maisto, S. A., Conigliaro, J., McNeil, M., Kraemer, K., O'Connor, M., & Kelley, M. E. (1999). Factor structure of the SOCRATES in a sample of primary care patients. *Addictive Behaviors*, *24*(6), 879–892. doi:10.1016/s0306-4603(99)00047-7

McCaul, M. E., Stitzer, M. L., Bigelow, G. E., & Liebson, I. A. (1984). Contingency management interventions: Effects on treatment outcome during methadone detoxification. *Journal of Applied Behavior Analysis*, *17*(1), 35–43. doi:10.1901/jaba.1984.17-35

McCrady, B. S. (2000). Alcohol use disorders and the Division 12 Task Force of the American Psychological Association. *Psychology of Addictive Behaviors*, *14*(3), 267–276. doi:10.1037//0893-164x.14.3.267

McCrady, B. S., & Epstein, E. E. (2009). *Overcoming alcohol problems: A couples focused program (therapist guide)*. Oxford, UK: Oxford University Press.

McCrady, B. S., Epstein, E. E., Cook, S., Jensen, N., & Hildebrandt, T. (2009). A randomized trial of individual and couple behavioral alcohol treatment for women. *Journal of Consulting and Clinical Psychology*, *77*(2), 243–256. doi:10.1037/a0014686

McDonell, M. G., Howell, D. N., McPherson, S., Cameron, J. M., Srebnik, D., Roll, J. M., & Ries, R. K. (2012). Voucher-based reinforcement for alcohol abstinence using the ethyl-glucuronide alcohol biomarker. *Journal of Applied Behavior Analysis*, *45*(1), 161–165. doi:10.1901/jaba.2012.45-161

Mendelson, J. H., & Mello, N. K. (1966). Experimental analysis of drinking behavior of chronic alcoholics. *Annals of the New York Academy of Sciences*, *133*(3), 828–845.

Meyers, R. J., & Miller, W. R. (2001). *A community reinforcement approach to addiction treatment*. New York, NY: Cambridge University Press.

Meyers, R. J., Miller, W. R., Smith, J. E., & Tonigan, J. S. (2002). A randomized trial of two methods for engaging treatment-refusing drug users through concerned significant others. *Journal of Consulting and Clinical Psychology*, *70*(5), 1182–1185. doi:10.1037//0022-006x.70.5.1182

Miller, W. R. (1983). Motivational interviewing with problem drinkers. *Behavioural Psychotherapy*, *11*(2), 147–172. doi:10.1017/S0141347300006583

Miller, W. R., Benefield, R. G., & Tonigan, J. S. (1993). Enhancing motivation for change in problem drinking: A controlled comparison of two therapist styles. *Journal of Consulting and Clinical Psychology*, *61*(3), 455–461. doi:10.1037//0022-006x.61.3.455

Miller, W. R., & Hester, R. K. (2003). Treating alcohol problems: Toward an informed eclecticism. In R. K. Hester & W. R. Miller (Eds.), *Handbook of alcoholism treatment approaches: Effective approaches* (pp. 1–12). Boston, MA: Allyn & Bacon.

Miller, W. R., & Kurtz, E. (1994). Models of alcoholism used in treatment: Contrasting AA and other perspectives with which it is often confused. *Journal of Studies on Alcohol and Drugs*, *55*(2), 159–166. doi:10.15288/jsa.1994.55.159

Miller, W. R., Meyers, R. J., & Hiller-Sturmhofel, S. (1999). The community-reinforcement approach. *Alcohol Research & Health*, *23*(2), 116–121.

Miller, W. R., Meyers, R. J., & Tonigan, J. S. (1999). Engaging the unmotivated in treatment for alcohol problems: A comparison of three strategies for intervention through family

members. *Journal of Consulting and Clinical Psychology*, *67*(5), 688–697. doi:10.1037//0022-006x.67.5.688

Miller, W. R., & Rollnick, S. (2012). *Motivational interviewing: Helping people change* (3rd ed.). New York, NY: Guilford Press.

Miller, W. R., & Rose, G. S. (2009). Toward a theory of motivational interviewing. *American Psychologist*, *64*(6), 527–537. doi:10.1037/a0016830

Miller, W. R., Sovereign, R. G., & Krege, B. (1988). Motivational interviewing with problem drinkers: II. The drinker's check-up as a preventive intervention. *Behavioural and Cognitive Psychotherapy*, *16*(4), 251–268. doi:10.1017/S0141347300014129

Miller, W. R., Zweben, A., DiClemente, C. C., & Rychtarik, R. C. (1992). *Motivational Enhancement Therapy manual: A clinical research guide for therapists treating individuals with alcohol abuse and dependence*. Rockville, MD: National Institute on Alcohol Abuse and Alcoholism.

Mitchell, D., Francis, J. P., & Tafrate, R. C. (2005). The psychometric properties of the Stages of Change Readiness and Treatment Eagerness Scale (SOCRATES) in a clinical sample of active duty military service members. *Military Medicine*, *170*(11), 960–963. doi:10.7205/milmed.170.11.960

Montgomery, H. A., Miller, W. R., & Tonigan, J. S. (1995). Does Alcoholics Anonymous involvement predict treatment outcome? *Journal of Substance Abuse Treatment*, *12*(4), 241–246. doi:10.1016/0740-5472(95)00018-Z

Monti, P. M. (2002). *Treating alcohol dependence: A coping skills training guide*. New York, NY: Guilford Press.

Monti, P. M., & MacKillop, J. (2007). Advances in the treatment of craving for alcohol and tobacco. In P. M. Miller & D. J. Kavanagh (Eds.), *Translation of addictions sciences into practice* (pp. 209–235). Amsterdam, Netherlands: Elsevier.

Monti, P. M., & O'Leary, T. A. (1999). Coping and social skills training for alcohol and cocaine dependence. *Psychiatric Clinics of North America*, *22*(2), 447–470, 10.1016/s0193-953x(05)70086-1

Monti, P. M., & Rohsenow, D. J. (1999). Coping-skills training and cue-exposure therapy in the treatment of alcoholism. *Alcohol Research & Health*, *23*(2), 107–115.

Moyers, T. B., Martin, T., Houck, J. M., Christopher, P. J., & Tonigan, J. S. (2009). From in-session behaviors to drinking outcomes: A causal chain for motivational interviewing. *Journal of Consulting and Clinical Psychology*, *77*(6), 1113–1124. doi:10.1037/a0017189

Moyers, T. B., Miller, W. R., & Hendrickson, S. M. L. (2005). How does motivational interviewing work? Therapist interpersonal skill predicts client involvement within motivational interviewing sessions. *Journal of Consulting and Clinical Psychology*, *73*(4), 590–598. doi:10.1037/0022-006X.73.4.590

Murphy, C. M., Stojek, M. K., Few, L. R., Rothbaum, A. O., & MacKillop, J. (2014). Craving as an alcohol use disorder symptom in DSM-5: An empirical examination in a treatment-seeking sample. *Experimental and Clinical Psychopharmacology*, *22*(1), 43–49. doi:10.1037/a0034535

Murphy, J., & MacKillop, J. (2006). Relative reinforcing efficacy of alcohol among college student drinkers. *Experimental and Clinical Psychopharmacology*, *14*(2), 219–227. doi:10.1037/1064-1297.14.2.219

Murphy, J. G., MacKillop, J., Skidmore, J. R., & Pederson, A. A. (2009). Reliability and validity of a demand curve measure of alcohol reinforcement. *Experimental and Clinical Psychopharmacology*, *17*(6), 396–404. doi:10.1037/a0017684

Nathan, P. E., Titler, N. A., Lowenstein, L. M., Solomon, P., & Rossi, A. M. (1970). Behavioral analysis of chronic alcoholism: Interaction of alcohol and human contact. *Archives of General Psychiatry*, *22*(5), 419–430. doi:10.1001/archpsyc.1970.01740290035005

Nowinski, J., Baker, S., & Carroll, K. M. (1992). *Twelve step facilitation therapy manual: A clinical research guide for therapists treating individuals with alcohol abuse and dependence* (Vol. 1). Washington, DC: National Institute on Alcohol Abuse and Alcoholism.

O'Farrell, T. J., & Clements, K. (2012). Review of outcome research on marital and family therapy in treatment for alcoholism. *Journal of Marital and Family Therapy*, *38*(1), 122–144. doi:10.1111/j.1752-0606.2011.00242.x

O'Farrell, T. J., & Fals-Stewart, W. (2006). *Behavioral couples therapy for alcoholism and drug abuse*. New York, NY: Guilford Press.

O'Malley, S. S., Krishnan-Sarin, S., Farren, C., Sinha, R., & Kreek, M. J. (2002). Naltrexone decreases craving and alcohol self-administration in alcohol-dependent subjects and activates the hypothalamo-pituitary-adrenocortical axis. *Psychopharmacology*, *160*, 19–29. doi:10.1007/s002130100919

Oslin, D. W., Cary, M., Slaymaker, V., Colleran, C., & Blow, F. C. (2009). Daily ratings measures of alcohol craving during an inpatient stay define subtypes of alcohol addiction that predict subsequent risk for resumption of drinking. *Drug and Alcohol Dependence*, *103*(3), 131–136. doi:10.1016/j.drugalcdep.2009.03.009

Ouimette, P. C., Finney, J. W., & Moos, R. H. (1997). Twelve-step and cognitive-behavioral treatment for substance abuse: A comparison of treatment effectiveness. *Journal of Consulting and Clinical Psychology*, *65*(2), 230–240.

Owens, M. D., Hallgren, K. A., Ladd, B. O., Rynes, K., McCrady, B. S., & Epstein, E. (2013). Associations between relationship satisfaction and drinking urges for women in alcohol behavioral couples and individual therapy. *Alcoholism Treatment Quarterly*, *31*(4), 415–430. doi:10.1080/07347324.2013.831668

Pagano, M. E., Friend, K. B., Tonigan, J. S., & Stout, R. L. (2004). Helping other alcoholics in Alcoholics Anonymous and drinking outcomes: Findings from Project MATCH. *Journal of Studies on Alcohol and Drugs*, *65*(6), 766–773. doi:10.15288/jsa.2004.65.766

Petry, N. M., Martin, B., Cooney, J. L., & Kranzler, H. R. (2000). Give them prizes, and they will come: Contingency management for treatment of alcohol dependence. *Journal of Consulting and Clinical Psychology*, *68*(2), 250–257.

Pisani, V. D., Fawcett, J., Clark, D. C., & McGuire, M. (1993). The relative contributions of medication adherence and AA meeting attendance to abstinent outcome for chronic alcoholics. *Journal of Studies on Alcohol and Drugs*, *54*(1), 115–119. doi:10.1037//0022-006x.68.2.250

Powers, M. B., Vedel, E., & Emmelkamp, P. M. G. (2008). Behavioral couples therapy (BCT) for alcohol and drug use disorders: A meta-analysis. *Clinical Psychology Review*, *28*(6), 952–962. doi:10.1016/j.cpr.2008.02.002

Prendergast, M., Podus, D., Finney, J., Greenwell, L., & Roll, J. (2006). Contingency management for treatment of substance use disorders: A meta-analysis. *Addiction*, *101*(11), 1546–1560. doi:10.1111/j.1360-0443.2006.01581.x

Project MATCH Research Group. (1997a). Matching alcoholism treatments to client heterogeneity: Project MATCH posttreatment drinking outcomes. *Journal of Studies on Alcohol*, *58*, 7–29.

Project MATCH Research Group. (1997b). Project MATCH secondary a priori hypotheses. *Addiction*, *92*, 1671–1698. doi:10.1111/j.1360-0443.1997.tb02889.x

Project MATCH Research Group. (1998a). Matching alcoholism treatments to client heterogeneity: Project MATCH three-year drinking outcomes. *Alcoholism: Clinical and Experimental Research*, *22*(6), 1300–1311. doi:10.1111/j.1530-0277.1998.tb03912.x

Project MATCH Research Group. (1998b). Therapist effects in three treatments for alcohol problems. *Psychotherapy Research*, *8*, 455–474. doi:10.1093/ptr/8.4.455

Rogers, C. R. (1959). A theory of therapy, personality, and interpersonal relationships as developed in the client-centered framework. In P. Koch (Ed.), *Psychology: The study of a science* (pp. 184–256). New York, NY: McGraw-Hill.

Roozen, H. G., Kerkhof, A. J. F. M., & Van den Brink, W. (2003). Experiences with an outpatient relapse program (community reinforcement approach) combined with naltrexone in the treatment of opioid-dependence: Effect on addictive behaviors and the predictive value of psychiatric comorbidity. *European Addiction Research*, *9*(2), 53–58. doi:10.1159/000068808

Rotunda, R. J., O'Farrell, T. J., Murphy, M., & Babey, S. H. (2008). Behavioral couples therapy for comorbid substance use disorders and combat-related posttraumatic stress disorder among male veterans: An initial evaluation. *Addictive Behaviors*, *33*(1), 180–187. doi:10.1016/j.addbeh.2007.06.001

Saha, T. D., Chou, S. P., & Grant, B. F. (2006). Toward an alcohol use disorder continuum using item response theory: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Psychological Medicine*, *36*(7), 931–941. doi:10.1017/S003329170600746X

Schuckit, M. A. (2009). Alcohol-use disorders. *Lancet*, *373*(9662), 492–501. doi:10.1016/S0140-6736(09)60009-X

Schuckit, M. A., Danko, G. P., Smith, T. L., Bierut, L. J., Bucholz, K. K., Edenberg, H. J., … Hinga, B. (2008). The prognostic implications of DSM-IV abuse criteria in drinking adolescents. *Drug and Alcohol Dependence*, *97*(1/2), 94–104. doi:10.1016/j.drugalcdep.2008.03.020

Schuckit, M. A., Smith, T. L., Danko, G. P., Kramer, J., Godinez, J., Bucholz, K. K., … Hesselbrock, V. (2005). Prospective evaluation of the four DSM-IV criteria for alcohol abuse in a large population. *American Journal of Psychiatry*, *162*(2), 350–360. doi:10.1176/appi.ajp.162.2.350

Schumm, J. A., O'Farrell, T. J., & Burdzovic, J. A. (2012). Behavioral couples therapy when both partners have a current alcohol use disorder. *Alcoholism Treatment Quarterly*, *30*(4), 407–421. doi:10.1080/07347324.2012.718963

Schumm, J. A., O'Farrell, T. J., Murphy, C. M., & Fals-Stewart, W. (2009). Partner violence before and after couples-based alcoholism treatment for female alcoholic patients. *Journal of Consulting and Clinical Psychology*, *77*(6), 1136–1146. doi:10.1037/a0017389

Sisson, R. W., & Azrin, N. H. (1986). Family-member involvement to initiate and promote treatment of problem drinkers. *Journal of Behavior Therapy and Experimental Psychiatry*, *17*(1), 15–21. doi:10.1016/0005-7916(86)90005-4

Skinner, B. F. (1938/1991). *The behavior of organisms: An experimental analysis*. Acton, MA: Copley.

Smith, J. E., Meyers, R. J., & Delaney, H. D. (1998). The community reinforcement approach with homeless alcohol-dependent individuals. *Journal of Consulting and Clinical Psychology*, *66*(3), 541–548. doi:10.1037//0022-006x.66.3.541

Stanton, M. D., & Shadish, W. R. (1997). Outcome, attrition, and family-couples treatment for drug abuse: A meta-analysis and review of the controlled, comparative studies. *Psychological Bulletin*, *122*(2), 170–191. doi:10.1037//0033-2909.122.2.170

Stitzer, M., & Petry, N. (2006). Contingency management for treatment of substance abuse. *Annual Review of Clinical Psychology*, *2*, 411–434. doi:10.1146/annurev.clinpsy.2.022305.095219

Timko, C., Moos, R. H., Finney, J. W., & Lesar, M. D. (2000). Long-term outcomes of alcohol use disorders: Comparing untreated individuals with those in Alcoholics Anonymous and formal treatment. *Journal of Studies on Alcohol and Drugs*, *61*(4), 529–540. doi:10.15288/jsa.2000.61.529

Tonigan, J. S., Connors, G. J., & Miller, W. R. (2002). Participation and involvement in Alcoholics Anonymous. In T. F. Babor & F. K. Del Boca (Eds.), *Matching alcoholism treatments to client heterogeneity* (pp. 184–204). New York, NY: Cambridge University Press.

Tonigan, J. S., Miller, W. R., & Connors, G. J. (2000). Project MATCH client impressions about Alcoholics Anonymous. *Alcoholism Treatment Quarterly*, *18*(1), 25–41. doi:10.1300/j020v18n01_02

UKATT Research Team. (2005). Effectiveness of treatment for alcohol problems: Findings of the randomised UK Alcohol Treatment Trial (UKATT). *BMJ*, *331*(7516), 331–541. doi:10.1136/bmj.331.7516.541

Vasilaki, E. I., Hosier, S. G., & Cox, W. M. (2006). The efficacy of motivational interviewing as a brief intervention for excessive drinking: A meta-analytic review. *Alcohol and Alcoholism*, *41*(3), 328–335. doi:10.1093/alcalc/agl016

Volpp, K. G., John, L. K., Troxel, A. B., Norton, L., Fassbender, J., & Loewenstein, G. (2008). Financial incentive-based approaches for weight loss: A randomized trial. *JAMA*, *300*(22), 2631–2637. doi:10.1001/jama.2008.804

Volpp, K. G., Loewenstein, G., Troxel, A. B., Doshi, J., Price, M., Laskin, M., & Kimmel, S. E. (2008). A test of financial incentives to improve warfarin adherence. *BMC Health Service Research*, *8*, 272. doi:10.1186/1472-6963-8-272

Waldron, H. B., Kern-Jones, S., Turner, C. W., Peterson, T. R., & Ozechowski, T. J. (2007). Engaging resistant adolescents in drug abuse treatment. *Journal of Substance Abuse Treatment*, *32*(2), 133–142. doi:10.1016/j.jsat.2006.07.007

Walitzer, K., Dermen, K., Shyhalla, K., & Kubiak, A. (2013). Couple communication among problem drinking males and their spouses: A randomized controlled trial. *Journal of Family Therapy*, *35*(3), 229–251. doi:10.1111/j.1467-6427.2013.00615.x

White, W. L. (2014). *Slaying the dragon: The history of addiction treatment and recovery in America* (2nd ed.). Bloomington, IN: Chestnut Health Systems.

Witkiewitz, K., & Marlatt, G. A. (2004). Relapse prevention for alcohol and drug problems: That was Zen, this is Tao. *American Psychologist*, *59*(4), 224–235. doi:10.1037/0003-066X.59.4.224

Witkiewitz, K., & Masyn, K. E. (2008). Drinking trajectories following an initial lapse. *Psychology of Addictive Behaviors*, *22*(2), 157–167. doi:10.1037/0893-164X.22.2.157

World Health Organization. (1992). *ICD-10 classification of mental and behavioral disorders: Clinical descriptions and diagnostic guidelines*. Geneva, Switzerland: World Health Organization.

Zemore, S. E., & Kaskutas, L. A. (2004). Helping, spirituality and Alcoholics Anonymous in recovery. *Journal of Studies on Alcohol and Drugs*, *65*(3), 383–391. doi:10.15288/jsa.2004.65.383

10

Psychotherapeutic Treatments for Male and Female Sexual Dysfunction Disorders

Jessica C. Emanu,[1] Isabelle Avildsen,[2] and Christian J. Nelson[3]

[1] *Department of Psychology, Long Island University, Brooklyn, USA*

[2] *Department of Psychology, The Graduate Center, City University of New York, New York, USA*

[3] *Department of Psychiatry and Behavioral Sciences, Memorial Sloan Kettering Cancer Center, New York, USA*

Sexual dysfunction is a common class of disorders and refers to difficulty experienced by an individual or a couple during any stage of a normal sexual experience, including in terms of physical pleasure, desire, preference, arousal, and orgasm (Simons & Carey, 2001). According to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5), sexual dysfunction comprises nine disorders categorized into four subgroups: sexual desire disorders (hypoactive sexual desire disorder [HSD], sexual aversion disorder); arousal disorders (male erectile disorder, female sexual arousal disorder); orgasm disorders (female orgasmic disorder, male orgasmic disorder, premature ejaculation); and pain disorders (dyspareunia, vaginismus). Varying subtypes of sexual dysfunction are used to designate the onset of the difficulty, which has implications for different etiologies of the disorders as well as treatments (American Psychiatric Association, 2013). "Lifelong" refers to a sexual problem that has been present from an individual's first sexual encounter, whereas "acquired" applies to sexual disorders that develop after a period of relatively normal sexual stimulation, situations, or partners (American Psychiatric Association, 2013). Lastly, "situational" refers to sexual difficulties that only occur with certain types of stimulations, situations, or partners (American Psychiatric Association, 2013).

Sexual dysfunctions globally are estimated to have a prevalence within the general population of 46% (Simons & Carey, 2001). However, epidemiological studies have yielded varying lifetime prevalence rates: in men, HSDs have the highest lifetime prevalence (26%), whereas premature ejaculation is estimated to occur in approximately 5% of the general population. A strong increase in both prevalence and incidence of problems with erections has been documented, particularly in men after the age 50. According to the DSM-5, approximately 13–21% of men aged 40–80 years complain of occasional problems with erections, and 2% of men younger than ages 40–50 years complain of frequent problems with erections. However, in men older than 60–70 years, 40–50% have significant problems with erections. In women, HSDs are most common (16%), while orgasmic disorders (4%) and dyspareunia (3%) are less frequent (Simons & Carey, 2001).

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

Determining prevalence rates of sexual dysfunctions cross-culturally has been more challenging, as sexual dysfunction is itself culturally influenced, and different cultures create standards of sexuality, sexual prowess, and behavior (Bhavsar & Bhugra, 2013). More work is needed to understand how cultural factors shape the thresholds and definitions of sexual dysfunction in different groups, as sexual dysfunction is associated with reduced quality of life, and impaired sexual and marital satisfaction (Frühauf, Gerger, Schmidt, Munder, & Barth, 2013). In a study conducted by Laumann, Paik, and Rosen (1999), it was revealed that women who experienced low sexual desire disorder, arousal disorder, and sexual pain disorder had low feelings of physical and emotional satisfaction and low feelings of happiness. Similarly, men with erectile dysfunction and low sexual desire disorder experienced a diminished quality of life. Additionally, sexual dysfunctions are typically caused or exacerbated by psychological distress, and, with this in mind, psychological interventions are important treatment options to explore.

10.1 Treatments for Sexual Dysfunction

In this chapter, we review the available psychotherapies for male and female sexual disorders in terms of both research support for the overall therapy and support for the putative mechanism of change that underlies the treatment. There have been several established therapies designed specifically for sexual dysfunctions, including cognitive–behavioral therapy (CBT), sex therapy, behavioral therapy, educational interventions, and other psychotherapies, which include mindfulness meditation therapies, hypnotherapy, and rational emotive therapy. We classify each treatment according to David and Montgomery's (2011; see also Chapter 1) nine-category taxonomy, which posits that an empirically validated treatment is supported by a strong theoretical base (mechanism of psychological change). In this view, to be considered efficacious, the therapeutic package must derive from the theory from which it is based. Therefore, the strongest treatments have empirical support for both the overall treatment and the mechanism of change that underlies that treatment. Accordingly, each treatment is evaluated and assigned to one of three levels: empirically well supported, preliminary data less than minimum standards, or mixed data and/or strong contradictory evidence.

10.2 Category I

10.2.1 Cognitive–Behavioral Therapy

CBT is a type of psychotherapeutic treatment developed by Aaron Beck (1996) that helps patients understand the thoughts and feelings that influence their behaviors (Avagianou, 2015). CBT has been used to treat a variety of disorders and is a short-term treatment that focuses on identifying and changing maladaptive thought patterns that have a negative influence on behavior. According to Beck (1996), maladaptive cognitions contribute to the maintenance of emotional distress and behavioral problems. Beck (1996) posits that these maladaptive cognitions include general beliefs or schemas about the world, the self, and the future, which give rise to specific and automatic thoughts in particular situations. According to cognitive–behavioral theory, therapeutic strategies to change these maladaptive cognitions lead to changes in emotional distress and

problematic behaviors (Beck, 1979). Adaptations have been made to standard CBT protocols to address sexual dysfunctions that aim to modify dysfunctional beliefs (cognitive restructuring), with a specific emphasis on homework assignments, out-of-session activities, psychoeducation, and acquisition of skills (Avagianou, 2015).

10.2.1.1 Evidence for therapy

10.2.1.1.1 Males

In total, two studies have examined the effectiveness of CBT for male sexual dysfunctions. McCabe, Price, Piterman, and Lording (2008) evaluated the effectiveness of an internet-based treatment program for erectile dysfunction by comparing outcome measures for men utilizing this program to a no-treatment control group. Eligibility consisted of the following: (1) persistent or recurrent difficulties attaining or maintaining an erection during sexual activity, (2) 18 years or older, (3) absence of major medical or psychiatric problems, (4) involvement in a stable heterosexual relationship, (5) willingness and motivation from partners to participate in treatment, and (6) regular internet access. As it was anticipated that many participants would already be using oral PDE5 inhibitors (phosphodiesterase type 5 inhibitors; pills used to treatment erectile dysfunction), medical treatments for erectile dysfunction were permitted. Men were instructed to maintain a record of frequency of medical treatment usage throughout the study and were also required to document cessation of medical treatment, as cessation may have influenced the responsiveness to the psychological treatment. The outcome measures were the International Index of Erectile Function (IIEF), the Kansas Marital Satisfaction Scale, the Index of Sexual Satisfaction, and the Self-Esteem and Relationship Questionnaire.

The treatment was a 10-week, internet-based CBT program for men with erectile dysfunction and their partners, entitled Rekindle, which focused on the resolution of psychological and relationship factors related to erectile dysfunction. Rekindle consisted of three main treatment components: sensate focus, communication exercises, and email contact with a therapist. Men and their partners spent approximately 2 weeks completing five modules, with earlier modules providing the foundation for later modules. Each of the modules had similar formats, beginning with a list of goals that couples were required to achieve, followed by communication exercises, sensate-focus activities, and a reminder that email contact was available for those seeking further support. Modules began with general body pleasuring and advanced to genital contact and then intercourse. The primary focus was to help the couples enjoy the sensations of the sexual interactions rather than focusing on their performance. Communication exercises were completed prior to each sensate-focus session and were designed to allow partners to express their feeling about the program and their relationships, as well as resolve any presenting problems. All modules concluded with a hurdle requirement, in which the couples compiled a list of statements that both needed to agree upon. Once a statement was agreed upon, each couple was given a username and password to access the following module.

Men and their partners were randomly allocated to either the treatment ($n = 24$) or the control condition ($n = 20$). All men completed questionnaire measures at pretest, at posttest (after the completion of the therapy for the treatment group, and after 10 weeks for the control group), and at 3-months follow-up (initial treatment group only). In total, 12 men and their partners in the treatment group completed the program, and 8 out of

the 12 completed follow-up, whereas 19 participants from the control group completed the posttest questionnaires.

From pretest to posttest, the treatment group reported significantly greater improvements in erectile functioning; however, other areas of sexual functioning did not change. After the completion of Rekindle, men in the treatment arm experienced a 41.66% increase in either fully erect or almost fully erect erections. At 3-month follow-up, men experienced further gains in erectile functioning, with a decrease in erectile dysfunction from 62.5% to 37.5%. Mixed model multivariate analysis of variance revealed a significant group-by-time interaction in overall relationship satisfaction, sexual relationship satisfaction, and sexual relationship quality, where the treatment group experienced significantly greater improvements than the control group.

Andersson et al. (2011) conducted a randomized controlled trial (RCT) comparing a guided internet-delivered cognitive–behavioral therapy (ICBT) for erectile dysfunction to an active control group. The ICBT consisted of seven weekly modules delivered via the internet, in which participants were given access to the treatment modules by completing homework assignments and reporting them to their therapists. Participants were recruited from media outlets, such as articles and interviews on TV and in newspapers. Both heterosexual and homosexual individuals were eligible for participation. Eligibility consisted of (1) having access to the internet, (2) being more than 18 years old, (3) having a score greater than 21 on the IIEF, and (4) having been in a stable relationship with a partner for at least 3 months.

The first module of ICBT focused on explaining erectile dysfunction from a biological, psychological, and social perspective, and information was given on how each of these perspectives influences the development and maintenance of erectile dysfunction. In the second module, participants were taught about the cognitive–behavioral perspective and how it relates to erectile dysfunction symptoms. Within the third module, participants were instructed to discuss the implications of their erectile dysfunction with their partners and together to conceptualize erectile dysfunction from a CBT perspective. Modules four through six focused on exposure tasks that gradually exposed the couple to sexual interaction. During this time, men were asked to rate their level of anxiety and note their intrusive thoughts, while simultaneously developing relaxation strategies for managing these thoughts and anxiety. The last module focused on relapse prevention by giving participants strategies that they could utilize alone or with a partner to maintain gains from the treatment. The active control-group participants had access to an online discussion forum, in which participants could send anonymous messages to one another for 7 weeks.

The primary outcome measure was the IIEF, and the secondary outcome measures consisted of the IIEF, the Relationship Assessment Scale, the Beck Anxiety Inventory, the Beck Depression Inventory–II, the World Health Organization Quality of Life–Brief Version, and the Clinical Global Impression Scale. All measures were delivered via the internet, except for the Clinical Global Impression Scale, which was administered via telephone. Using a mixed-effects model, a paired *t*-test revealed a trend toward improvement in the control group between pre- and posttest. After the control group received ICBT, within-group effects were significant on the IIEF, and the control group made similar improvements to the treatment group. At posttreatment, a significant association was found between number of completed modules and improvements on the IIEF–5.

10.2.1.1.2 Females

Three studies have examined the effectiveness of CBT for sexual dysfunction in women. Van Lankveld et al. (2006) compared CBT to a waitlist control group in an RCT of women with vaginismus. The 117 participants were randomized to a group-administered format of CBT, a bibliotherapy format of CBT, or a waitlist control condition. All conditions included pre- and posttreatment assessments at the same time intervals at pretreatment, posttreatment, 3-month follow-up, and 12-month follow-up. Participants were recruited by referral from the outpatient sexology clinics of Maastricht and Leiden university hospitals in the Netherlands or responded to newspaper ads. Eligibility criteria for the study were (1) being a heterosexual woman, (2) being aged 18 or older, (3) having a diagnosis of lifelong vaginismus, (4) being in a heterosexual relationship of at least 3 months' duration, and (5) being in good general health as per medical history. The treatment included sexual education, relaxation exercises, gradual exposure, cognitive therapy, and sensate focus exercises. The participants were given a manual on the treatment of vaginismus, a relaxation and sexual fantasy exercise CD-ROM, and a CD-ROM with instructions on the progressive relaxation technique. Additionally, they were given a diary to monitor the progress of their practice. In sessions, the participants were encouraged to try gradual exposure under applied relaxation, visual inspection of their own vagina with a handheld mirror, digital touching without penetration by both the participant and her partner, and then vaginal insertion by the participant and gradually by her partner and with a dilator. Participants began with the least anxiety-provoking exercise and gradually penetration was tried. Following sessions introduced the method of rational emotive self-analysis, and participants were encouraged to practice cognitive restructuring, identifying irrational cognitions, identifying irrational beliefs, self-talking, and making these into more rational cognitions. The group therapy consisted of ten 2-hour group sessions and each group consisted of six to nine couples. The bibliotherapy group received six biweekly 15-minute telephone contacts over the course of the treatment period, during which the assistant asked which manual chapters the participant had read, what needed further explanation, which exercises had been done, what difficulties had been encountered, how they had been overcome, and which cognitive strategies were used in this respect.

The primary outcome measure was behavioral functioning (the achievement of vaginal intercourse and noncoital penetration) and secondary outcome measures consisted of the Mini International Neuropsychiatric Interview, the Female Sexual Function Index, the Maudsley Marital Questionnaire, the Golombok Rust Inventory of Sexual Satisfaction, and the Sexual and Physical Abuse Questionnaire.

Use of a chi-square model determined that treated participants significantly more often reported successful intercourse at posttreatment than did waitlist participants. In the CBT groups, the format of delivery was not significant. Subjective sexual functioning measures revealed no main or interaction effects of treatment among the waitlist control group. At 12-month follow-up, 21% of those in group therapy and 15% of those in the bibliotherapy format reported successful intercourse. CBT also had significant effects on noncoital penetration behavior, with 79% of treated participants able to self-insert one finger at posttreatment compared with 36% of the control group participants.

Ravart, Trudel, Marchand, and Turgeon (1996) conducted an RCT that studied the efficacy of CBT for women with HSD compared to a waitlist control. The study recruited 74 women and their heterosexual male partners through two Montreal French

newspapers. The eligibility criteria were (1) female partner had presented a HSD diagnosis for at least 6 months; (2) the partners were between the ages of 20 and 55 years; (3) the partners had been living together for at least 1 year; (4) both partners expressed desire and motivation for treatment; (5) HSD was not associated with alcohol or drug abuse, conjugal violence, serious psychopathology or health problems, an ongoing sexual affair, sexual orientation, or deviation problems; and (6) both partners read and wrote in French. The study also excluded those diagnosed with the lifelong or global type of HSD. The treatment consisted of twelve 2-hour weekly sessions in a group of four to six couples. The sessions were conducted by a team of male and female clinical psychology graduate students and utilized the CBT program developed by Trudel and colleagues in 1996 (see Ravart et al., 1996). After every session, the couples were given homework assignments that included reading manuals. The therapists analyzed immediate and long-term causal factors related to HSD, psychoeducation for sexual information, communication skills training, problem-solving techniques for the couple, instruction on cognitive restructuring and rational emotive imagery, various intimacy-oriented and sensate-focus exercises, and sexual fantasy training.

The primary outcome measure was meeting criteria for HSD as per a semistructured clinical interview and measures from the Sexual History Form. Secondary outcome measures included the Multidimensional Sexuality Questionnaire (MSQ), the Negative Thoughts During Sex Questionnaire (NTDSQ), the Derogatis Sexual Fuctioning Inventory (DSFI), the Relationship Belief Inventory (RBI), and the Marital Attitude Survey (MAS). All participants were given these questionnaires pretreatment, posttreatment, and at 3-month follow-up intervals.

Results indicated that 68% of the women in the treatment group no longer met all diagnostic criteria for HSD, and 21% indicated their desire disorder was cured. This is in stark contrast to the 83% of women in the control group who still met criteria for HSD at the same time point. Additionally, after the waitlist control group received treatment, 79% no longer met the diagnostic criteria for HSD.

Trudel et al. (2001) devised an RCT to test the efficacy of CBT for women with HSD. The study tested an innovative, multimodal, short-term, cognitive–behavioral couple sex therapy program in a group format to treat HSD. The study recruited 77 couples through two Montreal newspapers. The eligibility criteria were (1) the partners had been living together for at least 1 year; (2) the subjects were between the ages of 20 and 55; (3) the subjects were French-speaking; (4) the subjects had no major psychological disorder as assessed by the Beck Depression Inventory, the IPAT Anxiety Scale, and a standardized clinical interview; and (5) HSD (as measured by the instruments in the next paragraph) had been present in the woman for at least 6 months. After an initial evaluation to confirm HSD diagnosis among the women, the couples were randomly assigned to either the treatment condition or a 12-week waitlist control group. The treatment consisted of twelve 2-hour weekly group couple sex therapy sessions that included four to six couples in each group. The sessions were held by a female and male therapist and followed the program laid out by Trudel et al. (see Ravart et al., 1996). Homework and reading were assigned at the end of each session. The techniques employed in the sessions were analyses of immediate and long-term factors related to HSD, sexual information, couple sexual intimacy exercises, sensate focus, communication skills training, emotional communication training, mutual reinforcement training, cognitive restructuring, and sexual fantasy training.

Variables included HSD symptoms as measured by a structured clinical interview, the Sexual History Inventory (SHI). Other outcome measures were the MSQ, the NTDSQ, the DSFI, the RBI, the MAS, the Sexual Arousability Inventory (SAI), the Sexual Behavior Inventory (SBI), the Dyadic Adjustment Scale (DAS), the Interpersonal Communication Questionnaire, and the Sexual Inventory (SII). Evaluations occurred for both groups pretreatment, posttreatment (or for the control group at the end of the 3-month waitlist and then posttreatment), a further 3 months posttreatment, and 1 year posttreatment.

HSD symptoms decreased from 100% pretreatment to 26% posttreatment, though they increased slightly to 36% at 3-month follow-up and remained at 36% at 1-year follow-up. As per self-report, 28% of the women at posttreatment considered themselves totally symptom free, and 31% at 3-month follow-up felt they were completely cured, which rose to 38% at 1-year follow-up. Treatment also significantly improved the quality of the couples' sexual and marital life, as measured by the SHI, the SBI (Pleasure Scale, Experimented Pleasure Scale, and Experience Scale), the SII, the MSQ's positive scales (Self-Esteem, Internal Sexual Control, Internal Sexual Awareness, Sexual Motivation, Sexual Assertion, External Sexual Control, and Sexual Satisfaction), the MSQ's negative scales (Sexual Preoccupation, Sexual Anxiety, Sexual Depression, Worries Over Sexual Image, and Fear of Sexuality), the DSFI, the NTDSQ, the RBI, the DAS, and the SAI. There was a positive effect in women on the MAS, the DSFI, and the SHI (Trudel et al., 2001).

10.2.1.2 Theory of disorder

According to cognitive–behavioral theory, cognitive schemas are responsible for the way in which individuals interpret and give meaning to different experiences (Avagianou, 2015; Beck, 1979; Turk, Meichenbaum, & Genest, 1983). In terms of male disorders, when a man experiences a negative sexual event, he develops schemas that generate negative thoughts (e.g., demand for performance, anticipation) and emotions (e.g., sadness, fear), thereby interfering with his sexual arousal (Avagianou, 2015). As a result, a negative thought pattern develops and maintains the individual's poor sexual performance. Some research suggests that men with sexual problems attribute negative sexual events to personal incompetence, thus interpreting those events as signs of personal failure, and this gives rise to beliefs of being different, powerless, and weak (Nobre & Pinot-Gouveia, 2009). Similarly, Quinta Gomes and Nobre (2012) investigated the role that early maladaptive schemas play in male sexual dysfunction in a community sample. The results revealed that men with sexual dysfunction had schemas that consisted of thoughts about being different, helpless, and incompetent. These findings suggest that the evaluation of an individual's core beliefs and a focus on cognitive techniques aimed at restructuring maladaptive beliefs and behavior patterns are integral in the treatment of sexual problems.

Bitzer and Brandenburg (2009) suggest that CBT may be useful for women as well. By looking at sexual dysfunction as a behavior learned by classical, operant conditioning and model learning, and the behavior sequence as characterized by specific sexual stimuli, responses, and contingencies, CBT can be useful in treating female sexual dysfunction. Sexual signals and bodily reactions may lead to positive or negative emotional consequences and experiences that are stored in the amygdala and hippocampus and that form an individual's sexual script. By helping individuals to become aware of these stimuli, the reactions they cause, and the consequences of the behavior, CBT can help

them learn what enhances and what inhibits sexual pleasure, learn to restructure their maladaptive beliefs, reduce avoidance behavior, and regulate the obsessional antisexual self-talk in which they are engaged (Ravart et al., 1996).

10.2.1.3 Summary

Based on David and Montgomery's (2011) framework for evaluating psychosocial interventions, CBT for male and female sexual dysfunctions can be considered a Category I psychotherapy. Support has been shown for both the treatment and the theory that underpins the treatment. Furthermore, empirical evidence validates the therapeutic package and the theory that supports the treatment.

10.3 Category III

10.3.1 Sex Therapy

Sex therapy is a psychotherapeutic treatment that focuses on immediate factors (e.g., fear of performance; concerns about one's own sexual adequacy, satisfaction, and marital relationship) within a couple's sexual interactions (Reynolds, 1977). Sex therapy consists of educational presentations, therapeutic discussions, and couples exercises (Frühauf et al., 2013; Price, Reynolds, Cohen, Anderson, & Schochet, 1981; Reynolds, 1977). Over a 2-week period, didactic presentations take place and the couple engages in exercises during daily meetings between the couple and a dual-sex therapy team (Frühauf et al., 2013; Weiner & Avery-Clark, 2014). In an effort to allow the couple to "think and feel" sexually without a preoccupation regarding performance, the couple is instructed to refrain from any sexual activity during the first few days of treatment and, instead, to engage in sensate-focus exercises (Weiner & Avery-Clark, 2014). In these exercises, couples caress areas of the body other than just the genitals or breasts, and, while erections commonly occur during these nondemanding pleasuring exercises, it is most important for the couple to focus on the fact that an erection is a natural physiological reaction that does not need to be achieved through a conscious effort (Weiner & Avery-Clark, 2014). Eventually, the couple is instructed to engage in sensate focus with genitals. During these exercises, the partners take turns providing manual stimulation to the genitals; the receiver provides verbal and nonverbal feedback concerning preferences in the positioning, pressure, direction, and rapidity of the caress. With an increase in communication, it is ascertained that both partners will begin to experience an increase in effective stimulation (Weiner & Avery-Clark, 2014).

Once a man is able to achieve a full erection during sensate exercises, the couple is instructed to engage in the "teasing technique," in which periods of manipulative play and cessation of penile stimulation are alternated (Weiner & Avery-Clark, 2014). This exercise is designed to demonstrate that there is no impairment in the erectile response to effective stimulation and to alleviate fear and anxiety regarding a loss of erection (Melnik, Soares, & Nasello, 2008; Weiner & Avery-Clark, 2014). After a week of these noncoital exercises, the couple is instructed to approach intercourse with spontaneous sexual activity with continued emphasis on mutual pleasuring rather than an erection or orgasm (Weiner & Avery-Clark, 2014).

10.3.1.1 Evidence for therapy

Two studies have examined the effectiveness of sex therapy for sexual dysfunction. Price et al. (1981) utilized a control group design to evaluate the effectiveness of a group treatment for erectile dysfunction in men without partners. The study involved 21 participants, recruited from the Human Sexuality Program at the Neuropsychiatric Unit at the University of Southern California. The men ranged from ages 25 to 61 years, with 19 being Caucasian, 1 African American, and 1 Hispanic. Eligibility consisted of (1) presenting a complaint of erectile dysfunction, (2) no steady sexual partner and not dating at a frequency greater than twice a week, and (3) a required fee of $120, which was repaid following the completion of the study. The outcome measures were the Minnesota Multiphasic Personality Inventory, the Background Information Questionnaire, the Negative Attitudes Toward Masturbation Scale, the Goals for Sex Therapy Scale for Males, the Erectile Difficulty Questionnaire, the Sexual Knowledge and Attitude Test, the California Inventory, the What's New Questionnaire, and the Participants Evaluation Questionnaire. Assessments were made pretreatment, 8 weeks posttreatment, and 6 months posttreatment.

Participants in the group treatment condition were seen by two therapists (one male and one female) on a weekly basis for eight 2-hour treatment sessions. Sessions began by collecting homework exercises and discussing progress, problems, and concerns regarding each homework assignment. Homework assignments consisted of increasing the man's awareness of his own body and the types of sensations he found pleasing. The content of each session was similar for the two treatment groups and included didactic presentations, audiovisual aids, homework exercises, and group discussions. Discussions typically focused on the male and female anatomy and physiology, myths about male and female sexuality and sex role stereotypes, and the importance of communication with partners about sexual difficulties and preferences. Throughout the progression of the treatment, group members practiced self-stimulation exercises in which they began to learn that they had the ability to gain, lose, and then regain erections. Additionally, men were instructed to practice communication skills on various topics with potential partners. Initially, men were instructed to refrain from sexual intercourse at all; however, at the conclusion, men were instructed to resume sexual contact at their own pace. Additionally, men were asked to prepare maintenance programs in which they identified the original problems that had prompted them to seek treatment, specific skills they had learned to mitigate these problems, possible problems that might arise in the future, and potential solutions.

Significant treatment group interactions were found for the total scores in the Erection Difficulty Questionnaire, the Goals for Sex Therapy Scale for Males, the Negative Attitudes Toward Masturbation Scale, and the Background Information Questionnaire, specifically concerning the level of satisfaction with one's overall sexual self-image. While the intervention group reported a decline in erection difficulties, this difference was not statistically significant, suggesting that the structured group treatment approach was less effective in producing specific behavioral changes in the frequency of erection difficulties. At 6-month follow-up, participants in the treatment conditions reported improved satisfaction with their changes in erectile functioning from pretreatment.

Kilmann and colleagues (1986) evaluated the effects of three group-treatment formats on 20 men and their partners with secondary erectile dysfunction. Following a

comprehensive medical and psychological screening, each couple was assigned to one of three treatment groups or one of two control groups, each of which consisted of twice-weekly sessions for a total of 20 hours. The treatment conditions included training in communication techniques, training in sexual techniques, and a combination treatment. One of the control groups was an attention placebo control and the other received no treatment. The no-treatment control group received sex education and treatment after a 5-week waitlist period. All three treatment groups experienced substantial gains in erectile functioning; therefore, no between-format differences were found to be statistically significant. Of the treated men, 81% reached the criterion of 80% experienced successful penetration and subsequent ejaculation at the 6-month follow-up.

While neither of these studies yielded significant differences favoring the sex group therapy over the control group, in a meta-analysis by Melnik and colleagues (2008), the active intervention group was favored, with 95% response for sex therapy and 0% for the waitlist group. In this view, the mean percentage of intercourse attempts per participant that were successful appeared to be greater for the sex group therapy compared to other types of group interventions, including rational emotive therapy, systematic desensitization, and Masters–Johnson modified sex therapy (Masters & Johnson, 1970).

10.3.1.2 Theory of disorder

The underlying rationale behind sex therapy is rooted in the belief that a redirection of the male's attention from sexual performance to sensual reception, combined with increased communication of preferences and reactions by both partners, will result in an erectile response that is an involuntary response to erotic stimulation. According to Masters and Johnson (1970), the dysfunction is typically maintained by a man's preoccupation with achieving an erection, which results in continued fear of erectile difficulty. In this view, such fear makes a man a "spectator" to his own sexual experience rather than an active participant, thereby inhibiting his ability to access the physical and psychological stimulation necessary for heightened sexual arousal and erections. This interference is frequently exacerbated by negative reactions from a sexual partner, which results in further detumescence and even greater performance anxiety and concerns.

10.3.1.3 Summary

Based on David and Montgomery's (2011) framework for evaluating psychosocial interventions, sex therapy for male and female sexual dysfunctions can be considered a Category III psychotherapy. While strong evidence exists for the theory of sex therapy, there is limited support for the therapeutic package.

10.4 Category IV

10.4.1 Behavioral Therapies

Behavioral therapy for sexual dysfunction consists of physical techniques that are utilized to help men develop skills necessary to improve sexual self-confidence (Cooper et al., 2015). Common behavioral techniques include the "stop–start technique," the "squeeze technique," and pelvic floor muscle rehabilitation (Cooper et al., 2015). The

stop–start technique involves stimulating the penis until the man feels the urge to ejaculate; once this feeling emerges, either the partner or the man discontinues the stimulation, thus allowing the urge to ejaculate to pass (Cooper et al., 2015). This technique is repeated multiple times in order for the man to learn how to recognize feelings of arousal in order to improve control over ejaculation (Cooper et al., 2015). Similarly, the squeeze technique teaches the man how to stimulate his penis until he feels the urge to ejaculate; he then squeezes the glans of the penis until the sensation passes. This is repeated before allowing ejaculation to occur (Cooper et al., 2015).

Behavior therapy can also be useful for sexual dysfunction in females as it views disorders as conditioned fear responses causing spasms or pain. Therefore, behavioral therapy can be useful by deconditioning these learned responses with gradual desensitization (Al-Sughayir, 2005).

10.4.1.1 Evidence for Therapy

10.4.1.1.1 Males

Cooper and colleagues (2015) conducted a meta-analysis comparing RCTs in adult men with premature ejaculation that evaluated behavioral therapy against a waitlist control or against another therapy. Additionally, studies were included if they compared behavioral therapies and a drug treatment against a drug treatment alone. Exclusion criteria consisted of (1) studies that had behavioral therapies in both arms, (2) theses and dissertations, and (3) non-English publications. Outcome measures included intravaginal ejaculatory latency time (IELT), other measures of ejaculatory latency, sexual satisfaction, control over ejaculation, relationship satisfaction, self-esteem, quality of life, treatment acceptability, and adverse events. Methodological quality was assessed using the Cochrane Risk of Bias assessment criteria and the completeness of the outcome data was considered low risk if the percentage of randomized participants excluded from the primary analysis was less than 30%. Selective reporting was considered low risk if IELT or ejaculatory latency was reported and if all outcomes referred to in the study methods were reported. Lastly, overall risk of bias for each study was classed as low or high risk depending on how the study was rated in three domains: allocation, concealment (blinding of outcome assessment), and completeness of outcome data. A total of 10 RCTs evaluating behavioral therapy for premature ejaculation were included.

The included studies assessed various types of behavioral therapy either individually or in combination, including the squeeze and stop–start techniques, stop–start aided by a stimulation device, education on sensuality and movement, sensate focus, and pelvic floor muscle rehabilitation. In total, four RCTs assessed non-IELT outcomes for behavioral therapies versus waitlist control. While one RCT significantly favored behavioral therapy (de Carufel & Trudel, 2006), the others were unclear as to whether there was a significant difference between the groups (Jern, 2014; Trudel & Proulx, 1987; Van Lankveld, Leusink, Van Diest, Gij, & Slob, 2009). De Carufel and Trudel (1987) found significant improvements in male perception of duration of intercourse and couples' sexual satisfaction with participants in the treatment arm compared with the waitlist control. Additionally, Trudel and Proulx (1987) reported a significant increase from baseline in sexual satisfaction for all three behavioral therapy groups (self-help book, self-help book plus therapist phone contact, and sexual therapy) with better results for self-help plus phone contact versus self-help alone. However, there were no data reported for the waitlist group. Van Lankveld and colleagues (2009) found that sexual desire improved

significantly more for participants in the behavioral treatment arm than the waitlist; however, between-group differences were not found in terms of improvement from baseline for sexual satisfaction, whereas self-confidence showed no significant difference either between groups or from baseline. Jern (2014) conducted an RCT using the stop–start technique aided by a handheld stimulation device, and the results revealed no significant improvement over waitlist or from baseline to posttreatment (via a composite score for ejaculatory latency, control, and relationship problems). Despite this, a significant improvement was found from baseline to 6-month follow-up; however, by this point, all participants had received treatment.

10.4.1.1.2 Females

Al-Sughayir (2005) conducted an RCT comparing the efficacy of hypnotherapy and behavior therapy for women with vaginismus. The trial recruited 36 women from the outpatient psychiatry clinic at King Abdulaziz University Hospital in Riyadh, Saudi Arabia. The inclusion criterion was meeting the criteria for vaginismus as per the DSM-IV (American Psychiatric Association, 1994). The hypnotherapy group received sessions once a week (45–69 minutes per session) until symptoms abated. Individuals in this arm were encouraged to practice self-hypnosis in between sessions. During the sessions, a trance was induced using standard eye fixation and arm levitation techniques, and faulty perceptions and cognitions were addressed. The behavioral therapy group also had sessions once a week (45–60 minutes per session) until symptoms abated. After the initial interview, the women in this group were seen with their spouses. During sessions, they were taught to recognize and reduce the woman's performance anxiety and increase pleasure and confidence, and were encouraged to practice muscle relaxation and desensitization techniques.

The outcome measures were the wife's sex-related anxiety, the wife's sexual satisfaction and the husband's sexual satisfaction as measured by personal and marital histories, an adapted version of the Brief Index of Sexual Functioning for Women, and the Brief Sexual Function Inventory.

While both treatments were significantly effective in reducing symptoms of the wife's sex-related anxiety, the wife's sexual satisfaction, and the husband's sexual satisfaction, between-group comparisons showed that hypnotherapy performed better than behavior therapy on all three variables. Additionally, the number of sessions needed to reduce symptomatology was significantly less in the hypnotherapy group.

10.4.1.2 Theory of Disorder

Behavior therapy views sexual dysfunction in terms of conditioned fear responses. For example, in women with vaginismus, behavior therapy views the conditioned fear response as the spasm of the vagina. Therefore, in order to properly treat this disorder, one would need to decondition the learned response through gradual desensitization.

10.4.1.3 Summary

Based on David and Montgomery's (2011) framework, behavior therapy for male and female sexual dysfunctions can be considered a Category IV psychotherapy. Empirical evidence suggests that there is limited support for both the treatment and the theory that underpins the treatment.

10.5 Category VIII

10.5.1 Psychoeducation

In the 1980s, psychoeducation was developed as a composite of numerous therapeutic elements to aid patients and their families in developing a precursory understanding of their illness before deciding on a long-term treatment (Bauml, Frobose, Kraemer, Rentrop, & Pitschel-Walz, 2006). Since then, psychoeducation has become a stand-alone treatment option with a focus on didactic skillful communication of key information to help empower patients and their families to understand and accept their illness in a successful manner (Bauml et al., 2006). In this view, psychoeducation for sexual dysfunction focuses on disseminating information about physiological and psychological changes that occur in the sexual response (Frühauf et al., 2013).

10.5.1.1 Evidence for Therapy

Goldman and Carroll (1990) studied the effect of a psychoeducational intervention as an adjunct to treatment of erectile dysfunction in couples between the ages of 55 and 75. The study recruited 20 couples that presented to a multidisciplinary center for erectile dysfunction. The inclusion criteria were that the couples (1) had to be heterosexual, (2) had to have been in an ongoing relation for at least 6 months, (3) had to have experienced secondary erectile dysfunction, and (4) had to have exhibited no major psychiatric disorder or severe marital distress at initial evaluation. Assessments were conducted pre- and posttreatment, and a qualitative interview was also conducted during the follow-up evaluation. Treatment consisted of an educational workshop, in which the goals were to increase the couple's knowledge about the sexual response cycle and normal changes associated with the cycle; to increase comfort levels with regard to discussion of sexuality; to increase satisfaction with expression of sexuality; and to increase the couple's acceptance of their sexual difficulties. Compared to the control condition, the treatment group had no statistically significant changes in scores from pre- to posttreatment on the Aging Sexuality Knowledge and Attitudes Scale, the Sexual Interaction Inventory, or measures of mean pleasure.

10.5.1.2 Theory of Disorder

The underlying rationale behind psychoeducation is that a basic level of understanding of their illness will enhance patients' empowerment by decreasing anxiety through providing knowledge (Bauml et al., 2006). By gaining this understanding, patients and their families tackle their illness in the most optimal way, as an established understanding of the illness allows patients to begin to have insight into their ailment, thus improving compliance and long-term successful cooperation with treatment (Bauml et al., 2006).

10.5.1.3 Summary

Based on David and Montgomery's (2011) framework for evaluating psychosocial interventions, psychoeducational therapy for male and female sexual dysfunctions can be considered a Category VIII psychotherapy. Despite invalidating evidence for the therapeutic package, there is limited support for the theory that underpins psychoeducation.

10.6 Other Therapies

Other therapies have been put forth as treatments for sexual dysfunction. They were not included in this review due to the fact that none have enough research yet to back up either the theory or the therapeutic package. For example, group mindfulness-based therapy has been researched as a therapeutic package in an RCT by Brotto and Basson (2014). However, the theoretical base for it has not garnered enough research. This type of theoretical model does seem convincing, as mindfulness-based therapies have been increasingly being used and valued, with mindfulness-based stress reduction having shown benefits for medical, psychological, and behavioral ailments as well as physical parameters and epigenetic changes. In addition, mindfulness-based cognitive therapy has been shown to alleviate anxiety and depression and prevent depression relapse in preliminary studies (Brotto & Basson, 2014). The proposed therapy teaches participants to attend to and accept the present moment and sexual stimuli, thus lessening the tendency to self-criticize and to follow distracting thoughts, which in turn not only increases awareness of sex responses but also lessens participants' own judgments that they are insufficient or substandard. This is all done in four 90-minute group sessions that include mindfulness meditation, cognitive therapy, and psychoeducation. Brotto and Basson's (2014) study found that, out of 117 women assigned to immediate treatment or delayed treatment, the treatment significantly improved sexual desire, sexual arousal, lubrication, sexual satisfaction, and overall sexual functioning. This type of theoretical model and therapeutic package indicates promising results, and more research should be done in order for it to be further utilized as a psychotherapy for women with sexual dysfunction.

Another therapy that was not included in its own right in the preceding sections but that has gained some attention is hypnotherapy. Hypnotherapy has preliminarily proved promising in reducing the symptomatology of female sexual dysfunction (Al-Sughayir, 2005; see Section 10.4.1.1.2). However, not enough research has been done on the psychological theory behind hypnotherapy to warrant it being considered a strong evidence-based psychotherapy to treat sexual dysfunction. Al-Sughayir's study pointed out that hypnotherapy can achieve rapid and emotional cognitive and attitudinal positive reconstructs; however, further research needs to be done.

The final type of type of therapy for women that needs more research is couples emotionally focused therapy (EFT). MacPhee, Johnson, and Van der Veer (1995) conducted a study with 49 couples in which the woman had inhibited sexual desire. The couples were randomly assigned to either EFT or a waitlist control. There were very modest differences between the treatment and control groups, although EFT was found to significantly increase one measure of sexual desire and decrease the level of depressive symptomatology.

10.7 Implications for Research

While there are several strong theoretical bases in the psychotherapies mentioned in this chapter, only CBT garners the title of a Category I psychotherapy according to David and Montgomery's (2011) framework. While sex therapy has a strong theoretical

base, the therapeutic package derived from that theory has not been well supported by research.

It may be important for future studies to first conduct qualitative research to delve into the issues that are most pertinent to male and female sexual dysfunction. Qualitative research will enable the theoretical bases to be made stronger, and it will be possible to design the therapeutic package such that it can be further tailored to each specific disorder.

Additionally, all future research studies should ensure that they gather all relevant information pertinent to why the anxiety or sexual dysfunction is occurring in order to help eliminate confounding variables. While some of the studies achieved this, not all did. Furthermore, most studies included a waitlist control, and it would be useful to have more studies comparing various psychotherapies so that the ones that are most efficacious can be focused on and improved upon.

Another area that lacks research about these sexual dysfunction disorders is how to adapt the various treatments to different cultures. Different cultures have different attitudes toward sexual activity, and ensuring that these treatments can be adjusted for each of them is key for research moving forward.

Lastly, much of the research is only inclusive of heterosexual males and females. Future research should conduct studies involving both heterosexual and homosexual males and females in order to be more comprehensive, inclusive, and generalizable.

10.8 Implications for Practice

The largest barriers to the treatment of sexual dysfunction disorders are the shame that is associated with having the disorder and the difficulty of the actual therapy and treatment itself. Many of these treatments involve issues that are uncomfortable for large numbers of people, and many of them involve people doing exercises they would not normally do. Therefore, it is vital to employ an extreme amount of understanding when working with people with these disorders.

Additionally, the majority of the studies have only focused on Western cultures. It is very important for multicultural competency to be realized for the existing treatments. Ensuring that all of the treatments are available in various languages and formats is crucial to ensuring that the treatments can be implemented in practice.

Another implication for practice is that, while psychologists can easily be trained in CBT and some of these other treatments, this is not the case for all treatments, and moreover it is sometimes the case that communities do not have access to both male and female therapists. As people with these disorders already feel shameful when they come forward, it is important that psychologists be available and that we find ways to reach out to individuals with these disorders. Telehealth may be useful to that end, as it not only eliminates some of the shame involved in going into a clinic but also eliminates the problem of trained professionals not being available due to location.

The majority of the proposed psychotherapies are manualized and short term. This is a positive implication for practice, first because more people can be trained in utilizing them and second because they are cost-effective as they are short-term therapies, making them less stressful on clinic resources.

10.9 Conclusions

There are few evidence-based interventions for sexual dysfunctions that are well developed, with only CBT classified as Category I. The evidence-based therapies for sexual dysfunctions are still in their infancy. However, this could be advantageous, as new psychotherapies and theories developed for sexual dysfunctions can take into account all of the critiques of existing models, ensuring that they are developed in an empirically sound way.

References

Al-Sughayir, M. A. (2005). Vaginismus treatment: Hypnotherapy versus behavior therapy. *Neurosciences (Riyadh, Saudi Arabia)*, *10*(2), 163–167.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders (*4th ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.

Andersson, E., Walén, C., Hallberg, J., Paxling, B., Dahlin, M., Almlöv, J., … Andersson, G. (2011). A randomized controlled trial of guided internet-delivered cognitive behavioral therapy for erectile dysfunction. *Journal of Sexual Medicine*, *8*(10), 2800–2809. doi:10.1111/j.1743-6109.2011.02391.x

Avagianou, P.-A. (2015). Cognitive behavioral therapy in sexual dysfunction. In M. Viigimaa, C. Vlachopoulos, & M. Doumas (Eds.), *Erectile dysfunction in hypertension and cardiovascular disease: A guide for clinicians* (pp. 205–213). New York, NY: Springer.

Bauml, J., Frobose, T., Kraemer, S., Rentrop, M., & Putschel-Walz, G. (2006). Psychoeducation: A basic psychotherapeutic intervention for patients with schizophrenia and their families. *Schizophrenia Bulletin*, *32*(1), 1–9. doi:10.1093/schbul/sbl017

Beck, A. T. (1979). *Cognitive therapy of depression*. New York, NY: Guilford Press.

Beck, A. T. (1996). Beyond belief: A theory of modes, personality, and psychopathology. In P. M. Salkovskis (Ed.), *Frontiers of cognitive therapy* (pp. 1–25). New York, NY: Guilford Press.

Bhavsar, V., & Bhugra, D. (2013). Cultural factors and sexual dysfunction in clinical practice. *Advances in Psychiatric Treatment*, *19*(2), 144–152. doi:10.1192/apt.bp.111.009852

Bitzer, J., & Brandenburg, U. (2009). Psychotherapeutic interventions for female sexual dysfunction. *Maturitas*, *63*(2), 160–163. doi:10.1016/j.maturitas.2009.02.012

Brotto, L. A., & Basson, R. (2014). Group mindfulness-based therapy significantly improves sexual desire in women. *Behaviour Research and Therapy, 57*, 43–54. doi:10.1016/j.brat.2014.04.001

Cooper, K., Martyn-St James, M., Kaltenthaler, E., Dickinson, K., Cantrell, A., Wylie, K., … Hood, C. (2015). Behavioral therapies for management of premature ejaculation: A systematic review. *Sexual Medicine*, *3*(3), 174–188. doi:10.1002/sm2.65

David, D., & Montgomery, G. H. (2011). The scientific status of psychotherapies: A new evaluative framework for evidence-based psychosocial interventions. *Clinical Psychology: Science and Practice*, *18*(2), 89–99. doi:10.1111/j.1468-2850.2011.01239.x

de Carufel, F., & Trudel, G. (2006). Effects of a new functional-sexological treatment for premature ejaculation. *Journal of Sex & Marital Therapy*, *32*(2), 97–114.

Frühauf, S., Gerger, H., Schmidt, H. M., Munder, T., & Barth, J. (2013). Efficacy of psychological interventions for sexual dysfunction: A systematic review and meta-analysis. *Archives of Sexual Behavior*, *42*(6), 915–933.

Goldman, A., & Carroll, J. L. (1990). Educational intervention as an adjunct to treatment of erectile dysfunction in older couples. *Journal of Sex & Marital Therapy*, *16*(3), 127–141. doi:10.1080/00926239008405259

Jern, P. (2014). Evaluation of a behavioral treatment intervention for premature ejaculation using a handheld stimulating device. *Journal of Sex & Marital Therapy*, *40*(5), 358–366. doi:10.1080/0092623X.2013.857376

Kilmann, P. R., Mills, K. H., Caid, C., Davidson, E., Bella, B., & Milan, R. J. (1986). Treatment of secondary orgasmic dysfunction: An outcome study. *Archives of Sexual Behavior*, *15*, 211–229. doi:10.1007/bf01542413

Laumann, E. O., Paik, A., & Rosen, R. C. (1999). Sexual dysfunction in the United States. *JAMA*, *281*(6), 537. doi:10.1001/jama.281.6.537

MacPhee, D. C., Johnson, S. M., & Van der Veer, M. M. (1995). Low sexual desire in women: the effects of marital therapy. *Journal of Sex & Marital Therapy*, *21*(3), 159–182.

Masters, W. H., & Johnson, V. E. (1970). *Human sexual inadequacy*. New York, NY: Little, Brown.

McCabe, M., Price, E., Piterman, L., & Lording, D. (2008). Evaluation of an internet- based psychological intervention for the treatment of erectile dysfunction. *International Journal of Impotence Research*, *20*(3), 324–330. doi:10.1038/ijir.2008.3

Melnik, T., Soares, B. G., & Nasello, A. G. (2008). The effectiveness of psychological interventions for the treatment of erectile dysfunction: Systematic review and meta-analysis, including comparisons to sildenafil treatment, intracavernosal injection, and vacuum devices. *Journal of Sexual Medicine*, *5*(11), 2562–2574. doi:10.1111/j.1743-6109.2008.00872.x

Nobre, P. J., & Pinto-Gouveia, J. (2009). Questionnaire of cognitive schema activation in sexual context: A measure to assess cognitive schemas activated in unsuccessful sexual situations. *Journal of Sex Research*, *46*(5), 425–437. doi:10.1080/00224490902792616

Price, S. C., Reynolds, B. S., Cohen, B. D., Anderson, A. J., & Schochet, B. V. (1981). Group treatment of erectile dysfunction for men without partners: A controlled evaluation. *Archives of Sexual Behavior*, *10*(3), 253–268. doi:10.1007/bf01543078

Quinta Gomes, A. L., & Nobre, P. (2012). Early maladaptive schemas and sexual dysfunction in men. *Archives of Sexual Behavior*, *41*(1), 311–320. doi:10.1007/s10508-011-9853-y

Ravart, M., Trudel, G., Marchand, A., & Turgeon, L. (1996). The efficacy of a cognitive behavioural treatment model for hypoactive sexual desire disorder: An outcome study. *Canadian Journal of Human Sexuality*, *5*(4), 279–293.

Reynolds, B. S. (1977). Psychological treatment models and outcome results for erectile dysfunction: A critical review. *Psychological Bulletin*, *84*(6), 1218–1238. doi:10.1037/0033-2909.84.6.1218

Simons, J. S., & Carey, M. P. (2001). Prevalence of sexual dysfunctions: Results from a decade of research. *Archives of Sexual Behavior*, *30*(2), 177–219. doi:10.1023/a:1002729318254

Trudel, G., Marchand, A., Ravart, M., Aubin, S., Turgeon, L., & Fortier, P. (2001). The effect of a cognitive-behavioral group treatment program on hypoactive sexual desire in women. *Sexual and Relationship Therapy*, *16*(2), 145–164. doi:10.1080/14681990120040078

Trudel, G., & Proulx, S. (1987). Treatment of premature ejaculation by bibliotherapy: An experimental study. *Journal of Sex & Marital Therapy*, *2*, 163–167.

Turk, D. C., Meichenbaum, D., & Genest, M. (1983). *Pain and behavioral medicine: A cognitive-behavioral perspective*. New York, NY: Guilford Press.

Van Lankveld, J. J. D. M., Leusink, P., Van Diest, S., Gijs, L., & Slob, A. K. (2009). Internet-based brief sex therapy for heterosexual men with sexual dysfunctions: A randomized controlled pilot trial. *Journal of Sexual Medicine*, *6*, 2224–2236. doi:10.1111/j.1743-6109.2009.01321.x

Van Lankveld, J. J. D. M., ter Kuile, M. M., de Groot, H. E., Melles, R., Nefs, J., & Zandbergen, M. (2006). Cognitive-behavioral therapy for women with lifelong vaginismus: A randomized waiting-list controlled trial of efficacy. *Journal of Consulting and Clinical Psychology*, *74*(1), 168–178. doi:10.1037/0022-006x.74.1.168

Weiner, L., & Avery-Clark, C. (2014). Sensate focus: Clarifying the Masters and Johnson's model. *Sexual and Relationship Therapy*, *29*(3), 307–319. doi:10.1080/14681994.2014.892920

11

The Psychological Treatment of Psychopathy

Theory and Research

Lisa K. Hecht,[1] Robert D. Latzman,[1] and Scott O. Lilienfeld[2,3]

[1] *Department of Psychology, Georgia State University, Atlanta, USA*

[2] *Department of Psychology, Emory University, Atlanta, USA*

[3] *Department of Psychology, University of Melbourne, Melbourne, Australia*

The psychological treatment of psychopathy is rife with controversy and conceptual disagreement. Indeed, researchers have yet to come to a clear consensus regarding the definition of psychopathy, including the boundaries of the construct and how best to assess its features (Lilienfeld, 1998; Lilienfeld, Watts, Smith, Berg, & Latzman, 2015). In the absence of an agreed-upon conceptualization, the literature on the treatment of psychopathy is abundant with competing approaches to assessing and diagnosing psychopathy, treatment methods, and outcome measures. Moreover, although a variety of treatment approaches have been described in the literature, few have been based on well-articulated etiological theories of psychopathy, and most involve nonexplicit or theoretically questionable mechanisms of change (Salekin, 2002; Salekin, Worley, & Grimes, 2010). As such, the scientific rigor of treatment designs and of the assessment of treatment efficacy is limited. This is not to say that the scientific treatment of psychopathy is unattainable, as treatment programs that are grounded firmly in scientifically informed etiological theories have evidenced at least some success (Andrews & Bonta, 2010; Antonowicz & Ross, 1994). Nevertheless, the scientific status of the treatment of psychopathy hinges largely on the development of an agreed-upon conceptualization of the construct, from which theoretically sound treatments can be advanced and rigorously tested.

The purpose of this chapter is to investigate the evidentiary basis of the treatment of psychopathy, in terms of both the effectiveness and the theoretical bases of alternative treatment approaches. First, before discussing these approaches, a brief discussion of psychopathy's differing conceptions over time is needed. These conceptual differences concerning the definition and measurement of psychopathy are especially pertinent to treatment evaluations, as differences in the operationalization of psychopathy bear directly on evaluations of efficacy. Second, we review long-standing negative views regarding psychopathy's treatability, as well as features of psychopathy that present potential barriers to its treatability. Third, we summarize methodological differences

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

among treatment investigations, some of which preclude clear-cut conclusions regarding psychopathy's amenability to treatment. Fourth, we review approaches to the treatment of psychopathy as well as empirical evidence for such approaches. Fifth and finally, we discuss the clinical and research implications of these findings. The present chapter aims to show that, in light of methodological issues rampant in this literature, compelling evidentiary support for specific approaches to the treatment of psychopathy is lacking. Nevertheless, several promising avenues to the scientific treatment of psychopathy exist (Lösel, 1998; Salekin, 2002; Salekin et al., 2010).

11.1 Conceptualizing and Measuring Psychopathy

In stark contrast to most domains of psychopathology research, marked disagreement exists in the field of psychopathy regarding the fundamental conceptualization and operationalization of the construct. This lack of consensus has persisted for decades, even in light of promising advances in the field concerning psychopathy's assessment and diagnosis (Lilienfeld, 1998; Lilienfeld et al., 2015), its neuroscientific correlates (Glenn & Raine, 2013), and the genetic and environmental architecture associated with psychopathy (Latzman, Patrick, Freeman, Schapiro, & Hopkins, 2017; Skeem, Polaschek, Patrick, & Lilienfeld, 2011). Ideally, interventions for psychopathy, indeed for all forms of psychopathology, would be designed based on a scientifically rigorous understanding of the construct. Nevertheless, although progress has been made, this goal has been largely unobtainable given that the field lacks a clear consensus regarding what psychopathy is.

The current view of psychopathy is based on research and theory developed over the past century. Hervey Cleckley's clinical description of psychopathy, first published the early 1940s, is almost certainly the most influential. Cleckley (1941/1988) offered a clear definition of psychopathy and its interpersonal, affective, and behavioral attributes, and vivified these features through descriptive case studies. Through his work with psychiatric patients, Cleckley delineated 16 criteria that he believed captured the essence of the prototypical psychopath. According to Cleckley, psychopathy is characterized by superficial charm, guiltlessness, callousness, dishonesty, egocentricity, lack of emotions such as love and anxiety, lack of insight, poor judgment, and failure to follow a coherent life plan. Although not explicitly linked to trait models of personality, Cleckley's criteria are clearly related to personality dispositions. An important aspect of Cleckley's conceptualization is the weight placed on seemingly positive social adjustment. According to Cleckley, the prototypical psychopath exhibits a chameleon-like nature. On the surface, he or she is charming and makes a positive impression on others, yet on the inside, he or she is deeply affectively impoverished. Moreover, although Cleckley recognized unmotivated antisocial behavior as associated with psychopathy, he did not regard antisocial and criminal behavior to be a necessary feature of the construct (Cleckley, 1941/1988). Although it is not clear to what extent Cleckley's descriptions of psychopathy directly influenced early editions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM; see Millon, 2011), a largely personality-based conceptualization of psychopathy appeared in the first version of the DSM as "sociopathic personality disturbance" (American Psychiatric Association, 1952, p. 38), which included individuals previously considered to have a "psychopathic personality" (American Psychiatric Association,

1952, p. 38). The description of "antisocial personality" appeared in the second version of the DSM (American Psychiatric Association, 1968, p. 43). This description was somewhat more in line with Cleckley's (1941/1988) psychopathy concept, and emphasized selfishness, irresponsibility, impulsivity, lack of loyalty, callousness, guiltnessness, failure to learn from punishment, and low frustration tolerance. Importantly, although recognized as a potential behavioral indicator, antisocial behavior was considered neither necessary nor sufficient to justify a diagnosis in the DSM-II (American Psychiatric Association, 1968).

Although Cleckley's views remain influential to this day, the field has drifted from his conceptualization. Cleckley provided no specific methods for a reliable diagnosis of psychopathy, and the lack of standardization of his criteria impeded early scientific progress. Moreover, the DSM-II's diagnoses of most mental disorders were notoriously unreliable, as classifications were based on clinical impressions of patients without explicit guidelines (Spitzer, Williams, & Skodol, 1980). With the field in need of reliable methods by which to assess all disorders, including psychopathy, competing conceptualizations of psychopathy placed a greater focus on indexing the behaviors rather than the personality features associated with the construct (Cloninger, 1978; Robins, 1966). This behavior-based approach was influenced primarily by the work of Robins (1966), who systematically examined over 500 individuals who, as juvenile delinquents 30 years prior, had received services at a child guidance clinic. Robins was interested in "sociopathic" personality disorder, and, although she noted difficulties in both terminology and definition, she considered sociopathic personality to be closely related to Cleckley's (1941/1988) concept of psychopathy. She advanced a list of 19 items specific to sociopathic personality, many with very specific requirements for assessment. Her list included several of Cleckley's psychopathy items as well as additional behavioral items such as drug and alcohol use and suicide attempts, as it was believed that these features could be more reliably assessed than affective criteria (e.g., lack of remorse). As a result of this work, and follow-up work by Feighner et al. (1972), the diagnosis of the condition shifted to include specific and explicit criteria. An illustration of this shift was the virtually wholesale removal of the personality-based descriptions from subsequent versions of the DSM. The DSM-III (American Psychiatric Association, 1980) included a primarily behavioral-based description of antisocial personality disorder (ASPD), which relied heavily on a history of readily observable antisocial behaviors that originated in childhood and persisted into adulthood (e.g., theft, vandalism, cruelty to animals). With the DSM-III-R (American Psychiatric Association, 1987) came the addition of the criterion "lacks remorse," and, although this criterion alluded to some psychopathic personality traits, the description remained behavioral overall. Although the DSM-IV field trial for ASPD revealed admittedly mixed evidence for the incremental contribution of personality-based criteria in the assessment of psychopathy and ASPD (Widiger et al., 1996), such features were not incorporated into the DSM-IV description of ASPD (American Psychiatric Association, 1994), which has remained identical in the DSM-5 (American Psychiatric Association, 2013). Nevertheless, psychopathy now appears in the DSM-5 but only as a specifier, termed "with psychopathic features" to denote a distinct variant of the diagnosis of ASPD in Section III, "Emerging Measures and Models." This specifier emphasizes the boldness features of psychopathy—namely, those linked to low levels of social fear and high emotional resilience (Anderson, Sellbom, Wygant, Salekin, & Krueger, 2014). This reemergence of the psychopathy construct in the

current version of the DSM represents a long awaited step forward; yet, the ASPD diagnosis, ostensibly the closest counterpart to psychopathy in the manual, is still conceptually and empirically wedded to antisocial behaviors. Although the terms "antisocial personality," "sociopathy," and "psychopathy" are theoretically related and are frequently used interchangeably, they are empirically and conceptually separable constructs (e.g., Walsh & Wu, 2008). Moreover, the many operationalizations of these constructs place varying emphases on antisocial behaviors versus personality features. Thus, when evaluating psychopathy treatment research, it is important to consider whether the operationalization of the construct is primarily based on behavior or personality.

An important development in the field of psychopathy, and particularly in the standardization of diagnostic criteria, began in the 1980s with the research program of Canadian psychologist Robert Hare, who initiated the construction of a reliable and construct-valid measure of psychopathy. The Psychopathy Checklist (PCL; Hare, 1980) initially relied heavily on Cleckley's criteria, but, through an iterative process of test construction, features related to positive social adjustment were largely eliminated, and additional features such as antisocial and criminal behaviors were included. After undergoing revisions, the PCL became the Psychopathy Checklist–Revised (PCL-R), now the most commonly used and extensively validated measure of psychopathy (Hare & Neumann, 2008). The PCL-R has been adapted and extended downwardly to adolescents (the Psychopathy Checklist: Youth Version, or PCL: YV; Forth, Kosson, & Hare, 2003) and to nonclinical samples (the Psychopathy Checklist: Screening Version, or PCL: SV; Hart, Cox, & Hare, 1995). Although the PCL-R was designed to index psychopathy as a single, total score (Hare & Neumann, 2008), research has increasingly focused on two broad factor-analytically-derived dimensions (Harpur, Hare, & Hakstian, 1989). Whereas Factor I assesses the core interpersonal and affective features of psychopathy, including grandiose sense of self-worth, lack of guilt, and callousness, Factor II assesses an antisocial and impulsive lifestyle (Hare, 1991/2003). More fine-grained factor models of the PCL-R have also been developed, such as three-factor (Cooke & Michie, 2001) and four-factor (Hare, 1991/2003) models. The PCL and its derivatives have contributed profoundly to the field of psychopathy research. Yet, several have criticized the field's heavy reliance on a single measure, observing that the PCL-R is an assessment tool that should not be substituted as a theoretical model for psychopathy (e.g., Logan, Rypdal, & Hoff, 2012; Skeem & Cooke, 2010). Moreover, the PCL tools place a significant emphasis on antisocial behavior, and the psychopathy construct assessed by these measures has drifted from Cleckley's 16 criteria.

The core features and structure of psychopathy continue to be debated. Specifically, consensus has yet to be reached on whether psychopathy is unidimensional or multidimensional at a higher-order level (e.g., Neumann, Hare, & Newman, 2007; Neumann, Malterer, & Newman, 2008). Further, opinions differ regarding the specific features of psychopathy. For example, one actively debated issue is whether certain adaptive features, such as boldness, are relevant to psychopathy (e.g., Lilienfeld et al., 2012; Lilienfeld et al., 2016; Lynam & Miller, 2012; Miller & Lynam, 2012; Patrick, Venables, & Drislane, 2013). Potentially most relevant when considering the evaluation of treatments, disagreement persists regarding whether antisocial behaviors are an integral feature of psychopathy (Hare & Neumann, 2010) or are simply correlates or sequelae of psychopathy (Skeem & Cooke, 2010).

Without an agreed-upon operationalization of psychopathy, prevalence rates are difficult to estimate. Moreover, prevalence rates assume a dichotomous cut point between "psychopath" and "nonpsychopath." Regardless of how psychopathy is conceptualized, taxometric research suggests psychopathy is dimensional rather than categorical, meaning that no clear diagnostic cut point exists (e.g., Edens, Marcus, Lilienfeld, & Poythress, 2006; Marcus, John, & Edens, 2004; cf. Harris, Rice, & Quinsey, 1994). Nonetheless, current estimates of the prevalence of psychopathy in adults are based largely on scores from the PCL-R or its variants. For example, a US study reported that 1.2% of a sample scored within 13 and 24 on the PCL: SV, an indication of "potential psychopathy" (Neumann & Hare, 2008). Similarly, a British study reported a community prevalence rate of 0.6% scoring higher than 13 on the PCL: SV (Coid et al., 2009). Prevalence rates in incarcerated adults are significantly higher; for example, high rates of ASPD (50–80%) have been observed in prison populations, with 20% of these individuals meeting criteria for psychopathy based on the PCL-R (Hare, 1998).

11.2 Is Psychopathy Treatable? A History of Negative Opinion

In light of the aforementioned debates regarding the conceptualization and definition of psychopathy, conclusions regarding psychopathy's amenability to treatment are tenuous. Traditionally, popular clinical opinion holds that psychopathy is untreatable, a belief similarly rampant among researchers for many years. Salekin (2002) aptly summarized the state of the field when he noted, "Clinical lore has led clinicians and researchers to believe that psychopathy is, essentially, an untreatable syndrome" (p. 79). Indeed, after years of working with individuals with psychopathy, Cleckley highlighted the elusiveness of successful treatment for psychopathy: "We do not at present have any kind of psychotherapy that can be relied upon to change the psychopath fundamentally" (p. 478). Later reviewers echoed this pessimistic sentiment (e.g., Suedfeld & Landon, 1978) and others have similarly questioned the treatability of psychopathy (e.g., Lösel, 1998), although many have noted that the lack of empirical support for specific treatment approaches does not necessarily provide evidence that psychopathy is untreatable (Blackburn, 1993; D'Silva, Duggan, & McCarthy, 2004; Lösel, 1998).

The challenges of conducting psychotherapy with individuals high in psychopathy have been documented for years (e.g., Doren, 1987; Eissler, 1949; Yochelson & Samenow, 1977). For psychotherapy to be effective, the patient should be able to form emotional connections with others, including the therapist; however, the classical psychopath is largely or entirely incapable of such interpersonal connectedness (e.g., Cleckley, 1941/1988; Hare, 1996; Yochelson & Samenow, 1977). Moreover, psychopaths' tendency to engage in pathological lying, manipulation, and deception, as well as their proneness to boredom and resistance to accepting responsibility for their actions, all present significant barriers to treatment progress (Lösel, 1998). Further, individuals with psychopathy are less motivated than their nonpsychopathic counterparts, expending less effort and time spent in treatment (e.g., Ogloff, Wong, & Greenwood, 1990). Moreover, unlike individuals with most forms of psychopathology, individuals with elevated scores on measures of psychopathy typically do not report subjective distress that reflects shame or guilt, or recognize their own difficulties, which may decrease their motivation to continue with treatment (Ogloff et al., 1990; Reid & Gacono, 2000).

Clearly, the nature of psychopathy makes psychotherapy difficult. Such obstacles undoubtedly fuel popular opinion that psychopathy is, at its core, untreatable. Nevertheless, potential difficulties in the therapeutic process do not render psychopathy untreatable. Rather, this question rests on the empirical status of treatment investigations. Indeed, despite widespread negative opinions regarding psychopathy's treatability, researchers have attempted to investigate its treatment response. Still, in comparison with other domains of treatment research, there is a striking dearth of scientific literature examining the therapeutic response of individuals with psychopathy. Further, and perhaps more problematic than the small quantity of empirical investigations, there is the large number of methodological issues in this limited literature. As such, the overall scientific quality of this literature is wanting, and informative meta-analyses and narrative reviews are difficult to conduct and interpret. Given the various issues enumerated in the following sections, it is challenging to evaluate the scientific literature on the treatment of psychopathy within extent models of evidentiary support (e.g., David & Montgomery, 2011).

11.3 Methodological Issues in Treatment Investigations

As noted, few evaluations of the treatment of psychopathy exist. Even more scarce are scientifically sound, controlled examinations of treatments designed specifically to target psychopathy (Lösel, 1998). Although a growing literature supports the effectiveness of treatments designed to reduce future violence in high-risk offenders (e.g., Di Placido, Simon, Witte, Gu, & Wong, 2006; Polaschek, Wilson, Townsend, & Daly, 2005; Wong, Gordon, Gu, Lewis, & Olver, 2012), few studies have explicitly considered psychopathy's responsiveness to treatment. In this section, we review methodological limitations specific to the treatment of psychopathy as well as barriers to progress in this area.

11.3.1 Lack of Contemporary, Empirically Rigorous Studies

The majority of the existing studies concerning the treatment of psychopathy were conducted prior to the 1980s; yet, as noted previously, the past few decades have seen significant gains regarding our understanding of psychopathy's etiology and assessment. As a result, the field is ripe for an influx of treatment evaluation studies based upon this surge of scientific progress in the field.

In 2002, Salekin conducted a meta-analysis of 42 studies of interventions for psychopathy, yet only six were published after 1980, further highlighting the need for contemporary treatment studies. Salekin (2002) reported that, on average, 62% of patients benefited from various forms of psychotherapy (which included psychoanalytic, cognitive–behavioral, therapeutic community, pharmacotherapeutic, and eclectic approaches) compared with 20% of those in the control groups. Nevertheless, critics have noted that most of the studies included in Salekin's meta-analysis contained serious methodological flaws: Many studies did not utilize a reliable, valid assessment tool for psychopathy; few assessed criminal behavior, violence, and/or aggression as outcomes; few utilized comparison groups; many had very small sample sizes; and effectiveness was frequently determined based on therapist opinion (Harris & Rice, 2006). Indeed, Salekin addressed such weaknesses by noting, "Though the studies in the current review may

be less than optimal in scientific rigor, their inclusion is considered to be both necessary and important given our current state of knowledge on psychopathy" (Salekin, 2002, p. 106).

Given the especially high prevalence rates of psychopathy in prisons (e.g., Hare, 1998), most treatment studies are conducted in forensic settings. Nevertheless, it is difficult to recruit equivalent comparison groups in prisons or forensic hospitals and, as such, few controlled treatment-efficacy studies have been conducted on personality-disordered, much less psychopathic, adults in these settings (Lösel, 2001). Rather, many treatment studies involve quasi-experimental designs and nonequivalent control groups (Lösel, 1998) and, as such, are susceptible to selection effects, among other limitations (Cook, Campbell, & Day, 1979). For example, rather than being randomly assigned, offenders might be placed in various treatment groups for a number of nonrandom reasons, including their behavior or staff preference/availability. Whereas controlled research examining treatment of offenders exists (e.g., Lösel, 1995), few controlled studies examine psychopathy specifically. More often, treatment studies involve broad groups of offenders, and psychopathy is not the explicit target of assessment or treatment.

11.3.2 Lack of Theoretically Grounded Treatments

Among the psychopathy literature, rarely are interventions specifically designed to target psychopathy. In a follow-up to their 2002 meta-analysis, Salekin and colleagues (2010) conducted a review of "second-generation" treatment studies, all of which utilized structured assessments of psychopathy (e.g., the PCL or its variants) in an effort to focus on more scientifically rigorous investigations. None of the eight adult studies reviewed included treatments designed to target psychopathy, and many were marked by other methodological weaknesses, such as the use of small samples or retrospective designs (Salekin et al., 2010). Moreover, the descriptions of the interventions in many investigations were brief and neglected to report important procedural or theoretical details, such as posited theoretical mechanisms of change or explicit treatment goals.

Relatedly, reviewers have repeatedly pointed out the need for more theoretically sound conceptualizations of psychopathy, which could, in turn, lead to the development of corresponding interventions designed based on the etiology of psychopathy (Lösel, 1998; Salekin, 2002; Salekin et al., 2010). Indeed, the development of effective treatment programs will presumably benefit from a better understanding of psychopathy's etiology. Many etiological theories of psychopathy exist, including learning theories focusing on modeling and conditioning (e.g., Bandura, 1973; Patterson, Dishion, & Chamberlain, 1993); social cognitive theories (e.g., Huesmann, 1988); response perseveration (Hare, 1970) and response modulation (e.g., Newman & Kosson, 1986); theories invoking personality dispositions, such as fearlessness or sensation-seeking (e.g., Eysenck, 1977; Lykken, 1995; Quay, 1965;); empathy theories (e.g., Gough, 1948); emotional processing theories (Blair, 2003; Hare, 1998); and environmental theories (e.g., McCord & McCord, 1964). Compared with the large number of theories, a limited number of theoretically grounded treatments exist. For example, few of the interventions employed in the 42 studies reviewed by Salekin (2002) were informed by an etiological theory of psychopathy.

11.3.3 Lack of Consensus Regarding Conceptualization and Assessment of Psychopathy

Inconsistencies in conceptual definitions of psychopathy affect both the classification of psychopathy and the assessment of treatment responsiveness. For example, treatment studies based on conceptualizations of psychopathy that place a greater emphasis on behavioral features tend to measure treatment progress based on criminal or antisocial behavior, potentially at the exclusion of other possible treatment gains. Indeed, whereas some argue that recidivism should be the primary outcome of interest (e.g., Harris & Rice, 2006; Wong & Hare, 2005), others argue that treatment studies should consider a wide variety of outcomes (e.g., Salekin, 2002; Salekin et al., 2010).

Similarly, the assessment of psychopathy varies greatly across studies. Whereas some treatment studies classify offenders based on legal definitions of psychopathy (e.g., Blackburn, 1993; Dolan & Coid, 1993), others use a diagnosis of ASPD, and still others rely on assorted measures of psychopathy. For example, some studies utilize the PCL or its variants; others, Cleckley's criteria and ratings; and still others, the Minnesota Multiphasic Personality Inventory (MMPI) Psychopathic Deviate scale, which correlates only weakly with the core interpersonal and affective deficits of psychopathy (Harpur et al., 1989). Even among studies employing the same or comparable measures, a wide variety of cut-offs are used to classify psychopathy. Such variation in methods renders meaningful comparison across studies difficult.

11.4 Evaluation of Psychopathy Treatments

In an attempt to increase the scientific grounding of the field of psychotherapy, David and Montgomery (2011) proposed a novel framework from which to distinguish scientific interventions from those that may produce improvement but lack theoretical substance. David and Montgomery noted that the American Psychological Association Division 12 criteria for empirically supported therapies do not necessitate research support for the theoretical mechanisms of change underlying interventions. They further observed that this omission opens the door for pseudoscientific interventions lacking in theory, and/or interventions relying solely on nonspecific factors, to be considered empirically supported. Thus, their framework emphasized the importance of examining empirical support for both the treatment package and the scientific theory from which it was drawn.

In light of the aforementioned issues in the psychopathy treatment literature, as noted earlier, it is difficult to apply the framework set forth by David and Montgomery (2011) to evaluate the scientific status of the treatment of psychopathy. Given the methodological weaknesses in this literature, it is difficult to identify empirically well-supported therapies, much less therapies based on theoretically sound or explicit mechanisms of change, as most studies do not provide explicit information regarding the active ingredients among various treatment approaches. Further, the vast majority of these treatment studies have been conducted in forensic settings, where offenders typically receive multiple types of treatment (Lösel & Köferl, 1989), rendering conclusions regarding the effects of any specific treatment difficult. As such, it is difficult to isolate specific mechanisms important for change. In the subsections that follow, we selectively

review the literature examining diverse types of treatments, with a particular focus on cognitive–behavioral, therapeutic community, and psychodynamic approaches, as these are the most prevalent in the treatment literature.

11.4.1 Cognitive–Behavioral Approaches

Treatments based in cognitive–behavioral theory have repeatedly been recommended for psychopathy (Andrews & Bonta, 2010; Serin & Kirychik, 1994; Wong & Hare, 2005). For example, based on etiological theories of psychopathy and aggression, Serin and Kirychik (1994) suggested that psychopaths are characterized by deficits in cognitive and social processing and that, through learning and rehearsal, they acquire violence and aggression as a dominant response. As a result, the authors developed a cognitive–behavioral treatment plan focusing on impulsivity as well as social and cognitive processing, aimed to reduce violence in psychopathic offenders. Although others have similarly developed treatment models for psychopathy based in cognitive–behavioral theory (e.g., Wong & Hare, 2005), few programs have been implemented based on these recommendations, despite the broad success of cognitive–behavioral programs in treating antisociality (e.g., Andrews & Bonta, 2010; Kazdin, Siegel, & Bass, 1992). Instead, most studies simply employ cognitive–behavioral techniques in treatment, without explicit links to theoretical mechanisms of change or etiological theories of psychopathy. Given this caveat, we use the term "cognitive-behavioral" loosely in this section, as the studies reviewed here draw on a variety of cognitive and behavioral methods. Notably, some studies reviewed in this section were conducted prior to the release of the first major texts on cognitive–behavioral modification (e.g., Kendall & Hollon, 1979; Mahoney, 1974; Meichenbaum, 1977), so the degree to which the techniques are consistent with contemporary cognitive–behavioral approaches is unclear.

Notwithstanding these theoretical limitations, among the limited psychopathy treatment literature, therapies based in cognitive–behavioral theory appear to be more promising than other approaches. In his meta-analytic review, Salekin (2002) found cognitive–behavioral therapies to have a success rate of 62%, followed closely by psychoanalytic therapies (59% success rate), compared with a 20% success rate in control groups. Yet, this statistic was based on only five studies (one of which was a case review study of three patients), which all used various forms of therapy involving cognitive and/or behavioral techniques with the goal of resocializing offenders. The interventions included group treatments focusing on skill acquisition, work programs, and highly structured programs involving authoritarian or directive behavioral control along with the identification and prevention of problematic feelings and behaviors. As such, some have criticized the classification of therapies in this review (e.g., Harris & Rice, 2006) and questioned the extent to which the "cognitive–behavioral" treatment studies employed cognitive–behavioral theory or methods, particularly when the term "cognitive–behavioral" had not yet been developed at the time the research was conducted (e.g., Craft, Stephenson, & Granger, 1964).

Nevertheless, in a follow-up review, Salekin and colleagues (2010) responded to this critique, noting that the treatments in the 2002 meta-analysis were categorized based on the techniques apparently used in the therapies. Based on reviews of treatment for antisocial behaviors, others have similarly suggested that cognitive–behavioral approaches may be most promising for antisocial, and specifically psychopathic, individuals (e.g.,

Lösel, 1998). Nevertheless, a closer inspection of treatment studies employing cognitive, behavioral, or cognitive–behavioral methods reveals a more complicated picture, from which it is difficult to draw definitive conclusions regarding effectiveness.

In terms of reductions in antisocial behaviors, cognitive–behavioral programs have demonstrated some success in individuals high in psychopathic traits. For example, Craft et al. (1964) compared the effectiveness of two treatment regimens in delinquents between the ages of 13 and 25 who were considered psychopathic based on scores on the MMPI Psychopathic Deviate scale. The "self-governing" regime encouraged permissiveness and independence, whereby "students" met with a small psychotherapy group three times per week and were encouraged to take ownership in the unit. In contrast, the "authoritarian" unit consisted of a much more strict and paternalistic regime with a directive atmosphere. At a follow-up at 14 months, youth from the authoritarian regime were faring better than those from the self-governing regime, exhibiting significantly fewer offenses after release and lower reinstitutionalization rates, as well as significant improvements in "clinical state" based on therapist interviews. Based on these results, and using similar treatment methods, Craft (1968) compared the effectiveness of authoritarian versus permissive treatment programs designed to treat psychopathy in several inpatient settings and found the authoritarian treatment to fare somewhat better based on reconviction rates and social adjustment (i.e., ability to hold a job, social wellbeing) posttreatment. Although in both studies the "authoritarian" treatment involved directive therapy and skills-building, it is unclear to what degree these interventions encompassed cognitive–behavioral techniques. Indeed, as mentioned earlier, Harris and Rice (2006) criticized Salekin's (2002) classification of Craft et al.'s (1964) authoritarian treatment as cognitive–behavioral. Moreover, neither study included a no-treatment control group, without which we can conclude only that the authoritarian treatment was related to better outcomes compared to the self-governing treatment. Without a no-treatment control group comparison, it is possible that the self-governing treatment was related to poorer outcomes, resulting in the authoritarian treatment appearing successful. These limitations notwithstanding, the above-reviewed investigations by Craft and colleagues are among the few to implement treatment designed to target psychopathic personality.

More recent research, conducted after the development of cognitive–behavioral therapy, has also provided some support for such techniques. In a small sample of nine psychiatric inpatient offenders classified as psychopathic based on the PCL-R, Hughes, Hogue, Hollin, and Champion (1997) employed a cognitive skill-based treatment designed to broadly increase adjustment. Notably, individuals scoring higher than 30 on the PCL-R were not admitted to the hospital. The authors supported this decision by citing research (Ogloff et al., 1990; Rice, Harris, & Cormier, 1992) that initially appeared to suggest that individuals with higher PCL-R scores may not benefit from treatment. The treatment involved a supportive ward; group work designed to address cognitive, emotional, and skill functioning; and additional support and treatment based on individual needs. The authors examined a broad range of outcomes through the use of 31 measures combined into a single global change score, noting that assessment of global change over time would be more informative than evaluating change on each measure, as the treatment included a small number of patients who participated in a range of different treatments. Further, some of the measures were not standardized for the type of patient in the sample, obscuring any interpretation of magnitude, rather than simply direction, of change. The global change score, which can perhaps be

criticized for its heterogeneity, included the assessment of problem-solving skills, attitudes, and impulsivity. Results indicated significant clinical gains in the global change measure; yet PCL psychopathy, and specifically Factor 1, was negatively associated with global change. Overall, although the treatment appears to have been broadly successful, higher psychopathy was associated with fewer treatment gains, implying individuals with marked psychopathic traits were less responsive to treatment. Combined with the elimination of individuals scoring greater than 30 on the PCL-R, these limitations preclude any conclusions regarding the effectiveness of this treatment among more severe cases. Further, the individuals in this small sample received varied treatment, as treatment was largely based on individual needs. Such differences in treatment, taken together with the use of a global change score, make it difficult to identify the relative contributions of therapeutic elements important for clinical change.

Promising results have also been reported in studies employing cognitive–behavioral methods among adolescents with psychopathic traits. Specifically, in a series of studies, Caldwell and colleagues (Caldwell, McCormick, Umstead, & Van Rybroek, 2007; Caldwell, Skeem, Salekin, & Van Rybroek, 2006; Caldwell, Vitacco, & Van Rybroek, 2006) examined the efficacy of a juvenile treatment center program designed to treat aggressive delinquent boys. The program, based on concepts of social control theory (Gottfredson & Hirschi, 1990; Sampson & Laub, 1997) and Sherman's (1993) theory of defiance, aimed to channel delinquent associations and activities through the development of interpersonal processes, skill acquisition, and social bonds. The treatment involved individual and group treatment, focusing on anger management, social skills, problem-solving, substance abuse, and sex offender treatment. Although the intervention was not specifically designed to treat psychopathy, participants were scored on the PCL: YV (Forth et al., 2003) based on an admission interview and file review. The studies by Caldwell and colleagues revealed that treatment was associated with improved behavior while participants were institutionalized as well as a significant reduction in violent recidivism postrelease. This relatively methodologically rigorous research is an important step forward, as it provides some grounds for optimism regarding psychopathy's amenability to treatment. Still, Caldwell and colleagues were unable to use a randomized treatment design, and treatment was not manualized. Further, the effects of the treatment on attitudes and personality traits associated with psychopathy are unknown.

Cognitive–behavioral programs may be preferable for use with individuals with psychopathic traits, even if the treatment target is not psychopathy or recidivism. For example, in an evaluation of coping skills versus interactional treatment in alcoholics, Kadden, Cooney, Getter, and Litt (1989) found that patients with higher pretreatment sociopathy scores exhibited decreased drinking rates after coping skills training, whereas interactional therapy was more effective for those with lower sociopathy scores. Conceptually related to psychopathy, sociopathy was assessed via the California Psychological Inventory Socialization Scale (Megargee, 1972). Nevertheless, this scale does not assess many of the core affective and interpersonal features of psychopathy and is instead more of an index of generalized antisocial behavior (Harpur et al., 1989). The coping skills program, modeled after cognitive–behavioral treatment programs, involved a highly structured skills training group that focused on skills for dealing with negative moods and drinking desires, as well as interpersonal, relaxation, and problem-solving skills. Group sessions involved didactic presentations by the therapists as well as behavioral rehearsal and homework exercises designed to practice skills learned in group (Monti, Abrams,

Kadden, & Cooney, 1989). Although the treatment was designed to target alcoholism, not psychopathy, the results provide some support for the success of cognitive–behavioral treatments among individuals with psychopathic traits.

Cognitive–behavioral approaches have also evidenced some success with regard to risk reduction in criminal samples with high levels of psychopathic traits. For example, Olver, Lewis, and Wong (2013) examined the effectiveness of a cognitive–behavioral treatment program among a sample of violent adult offenders in a psychiatric facility. The treatment, termed "ABC Program," is a 6- to 8-month high-intensity violence reduction program based in social learning principles. Founded upon the "what works" principles of correctional treatment (Andrews & Bonta, 2010), the program focuses on targeting on and intervening in criminogenic needs that are linked to violence (such as antisocial attitudes, anger problems, and relationship skills deficits) and promoting the acquisition of prosocial skills to reduce engagement in violent behaviors. Thus, although the program was not designed to target psychopathic traits per se, the intervention targets are related to psychopathy. Moreover, psychopathy as assessed by the PCL-R was examined in relation to therapeutic change and violent recidivism. Therapeutic change scores were negatively correlated with PCL-R dimensions, such that Factor 1 (encompassing the interpersonal and affective features of psychopathy) was a better predictor of decreased therapeutic change than Factor 2 (encompassing the antisocial features of psychopathy). Nevertheless, the authors observed reductions in violent recidivism, although the association between treatment change and violence was weaker after controlling for callous–unemotional features of psychopathy (Olver et al., 2013). Thus, the interpersonal features of psychopathy may render engagement in treatment difficult and thus stand in the way of treatment gains. It is important to note, however, that no control or comparison sample was involved in this study, thus weakening any conclusions with regard to treatment effectiveness. Nevertheless, this investigation lends promising support to the possibility that cognitive–behavioral approaches may exhibit some effectiveness with regard to risk reduction in psychopathic samples (for a review see Wong & Olver, 2015), although it is important for future research to consider the use of a nontreatment control group for comparison purposes.

Still, not all investigations of cognitive–behavioral approaches to psychopathy have yielded promising outcomes. In fact, some have suggested that certain treatments may be iatrogenic, particularly for individuals with high levels of the interpersonal and affective features of psychopathy. Specifically, Hare, Clark, Grann, and Thornton (2000) conducted a nonrandomized controlled study of 278 male offenders in several English prisons. All participants were scored on the PCL-R as part of the admissions process. Offenders participated in a short-term anger management program involving social skills training. After a 2-year follow-up, individuals with higher Factor 1 scores exhibited significantly higher rates of reconviction. The authors speculated that Factor 1 psychopaths may have increased their manipulative skill while in treatment. Nevertheless, without a control or comparison condition, it is impossible to know whether the treatment was causally associated with poor outcome. Moreover, the description of the treatment in this study is lacking, and it is possible that it varied across settings (Salekin et al., 2010). Furthermore, the authors noted that nonpsychopathic offenders did not benefit from the treatment, calling into question the appropriateness of the intervention (Hare et al., 2000). As such, it is difficult to conclude that this study provides evidence against the treatability of psychopathy.

Similarly, Seto and Barbaree (1999) suggested that sex offenders with psychopathic traits are adept at manipulating others during and after treatment. Specifically, Seto and Barbaree (1999) examined PCL-R psychopathy, treatment behavior, and recidivism rates among a sample of sex offenders in a cognitive–behavioral and relapse prevention program. The treatment involved daily 3-hour group sessions over a period of 5 months. The treatment focused on the identification and understanding of individual offense cycles by sequencing the thoughts, feelings, and behaviors preceding the commission of a sexual offense. Then, an individualized relapse prevention plan was enacted for each offender, which focused on the development of coping skills and/or avoidance strategies in accordance with individual triggers. Notably, the treatment was not designed to target psychopathy per se. Results revealed that offenders with higher PCL-R scores, who were rated to have behaved more positively in treatment, actually exhibited higher violent and/or sex offense recidivism rates. Nevertheless, these results did not hold up in a follow-up study (Barbaree, 2005), and others have questioned the meaning of the treatment behavior ratings in the Seto and Barbaree (1999) study. For example, Polaschek and Daly (2013) pointed out that the treatment behavior ratings in the original study, which were created by research assistants through the retrospective examination and aggregation of information from posttreatment reports, could not be replicated through the same process by two independent raters in a follow-up study (Langton, Barbaree, Harkins, & Peacock, 2006). Additionally, it was suggested that the treatment behavior ratings in Seto and Barbaree (1999) may have been biased by information unrelated to the treatment (Langton et al., 2006). Moreover, Olver and Wong (2009) examined the efficacy of a similar cognitive–behavioral relapse prevention program among psychopathic sex offenders and reported much more positive results. Although psychopathy was a predictor of treatment dropout, 73% of psychopathic offenders completed the program, and those who dropped out exhibited higher rates of violent, but not sexual, recidivism. Furthermore, positive treatment gains (as assessed by a violence risk scale) were associated with lower recidivism rates, indicating that individuals who were rated to have benefited more from treatment did not recidivate, although this finding may also merely reflect the fact that better adjusted participants were at lower risk for recidivism, independent of treatment. In light of these findings, it is difficult to conclude that treating psychopathy exacerbates psychopathic traits or creates a "more skilled" psychopath.

The aforementioned literature reveals that, whereas some investigations of cognitive–behavioral techniques for the treatment of psychopathy report some success, a closer inspection of these investigations reveals methodological limitations that preclude clear-cut conclusions. Moreover, cognitive–behavioral techniques may be more successful than other approaches among individuals with psychopathic traits, even if psychopathy is not the target of treatment.

11.4.2 Therapeutic Communities

The concept of the therapeutic community is one of the most frequently employed interventions for psychopathy. It was initially developed by Jones (1952) as a potential treatment for psychopathic inmates, on the basis that rehabilitation may occur if inmates are provided with an encouraging environment that fosters the adoption of responsibility for one's actions. Some authors (e.g., Hare, 1970) have also suggested that the therapeutic community creates a reshaped social environment capable of

changing psychopathic personality traits and behaviors. Nevertheless, the theoretical mechanisms within the therapeutic community that would bring about change in psychopathic personality traits are unclear.

After the therapeutic community first appeared, several modifications were instituted and numerous versions implemented. Certain components are relatively consistent among the various implementations. Specifically, therapeutic communities are distinctive in their establishment of an informal, supportive atmosphere in otherwise traditional institutions. For example, inmates are responsible for directing everyday activities, and facility staff serve as models for prosocial behavior and confront disruptive behaviors. Further, the residential community within the institution provides a supportive, therapeutic atmosphere. A critical component of the therapeutic community is the daily group meeting, at which all patients and staff are present. This meeting provides a cooperative, democratically based decision-making setting in which therapy can take place, potential conflicts can be discussed, and rules can be developed. Nevertheless, the therapeutic communities employed in the literature are not systematic treatments, and variety is commonplace. Moreover, many studies provide relatively brief descriptions of the therapeutic community, and some approaches are questionable in light of current ethical standards; for example, the inclusion of harsh disciplinary actions for misbehavior (e.g., seclusion) or the administration of alcohol and drugs (e.g., Rice, Harris, & Cormier, 1992). Not surprisingly, such approaches have attracted significant controversy and criticism (e.g., Polaschek & Daly, 2013; Skeem, Polaschek, & Manchak, 2009).

Although the therapeutic community is one of the most frequently used interventions for psychopathy, it has received little empirical support. In his 2002 meta-analysis, Salekin concluded that therapeutic communities were among the least effective treatments for psychopathy. Based on eight studies, Salekin (2002) found therapeutic communities to be associated with an average success rate of 25%, only slightly higher than that of the control conditions (20%). Although early studies using therapeutic communities boasted some success (e.g., Barker, Mason, & Wilson, 1969; Copas, O'Brien, Roberts, & Whiteley, 1984; Copas & Whiteley, 1976; Kiger, 1967), they were replete with methodological limitations, such as not classifying individuals as psychopathic or nonpsychopathic, not including a nontreatment control group, or not describing the treatment in sufficient detail.

Studies involving more rigorous scientific designs also do not provide much support for the effectiveness of the therapeutic community for psychopathic individuals. For example, Ogloff et al. (1990) examined a therapeutic community program in a forensic hospital. The authors split participants into subgroups based on PCL scores (those with a score of 27 or greater were classified as "psychopathic"; those who scored between 18 and 26 as "mixed"; and those who scored 17 or below as "nonpsychopathic") and found that psychopathic individuals exhibited less motivation in treatment and dropped out sooner than those in the other two groups, and also evidenced less clinical improvement at discharge (based on independent raters' reviews of clinical discharge summaries). These results are consistent with those from other therapeutic communities, which find psychopathy scores to be associated with poorer attendance and adherence to treatment (e.g., Hobson, Shine, & Roberts, 2000; Richards, Casey, & Lucente, 2003). The therapeutic community may not be sufficiently engaging or motivating to be successful for individuals with psychopathy. However, as Salekin and colleagues (2010) pointed out,

this study employed a retrospective design and only followed up a small portion of the participants ($n = 28$).

Although controversial, similarly to the findings regarding cognitive–behavioral programs with sex offenders (e.g., Seto & Barbaree, 1999), some research indicates that treatment of psychopathy using the therapeutic community may make psychopaths "worse" (Rice et al., 1992). One of the best-known examinations of the therapeutic community's effectiveness in reducing violence was conducted by Rice, Harris, and Cormier (1992). The authors retrospectively evaluated the 1960s Oak Ridge Social Therapy Unit, a hospitalization program in which 146 treated offenders were matched with an equal number of untreated offenders (based on age, criminal history, and index offense). All participants were scored on the PCL-R based on file information. Based on follow-up data roughly 10.5 years posttreatment, the authors concluded that the hospital treatment program resulted in increased risk of violent recidivism for psychopaths but decreased risk for nonpsychopaths. The authors speculated that the treatment provided a learning opportunity for both psychopaths and nonpsychopaths alike. Whereas the nonpsychopaths used the information to behave prosocially, the psychopaths used it to manipulate and exploit others (Harris & Rice, 2006; Rice et al., 1992).

Yet, several commentators have seriously questioned numerous elements of the treatment program, particularly its coerciveness, and highlighted the possibility that psychopaths were differentially harmed in the involuntary program, as they were exposed to more radical disciplinary action than were nonpsychopaths (e.g., Polaschek & Daly, 2013; Skeem et al., 2009). Specifically, in service of disrupting patients' unconscious defenses, treatment was intensive and included extreme measures, such as the administration of drugs (methedrine, LSD, scopolamine, and alcohol) and the use of marathon nude encounter sessions lasting up to 2 weeks. Despite the dubious treatment methods used in this study, it has repeatedly been cited as evidence that therapy makes psychopaths worse (e.g., Hare, 1993). Needless to say, this conclusion can be questioned.

Overall, the early optimism regarding the effectiveness of the therapeutic community in treating psychopathy appears to have dissolved. The treatment mechanism involved in therapeutic communities that would theoretically bring about change in psychopathic personality is unclear. Moreover, studies examining the effectiveness of therapeutic communities in reducing violence and antisocial behaviors have employed questionable and at best controversial techniques (e.g., Rice et al., 1992), raising questions about the evidentiary basis of this approach.

11.4.3 Psychodynamic Approaches

Given that psychoanalytic theory traditionally regards the development of a positive transference relationship between the therapist and client as an essential vehicle for improvement, it would seem unlikely that psychoanalytic approaches would be especially successful in the treatment of psychopaths, who have difficulty forming attachments with others. Yet, in his 2002 meta-analysis, Salekin found psychoanalytic therapies to be second only to cognitive–behavioral approaches in the effective treatment of psychopathy. He reported a success rate of 52% among 17 studies employing psychoanalytic methods. Nevertheless, a closer examination reveals a murky picture. First, only one of the studies classified by Salekin to be psychoanalytic involved a controlled design; the remaining 16 were case studies or collections of case studies. Thus,

many of these studies lack scientific rigor and involved unsystematically administered treatments whose posited mechanisms of change were often not explicitly described by the authors. Moreover, most studies omitted crucial details concerning the treatment methods used. Such issues preclude firm conclusions regarding the effectiveness of psychoanalytic approaches in the treatment of psychopathy.

In one of the few scientifically designed studies, Heaver (1943) examined the effects of psychoanalytic treatment among 40 hospitalized male patients. The patients were diagnosed with psychopathy based on Cheney's (1934) criteria: emotional immaturity or childishness, marked defects of judgment, inability to learn by experience, impulsive reactions without consideration for the feelings of others, and emotional instability characterized by rapid swings from elation to depression. Notably, these criteria could have captured individuals with conditions other than psychopathy, such as borderline personality disorder. Moreover, both the treatment and outcome measures were minimally, if at all, described. The results revealed 40% of the patients conformed to society's standards posttreatment, upon which the authors concluded that the treatment was effective. Nevertheless, the study included no comparison group, and, as mentioned, important information regarding treatment and outcome measures was omitted. As such, no conclusions can be drawn regarding the effectiveness of the treatment.

Several other treatments related to psychoanalytic theory have also been attempted with psychopathic individuals. Rooted in part in psychoanalytic theory, "psychodrama" relies heavily on the use of role-playing as a process by which the individual can be exposed to a greater variety of feelings and attitudes. Although the empirical support for this contention is questionable, some have suggested that such role-playing techniques provide a unique opportunity for psychopaths to break through their presumed defenses to alter their emotional experience, and potentially develop empathy (Carpenter & Sandberg, 1973). Psychodrama is often referred to as an "action" method and is closely related to action-oriented programs, which focus on decreasing boredom to increase treatment engagement. Action-oriented programs first appeared in the 1960s, in response to reports of little to no success in treating the most hard-core, psychopathic offenders within penal institutions. As a result of these repeated poor treatment outcomes, clinicians called for novel approaches. For example, Fox (1961) stated that "because [the psychopathic offender] is so difficult to reach, the usual and accepted methods of therapy are just not effective. Something unusual, unorthodox, and unexpected is needed to begin this relationship" (p. 476). Thus, based on theories that emphasized the psychopath's tendency to seek increased variety in environmental stimuli (e.g., Quay, 1965), treatment programs were developed that highlighted change, action, and novelty. The developers believed that such environments might engage psychopaths to such an extent that they could be effectively managed within institutions, and consequently benefit from regular programming (Ingram, Gerard, Quay, & Levinson, 1970).

Although the first report on psychodrama for psychopathy was promising (Corsini, 1958), it was a single case study. Numerous studies have examined psychodrama in correctional settings, yet few have examined the effectiveness of psychodrama in individuals with psychopathic traits using comparison groups. One such study was conducted by Maas (1966), who investigated the effectiveness of psychodrama in 46 adult female offenders, all of whom were classified as sociopathic based on Gough's (1960) Socialization Scale, which is a suboptimal measure of psychopathy. The experimental group received a combination of psychodrama and more traditional group therapy, whereas

the control group received no treatment. The Ego Identity Index (Block, 1961), which is based on the extent of interpersonal consistency in interactions with others, was administered pre- and posttreatment as an index of the individual's level of "ego diffusion." Although no outcome data were provided, a significant difference between groups revealed that the experimental group exhibited a stronger sense of personal identity. Given the lack of actual data provided, however, few firm conclusions can be drawn from this study.

To our knowledge, the study conducted by Ingram and colleagues (1970) is the only one to examine the efficacy of action-oriented therapy with psychopathic offenders. The authors compared 20 juvenile delinquents in action-oriented therapy, which included psychodrama, with 41 youth who received standard institutional counseling. The treatment, aimed at reducing boredom in psychopathic youth, emphasized excitement and novelty through various recreational activities and a "circus-like" atmosphere. Youth were rewarded with points and prizes for positive behavior and for winning competitions. Results indicated that youth in the action-oriented program spent significantly fewer days in administrative segregation, committed fewer violent offenses in the institution, and exhibited less negative institutional adjustment after transfer to another institution (defined as fewer instances of being absent without leave and fewer disciplinary transfers) than did youth in standard institutional counseling. Overall, the results suggest that psychopathic youth may be more engaged with treatments involving varied, exciting experiences, resulting in more positive institutional behavior. Yet, without long-term outcome data, the stability of these treatment gains over time and the extent to which the gains are maintained across settings (e.g., in the community, postrelease) are unknown.

In sum, psychoanalytic approaches to the treatment of psychopathy have received little support. Yet, because psychodynamic treatments are meant to directly effect personality change, it is theoretically reasonable to consider psychodynamic treatments for psychopathy, especially when conceptualizing psychopathy within a personality framework. Nevertheless, especially in view of psychopaths' presumed inability to form close attachments to others, including therapists, the theoretical basis for psychoanalytic techniques (which require the development of a transference relationship with the therapist) for psychopathy is scientifically questionable, rendering the proposed mechanisms of change in turn questionable. Further, the scientific rigor in the psychoanalytic treatment literature for psychopathy is wanting, and, as such, precludes clear-cut conclusions regarding the effectiveness of psychoanalytic approaches.

11.5 Implications for Research

Overall, the preceding review reveals that, although various attempts have been made to identify successful treatments for psychopathy, this goal has not yet been attained. This conclusion does not necessarily render psychopathy untreatable, although it suggests that successful treatments have yet to be discovered through rigorous scientific investigations. Indeed, it is possible that a successful treatment has already been developed but that the methodological difficulties pervading much of the aforementioned literature render conclusions regarding its efficacy premature. Alternatively, it may be that a successful treatment has yet to be developed and its efficacy demonstrated (Harris &

Rice, 2006). In either case, the burden of proof lies on investigators to demonstrate such efficacy for any specific psychopathy treatment. In our view, this burden has yet to be convincingly dealt with.

In spite of psychopathy's poor reputation with regard to treatability, researchers are pursuing new and creative treatment options as our understanding of brain–behavior relationships continues to grow and evolve. Specifically, neuroscience research increasingly points to various structural and functional deficits associated with psychopathy, such as decreased amygdala volume (e.g., Boccardi et al., 2011; Ermer, Cope, Nyalakanti, Calhoun, & Kiehl, 2012; Yang, Raine, Narr, Colletti, & Toga, 2009) and activation (e.g., Birbaumer et al., 2005; Rilling et al., 2007) and abnormalities in the prefrontal cortex (e.g., Gregory et al., 2012; Yang & Raine, 2009). Some researchers are hopeful that these advances in brain research on psychopathy may be able to inform intervention efforts in the future (e.g., Mobbs, Lau, Jones, & Frith, 2007), suggesting that the affected brain regions in people with psychopathy may serve as effective mechanisms of change, such that we may be able to modify their structural and/or functional deficits through various modes of noninvasive treatment, including repetitive transcranial magnetic stimulation (Glenn & Raine, 2013). Drawing on advances in neurobiology and cognitive neuroscience, cognitive remediation has also been suggested as a potential avenue for intervention (Baskin-Sommers, Curtain, & Newman, 2015). This treatment, still in the initial stages of development, uses cognitive training techniques to target attentional biases in psychopathic individuals who fail to recognize important affective, inhibitory, and punishment cues that interact with goal-directed behaviors. The stated goal of this approach would be to support individuals' amelioration of attentional biases and modification of reactionary behaviors. Nevertheless, these are theoretical treatments at this point, which have yet to enter the infancy stage of rigorous scientific testing that will be needed before any conclusions can be reached as to their effectiveness.

To scientifically evaluate various treatments using evaluative frameworks such as that proposed by David and Montgomery (2011), treatments that target theoretically sound mechanisms of change must first be developed. Indeed, therapies that are more closely tied to well-articulated etiological theories have the greatest chance of moving research in this area forward (Salekin, 2002; Salekin et al., 2010). As such, we must develop treatments that are (1) explicitly designed to target psychopathy and allied conditions and (2) are consistent with scientifically supported theories of the construct. One obstacle to this end is the lack of consensus regarding psychopathy's definition and essential features, an issue that has been repeatedly highlighted by researchers in the past (Blackburn, 1993; Harris & Rice, 2006; Lösel, 1998; Salekin, 2002; Salekin et al., 2010). Such disagreement leads to variability in the assessment of psychopathy as well as treatment targets, and hinders the emergence of consistent patterns of success or failure across studies. With so many theoretical models of psychopathy in existence, future research should consider how differing conceptualizations and operationalizations of psychopathy relate to measured treatment response (Salekin et al., 2010). Moreover, consensus must also be reached regarding the definition of efficacy in relation to psychopathy treatment. At the very least, the goals of therapy and the outcomes of interest must be tied more explicitly to psychopathy's core personality traits as opposed exclusively to its associated antisocial and criminal behaviors. Although recidivism is a theoretically and pragmatically important outcome of interest (Harris & Rice, 2006), it may

be fruitful to consider broad outcomes related to psychopathy and life functioning, such as job performance, interpersonal relationships, and engagement in enjoyable activities (Salekin et al., 2010).

In addition to enhancing the theoretical grounding of treatments, the scientific rigor of treatment investigations must be improved (Lösel, 1998; Salekin, 2002; Salekin et al., 2010). Since the mid-1990s, significant strides have been made in the development of reliable and valid measures of psychopathy. It will be important to rely more heavily on these established measures for baseline and outcome assessments in future treatment investigations. Studies involving nontreatment control groups will also be important. Moreover, studies should include detailed methodological information, particularly details specific to the treatment package and its posited theoretical mechanisms of change, to facilitate replication studies. In sum, prospective, controlled studies of psychopathy's treatment response are needed to evaluate the effectiveness of specific therapeutic packages. Such investigations are also necessary to advance conclusions regarding the evidentiary basis of the treatment of psychopathy.

11.6 Implications for Clinical Practice

Overall, the above-reviewed literature reveals little empirical support for various treatment approaches for psychopathy. Yet, it is important to emphasize the distinction between *invalidated* therapies, which have been systematically examined and shown to be ineffective, and *unvalidated* therapies, which have not been adequately systematically examined to draw conclusions (Arkowitz & Lilienfeld, 2006). Although treatment for psychopathy has received little evidentiary support thus far, methodological limitations in the existing literature prevent the conclusion that the available treatments are *invalidated*. Rather, the available treatments can only be considered *unvalidated*, further underscoring the need for more rigorous scientific investigations.

In the absence of a strong evidentiary basis for any specific psychopathy treatments, clinical intervention must rely on the best available evidence. As such evidence is lacking for psychopathy specifically, a first step may be to turn to supported treatments for antisocial behavior. Indeed, in forensic settings, where a higher percentage of psychopathic personalities exist, behavior control may be the most important target of treatment. Interventions based in social learning theories have exhibited success in offender groups (Andrews & Bonta, 2010) and, as such, may be appropriate for individuals with psychopathy (Lösel, 1998). Yet, solely targeting antisocial behavior without consideration for the etiology of psychopathy may impede the success of treatment. Research examining the treatment of antisocial behaviors reveals that the programs evidencing the most success are those based on scientifically sound hypotheses regarding the development and maintenance of such behaviors (Andrews & Bonta, 2010; Antonowicz & Ross, 1994). As such, interventions are more likely to be successful if underlying processes are considered (Beauchaine, Neuhaus, Brenner, & Gatzke-Kopp, 2008). Indeed, cognitive–behavioral interventions designed to target personality traits that have been identified as risk factors for youth substance misuse have evidenced promising and long-lasting effects on youth behavior postintervention (e.g., Conrod, Castellanos, & Mackie, 2008; Conrod, Stewart, Comeau, & Maclean, 2006; Conrod et al., 2000; Watt, Stewart, Birch, & Bernier, 2006). When conceptualizing psychopathy from a personality perspective,

such interventions appear particularly promising, given the focus on personality traits as risk factors for the development and expression of psychopathy.

11.7 Conclusions

In conclusion, although there is promising evidence that psychopathy may be somewhat treatable, the evidentiary support for any given treatment is minimal. Most interventions are not specifically designed to target psychopathy according to accepted theoretical models of the construct, and there is no shortage of methodological weaknesses in the psychopathy treatment literature. As such, most interventions are lacking theoretical and empirical basis and are not amenable to evaluative frameworks for empirical support (e.g., David & Montgomery, 2011).

Although popular opinion has generally regarded psychopathy as untreatable, the lack of evidentiary basis for specific interventions does not support this conclusion. Indeed, over 50 years ago, Chwast (1961) highlighted this sentiment in his review of problems in the treatment of psychopathic offenders with the following statement: "An interesting question does arise, however, in clarifying to what extent a prognosis of irreversibility reflects an admission of our own therapeutic incompetency and inadequacy rather than an asseveration that change is not possible in any environment under any set of circumstances" (p. 223). Rather, the psychopathy treatment literature demonstrates that successful treatment has yet to be demonstrated, underscoring the need for more research in this arena. In light of several sanguine reviews (Salekin, 2002; Salekin et al., 2010) and promising indications of effective treatments (e.g., Caldwell et al., 2007; Caldwell, Skeem et al., 2006; Caldwell, Vitacco, & Van Rybroek, 2006; Skeem, Monahan, & Mulvey, 2002), attitudes regarding psychopathy's amenability to treatment appear to be softening. Indeed, with major strides in research on the etiology and assessment of psychopathy occurring over the past few decades, we are now in a much better position to develop and evaluate theoretically grounded, evidence-based psychopathy treatments.

References

American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders* (1st ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd rev. ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.

Anderson, J. L., Sellbom, M., Wygant, D. B., Salekin, R. T., & Krueger, R. F. (2014). Examining the associations between DSM-5 Section III antisocial personality disorder

traits and psychopathy in community and university samples. *Journal of Personality Disorders*, *28*, 675–697. doi:10.1521/pedi_2014_28_134

Andrews, D. A., & Bonta, J. (2010). *The psychology of criminal conduct* (5th ed.). Cincinnati, OH: Anderson.

Antonowicz, D. H., & Ross, R. R. (1994). Essential components of successful rehabilitation programs for offenders. *International Journal of Offender Therapy and Comparative Criminology*, *38*(2), 97–104. doi:10.1177/0306624x9403800202

Arkowitz, H., & Lilienfeld, S. O. (2006). Psychotherapy on trial. *Scientific American Mind*, *17*(2), 42–49. doi:10.1038/scientificamericanmind0406-42

Bandura, A. (1973). Social learning analysis of aggression. In E. Ribes-Inesta & A. Bandura (Eds.), *Analysis of delinquency and aggression*. Oxford, UK: Lawrence Erlbaum Associates.

Barbaree, H. E. (2005). Psychopathy, treatment behavior, and recidivism: An extended follow-up of Seto and Barbaree. *Journal of Interpersonal Violence*, *20*(9), 1115–1131. doi:10.1177/0886260505278262

Barker, E. T., Mason, M. H., & Wilson, J. (1969). Defence-disrupting therapy. *Canadian Psychiatric Association Journal*, *14*, 355–359. doi:10.1177/070674376901400407

Baskin-Sommers, A. R., Curtin, J. J., & Newman, J. P. (2015). Altering the cognitive-affective dysfunctions of psychopathic and externalizing offender subtypes with cognitive remediation. *Clinical Psychological Science*, *3*(1), 45–57. doi:10.1177/2167702614560744

Beauchaine, T. P., Neuhaus, E., Brenner, S. L., & Gatzke-Kopp, L. (2008). Ten good reasons to consider biological processes in prevention and intervention research. *Development and Psychopathology*, *20*(3), 745–774.

Birbaumer, N., Veit, R., Lotze, M., Erb, M., Hermann, C., Grodd, W., & Flor, H. (2005). Deficient fear conditioning in psychopathy: A functional magnetic resonance imaging study. *Archives of General Psychiatry*, *62*(7), 799–805. doi:10.1001/archpsyc.62.7.799

Blackburn, R. (1993). *The psychology of criminal conduct: Theory, research and practice*. Chichester, UK: John Wiley & Sons.

Blair, R. J. R. (2003). Neurobiological basis of psychopathy. *British Journal of Psychiatry*, *182*(1), 5–7. doi:10.1192/bjp.182.1.5

Block, J. (1961). Ego identity, role variability, and adjustment. *Journal of Consulting Psychology*, *25*(5), 392–397. doi:10.1037/h0042979

Boccardi, M., Frisoni, G. B., Hare, R. D., Cavedo, E., Najt, P., Pievani, M., … Tiihonen, J. (2011). Cortex and amygdala morphology in psychopathy. *Psychiatry Research: Neuroimaging*, *193*(2), 85–92.

Caldwell, M., Skeem, J., Salekin, R., & Van Rybroek, G. (2006). Treatment response of adolescent offenders with psychopathy features: A 2-year follow-up. *Criminal Justice and Behavior*, *33*(5), 571–596. doi:10.1177/0093854806288176

Caldwell, M. F., McCormick, D. J., Umstead, D., & Van Rybroek, G. J. (2007). Evidence of treatment progress and therapeutic outcomes among adolescents with psychopathic features. *Criminal Justice and Behavior*, *34*(5), 573–587.

Caldwell, M. F., Vitacco, M., & Van Rybroek, G. J. (2006). Are violent delinquents worth treating? A cost–benefit analysis. *Journal of Research in Crime & Delinquency*, *43*(2), 148–168. doi:10.1177/0022427805280053

Carpenter, P., & Sandberg, S. (1973). "The things inside": Psychodrama with delinquent adolescents. *Psychotherapy: Theory, Research & Practice*, *10*(3), 245–247. doi:10.1037/h0087586

Cheney, C. O. (1934). *Outlines for psychiatric examinations*. New York, NY: New York State Department of Mental Hygiene.

Chwast, J. (1961). Reversibility–irreversibility: Problems in the treatment of offenders. *American Journal of Psychotherapy*, *15*, 221–232.

Cleckley, H. (1941/1988). *The mask of sanity*. St. Louis, MO: Mosby.

Cloninger, C. R. (1978). The antisocial personality. *Hospital Practice*, *13*, 97–106. doi:10.1080/21548331.1978.11707387

Coid, J., Yang, M., Ullrich, S., Roberts, A., Moran, P., Bebbington, P., … Hare, R. (2009). Psychopathy among prisoners in England and Wales. *International Journal of Law and Psychiatry*, *32*(3), 134–141. doi:10.1016/j.ijlp.2009.02.008

Conrod, P. J., Castellanos, N., & Mackie, C. (2008). Personality-targeted interventions delay the growth of adolescent drinking and binge drinking. *Journal of Child Psychology and Psychiatry*, *49*(2), 181–190.

Conrod, P. J., Stewart, S. H., Comeau, N., & Maclean, A. M. (2006). Efficacy of cognitive–behavioral interventions targeting personality risk factors for youth alcohol misuse. *Journal of Clinical Child & Adolescent Psychology*, *35*(4), 550–563. doi:10.1207/s15374424jccp3504_6

Conrod, P. J., Stewart, S. H., Pihl, R. O., Côté, S., Fontaine, V., & Dongier, M. (2000). Efficacy of brief coping skills interventions that match different personality profiles of female substance abusers. *Psychology of Addictive Behaviors*, *14*(3), 231–242. doi:10.1037//0893-164x.14.3.231

Cook, T. D., Campbell, D. T., & Day, A. (1979). *Quasi-experimentation: Design & analysis issues for field settings*. Boston, MA: Houghton Mifflin.

Cooke, D. J., & Michie, C. (2001). Refining the construct of psychopathy: Towards a hierarchical model. *Psychological Assessment*, *13*, 171–188. doi:10.1037/1040-3590.13.2.171

Copas, J. B., O'Brien, M., Roberts, J., & Whiteley, J. S. (1984). Treatment outcome in personality disorder: The effect of social psychological and behavioural variables. *Personality and Individual Differences*, *5*, 565–573. doi:10.1016/0191-8869(84)90031-x

Copas, J. B., & Whiteley, J. S. (1976). Predicting success in the treatment of psychopaths. *British Journal of Psychiatry*, *129*, 388–392. doi:10.1192/bjp.129.4.388

Corsini, R. (1958). Psychodrama with a psychopath. *Group Psychotherapy*, *11*, 33–39.

Craft, M. (1968). Psychopathic disorder: A second trial of treatment. *British Journal of Psychiatry*, *114*(512), 813–820. doi:10.1192/bjp.114.512.813

Craft, M., Stephenson, G., & Granger, C. (1964). A controlled trial of authoritarian and self-governing regimes with adolescent psychopaths. *American Journal of Orthopsychiatry*, *34*(3), 543–554. doi:10.1111/j.1939-0025.1964.tb02224.x

David, D., & Montgomery, G. H. (2011). The scientific status of psychotherapies: A new evaluative framework for evidence-based psychosocial interventions. *Clinical Psychology: Science and Practice*, *18*(2), 89–99. doi:10.1111/j.1468-2850.2011.01239.x

Di Placido, C., Simon, T. L., Witte, T. D., Gu, D., & Wong, S. C. (2006). Treatment of gang members can reduce recidivism and institutional misconduct. *Law and Human Behavior*, *30*(1), 93–114. doi:10.1007/s10979-006-9003-6

Dolan, B., & Coid, J. (1993). *Psychopathic and antisocial personality disorders: Treatment and research issues*. London, UK: Gaskell.

Doren, D. M. (1987). *Understanding and treating the psychopath*. Oxford, UK: John Wiley & Sons.

D'Silva, K., Duggan, C., & McCarthy, L. (2004). Does treatment really make psychopaths worse? A review of the evidence. *Journal of Personality Disorders*, *18*(2), 163–177. doi:10.1521/pedi.18.2.163.32775

Edens, J. F., Marcus, D. K., Lilienfeld, S. O., & Poythress, N. G. (2006). Psychopathic, not psychopath: Taxometric evidence for the dimensional structure of psychopathy. *Journal of Abnormal Psychology*, *115*, 131–144. doi:10.1037/0021-843x.115.1.131

Eissler, K. R. (1949). Some problems of delinquency. In K. R. Eissler (Ed.), *Searchlights on delinquency* (pp. 3–25). New York, NY: International Universities Press.

Ermer, E., Cope, L. M., Nyalakanti, P. K., Calhoun, V. D., & Kiehl, K. A. (2012). Aberrant paralimbic gray matter in criminal psychopathy. *Journal of Abnormal Psychology*, *121*(3), 649–658. doi:10.1037/a0026371

Eysenck, H. J. (1977). *Crime and personality* (3rd ed.). New York, NY: Routledge.

Feighner, J. P., Robins, E., Guze, S. B., Woodruff, R. A., Winokur, G., & Munoz, R. (1972). Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, *26*(1), 57–63. doi:10.1001/archpsyc.1972.01750190059011

Forth, A. E., Kosson, D., & Hare, R. D. (2003). *The Hare PCL: Youth Version*. Toronto, ON: Multi-Health Systems.

Fox, V. (1961). Psychopathy as viewed by a clinical psychologist. *Archives of Criminal Psychodynamics*, *4*, 472–479.

Glenn, A. L., & Raine, A. (2013). Neurocriminology: Implications for the punishment, prediction and prevention of criminal behaviour. *Nature Reviews Neuroscience*, *15*, 54–63. doi:10.1038/nrn3640

Gregory, S., Simmons, A., Kumari, V., Howard, M., Hodgins, S., & Blackwood, N. (2012). The antisocial brain: Psychopathy matters—A structural MRI investigation of antisocial male violent offenders. *Archives of General Psychiatry*, *69*(9), 962–972. doi:10.1001/archgenpsychiatry.2012.222

Gottfredson, M. R., & Hirschi, T. (1990). *A general theory of crime*. Stanford, CA: Stanford University Press.

Gough, H. G. (1948). A sociological theory of psychopathy. *American Journal of Sociology*, *53*(5), 359–366. doi:10.1086/220203

Gough, H. G. (1960). Theory and measurement of socialization. *Journal of Consulting Psychology*, *24*, 23–30. doi:10.1037/h0044749

Hare, R. D. (1970). *Psychopathy: Theory and research*. Chichester, UK: John Wiley & Sons.

Hare, R. D. (1980). A research scale for the assessment of psychopathy in criminal populations. *Personality and Individual Differences*, *1*(2), 111–119. doi:10.1016/0191-8869(80)90028-8

Hare, R. D. (1991/2003). *Manual for the revised psychopathy checklist* (2nd ed.). Toronto, ON: Multi-Health Systems.

Hare, R. D. (1993). *Without conscience: The disturbing world of the psychopaths among us*. New York, NY: Simon & Schuster.

Hare, R. D. (1996). Psychopathy: A clinical construct whose time has come. *Criminal Justice and Behavior*, *23*, 25–54. doi:10.1177/0093854896023001004

Hare, R. D. (1998). Psychopathy, affect and behavior. In D. J. Cooke, A. E. Forth, & R. D. Hare (Eds.), *Psychopathy: Theory, research and implications for society* (pp. 303–354). Dordrecht, Netherlands: Kluwer.

Hare, R. D., Clark, D., Grann, M., & Thornton, D. (2000). Psychopathy and the predictive validity of the PCL-R: An international perspective. *Behavioral Sciences & the Law*, *18*(5), 623–645. doi:10.1002/1099-0798(200010)18:5<623::aid-bsl409>3.0.co;2-w

Hare, R. D., & Neumann, C. S. (2008). Psychopathy as a clinical and empirical construct. *Annual Review of Clinical Psychology*, *4*, 217–246. doi:10.1146/annurev.clinpsy.3.022806.091452

Hare, R. D., & Neumann, C. S. (2010). The role of antisociality in the psychopathy construct: Comment on Skeem and Cooke (2010). *Psychological Assessment*, *22*, 446–454. doi:10.1037/a0013635

Harpur, T. J., Hare, R. D., & Hakstian, R. (1989). A two-factor conceptualization of psychopathy: Construct validity and implications for assessment. *Psychological Assessment*, *1*, 6–17. doi:10.1037//1040-3590.1.1.6

Harris, G. T., & Rice, M. E. (2006). Treatment of psychopathy. In C. J. Patrick (Ed.), *Handbook of psychopathy* (pp. 555–572). New York, NY: Guilford Press.

Harris, G. T., Rice, M. E., & Quinsey, V. L. (1994). Psychopathy as a taxon: Evidence that psychopaths are a discrete class. *Journal of Consulting and Clinical Psychology*, *62*(2), 387–397. doi:10.1037//0022-006x.62.2.387

Hart, S. D., Cox, D. N., & Hare, R. D. (1995). *The Hare Psychopathy Checklist: Screening version*. Toronto, ON: Multi-Health Systems.

Heaver, W. L. (1943). A study of forty male psychopathic personalities before, during and after hospitalization. *American Journal of Psychiatry*, *100*(3), 342–346.

Hobson, J., Shine, J., & Roberts, R. (2000). How do psychopaths behave in a prison therapeutic community? *Psychology, Crime & Law*, *6*(2), 139–154. doi:10.1080/10683160008410838

Huesmann, L. (1988). An information processing model for the development of aggression. *Aggressive Behavior*, *14*(1), 13–24. doi:10.1002/1098-2337(1988)14:1<13::aid-ab2480140104>3.0.co;2-j

Hughes, G., Hogue, T., Hollin, C., & Champion, H. (1997). First-stage evaluation of a treatment programme for personality disordered offenders. *Journal of Forensic Psychiatry*, *8*(3), 515–527. doi:10.1080/09585189708412273

Ingram, G. L., Gerard, R. E., Quay, H. C., & Levinson, R. B. (1970). An experimental program for the psychopathic delinquent: Looking in the "correctional wastebasket." *Journal of Research in Crime & Delinquency*, *7*(1), 24–30. doi:10.1177/002242787000700103

Jones, M. (1952). *Social psychiatry: A study of therapeutic communities*. London, UK: Tavistock.

Kadden, R. M., Cooney, N. L., Getter, H., & Litt, M. D. (1989). Matching alcoholics to coping skills or interactional therapies: Posttreatment results. *Journal of Consulting and Clinical Psychology*, *57*(6), 698–704.

Kazdin, A. E., Siegel, T. C., & Bass, D. (1992). Cognitive problem-solving skills training and parent management training in the treatment of antisocial behavior in children. *Journal of Consulting and Clinical Psychology*, *60*(5), 733–747. doi:10.1037//0022-006x.60.5.733

Kendall, P. C., & Hollon, S. D. (1979). Cognitive-behavioral interventions: Overview and current status. In P. C. Kendall & S. D. Hollon (Eds.), *Cognitive-behavioral interventions: Theory, research, and procedures*, 1–9. New York, NY: Academic Press.

Kiger, R. S. (1967). Treating the psychopathic patient in a therapeutic community. *Hospital & Community Psychiatry*, *18*, 191–196. doi:10.1176/ps.18.7.191

Langton, C. M., Barbaree, H. E., Harkins, L., & Peacock, E. J. (2006). Sex offenders' response to treatment and its association with recidivism as a function of psychopathy. *Sexual Abuse*, *18*(1), 99–120. doi:10.1177/107906320601800107

Latzman, R. D., Patrick, C. J., Freeman, H. D., Schapiro, S. J., & Hopkins, W. D. (2017). Etiology of triarchic psychopathy dimensions in chimpanzees (*Pan troglodytes*). *Clinical Psychological Science*, *5*(2), 341–354. doi:10.1177/2167702616676582

Lilienfeld, S. O. (1998). Methodological advances and developments in the assessment of psychopathy. *Behaviour Research and Therapy*, *36*, 99–125. doi:10.1016/s0005-7967(97)10021-3

Lilienfeld, S. O., Patrick, C. J., Benning, S. D., Berg, J., Sellbom, M., & Edens, J. F. (2012). The role of fearless dominance in psychopathy: Controversies, confusions, and clarifications. *Personality Disorders*, *3*, 327–340. doi:10.1037/a0026987

Lilienfeld, S. O., Smith, S. F., Sauvigné, K. C., Patrick, C. J., Drislane, L. E., Latzman, R. D., & Krueger, R. F. (2016). Is boldness relevant to psychopathic personality? Meta-analytic relations with non-psychopathy checklist-based measures of psychopathy. *Psychological Assessment*, *28*, 1172–1185. doi:10.1037/pas0000244

Lilienfeld, S. O., Watts, A. L., Smith, S. F., Berg, J. M., & Latzman, R. D. (2015). Psychopathy deconstructed and reconstructed: Identifying and assembling the personality building blocks of Cleckley's chimera. *Journal of Personality*, *83*, 593–610. doi:10.1111/jopy.12118

Logan, C., Rypdal, K., & Hoff, H. A. (2012). Understanding, treating and managing psychopathy: Moving on. *International Journal of Forensic Mental Health*, *11*(4), 239–241. doi:10.1080/14999013.2012.747574

Lösel, F. (1995). The efficacy of correctional treatment: A review and synthesis of meta-evaluations. In J. McGuire (Ed.), *What works: Reducing reoffending—Guidelines from research and practice* (pp. 79–111). Chichester, UK: John Wiley & Sons.

Lösel, F. (1998). Treatment and management of psychopaths. In D. J. Cooke, A. E. Forth, & R. D. Hare (Eds.), *Psychopathy: Theory, research and implications for society* (pp. 303–354). Dordrecht, Netherlands: Kluwer.

Lösel, F. (2001). Evaluating the effectiveness of correctional programs: Bridging the gap between research and practice. In G. A. Bernfeld, D. P. Farrington, & A. W. Leschied (Eds.), *Offender rehabilitation in practice* (pp. 67–92). Chichester, UK: John Wiley & Sons.

Lösel, F., & Köferl, P. (1989). Evaluation research on correctional treatment in West Germany: A meta-analysis. In H. Wegener, F. Lösel, & J. Haisch (Eds.), *Criminal behavior and the justice system* (pp. 334–355). Berlin, Germany: Springer.

Lykken, D. T. (1995). *The antisocial personalities*. Hillsdale, NJ: Psychology Press.

Lynam, D. R., & Miller, J. D. (2012). Fearless dominance and psychopathy. *Personality Disorders*, *3*(3), 341–353. doi:10.1037/a0028296

Maas, J. (1966). The use of actional procedures in group psychotherapy with sociopathic women. *International Journal of Group Psychotherapy*, *16*(2), 190–197. doi:10.1080/00207284.1966.11642924

Mahoney, M. J. (1974). *Cognition and behavior modification*. Cambridge, MA: Ballinger.

Marcus, D. K., John, S. L., & Edens, J. F. (2004). A taxometric analysis of psychopathic personality. *Journal of Abnormal Psychology*, *113*(4), 626–635. doi:10.1037/0021-843x.113.4.626

McCord, W., & McCord, J. (1964). *The psychopath: An essay on the criminal mind*. Oxford, UK: D. van Nostrand.

Megargee, E. I. (1972). *The California Psychological Inventory handbook*. San Francisco, CA: Jossey-Bass.

Meichenbaum, D. (1977). *Cognitive-behavior modification: An integrative approach*. New York, NY: Plenum Press.

Miller, J. D., & Lynam, D. R. (2012). An examination of the Psychopathic Personality Inventory's nomological network: A meta-analytic review. *Personality Disorders*, *3*(3), 305–326. doi:10.1037/a0024567

Millon, T. (2011). *Disorders of personality: Introducing a DSM/ICD spectrum from normal to abnormal*. Hoboken, NJ: John Wiley & Sons.

Mobbs, D., Lau, H. C., Jones, O. D., & Frith, C. D. (2007). Law, responsibility, and the brain. *PLOS Biology*, *5*(4), e103. doi:10.1371/journal.pbio.0050103

Monti, P. M., Abrams, D. B., Kadden, R. M., & Cooney, N. L. (1989). *Treating alcohol dependence: A coping skills training guide*. New York, NY: Guilford Press.

Neumann, C. S., & Hare, R. D. (2008). Psychopathic traits in a large community sample: Links to violence, alcohol use, and intelligence. *Journal of Consulting and Clinical Psychology*, *76*(5), 893–899. doi:10.1037/0022-006x.76.5.893

Neumann, C. S., Hare, R. D., & Newman, J. P. (2007). The super-ordinate nature of the Psychopathy Checklist–Revised. *Journal of Personality Disorders*, *21*(2), 102–117. doi:10.1521/pedi.2007.21.2.102

Neumann, C. S., Malterer, M. B., & Newman, J. P. (2008). Factor structure of the Psychopathic Personality Inventory (PPI): Findings from a large incarcerated sample. *Psychological Assessment*, *20*, 169–174. doi:10.1037/1040-3590.20.2.169

Newman, J. P., & Kosson, D. S. (1986). Passive avoidance learning in psychopathic and nonpsychopathic offenders. *Journal of Abnormal Psychology*, *95*(3), 252–256. doi:10.1037//0021-843x.95.3.252

Ogloff, J. R., Wong, S., & Greenwood, A. (1990). Treating criminal psychopaths in a therapeutic community program. *Behavioral Sciences & the Law*, *8*(2), 181–190. doi:10.1002/bsl.2370080210

Olver, M. E., Lewis, K., & Wong, S. C. (2013). Risk reduction treatment of high-risk psychopathic offenders: The relationship of psychopathy and treatment change to violent recidivism. *Personality Disorders*, *4*(2), 160–167. doi:10.1037/a0029769

Olver, M. E., & Wong, S. C. (2009). Therapeutic responses of psychopathic sexual offenders: Treatment attrition, therapeutic change, and long-term recidivism. *Journal of Consulting and Clinical Psychology*, *77*(2), 328–336. doi:10.1037/a0015001

Patrick, C. J., Venables, N. C., & Drislane, L. E. (2013). The role of fearless dominance in differentiating psychopathy from antisocial personality disorder: Comment on Marcus, Fulton, and Edens. *Personality Disorders*, *4*(1), 80–82. doi:10.1037/a0027173

Patterson, G. R., Dishion, T. J., & Chamberlain, P. (1993). Outcomes and methodological issues relating to treatment of antisocial children. In Giles, Thomas R. (Ed.), *Handbook of effective psychotherapy* (pp. 43–88). New York, NY: Springer.

Polaschek, D. L., & Daly, T. E. (2013). Treatment and psychopathy in forensic settings. *Aggression and Violent Behavior*, *18*(5), 592–603. doi:10.1016/j.avb.2013.06.003

Polaschek, D. L., Wilson, N. J., Townsend, M. R., & Daly, L. R. (2005). Cognitive-behavioral rehabilitation for high-risk violent offenders: An outcome evaluation of the violence prevention unit. *Journal of Interpersonal Violence*, *20*(12), 1611–1627. doi:10.1177/0886260505280507

Quay, H. C. (1965). Psychopathic personality as pathological stimulation-seeking. *American Journal of Psychiatry*, *122*(2), 180–183. doi:10.1176/ajp.122.2.180

Reid, W. H., & Gacono, C. (2000). Treatment of antisocial personality, psychopathy, and other characterologic antisocial syndromes. *Behavioral Sciences & the Law*, *18*(5), 647–662. doi:10.1002/1099-0798(200010)18:5<647::aid-bsl407>3.0.co;2-o

Rice, M. E., Harris, G. T., & Cormier, C. A. (1992). An evaluation of a maximum security therapeutic community for psychopaths and other mentally disordered offenders. *Law and Human Behavior*, *16*(4), 399–412. doi:10.1007/bf02352266

Richards, H. J., Casey, J. O., & Lucente, S. W. (2003). Psychopathy and treatment response in incarcerated female substance abusers. *Criminal Justice and Behavior*, *30*(2), 251–276. doi:10.1177/0093854802251010

Rilling, J. K., Glenn, A. L., Jairam, M. R., Pagnoni, G., Goldsmith, D. R., Elfenbein, H. A., & Lilienfeld, S. O. (2007). Neural correlates of social cooperation and non-cooperation as a function of psychopathy. *Biological Psychiatry*, *61*(11), 1260–1271. doi:10.1016/j.biopsych.2006.07.021

Robins, L. N. (1966). *Deviant children grown up: A sociological and psychiatric study of sociopathic personality*. Baltimore, MD: Williams & Wilkins.

Salekin, R. T. (2002). Psychopathy and therapeutic pessimism: Clinical lore or clinical reality? *Clinical Psychology Review*, *22*(1), 79–112. doi:10.1016/s0272-7358(01)00083-6

Salekin, R. T., Worley, C., & Grimes, R. D. (2010). Treatment of psychopathy: A review and brief introduction to the mental model approach for psychopathy. *Behavioral Sciences & the Law*, *28*(2), 235–266. doi:10.1002/bsl.928

Sampson, R. J., & Laub, J. H. (1997). A life-course theory of cumulative disadvantage and the stability of delinquency. *Developmental Theories of Crime and Delinquency*, *7*, 133–161.

Serin, R. C., & Kuriychuk, M. (1994). Social and cognitive processing deficits in violent offenders: Implications for treatment. *International Journal of Law and Psychiatry*, *17*(4), 431–441. doi:10.1016/0160-2527(94)90018-3

Seto, M. C., & Barbaree, H. E. (1999). Psychopathy, treatment behavior, and sex offender recidivism. *Journal of Interpersonal Violence*, *14*(12), 1235–1248. doi:10.1177/088626099014012001

Sherman, L. W. (1993). Defiance, deterrence, and irrelevance: A theory of the criminal sanction. *Journal of Research in Crime & Delinquency*, *30*(4), 445–473. doi:10.1177/0022427893030004006

Skeem, J. L., & Cooke, D. J. (2010). Is criminal behavior a central component of psychopathy? Conceptual directions for resolving the debate. *Psychological Assessment*, *22*, 433–445. doi:10.1037/a0008512

Skeem, J. L., Monahan, J., & Mulvey, E. P. (2002). Psychopathy, treatment involvement, and subsequent violence among civil psychiatric patients. *Law and Human Behavior*, *26*(6), 577–603. doi:10.1023/a:1020993916404

Skeem, J. L., Polaschek, D. L., & Manchak, S. (2009). Appropriate treatment works, but how? Rehabilitating general, psychopathic, and high-risk offenders. In J. L. Skeem, K. S. Douglas, & S. O. Lilienfeld (Eds.), *Psychological science in the courtroom: Consensus and controversy* (pp. 358–384). New York, NY: Guilford Press.

Skeem, J. L., Polaschek, D. L., Patrick, C. J., & Lilienfeld, S. O. (2011). Psychopathic personality: Bridging the gap between scientific evidence and public policy. *Psychological Science in the Public Interest*, *12*, 95–162. doi:10.1177/1529100611426706

Spitzer, R. L., Williams, J. B., & Skodol, A. E. (1980). DSM-III: The major achievements and an overview. *American Journal of Psychiatry*, *137*(2), 151–164. doi:10.1176/ajp.137.2.151

Suedfeld, P., & Landon, P. B. (1978). Approaches to treatment. *Psychopathic Behavior*, 347–376.

Walsh, A., & Wu, H. H. (2008). Differentiating antisocial personality disorder, psychopathy, and sociopathy: Evolutionary, genetic, neurological, and sociological considerations. *Criminal Justice Studies*, *21*(2), 135–152. doi:10.1080/14786010802159814

Watt, M., Stewart, S., Birch, C., & Bernier, D. (2006). Brief CBT for high anxiety sensitivity decreases drinking problems, relief alcohol outcome expectancies, and conformity drinking motives: Evidence from a randomized controlled trial. *Journal of Mental Health*, *15*(6), 683–695. doi:10.1080/09638230600998938

Widiger, T. A., Cadoret, R., Hare, R., Robins, L., Rutherford, M., Zanarini, M., … & Frances, A. (1996). DSM-IV antisocial personality disorder field trial. *Journal of Abnormal Psychology*, *105*(1), 3–16.

Wong, S. C., Gordon, A., Gu, D., Lewis, K., & Olver, M. E. (2012). The effectiveness of violence reduction treatment for psychopathic offenders: Empirical evidence and a treatment model. *International Journal of Forensic Mental Health*, *11*(4), 336–349. doi:10.1080/14999013.2012.746760

Wong, S. C., & Hare, R. D. (2005). *Guidelines for a psychopathy treatment program*. Toronto, ON: Multi-Health Systems.

Wong, S. C., & Olver, M. E. (2015). Risk reduction treatment of psychopathy and applications to mentally disordered offenders. *CNS Spectrums*, *20*(3), 303–310. doi:10.1017/s1092852915000322

Yang, Y., & Raine, A. (2009). Prefrontal structural and functional brain imaging findings in antisocial, violent, and psychopathic individuals: A meta-analysis. *Psychiatry Research: Neuroimaging*, *174*(2), 81–88. doi:10.1016/j.pscychresns.2009.03.012

Yang, Y., Raine, A., Narr, K. L., Colletti, P., & Toga, A. W. (2009). Localization of deformations within the amygdala in individuals with psychopathy. *Archives of General Psychiatry*, *66*(9), 986–994. doi:10.1001/archgenpsychiatry.2009.110

Yochelson, S., & Samenow, S. E. (1977). *The criminal personality* (Vols. 1 & 2). New York, NY: Jason Aronson.

12

The Treatment of Borderline Personality Disorder

Lorie A. Ritschel,[1] Colleen M. Cowperthwait,[2] Lindsay M. Stewart,[3] and Noriel E. Lim[3]

[1] *Department of Psychiatry, University of North Carolina School of Medicine, Chapel Hill, USA*

[2] *Department of Psychiatry and Behavioral Sciences, Duke University Medical Center, Durham, USA*

[3] *Department of Psychiatry and Behavioral Sciences, Emory University School of Medicine, Atlanta, USA*

Borderline personality disorder (BPD) is characterized by pervasive and often debilitating difficulties in the emotional, behavioral, interpersonal, intrapersonal, and cognitive spheres (Linehan, 1993). According to the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013), an individual must meet at least five of the following nine criteria to be diagnosed with BPD: (1) frantic efforts to avoid real or imagined abandonment; (2) a pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation; (3) markedly and persistently unstable self-image or sense of self; (4) impulsivity in at least two areas that are potentially self-damaging (e.g., substance abuse, binge eating, reckless driving); (5) recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior; (6) affective instability due to a marked reactivity of mood; (7) chronic feelings of emptiness; (8) inappropriate, intense anger or difficulty controlling anger; and (9) transient, stress-related paranoid ideation or severe dissociative symptoms. In order to meet the full criteria for a BPD diagnosis, these symptoms must be present by early adulthood and must be present across a variety of situations.

Epidemiological studies have yielded varying lifetime prevalence rates for BPD in the United States. Results from the National Comorbidity Survey ($n = 5{,}692$; Lenzenweger, Lane, Loranger, & Kessler, 2007) found that 1.2% of the adult population met criteria for BPD. In the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC; $n = 43{,}093$; Grant et al., 2008), 5.9% of adults met criteria for BPD, although that rate dropped to 2.7% when the specifier that BPD symptoms must be accompanied by significant distress or impairment was included (Trull, Jahng, Tomko, Wood, & Sher, 2010). Rates of BPD have consistently been found to be higher in clinical samples: approximately 10% of outpatients (Zimmerman, Rothschild, & Chelminski, 2005) and 15% of inpatients meet criteria for the diagnosis (Widiger & Weissman, 1991).

In terms of demographic correlates, many studies report that BPD is more common in women; however, results from the NESARC found no significant differences in rates of BPD across genders. Research assessing prevalence rates among different racial and ethnic populations has produced mixed results. Results from the NESARC found that

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

BPD was more common in Native Americans and less common among Hispanics (Grant et al., 2008). Some epidemiological studies, however, show no differences in prevalence rates of BPD between racial and ethnic groups (e.g., Lenzenweger et al., 2007). BPD appears to be more common in individuals in lower income brackets and in those with a high school degree or less than high school educations (Grant et al., 2008).

BPD is strongly associated with debilitating psychological and functional impairment and is highly comorbid with other psychiatric disorders. Zanarini and colleagues (1998) evaluated a sample of 379 patients with BPD and found considerable lifetime comorbidity: 96% of individuals with BPD also met criteria for a mood disorder, 64% met criteria for a substance use disorder, 88% met criteria for an anxiety disorder, and 53% met criteria for an eating disorder. The NESARC evaluated comorbidity of lifetime BPD and 12-month prevalence rates for other psychiatric disorders and found that patients demonstrated high rates of comorbidity with mood (51%), anxiety (60%), and substance use (51%) disorders. On average, individuals with BPD meet criteria for 3.4–4.2 additional psychiatric diagnoses (Harned, Rizvi, & Linehan, 2010). In addition, BPD is strongly associated with dysfunctional behaviors such as nonsuicidal self-injury (NSSI; e.g., cutting, burning) and chronic suicidality. Studies have shown that approximately 10% of individuals with BPD eventually die by suicide (Paris, Brown, & Nowlis, 1987; Paris & Zweig-Frank, 2001). In comparison to other psychiatric groups (e.g., other personality, mood, or anxiety disorders), individuals with BPD show greater interpersonal difficulties; are more impaired in occupational, home, and recreational activities; are higher users of both psychiatric and nonpsychiatric care; and have lower scores on measures of global satisfaction than their non-BPD peers (Ansell, Sanislow, McGlashan, & Grilo, 2007).

12.1 Treatments for Borderline Personality Disorder

In this chapter, we review the available psychotherapies for BPD in terms of both research support for the overall therapy and support for the putative mechanism of change that underlies the treatment. We then classify each treatment according to the nine-category taxonomy articulated by David and Montgomery (2011). These authors note that a "well-supported" psychotherapy must demonstrate empirical support in at least two different studies conducted by two different teams of researchers. In addition, they argue that in any scientifically oriented psychotherapy "there should be a correspondence between the mechanisms of treatment ('mechanism/theory of change') and the mechanisms of the disorder ('theory of disorder')" (p. 91). Thus, the strongest of the treatments has empirical support for both the overall treatment and the mechanism of change that underlies the treatment (i.e., Category I), and the weakest of the treatments shows strong contradictory evidence for both the treatment and the mechanism of change (i.e., Category IX).

Given the extent of the high-risk behaviors (e.g., suicide, NSSI) and substantial comorbidities that most patients exhibit, treatment for BPD is a complex endeavor. As such, relatively few comprehensive treatments for BPD exist. In the most recent Cochrane Review, Stoffers and colleagues (2012) found seven comprehensive treatments designed or adapted for BPD that had any research support: cognitive–behavioral therapy (Beck, Rush, Shaw, & Emery, 1979), dialectical behavior therapy (DBT; Linehan, 1993), dynamic

deconstructive therapy (Gregory et al., 2008), interpersonal psychotherapy (Klerman, Weissman, Rounsaville, & Chevron, 1984), mentalization-based therapy (MBT; Bateman & Fonagy, 2004, 2006), schema-focused therapy (Young, 1994), and transference-focused psychotherapy (Yeomans, Clarkin, & Kernberg, 2002). The authors note that a number of psychotherapies exist that are "noncomprehensive"; that is, they do not include individual psychotherapy as a substantial treatment component (for a review, see Stoffers et al., 2012). Less research has been conducted on the mechanisms of change in treatments for BPD and, problematically, not all therapies that do exist are based on a cogent theory of the development of BPD.

Given the paucity of empirically supported treatments for BPD and the lack of studies on treatment mechanisms, no existing psychotherapies for BPD meet the standards of Categories I (i.e., strong evidence for both the treatment and the mechanism of change) or III (i.e., preliminary or mixed evidence for the treatment and strong evidence for the mechanism of change) based on David and Montgomery's (2011) taxonomy. Fortunately, there also are no psychotherapies for which strong contradictory evidence exists (Categories V–IX). At the present time, two treatments meet the Category II criteria (DBT and MBT), and the rest meet the Category IV criteria. Category II includes well-supported therapies with equivocal (none, preliminary, or mixed) evidence in support of the theory of change, and Category IV includes treatments with equivocal (none, preliminary, or mixed) support for both the treatment and the theory of change. We will review the available evidence for each of these treatments, the mechanisms of change thought to underpin each treatment, and the available evidence in support of these purported mechanisms. We first review Category II treatments and then move to Category IV.

12.2 Category II

12.2.1 Dialectical Behavior Therapy

DBT is a multimodal, principle-based therapy developed to enhance emotion regulation by balancing acceptance of the individual's emotional experience with problem-solving to promote behavioral change. DBT is based on dialectical theory, Zen philosophy, and behaviorism. Standard outpatient DBT includes concomitant weekly individual and group skills training, as-needed telephone coaching, and weekly consultation team meetings for therapists. Clients are generally expected to make a 1-year commitment to treatment (Linehan, 1993). Individual therapy is highly structured, and each session begins with setting an agenda. DBT therapists prioritize treatment targets as follows: decreasing life-threating behaviors (e.g., suicidal or self-harming behaviors), decreasing therapy-interfering behaviors (e.g., dropout or nonattendance), decreasing quality of life-interfering behaviors (e.g., mood disorders, employment or relationship difficulties), and increasing behavioral skills (e.g., distress tolerance, mindfulness, interpersonal effectiveness, and emotion regulation). Weekly skills training groups focus on teaching skills developed to target emotional, behavioral, interpersonal, cognitive, and social dysregulation skill deficits. The individual therapist provides as-needed intersession telephone coaching focused on helping patients generalize skills into their natural environments. Weekly therapist consultation team meetings are considered a critical part of treatment and are designed to provide support for therapists, decrease therapist burnout, increase DBT treatment adherence, and promote effective skills use by DBT

therapists (for a succinct overview of standard outpatient DBT, see Rizvi, Steffel, & Carson-Wong, 2013).

12.2.1.1 Evidence for therapy

12.2.1.1.1 Adult samples

According to the latest Cochrane Review (Stoffers et al., 2012), DBT is the most robust psychological therapy for treating BPD and is effective in reducing suicide attempts, self-harm, and anger while improving general functioning. A number of randomized controlled trials (RCTs), open trials, and quasi-experimental studies of DBT's efficacy and effectiveness in treating BPD in outpatient settings have been conducted, and evidence in support of DBT has emerged from the treatment developer (Marsha Linehan) as well as numerous independent laboratories. Linehan and colleagues examined 1 year of outpatient DBT for women with BPD compared to treatment as usual (TAU; Linehan, Armstrong, Suarez, Allmon, & Heard, 1991; Linehan, Tutek, Heard, & Armstrong, 1994) and community treatment by experts (Linehan et al., 2006) and found that DBT outperformed TAU and treatment by experts on measures of frequency and severity of NSSI, inpatient psychiatric days, emergency room (ER) visits, and treatment dropout, as well as interpersonal outcomes such as anger and self- and interview-rated social adjustment. Other research groups have demonstrated similar results in RCTs conducted in a variety of outpatient settings: in a 1-year trial conducted in the Netherlands, Verheul and colleagues (2003) found that DBT was superior to clinical management for women with BPD; in a 6-month trial with female veterans, Koons and colleagues (2001) found that DBT was superior to TAU; in a 1-year trial with males and females with BPD, Turner (2000) found that DBT was superior to client-centered supportive psychotherapy; and, in a 1-year trial with college-aged men and women with NSSI or a suicide attempt and BPD traits, Pistorello, Fruzzetti, MacLane, Gallop, and Iverson (2012) found that DBT was superior to TAU. Across these four studies, attrition rates were lower than in the comparison conditions; participants showed greater improvements on measures of depression and social adjustment as well as severity and frequency of suicidal behaviors and NSSI; and participants showed greater reductions in emergency room visits and inpatient hospitalizations.

Three RCTs have been conducted in which DBT has been compared to an active treatment condition and found to be equal but not superior to the control conditions on various outcome measures. In one RCT, patients were randomized to DBT, transference-focused psychotherapy, or supportive psychotherapy (Clarkin, Levy, Lenzenweger, & Kernberg, 2007); in a second RCT, patients were randomized to DBT or psychiatric management (McMain et al., 2009). In both of those trials, significant improvements were seen across groups on a multitude of outcome measures; however, DBT did not significantly outperform other active treatments on outcomes such as frequency and severity of suicidal acts and NSSI, ER visits, hospitalizations, and inpatient days.

Effectiveness research shows that outpatient DBT has been successfully disseminated outside formal research settings and has shown comparable results to RCTs in terms of significant reductions in ER visits, inpatient admissions, and NSSI, as well as significant improvements in quality of life variables (e.g., Bohus et al., 2004; Comtois, Elwood, Holdcraft, & Simpson, 2007; Comtois, Kerbrat, Atkins, Harned, & Elwood, 2010; Evershed et al., 2003). No trials of DBT have demonstrated strong contradictory evidence against DBT as defined by David and Montgomery (2011). Therefore, the bulk

of the evidence to date, by a number of different research groups, substantiates DBT as a well-supported therapeutic package for the treatment of individuals with BPD in a variety of settings.

12.2.1.1.2 Youth samples

DBT for adolescents (DBT-A) includes several adaptations of standard DBT, including shortening the treatment, reducing the number of skills taught, adding skills specific to adolescents and their families, and including families in treatment (Rathus & Miller, 2014). Evidence for DBT with adolescents is preliminary. One RCT (Mehlum et al., 2014) has been conducted to evaluate DBT for adolescents with BPD traits (defined as at least two episodes of self-harm with or without suicidal intent and at least two DSM-5 criteria of BPD or one DSM-5 criteria at full threshold *and* two subthreshold criteria). Treatment comprised weekly individual and group therapy that occurred over the course of 19 weeks. Adolescents in both DBT-A and enhanced usual care demonstrated mean reductions in suicidal ideation severity and self-harm frequency, self- and interviewer-rated depression, and borderline symptoms. However, DBT-A significantly outperformed enhanced usual care on outcomes of self-harm frequency, severity of suicidal ideation, and depressive symptoms. There are also a number of nonrandomized, quasi-experimental trials comparing DBT-A to TAU (e.g., Fleischhaker et al., 2011; Rathus & Miller, 2002; Woodberry & Popenoe, 2008); across research groups, these trials demonstrate that DBT-A is associated with significant reductions in suicidal ideation, depression, anxiety, BPD symptoms, overall psychiatric symptoms, and functional impairment.

Although an adaptation of DBT for children has been described in the literature (Perepletchikova et al., 2011), no data have yet been reported for this age group.

12.2.1.2 Theory of the disorder

Linehan (1993) has proposed a biosocial theory to explain the etiology of BPD. According to this theory, the severe emotion dysregulation seen in BPD is a consequence of the transaction between innate biological dysfunction in an individual's emotion regulation system and an invalidating environment. Linehan posits that the specific deficits in an individual's emotion regulation system include increased baseline emotional sensitivity, increased reactivity to emotionally evocative stimuli, and a slow return to emotional baseline. An invalidating environment is any environment that communicates to the individual that his or her emotional experiences are wrong, too extreme, or unacceptable. According to the theory, there is insufficient modeling, coaching, and cheerleading of effective expressions of emotion, often in concert with either excessive punishment or intermittent reinforcement of extreme expressions of emotion. Over time, the biologically vulnerable individual and the environment shape and reinforce extreme behaviors in each other. This transaction leads to BPD symptomatology, characterized by emotion dysregulation and maladaptive behaviors by the individual in an attempt to regulate negative affect and/or inhibit emotional responses. As such, DBT was designed to teach individuals to regulate their emotions more effectively and without having to rely on extreme regulatory measures, such as self-harm or substance use.

Evidence for the biosocial theory is preliminary and equivocal. Individuals with BPD consistently report elevated emotional intensity in laboratory tasks (Bland, Williams, Scharer, & Manning, 2004; Yen, Zlotnick, & Costello, 2002). In addition, they report

greater frequency and intensity of negative emotions and fewer positive emotions in reaction to ordinary life events in naturalistic contexts (Ebner-Priemer et al., 2007). Further, individuals with BPD report more difficulties with affect control, or the perceived ability or skill to regulate emotion (Salsman & Linehan, 2012; Yen et al., 2002). Imaging studies in adults with BPD have suggested structural and functional abnormalities in structures within the brain thought to be involved in emotion regulation, including reduced volume in the hippocampus, amygdala, anterior cingulate cortex, and dorsolateral, prefrontal, and orbitofrontal cortexes as well as differential patterns of activation in response to emotional stimuli and faces compared to healthy controls (Courtney-Seidler, Klein, & Miller, 2013; Crowell, Beauchaine, & Linehan, 2009; Schmahl & Bremner, 2006; Sharp & Romero, 2007). These brain areas are involved in impaired neurotransmission of serotonin, which has been associated with impulsivity in BPD (Schmahl & Bremner, 2006; Sharp & Romero, 2007).

The findings regarding psychophysiological indicators of emotional reactivity and slow return to baseline have been less consistent. Studies of psychophysiological indicators of stress reactivity among adults with BPD find higher baseline cortisol, more cortisol reactivity, and delayed cortisol recovery in response to interpersonal stressors compared to healthy controls, indicating that altered reactivity of the stress response system may play a role in emotional dysregulation in individuals with BPD (Crowell et al., 2009; Lieb et al., 2004; Walter et al., 2008). With respect to indicators of emotion regulation capabilities, there is some evidence that individuals with BPD have higher baseline negative emotional intensity and lower baseline respiratory sinus arrhythmia than healthy controls or individuals with social anxiety disorder (Kuo & Linehan, 2009). However, the same study did not find support for the hypothesis of higher emotional reactivity in individuals with BPD, as indicated by greater changes from baseline to emotion-eliciting tasks, compared to either healthy controls or individuals with social anxiety disorder (Kuo & Linehan, 2009). No study to date has supported the idea that individuals with BPD demonstrate a slower return to baseline after an emotional prompt (Harned, Banawan, & Lynch, 2006).

There is some evidence for the association between an invaliding environment and later emotion dysregulation and BPD. Low parental affection (e.g., perceived paternal overprotection, low maternal care, and lack of empathy) and aversive parenting behavior (e.g., verbal abuse) are associated with elevated risk for the development of BPD in adulthood (Cohen, Crawford, Johnson, & Kasen, 2005; Johnson, Cohen, Chen, Kasen, & Brook, 2006). Low perceived quality of relationships between parents and young adults has been associated with high baseline cortisol and cortisol hyperreactivity after a discussion of family conflict among young adults with BPD, indicating a significant relationship between a perceived invalidating environment and physiological indicators of emotion dysregulation (Lyons-Ruth, Choi-Kain, Pechtel, Bertha, & Gunderson, 2011).

12.2.1.3 Theory of change

According to the DBT model, emotion dysregulation and skills deficits are the central problems for individuals with BPD, and skills acquisition in the service of emotion regulation is the primary mechanism of change. Recent mediation analyses (Neacsiu, Rizvi, & Linehan, 2010), dismantling studies (Linehan et al., 2015), and RCTs (Neacsiu, Eberle, Kramer, Wiesmann, & Linehan, 2014) provide preliminary support for this theory. Post hoc analyses of previous RCTs (Linehan et al., 1999, 2002, 2006) indicate that

DBT skills use fully mediates reductions in suicide attempts and depressive symptoms as well as increases in anger control, and skills use also partially mediates decreases in NSSI (Neacsiu et al., 2010). More recently, Linehan and colleagues (2015) conducted a component analysis comparing standard DBT, DBT skills training without individual DBT, and individual DBT without skills training. Both standard DBT and DBT skills without individual DBT significantly outperformed individual DBT without skills training on measures of NSSI frequency as well as depression and anxiety severity. These results indicate that DBT skills training, even in the absence of other DBT modalities, may drive reductions in maladaptive behaviors and improvements in emotion regulation (Neacsiu et al., 2014).

Few treatment studies among adults have demonstrated changes in emotion regulation using either subjective (self-reported or clinician-rated) or objective (physiological or neurological markers) measures over the course of treatment. Two small studies have utilized neuroimaging to demonstrate changes in reactivity to emotionally evocative pictorial stimuli over the course of treatment (Goodman et al., 2014; Schnell & Herpertz, 2007). Compared to pretreatment functional magnetic resonance imaging (fMRI) scans, adults with BPD who completed DBT demonstrated decreased amygdala activation to emotionally evocative pictures and improved amygdala habituation to repeated exposures to emotional pictures. These changes were associated with improved self-reported emotion regulation (Goodman et al., 2014). In another fMRI study, adults with BPD who completed a 12-week inpatient DBT treatment program and were clinician-rated treatment responders demonstrated neural changes in limbic and cortical regions associated with emotion regulation over the course of treatment, including reduced amygdala activation (Schnell & Herpertz, 2007). These studies demonstrate preliminary evidence that DBT is associated with improved emotion regulation and that improved self-reported emotion regulation over the course of treatment is associated with changes in relevant neural systems.

12.2.1.4 Summary

Based on David and Montgomery's (2011) framework for evaluating psychosocial interventions, DBT for adults with BPD can be considered a Category II psychotherapy: strong support exists for the treatment, and preliminary data support the theory that underpins the treatment. Evidence for the underlying theory of specific mechanisms of change is promising but preliminary. DBT-A can be considered a Category IV psychotherapy: preliminary data support the treatment, but no studies have examined skills acquisition as a mechanism of change in DBT-A.

12.2.2 Mentalization-Based Therapy

MBT (Bateman & Fonagy, 2004, 2006) is a psychodynamic treatment that draws from both attachment and cognitive theories. MBT is a manualized therapy that includes both individual and group components and aims to improve patients' ability to "mentalize," which is the "mental process by which an individual implicitly and explicitly interprets the actions of himself and others as meaningful on the basis of intentional mental states such as personal desires, needs, feelings, beliefs, and reasons" (Bateman & Fonagy, 2004, p. 21). Thus, mentalizing involves awareness of both the self and others from the perspectives of both observable behaviors and internal states. Patients are taught to learn to

see themselves as others see them while also knowing who they are implicitly and being able to develop a sense of self that remains constant even in the face of contradictory feedback from others. Treatment is conducted in individual and group therapy sessions and lasts approximately 18 months.

In MBT, borderline patients are hypothesized to have particular difficulty engaging in reflective functioning (i.e., mentalizing) in the context of interpersonal interactions. This difficulty is purported to have developed as a result of dysfunctional attachment relationships in the developmental years. According to the theory, strong attachment foundations promote the development of the ability to mentalize, allowing the individual to correctly infer relationships between observed behaviors and related intentional motives, states, or drives. Deficits in mentalization can be temporary (i.e., likely to arise in a time of distress) or more pervasive; either way, the inability to engage in mentalization leads to feeling overwhelmed or disconnected from others, resulting in emotional instability and a return to "prementalistic states." The three prementalizing states that are addressed in therapy are known as "psychic equivalence," "pretend mode," and "teleological stance." In psychic equivalence, thoughts and other mental states are perceived as concrete reality. When individuals enter this state, they can become highly inflexible to alternative views or can become overwhelmed by the intensity and perceived reality of their thoughts (e.g., flashbacks, negative self-directed cognitions). In pretend mode, there is little to no relationship between internal and external experiences. Pretend mode is hypothesized to be linked with the BPD symptoms of dissociation and emptiness. Finally, in teleological stance, individuals often assume that emotional problems can be solved through physical action (e.g., anger can be resolved by destroying something). Alternatively, individuals communicate their internal states to others via external actions (e.g., self-harm) and often expect others to do the same (e.g., statements of affection are insufficient; actions to demonstrate affection are expected). As such, the task of the MBT therapist is to help patients understand when their ability to mentalize "goes offline" and to help patients reengage in effective mentalization.

12.2.2.1 Evidence for therapy

12.2.2.1.1 Adult samples

In the first randomized trial of MBT, the treatment was delivered in a partial hospital program setting and was compared to routine psychiatric care (Bateman & Fonagy, 1999). After 18 months of treatment, patients in the MBT condition showed statistically superior outcomes on measures of suicidal and self-harming behaviors, depression, and interpersonal functioning. Notably, patients in this study were followed for an additional 8 years after the conclusion of active treatment (Bateman & Fonagy, 2008). Patients in the MBT condition continued to demonstrate superior outcomes in comparison to the control group; at follow-up, 18% of patients in the MBT condition continued to meet criteria for BPD as compared to 87% of patients in the TAU group. However, the authors noted that global functioning in both groups continued to be rather impaired.

Two RCTs of MBT have been completed in outpatient samples with BPD patients. In the first, 134 individuals with BPD were randomized to either MBT or Structured Clinical Management (Bateman & Fonagy, 2009). Patients in both conditions improved on all outcome variables, although the results favored MBT on some outcomes both in terms of the rapidity of change (e.g., reduced suicide attempts and severe self-harm) and extent of improvement (e.g., number of days hospitalized). The second RCT was completed by

an independent group of researchers (Jørgensen et al., 2013). A total of 58 patients were randomized to 2 years of either MBT or supportive group therapy. Participants in both groups demonstrated significant improvements on measures of depression, general and social functioning, and number of BPD symptoms endorsed. MBT outperformed supportive therapy on the general functioning measure.

MBT has been adapted for use with patients with eating disorders and symptoms of BPD (Robinson et al., 2014) as well as for major depression (Jakobsen et al., 2012), and RCTs are underway with both populations. No data have yet been published on these adaptations, however. No strong contradictory evidence against MBT has emerged in the literature.

12.2.2.1.2 Youth samples

One RCT has been conducted using MBT with adolescents (MBT-A; Rossouw & Fonagy, 2012). The treatment was delivered over a 1-year period and was compared to TAU in a sample of 80 self-harming youth with depression. Participants in both groups demonstrated significant reductions in NSSI and risk-taking behavior; MBT-A significantly outperformed TAU in terms of reducing NSSI, but there were no differences between groups on risk-taking behavior. MBT-A was associated with significantly greater decreases in depression over the course of treatment. Although an adaptation of MBT for use with children has been described in the clinical literature (Muller & Midgley, 2015), no studies have yet been conducted with this age group.

12.2.2.2 Theory of the disorder

According to the MBT theory, BPD develops as the result of disrupted attachments early in life that are combined with traumatic attachments throughout development. In this model, the attachment system becomes hypersensitive and hyperresponsive, impairing one's capacity to mentalize. Moreover, the ability to mentalize destabilizes even further during times of emotional arousal. Bateman and Fonagy (2010) have proposed a four-part model to explain the development of BPD. First, they hypothesize that individuals who will go on to develop BPD are born with biological vulnerabilities and/or are exposed to trauma early in life. Second, as a consequence of the combination of biological vulnerability and early childhood trauma, the development of the social and cognitive processes required to engage in the process of mentalizing is impaired. They propose that these difficulties become especially pronounced when the individual's emotional experience is incongruent with the emotions that a caregiver mirrors back to the child. Third, these social and/or cognitive deficits result in a hypersensitive attachment system. Fourth, this alteration in attachment destabilizes the individual's ability to modulate affect and attention, particularly in interpersonal contexts. By extension, the treatment is designed to improve patients' ability to regulate their own neuropsychological system (Fonagy & Bateman, 2006).

The evidence in support of this model is preliminary. Ample research supports the first two components of the theory. First, attachment is indeed disrupted in BPD (see Levy, 2005), and considerable evidence demonstrates that many individuals with BPD experienced early childhood trauma or neglect (Bandelow et al., 2005; Golier et al., 2003). In addition, as reviewed in Section 12.2.1.2, some evidence supports the theory that individuals with BPD are more vulnerable to their emotions and more reactive in the face of emotionally evocative or invalidating stimuli. In a review of the literature, Roepke,

Vater, Preißler, Heekeren, and Dziobek (2013) highlight a number of social and cognitive impairments in individuals with BPD, although they note that more research needs to be conducted to further elucidate the conditions under which these impairments occur. Less research has emerged, however, in support of the concept of the hypersensitive attachment system. Fonagy and Bateman (2006) review the conceptual and animal models in support of this aspect of the theory, but they note that the research to date is lacking in humans. Equivocal evidence exists regarding the ability of BPD patients to accurately perceive the emotions, thoughts, and intentions of others, although the majority of data suggest that impairments do exist (for a review, see Roepke et al., 2013).

12.2.2.3 Theory of change

Mechanisms of change have not been explicitly investigated in studies of MBT in adults, and Bateman and Fonagy (2009) have noted that future studies of MBT should attempt to fill this gap in the literature. The RCT of MBT-A (Rossouw & Fonagy, 2012) included two measures designed to assess mechanisms of change: The How I Feel questionnaire assessed ability to mentalize and the Experience of Close Relationships Inventory assessed attachment status. Results showed that participants in the MBT-A but not the TAU group showed significant improvements in the ability to mentalize, and participants in the MBT-A group showed significantly decreased attachment avoidance ratings as compared to the TAU group. Although formal mediation analyses were not conducted, results showed that changes in these two constructs over the course of treatment predicted lower NSSI at the end of treatment.

12.2.2.4 Summary

Based on David and Montgomery's (2011) framework for evaluating psychosocial interventions, MBT for adults can be considered a Category II psychotherapy. Support for the treatment has been demonstrated in two independent laboratories. In terms of support for the theory that underpins the treatment, considerable evidence supports parts of the theory, equivocal evidence supports other parts, and there are no data for yet other parts. No strong contradictory evidence has been published refuting the theory that underpins the treatment. MBT-A can be considered a Category IV psychotherapy: preliminary data support both the treatment and the theory that underpins the treatment. Evidence for the underlying theory for specific mechanisms of change is promising but preliminary.

12.3 Category IV

12.3.1 Schema-Focused Therapy

A derivative of Beck's cognitive therapy (Beck, Rush, Shaw, & Emery, 1979), schema-focused therapy (SFT; Young, 1994) was designed to help individuals who are unable to benefit from standard cognitive therapy due to their difficulties with attachment and their enduring, rigid beliefs, which are often associated with maladaptive coping strategies (McGinn & Young, 1996; Young, 1994; Young, Klosko, & Weishaar, 2003). Consistent with the overarching treatment target of cognitive therapy, SFT aims to modify maladaptive schemas (i.e., rigid and unhealthy beliefs about the self and the world).

Unlike standard cognitive therapy, SFT was developed as an integrative and more comprehensive treatment and utilizes techniques drawn from other treatment models. For instance, not only does SFT focus on changing maladaptive thoughts, identifying cognitive errors, practicing experimental tasks to test assumptions, and modifying unhealthy behaviors, it also uses emotion-focused, psychodynamic, and experiential approaches (e.g., gestalt's "empty-chair" technique, visual imagery). Because SFT is based on the notion that individuals with BPD did not experience healthy attachment experiences with parental figures, it highlights the importance of the therapeutic alliance and the role that therapists play in fulfilling individuals' unmet needs. Therapists work on compensating for the deficits in early childhood learning by attending to a person's need for validation and affection while staying within professional boundaries, an approach known as "limited reparenting." These techniques make up the core mechanisms of change and are central to SFT. SFT helps patients cope with various schema modes through encouragement, use of adaptive coping techniques, and learning through therapist modeling. SFT is a long-term treatment; on average, it requires a 3-year commitment, which is divided into three phases (bonding and emotion regulation, schema mode change, and development of autonomy). The initial treatment phase may require patients to schedule multiple sessions (two to three) per week, which is reduced to once weekly in later stages of treatment.

12.3.1.1 Evidence for therapy

Research on SFT has provided modest support for the effectiveness of the treatment (for a comprehensive review, see Sempertegui, Karreman, Arntz, & Bekker, 2013). One RCT has been conducted to examine the effectiveness of SFT against an active, non-SFT comparison condition. Giesen-Bloo and colleagues (2006) compared SFT to transference-focused psychotherapy (TFP); the participants in each condition received 3 years of treatment, and assessments were conducted at the end of each year. Participants in both groups showed significant improvements in quality of life, BPD severity, and general psychopathology. Survival analyses indicated that more patients in the SFT condition recovered and had more reliable improvements in BPD severity; moreover, the participants in SFT also had lower dropout rates. SFT showed moderate, positive effects on symptoms of impulsivity, fear of abandonment, NSSI, dissociation, and paranoid ideation. These trends were apparent after 1 year of treatment and extended throughout the full 3 years.

Two other RCTs of SFT have been conducted. Nadort and colleagues (2009) compared standard SFT to SFT with therapist telephone availability, in which clients were permitted to call their therapists between sessions for additional support. After 18 months of treatment, participants in both groups showed significant improvements on measures of borderline symptomology and a quality of life measure, and there were no differences between groups on any measures. The extent of reductions in BPD symptom severity and improvement in quality of life in this study were comparable to those of the Giesen-Bloo et al. (2006) RCT, although the disorders of the participants in this study were reportedly less severe (Nadort et al., 2009). In a second study, Farrell, Shaw, and Webber (2009) adapted SFT for use in a group format. In this study, 32 female participants were randomly assigned to either TAU (i.e., weekly individual eclectic/supportive therapy) or TAU plus group SFT. Participants received 14 months of treatment. Whereas all 16 participants who were assigned to TAU plus SFT completed the trial, only 12 of

the 16 participants in the TAU condition remained in the treatment. Patients who received TAU plus SFT had significant reductions (with large effect sizes) in general psychopathology, BPD severity, affective instability, and impulsivity, and had significant improvements in mental health status. Participants in the TAU plus SFT condition also showed high remission rates (94% vs. only 16% remission in the TAU group). This study provides additional evidence for the effectiveness of SFT in decreasing symptoms and improving functioning among patients with BPD. Taken together, these findings are encouraging, although it should be noted that support for standard SFT has not yet been replicated in a second RCT with an active (non-SFT) comparison condition or by researchers from an independent lab.

Although some clinical reports describe adaptations of SFT for adolescents (i.e., Geerdink, Jungman, & Scholing, 2012), no research to date has evaluated SFT in youth samples.

12.3.1.2 Theory of the disorder

Young (Young, 1994; Young et al., 2003) developed the concept of the "early maladaptive schema" (EMS), a particular type of schema that emerges from the combination of genetics, children's natural predispositions, and prolonged adverse family and social experiences. An EMS is believed to be a core representation of early childhood experiences that functions as "a template for processing and activating thoughts, feelings, and interpersonal behaviors" (Nysæter & Nordahl, 2008, p. 251). Young proposed that early experiences of abuse or trauma and unmet needs lead to the development of EMSs and the eventual emergence of BPD. He argued that an individual's healthy development is associated with five developmental tasks or needs: connectedness, autonomy, worthiness, reasonable expectations, and realistic limits. According to the theory, if these five developmental needs are not met, EMSs are likely to develop. Associated with these tasks, Young generated 18 common EMSs that can be classified under five domains: disconnection and rejection, impaired autonomy and performance, undesirability, restricted self-expression, and impaired limits.

As individuals with BPD often have difficulties associated with almost all EMSs, and because changes in their emotions are linked with rapid shifts in their schemas and associated coping strategies, Young proposed the concept of "schema modes." Inspired by transaction theory (Berne, 1961), Young suggested that individuals with BPD learn to respond to their environment based on their mood states, maladaptive schemas, and coping style (i.e., overcompensation, avoidance, and surrender). Young argued that helping an individual with BPD requires knowing how to respond to the particular mode the patient presents. He described five schema modes relevant to BPD: abandoned or helpless child mode, angry or impulsive child mode, the punitive parent mode, the detached protector mode, and the healthy adult mode. The ultimate goal of treatment is to reorganize these aspects of the self (Kellogg & Young, 2006).

Studies have shown some support for elements of the schema model; nevertheless, the findings are not particularly strong and some inconsistencies regarding the theoretical model remain. Research examining Young's concept of EMS shows that the link between childhood maltreatment and BPD is mediated by maladaptive schemas (e.g., mistrust, abandonment, defective self-control) and negative assumptions (Arntz, Dietzel, & Dreessen, 1999; Specht, Chapman, & Cellucci, 2009), thus providing support for the theory. The specificity of Young's schema domains to BPD has been

called into question; research to date does not show strong support for the schemas Young theorized to be especially predictive of BPD. For instance, research has shown that individuals with BPD tended to score higher on several schemas or schema domains compared to patients diagnosed with bipolar disorder (Nilsson, Jorgensen, Straarup & Licht, 2010), avoidant personality disorder, and obsessive–compulsive personality disorder (Jovev & Jackson, 2004), as well as healthy controls (Lawrence, Allen, & Chanen, 2011). Although patients with BPD do score significantly higher than controls on schemas associated with BPD, they also score higher than other groups on schemas or schema domains thought to be indicative of other personality disorders. Thus, patterns of scores on schemas and schema domains do not discriminate between patients with BPD and those with other personality disorders. While Specht and colleagues (2009) found that the schema domain of disconnection/rejection significantly predicted BPD severity, Carr and Francis (2010) found that none of Young's EMSs proved to be significant predictors of BPD symptoms. Inconsistencies in outcomes have also been found in research on schema modes; while some researchers have found that BPD patients scored significantly higher than controls on Young's hypothesized schema modes, they also scored higher on schema modes that, in theory, ought to be more closely related to other personality disorders.

Some aspects of the theory lack empirical evidence. For instance, no empirical data exist to support the concept behind "unmet developmental needs," nor is there evidence for how such unmet needs play a role in the development of BPD. Young's lack of explanation as to how he selected his five needs has also been met with criticism. The same could be said about the lack of research on the coping styles that were assumed to be central in Young's theory. Lastly, although studies have shown that patients do exhibit a pattern of switching from one schema mode to another, this behavior did not happen as immediately or quickly as previously hypothesized (Arntz, 2012; Arntz, Klokman, & Sieswerda, 2005; Lobbestael, Arntz, & Sieswerda, 2005; Sempertegui et al., 2013).

12.3.1.3 Theory of change

There are limited data on schema change resulting from SFT. Existing studies have used Young's Schema Questionnaire (YSQ) to assess change in maladaptive schemas and cognition. Of the four existing effectiveness studies that used the YSQ, one (Giesen-Bloo et al., 2006) did not specifically discuss schema change in the results or discussion sections of the manuscript; instead, YSQ scores were combined with other psychopathology or personality pathology questionnaires. Three other published reports have examined schema change in SFT. Results from the Nadort (2009) study showed support for the theory of change: Scores on the YSQ decreased over an 18-month period. The other two reports were from case series of one (Morrison, 2000) and six (Nordahl & Nysæter, 2005) patients; both showed positive trends of decreased scores on the YSQ over the course of treatment. Nevertheless, additional research is necessary.

12.3.1.4 Summary

Based on David and Montgomery's (2011) framework for evaluating psychosocial interventions, SFT for adults can be considered a Category IV psychotherapy. Preliminary but limited support has been shown for both the treatment and the theory that underpins the treatment.

12.3.2 Cognitive–Behavioral Therapy

Standard cognitive–behavioral therapy (CBT; Beck et al., 1979), which was originally developed to treat major depression, has been used to treat BPD. Adaptations have been made to the standard protocol to address the interpersonal difficulties and cognitive rigidity found in individuals with personality disorders (Beck, Freeman, & Davis, 2015). The focus of CBT for BPD is to help patients identify and modify distorted beliefs and schemas about the self, others, and the world. Similar to SFT, CBT includes a focus on modifying distorted schemas, but standard CBT relies more heavily on modifying distorted thoughts and beliefs than SFT and does not include schemas thought to be specific to the development of BPD (i.e., Young, 1994).

12.3.2.1 Evidence for therapy

Two published studies have examined the efficacy of CBT for BPD as the primary presenting problem (i.e., not for major depression with BPD as a comorbid disorder). Davidson and colleagues (2006) compared CBT plus TAU to TAU alone. Treatment lasted for 1 year, and patients attended an average of 27 individual therapy sessions. Participants in both conditions showed significant improvements on primary (i.e., suicidal acts, hospitalizations, and ER usage) and secondary (i.e., NSSI, depression, anxiety, and interpersonal functioning) outcome measures. There were no significant differences between conditions on the primary outcome measures; however, CBT was superior to TAU on measures of general distress and anxiety as well as on improving dysfunctional core beliefs. In a second study, Cottraux and colleagues (2009) compared CBT to supportive therapy over 1 year of treatment. There were no significant differences between the groups on any measure; some trend-level benefits favored CBT on measures of suicidality, anxiety, and impulsivity. It should be noted that this study included the same therapists in both conditions, which represents a significant confound in the design. No studies have been published examining the use of CBT for BPD in youth samples.

12.3.2.2 Theory of the disorder

The theory of the emergence and development of BPD according to the cognitive model is similar to what was described for SFT (see Section 12.3.1.2); that is, patients develop dysfunctional beliefs during childhood that influence their perceptions about themselves, others, and the environment (Beck et al., 2015). Once developed, these beliefs are thought to be highly inflexible and to inhibit one's ability to correctly process incoming information in adulthood (Arntz, 1994). In a series of studies of beliefs held by individuals with a variety of personality disorders, Arntz and colleagues (Arntz et al., 1999; Arntz, Dreessen, Schouten, & Weertman, 2004) found that individuals with BPD endorse a set of 20 assumptions about the self, others, and the world that appear to be stable across moods. Specifically, the authors also noted that four themes were reliably able to discriminate individuals with BPD from individuals with other personality disorders: loneliness, unlovability, rejection/abandonment, and a general view of the self as bad and deserving of punishment. In a similar study, Butler, Brown, Beck, and Grisham (2002) found that individuals with BPD could be discriminated from individuals with other personality disorders using the Personality Belief Questionnaire (Beck & Beck, 1991). Results from this study also suggested that individuals with BPD hold rigid beliefs that cluster around themes of helplessness, mistrust, rejection, and dependency.

12.3.2.3 Theory of change

According to the cognitive model, the primary mechanism of change in CBT is the modification of dysfunctional beliefs (Beck et al., 2015; Wenzel, Chapman, Newman, Beck, & Brown, 2006). Although this mechanism has been investigated in CBT for major depression, little research to date has been published on this topic in the treatment of BPD specifically. Brown, Newman, Charlesworth, Crits-Christoph, and Beck (2004) conducted an open trial of CBT for individuals with BPD and explicitly evaluated the extent to which dysfunctional beliefs changed over the course of treatment. Results from that study provided support for the theory; the majority of study participants showed significant improvement on the Personality Belief Questionnaire. More research is needed on this topic.

12.3.2.4 Summary

Based on David and Montgomery's (2011) framework for evaluating psychosocial interventions, CBT for adults can be considered a Category IV psychotherapy. Preliminary but limited support has been shown for both the treatment and the theory that underpins the treatment.

12.3.3 Transference-Focused Psychotherapy

TFP is a psychodynamic, manualized treatment based on object relations theory (Clarkin et al., 1999). Treatment is conducted via twice-weekly individual therapy sessions. Yeomans, Levy, and Caligor (2013) outline the therapeutic approach of TFP as follows: "(1) maintaining the frame of treatment; (2) containing and making use of the therapist's affective responses; and (3) engaging in the steps of the interpretive process" (p. 450). In order to maintain the frame of treatment, the therapist and client collaboratively develop a contract, which outlines treatment expectations and creates a safe place to reflect upon experiences and emotions. The process of containing and making use of the therapist's affective responses (i.e., countertransference) is believed to be particularly informative, as individuals with BPD often evoke strong emotions in others. In TFP, therapists are asked to be mindful of their reactions toward their clients and to maintain an accepting, reflective stance when processing these reactions (Yeomans et al., 2013). Finally, engaging in the steps of the interpretive process relies on the techniques of clarification, confrontation, and transference interpretation. Therapists use clarification to help the client identify feelings as well as their representations of self and others. Next, the therapist uses confrontation to point out any contradictions that may exist in the client's experience of an object relation dyad. Interpretation in TFP focuses attention upon the therapeutic relationship and the interactional patterns that occur within this context. The therapist aims to identify these patterns and link them to the client's other relationship patterns (Yeomans et al., 2013). The process of interpretation is thought to be the route to identity integration and, thus, improvement in psychological and social functioning. A primary goal of treatment is to increase the client's reflective functioning, which refers to one's ability to reflect and make inferences about behavior in the context of an attachment relationship (Fonagy et al., 1995).

12.3.3.1 Evidence for therapy

Equivocal results have emerged from three RCTs that have examined the efficacy of TFP in alleviating symptoms for individuals with BPD. As discussed in Section 12.3.1.1,

Giesen-Bloo et al. (2006) compared TFP and SFT over the course of 3 years. Participants in both conditions demonstrated similar initial improvements in psychopathology and patient functioning. After 3 years, however, patients who received SFT had more reliable improvements in the severity of their BPD symptoms as well as in their general functioning and quality of life. In addition, attrition rates were higher in TFP. Next, Clarkin et al. (2007) conducted a three-arm RCT to examine the differences in treatment outcomes for individuals with BPD after a year of TFP, DBT, or psychodynamic supportive therapy. The results of this study suggested stronger empirical support for TFP as compared to Giesen-Bloo et al.'s (2006) RCT: After a year of treatment, TFP was associated with significantly greater improvements across a variety of domains, including mood, anxiety, social functioning, suicidality, anger, and impulsivity. The improvements in these domains were found to be larger than the improvements associated with participation in DBT or psychodynamic supportive therapy.

In light of the somewhat mixed findings from the two RCTs described, Doering et al. (2010) conducted an RCT comparing TFP with treatment by community psychotherapists. The authors found that, after a year of treatment, TFP led to greater improvements in patient functioning across the following indices: number of BPD symptoms, psychosocial functioning, personality organization, suicide attempts, and the number and duration of inpatient hospitalizations. Surprisingly, NSSI did not change significantly for either treatment group. The authors noted that, compared with the other two available RCTs, participants in their study had less severe BPD symptomatology and less comorbidity of Axis I and Axis II disorders. They also noted that the frequency and severity of self-harm behaviors were low to begin with for their specific population.

A clinical trial is currently underway to evaluate TFP for adolescents (TFP-A). The goals of TFP-A are the same as those of TFP for adults and include working toward improvements in identity diffusion, unrealistic evaluations of self and others, and affective distortions (Normandin, Ensink, & Kernberg, 2015). Specific adaptations of TFP for use with adolescent populations include an emphasis on clear treatment parameters to decrease acting out or dangerous behaviors, support of normal development and identity formation, and neutrally addressing countertransference, which may be more likely to come up as therapists navigate interactions with both adolescents and their parents. No data are available yet regarding the efficacy of TFP-A.

12.3.3.2 Theory of the disorder

According to the TFP model, BPD is conceptualized as developing from a lack of identity integration, also referred to as "identity diffusion" (Clarkin et al., 2007). According to object relations theory, identity development begins with early interactions with caregivers, when individuals learn to integrate representations of themselves and others. Kernberg (1984) hypothesized that individuals with BPD have difficulty forming a cohesive identity of themselves and other people that includes both positive and negative elements. According to the theory, individuals are motivated to protect the positive views they have of themselves and others and thus aim to keep any negative views they have separate. As such, identity development is fractured and results in extreme and dichotomous thinking (e.g., people are either "good" or "bad"). As Clarkin, Yeomans, and Kernberg (2006) note, this failure to develop an integrated identity then contributes to the affective instability often seen in BPD.

There is a relative paucity of data in support of the TFP theory specifically; however, data do exist in support of portions of the theory. Clarkin and colleagues (2007) provided

a review of the data that indirectly support the object relations model. For example, the authors noted that individuals with BPD underperform on measures of effortful control, a construct that is inversely correlated with BPD symptomatology. They also noted that difficulties with reflective functioning are related to increased impulsivity. Levy (2000) found that individuals with less developed identity integration were more likely to use maladaptive strategies (e.g., self-harm, substance abuse, impulsive sex) to regulate negative emotions. Fonagy and colleagues (1996) found that 97% of patients with a history of abuse and low reflective functioning met criteria for BPD, whereas 17% of patients with a history of abuse but with high reflective functioning met diagnostic criteria for the disorder. This finding suggests the importance of this construct of reflective functioning (i.e., making inferences about self and others) in personality development.

12.3.3.3 Theory of change

TFP is theorized to help clients with BPD in the here and now by focusing on the transference and countertransference that occur between clients and therapists during treatment. Clients are taught to move away from their fractured sense of self and to move toward reflectiveness and integration, wherein they can think more flexibly about themselves and their environment (Levy, Clarkin et al., 2006). As they do so, their impulsivity and behavioral dysregulation decrease, and they begin to form more adaptive attachments, beginning with the therapist.

Levy, Meehan, and colleagues (2006) hypothesize that the mechanisms driving therapeutic change in TFP include a shift in attachment organization and an increase in reflective functioning. Improvements in identity integration are thought to occur over the course of treatment through the therapist's use of clarification, confrontation, and transference interpretations. Preliminary research suggests that increases in attachment security (as measured by the Adult Attachment Inventory) do occur after a course of TFP: In the three-arm study comparing TFP to DBT and supportive therapy, such increases were observed and were unique to TFP (Clarkin et al., 2007). The results of that study showed a threefold increase in the number of secure attachments after 1 year of TFP, whereas there were no clinically significant changes in the number of secure attachments for DBT or for psychodynamic supportive therapy. More research is needed to establish connections between these putative mechanisms of change and improvements in symptoms and functioning.

12.3.3.4 Summary

Based on David and Montgomery's (2011) framework for evaluating psychosocial interventions, TFP for adults and adolescents can be considered a Category IV psychotherapy. Preliminary but limited support has been shown for both the treatment and the theory that underpins the treatment. Some evidence suggests that other therapeutic approaches discussed in this chapter are more effective than TFP; however, no strong contradictory evidence has been published suggesting that TFP is iatrogenic.

12.4 Other Therapies

Several other therapies have been proposed as treatments for BPD that we elected not to include in this review for various reasons. First, none of them have garnered sufficient research evidence (for either the therapy itself or the theory of disorder or change) to be

strongly considered as a viable evidence-based treatment for BPD at this time. For example, interpersonal therapy (IPT; Klerman et al., 1984), which was developed for the treatment of major depression, has been adapted for use with patients with BPD (IPT-BPD; Markowitz, 2005; Markowitz, Skodol, & Bleiberg, 2006) and tested in one study (Bellino, Rinaldi, & Bogetto, 2010). In this trial, patients were randomized to receive fluoxetine plus clinical management or fluoxetine plus IPT-BPD. At 32 weeks, there were no differences between the groups on BPD remission rates, although some findings emerged favoring IPT-BPD on measures of anxiety, interpersonal relationships, impulsivity, and social functioning. No studies have examined potential mechanisms of change in IPT-BPD, although mechanisms have been proposed (Markowitz et al., 2006).

Second, some treatments have been proposed for BPD and a specific comorbid condition. For example, Dynamic Deconstructive Psychotherapy (DDP; Gregory, DeLucia-Deranja, & Mogle, 2010) was designed to treat co-occurring BPD and substance use disorders in weekly individual therapy sessions that occur over a 12–18-month period. Three studies have been conducted on DDP, all by the treatment developer. Preliminary data suggest that DDP is superior to TAU in reducing BPD symptoms and alcohol use; no studies have yet investigated hypothesized mechanisms of change. As another example, Bellino, Zizza, Rinaldi, and Bogetto (2007) compared CBT to IPT for patients who met criteria for both major depression and BPD. Participants in both conditions were also treated with fluoxetine to target depression. Both groups showed improvements on depression measures, but no explicit BPD-specific outcome measures were included in the study.

Finally, two group-based treatments have been developed for BPD and have shown some promise in early studies. These treatments have been offered either as stand-alone treatments or as adjuncts to ongoing individual therapy. Examples of group-based treatments are emotion regulation group training (Gratz & Gunderson, 2006) and Systems Training for Emotional Predictability and Problem Solving (Blum, Pfohl, St. John, Monahan, & Black, 2002).

12.5 Implications for Research

Given the challenges inherent in conducting treatment research, particularly with a chronically suicidal patient population, the progress and contributions made by existing treatment studies are significant and noteworthy. Yet, despite the increase in the number and variety of BPD treatment studies over the past decade, much has yet to be done to advance our knowledge about treatment efficacy and mechanisms of change (Stoffers et al., 2012). First, the amount of data supporting the effectiveness of psychotherapy for the treatment of BPD varies greatly by therapeutic approach. That is, whereas some psychotherapies have been well studied (e.g., DBT), other treatment approaches (e.g., DDP, IPT-BPD) have demonstrated only preliminary support. Thus, more studies are needed to evaluate less established approaches and to replicate outcomes from existing studies. Importantly, replication studies also need to be conducted by research groups not affiliated with the original treatment developer.

While it has been shown that active treatment is better than no treatment, and that using a particular therapeutic approach (e.g., DBT, SFT) yields better treatment outcomes than TAU, conducting more RCTs that compare several active treatments would

be useful to identify the strengths and weaknesses of different psychotherapies. In addition, data on the effectiveness of BPD treatment for chronically suicidal and self-harming adolescents are sparse; as such, there needs to be a push for more studies that assess the efficacy of BPD treatments in teens. Furthermore, because BPD is a chronic disorder, longitudinal research looking at the long-term outcomes of various treatment approaches is warranted.

When it comes to establishing empirical support for the theoretical foundation and mechanism of change in psychotherapy for BPD, more research is needed in general. As noted, a few studies have recently begun investigating theoretical components that were hypothesized to be crucial in driving change (e.g., evidence for biological predisposition for emotion regulation in DBT or presence of maladaptive schemas in SFT). Nevertheless, these studies are preliminary and inconsistent. Kazdin and colleagues (Kazdin, 2007; Kazdin & Nock, 2003) asserted that knowing how psychotherapy works is vital to maximizing treatment effects, which could, in turn, reduce treatment cost. Thus, additional studies that explicitly examine the mechanism of change in psychotherapy need to be equally prioritized.

Some scholars have also noted the importance of having researchers agree on a core battery of BPD outcomes for use in research (Stoffers et al., 2012). Having a standardized set of outcome measures would facilitate better comparisons between studies that utilize the same psychotherapy approach, as well as across treatment studies that examine different psychotherapies. Using the same set of measures across studies would also help to demonstrate whether a particular treatment is exclusively linked to its proposed mechanism of change (e.g., if DBT is specifically linked to increasing emotion regulation or if it could also indirectly lead to schema change, which is more central to SFT).

12.6 Implications for Practice

In terms of practice implications for patients with BPD, five key points are relevant. The first issue centers around issues of dissemination, implementation, and treatment effectiveness. The psychotherapies outlined in this chapter have their own complexities and generally require a fair amount of training in order to be delivered competently and adherently. As such, community-based mental health providers treating individuals with BPD must be prepared to make a substantial investment, in terms of both training in the treatments and the length and resource demands of treatment. When also considering the complexity of the patient population, the potential for service drift in these therapies is high without ongoing supervision and fidelity monitoring, both of which are relatively rare in community service settings. Thus, in order for these treatments to be administered as they were intended (and in a way that is consistent with the empirical evidence), therapists need to obtain appropriate training and, when possible, appropriate ongoing supervision. Although several studies have examined the cost-effectiveness of empirically supported treatments for BPD, especially with regard to decreased use of medical services and improved occupational functioning (e.g., Wagner et al., 2014), training and supervision costs are often not taken into account when evaluating the cost-effectiveness of psychotherapy (Van Asselt et al., 2008), so this is likely to be an ongoing practice-related issue.

In addition, these treatments are resource intensive; they often require multiple therapists (i.e., for individual and group therapies) and are delivered over long periods of time. It is often the case in community-based practice for treatments to be shortened or modified on the basis of limited resources (Linehan et al., 2015); as such, these adaptations may render the implementation method inconsistent with the evidence for the psychotherapy in question. Thus, therapists must be careful in making adaptations to the standard treatments and must be aware that certain adaptations may impact a critical element of the therapy. For example, until more dismantling studies are conducted, it will be impossible to know whether coaching calls are a critical part of DBT. By extension, if therapists elect to implement DBT without coaching calls, they may be eliminating an essential part of the treatment. At a minimum, therapists who are making adaptations to the treatment should be clear on the targets of treatment (e.g., changes in cognitive distortions, reductions in suicidality, improvements in reflective functioning), and progress toward these targets should be monitored.

Second, the choice of which of the aforementioned evidence-based treatments to implement with a patient with BPD may vary based on the severity of the high-risk behaviors present. The five therapies with the strongest empirical support vary in their degrees of targeting and managing high-risk symptoms of BPD. Specifically, DBT, MBT, and SFT focus on the function of self-harm and suicidal behavior, including serving to communicate unverbalized internal states or to regulate high-intensity negative emotion. Other treatments presented here, such as CBT and TFP, do not place as much emphasis on the explicit management of high suicidality and self-harm. Thus, providers may need to choose their intervention—or their need to refer a patient to a provider with the appropriate expertise—based on symptom severity.

Third, providers need to account for comorbid conditions when developing a patient's treatment plan. As the evidence reviewed in this chapter demonstrates, even after positive treatment trials, many individuals with BPD continue to experience global functioning impairment and psychiatric comorbidities. Across treatments, theories of how BPD develops consistently note the impact of trauma, invalidation, and early adverse experiences in the environment on the development of symptoms. It may be the case that, after treatment for interpersonal, behavioral, and emotional dysregulation, individuals with BPD need further treatment to specifically target trauma and/or symptoms of posttraumatic stress disorder or other comorbid conditions.

Fourth, given that interpersonal dysregulation is a common characteristic for individuals with BPD, attachment and commitment to the therapist need to be prioritized early in treatment. Four of the five treatments discussed in this chapter (DBT, MBT, SFT, and TFP) emphasize the importance of attachment relationships in the development of BPD as a disorder. In these treatments, the therapeutic alliance is used as the basis for forming adaptive interpersonal attachments and for providing appropriate validation and modeling of healthy relationships.

Finally, a key ethical and practice implication has to do with the use of particular treatment approaches for adolescents. Currently, only DBT and MBT have been adapted for and studied in youth samples. These are the only two treatments for adolescent BPD that have any support for both the therapeutic package and the theory, and research evidence is scant even in these two treatments. Thus, more research is needed, and the treatments should be applied in youth samples with the appropriate caveats to patients regarding treatment efficacy.

12.7 Conclusions

On the positive side, the available evidence suggests that several treatments are available for BPD and that these treatments are at least partially effective in reducing problematic behaviors and reducing distressing symptoms of BPD. On the negative side, although the literature on the general and comparative effectiveness of these therapies is growing, there is a paucity of information on the moderators and mechanisms by which these treatments effect change in patients. Moreover, there is little empirical support for the treatment of BPD in youth. Fortunately, no studies have been published that have demonstrated negative findings for any coherent psychotherapy designed to treat BPD (Bateman, 2012; Stoffers et al., 2012), although it is possible that some treatments have been studied and found to be iatrogenic and that such treatment results have not been published (i.e., the "file drawer" problem). As is the case with other psychiatric disorders, no treatment has been shown to be effective for all patients, and attrition remains a significant problem in this population. Thus, it may not make sense to identify the "best" treatment for BPD. Perhaps the better question that future research should address is which treatment approach is best for a particular individual, for a specific setting, and for a given condition or situation. Of course, this personalized approach requires a better understanding of the mechanisms that underpin these treatments. In sum, great strides have been made in the treatment of BPD over the past few decades, and there is still a long way to go.

References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.

Ansell, E. B., Sanislow, C. A., McGlashan, T. H., & Grilo, C. M. (2007). Psychosocial impairment and treatment utilization by patients with borderline personality disorder, other personality disorders, mood and anxiety disorders, and a healthy comparison group. *Comprehensive Psychiatry*, *48*(4), 329–336. doi:10.1016/j.comppsych.2007.02.001

Arntz, A. (1994). Treatment of borderline personality disorders: A challenge for cognitive behavioural therapy. *Behaviour Research and Therapy*, *32*, 419–430. doi:10.1016/0005-7967(94)90005-1

Arntz, A. (2012). Imagery rescripting as a therapeutic technique: Review of clinical trials, basic studies, and research agenda. *Journal of Experimental Psychopathology*, *3*(2), 189–208. doi:10.5127/jep.024211

Arntz, A., Dietzel, R., & Dreessen, L. (1999). Assumptions in borderline personality disorder: Specific stability and relationship with etiological factors. *Behaviour Research and Therapy*, *37*, 545–557. doi:10.1016/S0005-7967(98)00152-1

Arntz, A., Dreessen, L., Schouten, E., & Weetman, A. (2004). Beliefs in personality disorders: A test with the Personality Disorder Belief Questionnaire. *Behaviour Research and Therapy*, *42*, 1215–1225. doi:10.1016/j.brat.2003.08.004

Arntz, A., Klokman, J., & Sieswerda, S. (2005). An experimental test of the schema mode model of borderline personality disorder. *Journal of Behavior Therapy and Experimental Psychiatry*, *36*(3), 226–239. doi:10.1016/j.jbtep.2005.05.005

Bandelow, B., Krause, J., Wedekind, D., Broocks, A., Hajak, G., & Rüther, E. (2005). Early traumatic life events, parental attitudes, family history, and birth risk factors in patients with borderline personality disorder and healthy controls. *Psychiatry Research*, *134*(2), 169–179. doi:10.1016/j.psychres.2003.07.008

Bateman, A., & Fonagy, P. (1999). Effectiveness of partial hospitalization in the treatment of borderline personality disorder: A randomized controlled trial. *American Journal of Psychiatry*, *156*, 1563–1569. doi:10.1176/ajp.156.10.1563

Bateman, A., & Fonagy, P. (2004). *Psychotherapy for borderline personality disorder: Mentalisation based treatment*. Oxford, UK: Oxford University Press.

Bateman, A., & Fonagy, P. (2006). *Mentalization-based treatment for borderline personality disorder: A practical guide*. Oxford, UK: Oxford University Press.

Bateman, A., & Fonagy, P. (2008). 8-year follow-up of patients treated for borderline personality disorder: Mentalization-based treatment versus treatment as usual. *American Journal of Psychiatry*, *165*(5), 631–638. doi:10.1176/appi.ajp.2007.07040636

Bateman, A., & Fonagy, P. (2009). Randomized controlled trial of outpatient mentalization-based treatment versus structured clinical management for borderline personality disorder. *American Journal of Psychiatry*, *166*, 1355–1364. doi:10.1176/appi.ajp.2009.09040539

Bateman, A., & Fonagy, P. (2010). Mentalization based treatment for borderline personality disorder. *World Psychiatry*, *9*(1), 11–15. doi:10.1002/j.2051-5545.2010.tb00255.x

Bateman, A. W. (2012). Treating borderline personality disorder in clinical practice. *American Journal of Psychiatry*, *169*(6), 560–563. doi:10.1176/appi.ajp.2012.12030341

Beck, A. T., & Beck, J. S. (1991). *The Personality Belief Questionnaire*. Unpublished. Bala Cynwyd, PA: Beck Institute for Cognitive Therapy and Research.

Beck, A. T., Freeman, A., & Davis, D. D. (2015). *Cognitive therapy of personality disorders* (3rd ed.). New York, NY: Guilford Press.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York, NY: Guilford Press.

Bellino, S., Rinaldi, C., & Bogetto, F. (2010). Adaptation of interpersonal psychotherapy to borderline personality disorder: A comparison of combined therapy and single pharmacotherapy. *Canadian Journal of Psychiatry/Revue Canadienne de Psychiatrie*, *55*, 74–81. doi:10.1177/070674371005500203

Bellino, S., Zizza, M., Rinaldi, C., & Bogetto, F. (2007). Combined therapy of major depression with concomitant borderline personality disorder: Comparison of interpersonal and cognitive psychotherapy. *Canadian Journal of Psychiatry/Revue Canadienne de Psychiatrie*, *52*(11), 718–725. doi:10.1177/070674370705201106

Berne, E. (1961). *Transactional analysis in psychotherapy*. New York, NY: Grove Press.

Bland, A. R., Williams, C. A., Scharer, K., & Manning, S. (2004). Emotion processing in borderline personality disorders. *Issues in Mental Health Nursing*, *25*, 655–672. doi:10.1080/01612840490486692

Blum, N., Pfohl, B., St. John, D., Monahan, P., & Black, D. (2002). STEPPS: A cognitive-behavioral systems-based group treatment for outpatients with borderline personality disorder: A preliminary report. *Comprehensive Psychiatry*, *43*, 301–310. doi:10.1053/comp.2002.33497

Brown, G. K., Newman, C. F., Charlesworth, S. E., Crits-Christoph, P., & Beck, A. T. (2004). An open clinical trial of cognitive therapy for borderline personality disorder. *Journal of Personality Disorders*, *18*(3), 257–271. doi:10.1521/pedi.18.3.257.35450

Bohus, M., Haaf, B., Simms, T., Limberger, M. F., Schmahl, C., Unckel, C., … Linehan, M. M. (2004). Effectiveness of inpatient dialectical behavioral therapy for borderline personality disorder: A controlled trial. *Behaviour Research and Therapy*, *42*, 487–499. doi:10.1016/S0005-7967(03)00174-8

Butler, A. C., Brown, G. K., Beck, A. T., & Grisham, J. R. (2002). Assessment of dysfunctional beliefs in borderline personality disorder. *Behaviour Research and Therapy*, *40*(10), 1231–1240. doi:10.1016/S0005-7967(02)00031-1

Carr, S. N., & Francis, A. J. (2010). Do early maladaptive schemas mediate the relationship between childhood experiences and avoidant personality disorder features? A preliminary investigation in a non-clinical sample. *Cognitive Therapy and Research*, *34*(4), 343–358. doi:10.1007/s10608-009-9250-1

Clarkin, J. F., Levy, K. N., Lenzenweger, M. F., & Kernberg, O. F. (2007). Evaluating three treatments for borderline personality disorder: A multiwave study. *American Journal of Psychiatry*, *164*(6), 922–928. doi:10.1176/appi.ajp.164.6.922

Clarkin, J. F., Yeomans, F. E., & Kernberg, O. F. (1999). *Psychotherapy for borderline personality*. New York, NY: John Wiley & Sons.

Clarkin, J. F., Yeomans, F. E., & Kernberg, O. F. (2006). *Psychotherapy for borderline personality: Focusing on object relations*. Washington, DC: American Psychiatric Association.

Cohen, P., Crawford, T. N., Johnson, J. G., & Kasen, S. (2005). The Children in the Community Study of developmental course of personality disorder. *Journal of Personality Disorders*, *19*(5), 466–486. doi:10.1521/pedi.2005.19.5.466

Comtois, K. A., Elwood, L., Holdcraft, L. C., & Simpson, T. L. (2007). Effectiveness of dialectical behavior therapy in a community mental health center. *Cognitive and Behavioral Practice*, *14*(4), 406–414. doi:10.1016/j.cbpra.2006.04.023

Comtois, K. A., Kerbrat, A. H., Atkins, D. C., Harned, M. S., & Elwood, L. (2010). Recovery from disability for individuals with borderline personality disorder: A feasability trial of DBT-ACES. *Psychiatric Services*, *61*(11), 1106–1111. doi:10.1176/appi.ps.61.11.1106

Cottraux, J., Note, I. D., Boutitie, F., Milliery, M., Genouihlac, V., Yao, S. N., … Gueyffier, F. (2009). Cognitive therapy versus Rogerian supportive therapy in borderline personality disorder. *Psychotherapy and Psychosomatics*, *78*, 307–316. doi:10.1159/000229769

Courtney-Seidler, E. A., Klein, D., & Miller, A. L. (2013). Borderline personality disorder in adolescents. *Clinical Psychology: Science and Practice*, *20*, 425–444. doi:10.1111/cpsp.12051

Crowell, S. E., Beauchaine, T. P., & Linehan, M. M. (2009). A biosocial developmental model of borderline personality: Elaborating and extending Linehan's theory. *Psychological Bulletin*, *135*(3), 495–510. doi:10.1037/a0015616

David, D., & Montgomery, G. H. (2011). The scientific status of psychotherapies: A new evaluative framework for evidence-based psychosocial interventions. *Clinical Psychology: Science and Practice*, *18*(2), 89–99. doi:10.1111/j.1468-2850.2011.01239.x

Davidson, K., Norrie, J., Tyrer, P., Gumley, A., Tata, P., Murray, H., & Palmer, S. (2006). The effectiveness of cognitive behavior therapy for borderline personality disorder: Results from the Borderline Personality Disorder Study of Cognitive Therapy (BOSCOT) trial. *Journal of Personality Disorders*, *20*(5), 450–465. doi:10.1521/pedi.2006.20.5.450

Doering, S., Horz, S., Rentrop, M., Fischer-Kern, M., Schuster, P., Benecke, C., … Buchheim, P. (2010). Transference-focused psychotherapy v. treatment by community

psychotherapists for borderline personality disorder: Randomised controlled trial. *British Journal of Psychiatry*, *196*(5), 389–395. doi:10.1192/bjp.bp.109.070177

Ebner-Priemer, U. W., Welch, S. S., Grossman, P., Reisch, T., Linehan, M. M., & Bohus, M. (2007). Psychophysiological ambulatory assessment of affective dysregulation in borderline personality disorder. *Psychiatry Research*, *150*, 265–275. doi:10.1016/j.psychres.2006.04.014

Evershed, S., Tennant, A., Boomer, D., Rees, A., Barkham, M., & Watson, A. (2003). Practice-based outcomes of dialectical behaviour therapy targeting anger and violence, with male forensic patients: A pragmatic and non-contemporaneous comparison. *Criminal Behaviour and Mental Health*, *13*, 198–213. doi:10.1002/cbm.542

Farrell, J. M., Shaw, I. A., & Webber, M. A. (2009). A schema-focused approach to group psychotherapy for outpatients with borderline personality disorder: A randomized controlled trial. *Journal of Behavior Therapy and Experimental Psychiatry*, *40*(2), 317–328. doi:10.1016/j.jbtep.2009.01.002

Fleischhaker, C., Böhme, R., Sixt, B., Brück, C., Schneider, C., & Schulz, E. (2011). Dialectical behavioral therapy for adolescents: A clinical trial for patients with suicidal and self-injurious behavior and borderline symptoms with a one-year follow-up. *Child and Adolescent Psychiatry and Mental Health*, *5*(3), 3. doi:10.1186/1753-2000-5-3

Fonagy, P., & Bateman, A. W. (2006). Mechanisms of change in mentalization-based treatment of BPD. *Journal of Clinical Psychology*, *62*(4), 411–430. doi:10.1002/jclp.20241

Fonagy, P., Leigh, T., Steele, M., Steele, H., Kennedy, R., Mattoon, G., … Gerber, A. (1996). The relation of attachment status, psychiatric classification, and response to psychotherapy. *Journal of Consulting and Clinical Psychology*, *64*(1), 22–31. doi:10.1037/0022-006X.64.1.22

Fonagy, P., Steele, M., Steele, H., Leigh, T., Kennedy, R., Mattoon, G., & Target, M. (1995). Attachment, the reflective self, and borderline states: The predictive specificity of the Adult Attachment Interview and pathological emotional development. In S. Goldberg, J. Kerr, & R. Muir (Eds.), *Attachment theory: Social, developmental, and clinical perspectives* (pp. 233–278). Hillsdale, NJ: Analytic Press.

Geerdink, M., Jongman, E., & Scholing, A. (2012). Schema therapy in adolescents. In M. van Vreeswijk, J. Broersen, & M. Nadort (Eds.), *The Wiley Blackwell handbook of schema therapy: Theory, research and practice* (pp. 391–396). Oxford, UK: Wiley-Blackwell.

Giesen-Bloo, J., Van Dyck, R., Spinhoven, P., Van Tilburg, W., Dirksen, C., Van Asselt, T., … Arntz, A. (2006). Outpatient psychotherapy for borderline personality disorder: Randomized trial of schema-focused therapy vs. transference-focused psychotherapy. *Archives of General Psychiatry*, *63*, 649–658. doi:10.1001/archpsyc.63.6.649

Golier, J. A., Yehuda, R., Bierer, L. M., Mitropoulou, V., New, A. S., Schmeidler, J., … Siever, L. J. (2003). The relationship of borderline personality disorder to posttraumatic stress disorder and traumatic events. *American Journal of Psychiatry*, *160*(11), 2018–2024. doi:10.1176/appi.ajp.160.11.2018

Goodman, M., Carpenter, D., Tang, C. Y., Goldstein, K. E., Avedon, J., Fernandez, N., … Hazlett, E. A. (2014). Dialectical behavior therapy alters emotion regulation and amygdala activity in patients with borderline personality disorder. *Journal of Psychiatric Research*, *57*, 108–116. doi:10.1016/j.jpsychires.2014.06.020

Grant, B. F., Chou, S. P., Goldstein, R. B., Huang, B., Stinson, F. S., Saha, T. D., … Ruan, W. J. (2008). Prevalence, correlates, disability, and comorbidity of DSM-IV borderline personality disorder: Results from the Wave 2 National Epidemiologic Survey on Alcohol

and Related Conditions. *Journal of Clinical Psychiatry*, *69*(4), 533–545. doi:10.4088/JCP.v69n0404

Gratz, K. L., & Gunderson, J. G. (2006). Preliminary data on an acceptance-based emotion regulation group intervention for deliberate self-harm among women with borderline personality disorder. *Behavior Therapy*, *37*(1), 25–35. doi:10.1016/j.beth.2005.03.002

Gregory, R. J., Chlebowski, S., Kang, D., Remen, A. L., Soderberg, M. G., Stepkovitch, J., & Virk, S. (2008). A controlled trial of psychodynamic psychotherapy for co-occurring borderline personality disorder and alcohol use disorder. *Psychotherapy: Theory, Research & Practice*, *45*(1), 28–41. doi:10.1037/0033-3204.45.1.28

Gregory, R. J., DeLucia-Deranja, E., & Mogle, J. A. (2010). Dynamic deconstructive psychotherapy versus optimized community care for borderline personality disorder co-occurring with alcohol use disorders: A 30-month follow-up. *Journal of Nervous and Mental Disease*, *198*(4), 292–298. doi:10.1097/NMD.0b013e3181d6172d

Harned, M. S., Banawan, S. F., & Lynch, T. R. (2006). Dialectical behavior therapy: An emotion-focused treatment for borderline personality disorder. *Journal of Contemporary Psychotherapy*, *36*, 67–75. doi:10.1007/s10879-006-9009-x

Harned, M. S., Rizvi, S. L., & Linehan, M. M. (2010). Impact of co-occurring posttraumatic stress disorder on suicidal women with borderline personality disorder. *American Journal of Psychiatry*, *167*, 1210–1217. doi:10.1176/appi.ajp.2010.09081213

Jakobsen, J. C., Gluud, C., Kongerslev, M., Larsen, K. A., Sørensen, P., Winkel, P., … Simonsen, E. (2012). "Third wave" cognitive therapy versus mentalization-based therapy for major depressive disorder: A protocol for a randomised clinical trial. *BMC Psychiatry*, *12*(1), 232–241. doi:10.1186/1471-244X-12-232

Johnson, J. G., Cohen, P., Chen, H., Kasen, S., & Brook, J. S. (2006). Parenting behaviors associated with risk for offspring personality disorder during adulthood. *Archives of General Psychiatry*, *63*(5), 579–587. doi:10.1001/archpsyc.63.5.579

Jørgensen, C. R., Freund, C., Bøye, R., Jordet, H., Andersen, D., & Kjølbye, M. (2013). Outcome of mentalization-based and supportive psychotherapy in patients with borderline personality disorder: A randomized trial. *Acta Psychiatrica Scandinavica*, *127*(4), 305–317. doi:10.1111/j.1600-0447.2012.01923.x

Jovev, M., & Jackson, H. J. (2004). Early maladaptive schemas in personality disordered individuals. *Journal of Personality Disorders*, *18*(5), 467–478. doi:10.1521/pedi.18.5.467.51325

Kazdin, A. E. (2007). Mediators and mechanism of change in psychotherapy research. *Annual Review of Clinical Psychology*, *3*, 1–27. doi:10.1146/annurev.clinpsy.3.022806.091432

Kazdin, A. E., & Nock, M. K. (2003). Delineating mechanisms of change in child and adolescent therapy: Methodological issues and research recommendations. *Journal of Child Psychology and Psychiatry*, *44*, 1116–1129. doi:10.1111/1469-7610.00195

Kellogg, S. H., & Young, J. E. (2006). Schema therapy for borderline personality disorder. *Journal of Clinical Psychology*, *62*(4), 445–458. doi:10.1002/jclp.20240

Kernberg, O. (1984). *Severe personality disorders: Psychotherapeutic strategies*. New Haven, CT: Yale University Press.

Klerman, G. L., Weissman, M. M., Rounsaville, B., & Chevron, E. S. (1984). *Interpersonal psychotherapy for depression*. Lanham, MD: Rowman & Littlefield.

Koons, C. R., Robins, C. J., Tweed, J. L., Lynch, T. R., Gonzalez, A. M., Morse, J. Q., … Bastian, L. A. (2001). Efficacy of dialectical behavior therapy in women veterans with

borderline personality disorder. *Behavior Therapy*, *32*(2), 371–390. doi:10.1016/S0005-7894(01)80009-5

Kuo, J. R., & Linehan, M. M. (2009). Disentangling emotion processes in borderline personality disorder: Physiological and self-reported assessment of biological vulnerability, baseline intensity, and reactivity to emotionally evocative stimuli. *Journal of Abnormal Psychology*, *118*(3), 531–544. doi:10.1037/a0016392

Lawrence, K. A., Allen, J. S., & Chanen, A. M. (2011). A study of maladaptive schemas and borderline personality disorder in young people. *Cognitive Therapy and Research*, *35*(1), 30–39. doi:10.1007/s10608-009-9292-4

Lenzenweger, M. F., Lane, M. C., Loranger, A. W., & Kessler, R. C. (2007). DSM-IV personality disorders in the National Comorbidity Survey Replication. *Biological Psychiatry*, *62*(6), 553–564. doi:10.1016/j.biopsych.2006.09.019

Levy, K. N. (2000). *Attachment style, representations of self and others, and affect regulation: Implications for the experience of depression* (Doctoral thesis). Retrieved from Dissertation Abstracts International: Section B: The Sciences & Engineering (4895).

Levy, K. N. (2005). The implications of attachment theory and research for understanding borderline personality disorder. *Developmental Psychopathology*, *17*(4), 959–986. doi:10.1017/S0954579405050455

Levy, K. N., Clarkin, J. F., Yeomans, F. E., Scott, L. N., Wasserman, R. H., & Kernberg, O. F. (2006). The mechanisms of change in the treatment of borderline personality disorder with transference-focused psychotherapy. *Journal of Clinical Psychology*, *62*, 481–502. doi:10.1002/jclp.20239

Levy, K. N., Meehan, K. B., Kelly, K. M., Reynoso, J. S., Clarkin, J. F., & Kernberg, O. F. (2006). Change in attachment patterns and reflective function in a randomized control trial of transference-focused psychotherapy for borderline personality disorder. *Journal of Consulting and Clinical Psychology*, *74*, 1027–1040. doi:10.1037/0022-006X.74.6.1027

Lieb, K., Rexhausen, J. E., Kahl, K. G., Schweiger, U., Philipsen, A., Hellhammer, D. H., & Bohus, M. (2004). Increased diurnal salivary cortisol in women with borderline personality disorder. *Journal of Psychiatric Research*, *38*(6), 559–565. doi:10.1016/j.jpsychires.2004.04.002

Linehan, M. M. (1993). *Cognitive-behavioral treatment of borderline personality disorder*. New York, NY: Guilford Press.

Linehan, M. M., Armstrong, H. E., Suarez, A., Allmon, D., & Heard, H. L. (1991). Cognitive-behavioral treatment of chronically parasuicidal borderline patients. *Archives of General Psychiatry*, *48*(12), 1060–1064. doi:10.1001/archpsyc.1991.01810360024003

Linehan, M. M., Comtois, K. A., Murray, A. M., Brown, M. Z., Gallop, R. J., Heard, H. L., … Lindenboim, N. (2006). Two-year randomized controlled trial and follow-up of dialectical behavior therapy vs. therapy by experts for suicidal behaviors and borderline personality disorder. *Archives of General Psychiatry*, *63*, 757–766. doi:10.1001/archpsyc.63.7.757

Linehan, M. M., Dimeff, L. A., Reynolds, S. K., Comtois, K. A., Welch, S. S., Heagerty, P., & Kivlahan, D. R. (2002). Dialectical behavior therapy versus comprehensive validation therapy plus 12-step for the treatment of opioid dependent women meeting criteria for borderline personality disorder. *Drug and Alcohol Dependence*, *67*(1), 13–26. doi:10.1016/S0376-8716(02)00011-X

Linehan, M. M., Korslund, K. E., Harned, M. S., Gallop, R. J., Lungu, A., Neacsiu, A. D., … Murray-Gregory, A. M. (2015). Dialectical behavior therapy for high suicide risk in individuals with borderline personality disorder: A randomized clinical trial and

component analysis. *JAMA Psychiatry*, *72*(5), 475–482. doi:10.1001/jamapsychiatry.2014.3039

Linehan, M. M., Schmidt, H., III, Dimeff, L. A., Craft, J. C., Kanter, J., & Comtois, K. A. (1999). Dialectical behavior therapy for patients with borderline personality disorder and drug-dependence. *American Journal on Addictions*, *8*, 279–292. doi:10.1080/105504999305686

Linehan, M. M., Tutek, D. A., Heard, H. L., & Armstrong, H. E. (1994). Interpersonal outcome of cognitive behavioral threatment for chronically suicidal borderline patients. *American Journal of Psychiatry*, *151*(12), 1771–1776. doi:10.1176/ajp.151.12.1771

Lobbestael, J., Arntz, A., & Sieswerda, S. (2005). Schema modes and childhood abuse in borderline and antisocial patients. *Journal of Behavior Therapy and Experimental Psychiatry*, *36*(3), 240–253. doi:10.1016/j.jbtep.2005.05.006

Lyons-Ruth, K., Choi-Kain, L., Pechtel, P., Bertha, E., & Gunderson, J. (2011). Perceived parental protection and cortisol responses among young females with borderline personality disorder and controls. *Psychiatry Research*, *189*, 426–432. doi:10.1016/j.psychres.2011.07.038

Markowitz, J. C. (2005). Interpersonal therapy of personality disorders. In J. M. Oldham, A. E. Skodol, & B. S. Bender (Eds.), *Textbook of personality disorders* (pp. 321–334). Washington, DC: American Psychiatric Press.

Markowitz, J. C., Skodol, A. E., & Bleiberg, K. (2006). Interpersonal psychotherapy for borderline personality disorder: Possible mechanisms of change. *Journal of Clinical Psychology*, *62*(4), 431–444. doi:10.1002/jclp.20242

McGinn, L. K., & Young, J. E. (1996). Schema-focused therapy. In P. H. Salkovskis (Ed.), *Frontiers of cognitive therapy* (pp. 182–207). New York, NY: Guilford Press.

McMain, S. F., Links, P. A., Gnam, W. H., Guimond, T., Cardish, R. J., Korman, L., & Streiner, D. L. (2009). A randomized trial of dialectial behavior therapy versus general psychiatric management for borderline personality disorder. *American Journal of Psychiatry*, *166*(12), 1365–1374. doi:10.1176/appi.ajp.2009.09010039

Mehlum, L., Tørmoen, A. J., Ramberg, M., Haga, E., Diep, L. M., Laberg, S., … Grøholt, B. (2014). Dialectical behavior therapy for adolescents with repeated suicidal and self-harming behavior: A randomized trial. *Journal of the American Academy of Child & Adolescent Psychiatry*, *53*(10), 1082–1091. doi:10.1016/j.jaac.2014.07.003

Morrison, N. (2000). Schema-focused cognitive therapy for complex long-standing problems: A single case study. *Behavioural and Cognitive Psychotherapy*, *28*(3), 269–283. doi:10.1017/S1352465800003076

Muller, N., & Midgley, N. (2015). Approaches to assessment in time-limited mentalization-based therapy for children (MBT-C). *Frontiers in Psychology*, *6*, 1–10. doi:10.3389/fpsyg.2015.01063

Nadort, M., Arntz, A., Smit, J. H., Giesen-Bloo, J., Eikelenboom, M., Spinhoven, P., … & Van Dyck, R. (2009). Implementation of outpatient schema therapy for borderline personality disorder with versus without crisis support of the therapist outside office hours: A randomized trial. *Behaviour Research and Therapy*, *47*, 961–973. doi:10.1016/j.brat.2009.07.013

Neacsiu, A. D., Eberle, J. W., Kramer, R., Wiesmann, T., & Linehan, M. M. (2014). Dialectical behavior therapy skills for transdiagnostic emotion dysregulation: A pilot randomized controlled trial. *Behaviour Research and Therapy*, *59*, 40–51. doi:10.1016/j.brat.2014.05.005

Neacsiu, A. D., Rizvi, S. L., & Linehan, M. M. (2010). Dialectical behavior therapy skills use as a mediator and outcome of treatment for borderline personality disorder. *Behaviour Research and Therapy*, *48*, 832–839. doi:10.1016/j.brat.2010.05.017

Nilsson, A. K. K., Jørgensen, C. R., Straarup, K. N., & Licht, R. W. (2010). Severity of affective temperament and maladaptive self-schemas differentiate borderline patients, bipolar patients, and controls. *Comprehensive Psychiatry*, *51*(5), 486–491. doi:10.1016/j.comppsych.2010.02.006

Nordahl, H. M., & Nysæter, T. O. (2005). Schema therapy for patients with borderline personality disorder: A single case series. *Journal of Behavior Therapy and Experimental Psychiatry*, *36*(3), 254–264. doi:10.1016/j.jbtep.2005.05.007

Normandin, L., Ensink, E. K., & Kernberg, O. F. (2015). Transference-focused psychotherapy for borderline adolescents: A neurobiologically informed psychodynamic psychotherapy. *Journal of Infant, Child, and Adolescent Psychotherapy*, *14*, 98–110. doi:10.1080/15289168.2015.1006008

Nysæter, T. E., & Nordahl, H. M. (2008). Principles and clinical application of schema therapy for patients with borderline personality disorder. *Nordic Psychology*, *60*(3), 249–263. doi:10.1027/1901-2276.60.3.249

Paris, J., Brown, R., & Nowlis, D. (1987). Long-term follow-up of borderline patients in a general hospital. *Comprehensive Psychiatry*, *28*, 530–535. doi:10.1016/0010-440X(87)90019-8

Paris, J., & Zweig-Frank, H. (2001). A 27-year follow-up of patients with borderline personality disorder. *Comprehensive Psychiatry*, *42*(6), 482–487. doi:10.1053/comp.2001.26271

Perepletchikova, F., Axelrod, S. R., Kaufman, J., Rounsaville, B. J., Douglas-Palumberi, H., & Miller, A. L. (2011). Adapting dialectical behaviour therapy for children: Towards a new research agenda for paediatric suicidal and non-suicidal self-injurious behaviours. *Child and Adolescent Mental Health*, *16*(2), 116–121. doi:10.1111/j.1475-3588.2010.00583.x

Pistorello, J., Fruzzetti, A. E., MacLane, C., Gallop, R. G., & Iverson, K. M. (2012). Dialectical behavior therapy applied to college students: A randomized clinical trial. *Journal of Consulting and Clinical Psychology*, *80*(6), 982–994. doi:10.1037/a0029096

Rathus, J. H., & Miller, A. L. (2002). Dialectical behavior therapy adapted for suicidal adolescents. *Suicide and Life-Threatening Behavior*, *32*(2), 146–157. doi:10.1521/suli.32.2.146.24399

Rathus, J. H., & Miller, A. L. (2014). *DBT skills manual for adolescents*. New York, NY: Guilford Press.

Rizvi, S. L., Steffel, L. M., & Carson-Wong, A. (2013). An overview of dialectical behavior therapy for professional psychologists. *Professional Psychology: Research and Practice*, *44*(2), 73–80. doi:10.1037/a0029808

Robinson, P., Barrett, B., Bateman, A., Hakeem, A., Hellier, J., Lemonsky, F., … Fonagy, P. (2014). Study protocol for a randomized controlled trial of mentalization based therapy against specialist supportive clinical management in patients with both eating disorders and symptoms of borderline personality disorder. *BMC Psychiatry*, *14*(1), 51. doi:10.1186/1471-244X-14-51

Roepke, S., Vater, A., Preißler, S., Heekeren, H. R., & Dziobek, I. (2013). Social cognition in borderline personality disorder. *Frontiers in Neuroscience*, *6*, 195. doi:10.3389/fnins.2012.00195

Rossouw, T. I., & Fonagy, P. (2012). Mentalization-based treatment for self-harm in adolescents: A randomized controlled trial. *Journal of the American Academy of Child & Adolescent Psychiatry*, *51*(12), 1304–1313. doi:10.1016/j.jaac.2012.09.018

Salsman, N. L., & Linehan, M. M. (2012). An investigation of the relationships among negative affect, difficulties in emotion regualtion, and features of borderline personality disorder. *Journal of Psychopathology and Behavioral Assessment*, *34*, 260–267. doi:10.1007/s10862-012-9275-8

Schmahl, C., & Bremner, J. D. (2006). Neuroimaging in borderline personality disorder. *Journal of Psychiatric Research*, *40*(5), 419–427. doi:10.1016/j.jpsychires.2005.08.011

Schnell, K., & Herpertz, S. C. (2007). Effects of dialectic-behavioral-therapy on the neural correlates of affective hyperarousal in borderline personality disorder. *Journal of Psychiatric Research*, *41*, 837–847. doi:10.1016/j.jpsychires.2006.08.011

Sempertegui, G. A., Karreman, A., Arntz, A., & Bekker, M. H. J. (2013). Schema therapy for borderline personality disorder: A comprehensive review of its empirical foundations, effectiveness and implementation possibilities. *Clinical Psychology Review*, *33*, 426–447. doi:10.1016/j.cpr.2012.11.006

Sharp, C., & Romero, C. (2007). Borderline personality disorder: A comparison between children and adults. *Bulletin of the Menninger Clinic*, *71*(2), 85–114. doi:10.1521/bumc.2007.71.2.85

Specht, M. W., Chapman, A., & Cellucci, T. (2009). Schemas and borderline personality disorder symptoms in incarcerated women. *Journal of Behavior Therapy and Experimental Psychiatry*, *40*(2), 256–264. doi:10.1016/j.jbtep.2008.12.005

Stoffers, J. M., Völlm, B. A., Rücker, G., Timmer, A., Huband, N., & Lieb, K. (2012). Psychological therapies for people with borderline personality disorder. *Cochrane Database of Systematic Reviews*, *8*, CD005652. doi:10.1002/14651858.CD005652.pub2

Trull, T. J., Jahng, S., Tomko, R. L., Wood, P. K., & Sher, K. J. (2010). Revised NESARC personality disorder diagnoses: Gender, prevalence, and comorbidity with substance dependence disorders. *Journal of Personality Disorders*, *24*(4), 412–426. doi:10.1521/pedi.2010.24.4.412

Turner, R. M. (2000). Naturalistic evaluation of dialectical behavior therapy-oriented treatment for borderline personality disorder. *Cognitive and Behavioral Practice*, *7*(4), 413–419. doi:10.1016/S1077-7229(00)80052-8

Van Asselt, A. D., Dirksen, C. D., Arntz, A., Giesen-Bloo, J. H., Van Dyck, R., Spinhoven, P., … Severens, J. L. (2008). Out-patient psychotherapy for borderline personality disorder: Cost-effectiveness of schema-focused therapy v. transference-focused psychotherapy. *British Journal of Psychiatry*, *192*(6), 450–457. doi:10.1192/bjp.bp.106.033597

Verheul, R., Van den Bosch, L. M. C., Koeter, M. W. J., de Ridder, M. A. J., Stijnen, T., & Van den Brink, W. (2003). Dialectical behaviour therapy for women with borderline personality disorder: 12-month, randomised clinical trial in the Netherlands. *British Journal of Psychiatry*, *182*(2), 135–140. doi:10.1192/bjp.182.2.135

Wagner, T., Fydrich, T., Stiglmayr, C., Marschall, P., Salize, H. J., Renneberg, B., … Roepke, S. (2014). Societal cost-of-illness in patients with borderline personality disorder one year before, during and after dialectical behavior therapy in routine outpatient care. *Behaviour Research and Therapy*, *61*, 12–22. doi:10.1016/j.brat.2014.07.004

Walter, M., Bureau, J. F., Holmes, B. M., Bertha, E. A., Hollander, M., Wheelis, J., … Lyons-Ruth, K. (2008). Cortisol response to interpersonal stress in young adults with

borderline personality disorder: A pilot study. *European Psychiatry*, *23*(3), 201–204. doi:10.1016/j.eurpsy.2007.12.003

Wenzel, A., Chapman, J. E., Newman, C. F., Beck, A. T., & Brown, G. K. (2006). Hypothesized mechanisms of change in cognitive therapy for borderline personality disorder. *Journal of Clinical Psychology*, *62*(4), 503–516. doi:10.1002/jclp.20244

Widiger, T. A., & Weissman, M. M. (1991). Epidemiology of borderline personality disorder. *Psychiatric Services*, *42*(10), 1015–1021. doi:10.1176/ps.42.10.1015

Woodberry, K. A., & Popenoe, E. J. (2008). Implementing dialectical behavior therapy with adolescents and their families in a community outpatient clinic. *Cognitive and Behavioral Practice*, *15*(3), 277–286. doi:10.1016/j.cbpra.2007.08.004

Yen, S., Zlotnick, C., & Costello, E. (2002). Affect regulation in women with borderline personality disorder traits. *Journal of Nervous and Mental Disease*, *190*(10), 693–696. doi:10.1097/01.NMD.0000034744.11140.99

Yeomans, F. E., Clarkin, J. F., & Kernberg, O. F. (2002). *A primer of transference-focused psychotherapy for the borderline patient*. Northvale, NJ: Jason Aronson.

Yeomans, F. E., Levy, K. N., & Caligor, E. (2013). Transference-focused psychotherapy. *Psychotherapy*, *50*, 449–453. doi:10.1037/a0033417

Young, J. E. (1994). *Cognitive therapy for personality disorders: A schema-focused approach* (Rev. ed.). Sarasota, FL: Professional Resource Press.

Young, J. E., Klosko, J. S., & Weishaar, M. E. (2003). *Schema therapy: A practitioner's guide*. New York, NY: Guilford Press.

Zanarini, M. C., Frankenburg, F. R., Dubo, E. D., Sickel, A. E., Trikha, A., Levin, A., & Reynolds, V. (1998). Axis II comorbidity of borderline personality disorder. *Comprehensive Psychiatry*, *39*(5), 296–302. doi:10.1016/S0010-440X(98)90038-4

Zimmerman, M., Rothschild, L., & Chelminski, I. (2005). The prevalence of DSM-IV personality disorders in psychiatric outpatients. *American Journal of Psychiatry*, *162*, 1911–1918. doi.org/10.1176/appi.ajp.162.10.1911

13

The Treatment of Dissociation

An Evaluation of Effectiveness and Potential Mechanisms

Reed Maxwell,[1] Harald Merckelbach,[2] Scott O. Lilienfeld,[3] and Steven Jay Lynn[4]

[1] *Psychiatry Department, Weil-Cornell Medical College, New York, USA*
[2] *Clinical Psychological Science, Maastricht University, Maastricht, Netherlands*
[3] *Department of Psychology, Emory University, Atlanta, USA*
[4] *Psychology Department, Binghamton University, Binghamton, USA*

As evidence mounts for the efficacy and effectiveness of some psychotherapies in comparison with others, best practices among clinicians necessitate the use of evidence-based (i.e., research-supported) techniques across clinical settings when and where possible (American Psychological Association Presidential Task Force on Evidence-Based Practice, 2006; Lilienfeld, Ritschel, Lynn, Cautin, & Latzman, 2014). In the best-case scenario, therapists employ empirically supported treatments (ESTs) or more general research-supported strategies and methods within the broader context of evidence-based practice in psychology. The latter approach integrates research evidence, clinical expertise, and patient preferences and values—ideally, with research evidence at the apex—to guide assessment, case conceptualization, and treatment (Lilienfeld, Ritschel, Lynn, Cautin, & Latzman, 2013). When using evidence-based practice, therapists employ their accumulated knowledge of research and practice, along with clinical skills, to converge on the most appropriate research-supported treatments and techniques (i.e., "what treatment, by whom, is most effective for this individual with that specific problem, and under which set of circumstances"; Paul, 1967, p. 44). Clinical scientists then expertly deliver those treatment protocols and techniques in the context of cultivating strong therapeutic relationships (Norcross & Wampold, 2011) and leveraging transtheoretical (aka "nonspecific") factors, such as positive treatment expectancies (Prochaska & Norcross, 2013).

Provided that therapists meet these expectations, therapy ideally generates patient gains comparable to clinically significant outcomes (e.g., individuals no longer meet criteria for a diagnosis that served as a key selection criterion) observed in controlled trials of the treatments applied to participants with presentations similar to those of the patients (Kendall, Flannery-Schroeder, & Ford, 1999). Although questions persist regarding the extent to which controlled research protocols transfer to the more complex milieu of real-world clinical practices, at least some of these concerns may

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

be allayed as studies elucidate mechanisms of change (Kazdin, 2008) and increasingly demonstrate promising transfer from efficacy trials to effectiveness trials (Hunsley, Elliott, & Therrien, 2014). The former trials examine the outcome of psychotherapy under controlled conditions, whereas the latter trials examine the outcome of psychotherapy as practiced in real-world settings. At present, the evidence-based practice model of psychotherapy delivery remains the ideal in mental health care (Lilienfeld et al., 2013).

Paradoxically, the numbers of clinicians, educators, and students who are aware of, endorse, and adhere to evidence-based practice remain troublingly small (Lilienfeld et al., 2013). Lest the promises of an evidence-based approach fall on deaf ears, or no ears at all, large-scale efforts to disseminate ESTs to clinicians and consumers have received significant backing (McHugh, Murray, & Barlow, 2009). Division 12 (Society of Clinical Psychology) of the American Psychological Association maintains a list of dozens of ESTs for diverse diagnoses listed in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5), including schizophrenia, depression, bipolar disorder, panic disorder, eating disorders, attention-deficit hyperactivity disorder, and more (American Psychiatric Association, 2013).

With respect to the metrics for ESTs, Division 12 adheres to criteria for what were initially termed "empirically validated" treatments, outlined in Chambless and Hollon (1998). Specifically, Division 12 distinguishes treatments with "strong" research support from those with "modest" research support. Strongly supported, or "well-established," treatments have been shown to be efficacious by means of (1) two independently conducted randomized controlled trials (RCTs) demonstrating superiority to, or equivalence with, placebo, pill, or treatment as usual, or (2) a large series of single-case-design experiments ($n > 9$). Modestly supported, or "probably efficacious," treatments have been shown to be efficacious by means of (1) a smaller series of single-case designs; (2) one or more RCTs conducted by a single investigator or investigating team; or (3) two non-RCT experiments showing superiority to waitlist or control groups. Debate persists about whether these standards are optimal and which treatments meet these standards (e.g., Chambless & Ollendick, 2001).

A treatment may appear to be effective for many reasons (e.g., regression to the mean, nonspecific effects, demand characteristics; see Lilienfeld et al., 2014) that have little or nothing to do with the hypothesized mechanisms of treatment success (David & Montgomery, 2011). Accordingly, and as per David and Montgomery's (2011) framework, we will consider the level of empirical support on a continuum ranging from well supported or evidence based to absent and even contradictory evidence for both treatment effectiveness and mechanisms of the diverse interventions we review herein. Per David and Montgomery (2011), nine categories of empirical support for psychotherapies can be established from combinations of research support for interventions and the theoretical frameworks in which they fall. A treatment protocol achieves the highest category of empirical support (i.e., Category I: evidence-based psychotherapies) when both its interventions and its theoretical framework (i.e., proposed mechanisms of effects) receive research support roughly consistent with the highest standards of support outlined by Division 12 (i.e., Chambless & Hollon, 1998). At the other extreme, Category IX (bad-theory-and bad-intervention-driven psychotherapies) includes treatment protocols with empirical literature bases strongly disfavoring both their interventions and their proposed mechanisms of change.

Treatments for dissociative symptoms are not, yet, represented among ESTs. Part of the problem is that the very existence of some of the dissociative disorders, most notably multiple personality disorder, or, as it is called today, dissociative identity disorder (DID), has been the subject of intense scientific debate (e.g., Lilienfeld et al., 1999; Lynn, Lilienfeld, Merckelbach, Giesbrecht, & Van der Kloet, 2012; Lynn et al., 2014; Paris, 2012). With this caveat in mind, the present chapter compiles and synthesizes the state of the empirical evidence for the effectiveness of treatments for dissociative symptoms in adulthood, and argues for caution when treating dissociative symptoms, as workers in the field have expressed concern that some of these symptoms may arise as a function of the very techniques used to treat them (see Lilienfeld, 2007; Lilienfeld et al., 1999; Lynn, Condon, & Colletti, 2013; Spanos, Weekes, Menary, & Bertrand, 1986). Indeed, many DID patients show few or no clear-cut signs of major dissociative symptoms (e.g., different so-called "alter" personalities) prior to psychotherapy (Kluft, 1984), raising the possibility that such manifestations are often generated by treatment. Given the controversial nature of dissociative disorders, we will proceed with the understanding that, although patients may present with dissociative symptoms, questions linger regarding the trauma-based genesis of these symptoms and the validity of the diagnoses of DID and dissociative amnesia, in particular (see Lilienfeld et al., 1999; Lynn et al., 2012; Spanos et al., 1986; but for a contrary perspective see Dalenberg et al., 2012).

We organize our review in terms of dissociative disorders, as that is the context in which dissociative symptoms are described in the treatment literature. We also examine the admittedly limited evidence for mechanisms of change proposed or implied in the treatments reviewed and evaluate the strength of this evidence. We begin with a review of the DSM-5 dissociative disorders.

13.1 A Review of the DSM-5 Dissociative Disorders

The DSM-5 defines dissociative disorders as "disruption[s] of and/or discontinuity in the normal integration of consciousness, memory, identity, emotion, perception, body representation, motor control, and behavior" (American Psychiatric Association, 2013, p. 291). The DSM-5 differentiates among several subtypes of dissociative disorders and symptoms. Dissociative symptoms may be "positive" (e.g., fragmentation of identity, depersonalization, derealization) or "negative" (e.g., amnesia). Depersonalization/derealization disorder (DP/DR) is characterized by clinically significant persistent or recurring experiences of detachment from one's mind, self, or body (depersonalization) or feelings of unreality (derealization); dissociative amnesia is characterized by a profound inability to recall autobiographical information for an event or period of time, a particular aspect of an event, or details about one's identity and life history (thought to occur most often as a result of psychological trauma); and DID is characterized by (1) the presence of two or more distinct personality states or a subjectively experienced episode of what the patient believes to be possession by a spirit demon or other agent and (2) recurrent episodes of amnesia. Among these disorders, DID is thought to be the most severe, and its presentation has been associated with exposure to extreme trauma (especially physical, sexual, and/or other abuse) in childhood (Dorahy et al., 2014; but see Lynn et al., 2012, for a contrary view). Although the DSM-5 reclassified dissociative fugue as a subtype of dissociative amnesia, we will review treatments for its symptoms

separately in light of its standing as a distinct disorder in the previous three editions of the DSM (i.e., DSM-III-R, DSM-IV, and DSM-IV-TR; American Psychiatric Association, 1987, 1994, 2000). The DSM-5 consolidates the previously separately diagnosed disorders depersonalization and derealization into a single disorder, the aforementioned DP/DR (Lynn et al., 2016).

Transient (i.e., fleeting and/or "normal") dissociative experiences may occur in upwards of 50% of the general population (Ross, Joshi, & Currie, 1990). Estimates of lifetime prevalence rates for pathological dissociation in general and clinical samples range widely from 3% (Maaranen et al., 2005) to 10% (Sar, 2011), with dissociative disorders not otherwise specified (DDNOS) being the most prevalent and DID being the least prevalent (Johnson, Cohen, Kasen, & Brook, 2006). Within inpatient and outpatient settings, approximately half of all dissociative disorder diagnoses are DID. Rates of dissociative disorders tend to be higher among special populations (e.g., 39% of people with substance dependencies: Ross et al., 1992; 55% of women in prostitution: Ross, Anderson, Heber, & Norton, 1990; and 80% of exotic dancers: Ross, Anderson et al., 1990). Additionally, dissociative symptoms accompany almost all psychiatric diagnoses (Sar & Ross, 2006), including borderline personality disorder (Sar, Akyüz, Kugu, Ozturk, & Ertem-Vehid, 2006; Sar, Kundakçı et al., 2003), conversion disorder (Şar, Akyüz, Kundakçı, Kızıltan, & Doğan, 2004), and obsessive–compulsive disorder (Rufer, Fricke, Held, Cremer, & Hand, 2006). Notably, depersonalization and derealization symptoms commonly co-occur with anxiety disorders (e.g., panic disorder; Hunter, Phillips, Chalder, Sierra, & David, 2003).

Importantly, "comorbid" (co-occurring) dissociative symptoms predict poor treatment outcome and worse prognosis (e.g., borderline personality disorder: Kleindienst et al., 2011; obsessive–compulsive disorder: Rufer et al., 2006; posttraumatic stress disorder: Lanius, Brand, Vermetten, Frewen, & Spiegel, 2012; schizophrenia: Yu et al., 2010; panic disorder: Michelson, June, Vives, Testa, & Marchione, 1998; major depressive disorder: Soffer-Dudek, 2014). These findings raise the important question of whether dissociative symptoms reflect a flawed emotion regulation system, an issue to which we will return below.

Most evidence has not supported differences in the prevalence of dissociative disorders between genders (Spitzer et al., 2003; Tolmunen et al., 2007). Overall, studies from various countries indicate that dissociative disorders constitute a common mental health problem in both clinical practice and the community (Sar, 2011).

Psychotherapeutic treatments for each dissociative disorder have received what one might fairly call "minimal" attention in the literature, but some disorders are somewhat better represented than are others (e.g., DID vs. dissociative fugue). On the whole, there is a pressing need for more treatments for dissociative symptoms. Case studies are far more prevalent than controlled between-group studies, and there are no controlled trials (RCTs) of any treatment for any dissociative disorder (see Brand, Classen, McNary, & Zaveri, 2009; Brand, Lanius, Vermetten, Loewenstein, & Spiegel, 2012).

13.2 Treatments for Depersonalization/Derealization Disorder

Until 2003, publications concerning psychotherapies for DP/DR symptoms were limited to a small number of case studies and one larger case series (Ackner, 1954). These early

reports described successful outcomes using psychodynamic techniques alone (Torch, 1987), psychodynamic techniques with a pharmacological intervention (e.g., abreaction using diazepam; Ballard, Mohan, & Handy, 1992), behavioral methods (e.g., negative reinforcement; Blue, 1979), family therapy (Cattell & Cattell, 1974), and imaginal exposure (Sookman & Solyom, 1978).

One case study suggested that in vivo exposure did not alleviate symptoms (Sookman & Solyom, 1978). Torch (1987) noted that early psychodynamic clinicians regarded patients with depersonalization symptoms as either unsuitable for treatment or in need of greatly extended treatment (e.g., Schilder, 1939). He employed a psychodynamic approach to help a young adult male patient resolve a conflict presumed to trigger depersonalization, conceptualized as a psychological defense against obsessional self-scrutiny and feelings of worthlessness arising from failures to meet externally imposed parental demands during childhood. Torch reported that, as the intervention facilitated greater self-acceptance and transferred a sense of responsibility for childhood feelings of worthlessness from the patient to the parents, the hypothesized dissociative defense became unnecessary, and depersonalization symptoms lessened. Torch (1987) concluded that psychodynamic psychotherapy for depersonalization "must address issues of low self-esteem and compensatory hypervigilance with the self as an obsessional focus" (pp. 142–143).

Ballard et al. (1992) reported positive results in a case study of a middle-aged female patient with primary depersonalization symptomatology and an extensive history of unsuccessful psychiatric and pharmacological treatments. Consistent with Torch (1987), Ballard et al. (1992) observed "obsessional characteristics" in their patient as well as "discordance of a parent–child relationship" (p. 125) and also conceptualized depersonalization as a psychological defense. In this case, depersonalization purportedly defended the patient against an unfavorable self-image characterized by repudiated aggressive and sexual drives (i.e., an Electra complex) toward her mother and father, respectively. After several sessions of "explorative psychotherapy," the authors employed an abreaction technique using intravenous diazepam during which the patient expressed strong anger toward her mother. It should be noted, however, that there is little or no scientific support for such medication-induced abreaction methods, which appear to merely lower the threshold for reporting all symptoms, genuine or not (Piper, 1993). In subsequent sessions, the patient denied further depersonalization symptoms but reported substantial anxiety (e.g., panic attacks), after which the therapy focused on "anxiety management training." The authors noted that their patient's apparently positive response to abreaction was consistent with previous findings purporting resolution of depersonalization symptoms after use of an ether abreaction technique (Sargant & Shorvon, 1945).

In both case studies (Ballard et al., 1992; Torch, 1987), the authors conceptualized depersonalization as a defense against anxieties engendered by unwelcome discrepancies between the patient's sense of self as it "should be" and the patient's actual thoughts, feelings, and behaviors. These conceptualizations have theoretical precedent in the psychodynamic literature concerning the functions of depersonalization symptomatology. Frances, Sacks, and Aronoff (1977) ascribed depersonalization symptoms to disruptions in the stability and cohesiveness of self-representations (i.e., "intrapsychic conflict within the ego"). Their approach links symptoms to unique disruptions in self-concept associated with borderline, psychotic, narcissistic, and neurotic pathologies, and remediation

of these disruptions is expected to reduce depersonalization symptoms. Although this approach has not been evaluated empirically, it may receive circumstantial support from epidemiological data indicating a higher prevalence of depersonalization symptoms among individuals with personality pathology (e.g., Hunter, Sierra, & David, 2004).

The mechanism or mechanisms of change posited by this psychodynamic framework may be roughly characterized as the reduction of depersonalization via correction or reduction of anxiety-inducing discrepancies between the patient's "ideal" self and the patient's actual feelings, thoughts, and experiences. In this model, depersonalization symptomatology (e.g., detachment from the self) defends the patient against anxiety wrought by these seemingly unresolvable discrepancies. Resolution of these discrepancies via psychotherapy reduces the patient's need for the defense; thus, symptoms begin to remit. Nevertheless, the copresence of depersonalization and personality pathology implies neither a necessary defensive role for depersonalization nor a causal relation between dissociative and personality pathology in one direction or the other. To date, the causal links between dissociative symptoms and personality pathology have not been examined empirically.

Early case studies using behavioral techniques to treat DP/DR also reported encouraging results. Blue (1979) observed positive outcomes using directive therapy to treat a middle-aged woman with a history of unsuccessful pharmacological (e.g., tranquilizers, antidepressants) and psychotherapeutic (e.g., cognitive–behavioral therapy [CBT]) treatments for chronic anxiety and depersonalization symptoms. Directive therapy postulates that psychopathology persists when its expression is reinforced in social settings (Haley, 1963). Blue (1979) assumed that the patient intended to control both her depersonalization symptoms and "other people" (p. 905), including the therapist. Blue asked the patient to perform an unpleasant household chore whenever she experienced depersonalization. Blue reasoned that the patient's symptoms had been positively reinforced by others' responses. The patient had thereby learned to "control" others' behaviors (i.e., elicit reinforcement) by means of symptom manifestation. By associating symptom presentation with an unpleasant task and loss of control, psychotherapy compelled the patient to cease her symptomatology to regain autonomy. After a brief intervention using this technique, the patient's symptoms decreased substantively and remained remitted 3 months after termination.

The potential mechanism of change hypothesized by this unconventional treatment may be conceptualized as the reduction of symptoms via the modification of symptom reinforcement contingencies in the patient's social milieu along with the removal of secondary gain. The fact that Blue (1979) reported variations in symptoms as a function of reinforcement provides tentative support for the role of reinforcement in this case. Nevertheless, Blue's case study, like other case studies we review, was not equipped with control conditions (e.g., waitlist, no reinforcement, placebo) to rule out alternative explanations, such as regression toward the mean, expectation/attention effects, or naturally occurring fluctuations in symptoms over time. Moreover, the treatment may have merely reduced the patient's willingness to report depersonalization symptoms rather than reduced these symptoms per se.

Sookman and Solyom (1978) treated two patients with depersonalization and co-occurring anxiety using imaginal and in vivo exposure techniques, respectively. The patients were middle-aged individuals with extensive histories of depersonalization symptoms with obsessional tendencies (e.g., compulsive checking of household

appliances) and elevated anxiety. Unlike the case studies described thus far, these authors incorporated pre- and postintervention outcome measures to assess change in anxiety and depersonalization symptoms after termination of psychotherapy. The first patient received 10 weeks of two 1-hour treatment sessions "in addition to 8 flooding sessions for her agoraphobia" (Sookman & Solyom, 1978, p. 1544). The patient underwent imaginal flooding using "taped narratives of depersonalization episodes based on [the patient's] description of her anxiety-provoking thoughts and sensorial experiences" during "intense attacks of depersonalization, derealization, and panic" (p. 1544). The authors reported that the patient's depersonalization was reduced by 83% on "psychiatric ratings" and by 67% on "self-rating" after psychotherapy. Other anxious, depressive, and obsessional symptoms also improved.

The second patient received in vivo exposure over 2.5 months with five 1-hour treatment sessions per week and several prolonged (2- to 3-hour) flooding treatments. During flooding, the patient was exposed to real-world situations that provoked depersonalization symptoms and was prompted not to perform rituals to terminate these symptoms (i.e., response prevention). A thought-stopping technique was employed to decrease ruminations about symptoms. Although the intervention did not appear to impact depersonalization symptoms, the patient's anxiety, depression, and obsessive symptoms showed moderate improvements that persisted for 6 months.

In the former case, the potential mechanism of symptom reduction appears to have been habituation to depersonalization cues and diminution of the patient's anxiety and fear responses to these cues through repeated exposure to triggering scenarios. However, to date, this mechanism has not been examined systematically with regard to the targeted symptoms.

Although these single case studies using psychodynamic and behavioral interventions yielded apparently promising results, mixed results from an early influential case series (54 patients treated with undefined "psychotherapy"; Ackner, 1954) may have contributed to a premature consensus regarding a poor prognosis for DP/DR, which stifled treatment research (Hunter, 2013; Hunter et al., 2003). Nonetheless, individuals with DP/DR have continued to seek help through psychotherapy, and clinicians have continued to provide it. Simeon, Knutelska, Nelson, and Guralnik (2003) presented a case series of 117 patients formally diagnosed with depersonalization using the Structured Clinical Interview for DSM-IV Dissociative Disorders (Steinberg, 1994a). The authors reported that most individuals described histories of psychotherapeutic and pharmacological interventions of variable types, durations, and intensities. A large number of these individuals stated that psychotherapy had facilitated greater understanding of their symptoms and ability to cope with them; however, only CBT and hypnosis were associated with reports of sporadic improvements (i.e., "definitely better") in DP/DR symptoms. Steinberg (1991) also claimed that hypnotherapy may be useful in treating DP/DR symptoms. Nevertheless, the sanguine appraisals of some authors notwithstanding (e.g., Brand, 2016), numerous authors have raised serious concerns about the potential suggestive or iatrogenic effects of using hypnosis and allied suggestive procedures with dissociative patients (Lilienfeld et al., 1999; Lynn et al., 2013).

Simeon (2004) observed that no intervention had received empirical support at the time of her review of treatments for DP/DR (e.g., psychodynamic, cognitive–behavioral). She suggested that the case studies employing psychodynamic techniques (e.g., Ballard et al., 1992; Torch, 1987) showed some promise in treating less chronic

and less unremitting DP/DR symptoms. For individuals with poorly processed trauma histories (e.g., patients with prolonged life stress; patients with unresolved bereavement issues), Simeon noted that trauma-focused therapies (e.g., exposure and cognitive processing therapy; Resick & Schnicke, 1992) may provide some relief for individuals with symptoms that appear to be related to these traumas.

Since Simeon's review, no publications have addressed the use of cognitive processing therapy in treating DP/DR. Simeon observed that a cognitive–behavioral approach developed by Hunter et al. (2003; see next paragraph) showed some promise in treating DP/DR. Finally, she noted that miscellaneous techniques tailored to other aspects of patients' presentations (e.g., relaxation and meditation techniques for patients with high arousal; conversely, techniques that heighten arousal for patients with low arousal) should also be incorporated when indicated. None of the studies used mediational analyses to provide a formal evaluation of hypothesized mechanisms of treatment gains.

Since Simeon's (2004) review, the cognitive–behavioral approach developed by Hunter et al. (2003) has received preliminary empirical support. Hunter and colleagues contended that DP/DR evidences important dissimilarities with other dissociative disorders (e.g., absence of memory loss; no loss of awareness of the self or external world; weak relationship with trauma history) and compelling similarities with anxiety disorders (e.g., cognitive symptoms associated with increased arousal; dizziness; avoidance of provoking situations). In particular, the authors observed a strong link between DP/DR symptoms and panic symptoms (e.g., overlap with DSM-IV criteria; onset of DP/DR symptoms in tandem with onset of panic disorder).

Hunter and colleagues developed a cognitive model of DP/DR disorder in which catastrophic misinterpretations of DP/DR symptoms (e.g., signs of "madness," loss of control) exacerbate anxiety and subsequently DP/DR symptoms (see Clark, 1986). To manage symptoms in the short run, patients may adopt excessive self-monitoring and checking behaviors, avoidant (i.e., "safety") behaviors, and so on. Nevertheless, these behaviors perpetuate a vicious cycle of anxious vigilance toward, and catastrophic misinterpretations of, symptoms, distress, and further anxious vigilance. The authors advanced a model that incorporates psychoeducation/normalizing, diary-keeping (to highlight symptom variability), avoidance-reduction and self-focused attention, and challenging catastrophic assumptions.

Hunter, Baker, Phillips, Sierra, and David (2005) evaluated this CBT protocol with 21 patients diagnosed with DP/DR disorder. They delivered treatment in an outpatient setting, and each patient received between 4 and 20 psychotherapy sessions. Therapy consisted of three phases: Phase 1 focused on symptom reduction and psychoeducation; phase 2 targeted symptom avoidance, "safety behaviors," and self-focused attention; and phase 3 focused on progress maintenance and relapse prevention.

The authors reported improvements in functioning posttreatment and after 6 months. Significant reductions in clinician ratings on the Present State Examination (Wing, Cooper, & Sartorius, 1974) were also observed, and 29% of participants no longer met criteria for DP/DR at the conclusion of psychotherapy. The authors concluded that their protocol might possess some efficacy; however, RCTs would be required to confirm their findings and identify mechanisms associated with treatment gains. Moreover, it is unclear which components of the multifaceted intervention, in isolation or in combination, contributed to apparent treatment changes.

Most recently, Hunter (2013) reviewed the CBT model of DP/DR and introduced the model's approach to assessment, case conceptualization, and intervention using a case study. Yet the CBT model is not without its critics. For example, some researchers have noted that the relationship between depersonalization and anxiety may not be as "special" as it appears (Sierra, Medford, Wyatt, & David, 2012). Nevertheless, CBT remains the most empirically supported psychotherapy for DP/DR disorder. Still, uncontrolled case studies and empirical investigations that do not control for nonspecific treatment effects, regression to the mean, demand characteristics, naturally occurring fluctuations in symptoms over time, and other possible contributors to symptom alleviation (see Lilienfeld et al., 2014) leave many questions unanswered regarding the mechanisms responsible for apparent treatment success. They also leaved unresolved the question of whether the treatment produces gains above and beyond no treatment.

Since Simeon's (2004) review, anecdotal support for psychodynamic approaches continues to appear in the literature. For example, Gentile, Snyder, and Gillig (2014) observed that an eclectic approach that alternates between supportive and psychodynamic approaches might be indicated for some patients with DP/DR. According to these authors, supportive techniques (e.g., crisis intervention, reinforcement of existing coping strategies) may better fit patients experiencing acute DP/DR symptoms in tandem with severe anxiety/depression, whereas psychodynamic techniques (e.g., self-examination and self-reflection) may better fit patients experiencing milder symptoms. However, as no RCTs of psychodynamic or any treatment for dissociation have been conducted, additional empirical work is required before psychodynamic psychotherapy can be recommended as being supported by research. Lastly, it is unclear whether early studies reporting successful treatments for depersonalization disorder addressed depersonalization symptoms solely, or both depersonalization and derealization symptoms. Future research might determine whether existing interventions are helpful for both DP and DR, or whether separate interventions for each symptom constellation are needed.

13.3 Treatments for Dissociative Identity Disorder

In 2009, the International Society for the Study of Trauma and Dissociation commissioned a third revision of its influential "Guidelines for Treating Dissociative Identity Disorder," and the resulting document was published in 2011 (International Society for the Study of Trauma and Dissociation, 2011). The authors noted that specialized DID/DDNOS (i.e., variants of DDNOS with similarities to DID) treatment guidelines should supplement rather than supplant established general psychotherapeutic and pharmacological principles. Although the guidelines assert that research on treatments for DID remains in its infancy, expert consensus and outcome studies have yielded a broad rubric for case conceptualization and prognosis.

Nevertheless, the guidelines were not compiled with the consultation of experts skeptical of the traumatic origin of the symptoms of DID, despite the fact that the evidence for such a universal origin is at best equivocal and highly controversial (e.g., Lynn et al., 2014). Nor did the guidelines voice appropriately strong reservations regarding the use of some of the highly suggestive treatments recommended, such as encouraging communication among supposed alters, which could reinforce the manifestation of alter identities. Accordingly, the guidelines do not represent a genuine or representative

scientific consensus among experts. The guidelines recommend that clinicians assist patients in achieving integrated functioning of the patient's multiple identities in the context of the "whole person" responsible for the behavior of all identities: Treatment should facilitate coordination and communication among the patient's identities whenever possible until these identities "fuse" (i.e., lose separateness; Kluft, 1993). However, in some cases, "resolution" (i.e., a degree of coordination or cooperation among alternate identities or "alters" that promotes healthy functioning) may be a more tenable outcome. The guidelines contend that a flexible, phase-oriented, and temporally unconstrained approach to treatment facilitates improvement more than a time-limited and narrow approach. In general, outcome studies suggest that patients benefit from more comprehensive psychotherapies (Groenendijk & Van der Hart, 1995), and 16–33% of treated patients are said to achieve final fusion (Coons & Bowman, 2001; Coons & Sterne, 1986; Ellason & Ross, 1997). Although higher-functioning patients tend to benefit most rapidly from treatment, patients with comorbid diagnoses (e.g., mood disorders; personality disorders) and more extensive psychiatric histories also show improvement over time (Loewenstein, 1994; Loewenstein & Putnam, 2004).

The guidelines recommend three phases to address symptom management, trauma processing, and identity fusion: a first phase for establishing safety, stabilization, and symptom reduction; a second phase for confronting, working through, and integrating traumatic memories; and a third phase for identity integration and rehabilitation. These phases are consistent with expert recommendations in the treatment of other complex trauma-related disorders (e.g., Courtois & Ford, 2009). However, researchers have not yet compared this phase-oriented approach with any number of other approaches that do not focus on traumatic memories and working with "identities."

According to the guidelines, individual-oriented psychodynamic psychotherapy is the most commonly recommended treatment for DID, but use of adjunctive techniques and interventions—ranging from CBT to dialectical behavior therapy to family therapy to expressive therapy to behavior therapy (e.g., exposure therapy) to hypnotherapy—is authorized and often encouraged. The guidelines advise against group psychotherapy as a primary treatment modality for DID and further advise against electroconvulsive therapy. Although the guidelines present what the authors consider circumstantial evidence for the usefulness of hypnosis in facilitating psychotherapy for patients with DID (e.g., a positive correlation between hypnotic suggestibility and DID symptomatology; a positive relation between hypnotic suggestibility and positive outcomes in hypnotherapy for DID; Frischholz, Lipman, Braun, & Sachs, 1992), the guidelines do not present any evidence (e.g., case studies, controlled studies) for beneficial effects in this context. Moreover, there exists substantial concern that hypnosis is a highly risky, suggestive procedure in this context, as it may inadvertently fuel the creation of new memories and believed-in identities (see Lynn, Rhue, & Kirsch, 2010). Evidence suggests that these guidelines have contributed to effective cross-cultural treatment of DID by international clinicians (Brand, Myrick et al., 2012; Spiegel et al., 2011). Nevertheless, no controlled studies directly compare interventions that are based on the guidelines with interventions that are not.

Absent RCTs and studies that compare treatment with no treatment or waitlist comparisons, the guidelines present at best preliminary evidence for the value of psychotherapy for DID. Moreover, there is no clear basis to warrant the use of one treatment modality in lieu of another (e.g., hypnotherapy, cognitive analytic therapy). Importantly,

researchers skeptical of the posttraumatic model of dissociation (i.e., in which trauma predictably engenders dissociative symptoms in vulnerable individuals; Gee, Allen, & Powell, 2003; Lilienfeld, 2007; Lynn et al., 2012) have drawn on a large body of circumstantial but converging evidence to contend that certain treatments for DID may cause harm to patients. Specifically, techniques that are highly suggestive in nature can arguably engender and/or reinforce the presentation of multiple identities. These techniques are ones that encompass bringing forth and "searching for" purported "personalities" or "identities"; using techniques such as diaries, journals, and hypnosis and other imagery-based techniques to recover supposedly repressed and/or dissociated memories; and inviting alters to interact with one another and report separate "histories" (see Lynn et al., 2013). Indeed, case reports in the literature describe florid posttraumatic stress symptomatology and suicide attempts in DID patients during or soon after treatment focused on memory recovery (e.g., Fetkewicz, Sharma, & Merskey, 2000). Such pre–post comparisons do not demonstrate causality, but they raise serious concerns regarding a potential iatrogenic role of highly suggestive interventions, especially in view of abundant laboratory data indicating that such interventions can induce confidently held false memories in sizeable minorities of individuals.

More broadly, the question of iatrogenic harm persists aside from other questions about the validity of DID (see Boysen & VanBergen, 2013; for a critical reply, see Brand, Loewenstein & Spiegel, 2013), and we encourage readers to consult the literature for perspectives from both sides. Nevertheless, we strongly recommend that therapists eschew suggestive and memory recovery techniques in favor of empirically supported interventions to treat specific dysfunctions in emotional regulation, the psychological repercussions of trauma (e.g., posttraumatic disorder [PTSD]), and problems in identity and interpersonal relationships.

Brand and Loewenstein (2014; see also Brand, 2016) countered the claim of iatrogenic harm with a review of the largest treatment outcome study to date of patients with DID and DDNOS: the Treatment of Patients with Dissociative Disorders study (Brand, McNary et al., 2013). Brand and colleagues' positive review gives credence to clinicians' claims that psychotherapy for these disorders helps patients. Nevertheless, Brand and colleagues observed a number of serious methodological weaknesses across these publications, including regression to the mean, limited sample sizes, and nonrandomized designs, but they concluded that the overall findings were promising.

Regarding case studies, Brand and Loewenstein (2014) found reports of positive treatment outcomes across countries (Cagiada, Camaido, & Pennan, 1997; Hove, Langfeldt, Boe, Haslerud, & Stoereth, 1997; Martinez-Taboas & Rodrigues-Cay, 1997; Sar, Ozturk, & Kundakçı, 2002; Sar & Tutkun, 1997; Van der Hart & Boon, 1997) and theoretical orientations. Clinicians have reported success in treating DID using CBT (Caddy, 1985; Martinez-Taboas & Rodrigues-Cay, 1997), phenomenological therapy (Ellerman, 1998), contextual therapy (Gold et al., 2001), cognitive analytic therapy (Kellett, 2005), and feminist therapy (Riggs & Bright, 1997). We will review these case reports and others purporting to show success in treating DID with psychotherapy, and we will elucidate the apparent yet untested mechanisms of change within each report when potential mechanisms can be inferred or hypothesized.

Caddy (1985) treated a 29-year-old female patient who presented with two alters and co-occurring anxiety and personality-related problems. Caddy allotted 3 months for the development of a collaborative therapeutic relationship, 3 weeks for training in

progressive muscle relaxation, 3 months for systematic desensitization training, assertiveness training over the 6th month of treatment, and extensive cognitive restructuring for the patient and each "alternate" over 14 months. Caddy observed that "it was the lack of internal consistency, the conflict ambiguity that existed between many of the belief systems and the behavioral routines of [the patient] and her alternates that created the … difficulties she experienced." Furthermore, Caddy argued that these problems of internal consistency provoked the need for "the dissociative processes that produced [the alternates]" (p. 286). Under Caddy's guidance, the patient began to restructure her cognitions about her worth and behave in ways consistent with these restructurings. For example, the patient began to terminate destructive relationships and make lifestyle choices more consistent with her voiced religious values. Caddy observed that the patient's dissociative experiences (i.e., presentations of alters) decreased as her coping skills and restructured cognitions increased and took hold. Caddy noted that the patient's treatment gains persisted years after therapy.

The hypothesized mechanism of symptom reduction appeared to be the enhancement of cohesion between the patient's daily behavioral routines and belief systems via cognitive restructuring. As the patient's behavior began to conform more to her values and beliefs, alters associated with discrepant behaviors presented less frequently. Still, this potential mechanism has not been evaluated, and the treatment, like many others reviewed to this point, contains numerous component interventions (e.g., desensitization, assertiveness training) that were not assayed independently or interactively by means of dismantling designs, thereby constraining empirically based inferences regarding moderators and/or mediators of treatment gains.

With the purported success of Caddy's (1985) cognitive intervention in mind, Ross and Gahan (1988) produced a general cognitive analysis of DID to guide cognitive conceptualization and treatment. According to Ross and Gahan, eight core assumptions learned through early childhood abuse (e.g., different parts of the self are separate selves; the past is present; the primary personality must be punished) are common among many DID patients and are often evident within alters' self-statements or evaluations of one another. Ross and Gahan posited that use of cognitive restructuring techniques to challenge and correct alters' cognitive distortions from these eight assumptions may reduce dissociative symptoms and foster final fusion. Ross and Gahan contended that cognitive interventions should be used alongside abreaction, negotiation among alters, and other techniques extraneous to CBT.

Ross (1997) later concluded that DID may be understood as a disorder of attachment to the perpetrator of early childhood abuse. His cognitive analysis coincides with the psychodynamic workup of Liotti (1999), who similarly theorized that "the construction of multiple, dissociated working models of self and the attachment figure in the context of early disorganized attachment prompts us to regard [DID] … as the sign and substance of a primary breakdown in the intersubjective processes … that normally generate an integrated and coherent sense of self" (p. 292). However, whereas Liotti refrained from extrapolating concrete avenues for psychotherapy, Ross contended that "the clinical problem requiring solution is the blockage of information flow … the purpose of amnesia barriers and abnormal personification of parts of the self is … to keep modules responsible for attachment uncontaminated by traumatic information" (p. 375). Ross again concluded that cognitive techniques, combined with other approaches, may facilitate resolution of this attachment problem.

Ross' cognitive and functional analysis of DID (i.e., symptoms function to preserve attachment to perpetrator(s)) has not been empirically applied in a published case study. The potential mechanism of therapeutic change may be characterized as symptom reduction by way of cognitive restructuring that addresses the problem of attachment to the perpetrator of early childhood abuse. Nevertheless, this potential mechanism has not been evaluated, and Ross' eight core assumptions have not been validated or subjected to empirical scrutiny.

Martinez-Taboas and Rodriguez-Cay (1997) used psychotherapy and a pharmacological intervention to treat a 29-year-old female DID patient with a history of anxiety, depression, and numerous alters that the therapists concluded represented abusive people from her childhood (e.g., an alter identified as a sexual abuser). The clinicians treated the patient in an initial stage focusing on confirming the DID diagnosis and communicating effectively with principal alters; a second stage targeting dissociative defenses and replacing them with "more mature ones"; and a third stage using behavioral, cognitive, and experiential therapies to "consolidate her new self" (p. 145). The clinicians encouraged the patient to actively communicate with her alters, which appeared to help her access previously avoided negative affect (i.e., deactivate the patient's dissociative defenses). The clinicians also educated the patient's alters about their roles in the patient's life (e.g., "their 'mission' of duplicating the actions of [the patient's] abusers," p. 145), which appeared to dissuade the alters from persisting. Most of the alters purportedly fused with the patient's identity, and those that did not presented less frequently over time. Finally, the authors used CBT and other therapeutic modalities to help the patient develop social skills and restructure maladaptive schemas developed in childhood (e.g., a fear of abandonment). Over the course of therapy, the patient used psychotropic medication to manage depressive and anxious symptoms. The clinicians reported that the patient's gains persisted for 2 years after formal termination.

According to Martinez-Taboas and Rodriguez-Cay (1997), the mechanisms of change appear to be threefold: (1) exposure to previously avoided negative affect via "communication" with alters; (2) functional analysis of the alters' identities in the context of the patient's whole experience; and (3) cognitive restructuring of maladaptive beliefs associated with early childhood trauma(s). Skeptics of this treatment could legitimately contend that the focus on "communication with alters" was a highly risky and potentially iatrogenic strategy and that the seemingly positive effects of treatment could be attributed to the psychopharmacology or any number of the varied interventions.

Regarding DID treatment, Ellerman (1998) asserted that "my experience … indicates that effective treatment does not hinge upon understanding either traumatic precipitants or the adaptive role of dissociation in mitigating childhood trauma" (p. 70). Rather, Ellerman argued that treatment requires the therapist to behave "as if" the patient's narrative is accurate in order to restructure it therapeutically in collaboration with the patient. These restructurings are designed to dispense with extensive reviews and reconstructions of past traumas, integrate the patient's disconnected memories into a coherent whole, and ultimately facilitate final fusion. Ellerman treated a 51-year-old male with a 30-year history of multiple alters associated with chemical dependencies and antisocial behaviors. First, Ellerman ascertained the identities and functions of the patient's alters (e.g., "persecutor"). Then, he assumed the role of "therapeutic storyteller" to reshape the patient's personal story in ways that dispensed with DID and assorted alters as explanations for the patient's experiences. These narrative changes "[replaced] metaphors of

division with images of unity—leading [the patient] to renounce the illusion of separateness" (p. 75). Ellerman also encouraged the alters to share their memories with the patient, and he shaped the patient's narrative so that a consolidation of memories would result in final fusion (i.e., an experience of himself as an undivided person). According to the author, this collaborative, present-focused storytelling reduced the patient's dissociative symptoms and facilitated healthier functioning.

The mechanism of therapeutic change may be characterized as the reduction of symptoms via (1) systematic and collaborative reattribution of DID symptoms to causal agents other than alters and (2) dissociation of alters from events in episodic memory and reunification of previously separated memories into a single narrative attributed to the whole patient. This potential mechanism, although interesting as it conceptualizes the patient's alters as metaphors in a narrative context and maintains a present focus, has not yet been subjected to empirical inquiry. Nevertheless, narrative interventions, which do not assume that alters represent literal divisions in personality, open up an alternative, non-trauma-oriented pathway to the treatment of DID.

Gold et al. (2001) used a similar present-focused approach, dispensing with extensive reviews of both historical traumas and alters. The authors' contextual approach relates DID symptomatology to long-standing deficits in life and coping skills that "[reflect] the chaotic and inconsistent interpersonal environment in which the individual with DID was reared" (p. 9). This contextual approach contends that maladaptive learning histories associated with the dysfunctional family context of childhood trauma, rather than trauma per se, engender and maintain dissociative problems. Thus, the authors aimed to correct ineffective learning histories and effect helpful changes in interpersonal functioning, self-understanding, and daily living skills to reduce symptoms including identity fragmentation. The authors presented a small case series ($n = 3$) with accompanying pretreatment, posttreatment, and follow-up data indicating improvements in functioning. The authors suggest that addressing and improving life and coping skills decreases DID symptoms. Nevertheless, this mechanism has not been evaluated in the context of dissociative symptoms.

Another case illustrates a learning approach to the treatment of dissociative symptomatology. Kohlenberg (1973) manipulated reinforcement contingencies—reduced attention to alter displays—and found that this intervention was followed by the disappearance of alter manifestations. The close link between reinforcement and the symptoms suggests that reinforcement might be a viable mechanism that contributes to the symptoms of DID.

Hypnotic treatments for DID are controversial, given the risk of suggestive techniques in psychotherapy. Still, noting the apparent historical success of hypnosis in treating DID (e.g., Allison, 1974; Despine, 1840; Janet, 1889/1973; Phillips & Frederick, 1995) and high levels of hypnotic suggestibility among DID patients, Kluft (2012) applied hypnotically facilitated psychodynamic techniques in treating an adult female professional with an extensive history of sexual trauma and presenting DID. Kluft reviewed 21 hypnotically informed interventions for use in systematically accessing alters in session and facilitating safe and protective forays into unpleasant affect (e.g., shame). Although Kluft did not operationalize hypnosis as a primary intervention or mechanism of therapeutic change, he reported that "my clinical experience in the successful integration of over 180 DID patients indicates that the quality and efficacy of the treatment I can provide is profoundly improved by the use of hypnotherapeutic interventions" (p. 153).

Relatedly, Smith (1996) observed that "psychotherapeutic treatment of persons with DID frequently includes judicious use of hypnosis" (p. 222). Both practitioners conjectured that hypnotherapeutic interventions capitalize on DID patients' frequently elevated hypnotic suggestibility to bolster treatment gains yielded by other psychotherapeutic techniques.

On the other hand, Fine (2012) posited a central role for hypnosis in treating DID. Fine observed that "any therapy for [dissociative disorders] is, by necessity, a hypnotically driven therapy; all [cognitive–behavioral] therapy for DID is a cognitive–behavioral hypnotherapy" (p. 332). According to Fine, DID alters are disparate "entrenched trance states" (p. 337) amenable to integration by way of cognitive–behavioral hypnotherapy. Moreover, the use of formalized hypnotic techniques facilitates patient mastery of dysfunctional autohypnotic states that characterize DID symptomatology. Hypnotic techniques, in conjunction with standard cognitive–behavioral techniques (e.g., cognitive restructuring) and abreactive work, increase fluidity between and among alters, reduce affect intensity, and increase cognitive flexibility.

According to Fine, DID can be understood primarily as a disorder of hypnotic faculties. Thus, the core mechanisms of therapeutic change (e.g., patient mastery of autohypnotic states gone awry via hypnotic techniques) are hypnotic. Nevertheless, this potential mechanism has not been evaluated, and it is difficult, if not impossible, to evaluate it empirically without a clear delineation of what constitutes a "trance" or an "autohypnotic state." Moreover, studies have not compared the same interventions administered in a hypnotic versus a nonhypnotic context. An important risk that hypnosis carries is that hypnotically elicited memories may be held with unwarranted confidence ("memory hardening") regardless of their accuracy (Lynn et al., 2010), so a strong argument can be advanced that hypnotic techniques should be avoided in the treatment of dissociative symptoms for most purposes other than relaxation (Lilienfeld, 2007).

With respect to contemporary psychodynamic interventions, Burton and Lane (2001) reviewed transference and countertransference components in DID treatment using a relational psychoanalytic approach. The authors noted that contemporary relational psychoanalytic practitioners have shown increasing interest in the transference–countertransference matrices (i.e., complex reactions between therapist and patient) arising in session with DID patients. The authors observed that "the technical use of empathy, enactment (i.e., externalization of the patient's internal experience of the therapist) … and projective identification (i.e., the patient's unconscious inducement of the therapist to assume a particular role) with all alter personalities form the core of the treatment within the relational (psychoanalytic) model" (p. 318).

The authors contend that final fusion is most efficiently facilitated by means of direct contact with alters and a relational approach to the management of the transference–countertransference matrix of therapist–alter relationships. Relational therapy purportedly enables the patient to develop a capacity to sustain internal conflict with subsequent reductions in symptoms. The authors identified dependent, aggressive, and eroticized transferences and corresponding countertransferences as particularly common challenges in psychotherapy with DID patients.

These authors' conjectures may receive circumstantial support from findings indicating that the therapeutic alliance yields stronger effects for dissociative disorder treatment outcomes than for other patient population outcomes (Cronin, Brand, & Mattanah, 2014). More specifically, self-rated alliance scores statistically predicted

better outcomes after controlling for patient adaptive capacities. Nevertheless, ascribing a causal role to the therapeutic alliance in promoting positive findings must remain highly tentative, as the therapeutic alliance may reflect positive changes in therapy rather than be the cause of such changes (Lilienfeld et al., 2013). Moreover, no studies have compared treatments that involve communication with supposed alters with treatments that eschew such communication, or therapies that focus on transference with treatments that do not.

Kellett (2005) used an A/B single-case experimental design (Turpin, 2001) to test the effectiveness of cognitive analytic therapy (CAT) for DID. Employing methods "[representing] good practice" (p. 2) of a single-case experimental design, the author examined the effects of CAT on state and trait dissociation, as well as general aspects of mental health and personality. Observations on multiple validated self-report measures were taken daily during baseline assessment (A), treatment (B), and 6-month follow-up periods. CAT assumes that DID symptoms arise by way of dissociated self-states (e.g., abandoning, abusing, contemptuous) and associated dysfunctional state-switching (i.e., the multiple self states model; see Ryle, 1997). Dissociated self-states supposedly produce behaviors, interactions, and affects discrete from those of other self-states, and each self-state exhibits amnesia about the others. CAT first employs a three-session assessment period yielding a narrative reformulation of the patient's troubles. The therapist provides a written narrative to clarify symptoms and reinforce a treatment focus on impaired functioning. CAT then requires therapist and patient to construct a sequential diagrammatic reformulation (SDR) of the patient's dissociated self-states and use the diagram to "[create] greater reflective capacity in the patient, [manage] transference in the sessions and [label] exits for the self-states more productive to mental health" (Kellett, 2005, p. 63). At the termination of CAT, patient and therapist each write a "goodbye letter" summarizing therapeutic achievements, key insights, and future challenges. In a case study, Kellet (2005) employed CAT with a female patient with DID and an extensive history of psychological distress (e.g., self-reported early childhood sexual abuse, self-harm, inpatient hospitalization). Kellet observed reductions in state dissociation symptoms (as well as other mental health symptoms) over the course of treatment, including sudden gains following the narrative reformulation and construction of the SDR.

The core mechanism of treatment might be characterized as reduction of symptoms via collaborative functional analysis of the patient's dissociated self-states. However, the case study did not include a validated measure of "self-states," and the overall treatment included multiple components, such as extensive assessment, that could produce insight and enhance treatment expectancies, reformulation of self-states, and a focus on impaired functioning, with apparent efforts to build rapport with the patient. Hence, attributing improvement to the intervention itself is difficult or impossible.

In an especially bizarre study, Bull, Ellason, and Ross (1998) reviewed 47 incidents of exorcism conducted on 15 patients with DID. The authors interviewed these patients using the Exorcism Experiences Questionnaire (i.e., a battery of inquiries about a patient's experiences of exorcism; Bowman, 1993) and recorded five types of exorcism derived from configurations of eight methodological factors: (1) patient permission, (2) noncoercion, (3) patient active participation, (4) exorcist understanding of DID dynamics, (5) contextualization of exorcism within psychotherapy, (6) compatibility

of procedure with patient spirituality, (7) incorporation of patient belief system, and (8) use of patient-initiated, self-conducted exorcism. It perhaps goes without saying that the use of exorcism is exceedingly problematic, both scientifically and ethically, especially given its explicit or at least implicit presumption of demonic possession as a cause of dissociative symptoms. The authors delineated the five types by the number of methodological factors employed (e.g., the first type employed no factors; the fifth type employed all factors). Analysis of all incidents yielded 23 mixed and 24 positive experiences, and more positive experiences occurred for types of exorcism that employed more methodological factors. Type 5 exorcisms (i.e., all factors employed) comprised all 24 patient-reported "very positive" exorcism experiences, whereas Type 1 (i.e., no factors used) exorcisms comprised three patient-reported "very negative" exorcism experiences. Patients reported mixed experiences for intermediate types employing some but not all factors. The authors concluded that noncoercive exorcism allowing for patient choice and autonomy in tandem with psychotherapy may be helpful for some patients with DID.

These conclusions are at best dubious. Although mechanisms of treatment cannot be inferred readily due to the heterogeneity of the cases and only vaguely described procedures, it seems reasonable to posit that therapeutic gains in positive incidents were mediated by (1) inclusion of spiritual patients' beliefs and associated expectations and (2) enhanced therapeutic alliance between therapist and patient via incorporation of the patient's spiritual worldview. These mechanisms have not been evaluated systematically, and serious ethical questions can be raised regarding "performing exorcisms" with patients of any ilk.

Among the approaches used across the case reports reviewed, CAT (Kellett, 2005) and contextual therapy (Gold et al., 2001) boast the strongest, albeit limited, empirical support: More and better controlled case reports of successful treatment are needed before these interventions can be considered to be supported by research. Although our review of case reports and clinical conjectures highlights treatments of DID symptomatology with a sole focus on the patient, studies have shown that treatments tailored to improve DID patients' social contexts (e.g., family systems interventions: Benjamin & Benjamin, 1992; Chiappa, 1994; Twombly, 2013; Miller's feminist relational model: Riggs & Bright, 1997; and group psychotherapy: Fine & Madden, 2000; Ross, 2008) have shown promise in helping patients cope with symptoms. Mechanisms of change in such contextual interventions are beyond the scope of this review. Interested readers are advised to consult the literature for appropriate reviews of such mechanisms (e.g., Burlingame, MacKenzie, & Strauss, 2004; Hoffman, 1981; Yalom & Lecszcz, 2005), although systematic investigations of mechanisms have not been reported.

As noted, Brand, Classen, McNary, and Zaveri (2009) reviewed a body of dissociative disorder treatment literature including three nonrandomized treatment outcome studies for DID (i.e., Choe & Kluft, 1995; Ellason & Ross, 1996, 1997, 2004; Gantt & Tinnin, 2007; Ross & Haley, 2004). These studies included clinical populations with co-occurring DID and other diagnoses (e.g., DID with co-occurring borderline personality disorder) recruited from inpatient and outpatient trauma programs. Per Brand and colleagues, descriptions of the treatments in these studies were sparse, with eclectic techniques, and outcome data ranged from pre- and posttreatment (Choe & Kluft, 1995; Ellason & Ross, 2004) to multiple months and years posttreatment. Brand and associates

observed that patients with DID who became integrated over the course of treatments reported greater reductions in symptoms of co-occurring diagnoses. However, the studies lacked control groups and random or systematic selection of patients. Other methodological limitations included high dropout rates and small sample sizes. Brand, McNary, and colleagues (2013) conducted a naturalistic observational 30-month follow-up study of an international sample of patients ($N = 226$) with DID or DDNOS diagnoses. Treatments delivered by community providers were eclectic. In the study, 119 of 226 patients provided outcome data at 30-month follow-up, and patients reported reductions in dissociation, PTSD symptoms, drug use, general distress, physical pain, and depressive symptoms over the course of treatment. Providers also reported improvements in patients' adaptive functioning (e.g., socializing; volunteering) and reductions in patients' self-injurious behavior over the course of treatment. Nevertheless, the authors cautioned that the lack of a control condition and selection bias (i.e., selection of patients especially well suited to treatment) limit the generalizability of these results.

Beyond case reports and the aforementioned large-scale treatment outcome studies (i.e., Brand, Classen, Lanius et al., 2009; Brand, McNary et al., 2013), absent from the literature are finer-grained studies of specific DID interventions, in isolation and in combination, with adequate sample sizes on which to base strong inferences, and systematic examination of potential mechanisms of treatment. Although we might be tempted to conclude from these large-scale outcome studies that psychotherapy yields reductions in symptoms and improvements in functioning, we cannot yet evaluate the efficacy or effectiveness of any particular intervention (e.g., cognitive restructuring; hypnotherapy) in comparison with another intervention or treatment as usual. In an editorial observing that published trials of treatment outcomes for dissociative disorders have been uncontrolled and methodologically lacking, Brand (2012) attributed this paucity of research to an absence of funding and to the high complexity of dissociative disorder patients (e.g., multiple co-occurring psychiatric diagnoses alongside DID diagnosis). She noted that the National Institute of Mental Health has yet to fund a treatment outcome study for DID or DDNOS.

13.4 Treatments for Dissociative Fugue and Dissociative Amnesia

Although epidemiological studies of dissociative disorder prevalence have shown that dissociative amnesia (DA) and dissociative fugue (DF) are the most and least commonly diagnosed dissociative disorders in the general population, respectively (Ross, 1991; Ross, Joshi, & Currie, 1990), publications examining treatments for DA and DF are few enough to warrant combined coverage. Prior to 2000, only one study of DF had appeared in the literature (Coons, 1999). Experiences of DA include blackouts or "time loss," perplexing changes in relationships with others, fragmentary recall of life history, fugue-like episodes, and trance-like amnesia episodes (i.e., "microamnesias") (Loewenstein, 1991; Steinberg, 1994b). Often co-occurring with histories of trauma (e.g., disaster; Cardena & Spiegel, 1993) and overwhelmingly stressful life events, amnesic and other dissociative experiences may function to defend the self against overwhelming traumatic memories and affect (e.g., Putnam, 1991; Schacter & Kihlstrom, 1989) or against suicidal ruminations (e.g., Gudjonsson & Haward, 1982). Nevertheless, these causal assumptions have hardly gone unquestioned (e.g., Lynn et al., 2014).

In light of the assumed relationship between dissociative experiences and trauma, Loewenstein (1996) contended that interventions for both DA and DF would do well to adhere to the triphasic model of treatment employed for trauma disorders in general. Psychodynamic conceptualizations have posited that the persistence and severity of amnesia parallel the intolerableness of traumatic memories and affect (e.g., Terr, 1991). Although clinical lore (e.g., Brown, 1919; Fisher, 1943; Gudjonsson & Haward, 1982; Kardiner & Spiegel, 1947; Steele & Colrain, 1990) holds that overzealous or ill-timed efforts to uncover traumatic material risk exacting significant psychological stress on the patient, researchers have not evaluated this claim. As in the treatment of DID, use of adjunctive hypnotherapeutic techniques purportedly facilitate recall, modulate symptom intensity, and otherwise improve outcomes in cases of DA and DF (Brown & Fromm, 1986). However, the role of hypnosis is difficult to isolate and has not been evaluated to date, as it is typically implemented in the context of complex, multicomponent interventions (Jasper, 2003). Other interventions (e.g., supportive psychotherapy and mindfulness; Jha & Sharma, 2015) show promise in helping patients with DF to acknowledge and accept traumatic memories, and some case studies describe successful CBT for DA (Cassel & Humphreys, 2016). Also, some authors have advocated the use of lorazepam, or amytal or other barbiturates to recover memories (e.g., Seo, Shin, Kim, & Kim, 2013), although (as noted in Section 13.3) the latter approach may create false memories (see Gudjonsson, Kopelman, & MacKeith, 1999; Piper, 1993).

Critics have suggested that studies have not ruled out alternative explanations for the presentation of DA and DF, such as neurological conditions (e.g., seizures, traumatic brain injury), malingering, and factitious disorders (Giesbrecht, Merckelbach, Van Oorsouw, & Simeon, 2010; Kihlstrom, 2005). Moreover, DA and DF often resolve spontaneously in a short time (e.g., within a week) or in response to simple suggestions for recall (Abeles & Schilder, 1935; Herman, 1938; Loewenstein, 1996), rendering attributions of treatment gains to psychotherapy problematic. In long-standing, chronic amnesia, researchers suggest that amnesia may diminish alongside treatment of co-occurring and reinforcing psychopathology (e.g., PTSD; Brende, 1985; Van der Kolk, 1986; for contraindications see Colrain & Steele, 1991). Nevertheless, no controlled studies of potential treatment mechanisms have been conducted.

13.5 Innovative Treatments for Dissociation

Consistent with perspectives that contend that dissociative symptoms may sometimes arise in the absence of trauma (e.g., Lilienfeld et al., 1999; Van der Kloet, Merckelbach, Giesbrecht, & Lynn, 2012), nontraumacentric therapies for dissociation have received attention in recent years. To claim that A is a causal factor contributing to B, several criteria have to be met (e.g., Hill, 1965). First, A must correlate with B: the stronger, the better. Second, this correlation must be consistently found by—to paraphrase epidemiologist Austin Bradford Hill (1965)—different researchers, in different places, circumstances, and times. Third, the A–B link should not be part of a diffuse, undifferentiated network, but rather should be specific (it is A with B, not A with B, C, D, etc.). Fourth, the alleged causal link between A and B should be coherent with extant knowledge, or at least it should not conflict with known facts. And, fifth and finally, experimental or quasi-experimental data should indicate that an increase in A is followed by an increase

in B, whereas a reduction of A is followed by a reduction of B. From a therapeutic perspective, the latter is of course particularly relevant.

13.5.1 Sleep-Related Interventions

Starting with the seminal work of Watson (2001) demonstrating that, in undergraduates, dissociative symptoms correlate with certain sleep experiences (e.g., nightmares), many researchers have replicated the sleep disturbance–dissociation link. In their meta-analysis, Van der Kloet, Merckelbach et al. (2012) reviewed 23 studies (with a total *N* of > 5,600) by different researchers using different measures of sleep problems and dissociative symptomatology. Almost all studies found significant correlations (*r*'s in the 0.30–0.55 range) between deviant sleep experiences and dissociative symptoms, such that the more individuals experienced sleep disturbances, the higher they scored on self-reported dissociative symptoms. More recent studies have extended this finding in three ways: (1) path analyses suggest that sleep abnormalities act as temporal and perhaps causal antecedents of dissociative symptoms (Van Heugten-Van der Kloet, Merckelbach, Giesbrecht, & Broers, 2014), (2) sleep problems are prominent in PTSD and DID patients (Van Heugten-Van der Kloet, Huntjens, Giesbrecht, T., & Merckelbach, 2014), and (3) increased dissociative pathology is evident in patients with insomnia (Van der Kloet et al., 2013). In his original study, Watson (2001) noted that the correlation of dissociativity with atypical sleep reports was much stronger than that between neuroticism and atypical sleep reports. Subsequent studies have also found supportive evidence for the specificity of the sleep disturbance–dissociation link. Thus, in their review, Van der Kloet, Merckelbach et al. (2012) concluded that "the connection between sleep and dissociation is specific in the sense that unusual sleep phenomena that are difficult to control, including nightmares and waking dreams, are related to dissociative symptoms, but lucid dreaming—dreams that are controllable—are only weakly related to dissociative symptoms" (p. 164).

The connection between sleep problems and dissociative symptoms is perfectly in line with studies concerning the critical role of sleep in emotion regulation. Thus, a broad literature documents that sleep, notably REM sleep, is crucial in processing emotional experiences. Summarizing studies in this domain, Walker and Van der Helm (2009) suggested that the "state of REM provides an optimal biological theater, within which, can be achieved a form of affective 'therapy'" (p. 731). Specifically, increased activity within limbic and paralimbic structures (e.g., the hippocampus and the amygdala) during REM sleep may first reactivate previously acquired affective experiences. In addition, REM sleep, which involves dominant theta oscillations within subcortical as well as cortical nodes, may offer large-scale network cooperation at night, allowing the integration and, as a consequence, greater understanding of recently experienced emotional events in the context of stored semantic memory.

There is now good experimental evidence that, when sleep problems are elicited by extensive sleep deprivation, dissociative symptoms increase. Giesbrecht, Smeets, Leppink, Jelicic, and Merckelbach (2015) deprived 25 healthy volunteers of sleep for 1 night and measured dissociative symptoms every 6 hours. The authors observed that dissociative symptoms increased steeply during the night, as sleep debt grew. This pattern was replicated by VanHeugten-Van der Kloet, Giesbrecht, and Merckelbach (2015), who sleep deprived nonsymptomatic volunteers and observed that their sleep

disruptions fueled distress, degraded memory, and attentional control while dissociative symptoms increased in intensity.

There is also evidence for the converse pattern: Normalizing the sleep–wake cycle in patients is followed by a reduction in dissociative symptoms. Van der Kloet, Giesbrecht, Merckelbach, Lynn, and de Zutter (2012) followed a group of inpatients ($N = 195$) with mood and anxiety disorders and/or substance abuse problems for 6–8 weeks. The therapeutic environment included CBT but also adhered to a strict sleep hygiene regime. Patients were awakened in the morning, had no access to alcohol, were not allowed to drink caffeine or engage in fitness activities in the evening, and so forth. Patients completed measures of sleep problems, general psychopathology, and dissociative symptoms on their first days in the clinic and 1 day before discharge. Over the course of the treatment period, sleep patterns normalized, and this was accompanied by an overall reduction in psychopathological symptoms. The beneficial effect of sleep normalization on dissociative symptoms was particularly evident, and that decline in dissociative symptoms was more pronounced than the decline in global psychopathology. This example suggests that treatment of dissociative symptoms might benefit from tracking sleep problems in patients and addressing them using simple sleep hygiene interventions.

Poerio, Kellett, and Totterdell (2016) employed experience sampling to study the dynamics of sleep, daydreaming, and dissociative symptoms in a patient with DP/DR over 40 consecutive days. Poor self-reported sleep quality was followed by an increase in dissociative symptoms. Daydreaming played a role in this symptomatological cascade, providing further evidence for the idea that a disturbed sleep–wake cycle undermines cognitive control such that dream-like mentation—in the form of daydreaming—intrudes into consciousness and fuels dissociativity. The authors commented on the therapeutic implication of this formulation: "Sleep hygiene, and daydream content interventions could occur in the initial phase of the treatment, so that dissociation is reduced and the patient is stabilized" (Poerio et al., 2016, p. 10). Lynn et al. (2013) presented a case study of DID in which they alleviated virtually all major dissociative symptoms using a multicomponent treatment that included behavioral, cognitive–behavioral, mindfulness, and behavioral activation interventions, as well as strategies that specifically targeted sleep-related problems. Conversations with alters and other suggestive techniques were eschewed, as the interventions focused on emotion regulation and contending skillfully with problems in everyday life.

13.5.2 Emotion Regulation Interventions

Interventions that target emotion regulation in patients with dissociative symptomatology may serve as a valuable therapeutic add-on. An important consideration in this respect is that both dissociative symptoms and sleep disturbances overlap with a trait known as alexithymia, which can best be conceptualized as an impairment in the ability to accurately evaluate internal states and experiences (Bauermann, Parker, & Tayler, 2008; Merckelbach, Boskovic, Pesy, Dalsklev, & Lynn, 2017). Such impairment may contribute to a highly volatile symptom presentation, as lack of evaluation of internal states may hamper regulation of emotions (Brady, Bujarski, Feldner, & Pyne, 2017). Alexithymia may be reduced by mentalization-based therapies or, more generally, interventions that help patients to recognize and verbalize their emotions

(Ogrodniczuk, Sochting, Piper, & Joyce, 2012). There is literature on the beneficial effects of mentalization-based therapies in borderline personality disorder and preliminary indications that this type of intervention may be promising in borderline personality disorder patients with dissociative symptoms (Korzekwa, Dell, & Pain, 2009). Clearly, from a therapeutic point of view, the links among sleep disturbances, alexithymia, and dissocative pathology are worth exploring in future research. Furthermore, systematic studies are required to articulate more precise therapeutic interventions.

13.6 Conclusions

The treatment of dissociative disorders has received scant attention, arguably less than any other major diagnostic class in the DSM-5. RCTs using attention-placebo or even waitlist controls have been glaringly absent, and case studies have been largely unsystematic, typically failing to meet scientific standards for single-case-study designs (Chambless & Hollon, 1998). Accordingly, not a single treatment for DID rises to the level of well supported or evidence based (Category I; David & Montgomery, 2011). At the best, a few treatments may rise to the level of preliminary effectiveness with no systematic investigation of mechanisms of change (Category IV; David & Montgomery, 2011).

Specifically, although some findings appear to be promising, such as CBT for DP/DR (Hunter, 2013), CBT plus sleep hygiene treatments for dissociative symptoms (Van der Kloet, Giesbrecht et al., 2012), and eclectic treatments for DID (Brand, Classen, Lanius et al., 2009), they must be regarded as tentative, as RCTs have not been conducted, replications have not been attempted, and numerous factors discussed previously, including regression to the mean, natural fluctuations in symptoms over time, demand characteristics, nonspecific factors, and other alternative explanations for apparent treatment success (see Lilienfeld et al., 2014), have not been ruled out. Indeed, across dissociative disorders, evidence for mechanisms of change in psychotherapy comes almost entirely from case reports, and hypothesized mechanisms are typically not clearly specified; when they are specified, they tend to be heterogeneous in nature and idiosyncratic, even within a specific diagnosis, and not evaluated in terms of clinical or research outcomes. Although these hypothesized mechanisms may inform interventions and theory-building in controlled studies, each mechanism we have reviewed remains conjectural. Moreover, many of the treatments are multifaceted, and the active/effective components unique to the intervention, to the extent they exist, have not been identified or isolated. Nor have researchers compared the efficacy of treatments with that of alternative treatments, and it is also unclear whether interventions perform better than no treatment (e.g., waitlist). Moreover, numerous legitimate concerns have been raised about possible iatrogenic effects of treatments for dissociation, and these potential effects should be explored systematically in future research, as some therapies that focus on memory recovery and that use suggestive techniques probably qualify for Category IX (i.e., bad-theory- and bad-intervention-driven psychotherapies) in David and Montgomery's (2011) framework.

In conclusion, no interventions have met criteria (David & Montgomery, 2011) to be considered evidence based, and potential mechanisms that undergird successful treatments have not been evaluated systematically. This state of affairs is unfortunate, as

many individuals suffer from dissociative symptoms and would benefit from empirically supported interventions. Clearly, the collection of more and better data on the treatment of dissociative disorders needs to be accorded higher priority in psychotherapy outcome research.

References

Abeles, M., & Schilder, P. (1935). Psychogenic loss of personal identity. *Archives of Neurology & Psychiatry*, *34*, 587–604. doi:10.1001/archneurpsyc.1935.02250210108008

Ackner, B. (1954). Depersonalization. *British Journal of Psychiatry*, *100*, 854–872. doi:10.1192/bjp.100.421.854

Allison, R. B. (1974). A new treatment approach for multiple personalities. *American Journal of Clinical Hypnosis*, *17*, 15–32. doi:10.1080/00029157.1974.10403703

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd rev. ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed. TR). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.

American Psychological Association Presidential Task Force on Evidence-Based Practice. (2006). Evidence-based practice in psychology. *American Psychologist*, *61*, 271–285.

Ballard, C. G., Mohan, R. N., & Handy, S. (1992). Chronic depersonalization neurosis au Shorvon: A successful intervention. *British Journal of Psychiatry*, *160*, 123–125. doi:10.1192/bjp.160.1.123

Bauermann, T. M., Parker, J. D. A., & Taylor, G. J. (2008). Sleep problems and sleep hygiene in young adults with alexithymia. *Personality and Individual Differences*, *45*, 318–322. doi:10.1016/j.paid.2008.04.019

Benjamin, L. R., & Benjamin, R. (1992). An overview of family treatment in dissociative disorders. *Dissociation*, *5*, 236–241.

Blue, F. R. (1979). Use of directive therapy in the treatment of depersonalization. *Psychological Reports*, *45*, 904–906. doi:10.2466/pr0.1979.45.3.904

Bowman, E. S. (1993). Clinical and spiritual effects of exorcism in fifteen patients with multiple personality disorder. *Dissociation*, *6*(4), 222–238.

Boysen, G., & VanBergen, A. (2013). A review of published research on adult dissociative identity disorder. *Journal of Nervous and Mental Disease*, *201*, 5–11. doi:10.1097/nmd.0b013e31827aaf81

Brady, R. E., Bujarski, S. J., Feldner, M. T., & Pyne, J. M. (2017). Examining effects of alexithymia on the relation between posttraumatic stress disorder and over-reporting. *Psychological Trauma*, *9*, 80–87. doi:10.1037/tra0000155

Brand, B. L. (2012). What we know and what we need to learn about the treatment of dissociative disorders. *Journal of Trauma and Dissociation*, *13*(2), 387–396. doi:10.1080/15299732.2012.67255

Brand, B. L. (2016). The necessity of clinical training in trauma and dissociation. *Journal of Depression and Anxiety*, *5*, 251. doi:10.4172/2167-1044.1000251

Brand, B. L., Classen, C., Lanius, R., Loewenstein, R., McNary, S., Pain, C., & Putnam, F. (2009). A naturalistic study of dissociative identity disorder and dissociative disorder not otherwise specified patients treated by community clinicians. *Psychological Trauma*, *1*, 153–171. doi:10.1037/a0016210

Brand, B. L., Classen, C. C., McNary, S. W., & Zaveri, P. (2009). A review of dissociative disorder treatment studies. *Journal of Nervous and Mental Disease*, *197*, 646–654.

Brand, B. L., Lanius, R., Vermetten, E., Loewenstein, R. J., & Spiegel, D. (2012). Where are we going? An update on assessment, treatment, and neurobiological research in dissociative disorders as move toward the DSM-5. *Journal of Trauma & Dissociation*, *13*, 9–31. doi:10.1080/15299732.2011.620687

Brand, B. L., & Loewenstein, R. J. (2014). Does phasic trauma treatment make patients with dissociative identity disorder treatment more dissociative? *Journal of Trauma & Dissociation*, *15*, 52–65. doi:10.1080/15299732.2013.828150

Brand, B. L., Loewenstein, R. J., & Spiegel, D. (2013). Patients with DID are found and researched more widely than Boysen and VanBergen recognized. *Journal of Nervous and Mental Disease*, *201*, 440. doi:10.1097/nmd.0b013e31828e10d1

Brand, B. L., McNary, S. W., Myrick, A. C., Classen, C., Lanius, R., Loewenstein, R. J., … Putnam, F. W. (2013). A longitudinal naturalistic study of patients with dissociative disorders treated by community clinicians. *Psychological Trauma: Theory, Research, Practice, and Policy*, *5*(4), 301–308. doi:10.1037/a0027654

Brand, B. L., Myrick, A. C., Loewenstein, R. J., Classen, C. C., Lanius, R., McNary, S. W., … Putnam, F. W. (2012). A survey of practices and recommended treatment interventions among expert therapist treating patients with dissociative identity disorder and dissociative disorder not otherwise specified. *Psychological Trauma*, *4*, 490–500. doi:10.1037/a0026487

Brende, J. O. (1985). The use of hypnosis in post-traumatic conditions. In W. E. Kelly (Ed.), *Post-traumatic stress disorder and the war veteran patient* (pp. 193–210). New York, NY: Brunner/Mazel.

Brown, D. P., & Fromm, E. (1986). *Hypnotherapy and hypnoanalysis*. Hillsdale, NJ: Lawrence Erlbaum Associates.

Brown, W. (1919). Hypnosis, suggestion, and dissociation. *BMJ*, *191*, 734–736. doi:10.1136/bmj.1.3050.734

Bull, D. L., Ellason, J. W., & Ross, C. A. (1998). Exorcism revisited: Positive outcomes with dissociative identity disorder. *Journal of Psychology & Theology*, *26*, 188–196.

Burlingame, G. M., MacKenzie, K. R., & Strauss, B. (2004). Small group treatment: Evidence for effectiveness and mechanisms of change. In M. L. Lambert (Ed.), *Bergin and Garfield's handbook of psychotherapy and behavior change* (5th ed., pp. 647–696). Hoboken, NJ: John Wiley & Sons.

Burton, N., & Lane, R. C. (2001). The relational treatment of dissociative identity disorder. *Clinical Psychology Review*, *21*, 301–320. doi:10.1016/s0272-7358(99)00049-5

Caddy, G. R. (1985). Cognitive behavior therapy in the treatment of multiple personality disorder. *Behavior Modification*, *9*, 267–292. doi:10.1177/01454455850093001

Cagiada, S., Camaido, L., & Pennan, A. (1997). Successful integrated hypnotic and psychopharmacological treatment of war-related post-traumatic psychological and aomatoform dissociative disorder of two years duration (psychogenic coma). *Dissociation*, *10*, 182–189.

Cardena, E., & Spiegel, D. (1993). Dissociative reactions to the San Francisco Bay Area earthquake of 1989. *American Journal of Psychiatry*, *150*, 474–478. doi:10.1176/ajp.150.3.474

Cassel, A., & Humphreys, K. (2016). Psychological therapy for psychogenic amnesia: Successful treatment in a single case study. *Neuropsychological Rehabilitation*, *26*, 374–391. doi:10.1080/09602011.2015.1033431

Cattell, J. P., & Cattell, J. S. (1974). Depersonalization: Psychological and social perspectives. In S. Arieti & E. B. Brody (Eds.), *American handbook of psychiatry* (Vol. 3, pp. 766–799). New York, NY: Basic Books.

Chambless, D. L., & Hollon, S. D. (1998). Defining empirically supported therapies. *Journal of Consulting and Clinical Psychology*, *66*, 7–18. doi:10.1037//0022-006x.66.1.7

Chambless, D. L., & Ollendick, T. H. (2001). Empirically supported psychological interventions: Controversies and evidence. *Annual Review of Psychology*, *52*, 685–716. doi:10.1146/annurev.psych.52.1.685

Chiappa, F. (1994). Effective management of family and individual interventions in the treatment of dissociative disorders. *Dissociation*, *7*, 185–190.

Choe, B. M., & Kluft, R. P. (1995). The use of the DES in studying treatment outcome with dissociative identity disorder: A pilot study. *Dissociation*, *8*, 160–164.

Clark, D. M. (1986). A cognitive approach to panic. *Behaviour Research and Therapy*, *24*, 461–470. doi:10.1016/0005-7967(86)90011-2

Colrain, J., & Steele, K. (1991). Treatment protocols for spontaneous abreactive memory work. In *Proceedings of the eighth international conference on multiple personality disorder and dissociation* (pp. 6–8). Chicago, IL: University of Chicago Press.

Coons, P. M. (1999). Psychogenic or dissociative fugue: A clinical investigation of five cases. *Psychological Reports*, *84*, 881–886. doi:10.2466/pr0.1999.84.3.881

Coons, P. M., & Bowman, E. S. (2001). Ten-year follow-up study of patients with dissociative identity disorder. *Journal of Trauma & Dissociation*, *2*, 73–89. doi:10.1300/j229v02n01_09

Coons, P. M., & Sterne, A. L. (1986). Initial and follow-up psychological testing on a group of patients with multiple personality disorder. *Psychological Reports*, *58*, 43–49. doi:10.2466/pr0.1986.58.1.43

Courtois, C. A., & Ford, J. D. (2009). *Treating complex traumatic stress disorders: An evidence-based guide*. New York, NY: Guilford Press.

Cronin, E., Brand, B. L., & Mattanah, J. F. (2014). The impact of the therapeutic alliance on treatment outcome in patients with dissociative disorders. *European Journal of Psychotraumatology*, *5*, 301–320.

Dalenberg, C. J., Brand, B. L., Gleaves, D. H., Dorahy, M. J., Loewenstein, R. J., Cardeña, E., … Spiegel, D. (2012). Evaluation of the evidence for the trauma and fantasy models of dissociation. *Psychological Bulletin*, *138*, 550–588. doi:10.1037/a0027447

David, D., & Montgomery, G. H. (2011). The scientific status of psychotherapies: A new evaluative framework for evidence-based psychosocial interventions. *Clinical Psychology: Science and Practice*, *18*, 89–99. doi:10.1111/j.1468-2850.2011.01239.x

Despine, C. H. A. (1840). *De l'emploi du magnetisme animal et des eaux minerales dans le traitement des maladies nerveuses, suivi d'une observation tres curieuse de guerison de nevropathie* [A study of the uses of animal magnetism in the treatment of disorders of the nervous system followed by a case of a highly unusual cure of neuropathy]. Paris, France: Ballière.

Dorahy, M. J., Brand, B. L., Vedat, Ş., Krüger, C., Stavropoulos, P., Martinez-Taboas, A., … Middleton, W. (2014). Dissociative identity disorder: An empirical overview. *Australian & New Zealand Journal of Psychiatry*, *48*, 402–417.

Ellason, J. W., & Ross, C. A. (1996). Millon Clinical Multiaxial Inventory-II follow-up of patients with dissociative identity disorder. *Psychological Reports*, *78*, 707–716. doi:10.2466/pr0.1996.78.3.707

Ellason, J. W., & Ross, C. A. (1997). Two-year follow-up of inpatients with dissociative identity disorder. *American Journal of Psychiatry*, *154*, 832–839. doi:10.1176/ajp.154.6.832

Ellason, J. W., & Ross, C. A. (2004). SCL-90-R norms for dissociative identity disorder. *Journal of Trauma & Dissociation*, *5*, 85–91. doi:10.1300/j229v05n03_06

Ellerman, C. P. (1998). The phenomenological treatment of dissociative identity disorder. *Psychotherapy*, *28*, 69–79.

Fetkewicz, J., Sharma, V., & Merskey, H. (2000). A note on suicidal deterioration with recovered memory treatment. *Journal of Affective Disorders*, *58*, 155–159. doi:10.1016/s0165-0327(98)00193-1

Fine, C. G. (2012). Cognitive behavioral hypnotherapy for dissociative disorders. *American Journal of Clinical Hypnosis*, *54*, 331–352. doi:10.1080/00029157.2012.656856

Fine, C. G., & Madden, N. E. (2000). Group psychotherapy in the treatment of dissociative identity disorder and allied dissociative disorders. In R. H. Klein & V. L. Schermer (Eds.), *Group psychotherapy for psychological trauma* (pp. 298–325). New York, NY: Guilford Press.

Fisher, C. (1943). Hypnosis in treatment of neurosis due to war and other causes. *War Medicine*, *4*, 565–576.

Frances, A., Sacks, M., & Aronoff, M. S. (1977). Depersonalization: A self-relations perspective. *International Journal of Psychoanalysis*, *58*, 325–331.

Frischholz, E. J., Lipman, L. S., Braun, B. G., & Sachs, R. G. (1992). Psychopathology, hypnotizability, and dissociation. *American Journal of Psychiatry*, *149*, 1521–1525. doi:10.1176/ajp.149.11.1521

Gantt, L., & Tinnin, L. W. (2007). Intensive trauma therapy of PTSD and dissociation: An outcome study. *The Arts in Psychotherapy*, *34*, 69–80. doi:10.1016/j.aip.2006.09.007

Gee, T., Allen, K., & Powell, R. A. (2003). Questioning premorbid dissociative symptomatology in dissociative identity disorder: Comment on Gleaves, Hernandez and Warner (1999). *Professional Psychology: Research and Practice*, *34*, 114–116. doi: 10.1037//0735-7028.34.1.114

Gentile, J. P., Snyder, M., & Gillig, P. M. (2014). Stress and trauma: Psychotherapy and pharmacotherapy for depersonalization/derealization disorder. *Innovations in Clinical Neuroscience*, *11*, 37–41.

Giesbrecht, T., Merckelbach, H., Van Oorsouw, K., & Simeon, D. (2010). Skin conductance and memory fragmentation after exposure to an emotional film clip in depersonalization disorder. *Psychiatry Research*, *177*, 342–349. doi:10.1016/j.psychres.2010.03.010

Giesbrecht, T., Smeets, T., Leppink, J., Jelicic, M., & Merckelbach, H. (2007). Acute dissociation after 1 night of sleep loss. *Journal of Abnormal Psychology*, *116*, 599–606. doi:10.1037/0021-843x.116.3.599

Gold, S. N., Elhai, J. D., Rea, B. D., Weiss, D., Masino, T., Morris, S. L., & McIninch, J. (2001). Contextual treatment of dissociative identity disorder: Three case studies. *Journal of Trauma & Dissociation*, *2*, 5–36. doi:10.1300/j130v02n04_02

Groenendijk, I., & Van der Hart, O. (1995). Treatment of DID and DDNOS patients in a regional institute for ambulatory mental health care in the Netherlands: A survey. *Dissociation: Progress in the Dissociative Disorders*, *8*, 73–83.

Gudjonsson, G. H., & Haward, L. R. C. (1982). Hysterical amnesia as an alternative to suicide. *Medicine, Science and the Law*, *22*, 68–72. doi:10.1177/002580248202200113

Gudjonsson, G. H., Kopelman, M. D., & MacKeith, J. A. (1999). Unreliable admissions to homicide: A case of misdiagnosis of amnesia and misuse of abreaction technique. *British Journal of Psychiatry*, *174*, 455–459. doi:10.1192/bjp.174.5.455

Haley, J. (1963). *Strategies of psychotherapy*. New York, NY: Grune & Stratton.

Herman, M. (1938). The use of intravenous sodium amytal in psychogenic amnesic states. *Psychiatric Quarterly*, *12*, 738–742. doi:10.1007/bf01562835

Hill, A. B. (1965). The environment and disease: Association or causation? *Proceedings of the Royal Society of Medicine*, *58*, 295–300.

Hoffman, L. (1981). *Foundations of family therapy: A conceptual framework for systems change*. New York, NY: Basic Books.

Hove, I., Langfeldt, T. S., Boe, T., Haslerud, J., & Stoerseth, F. (1997). Working with dissociative identity disorder in Stavanger, Norway. *Dissociation*, *10*, 166–173.

Hunsley, J., Elliott, K., & Therrien, Z. (2014). The efficacy and effectiveness of psychological treatments for mood, anxiety, and related disorders. *Canadian Psychology/Psychologie Canadienne*, *55*(3), 161–176. doi:10.1037/a0036933

Hunter, E. C. M. (2013). Understanding and treating depersonalization disorder. In F. Kennedy, H. Kennerley, & D. Pearson (Eds.), *Cognitive behavioural approaches to the understanding and treatment of dissociation*. New York, NY: Routledge.

Hunter, E. C. M., Baker, D., Phillips, M. L., Sierra, M., & David, A. S. (2005). Cognitive-behaviour therapy for depersonalization disorder: An open study. *Behaviour Research and Therapy*, *43*, 1121–1130. doi:10.1016/j.brat.2004.08.003

Hunter, E. C. M., Phillips, M. L., Chalder, T., Sierra, M., & David, A. S. (2003). Depersonalisation disorder: A cognitive-behavioural conceptualisation. *Behaviour Research and Therapy*, *41*, 1451–1467. doi:10.1016/s0005-7967(03)00066-4

Hunter, E. C. M., Sierra, M., & David, A. S. (2004). The epidemiology of depersonalization and derealization. *Social Psychiatry and Psychiatric Epidemiology*, *39*, 9–18.

International Society for the Study of Trauma and Dissociation. (2011). Guidelines for treating dissociative identity disorder in adults, third revision. *Journal of Trauma & Dissociation*, *12*, 115–187. doi:10.1080/15299732.2011.537247

Janet, P. (1889/1973). *L'automatisme psychologique* [Psychological automatism]. Paris: Centre National de las Recherche Scientifique.

Jasper, F. J. (2003). Working with dissociative fugue in a general psychotherapy practice: A cautionary tale. *American Journal of Clinical Hypnosis*, *45*, 311–322. doi:10.1080/00029157.2003.10403544

Jha, M., & Sharma, V. (2015). Dissociative fugue disorder: Identification and psychological intervention. *Psychological Studies*, *60*, 17–24. doi:10.1007/s12646-014-0287-1

Johnson, J. G., Cohen, P., Kasen, S., & Brook, J. S. (2006). Dissociative disorders among adults in the community, impaired functioning, and Axis I and II comorbidity. *Journal of Psychiatric Research*, *40*, 131–140. doi:10.1016/j.jpsychires.2005.03.003

Kardiner, A., & Spiegel, H. (1947). *War, stress, and neurotic illness*. New York, NY: Hoeber.

Kazdin, A. E. (2008). Evidence-based treatment and practice: New opportunities to bridge clinical research and practice, enhance the knowledge base, and improve patient care. *American Psychologist*, *63*, 146–159. doi:10.1037/0003-066x.63.3.146

Kellett, S. (2005). The treatment of dissociative identity disorder with cognitive analytic therapy: Experimental evidence of sudden gains. *Journal of Trauma & Dissociation*, *6*, 55–81. doi:10.1300/j229v06n03_03

Kendall, P. C., Flannery-Schroeder, E. C., & Ford, J. D. (1999). Therapy outcome research methods. In P. C. Kendall & J. N. Butcher (Eds.), *Handbook of research methods in clinical psychology* (2nd ed., pp. 330–363). Hoboken, NJ: John Wiley & Sons.

Kihlstrom, J. F. (2005). Dissociative disorders. *Annual Review of Clinical Psychology*, *1*, 227–253. doi:10.1146/annurev.clinpsy.1.102803.143925

Kleindienst, N., Limberger, M. F., Ebner-Priemer, U. W., Keibel-Mauchnik, J., Dyer, A., Berger, M., … Bohus, M. (2011). Dissociation predicts poor response to dialectical behavioral therapy in female patients with borderline personality disorder. *Personality Disorders*, *25*, 432–447. doi:10.1521/pedi.2011.25.4.432

Kluft, R. P. (1984). Treatment of multiple personality disorder: A study of 33 cases. *Psychiatric Clinics of North America*, *7*, 9–29.

Kluft, R. P. (1993). The initial stages of psychotherapy in the treatment of multiple personality disorder. *Dissociation*, *6*, 145–161.

Kluft, R. P. (2012). Hypnosis in the treatment of dissociative identity disorder and allied states: An overview and case study. *South African Journal of Psychology*, *42*, 146–155. doi:10.1177/008124631204200202

Kohlenberg, R. J. (1973). Behavioristic approach to multiple personality: A case study. *Behavior Therapy*, *4*, 137–140. doi:10.1016/s0005-7894(73)80086-3

Korzekwa, M. I., Dell, P. F., & Pain, C. (2009). Dissociation and borderline personality disorder: An update for clinicians. *Current Psychiatry Reports*, *11*, 82–88. doi:10.1007/s11920-009-0013-1

Lanius, R. A., Brand, B., Vermetten, E., Frewen, P. A., & Spiegel, D. (2012). The dissociative subtype of posttraumatic stress disorder: Rationale, clinical and neurobiological evidence, and implications. *Depression and Anxiety*, *29*, 701–708. doi:10.1002/da.21889

Lilienfeld, S. O. (2007). Psychological treatments that cause harm. *Perspectives on Psychological Science*, *2*, 53–70. doi:10.1111/j.1745-6916.2007.00029.x

Lilienfeld, S. O., Kirsch, I., Sarbin, T. R., Lynn, S. J., Chaves, J. F., Ganaway, G. K., & Powell, R. A. (1999). Dissociative identity disorder and the sociocognitive model: Recalling the lessons of the past. *Psychological Bulletin*, *125*, 507–523. doi:10.1037//0033-2909.125.5.507

Lilienfeld, S. O., Ritschel, L. A., Lynn, S. J., Cautin, R. L., & Latzman, R. D. (2013). Why many clinical psychologists are resistant to evidence-based practice: Root causes and constructive remedies. *Clinical Psychology Review*, *33*, 883–900. doi:10.1016/j.cpr.2012.09.008

Lilienfeld, S. O., Ritschel, L. A., Lynn, S. J., Cautin, R. L., & Latzman, R. D. (2014). Why ineffective therapies work: A taxonomy of causes of spurious therapeutic effectiveness. *Perspectives on Psychological Science*, *9*, 355–387. doi:10.1177/1745691614535216

Liotti, G. (1999). Disorganization of attachment as a model for understanding dissociative psychopathology. In J. Solomon & C. George (Eds.), *Attachment disorganization* (pp. 291–317). New York, NY: Guilford Press.

Loewenstein, R. J. (1991). Psychogenic amnesia and psychogenic fugue: A comprehensive review. In A. Tasman & S. Goldfinger (Eds.), *American Psychiatric Press annual review of psychiatry* (Vol. 10, pp. 189–222). Washington, DC: American Psychiatric Press.

Loewenstein, R. J. (1994). Diagnosis, epidemiology, clinical course, treatment, and cost effectiveness of treatment for dissociative disorders and MPD: Report submitted to the Clinton Administration Task Force on Health Care Financing Reform. *Dissociation*, *7*, 3–11.

Loewenstein, R. J. (1996). Dissociative amnesia and dissociative fugue. In L. K. Michelson & W. J. Ray (Eds.), *Handbook of dissociation: Theoretical, empirical, and clinical perspectives* (pp. 307–336). New York, NY: Springer.

Loewenstein, R. J., & Putnam, F. W. (2004). The dissociative disorders. In B. J. Sadock & V. A. Sadock (Eds.), *Kaplan and Sadock's comprehensive textbook of psychiatry* (8th ed., pp. 1844–1901). Philadelphia, PA: Lippincott Williams and Wilkins.

Lynn, S. J., Condon, L., & Colletti, G. (2013). The treatment of dissociative identity disorder: Questions and considerations. In W. O'Donohue & S. O. Lilienfeld (Eds.), *Case studies in clinical psychological science: Bridging the gap from science to practice* (pp. 329–351). Oxford, UK: Oxford University Press.

Lynn, S. J., Lilienfeld, S. O., Merckelbach, H., Giesbrecht, T., McNally, R. J., Loftus, E. F., … Malaktaris, A. (2014). The trauma model of dissociation: Inconvenient truths and stubborn fictions. Comment on Dalenberg et al. (2012). *Psychological Bulletin*, *3*, 896–910. doi:10.1037/a0035570

Lynn, S. J., Lilienfeld, S. O., Merckelbach, H., Giesbrecht, T., & Van der Kloet, D. (2012). Dissociation and dissociative disorders: Challenging conventional wisdom. *Current Directions in Psychological Science*, *21*, 48–53. doi:10.1177/0963721411429457

Lynn, S. J., Lilienfeld, S. O., Merckelbach, H., Maxwell, R., Baltman, J., & Giesbrecht, T. (2016). Dissociative disorders. In J. E. Maddux & B. A. Winstead (Eds.), *Psychopathology: Foundations for a contemporary understanding* (pp. 298–318). London, UK: Routledge.

Lynn, S. J., Rhue, J. W., & Kirsch, I. (Eds.). (2010). *Handbook of clinical hypnosis* (2nd ed.). Washington, DC: American Psychological Association.

Maaranen, P., Tanskanen, A., Honkalampi, K., Haatainen, K., Hintikka, J., & Viinamäki, H. (2005). Factors associated with pathological dissociation in the general population. *Australian & New Zealand Journal of Psychiatry*, *39*, 387–394. doi:10.1080/j.1440-1614.2005.01586.x

Martinez-Taboas, A., & Rodriguez-Cay, J. R. (1997). Case study of a Puerto Rican woman with dissociative identity disorder. *Dissociation*, *10*, 141–147.

McHugh, R. K., Murray, H. W., & Barlow, D. H. (2009). Balancing fidelity and adaption in the dissemination of empirically-supported treatments: The promise of transdiagnostic interventions. *Behaviour Research and Therapy*, *47*, 946–953. doi:10.1016/j.brat.2009.07.005

Merckelbach, H., Boskovic, I., Pesy, D., Dalsklev, M., & Lynn, S. J. (2017). Symptom overreporting and dissociative experiences: A qualitative review. *Consciousness and Cognition*, *49*, 132–144. doi:10.1016/j.concog.2017.01.007

Michelson, L., June, K., Vives, A., Testa, S., & Marchione, N. (1998). The role of trauma and dissociation in cognitive-behavioral psychotherapy outcome and maintenance for panic disorder with agoraphobia. *Behaviour Research and Therapy*, *36*, 1011–1050. doi:10.1016/s0005-7967(98)00073-4

Norcross, J. C., & Wampold, B. E. (2011). Evidence-based therapy relationships: Research conclusions and clinical practices. *Psychotherapy*, *48*, 98–102. doi:10.1037/a0022161

Ogrodniczuk, J. S., Sochting, I., Piper, W. E., & Joyce, A. S. (2012). A naturalistic study of alexithymia among psychiatric outpatients treated in an integrated group therapy program. *Psychology and Psychotherapy*, *85*, 278–291. doi:10.1111/j.2044-8341.2011.02032.x

Paris, J. (2012). The rise and fall of dissociative identity disorder. *Journal of Nervous and Mental Disease*, *200*, 1076–1079. doi:10.1097/nmd.0b013e318275d285

Paul, G. L. (1967). Strategy outcome research in psychotherapy. *Journal of Consulting Psychology*, *31*, 109–118. doi:10.1037/h0024436

Phillips, M., & Frederick, C. (1995). *Healing the divided self: Clinical and Ericksonian hypnotherapy for post-traumatic and dissociative conditions*. New York, NY: Norton.

Piper, A. (1993). "Truth serum" and "recovered memories" of sexual abuse: A review of the evidence. *Journal of Psychiatry & Law*, *21*, 447–471.

Poerio, G. L., Kellet, S., & Totterdell, P. (2016). Tracking potentiating states of dissociation: An intensive clinical case study of sleep, daydreaming, mood, and depersonalization/derealization. *Frontiers in Psychology*, *7*, 1231. doi:10.3389/fpsyg.2016.01231

Prochaska, J., & Norcross, J. (2013). *Systems of psychotherapy: A transtheoretical analysis*. Stamford, CT: Cengage Learning.

Putnam, F. W. (1991). Dissociative phenomena. In A. Tasman & S. Goldfinger (Eds.), *American Psychiatric Press annual review of psychiatry* (Vol. 10, pp. 145–160). Washington, DC: American Psychiatric Press.

Resick, P. A., & Schnicke, M. K. (1992). Cognitive processing therapy for sexual assault victims. *Journal of Consulting and Clinical Psychology*, *60*, 748–756. doi:10.1037//0022-006x.60.5.748

Riggs, S. R., & Bright, M. A. (1997). Dissociative identity disorder: A feminist approach to inpatient treatment using Jean Baker Miller's relational model. *Archives of Psychiatric Nursing*, *11*, 218–224. doi:10.1016/s0883-9417(97)80029-5

Ross, C. A. (1991). Epidemiology of multiple personality disorder and dissociation. *Psychiatric Clinics of North America*, *14*, 503–517.

Ross, C. A. (1997). Cognitive therapy of dissociative identity disorder. In P. S. Appelbaum, L. A. Uyehara, & M. R. Elin (Eds.), *Trauma and memories: Clinical and legal controversies* (pp. 361–377). Oxford, UK: Oxford University Press.

Ross, C. A. (2008). Group therapy for dissociative disorders and addiction. *Journal of Groups in Addiction & Recovery*, *3*, 323–346. doi:10.1080/15560350802424795

Ross, C. A., Anderson, G., Heber, S., & Norton, G. R. (1990). Dissociation and abuse among multiple-personality disorder patients, prostitutes, and exotic dancers. *Psychiatric Services*, *41*, 328–330. doi:10.1176/ps.41.3.328

Ross, C. A., & Gahan, P. (1988). Cognitive analysis of multiple personality disorder. *American Journal of Psychiatry*, *42*, 229–239.

Ross, C. A., & Haley, C. (2004). Acute stabilization and three-month follow-up in a trauma program. *Journal of Trauma & Dissociation*, *5*, 103–112. doi:10.1300/j229v05n01_06

Ross, C. A., Joshi, S., & Currie, R. (1990). Dissociative experiences in the general population. *American Journal of Psychiatry*, *147*, 1547–1552. doi:10.1176/ajp.147.11.1547

Ross, C. A., Kronson, J., Koensgen, S., Barkman, K., Clark, P., & Rockman, G. (1992). Dissociative comorbidity in 100 chemically dependent patients. *Psychiatric Services*, *43*, 840–842. doi:10.1176/ps.43.8.840

Rufer, M., Fricke, S., Held, D., Cremer, J., & Hand, I. (2006). Dissociation and symptom dimensions of obsessive-compulsive disorder. *European Archives of Psychiatry and Clinical Neuroscience*, *256*, 146–150.

Ryle, A. (1997). The structure and development of borderline personality disorder: A proposed model. *British Journal of Psychiatry*, *170*, 82–87. doi:10.1192/bjp.170.1.82

Sar, V. (2011). Epidemiology of dissociative disorders: An overview. *Epidemiology Research International*, *2011*, 1–8. doi:10.1155/2011/404538

Sar, V., Akyüz, G., Kugu, N., & Ertem-Vehid, H. (2006). Axis I dissociative disorder comorbidity in borderline personality disorder and reports of childhood trauma. *Journal of Clinical Psychology*, *67*, 1583–1590. doi:10.4088/jcp.v67n1014

Şar, V., Akyüz, G., Kundakçı, T., Kızıltan, E., & Doçan, O. (2004). Childhood trauma, dissociation, and psychiatric comorbidity in patients with conversion disorder. *American Journal of Psychiatry*, *161*, 2271–2276. doi:10.1176/appi.ajp.161.12.2271

Sar, V., Kundakçı, T., Kızıltan, E., Yargic, I. L., Tutkun, H., Bakim, B., … Özdemir, Ö. (2003). The Axis-I dissociative disorder comorbidity of borderline personality disorder among psychiatric outpatients. *Journal of Trauma & Dissociation*, *4*, 119–136. doi:10.1300/j229v04n01_08

Sar, V., Ozturk, E., & Kundakçı, T. (2002). Psychotherapy of an adolescent with dissociative identity disorder: Change in Rorschach patterns. *Journal of Trauma & Dissociation*, *3*, 81–95. doi:10.1300/j229v03n02_05

Sar, V., & Ross, C. A. (2006). Dissociative disorders as a confounding factor in psychiatric research. *Psychiatric Clinics of North America*, *29*, 129–144. doi:10.1016/j.psc.2005.10.008

Sar, V., & Tutkun, H. (1997). Treatment of dissociative identity disorder in Turkey: A case study. *Dissociation*, *10*, 148–156.

Sargant, W., & Shorvon, H. J. (1945). Acute war neurosis: Special reference to Pavlov's experimental observations and the mechanism of abreaction. *Archives of Neurology & Psychiatry*, *54*, 231–240. doi:10.1001/archneurpsyc.1945.02300100003001

Schacter, D. L., & Kihlstrom, J. F. (1989). Functional amnesia. In F. Boller & J. Grafman (Eds.), *Handbook of neuropsychology* (pp. 508–514). Amsterdam, Netherlands: Elsevier.

Schilder, P. (1939). The treatment of depersonalization. *Bulletin of the New York Academy of Medicine*, *15*, 258–266.

Seo, Y., Shin, M., Kim, S., & Kim, J. (2013). Effectiveness of lorazepam-assisted interviews in an adolescent with dissociative amnesia: A case report. *Neural Regeneration Research*, *15*, 186–190.

Sierra, M., Medford, N., Wyatt, G., & David, A. S. (2012). Depersonalization disorder and anxiety: A special relationship? *Psychiatry Research*, *197*, 123–127. doi:10.1016/j.psychres.2011.12.017

Simeon, D. (2004). Depersonalization disorder: A contemporary overview. *CNS Drugs*, *18*, 343–354. doi:10.2165/00023210-200418060-00002

Simeon, D., Knutelska, M., Nelson, D., & Guralnik, O. (2003). Feeling unreal: A depersonalization disorder update of 117 cases. *Journal of Clinical Psychiatry*, *64*, 990–997. doi:10.4088/jcp.v64n0903

Smith, W. (1996). The use of hypnosis in diagnosis and treatment. In J. L. Spira & I. D. Yalom (Eds.), *Treating dissociative identity disorder* (pp. 219–238). San Francisco, CA: Jossey-Bass.

Spanos, N. P., Weekes, J. R., Menary, E., & Bertrand, L. (1986). Hypnotic interview and age regression procedures in the elicitation of multiple personality symptoms: A simulation study. *Psychiatry: Interpersonal and Biological Processes*, *49*, 298–311. doi:10.1080/00332747.1986.11024330

Spiegel, D., Loewenstein, R. J., Lewis-Fernàndez, R., Sar, V., Simeon, D., Vermetten, E., … Dell, P. F. (2011). Dissociative disorders in DSM-5. *Depression and Anxiety*, *28*, E17–E45. doi:10.1002/da.20923

Spitzer, C., Klauer, T., Grabe, H. J., Lucht, M., Stieglitz, R. D., Schneider, W., & Freyberger, H. J. (2003). Gender differences in dissociation. *Psychopathology*, *36*, 65–70. doi:10.1159/000070360

Soffer-Dudek, N. (2014). Dissociation and dissociative mechanisms in panic disorder, obsessive-compulsive disorder, and depression: A review and heuristic framework. *Psychology of Consciousness*, *1*, 243–270. doi:10.1037/cns0000023

Sookman, D., & Solyom, L. (1978). Severe depersonalization treated by behavior therapy. *American Journal of Psychiatry*, *135*, 1543–1545. doi:10.1176/ajp.135.12.1543

Steele, K., & Colrain, J. (1990). Abreactive work with sexual abuse survivors: Concepts and techniques. In M. Hunter (Ed.), *The sexually abused male* (Vol. 2, pp. 1–155). Lexington, MA: Lexington Books.

Steinberg, M. (1991). The spectrum of depersonalization: Assessment and treatment. In A. Tasman & S. M. Goldfinger (Eds.), *American Psychiatric Press review of psychiatry* (Vol. 10, pp. 223–247). Arlington, VA: American Psychiatric Press.

Steinberg, M. (1994a). *The Structured Clinical Interview for DSM-IV Dissociative Disorders-Revised (SCID-D-R)*. Washington, DC: American Psychiatric Press.

Steinberg, M. (1994b). Systematizing dissociation: Symptomatology and diagnostic assessment. In D. Spiegel (Ed.), *Dissociation: Culture, mind, and body* (pp. 59–90). Washington, DC: American Psychiatric Press.

Terr, L. (1991). Childhood traumas: An outline and overview. *American Journal of Psychiatry*, *148*, 10–20. doi:10.1176/ajp.148.1.10

Tolmunen, T., Maaranen, P., Hintikka, J., Kylm, J., Rissanen, M., Honkalampi, K., … Kaukkanen, E. (2007). Dissociation in a general population of Finnish adolescents. *Journal of Nervous and Mental Disease*, *195*, 614–617. doi:10.1097/nmd.0b013e318093f487

Torch, E. M. (1987). The psychotherapeutic treatment of depersonalization disorder. *Hillside Journal of Clinical Psychology*, *9*, 133–151.

Turpin, G. (2001). Single case methodology and psychotherapy evaluation: From research to practice. In C. Mace, S. Moorey, & B. Roberts (Eds.), *Evidence in the psychological therapies: A critical guide for practitioners* (pp. 91–113). Philadelphia, PA: Taylor & Francis.

Twombly, J. H. (2013). Integrating IFS with phase-oriented treatment of clients with dissociative disorder. In M. Sweezy & E. Ziskind (Eds.), *Internal family systems therapy* (pp. 72–89). New York, NY: Routledge.

Van der Hart, O., & Boon, S. (1997). Treatment strategies for complex dissociative disorders: Two Dutch case examples. *Dissociation*, *10*, 157–165.

Van der Kloet, D., Giesbrecht, T., Franck, E., Van Gastel, A., De Volder, I., Van Den Eede, F., … Merckelbach, H. (2013). Dissociative symptoms and sleep parameters: An all-night polysomnography study in patients with insomnia. *Comprehensive Psychiatry*, *54*, 658–664. doi:10.1016/j.comppsych.2012.12.025

Van der Kloet, D., Giesbrecht, T., Lynn, S. J., Merckelbach, H., & de Zutter, A. (2012). Sleep normalization and decrease in dissociative experiences: Evaluation in an inpatient sample. *Journal of Abnormal Psychology*, *121*, 140–150. doi:10.1037/a0024781

Van der Kloet, D., Merckelbach, H., Giesbrecht, T., & Lynn, S. J. (2012). Fragmented sleep, fragmented mind: The role of sleep in dissociative symptoms. *Perspectives on Psychological Science*, *21*, 48–53.

Van der Kolk, B. A. (1986). The drug treatment of post-traumatic stress disorder. *Journal of Affective Disorders*, *13*, 203–213. doi:10.1016/0165-0327(87)90024-3

Van Heugten-Van der Kloet, D., Giesbrecht, T., & Merckelbach, H. (2015). Sleep loss increases dissociation and affects memory for emotional stimuli. *Journal of Behavior Therapy and Experimental Psychiatry*, *45*, 9–17. doi:10.1016/j.jbtep.2014.11.002

Van Heugten-Van der Kloet, D., Huntjens, R., Giesbrecht, T., & Merckelbach, H. (2014). Self-reported sleep disturbances in patients with dissociative identity disorder and post-traumatic stress disorder and how they relate to cognitive failures and fantasy proneness. *Frontiers in Psychiatry*, *5*, 19. doi:10.3389/fpsyt.2014.00019

Van Heugten-Van der Kloet, D., Merckelbach, H., Giesbrecht, T., & Broers, N. (2014). Night-time experiences and daytime dissociation: A path analysis modeling study. *Psychiatry Research*, *216*, 236–241. doi:10.1016/j.psychres.2013.12.053

Walker, M. P., & Van der Helm, E. (2009). Overnight therapy? The role of sleep in emotional brain processing. *Psychological Bulletin*, *135*, 731–748. doi:10.1037/a0016570

Watson, D. (2001). Dissociations of the night: Individual differences in sleep-related experiences and their relation to dissociation and schizotypy. *Journal of Abnormal Psychology*, *110*, 526–535. doi:10.1037//0021-843x.110.4.526

Wing, J. K., Cooper, J. E., & Sartorius, N. (1974). *The description and manual for the PSE and CATEGO system*. London, UK: Cambridge University Press.

Yalom, I. D., & Leszcz, M. (2005). *Theory and practice of group psychotherapy*. New York, NY: Basic Books.

Yu, J., Ross, C. A., Keyes, B. B., Li, Y., Dai, Y., Zhang, T., … Xiao, Z. (2010). Dissociative disorders among Chinese inpatients diagnosed with schizophrenia. *Journal of Trauma & Dissociation*, *11*, 358–372. doi:10.1080/15299731003793468

14

Psychotherapy for Schizophrenia-Spectrum Disorders

Stacy Ellenberg,[1] Steven Jay Lynn,[1] and Gregory P. Strauss[2]
[1] *Department of Psychology, Binghamton University, Binghamton, USA*
[2] *Department of Psychology, University of Georgia, Athens, USA*

Schizophrenia and related psychotic disorders are debilitating, chronic mental disorders characterized by persistent delusions, hallucinations, disorganized thoughts and behavior, and negative symptoms (American Psychiatric Association, 2013). Psychotic disorders include schizophrenia, schizophreniform disorder, schizotypal (personality) disorder, delusional disorder, brief psychotic disorder, substance/medication-induced psychotic disorder, schizoaffective disorder, and what used to be termed psychosis NOS: other specified/unspecified schizophrenia-spectrum and other psychotic disorders (APA, 2013). Schizotypal personality disorder is listed in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) under schizophrenia-spectrum disorders as well as personality disorders.

Schizophrenia is characterized by at least 6 months (including prodromal and residual symptoms) of the following symptoms: (1) delusions, (2) hallucinations, (3) disorganized speech, (4) grossly disorganized/catatonic behavior, and (5) negative symptoms. Further, individuals must have experienced at least two of the above criteria for a majority of the time for 1 month, which must include delusions, hallucinations, or disorganized speech. Severe negative symptoms can occur in the prodromal and residual stages (American Psychiatric Association, 2013). The main focus of this chapter is on schizophrenia, although many of the clinical trials reviewed include a variety and combination of schizophrenia spectrum disorders (e.g., schizoaffective disorder, psychosis NOS).

Delusions are defined as strongly held beliefs that are clearly implausible or not based in reality. Delusions can be categorized as, for example, delusions of grandeur, erotomatic delusions, jealous delusions, somatic delusions, or religious delusions. Hallucinations are perceptual experiences that occur absent an external stimulus. Auditory hallucinations are most likely to occur in schizophrenia (National Institute of Mental Health, 2015). Disorganized speech, also known as formal thought disorder, occurs when an individual speaks tangentially or uses neologisms, or the flow of associations derails into a loosely or completely unrelated topic. Disorganized behavior can include catatonia and abnormal motor behaviors, which encompass excessive movement, such

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

as stereotyped behaviors or tics, or excessive lack of movement, such as mutism or stupor (American Psychiatric Association, 2013). Negative symptoms include anhedonia, alogia, avolition, asociality, and blunted affect (American Psychiatric Association, 2013). Significant functional impairment is also one of the central features of schizophrenia, thereby placing a burden on patients, families, and the community.

Per the DSM-5, the lifetime prevalence of schizophrenia is estimated to be 0.3–0.7%. Of that percentage, 5–6% complete suicide. Only 20% of individuals with schizophrenia will experience a remission in their lifetime (American Psychiatric Association, 2013). Individuals with schizophrenia represent 121.9 of every 100,000 hospital inpatient stays in the United States. Further, within 30 days of discharge from inpatient hospitalization, 18.6% of patients are rehospitalized with any psychosis, and 22.4% are rehospitalized for any reason (Heslin & Weiss, 2015).

A late winter or spring birth, an upbringing in an urban environment, increased paternal age, being an immigrant, being single, bullying, social isolation, early stressors, low birth weight, and substance use all are risk factors linked to schizophrenia (Bebbington et al., 2004; Byrne, Agerbo, Eaton, & Mortensen, 2004; Hultman, Ohman, Cnattingius, Wieselgren, & Lindstr, 1997; Kuepper et al., 2011; Malaspina et al., 2001; Mander & Kingdon, 2015; Resnick, Bond, & Mueser, 2003; Sundquist, Frank & Sundquist, 2004; Veling et al., 2008). Schizophrenia is equally likely to manifest in both women and men, but men typically experience a more chronic course and more negative symptoms (Strauss, Harrow, Grossman, & Rosen, 2010). Female onset of psychosis typically occurs in the late twenties, whereas males tend to develop psychosis earlier in life, during the early to mid-twenties (American Psychiatric Association, 2013). Females tend to experience better long-term functional and symptomatic outcomes than males (Grossman, Harrow, Rosen, Faull, & Strauss, 2008).

Cognitive deficits are highly prevalent in schizophrenia and include deficits in processing speed, working and declarative memory, language abilities, and attentional abilities (American Psychiatric Association, 2013; Mander & Kingdon, 2015). Individuals with schizophrenia commonly exhibit cognitive deficits early in childhood that impede social and academic progress, implying a developmental component. Better cognitive performance in processing speed, working memory, attention, problem-solving, and visual memory are associated with better functional outcomes (Schmidt, Mueller, & Roder, 2011).

Individuals with schizophrenia also often exhibit social cognitive deficits. Green, Horan, and Lee (2015) classified social cognitive deficits into four broad categories. A deficit in mentalization, also known as theory of mind, which represents the ability to recognize and identify that another individual has thoughts and emotions, can lead to the perception of others' neutral behaviors as malicious, which can exacerbate persecutory delusions (American Psychiatric Association, 2013). Social cue perception, defined by incorrect perception of vocal and facial cues, is also frequently impaired in schizophrenia, as is experience sharing, which is a deficit in normative activation of the brain in response to observation of others' actions. Green et al. (2015) propose that schizophrenia encompasses deficits in emotion regulation, which refers to the ability to implement strategies to exert cognitive control over the experience of emotion. However, increases in social cognition can ameliorate the effect of cognitive deficits on functioning (Schmidt et al., 2011). Further, individuals with schizophrenia often exhibit limited insight into their illness, which is a strong predictor of medication

noncompliance, aggression, relapse, and a poorer prognosis in general (American Psychiatric Association, 2013).

Brain abnormalities are common and typically characterized by reduced overall brain volume, enlarged ventricles with age, enlarged cerebrospinal fluid spaces, and underdeveloped frontal and medial temporal lobes (American Psychiatric Association, 2013; Ho et al., 2003). Brain deterioration, as indicated by enlarged ventricular structures and reduced frontal lobes, is related to poorer functional outcomes (Ho et al., 2003).

Schizophrenia has been ranked as one of the most disabling of all disorders (Schmidt et al., 2011). Substance use and mood symptoms are highly comorbid with schizophrenia. Approximately half of individuals with schizophrenia report symptoms of depression (American Psychiatric Association, 2013). Further, anxiety is commonly comorbid with psychosis, with 25% of individuals possibly having comorbid obsessive–compulsive disorder, 20% having comorbid panic disorder, 30% having posttraumatic stress disorder, 40% having substance misuse, and 50% having a personality disorder (Keown, Holloway & Kuipers, 2002, 2005; Kuipers et al., 2006; Mander & Kingdon, 2015; Mueser et al., 1998; Scott et al., 1998; Turnbull & Bebbington, 2001). Anxiety, phobia, dissociative symptoms, depression, anger, and disturbed sleep also commonly co-occur in schizophrenia. Over half of individuals with schizophrenia meet criteria for tobacco use disorder as well (American Psychiatric Association, 2013). Moreover, Mueser et al. (1998) found that 98% of schizophrenic patients reported a history of trauma, which is associated with increased rates of rehospitalization (Doering et al., 1998).

The costs of psychosis to the US economy are estimated to be as high as $317 billion yearly (Insel, 2008). These costs encompass losses in economic productivity due to disability, costs of high rehospitalization rates, and indirect costs due to medication noncompliance.

14.1 Treatment for Schizophrenia

It was not until the 1960s that psychosocial interventions were considered viable treatments for schizophrenia. Today, a multitude of psychological interventions, supplemented with medications, are highly effective in reducing positive symptoms but often fail to produce favorable functional outcomes or improvements in negative symptoms of schizophrenia (Mander & Kingdon, 2015). Individuals with schizophrenia and other psychosis-spectrum disorders exhibit limited insight, cognitive impairments, and high levels of stress that interfere with treatment and engender a high rate of attrition (Dickerson & Lehman, 2011). Still, a variety of psychotherapeutic interventions have been developed, which borrow from interventions that target less severe mental illness, with treatment aimed at returning the patient to a baseline or minimal level of adaptive functioning, such as maintaining independence and carrying out activities of daily living.

Although a plethora of interventions exist, our review will discuss only the most widely used treatments with the most substantive bodies of empirical research. A Patient Outcomes Research Team (PORT) National Institute of Mental Health schizophrenia publication (Kreyenbuhl, Buchanan, Dickerson, & Dixon, 2010) reviewed the literature on psychotherapy for schizophrenia. PORT reviews are developed by two teams of experts and nonexperts who obtain information from rigorous studies to draw conclusions regarding the evidence base of treatments. The authors of the

PORT review listed the following modalities as evidence-based treatments: cognitive–behavioral therapy (Beck, 1979), skills training, Supported Employment, Assertive Community Treatment, family-based services, and token economy interventions. Our review focuses on individual and group psychotherapies and does not evaluate family and institution-based treatments, such as Supported Employment for Schizophrenia, in depth, although we touch on the effectiveness of these other approaches at the end of our review.

We adopt David and Montgomery's (2011) nine-category framework, which classifies interventions in terms of levels of both (1) empirical support, ranging from no support to moderate to strong support for treatment package effectiveness, and (2) mechanisms hypothesized to mediate or moderate treatment effects based on the same three levels of classification. The framework not only considers absolute efficacy (i.e., therapeutic package better than a comparison condition) but also considers efficacy relative to another evidence-based psychological treatment (relative efficacy) and specific efficacy (i.e., performs better than a pill and/or medical or psychological placebo or equivalent to or better than active standard psychological therapies).

14.2 Category II

No interventions meet the criteria for a Category I treatment (substantial support for both the treatment package and treatment mechanisms). Category II therapies have a well-supported treatment package with equivocal, preliminary, or mixed findings regarding treatment mechanism. In the following sections, we argue that cognitive–behavioral therapy (CBT) and social skills training (SST) merit a tentative Category II designation. Although the literature is not exclusively positive, which is not surprising given the heterogeneity of conditions and interventions, we base this designation on the overall patterns of findings reported in the literature.

14.2.1 Cognitive–Behavioral Therapy

In the 1950s, before Aaron Beck targeted anxiety and depression with CBT, he focused on the treatment of faulty beliefs and delusional guilt in schizophrenia (Beck, 1952; Mander & Kingdon, 2015). CBT for schizophrenia enjoyed a second rise in interest during the 1980s when researchers (Tarrier, Harwood, Yusupoff, Beckett, & Baker, 1990) devised CBT for psychosis that targeted coping mechanisms to alleviate symptoms and enhance functioning (Mander & Kingdon, 2015). Elis' rational emotive behavior therapy has also been proposed as a framework for treating psychotic disorders (Kingdon & Turkington, 2006)

The term CBT encompasses a multitude of therapies, including acceptance and commitment therapy (ACT), metacognitive training, SST, cognitive remediation, and group CBT. The overlap among these treatments is significant, so the separation of the treatments is somewhat arbitrary. Nevertheless, we adopt the terms used by the researchers themselves. Accordingly, this section only includes treatments that researchers label CBT. Like other psychotherapies we review, CBT is intended to be used as an adjunct treatment to case management and psychopharmacological interventions in the treatment of psychosis.

CBT for schizophrenia and other psychosis-spectrum disorders typically ranges from 6 to 20 sessions over 6 weeks to 9 months to several years, and is preferably administered by a doctoral-level clinician (Bellack, 2002; Dickerson & Lehman, 2011). Individual CBT is most common, but group CBT is an important modality as well (Dickerson & Lehman, 2011). The ideal patients for CBT are those with lifelong psychosis and whose distress is caused by positive symptoms but who embrace less than complete conviction in their delusions (Dickerson & Lehman, 2011).

Therapy begins with establishing an empathic working alliance, which is deemed pivotal. Cognitive techniques are used to dismantle delusions gently. As initial distress subsides, therapists reshape thoughts and distorted cognitive beliefs associated with the development and maintenance of symptoms. CBT challenges the validity of delusions, loosening up tightly held convictions regarding delusions and correcting automatic thought processes that lead to faulty beliefs regarding the validity of hallucinations and delusions (Barlow, 2014). No two studies are alike, and any study may endorse a wide variety of these techniques, which makes it difficult to pin down the components of treatment that contribute most to overall outcome (Dickerson & Lehman, 2011).

14.2.1.1 Components of the therapy

Common components of CBT include a strong therapeutic alliance, psychoeducation, cognitive–behavioral techniques, stress reduction, and relapse prevention (Bellack, 2002). The following methods have all been used in CBT for psychosis: thought focusing/reattribution, belief modification, normalizing of symptoms, reality testing, activity-scheduling, psychoeducation, analysis of symptoms, building coping skills, problem-solving, and relaxation training (Dickerson & Lehman, 2011; Durham et al., 2003; Turkington et al., 2008).

Belief modification, reality-testing, and providing alternative explanations encourage patients to develop an alternative formulation of hallucinations and delusions. Reality-testing involves having patients go into the "real world" to test the validity of delusions (Dickerson, 2000). Using verbal challenges, the therapist suggests, but does not assert, an alternative explanation for the patient's delusion and explores how the delusional belief has affected the patient's broader skein of beliefs and actions. The therapist then asks the patient questions regarding evidence for and against a delusional belief, starting with the least and working up to the most threatening delusions. Focusing and reattribution are used to reduce the occurrence and distress associated with auditory hallucinations (Dickerson, 2000). Patients are asked to elaborate on their experience of a hallucination; to describe its content and qualities in detail and in terms of loudness, vividness, duration, and frequency; and to record these qualities at home. Finally, patients are asked about their beliefs and reactions to the hallucination and instructed to record what happened immediately before its occurrence (Dickerson, 2000; Dickerson & Lehman, 2011). The goal of these exercises is to facilitate the recognition that the hallucinations originate within the patient, rather than an external source (Dickerson & Lehman, 2011). Coping skills can include behavioral activation, relaxation training, attention switching, and self-statements, which can be practiced at home and in session (Dickerson, 2000). Self-statements include phrases that can be repeated in response to high anxiety and underscore the fact that anxiety is temporary and that the individual will persist despite the anxiety. The therapist normalizes symptoms, "decatastrophizes" the diagnosis,

explains how life circumstances might have exacerbated or preceded the psychosis, and provides reassurance that the symptoms are a part of the human experience (Dickerson, 2000). Indeed, researchers have estimated that 25–40% of the population experience voices (Mander & Kingdon, 2015). Finally, homework is an integral component, as patients are encouraged to practice cognitive strategies in everyday life (Bellack, 2002). As patients are gently guided through the process of rationalization of delusions and are taught new coping skills, they can move into the phase in which treatment gains are maintained, and the final focus of the therapy becomes relapse prevention (Valmaggia, Van der Gaag, Tarrier, Pijnenborg, & Slooff, 2005).

14.2.1.2 Evidence for the therapy

Research has focused mainly on adult inpatients and outpatients and has most often included varying disorders on the psychotic spectrum (e.g., schizoaffective disorder, delusional disorder, schizophrenia, other psychosis/bipolar disorders, and psychosis NOS). Studies differ in terms of patient age, duration of illness, socioeconomic status, level of symptom severity, comorbid symptoms, inpatient/outpatient setting, group/individual context, and level of cognitive functioning. Due to the heterogeneity of techniques and comparison groups, it is difficult to derive unambiguous conclusions regarding the effectiveness of CBT. Although researchers have conducted qualitative reviews and meta-analyses, they have reported mixed findings and diverse evaluations of the literature. Still, PORT (Kreyenbuhl et al., 2010) recommends CBT as an evidence-based treatment for individuals with chronic psychosis who are taking medications (Kreyenbuhl et al., 2010; Jauhar et al., 2014). Further, the American Psychiatric Association recommends CBT as a treatment for schizophrenia (American Psychological Association [APA] Presidential Task Force on Evidence-Based Practice, 2006; Jauhar et al., 2014). Nevertheless, a recent Cochrane Review concluded that CBT cannot be considered superior to other psychosocial interventions for schizophrenia (Jones, Hacker, Cormac, Meaden & Irving, 2012).

14.2.1.2.1 Reviews

Several qualitative reviews and meta-analyses have assessed the effectiveness of CBT for psychosis. The findings vary but indicate that CBT is effective for schizophrenia overall, with a modest effect size. For example, Wykes, Steel, Everitt, and Tarrier (2008) examined 34 clinical trials for psychosis and found significant effects for the following outcomes: social functioning (effect size = 0.38), positive symptoms (0.37), negative symptoms (0.44), and mood (0.36). Although it did not achieve significance, the authors reported an effect size of 0.44 pertaining to overall improvement on the targeted symptom. Because the meta-analysis included nonblinded studies, the effect sizes may be exaggerated. Moreover, the effect sizes were based on varying numbers of clinical trials. Additionally, when the researchers restricted their analysis to methodologically rigorous studies, positive symptoms was the only variable that remained significant (Dickerson & Lehman, 2011; Wykes et al., 2008). Further, Turner, Van der Gaag, Karyotaki, and Cuijpers (2014) conducted a meta-analysis of 48 randomized controlled trials (RCTs) and found that CBT reduced positive symptoms and was more efficacious than other interventions, with an effect size of 0.16. Nevertheless, when the authors accounted for researcher allegiance, the findings were no longer significant. Additionally, CBT, SST, and cognitive remediation all improved overall outcomes significantly, but, again,

statistical significance was not maintained when the researchers controlled for allegiance effects (Turner et al., 2014).

Bustillo, Lauriello, Horan, and Keith (2001) reviewed five RCTs of CBT for schizophrenia that examined primary outcomes (positive symptom reduction) and secondary outcomes (reduction in negative symptoms and improvement in social functioning). Comparison conditions included supportive counseling, befriending, treatment as usual (TAU), and structured activities with a therapist. Befriending is supportive counseling that matches for time spent with therapist, session frequency, and time elapsed between sessions. Three of the studies examined only medication-resistant patients with schizophrenia. The authors concluded that CBT shows promise over comparison conditions in improving primary outcomes for medication-resistant patients. One study found that rates of rehospitalization were reduced by 9 months (Buchkremer, Klingberg, Holle, Mönking, & Hornung, 1997), and another study documented improvements in negative and depressive symptoms (Sensky et al., 2000). Nevertheless, social functioning and relapse were not improved in these five RCTs (Bustillo et al., 2001). Bellack (2002) compiled findings from multiple meta-analyses and reported that, whereas CBT is effective in improving overall symptoms, in reducing strong convictions over delusions, and in reducing the distress related to hallucinations and frequency of hallucinations, insufficient evidence exists to conclude that CBT reduces relapse rates.

Gaudiano (2005) reviewed the literature on CBT for psychosis and concluded that studies have indicated overall positive results across multiple settings (i.e., inpatient vs. outpatient) and for varying subtypes (i.e., acute vs. first-episode psychosis) of psychosis. Indeed, the literature indicates that CBT is promising for early intervention psychosis, comorbid psychosis, and acute psychosis. Gaudiano (2005) discovered that CBT was superior in comparison to waitlist conditions, although more research comparing CBT with active treatments is needed. Moderators and mediators of CBT should also be further researched to identify (1) who benefits most from CBT and (2) through which components of CBT individuals benefit. Further, researchers should include a wider array of outcome measures, including measures of quality of life, and examine the generalizability and duration of the effects of CBT. Finally, Gaudiano (2005) cautioned that the heterogeneity of treatment protocols further complicates conclusions to be drawn regarding the efficacy of CBT. Gaudiano (2005) concluded that research on CBT for psychosis remains in a preliminary stage, but the literature consistently shows promising and supportive results in favor of using CBT.

Gaudiano (2006) conducted a meta-analysis of 12 RCTs comparing CBT with routine care and examined clinical significance and reliable change, compared with statistical change. Gaudiano (2006) contended that statistical change may not be an appropriate analysis in treatment outcome research, as clinical significance captures a more realistic index of change. Gaudiano (2006) examined clinically significant change, defined as a change of two standard deviations from original scores, and reliable change, which assesses whether a significant score change has occurred for a particular outcome measure. Gaudiano (2006) found reliable change in 42% of CBT conditions and in 25% of routine care conditions on at least one measure of psychosis. However, CBT and routine care varied little, with a 48% clinical change for CBT and a 52% change for routine care (Gaudiano, 2006).

Lynch, Laws, and McKenna (2010) conducted a meta-analysis that included nine RCTs related to schizophrenia, bipolar disorder, and depression. Although the effect size

was small, CBT was effective in reducing depression and relapse, but it was not effective in reducing psychotic symptoms or preventing relapse. Specifically, the effect size for positive symptoms changed from −0.19 favoring CBT to −0.08 when only blinded studies were considered. The effect size for negative symptoms remained at −0.02 regardless of blinding (Lynch et al., 2010).

Sarin, Wallin, and Widerlöv (2011) performed a meta-analysis of 22 RCTs in which they examined follow-up effects of CBT versus TAU and other psychological (e.g., psychoeducation, family intervention, befriending) treatments at 3 to 15 months. Although the treatment effect was small, they found that CBT improved outcomes significantly on positive (Hedges' $g = -1.01$), negative ($g = -0.20$), and general symptoms ($g = -4.80$) at follow-up, but CBT failed to outperform TAU and other treatments immediately after the intervention was completed. A negative *g* effect size favored CBT. The effects of CBT may require time for benefits to consolidate. Indeed, patients who received 20 or more sessions displayed improved outcomes over those who received fewer sessions (Barlow, 2014; Sarin et al., 2011).

A Cochrane Review based on a meta-analysis of 20 RCTs reported that CBT was comparable to other treatment modalities (e.g., family therapy, medication management, relaxation, supportive therapy, psychoeducation) with regard to rates of relapse and rehospitalization, positive symptoms, social functioning, and global functioning (Jones et al., 2012). Affective outcomes were favored by CBT in the long term. CBT was superior to other treatments in the long term only in reducing positive symptoms. CBT showed improvements in the short term for global functioning and social functioning, and an advantage was reported for CBT over other treatments in ameliorating negative symptoms, problem behaviors, relapse, and rehospitalization rates (Jones et al., 2012). Despite the generally negative results from this Cochrane Review, the authors contend that more research is needed to draw definitive conclusions. Nevertheless, it is apparent that CBT benefits individuals with schizophrenia by reducing affective symptoms, which is especially relevant for a diagnosis with such a high comorbidity with depression.

Jauhar et al. (2014) examined 52 RCTs of CBT, which included ACT and CBT that incorporated skills from other treatment modalities, including SST, motivational interviewing, family engagement, and basic behavior therapy. ACT employs transdiagnostic treatment strategies aimed at improving acceptance of symptoms and increasing mindfulness. ACT's approach is not to attempt directly to diminish symptoms but to improve emotion regulation and decrease experiential avoidance to produce symptom change. Preliminary findings have suggested ACT is able to reduce rehospitalization rates and distress associated with hallucinations (Bach & Hayes, 2002; Gaudiano & Herbert, 2006).

Jauhar et al.'s (2014) findings supported Wykes et al. (2008) in that there was an overall reduction in symptoms with an effect size of −0.33; however, when only blinded trials were included, the effect size diminished to −0.15 (Hedges' *g*). Notably, the negative effect size indicates that the effect size favors CBT. The effect size was −0.25 for positive symptoms (reduced to −0.10) and −0.13 for negative symptoms (reduced to −0.02; Jauhar et al., 2014). The authors conclude that CBT should not be recommended as an effective treatment for positive and negative symptoms due to these small effect sizes.

14.2.1.2.2 Randomized controlled trials

Researchers have conducted RCTs and open trials with positive results. According to the most recent Cochrane Review, researchers have conducted 20 RCTs comparing CBT

with another treatment or comparison condition (TAU or waitlist) (Jones et al., 2012). The following studies portray the mixed findings regarding CBT for psychosis. The list of clinical trials is not exhaustive. See Table 14.1 for a summary.

Drury, Birchwood, Cochrane, and MacMillan (1996) randomly assigned 40 inpatients with any form of psychosis (other than mania) to either 12 weeks of cognitive therapy or a recreation and support group. Both groups showed significant improvements in positive symptoms, with cognitive therapy improving symptoms more rapidly and to a greater extent over the 12 weeks (Drury et al., 1996). The degree to which participants were convinced of and preoccupied with delusions improved significantly in both groups, and did so to a greater extent in the cognitive treatment condition. Both groups improved in negative symptoms and thought disorganization. No group differences were noted with respect to negative symptoms at a 9-month follow-up but positive symptoms were still improved significantly for the cognitive treatment condition. Finally, cognitive therapy improved patients' perceived control over hallucinations and delusions.

Haddock et al. (1999) compared CBT with supportive counseling plus psychoeducation with 21 inpatients with schizophrenia and schizoaffective disorder in an acute, early stage. Participants were randomized to conditions, and treatment providers were blinded to conditions. There were no differences between the treatments with regard to symptoms or length of stay in hospital. Both groups had significant reductions in psychiatric symptoms.

Lewis et al. (2002) conducted a blinded RCT that compared CBT with both supportive counseling and TAU among 172 in- and outpatients with schizophrenia, schizophreniform disorder, schizoaffective disorder, and delusional disorder. Participants were provided with 5 weeks of treatment and 2 weeks of "booster sessions." Of the three groups, CBT provided the most rapid improvement in the rate at which participants reached remission, including "resolution of auditory hallucinations." CBT was also superior to TAU in improving scores on the Positive and Negative Syndrome Scale (PANSS; Kay, Flszbein, & Opfer, 1987) and the Psychotic Symptom Rating Scale (PSYRATS; Haddock, McCarron, Tarrier, & Faragher, 1999) at 5 weeks, but these positive findings did not replicate at the final test (Lewis et al., 2002).

Durham et al. (2003) examined differences between CBT plus TAU, supportive psychotherapy plus TAU, and TAU. The researchers were blinded to conditions. The results were not significant, yet 25% improvements were seen on the PANSS in the CBT plus TAU group. Further, twice as many participants in the CBT plus TAU group had a 25% improvement on the PANSS compared to the two other groups. The CBT plus TAU group also rated themselves as more improved than the other two groups, but strangely also rated themselves as having deteriorated to some degree compared with the other two groups. Perhaps an increase in insight was responsible for these seemingly discrepant findings.

Rector, Seeman, and Segal (2003) examined 20 sessions (6 months) of individual CBT plus "enriched TAU" (medication management, case management, psychoeducation groups) versus just enriched TAU in 42 outpatients with schizophrenia and schizoaffective disorder. The researchers found that the CBT plus enriched TAU group and the TAU group had improved positive and negative symptoms at follow-up (immediately after treatment), but the difference between the conditions was not significant: Both groups improved in general symptomatology, with CBT plus enriched TAU showing greater (but nonsignificant) improvements. At 6 months posttreatment, the CBT plus enriched

Table 14.1 Summary of cognitive–behavioral randomized controlled trials included in this review.

Authors and Year	*N*	Experimental Group	Comparison Group	Sessions/Weeks of Treatment	Absolute Efficacy (vs. Comparison)	Relative Efficacy	Specific Efficacy
Drury, Birchwood, Cochrane, & MacMillan (1996)	40 inpatients	Cognitive therapy	Recreation and support group	12 weeks	Both improved: positive/negative sxs, thought disorganization. Superior to comparison in positive sxs, preoccupation of delusions, perceived control over positive sxs.		
Haddock, Tarrier et al. (1999)	20 inpatients	CBT	Supportive counseling/ psychoeducation	4 months	Superior to comparison in relapse rates (not significant). No difference between groups for length of stay at hospital; positive/negative sxs.		
Lewis et al. (2002)	172 in/outpatients	CBT	Supportive counseling or routine care	5 weeks + 2 weeks of booster sessions	Superior to comparisons in speed of recovery; positive/negative sxs (only shown at 5 weeks; not after).		
Durham et al. (2003)	66 in/outpatients	CBT+TAU	TAU+supportive therapy or TAU	9 months	Superior to comparison in positive/negative sxs and ratings of improvement (not significant).	Superior to standard treatment in positive/negative sxs and ratings of improvement (not significant).	
Rector, Seeman, & Segal (2003)	42 outpatients	CBT+enriched TAU	Enriched TAU	20 sessions/ 6 months		Superior to comparison in overall/negative sxs (not significant); superior to comparison in negative sxs at 6-month follow-up.	
Bechdolf et al. (2004)	88 inpatients	Group CBT	Group psychoeducation	16 sessions/ 8 weeks	Both improved: clinical sxs (psychoeducation to a greater extent). Superior to comparison in rehospitalization rate.		

Valmaggia, Van der Gaag, Tarrier, Pijnenborg, & Slooff (2005)	58 inpatients	CBT	Supportive counseling	16 sessions/ 22 weeks	Superior to comparison at follow-up in positive sxs/illness insight (not maintained at 6-month follow-up).	
Jackson et al. (2005)	79 inpatients with first-episode psychosis	Cognitively oriented psychotherapy for early psychosis	Patients who did not agree to treatment or patients who were not offered treatment	1–34 sessions	Not superior to comparisons in rehospitalization.	
Turkington et al. (2008)	59 outpatients	CBT	Befriending	20 sessions/ 9 months	Superior to comparison after 5 years in overall and negative sxs.	
Penn et al. (2009)	65 outpatients	Group CBT	Enhanced supportive therapy	12 sessions/ 12 weeks	Superior to comparison in overall sxs. Comparison superior to experimental in improving negative beliefs.	
Velligan et al. (2014)	166 outpatients	CBT; Cognitive Adaptation Training; multimodal cognitive training	TAU	9 months	Cognitive Adaptation Training superior to CBT in positive sxs/associated distress; functional outcomes.	
de Jong et al. (2014)	78 outpatients	Group CBT	Standard support group	12 sessions/ 6 months	Not superior to comparison in rate of job satisfaction/employment.	
Freeman et al. (2015)	150 outpatients	CBT	Standard care	8 weeks		Superior to standard care: worry ratings, persecutory delusion.

CBT = cognitive–behavioral therapy; sxs = symptoms; TAU = treatment as usual.

TAU group exhibited a significantly greater improvement in negative symptoms compared with the TAU group. The researchers replicated improved negative symptoms at follow-up in a CBT group in a study that examined outcomes at 5 years in a study comparing CBT to befriending (Turkington et al., 2008). After 20 sessions over 9 months, 59 outpatients with schizophrenia improved significantly in overall and negative symptoms when assigned to CBT versus befriending. Raters were blinded to treatment condition.

Bechdolf et al. (2004) compared group CBT with group psychoeducation. The authors noted that the group CBT had elements of affective, cognitive, and psychomotor intervention. Participants assigned to CBT had significantly fewer rehospitalizations compared with participants assigned to psychoeducation during the follow-up period. Both groups' symptoms improved significantly, and, surprisingly, the psychoeducation group did so to a greater extent (Bechdolf et al., 2004).

Although Bechdolf et al. (2005) did not conduct an RCT, they found that CBT decreased prodromal symptoms and improved social adjustment in outpatients with prodromal psychotic symptoms (Bechdolf et al., 2005). When compared with a separate naturalistic study (Hambrecht, Lammertink, Klosterkötter, Matuschek, & Pukrop, 2002), people who had received CBT experienced a slower progression into psychosis.

Valmaggia et al. (2005) examined CBT versus supportive counseling. The participants experienced cognitive deficits and were resistant to atypical antipsychotics. The researchers found that CBT was superior to supportive counseling at the end of treatment with regard to auditory hallucinations, interpretations of auditory hallucinations, knowledge of the origin of auditory hallucinations, and the veridicality of auditory hallucinations. Although the level of conviction regarding voices and the belief in the origin of auditory hallucinations decreased significantly, group differences failed to replicate at follow-up. Perhaps the lack of significant findings at follow-up was due to the low level of cognitive functioning of the sample (Valmaggia et al., 2005).

Jackson et al. (2005) examined "cognitively oriented psychotherapy for early psychosis" (COPE, which the authors consider a form of CBT) among 79 inpatients experiencing their first psychotic episode. The researchers compared outcomes for people who agreed to treatment with a comparison group of people who did not agree to treatment, and a third group that was not offered treatment. The treatment package addressed deficits in functional abilities, such as activities of daily living, relationship tasks, and other aspects of occupational and developmentally appropriate functioning. Participants were assessed at 6 and 12 months. The number of sessions ranged from 1 to 34 and depended largely on the mental state of the patient. The researchers found no differences among the groups in rehospitalization rate, and no differences in other outcomes, including depression, general symptomatology, quality of life, or positive and negative symptoms. By the fourth year, COPE participants had more rehospitalizations. The authors note, however, that positive symptoms and the accompanying distress were not the focus of therapy.

Penn et al. (2009) conducted a blinded RCT that compared group CBT to enhanced supportive therapy. The enhanced supportive therapy condition focused on the therapeutic alliance, establishing goals, and building social skills. Participants were assessed at 3 months and 12 months posttreatment. CBT was superior to supportive therapy in improving overall symptoms as measured by the PANSS at 12 months, and supportive therapy was superior to CBT in improving negative beliefs. The mechanisms underlying

these unusual findings, and why supportive therapy provided benefits that CBT did not, are not understood.

De Jong et al. (2014) compared group CBT with a standard support. Outcomes were measured through job satisfaction and employment. The researchers found that CBT did not significantly improve job satisfaction, but they found a relation between metacognitive ability and treatment, in that metacognitive abilities predicted job satisfaction. This relation was evident only in the CBT group.

Velligan et al. (2014) randomly assigned 166 outpatients with schizophrenia or schizoaffective disorder to 9 months of CBT, Cognitive Adaptation Training (CAT), multimodal cognitive training (a combination of CBT and CAT), or TAU. CAT involves a regimented process of placing cues (e.g., signs, alarms) in one's natural environment that prompt regular activities of daily living. The researchers found that the CAT condition was superior to CBT. CAT improved hallucinations and accompanying distress, as well as the ability to function normally in the community and tend to activities of daily living. Adding CBT to CAT did not provide any additional benefit, and the CBT condition alone did not improve any outcomes. However, the CAT condition emphasized medication compliance, and therapists may have been more familiar with CAT than they were with CBT (Velligan et al., 2014). Although other factors may account for the improvement associated with CAT, they do not account for the fact that the CBT condition produced no improvements in target measures.

Freeman et al. (2015) compared 8 weeks of standard care to 8 weeks of a specialized form of CBT designed to reduce worry with standard care among an almost exclusively outpatient sample of individuals diagnosed with schizophrenia, schizoaffective disorder, or delusional disorder. Raters were blinded to treatment condition. By the end of treatment, CBT had significantly improved persecutory delusions and worry versus the comparison group; this difference was maintained at 24-week follow-up (Freeman et al., 2015). This study qualifies as a rigorous RCT that provides support for CBT as superior to standard care for treating delusions and worry.

The studies reviewed produced no definitive conclusions regarding whether CBT reduces rehospitalization, positive symptoms, and negative symptoms compared with an active comparison condition or standard treatment. It is difficult to find trends in the effectiveness of CBT, as many of its positive effects either did not persist through follow-up or only appeared much later on. The heterogeneity of these studies further increases the challenge to derive conclusive results regarding the effectiveness of CBT. Indeed, many of the studies assessed a wide range of participants (i.e., inpatients, outpatients, different age groups, different stages of psychosis) as well as a variety of implementations of CBT (e.g., group CBT, cognitive therapy only, CBT plus TAU). Overall, although the findings were not consistent across studies, positive and negative symptoms appeared to improve more in treatment compared with comparison groups, and beliefs surrounding positive symptoms improved in CBT more than in comparison groups.

14.2.1.3 Theory of the disorder

Researchers have proposed a variety of theories to explain the development and maintenance of psychosis. Meehl (1962) first advanced the stress-vulnerability model and argued that genetic predispositions and stressful life events interact to produce the symptoms of schizophrenia. The CBT model asserts that distress associated with

schizophrenia can be alleviated through reinterpretation of symptoms, their meaning, and their consequences (Jones et al., 2012). Thus, CBT is aligned with the stress-vulnerability model in that it seeks to alleviate stress that exacerbates symptoms, rather than eliminate the symptoms themselves, which are thought to be the product mainly of biological predispositions. As psychosocial stress and biological symptoms interact, CBT seeks first to eliminate the psychosocial stress, with the assumption that associated biological symptoms will decrease as a result.

Garety and Freeman (2013) proposed that the following factors contribute to the development of delusions: reasoning and information-processing biases, beliefs about the self and others, and emotional and social stressors (e.g., stigma). Individuals with schizophrenia also display cognitive deficits, which can lead to faulty information-processing, including reasoning, attributional biases, jumping to conclusions, and belief inflexibility (Mander & Kingdon, 2015). Delusions are maintained by a reasoning bias called "jumping to conclusions" (JTC) that involves relying on faulty or scarce data and failing to consider alternative explanations for delusory beliefs (Garety et al., 2015; Kuipers et al., 2006). According to Garety et al. (2015), links exists among negative symptoms, JTC, and poor working memory. Further, JTC is related to a poorer prognosis (Garety et al., 2015; Kuipers et al., 2006).

In CBT, delusions are thought to serve protective or adaptive functions (e.g., delusions of grandeur provide a sense of importance) and hallucinations stem from or are exacerbated by significant life stressors (Kuipers et al., 2006). Kuipers et al. (2006) reported that individuals with psychosis are more likely to externalize negative events onto others, as opposed to blaming themselves. Kuipers et al. (2006) also propose a "self-monitoring problem," which can engender delusions of control and hallucinations, and suggest that increased dopamine can amplify the salience of events, thereby increasing the likelihood that they will be interpreted erroneously as ideas of reference (Kapur, 2003; Kuipers et al., 2006).

Freeman and Garety (1999) proposed that anxiety, particularly "meta-worry" (i.e., worry about worry; fear of loss of control of thoughts, persecutory delusions; Wells, 1995), maintains psychosis and is therefore a worthy target of CBT. Freeman and Garety (1999) reported that, as distress regarding delusions increased, so did meta-worry. Moreover, anxiety often exacerbates psychotic symptoms and can fuel the search for solutions to problems via delusional explanations (Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001; Slade, 1972).

Additionally, social isolation increases the conviction of delusions and reduces the likelihood of considering alternative explanations (Garety et al., 2001; Looijestijn, Blom, Aleman, Hoek, and Goekoop, 2015; Selten, Van der Ven, Rutten, & Cantor-Graae, 2013). Low self-esteem, typically present in psychosis, further promotes maladaptive beliefs and assumptions concerning the self and others, which can devolve into degrading hallucinations and negative delusions (Close & Garety, 1998; Garety et al., 2001). Garety et al. (2001) contended that positive symptoms (e.g., delusions and hallucinations) can develop in a manner delineated by the cognitive and affective model and the affective disturbance model. The cognitive and affective model postulates that cognitive processes are disturbed when an individual with a biological predisposition for psychotic symptoms encounters a triggering event (similar to the stress-vulnerability model proposed by Meehl [1962]) that engenders sensory disturbances, with material from memory intrusively entering consciousness.

The affective disturbance model holds that psychosis develops when negative emotions exacerbate specific cognitive deficits (e.g., JTC, externalizing attributional biases, deficits in theory of mind) that trigger auditory hallucinations, for example (Garety et al., 2001). Further, adversity in childhood engenders negative self-schemas, which can increase the likelihood of the development of psychosis (Birchwood, Meaden, Trower, Gilbert, & Plaistow, 2000). Finally, stressors that accompany a diagnosis of schizophrenia, such as stigma and negative self-expectancies, can promote secondary depression (Garety et al., 2001).

Looijestijn et al. (2015) proposed a similar developmental model to explain hallucinations. The authors refer to the "social-defeat hypothesis" (Selten et al., 2013), which links social isolation to increased dopamine activity, which, in turn, correlates with increased risk for psychosis. Moreover, increased social isolation is a significant stressor that further increases the probability of psychosis through what the authors term "stress-induced false-positive identification of threats," wherein faulty attractor networks (or networks responsible for perception) and deficiencies in GABA and NMDA engender delusions and hallucinations. "Noradrenergic stimulation" (e.g., increased dopamine) by stress triggers alterations in the "nose-to-signal ratios" (e.g., enhanced and unreality-based threat perception) due to an accentuation of an inherent bias toward threat within human cognitive networks (to prepare humans for threat).

Bentall (1990, 2013) proposed that hallucinations can be triggered or exacerbated by stress, biological predispositions, social isolation/sensory deprivation, suggestion, reinforcement, intellectual deficits in language processing, and specific environmental stimulation (e.g., Barber, 1970; Bentall, 1990; Slade, 1976; Tarrier, 1987). Due to impaired reality-testing, individuals fail to identify a hallucination as occurring within themselves, as opposed to an external source, yielding to questions regarding whether thoughts are out of their control or whether they could be broadcast for others to hear (Bentall, 1990). Mintz and Alpert (1972) evaluated this hypothesis using the "White Christmas" test, in which participants are asked to imagine the song "White Christmas" being played in their minds. Participants who reported auditory hallucinations were more likely to report having actually heard the song "White Christmas" being played, and more likely to believe that it was actually played, compared with healthy participants.

Bentall (1990) also contended that hallucinators may experience a bias for sensory stimuli such that they overreport the experience of a stimulus (Bentall, 1990). Further, "seepage" theory proposes that hallucinations are produced by "seepages" from unconscious to conscious awareness, and reflect a deficit in the filter between the unconscious and conscious contents (Bentall, 1990). Another possibility is that hallucinations are manifestations of subvocalizations of words, but this theory fails to explain hallucinations other than auditory hallucinations (Bentall, 1990; Done, Frith, & Owens, 1986). Nevertheless, tasks such as reading, writing, and other tasks involving verbal ability suppress auditory hallucinations, perhaps because they interfere with subvocalizations that develop into hallucinations (Bentall, 1990; Margo, Helmsley, & Slade, 1981; Slade, 1974). Further, Morrison and Haddock (1997) found that hallucinations are associated with increased self-focus, so increased attention directed to psychological processes could increase hallucinations.

A common thread that joins the theories we have reviewed is that interpreting hallucinations as arising from an external source evokes distress (Bentall, 1990; Morrison, 1998; Morrison & Renton, 2001), including anxious, paranoid, angry reactions that engender

maladaptive coping behaviors and relapse and exacerbate persecutory delusions (Birchwood, 1995; Gumley & Power, 2000).

14.2.1.4 Theory of change

Given this theoretical backdrop, it is not surprising that distress, negative interpretations, and cognitive biases are focal points of CBT for schizophrenia (Garety et al., 2001; Mander & Kingdon, 2015). For example, CBT uses cognitive restructuring to address negative symptoms hypothesized to be mediated by poor self-efficacy and defeatist beliefs (Dickerson & Lehman, 2011). However, very few studies to date have conducted mediational analyses on potential mechanisms of change. The preliminary findings are as follows.

Davis, Eicher, and Lysaker (2011) examined differences between CBT and supportive psychotherapy among 62 outpatients with schizophrenia and schizoaffective disorder over 26 sessions delivered once weekly. CBT was partly aimed at improving metacognition (i.e., the ability to think about one's thought processes). The researchers found that metacognition was correlated with the therapeutic alliance and that a successful alliance predicted improvements in symptoms, performance during rehabilitation, and quality of life. Further, improvements in metacognition mediated the relation between neurocognitive deficits and outcome, such that metacognitive skills could compensate for the debilitating effects of neurocognitive deficits (Davis et al., 2011).

Evidence suggests that CBT effectively militates against harmful, negative expectancies and defeatist beliefs that act as self-fulfilling prophecies, as some individuals do not initiate potentially rewarding activities due to their beliefs that they will fail if they attempt to do so (Granholm, Holden, Link, McQuaid & Jeste, 2013). Granholm et al. (2013) examined whether addressing defeatist beliefs improved outcomes among 79 outpatients with schizophrenia and schizoaffective disorder randomly assigned to 36 weeks of either group CBT with SST or goal-focused supportive group therapy. The researchers found that addressing defeatist beliefs in the group CBT condition, through thought-challenging, problem-solving, and SST, was effective in reducing defeatist beliefs in patients with severe defeatist attitudes, whereas the comparison condition was effective in helping patients with less severe defeatist beliefs (Granholm et al., 2013). Importantly, reduction of defeatist beliefs significantly predicted improvement in self-reported everyday functioning for CBT plus SST at 18 months, whereas this did not occur for the supportive group therapy condition. This finding provides evidence that improving defeatist beliefs is a mechanism of change operative in CBT (albeit combined with SST). The authors argue that the CBT component may have been responsible for this change, whereas SST, which addressed social competence, could act as an independent mediator of treatment gains (Granholm et al., 2013).

To further corroborate the findings that change in defeatist beliefs mediates symptom outcome, Grant and Beck (2009) conducted an assessment study of 77 individuals with schizophrenia or schizoaffective disorder in which participants completed a battery of cognitive tasks. The tasks included tests of memory, processing speed, and attention. Participants were also assessed for defeatist beliefs, functioning, and psychosis symptom severity. Grant and Beck (2009) conduced mediational analyses and discovered that defeatist beliefs mediated the impact of cognitive impairment on overall functioning and negative symptoms. Identifying defeatist beliefs as a mediator further validates the use of CBT for schizophrenia, as it addresses defeatist beliefs.

Turkington et al. (2008) also addressed negative symptoms, such as blunted affect and nonresponsiveness (Scale for the Assessment of Negative Symptoms; Andreasen, 1989) in a study examining CBT versus befriending. CBT included activities aimed at either directly improving functioning (i.e., activity scheduling, voice diaries) or using cognitive–behavioral techniques indirectly (i.e., alternative explanations of origin of voices, normalizing of symptoms). The researchers reported decrements in negative symptoms in the CBT condition and postulated that the activities in the CBT condition led to a "switching on of the prefrontal cortex," although the researchers provided no psychophysiological evidence for this claim. The authors further propose that CBT also may have reduced stigma, which facilitated social contact (Turkington et al., 2008).

Freeman et al. (2015) reported that CBT may reduce delusions by diminishing worry associated with delusions. In this blinded study, 150 participants, mostly outpatients with psychosis, were randomized to either CBT or standard care. CBT significantly decreased worry and persecutory delusions compared with standard care at 8 weeks and 24 weeks. Reduction in worry accounted for 66% of the improvements seen in delusions based on a mediation analysis (Freeman et al., 2015).

Gaudiano, Herbert, and Hayes (2010) used mediation analyses to study mechanisms of change for a particular subtype of CBT (ACT) in inpatients with psychosis. Participants were assigned to either ACT or TAU, and ACT was shown to be significantly superior to TAU. Believability of hallucinations significantly mediated the relationship at posttreatment between treatment condition and hallucination distress. More specifically, participants assigned to ACT showed significant improvement in distress over TAU, and this was partially attributed to ACT's ability to alter believability of hallucinations.

In summary, CBT reduces hallucinations by modifying beliefs regarding their origins and the perceived reality of the hallucinations. When conviction in hallucinations diminishes, accompanying distress, maladaptive behaviors, and frequency of hallucinatory symptoms are reduced (Dickerson, 2000; Chadwick & Birchwood, 1994; Chadwick & Lowe, 1990).

Moreover, using CBT focusing and/or attribution techniques can reduce hallucinations and the accompanying distress. An RCT of 34 patients with schizophrenia with hallucinations showed that frequency and distress surrounding auditory hallucinations were reduced in both conditions, one using distraction and another using focusing (Dickerson, 2000; Haddock, Slade, Bentall, Reid, & Faragher, 1998). Therapy was conducted over 20 hour-long sessions. Self-esteem was improved more in the focusing treatment condition. These gains were not maintained at the 2-year follow-up, assessors were not blinded to the condition, and the study lacked a no-treatment, waitlist, or placebo comparison condition. CBT appears to be effective by improving reasoning abilities, such that individuals are given the skills to not use a JTC bias, to consider alternative explanations, to use reasoning skills to avoid interpreting neutral events as malicious, and to avoid feelings of loss of control over their thoughts and actions (de Jong et al., 2014).

Very few studies have analyzed brain mechanisms associated with treatment change; only two studies to date have done so. Kumari et al. (2011) reported that 22 patients with schizophrenia exhibited decreased neural responses to threatening stimuli following 6–8 months of CBT. Further, Mason, Peters, Dima, Williams, and Kumari (2015) documented increased connectivity in brain regions responsible for top-down

regulation and in brain areas associated with decreased persecutory beliefs (i.e., the amygdala and the inferior parietal lobule). Candida et al. (2016) suggested that these findings implicate CBT's role in emotion regulation and decreased reactivity to threat.

14.2.1.5 Summary

Meta-analyses, qualitative reviews, and individual studies provide no consensus regarding whether CBT is superior to other interventions. Overall, effect sizes are modest when comparing CBT to a control group, ranging from 0.38 for positive symptoms (Wykes et al., 2008) to −0.08 (Lynch et al., 2010; see Section 14.2.1.2.1 for more data), and further research is needed to identify the core components of treatments that are most effective. Moreover, research has generally not controlled for nonspecific factors and evaluated treatment gains over relatively long time intervals. The heterogeneity of disorders and treatment packages in studies is an issue as well, and research variously compares CBT with TAU, other interventions, or waitlist. Variability in specific and likely nonspecific factors renders it difficult to ascertain change mechanisms responsible for treatment gains. It is also difficult to (1) confirm CBT's superiority over alternative treatments, as comparative studies are lacking, and (2) pinpoint change mechanisms of CBT when variability exists in delivery and comparison groups across studies.

Researchers have documented mixed findings for CBT with respect to comparison conditions (e.g., standard care, TAU; Durham et al., 2003; Rector et al., 2003; Turkington et al., 2008; Valmaggia et al., 2005), but, overall, multiple meta-analyses and reviews have concluded that CBT produces superior outcomes in treating positive, negative, and overall symptomatology for psychosis (Jauhar et al., 2014; Sarin et al., 2011; Wykes et al., 2008). Based on David and Montgomery's (2011) taxonomy of empirically supported psychotherapies, CBT for psychosis can be considered Category II, because CBT's therapeutic package has been empirically validated by at least two different investigators or investigating teams (Freeman et al., 2015; Penn et al., 2009; Turkington, Kingdon, & Weiden, 2006). That said, there is significant variability among studies in terms of outcomes and when treatment effects are manifested and the longevity of treatment effects at follow-up is questionable, so this categorization should be regarded as tentative, and we must await further research for more definitive conclusions. Additionally, the mechanisms of change that potentially moderate or mediate CBT-related treatment gains have not been ascertained with certainty, and very few studies have focused on proposed mechanisms of change (Freeman et al., 2015). We therefore regard the literature on mechanisms of change as promising yet preliminary.

14.2.2 Social Skills and Social Cognition Training

SST is a behavioral therapy that was developed in the 1970s, and it has changed little since its inception. SST is typically administered in a 45–90-minute group format of 4–12 patients, facilitated by two bachelor's- or master's-level clinicians. Groups typically meet one to five times per week (Kopelowicz, Liberman, & Zarate, 2006). SST aims to improve social competence, including social perception, social cognition (i.e., ability to accurately assess emotional displays of others), processing of social information (i.e., ability to correctly interpret words/actions of others), and appropriate displays of expression (Bennett & Bellack, 2015; Kopelowicz et al., 2006). Other skills SST teaches

include affiliative skills (i.e., appropriate self-disclosure), interactional skills (i.e., initiating and maintaining a conversation), instrumental role skills (i.e., learning how to function in society), and how to behave according to social norms (Kopelowicz et al., 2006). Overall, SST for schizophrenia aims to improve abilities to assess others, process what is occurring in a social interaction properly, and respond appropriately (Pfammatter, Junghan, & Brenner, 2006).

Almost all research to date is based on Liberman et al.'s (1993) Social and Independent Living Skills training modules (Pfammatter et al., 2006). SST is intended to be administered alongside case management and pharmacotherapy. The therapy uses behavioral principles of learning by repeated rehearsal of real-world scenarios that are thought to translate to everyday life. Therapists model appropriate social behavior and provide feedback to patients in a group context. Therapy also can involve homework, goal-setting, and problem-solving (Kopelowicz et al., 2006). SST addresses social skills deficits in psychosis specifically, and is designed to be implemented directly into patients' lives in a straightforward, practical manner (Bennett & Bellack, 2015). Activities can involve role-playing scenarios of job interviews, refusing drugs, starting a conversation with a stranger, and initiating safe sex.

14.2.2.1 Evidence for the therapy

14.2.2.1.1 Reviews

The PORT recommendation, which uses two "evidence review groups" to identify evidence-based treatments (Kreyenbuhl et al., 2010), has recommended SST as an evidence-based treatment for schizophrenia. Further, the APA (APA Presidential Task Force on Evidenced-Based Practice, 2006) has listed SST as an evidence-based treatment.

Bellack (2002) reviewed four meta-analyses and eight reviews and concluded that SST exerts significant effects on improving behavioral skills and improves social functioning, patient satisfaction, and self-efficacy. However, the author notes that the reviews arrived at inconsistent conclusions. The author also concluded that SST produces no lasting positive effects on symptomatology or relapse prevention.

Pilling et al. (2002) reported little evidence for the efficacy of SST on relapse reduction, dropout rates, global adjustment measures, social functioning, or overall quality of life. The meta-analysis included only RCTs (not quasi-experimental) that were limited to individuals with psychotic disorders.

Kopelowicz et al.'s (2006) review of SST concluded that SST significantly improves patients' social skills for up to 2 years. Nevertheless, the authors noted that the results are "discouraging" with regard to transfer and generalization of treatment gains to the real world.

Pfammatter et al. (2006) performed a meta-analysis on 19 RCTs that included observational and quasi-experimental studies, and concluded that SST significantly improves social functioning ($g = 0.32$), general symptoms ($g = 0.47$), and assertiveness ($g = 0.43$). The reviewers also reported that two RCTs demonstrated "considerably" decreased rehospitalization rates during the follow-up period ($g = 0.47$; Benton & Schroeder, 1990; Corrigan, 1991).

Kurtz and Mueser (2008) conducted a meta-analysis of 23 RCTs with schizophrenic and schizoaffective patients and evaluated proximal and distal effects of training and participant variables. The reviewers found that patients were most affected proximally,

with content mastery of skills evidencing the strongest effect (Cohen's $d = 1.2$). Participants also improved in social and independent functioning ($d = 0.52$), "psychosocial functioning" ($d = 0.52$), and negative symptoms ($d = 0.4$).

A recent Cochrane Review (Almerie et al., 2015) evaluated RCTs that compared SST with TAU or "discussion groups," which involve discussion of heterogeneous topics. The authors noted that most of the clinical trials were conducted in China and across seven outcome measures; the trials found significant differences between SST and TAU in favor of SST in terms of social functioning, relapse rates up to 12 months, rehospitalization rates, negative and positive symptom improvement, general symptoms, and overall functioning. No significant differences were seen for early treatment dropout, employment, or quality of life compared with TAU. No significant differences were reported between SST and discussion groups for social functioning, relapse, overall functioning, mental state, early treatment dropout, or quality of life. Thus, the authors concluded that there is "very low quality" evidence to support SST. Multiple reviews suggest that SST improves functional and social skills, although concerns abide regarding the generalization of skills to everyday life.

14.2.2.1.2 Randomized controlled trials

Researchers have conducted many RCTs for SST, mostly during the 1990s. We discuss a number of exemplary RCTs (see Table 14.2 for a summary). A variety of studies combine SST and another treatment modality. For example, CBT and SST can be combined to create cognitive–behavioral social skills training (CBSST). Granholm, Ben-Zeev, and Link (2009) developed CBSST for treating middle- and older-aged adults. Very few trials to date have assessed CBSST to determine its comparability to regular SST, but CBSST has shown initially promising results in improving functional outcomes (Granholm et al., 2009, 2013). We have excluded such "combined" (apart from coadministered medication) studies to more precisely isolate the effects of SST. Combining SST with a CBT-based treatment appears to amplify the positive effects on social functioning (Pfammatter et al., 2006); thus, combining CBT and SST may be better than either alone.

Eckman et al. (1992) compared SST with supportive group psychotherapy. Researchers were "generally blind" to the condition. Participants were trained on two modules from the University of California, Los Angeles' Social and Independent Living Skills Program, which taught medication and symptom self-management twice weekly over 6 months. Medication and symptom self-management referred to patients' ability to complete tasks such as obtaining transportation, seeking treatment when noticing warning signs of relapse, and speaking about medication concerns with their treatment providers. Raters assessed patients on their ability to manage medication and symptoms through patients' performance in role-plays. Individuals assigned to SST showed significantly improved skills (e.g., medication and symptom self-management) 6 months after the intervention, whereas participants in supportive group therapy did not evidence comparable gains. Differences between the SST group and the comparison condition with respect to symptoms failed to achieve significance. Negative symptoms did not decrease over the 1.5-year assessment period (Eckman et al., 1992).

Glynn et al. (2002) compared "clinic-based" skills training with clinic-based skills training with additional "in vivo amplified skills training" to facilitate generalization of skills to the community. Participants also received either haloperidol or risperidone;

Table 14.2 Summary of social skills training randomized controlled trials included in this review.

Authors and Year	*N*	Experimental Group	Comparison Group	Sessions/Weeks of Treatment	Absolute Efficacy (vs. Comparison)	Relative Efficacy	Specific Efficacy
Eckman et al. (1992)	41 male outpatient veterans with schizophrenia	SST	Supportive group psychotherapy	Twice weekly/ 6 months	Superior to control in improvement of skills (medication and symptom management). No difference in sxs reduction.		
Glynn et al. (2002)	63 outpatients with schizophrenia or schizoaffective disorder	Clinic-based skills training + in vivo amplified skills training	Clinic-based skills training	60 weeks	Superior to control in role functioning and family relationships.		
Horan et al. (2009)	31 outpatients with schizophrenia or schizoaffective disorder	Social cognitive skills training	Illness/relapse management skills group	12 sessions	Superior to control in perception of facial affect. Both groups improved in neurocognitive abilities.		
Rus-Calafell, Gutiérrez-Maldonado, Ortega-Bravo, Ribas-Sabaté, & Caqueo Urízar (2013)	31 outpatients with schizophrenia or schizoaffective disorder aged between 18 and 55	SST	TAU	16 sessions	Superior to TAU in improvement of negative sxs and overall functioning.		
Davis (2014)	27 male outpatients with schizophrenia	Social cognitive skills training + oxytocin	Social cognitive skills training + placebo	12 sessions			Improvement in empathic accuracy.

SST = social skills training; sxs = symptoms; TAU = treatment as usual.

assessors were naive to medication allocation. Individuals assigned to the SST with in vivo training showed significantly improved "instrumental role functioning" and close family relationships as measured by the Social Adjustment Scale–II (Schooler, Hogarty, & Wiessman, 1979). Participants assigned to this group also showed faster improvement across 60 weeks. Medication did not produce significant results.

Horan et al. (2009) compared "social cognitive skills training" with a comparison group who were taught illness and relapse management skills. Social cognitive skills training provided training in facial affect and nonverbal communication perception, as well as discussion regarding how paranoia impacts perceptions of others' intentions toward the participant (Horan et al., 2009). The experimental condition showed significant improvements in perception of facial affect using a facial emotion identification test, with an effect size of $d = 0.21$ between groups. A significant change was not evident in the comparison condition. Both groups improved in neurocognitive abilities, as measured by the MATRICS Consensus Cognitive Battery (Nuechterlein & Green, 2006). Only assessors of attributional style, using the Ambiguous Intentions Hostility Questionnaire (Combs, Penn, Wicher, & Waldheter, 2007), were naive to condition (Horan et al., 2009).

The same research group conducted a double-blind placebo-controlled study comparing social cognitive skills training augmented with oxytocin nasal spray to an otherwise identical placebo nasal spray. Both oxytocin and placebo were administered 30 minutes prior to each of the 12 sessions. This study found that administration of oxytocin enhanced the treatment's effectiveness, as measured by significant improvement over placebo in empathic accuracy (the ability to accurately assess how another individual may be feeling). These effects persisted for 1 month (Davis et al., 2014).

Rus-Calafell et al. (2013) compared SST and TAU and found that the SST condition showed significant improvements over TAU in reduction of negative symptoms, as measured by the PANSS, and improvements in functioning (withdrawal and interpersonal communication), as measured by the Social Functioning Scale (Birchwood, Smith, Cochrane, Wetton, & Copestake, 1990). These gains were also present at a 6-month follow-up. These findings contrast with those of Eckman et al. (1992), whose participants did not show reductions in negative symptoms during a 1.5-year follow-up.

14.2.2.2 Theory of the disorder

SST operates on the premise that schizophrenia engenders debilitating impairments in social cognition, including processing of emotion, perception abilities, theory of mind, and attributional biases (Horan et al., 2009) that produce social isolation (Rus-Calafell et al., 2013). Individuals with schizophrenia may lack the ability to integrate prior and current events, and thus lack the ability to use prior experiences to inform current behaviors. Further, social skills deficits are common in schizophrenia-spectrum disorders, including difficulties in communicating, navigating conflicts, expressing opinions, and forming social connections with new people (Ottavi et al., 2014).

14.2.2.3 Theory of change

SST does not directly target symptoms but rather instills life skills with the assumption that improving social conditions, problem-solving, and mitigating social stress will alleviate symptoms and enhance effective communication, assertiveness, and self-efficacy (Bennett & Bellack, 2015; Kopelowicz et al., 2006). Proponents of SST argue that neurocognitive deficits can be mediated by social skills. Indeed, research suggests that social

cognition mediates the impact of neurocognitive deficits on overall functioning (Brekke, Kay, Lee, & Green, 2005; Horan et al., 2009) and neurocognitive deficits moderate social competence (Bellack, 2002). Further, SST has proven effective in facilitating patients' abilities to medicate themselves, which increases perceptions of responsibility and control over their illness and increases insight into their condition (Day et al., 2005; Eckman, Liberman, Phipps, & Blair, 1990; Kopelowicz et al., 2006). Researchers have hypothesized that increases in social competence foster self-esteem, confidence, and understanding of others.

In an unpublished dissertation, Quinlan (2014) conducted a mediational analysis to examine mechanisms of change for CBSST compared with neurocognitive training. Quinlan did not find that improvement in defeatist beliefs mediated the relation between group assignment and outcome variables. Overall, the mechanisms of action for SST are not fully understood and scant mechanistic research has been conducted.

14.2.2.4 Summary

SST can improve social competence for individuals with schizophrenia. The intervention serves as a "practice arena" and a safe space to develop skills before implementing them in the real world. However, as Pfammatter et al. (2006) note, research regarding the transfer of treatment effects to everyday life is slim, and more research is clearly needed. The aforementioned studies provide promising results regarding the efficacy of SST in initiating improvements in negative symptoms, interpersonal relationships, overall functioning, social withdrawal, perception of facial affect, and medication management. Additional research is needed to identify which outcomes persist through follow-up, as studies have yielded varying conclusions.

With regard to David and Montgomery's (2011) taxonomy of effective psychotherapies, SST for psychosis can be regarded as a Category II intervention. Promising results derived from multiple meta-analyses and RCTs boast significant findings for active treatments over comparison conditions on diverse functional outcomes (Glynn et al., 2002; Horan et al., 2009; Rus-Calafell et al., 2013), and multiple reviews and meta-analyses have supported SST as an empirically based treatment (Bellack, 2002; Kurtz & Mueser, 2008; Pfammatter et al., 2006). Thus, SST has well-supported evidence for its therapeutic package from two independent research groups (Horan et al., 2009; Rus-Calafell et al., 2013) and equivocal evidence (preliminary) for its theory (Brekke et al., 2005; Horan et al., 2009), warranting its classification as a Category II treatment in David and Montgomery's (2011) scheme. Nevertheless, concern has been expressed regarding the ability of SST to transfer treatment gains to everyday life. SST's inability to transfer to skills outside of session further complicates the process of categorizing the intervention. Accordingly, the placement of SST in Category II should be taken to be provisional.

14.3 Category IV

Category IV therapies have mixed data on their treatment package and preliminary data on their mechanisms.

14.3.1 Metacognitive Therapy

Although metacognitive therapy (MCT) can be considered under the umbrella of CBT, recent studies suggest that it is often practiced as a single entity, apart from CBT.

Thus, we will discuss the literature on MCT under a separate rubric. The most recent Cochrane Review (Jones et al., 2012) on CBT and other psychosocial treatments and the PORT Review (Kreyenbuhl et al., 2010) have not designated MCT as an empirically based treatment for schizophrenia. In fact, they do not even mention MCT, probably because many reviewers would not consider it a modality distinct from CBT. Further, the APA does not list MCT as an empirically based treatment for schizophrenia (APA Presidential Task Force on Evidence-Based Practice, 2006). Nevertheless, we will review a considerable body of evidence relevant to the effectiveness of this method, as it has been somewhat extensively researched.

Moritz and Woodward (2007) developed MCT as a manualized treatment (available cost-free online) in two modules of eight 1-hour sessions conducted in group format, typically with 4–10 patients (Favrod et al., 2014; Moritz et al., 2013). MCT includes elements of cognitive remediation and CBT (Moritz et al., 2013). Similarly to CBT, it addresses cognitive biases and delusional beliefs, such as JTC; belief inflexibility; overconfidence in memory errors; attributional biases; deficits in theory of mind; and depressive cognitive schemas (Lam et al., 2015; Moritz et al., 2013). MCT differs from other cognitive therapies in that it focuses on the "social cognitive aspect of recovery" as well as metacognition (Moritz, Vitzthum, Randjbar, Veckenstedt, & Woodward, 2010, p. 562), which is the ability to think about thinking and reflect critically on thoughts (Balzan & Galletly, 2015; Moritz et al., 2013). Further, whereas CBT focuses on individuals' specific maladaptive beliefs, MCT targets general cognitive biases, addressing how delusions are formed and what purpose they may serve (Dickerson & Lehman, 2011). For example, therapists provide information on how delusions protect individuals from the negative aspects of psychosis (e.g., delusions of grandeur provide a sense of importance and a shield against low self-image) but are merely short-term coping mechanisms (Moritz et al., 2010). The social cognitive aspect of therapy requires that patients reflect on the consequences of their behaviors for interpersonal relationships (Balzan & Galletly, 2015; Moritz et al., 2013). Participants are also provided with exercises that target biases via corrective experiences that teach coping and information-processing skills and how to incorporate them into everyday life (Balzan & Galletly, 2015; Moritz et al., 2013). For example, Moritz et al. (2010) explain that a common, colloquial "urban legend or myth" might be discussed wherein members, for example, provide evidence for and against the belief that the $1 bill contains hidden messages alluding to secret societies governing the US government. Therapists may also encourage members to exercise "detached mindfulness" (Moritz et al., 2010) to reduce experiential avoidance (e.g., actively avoiding uncomfortable emotions or experiences) and emphasize positive events in daily living to build self-esteem (Moritz et al., 2010). To avert a psychotic episode, MCT aims to increase awareness of cognitive biases and cultivate problem-solving skills to foster metacognitive awareness, including the ability to think cogently about mental activities as they emerge.

14.3.1.1 Evidence for the therapy

14.3.1.1.1 Reviews

Dickerson and Lehman (2011) concluded that only CBT can be considered an evidence-based treatment for schizophrenia and that MCT is still in its initial phases. Nevertheless, they note that MCT is promising, as it is readily accepted by patients, feasible and cost-effective to conduct, and reduces symptoms and cognitive biases

while it enhances self-efficacy. Pankowski, Kowalsi, and Gawęda's (2016) recent review provided an even more sanguine assessment of the literature (2009–2015) in their evaluation of 14 trials in which researchers compared MCT with an active comparison condition. Of the studies reviewed, 11 were RCTs, and most included assessors blinded to treatment condition. These authors reported the greatest improvements for delusions, their associated distress, and level of conviction held regarding delusions, implying that MCT moderates cognitive biases, including JTC, catastrophizing, and emotion-based reasoning (Pankowski et al., 2016). Positive effects were evident for up to 6 months, although MCT exerted a smaller effect on hallucinations. The review also reported that, in some studies, MCT improved neurocognitive abilities such as attention and memory. Finally, Pankowski et al. (2016) concluded that studies showed improvement in insight, and one study documented treatment gains in the large effect size range (Gawęda, Krężołek, Olbryś, Turska, & Kokoszka, 2015).

14.3.1.1.2 Randomized controlled trials

The following RCTs summarize major findings in the literature for MCT for psychosis. Discrepancies in the literature are evident, as the following findings suggest; discrepancies in findings may be attributable to the high variability in administration of MCT, iterations of MCT, comparison groups, administration techniques, and "dosages" of treatment. See Table 14.3 for a summary.

Aghotor, Pfueller, Moritz, Weisbrod, and Roesch-Ely (2010) compared 4 weeks of twice-weekly group MCT to a once-weekly active comparison condition. The active comparison group simply discussed newspaper stories. The comparison condition was intended to improve social interactions, thought blocking, and cognitive abilities. Assessors were naive to condition. Patients were assessed on the PANSS and a computer task to measure JTC. MCT showed improvements over the comparison group in all PANSS subscales. Participants in MCT showed improvements on the PANSS positive subscale with a medium effect size ($d = 0.43$), and a small effect size ($d = 0.24$) with respect to the total score. Participants in MCT improved on the JTC task but the effect did not achieve significance (Aghotor et al., 2010).

Kumar et al. (2010) examined group MCT compared with TAU. The TAU group attended the groups regularly held by the inpatient unit. MCT was held twice weekly for 4 weeks. The researchers found that MCT was superior to TAU in improvement of positive symptoms as measured by the PANSS, and, though this difference was not significant, medium to large effect sizes were detected between groups ($d = 0.68$; Kumar et al., 2010).

Moritz et al. (2013) conducted a study comparing group MCT to a cognitive remediation comparison group. The participants were assessed immediately after treatment, at 4 weeks posttreatment, and at a follow-up of 6 months. Participants assigned to the experimental condition were provided with 45–60-minute sessions of a manualized treatment. Researcher assessors were blinded to conditions. The participants were administered cognitive remediation through a computerized program called CogPack, which included a battery of neuropsychological tests, including on memory and selective attention, and the participants were provided with feedback at the end of each session. Participants in MCT showed significantly improved symptomatology as compared with cognitive remediation, as measured by the PANSS Delusion subscale at follow-up, the PANSS Positive subscale at posttreatment, and the PSYRATS Delusion subscale at

Table 14.3 Summary of metacognitive therapy randomized controlled trials included in this review.

Authors and Year	*N*	Experimental Group	Comparison Group	Sessions/Weeks of Treatment	Absolute Efficacy (vs. Comparison)	Relative Efficacy	Specific Efficacy
Aghotor, Pfueller, Moritz, Weisbrod, & Roesch-Ely (2010)	30 inpatients with a schizophrenia-spectrum disorder with prior or current delusions	MCT	Active comparison Group	Twice weekly/ 4 weeks	Superior to comparison group in positive/negative sxs. Improvement on "jumping to conclusions" task (not significant).		
Kumar et al. (2010)	16 male inpatients with paranoid schizophrenia	MCT	TAU	Twice weekly/ 4 weeks	Superior to TAU in positive sxs (not significant).		
Moritz et al. (2013)	150 in/outpatients with schizophrenia-spectrum disorders	MCT	Cognitive remediation	4–8 weeks	Superior to comparison in positive sxs. Comparison superior in skills in immediate memory recall. No difference in negative sxs.		
Favrod et al. (2014)	52 participants with schizophrenia or schizoaffective disorder	MCT+TAU	TAU	2 months	Superior to TAU in positive sxs.		
Briki et al. (2014)	50 in/outpatients with schizophrenia-spectrum disorders	MCT	Supportive therapy (active verbal therapy group)	Twice weekly/ 8 weeks	Superior to comparison in positive sxs. Improvement in awareness and attribution of hallucinations over comparison (not significant).		
Kuokkanen, Lappalainen, Repo-Tiihonen, & Tiihonen (2014)	33 male inpatients with schizophrenia with a history of violence	MCT	TAU	8 sessions twice weekly	Superior to control in positive sxs. Not significant at 6 months. TAU worsened sxs.		
Van Oosterhout et al. (2014)	111 patients (mostly outpatients) with a schizophrenia-spectrum disorder	MCT	MCT+TAU	8 sessions twice weekly	Both groups improved in overall sxs (not significant differences). TAU superior to TAU in paranoid delusions.		
Lam et al. (2015)	80 in/outpatients with schizophrenia-spectrum disorders	MCT	TAU (general treatment with an occupational therapist)	Twice weekly/ 8 weeks	Superior to TAU in self-reflectiveness and insight. No differences in self-certainty.		

MCT = metacognitive therapy; sxs = symptoms; TAU = treatment as usual.

both posttreatment and follow-up (Moritz et al., 2013). The researchers reported that attendance in MCT groups and improvement on the PANSS Positive subscale were correlated. Further, MCT participants reported increases in self-esteem and improvements in communicating with others. The comparison condition significantly improved skills in immediate memory recall at 4 weeks, but this gain was not maintained at follow-up. Neither intervention affected negative symptoms (Moritz et al., 2013).

Favrod et al. (2014) examined differences between group MCT with TAU and TAU alone. The participants were assessed with naive raters at 2 and 6 months posttreatment. Significant findings for the MCT plus TAU group were seen for the PSYRATS Delusion subscale and the PANSS Positive subscale at both 2 and 6 months compared with TAU alone.

Briki et al. (2014) compared group MCT with supportive therapy. The assessors were naive to treatment condition. Individuals in the MCT group showed significant improvement over supportive therapy on the PANSS Positive subscale with a medium effect size ($d = -0.61$). Insight awareness and attribution scores for hallucinations, as measured by the Scale to Assess Unawareness of Mental Disorder (Amador et al., 1993), did not improve over supportive therapy, although the differences trended toward significance.

Kuokkanen, Lappalainen, Repo-Tiihonen, and Tiihonen (2014) compared group MCT to TAU. Individuals in the MCT condition showed significant improvements over TAU in measures of suspiciousness, as measured by the PANSS Suspiciousness and PANSS Sum scores, with the greatest improvements at 3 months posttreatment, although the findings did not replicate at 6 months. The TAU group showed worsened symptoms from baseline to 3 months (Kuokkanen et al., 2014).

Van Oosterhout et al. (2014) compared group MCT plus TAU to TAU alone. Assessors were naive to condition. Both MCT plus TAU and TAU showed improvement in overall symptomatology, but no significant differences were found. Subjective experiences of delusions and cognitive insight were unchanged as well. In fact, TAU improved significantly over MCT plus TAU on the B subscale of the Green Paranoid Thought Scale (Green et al., 2008), which measures paranoid delusions (Van Oosterhout et al., 2014).

Lam et al. (2015) investigated the effectiveness of MCT compared with TAU (i.e., "general treatment with an occupational therapist"; p. 40). Participants assigned to MCT received twice-weekly sessions for 8 consecutive weeks and exhibited significant improvements compared with TAU in self-reflectiveness as measured by the Beck Cognitive Insight Scale (BCIS; Beck, Baruch, Balter, Steer, & Warman, 2004) and by the BCIS composite score as compared to TAU alone. No significant changes were observed for self-certainty as measured by the BCIS self-certainty subscore (Lam et al., 2015).

14.3.1.1.3 Nonrandomized controlled studies

Gawęda et al. (2015) compared group MCT plus TAU to TAU among 44 outpatients with schizophrenia. Treatment assessors administered the PSYRATS and were naive to condition. MCT was administered twice weekly over 4 weeks. Participants in the MCT plus TAU group showed significant improvements over TAU alone in illness insight, delusion frequency and conviction, and cognitive biases, including catastrophization, emotion-based reasoning, and JTC. Overall symptomatology severity was improved in MCT plus TAU over TAU with a medium effect size ($d = 0.54$), but this did not achieve

significance. Participants in the MCT plus TAU group did not exhibit improvements in attentional abilities, global functioning, or belief flexibility.

Balzan and Galletly (2015) examined the effectiveness of nine individual sessions of MCT+ over 4 weeks with one patient diagnosed with delusional disorder and a second patient diagnosed with schizophrenia. MCT+ combined basic MCT with CBT techniques. Participants did not take antipsychotic medication. Assessors were blind to conditions. MCT improved scores on the PANSS, the PANSS Positive subscale, the PANSS Delusions item, the PSYRATS, and the SAPS Delusion subscales. Participants reported reductions in distress, preoccupation, and belief in delusions (Balzan & Galletly, 2015), although the authors do not note whether these findings, based on reductions in raw scores, were statistically significant.

14.3.1.2 Theory of the disorder

Both MCT and CBT assert that cognitive biases should be addressed, instead of symptoms. However, MCT additionally holds that individuals with schizophrenia exhibit a monocausal attribution style, which Moritz et al. (2010) describe as externalizing blame onto others to protect self-esteem and subsequently isolate from social contacts (Favrod et al., 2014; Moritz et al., 2010). Further, individuals with schizophrenia experience memory deficits, which increase confidence in incorrect or false memories (Moritz et al., 2010). Despite such increases in confidence, individuals with schizophrenia often report being indecisive (Freeman et al., 2006). Their inability to critically analyze thoughts and cognitions increases vulnerability to developing delusions based on faulty reasoning biases discussed previously.

14.3.1.3 Theory of change

The creators of MCT argue that MCT extends beyond CBT in terms of focus on increasing insight into one's illness and metacognitive awareness (Moritz et al., 2010). MCT holds that addressing cognitive biases responsible for transforming beliefs into delusions can treat delusions. Enhanced metacognition increases awareness of cognitive biases and how such biases develop into delusions. Via examples, individuals are taught to think critically to acquire more information before reaching a conclusion and implement logical reasoning processes to minimize cognitive biases and resultant delusions (Aghotor et al., 2010; Moritz et al., 2010; Moritz & Woodward, 2007).

Moritz et al. (2013) reported a correlation between frequency of MCT group attendance and improvements on the PANSS Positive subscale, an outcome they related to the possible acquisition of reasoning skills that facilitated participants' ability to "exercise greater caution in their judgments" (Moritz et al., 2013, p. 67). Although self-esteem increased in the MCT group compared with the comparison condition, this difference did not achieve significance.

MCT also may increase amenability to engage in treatment (Favrod et al., 2014). Menon, Andersen, Quilty, and Woodward (2015) suggest that MCT is effective in reducing delusions because the group setting facilitates discussion of cognitive biases, negative self-appraisals of delusions, and flexibility in changing maladaptive beliefs about delusions.

The literature on the mechanisms of change for MCT in general is sparse, particularly regarding MCT for schizophrenia. Nevertheless, researchers have reported that metacognitive awareness mediates the effect of neurocognitive deficits on social skills

when controlling for symptoms (Lysaker et al., 2010; Van Donkersgoed et al., 2014): Through enhanced metacognition, individuals with schizophrenia may negate the negative impact of neurocognitive deficits on social skills. Moreover, metacognitive awareness benefits social contacts (Lysaker et al., 2013).

Morrison et al. (2014) conducted a pilot trial in which they provided 10 individuals with schizophrenia with 12 sessions of MCT and found that MCT increased metacognitive beliefs and decreased psychotic symptoms significantly. Metacognitive beliefs were measured with the Metacognition Questionnaire–30 (Wells & Cartwright-Hatton, 2004) and psychosis was measured with the PANSS and PSYRATS. Mediational analyses were not conducted to establish temporal precedence, so the authors were unable to conclude that changes in metacognition mediated symptom change.

To the authors' knowledge, mediational analyses have not shown that reductions in JTC mediate change in MCT, as the theory suggests. So et al. (2015) performed mediational analyses, which revealed that, although JTC did not mediate the impact of MCT on psychotic symptoms, belief flexibility mediated the impact of MCT on positive symptoms as measured by the PANSS and PSYRATS.

The mechanisms of change for MCT for psychosis are in need of further research. Overall, the extant findings suggest that increases in metacognitive awareness related to MCT may mediate the impact of neurocognitive deficits on social skills, significantly improve symptoms, and improve belief flexibility. Nevertheless, very few studies have verified these findings, so conclusions about potential mediation must remain tentative.

14.3.1.4 Summary

The studies listed in the preceding sections provide preliminary information to classify MCT as an empirically supported treatment for the positive symptoms of psychosis. It is important to note that many studies framed MCT as a superior treatment without significant results to justify doing so. Multiple researchers from independent laboratories have established MCT's efficacy over comparison conditions in improving positive symptoms of psychosis as measured by the PANSS Positive subscale (Aghotor et al., 2010; Briki et al., 2014; Favrod et al., 2014; Kuokkanen et al., 2014), although this finding was not replicable in all studies (Kumar et al., 2010). MCT is categorized most appropriately as a Category IV treatment in David and Montgomery's (2011) scheme. Although at least two studies (Favrod et al., 2014; Kuokkanen et al., 2014), conducted by independent research teams, have documented the effectiveness of MCT in improving positive symptoms as measured by the PANSS, some studies have shown contradictory (Van Oosterhout et al., 2014) and null (Kumar et al., 2010) findings, and the change mechanisms are not well understood. The future of MCT for schizophrenia holds promise, but further research is necessary to elucidate mechanisms of change. In sum, the theory and therapeutic package of MCT can best be categorized as preliminary, equivocal evidence in support of the effectiveness of the intervention.

14.4 Other Therapies

Other therapies for schizophrenia are more family, vocational, or community oriented than those we reviewed. PORT has recommended the following interventions as

evidence-based treatments for schizophrenia: Assertive Community Treatment, Supported Employment, token economy interventions, and family-based services (Kreyenbuhl et al., 2010). Assertive Community Treatment is described as "intensive case management," and it is aimed at improving functional outcomes for individuals with schizophrenia. This approach shows promise in reducing rehospitalization rates for individuals with serious mental illness (Salkever et al., 1999). Supported Employment, which focuses solely on helping individuals with schizophrenia to obtain and retain work, has shown promising results in helping individuals with schizophrenia to obtain jobs (Lehman et al., 2002). Token economy programs involve setting up behavioral contingencies in one's environment such that behavioral action is met with reinforcers (tokens). Behaviors can include basic activities of living such as hygiene, or participation in work. Interestingly, most research studies on token economy interventions on schizophrenia took place over 20 years ago, but these studies strongly point to token economy's effectiveness in improving functional outcomes as well as symptomatology (Dickerson, Tenhula, & Green-Paden, 2005; Li & Wang, 1994).

Family-based treatments take a systemic approach, employing family members to assist in treating the afflicted family member. Family-based treatment is a broad term for any treatment that encourages contact and emotional support from family members. One form of this treatment is Family Psychoeducation for Schizophrenia, which simply educates family members on the individual's diagnosis and how to best help the family member. This treatment shows promising results in improving symptoms and functional outcomes (Dyck et al., 2000).

Another commonly used treatment, cognitive remediation, focuses solely on improving cognitive functioning. Cognitive skills include attentional abilities, learning, memory, and processing speed. Research on cognitive remediation has also shown very promising findings, with effects lasting up to 6 months posttreatment (Medalia, Ravheim, & Casey, 2001; Wykes et al., 2007).

14.5 Implications for Research

CBT is the intervention with the highest level of evidence-based support, with multiple RCTs and meta-analyses pointing to reductions in symptoms of general psychopathology and positive symptoms through follow-up, and reduced severity in hallucinations at posttreatment (Pfammatter et al., 2006). SST also can boast support for its treatment package. Future studies should evaluate CBT and SST in head-to-head comparisons.

The proposed mechanisms of change have received scant empirical scrutiny. Study of specific mechanisms of action is complicated by ongoing medication changes and poor medication and treatment compliance. Severe psychotic symptoms often interfere with treatment and the ability to transfer treatment gains outside therapy. Moreover, severe symptoms may preclude the ability to generalize the effectiveness of treatment to more severely psychotic individuals. Because there is tremendous heterogeneity among symptoms in psychotic disorders, adopting the Research Domain of Criteria framework, which encourages the study of individuals not by diagnosis but by collections of symptoms (e.g., only people who hear voices), could allow for more accurate identification of mechanisms of action and identification of the most appropriate treatment or treatment combination based on clinical presentation. Future researchers would also do well to

examine schizophrenia independent from other psychotic symptoms and to examine the effectiveness of psychotherapy across the entire range of severity of psychotic symptoms.

We suggest that future studies include comparison conditions that permit examination of the role of nonspecific effects and take care to clearly indicate when findings fail to reach conventional levels of statistical or clinical significance (e.g., change in diagnosis from psychotic to not psychotic or enhanced gains in functioning) and temper conclusions appropriately. Moreover, researchers would do well to account for allegiance effects and ensure that key personnel are blind to treatment conditions when appropriate. Examining brain changes associated with treatment gains is a fruitful area for research on correlates and mediators of successful interventions.

14.6 Implications for Clinical Practice

As discussed, heterogeneity in symptoms complicates the identification of optimally effective treatments for a given patient. Fortunately, CBT is effective in improving the lives of many individuals with psychosis, due largely to reductions in distress. Individuals with delusions often hold strong convictions regarding their beliefs, as they serve a functional role in their lives (e.g., delusions of grandeur engender beliefs in self-importance), even as delusions elicit significant distress. Thus, care should be exercised in dismantling such beliefs and replacing them with a sense of purpose derived from activities that foster self-esteem, such as employment, volunteer work, community engagement, hobbies, or increased social activities. Further, some individuals in the patient's social sphere may not empathize properly with the patient, due to lack of patience and understanding. Accordingly, it is imperative that the therapeutic alliance provides patients with empathy and positive reinforcement.

14.7 Conclusion

Schizophrenia is characterized by debilitating symptoms. Research on schizophrenia has produced quite promising results in improving functional and symptomatic outcomes, but evaluating the success of even the most effective interventions is complicated by the complexity of the disorder and the heterogeneity that presents in its population. Nevertheless, people with schizophrenia are in great need of social, emotional, occupational, and physical support, so treatment research for schizophrenia should persist despite the complexity of doing so. Symptoms of psychosis, including auditory hallucinations and persistent delusions, can serve as barriers to treatment, not evident in other psychological ailments, and lower treatment compliance and willingness to enter treatment in the first place. Psychotherapy process and outcome research will therefore likely gravitate toward technology-based interventions capable of reaching isolated and functionally impaired individuals. In paving these future paths for the betterment of individuals with serious mental illness, considerations must be made for their profoundly distressing symptoms, but extreme care must be taken not to allow societally based, stigmatizing biases against the mentally ill to dictate how and to what extent empirically based treatments will be implemented.

References

Aghotor, J., Pfueller, U., Moritz, S., Weisbrod, M., & Roesch-Ely, D. (2010). Metacognitive training for patients with schizophrenia (MCT): Feasibility and preliminary evidence for its efficacy. *Journal of Behavior Therapy and Experimental Psychiatry*, *41*(3), 207–211. doi:10.1016/j.jbtep.2010.01.004

Almerie, M. Q., Okba Al Marhi, M., Jawoosh, M., Alsabbagh, M., Matar, H. E., Maayan, N., & Bergman, H. (2015). Social skills programmes for schizophrenia. *Cochrane Database Systematic Review*, *6*, CD009006. doi:10.1002/14651858.CD009006.pub2

Amador, X. F., Strauss, D. H., Yale, S. A., Flaum, M. M., Endicott, J., & Gorman, J. M. (1993). Assessment of insight in psychosis. *American Journal of Psychiatry*, *150*, 873–879. doi:10.1176/ajp.150.6.873

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.

American Psychological Association Presidential Task Force on Evidence-Based Practice. (2006). Evidence-based practice in psychology. *American Psychologist*, *61*, 271–285. doi:10.1037/0003-066x.61.4.271

Andreasen, N. C. (1989). Scale for the Assessment of Negative Symptoms (SANS). *British Journal of Psychiatry*, *155*(7), 53–58.

Bach, P., & Hayes, S. C. (2002). The use of acceptance and commitment therapy to prevent the rehospitalization of psychotic patients: A randomized controlled trial. *Journal of Consulting and Clinical Psychology*, *70*(5), 1129–1138. doi:10.1037//0022-006x.70.5.1129

Balzan, R. P., & Galletly, C. (2015). Metacognitive therapy (MCT+) in patients with psychosis not receiving antipsychotic medication: A case study. *Frontiers in Psychology*, *6*, 1–6. doi:10.3389/fpsyg.2015.00967

Barber, T. (1970). Hypnosis, suggestions, and auditory-visual hallucination. In W. Keup (Ed.), *Origins and mechanisms of hallucinations* (pp. 167–180). New York, NY: Plenum Press.

Barlow D. H. (2014). *Clinical handbook of psychological disorders* (5th ed.). New York NY: Guilford Press.

Bebbington, P. E., Bhugra, D., Brugha, T., Singleton, N., Farrell, M., Jenkins, R., … Meltzer, H. (2004). Psychosis, victimisation and childhood disadvantage. *British Journal of Psychiatry*, *185*(3), 220–226. doi:10.1192/bjp.185.3.220

Bechdolf, A., Knost, B., Kuntermann, C., Schiller, S., Klosterkötter, J., Hambrecht, M., & Pukrop, R. (2004). A randomized comparison of group cognitive-behavioural therapy and group psychoeducation in patients with schizophrenia. *Acta Psychiatrica Scandinavica*, *110*(1), 21–28. doi:10.1111/j.1600-0447.2004.00300.x

Bechdolf, A., Veith, V., Schwarzer, D., Schormann, M., Stamm, E., Janssen, B., … Klosterkötter, J. (2005). Cognitive-behavioral therapy in the pre psychotic phase: An exploratory study. *Psychiatry Research*, *136*(2), 251–255. doi:10.1016/j.psychres.2004.06.021

Beck, A. (1952). Successful outpatient psychotherapy of a chronic schizophrenic with a delusion based on borrowed guilt. *Psychiatry*, *15*, 305–312. doi:10.1080/00332747.1952.11022883

Beck, A. T., Baruch, E., Balter, J. M., Steer, R. A., & Warman, D. M. (2004). A new instrument for measuring insight: The Beck Cognitive Insight Scale. *Schizophrenia Research*, *68*(2), 319–329. doi:10.1016/s0920-9964(03)00189-0

Beck, J. S. (1979). *Cognitive therapy of depression*. New York: Guilford Press.
Bellack, A. S. (2002). Skills training for people with severe mental illness. *Psychiatric Rehabilitation Journal*, *27*(4), 375–391. doi:10.2975/27.2004.375.391
Bennett, M., & Bellack, A. (2015). *Recovery coach manual*. New York, NY: Center for Practice Innovations.
Bentall, R. P. (1990). The illusion of reality: A review and integration of psychological research on hallucinations. *Psychological Bulletin*, *107*(1), 82–95. doi:10.1037//0033-2909.107.1.82
Bentall, R. P. (Ed.). (2013). *Reconstructing schizophrenia*. London, UK: Routledge.
Benton, M. K., & Schroeder, H. E. (1990). Social skills training with schizophrenics: A meta analytic evaluation. *Journal of Consulting and Clinical Psychology*, *58*(6), 741–747. doi:10.1037//0022-006x.58.6.741
Birchwood, M. (1995). Early intervention in psychotic relapse: Cognitive approaches to detection and management. *Behaviour Change*, *12*, 2–19. doi:10.1017/s0813483900004344
Birchwood, M., Meaden, A., Trower, P., Gilbert, P., & Plaistow, J. (2000). The power and omnipotence of voices: Subordination and entrapment by voices and significant others. *Psychological Medicine*, *30*(2), 337–344. doi:10.1017/s0033291799001828
Birchwood, M., Smith, J. O., Cochrane, R., Wetton, S., & Copestake, S. O. N. J. (1990). The Social Functioning Scale: The development and validation of a new scale of social adjustment for use in family intervention programmes with schizophrenic patients. *British Journal of Psychiatry*, *157*(6), 853–859. doi:10.1192/bjp.157.6.853
Brekke, J., Kay, D. D., Lee, K. S., & Green, M. F. (2005). Biosocial pathways to functional outcome in schizophrenia. *Schizophrenia Research*, *80*(2), 213–225. doi:10.1016/j.schres.2005.07.008
Briki, M., Monnin, J., Haffen, E., Sechter, D., Favrod, J., Netillard, C., … Bonin, B. (2014). Metacognitive training for schizophrenia: A multicentre randomised controlled trial. *Schizophrenia Research*, *157*(1), 99–106. doi:10.1016/j.schres.2014.06.005
Buchkremer, G., Klingberg, S., Holle, R., Mönking, H. S., & Hornung, W. P. (1997). Psychoeducational psychotherapy for schizophrenic patients and their key relatives or care-givers: Results of a 2-year follow-up. *Acta Psychiatrica Scandinavica*, *96*(6), 483–491. doi:10.1111/j.1600-0447.1997.tb09951.x
Bustillo, J. R., Lauriello, J., Horan, W. P., & Keith, S. J. (2001). The psychosocial treatment of schizophrenia: An update. *American Journal of Psychiatry*, *158*(2), 163–175. doi:10.1176/appi.ajp.158.2.163
Byrne, M., Agerbo, E., Eaton, W. W., & Mortensen, P. B. (2004). Parental socio-economic status and risk of first admission with schizophrenia. *Social Psychiatry and Psychiatric Epidemiology*, *39*(2), 87–96. doi:10.1007/s00127-004-0715-y
Candida, M., Campos, C., Monteiro, B., Rocha, N. B. F., Paes, F., Nardi, A. E., & Machado, S. (2016). Cognitive-behavioral therapy for schizophrenia: An overview on efficacy, recent trends and neurobiological findings. *Medical Express*, *3*(5), 1–10. doi:10.5935/medicalexpress.2016.05.01
Chadwick, P., & Birchwood, M. (1994). The omnipotence of voices: A cognitive approach to auditory hallucinations. *British Journal of Psychiatry*, *164*(2), 190–201. doi:10.1192/bjp.164.2.190
Chadwick, P., & Lowe, C. (1990). Measurement and modification of delusional beliefs. *Journal of Consulting and Clinical Psychology*, *58*, 225–232. doi:10.1037//0022-006x.58.2.225

Close, H. & Garety, P. A. (1998). Cognitive assessment of voices: Further developments in understanding the emotional impact of voices. *British Journal of Clinical Psychology*, *37*, 173–188. doi:10.1111/j.2044-8260.1998.tb01292.x

Combs, D. R., Penn, D. L., Wicher, M., & Waldheter, E. (2007). The Ambiguous Intentions Hostility Questionnaire (AIHQ): A new measure for evaluating hostile social-cognitive biases in paranoia. *Cognitive Neuropsychiatry*, *12*(2), 128–143. doi:10.1080/13546800600787854

Corrigan, P. W. (1991). Social skills training in adult psychiatric populations: A meta-analysis. *Journal of Behavior Therapy and Experimental Psychiatry*, *22*(3), 203–210. doi:10.1016/0005-7916(91)90017-y

David, D., & Montgomery, G. (2011). The scientific status of psychotherapies: A new evaluative framework for evidence-based psychosocial interventions. *Clinical Psychology: Science and Practice*, *18*, 88–99. doi:10.1111/j.1468-2850.2011.01239.x

Davis, L. W., Eicher, A. C., & Lysaker, P. H. (2011). Metacognition as a predictor of therapeutic alliance over 26 weeks of psychotherapy in schizophrenia. *Schizophrenia Research*, *129*(1), 85–90. doi:10.1016/j.schres.2011.02.026

Davis, M. C., Green, M. F., Lee, J., Horan, W. P., Senturk, D., Clarke, A. D., & Marder, S. R. (2014). Oxytocin-augmented social cognitive skills training in schizophrenia. *Neuropsychopharmacology*, *39*(9), 2070–2077. doi:10.1038/npp.2014.68

Day, J. C., Bentall, R. P., Roberts, C., Randall, F., Rogers, A., Cattell, D., Healy, D., … Power, C. (2005). Attitudes toward antipsychotic medication: The impact of clinical variables and relationships with health professionals. *Archives of General Psychiatry*, *62*(7), 717–724. doi:10.1001/archpsyc.62.7.717

De Jong, S., Renard, S. B., Van Donkersgoed, R. J. M., Van der Gaag, M., Wunderink, L., Pijnenborg, G. H. M., & Lysaker, P. H. (2014). The influence of adjunctive treatment and metacognitive deficits in schizophrenia on the experience of work. *Schizophrenia Research*, *157*(1), 107–111. doi:10.1016/j.schres.2014.04.017

Dickerson, F. B. (2000). Cognitive behavioral psychotherapy for schizophrenia: A review of recent empirical studies. *Schizophrenia Research*, *43*(2), 71–90. doi:10.1016/s0920-9964(99)00153-x

Dickerson, F. B., & Lehman, A. F. (2011). Evidence-based psychotherapy for schizophrenia: 2011 update. *Journal of Nervous and Mental Disease*, *199*(8), 520–526. doi:10.1097/nmd.0b013e318225ee78

Dickerson, F. B., Tenhula, W. N., & Green-Paden, L. D. (2005). The token economy for schizophrenia: Review of the literature and recommendations for future research. *Schizophrenia Research*, *75*(2), 405–416. doi:10.1016/j.schres.2004.08.026

Doering, S., Müller, E., Köpcke, W., Pietzcker, A., Gaebel, W., Linden, M., … Schüssler, G. (1998). Predictors of relapse and rehospitalization in schizophrenia and schizoaffective disorder. *Schizophrenia Bulletin*, *24*(1), 87–98. doi:10.1093/oxfordjournals.schbul.a033316

Done, D., Frith, C., & Owen, D. (1986). Reducing persistent auditory hallucinations by wearing an ear plug. *British Journal of Clinical Psychology*, *25*, 151–152. doi:10.1111/j.2044-8260.1986.tb00687.x

Drury, V., Birchwood, M., Cochrane, R., & MacMillan, F. (1996). Cognitive therapy and recovery from acute psychosis: A controlled trial. I. Impact on psychotic symptoms. *British Journal of Psychiatry*, *169*(5), 593–601. doi:10.1192/bjp.169.5.593

Durham, R. C., Guthrie, M., Morton, R. V., Reid, D. A., Treliving, L. R., Fowler, D., & MacDonald, R. R. (2003). Tayside–Fife clinical trial of cognitive–behavioural therapy for medication-resistant psychotic symptoms. *British Journal of Psychiatry*, *182*(4), 303–311. doi:10.1192/bjp.182.4.303

Dyck, D. G., Short, R. A., Hendryx, M. S., Norell, D., Myers, M., Patterson, T., … McFarlane, W. R. (2000). Management of negative symptoms among patients with schizophrenia attending multiple-family groups. *Psychiatric Services*, *51*(4), 513–519. doi:10.1176/appi.ps.51.4.513

Eckman, T., Wirshing, W., Marder, S., Liberman, R., Johnston-Cronk, K., Zimmermann, K., & Mintz, J. (1992). Technique for training schizophrenic patients in illness self management: A controlled trial. *American Journal of Psychiatry*, *149*(11), 1549–1555. doi:10.1176/ajp.149.11.1549

Eckman, T. A., Liberman, R. P., Phipps, C. C., & Blair, K. E. (1990). Teaching medication management skills to schizophrenic patients. *Journal of Clinical Psychopharmacology*, *10*(1), 33–38. doi:10.1097/00004714-199002000-00006

Favrod, J., Rexhaj, S., Bardy, S., Ferrari, P., Hayoz, C., Moritz, S., … Bonsack, C. (2014). Sustained antipsychotic effect of metacognitive training in psychosis: A randomized-controlled study. *European Psychiatry*, *29*(5), 275–281. doi:10.1016/j.eurpsy.2013.08.003

Freeman, D., Dunn, G., Startup, H., Pugh, K., Cordwell, J., Mander, H., … Kingdon, D. (2015). Effects of cognitive behaviour therapy for worry on persecutory delusions in patients with psychosis (WIT): A parallel, single-blind, randomised controlled trial with a mediation analysis. *Lancet Psychiatry*, *2*(4), 305–313. doi:10.1016/s2215-0366(15)00039-5

Freeman, D., & Garety, P. A. (1999). Worry, worry processes and dimensions of delusions: An exploratory investigation of a role for anxiety processes in the maintenance of delusional distress. *Behavioural and Cognitive Psychotherapy*, *27*(1), 47–62.

Freeman, D., Garety, P., Kuipers, E., Colbert, S., Jolley, S., Fowler, D., Dunn, G., & Bebbington, P. (2006). Delusions and decision-making style: Use of the Need for Closure Scale. *Behaviour Research and Therapy*, *44*(8), 1147–1158. doi:10.1016/j.brat.2005.09.002

Garety, P., Waller, H., Emsley, R., Jolley, S., Kuipers, E., Bebbington, P., … Freeman, D. (2015). Cognitive mechanisms of change in delusions: An experimental investigation targeting reasoning to effect change in paranoia. *Schizophrenia Bulletin*, *41*(2), 400–410. doi:10.1093/schbul/sbu103

Garety, P. A., & Freeman, D. (2013). The past and future of delusions research: From the inexplicable to the treatable. *British Journal of Psychiatry*, *203*(5), 327–333. doi:10.1192/bjp.bp.113.126953

Garety, P. A., Kuipers, E., Fowler, D., Freeman, D., & Bebbington, P. E. (2001). A cognitive model of the positive symptoms of psychosis. *Psychological Medicine*, *31*(2), 189–195.

Gaudiano, B. A. (2005). Cognitive behavior therapies for psychotic disorders: Current empirical status and future directions. *Clinical Psychology: Science and Practice*, *12*(1), 33–50. doi:10.1093/clipsy.bpi004

Gaudiano, B. A. (2006). Is symptomatic improvement in clinical trials of cognitive-behavioral therapy for psychosis clinically significant*? Journal of Psychiatric Practice*, *12*(1), 11–23. doi:10.1097/00131746-200601000-00003

Gaudiano, B. A., & Herbert, J. D. (2006). Acute treatment of inpatients with psychotic symptoms using acceptance and commitment therapy: Pilot results. *Behaviour Research and Therapy*, *44*(3), 415–437. doi:10.1016/j.brat.2005.02.007

Gaudiano, B. A., Herbert, J. D., & Hayes, S. C. (2010). Is it the symptom or the relation to it? Investigating potential mediators of change in acceptance and commitment therapy for psychosis. *Behavior Therapy*, *41*(4), 543–554. doi:10.1016/j.beth.2010.03.001

Gawęda, Ł., Krężołek, M., Olbryś, J., Turska, A., & Kokoszka, A. (2015). Decreasing self reported cognitive biases and increasing clinical insight through meta-cognitive training in patients with chronic schizophrenia. *Journal of Behavior Therapy and Experimental Psychiatry*, *48*, 98–104. doi:10.1016/j.jbtep.2015.02.002

Glynn, S., Marder, S., Liberman, R., Blair, K., Wirshing, W., Wirshing, D., Ross, D., & Mintz, J. (2002). Supplementing clinic-based skills training with manual-based community support sessions: Effects on social adjustment of patients with schizophrenia. *American Journal of Psychiatry*, *159*(5), 829–837. doi:10.1176/appi.ajp.159.5.829

Granholm, E., Ben-Zeev, D., & Link, P. C. (2009). Social disinterest attitudes and group cognitive-behavioral social skills training for functional disability in schizophrenia. *Schizophrenia Bulletin*, *35*(5), 874–883. doi:10.1093/schbul/sbp072

Granholm, E., Holden, J., Link, P. C., McQuaid, J. R., & Jeste, D. V. (2013). Randomized controlled trial of cognitive behavioral social skills training for older consumers with schizophrenia: Defeatist performance attitudes and functional outcome. *American Journal of Geriatric Psychiatry*, *21*(3), 251–262. doi:10.1016/j.jagp.2012.10.014

Grant, P. M., & Beck, A. T. (2009). Defeatist beliefs as a mediator of cognitive impairment, negative symptoms, and functioning in schizophrenia. *Schizophrenia Bulletin*, *35*(4), 798–806. doi:10.1093/schbul/sbn008

Green, C. E. L., Freeman, D., Kuipers, E., Bebbington, P., Fowler, D., Dunn, G., & Garety, P. A. (2008). Measuring ideas of persecution and social reference: The Green et al. Paranoid Thought Scales (GPTS). *Psychological Medicine*, *38*(1), 101–111. doi:10.1017/s0033291707001638

Green, M. F., Horan, W. P., & Lee, J. (2015). Social cognition in schizophrenia. *Nature Reviews Neuroscience*, *16*, 620–631. doi:10.1038/nrn4005

Grossman, L. S., Harrow, M., Rosen, C., Faull, R., & Strauss, G. P. (2008). Sex differences in schizophrenia and other psychotic disorders: A 20-year longitudinal study of psychosis and recovery. *Comprehensive Psychiatry*, *49*, 523–529. doi:10.1016/j.comppsych.2008.03.004

Gumley, A., & Power, K. (2000). Is targeting cognitive therapy during relapse in psychosis feasible? *Behavioural and Cognitive Psychotherapy*, *28*(2), 161–174.

Haddock, G., McCarron, J., Tarrier, N., & Faragher, E. B. (1999). Scales to measure dimensions of hallucinations and delusions: The Psychotic Symptom Rating Scales (PSYRATS). *Psychological Medicine*, *29*(4), 879–889. doi:10.1017/s0033291799008661

Haddock, G., Slade, P., Bentall, R., Reid, D., & Faragher, E. (1998). A comparison of the long-term effectiveness of distraction and focusing in the treatment of auditory hallucinations. *British Journal of Medical Psychology*, *71*, 339–349. doi:10.1111/j.2044-8341.1998.tb00996.x

Haddock, G., Tarrier, N., Morrison, A. P., Hopkins, R., Drake, R., & Lewis, S. (1999). A pilot study evaluating the effectiveness of individual inpatient cognitive-behavioural therapy in

early psychosis. *Social Psychiatry and Psychiatric Epidemiology*, *34*(5), 254–258. doi:10.1007/s001270050141

Hambrecht, M., Lammertink, M., Klosterkötter, J., Matuschek, E., & Pukrop, R. (2002). Subjective and objective neuropsychological abnormalities in a psychosis prodrome clinic. *British Journal of Psychiatry*, *181*(43), S30–S37. doi:10.1192/bjp.181.43.s30

Heslin, K. C., & Weiss, A. J. (2015). Hospital readmissions involving psychiatric disorders, 2012. *Statistical Brief 189*. Rockville, MD: Agency for Healthcare Research and Quality. Retrieved October 3, 2017, from www.hcup-us.ahrq.gov/reports/statbriefs/sb189-Hospital-Readmissions-Psychiatric-Disorders-2012.jsp

Ho, B. C., Andreasen, N. C., Nopoulos, P., Arndt, S., Magnotta, V., & Flaum, M. (2003). Progressive structural brain abnormalities and their relationship to clinical outcome: A longitudinal magnetic resonance imaging study early in schizophrenia. *Archives of General Psychiatry*, *60*(6), 585–594. doi:10.1001/archpsyc.60.6.585

Horan, W. P., Kern, R. S., Shokat-Fadai, K., Sergi, M. J., Wynn, J. K., & Green, M. F. (2009). Social cognitive skills training in schizophrenia: An initial efficacy study of stabilized outpatients. *Schizophrenia Research*, *107*(1), 47–54. doi:10.1016/j.schres.2008.09.006

Hultman, C. M., Ohman, A., Cnattingius, S., Wieselgren, I. M., & Lindstr, L. H. (1997). Prenatal and neonatal risk factors for schizophrenia. *British Journal of Psychiatry*, *170*(2), 128–133. doi:10.1192/bjp.170.2.128

Insel, T. R. (2008). Assessing the economic costs of serious mental illness. *American Journal of Psychiatry*, *165*(6), 663–665. doi:10.1176/appi.ajp.2008.08030366

Jackson, H., McGorry, P., Edwards, J., Hulbert, C., Henry, L., Harrigan, S., … Power, P. (2005). A controlled trial of cognitively oriented psychotherapy for early psychosis (COPE) with four-year follow-up readmission data. *Psychological Medicine*, *35*(9), 1295–1306. doi:10.1017/s0033291705004927

Jauhar, S., McKenna, P. J., Radua, J., Fung, E., Salvador, R., & Laws, K. R. (2014). Cognitive behavioural therapy for the symptoms of schizophrenia: Systematic review and meta analysis with examination of potential bias. *British Journal of Psychiatry*, *204*(1), 20–29. doi:10.1192/bjp.bp.112.116285

Jones, C., Hacker, D., Cormac, I., Meaden, A., & Irving, C. B. (2012). Cognitive behavioural therapy versus other psychosocial treatments for schizophrenia (review). *Cochrane Database of Systematic Reviews*, *4*, CD008712. doi:10.1002/14651858.CD008712.pub2

Kapur, S. (2003). Psychosis as a state of aberrant salience: A framework linking biology, phenomenology, and pharmacology in schizophrenia. *American Journal of Psychiatry*, *160*, 13–23. doi:10.1176/appi.ajp.160.1.13

Kay, S. R., Flszbein, A., & Opfer, L. A. (1987). The Positive and Negative Syndrome Scale (PANSS) for schizophrenia. *Schizophrenia Bulletin*, *13*(2), 261–276. doi:10.1093/schbul/13.2.261

Keown, P., Holloway, F., & Kuipers, E. (2002). The prevalence of personality disorders, psychotic disorders and affective disorders amongst the patients seen by a community mental health team in London. *Social Psychiatry and Psychiatric Epidemiology*, *37*(5), 225–229. doi:10.1007/s00127-002-0533-z

Keown, P., Holloway, F., & Kuipers, E. (2005). The impact of severe mental illness, co-morbid personality disorders and demographic factors on psychiatric bed use. *Social Psychiatry and Psychiatric Epidemiology*, *40*(1), 42–49. doi:10.1007/s00127-005-0842-0

Kingdon, D., & Turkington, D. (2006). The ABCs of cognitive-behavioral therapy for schizophrenia. *Psychiatric Times*, *23*(7), 49–50.

Kopelowicz, A., Liberman, R. P., & Zarate, R. (2006). Recent advances in social skills training for schizophrenia. *Schizophrenia Bulletin*, *32*(1), S12–S23. doi:10.1093/schbul/sbl023

Kreyenbuhl, J., Buchanan, R., Dickerson, F., & Dixon, L. (2010). The Schizophrenia Patient Outcomes Research Team (PORT): Updated treatment recommendations 2009. *Schizophrenia Bulletin*, *36*(1), 94–103. doi:10.1093/schbul/sbp130

Kuepper, R., Van Os, J., Lieb, R., Wittchen, H. U., Höfler, M., & Henquet, C. (2011). Continued cannabis use and risk of incidence and persistence of psychotic symptoms: 10 year follow-up cohort study. *BMJ, 342*, d738. https://doi.org/10.1136/bmj.d738

Kuipers, E., Garety, P., Fowler, D., Freeman, D., Dunn, G., & Bebbington, P. (2006). Cognitive, emotional, and social processes in psychosis: Refining cognitive behavioral therapy for persistent positive symptoms. *Schizophrenia Bulletin*, *32*(1), S24–S31.

Kumar, D., Zia Ul Haq, M., Dubey, I., Dotivala, K. N., Veqar Siddiqui, S., Prakash, R., … Nizamie, S. H. (2010). Effect of meta-cognitive training in the reduction of positive symptoms in schizophrenia. *European Journal of Psychotherapy & Counselling*, *12*(2), 149–158. doi:10.1080/13642537.2010.488875

Kumari, V., Fannon, D., Peters, E. R., Sumich, A. L., Premkumar, P., Anilkumar, A. P., … Kuipers, E. (2011). Neural changes following cognitive behaviour therapy for psychosis: A longitudinal study. *Brain*, *134*(8), 2396–2407. doi:10.1093/brain/awr154

Kuokkanen, R., Lappalainen, R., Repo-Tiihonen, E., & Tiihonen, J. (2014). Metacognitive group training for forensic and dangerous non-forensic patients with schizophrenia: A randomised controlled feasibility trial. *Criminal Behaviour and Mental Health*, *24*(5), 345–357. doi:10.1002/cbm.1905

Kurtz, M., & Mueser, K. (2008). A meta-analysis of controlled research on social skills training for schizophrenia. *Journal of Consulting and Clinical Psychology*, *76*(3), 491–504.

Lam, K., Ho, C. P., Wa, J., Chan, S., Yam, K., Yeung, O. S., … Balzan, R. (2015). Metacognitive training (MCT) for schizophrenia improves cognitive insight: A randomized controlled trial in a Chinese sample with schizophrenia-spectrum disorders. *Behaviour Research and Therapy*, *64*, 38–42. doi:10.1016/j.brat.2014.11.008

Lehman, A. F., Goldberg, R., Dixon, L. B., McNary, S., Postrado, L., Hackman, A., & McDonnell, K. (2002). Improving employment outcomes for persons with severe mental illnesses. *Archives of General Psychiatry*, *59*(2), 165–172. doi:10.1001/archpsyc.59.2.165

Lewis, S., Tarrier, N., Haddock, G., Bentall, R., Kinderman, P., Kingdon, D., … Dunn, G. (2002). Randomised controlled trial of cognitive behavioural therapy in early schizophrenia: Acute-phase outcomes. *British Journal of Psychiatry*, *181*(43), S91–S97. doi:10.1192/bjp.181.43.s91

Li, F., & Wang, M. (1994). A behavioral training programme for chronic schizophrenic patients: A three-month randomised controlled trial in Beijing. *British Journal of Psychiatry*, *165*(Suppl. 24), 32–37.

Liberman, R., Wallace, G., Blackwell, G., Eckmann, T., Vaccaro, J., & Kuehnel, T. (1993). Innovations in skills training for the seriously mentally ill: The UCLA Social and Independent Living Skills Modules. *Innovations Research*, *2*, 43–60.

Looijestijn, J., Blom, J. D., Aleman, A., Hoek, H. W., & Goekoop, R. (2015). An integrated network model of psychotic symptoms. *Neuroscience & Biobehavioral Reviews*, *59*, 238–250. doi:10.1016/j.neubiorev.2015.09.016

Lynch, D., Laws, K. R., & McKenna, P. J. (2010). Cognitive behavioural therapy for major psychiatric disorder: Does it really work? A meta-analytical review of well-controlled trials. *Psychological Medicine*, *40*(1), 9–24. doi:10.1017/s003329170900590x

Lysaker, P., Gumley, A., Luedtke, B., Buck, K., Ringer, J., Olesek, K., … Dimaggio, G. (2013). Social cognition and metacognition in schizophrenia: Evidence of their independence and linkage with outcomes. *Acta Psychiatrica Scandinavica*, *127*(3), 239–247. doi:10.1111/acps.12012

Lysaker, P., Shea, A., Buck, K., Dimaggio, G., Nicolò, G., Procacci, M., Salvatore, G., & Rand, K. (2010). Metacognition as a mediator of the effects of impairments in neurocognition on social function in schizophrenia-spectrum disorders. *Acta Psychiatrica Scandinavica*, *122*(5), 405–413. doi:10.1111/j.1600-0447.2010.01554.x

Malaspina, D., Harlap, S., Fennig, S., Heiman, D., Nahon, D., Feldman, D., & Susser, E. S. (2001). Advancing paternal age and the risk of schizophrenia. *Archives of General Psychiatry*, *58*(4), 361–367.

Mander, H., & Kingdon, D. (2015). The evolution of cognitive–behavioral therapy for psychosis. *Psychology Research and Behavior Management*, *8*, 63–69. doi:10.2147/prbm.s52267

Margo, A., Hemsley, D., & Slade, P. (1981). The effects of varying auditory input on schizophrenic hallucinations. *British Journal of Psychiatry*, *139*, 122–127. doi:10.1192/bjp.139.2.122

Mason, L., Peters, E. R., Dima, D., Williams, S. C., & Kumari, V. (2015). Cognitive behavioural therapy normalizes functional connectivity for social threat in psychosis. *Schizophrenia Bulletin*, *42*(3), 684–692. doi:10.1093/schbul/sbv153

Medalia, A., Revheim, N., & Casey, M. (2001). The remediation of problem-solving skills in schizophrenia. *Schizophrenia Bulletin*, *27*(2), 259–267. doi:10.1093/oxfordjournals.schbul.a006872

Meehl, P. E. (1962). Schizotaxia, schizotypy, schizophrenia. *American Psychologist*, *17*(12), 827–838. doi:10.1037/h0041029

Menon, M., Andersen, D., Quilty, L., & Woodward, T. (2015). Individual factors predicted to influence outcome in group CBT for psychosis (CBTp) and related therapies. *Frontiers in Psychology*, *6*, 1–5. doi:10.3389/fpsyg.2015.01563

Mintz, S., & Alpert, M. (1972). Imagery vividness, reality testing, and schizophrenic hallucinations. *Journal of Abnormal Psychology*, *19*, 310–316. doi:10.1037/h0033209

Moritz, S., Veckenstedt, R., Bohn, F., Hottenrott, B., Scheu, F., Randjbar, S., … Roesch-Ely, C. (2013). Complementary group metacognitive training (MCT) reduces delusional ideation in schizophrenia. *Schizophrenia Research*, *151*(1), 61–69. doi:10.1016/j.schres.2013.10.007

Moritz, S., Vitzthum, F., Randjbar, S., Veckenstedt, R., & Woodward, T. S. (2010). Detecting and defusing cognitive traps: Metacognitive intervention in schizophrenia. *Current Opinion in Psychiatry*, *23*(6), 561–569. doi:10.1097/yco.0b013e32833d16a8

Moritz, S., & Woodward, T. (2007). Metacognitive training in schizophrenia: From basic research to knowledge translation and intervention. *Current Opinion in Psychiatry*, *20*, 619–625. doi:10.1097/yco.0b013e3282f0b8ed

Morrison, A. (1998). A cognitive analysis of auditory hallucinations: Are voices to schizophrenia what bodily sensations are to panic? *Behavioural and Cognitive Psychotherapy*, *26*, 289–302.

Morrison, A., & Haddock, G. (1997). Self-focused attention in schizophrenic patients and normal subjects: A comparative study. *Personality and Individual Differences*, *23*, 937–941. doi:10.1016/s0191-8869(97)00130-x

Morrison, A., & Renton, J. C. (2001). Cognitive therapy for auditory hallucinations: A theory based approach. *Cognitive and Behavioral Practice*, *8*(2), 147–160. doi:10.1016/s1077-7229(01)80020-1

Morrison, A. P., Pyle, M., Chapman, N., French, P., Parker, S. K., & Wells, A. (2014). Metacognitive therapy in people with a schizophrenia-spectrum diagnosis and medication resistant symptoms: A feasibility study. *Journal of Behavior Therapy and Experimental Psychiatry*, *45*(2), 280–284. doi:10.1016/j.jbtep.2013.11.003

Mueser, K. T., Goodman, L. B., Trumbetta, S. L., Rosenberg, S. D., Osher, F. C., Vidaver, R., … Foy, D. W. (1998). Trauma and posttraumatic stress disorder in severe mental illness. *Journal of Consulting and Clinical Psychology*, *66*(3), 493–499. doi:10.1037//0022-006x.66.3.493

National Institute of Mental Health. (2015). *Schizophrenia*. Retrieved September 24, 2017, from https://www.nimh.nih.gov/health/publications/schizophrenia-booklet/index.shtml

Nuechterlein, K. H., & Green, M. F. (2006). *MATRICS consensus cognitive battery*. Los Angeles, CA: MATRICS Assessment.

Ottavi, P., D'Alia, D., Lysaker, P., Kent, J., Popolo, R., Salvatore, G., & Dimaggio, G. (2014). Metacognition-oriented social skills training for individuals with long-term schizophrenia: Methodology and clinical illustration. *Clinical Psychology & Psychotherapy*, *21*(5), 465–473. doi:10.1002/cpp.1850

Pankowski, D., Kowalski, J., & Gawęda, Ł. (2016). The effectiveness of metacognitive training for patients with schizophrenia: A narrative systematic review of studies published between 2009 and 2015. *Psychiatria Polska*, *50*(4), 787–803. doi:10.12740/pp/59113

Penn, D. L., Meyer, P. S., Evans, E., Wirth, R. J., Cai, K., & Burchinal, M. (2009). A randomized controlled trial of group cognitive-behavioral therapy vs. enhanced supportive therapy for auditory hallucinations. *Schizophrenia Research*, *109*(1), 52–59. doi:10.1016/j.schres.2008.12.009

Pfammatter, M., Junghan, U. M., & Brenner, H. D. (2006). Efficacy of psychological therapy in schizophrenia: Conclusions from meta-analyses. *Schizophrenia Bulletin*, *32*(Suppl. 1), S64–S80. doi:10.1093/schbul/sbl030

Pilling, S., Bebbington, P., Kuipers, E., Garety, P., Geddes, J., Martindale, B., … Morgan, C. (2002). Psychological treatments in schizophrenia: II. Meta-analyses of randomized controlled trials of social skills training and cognitive remediation. *Psychological Medicine*, *32*(5), 783–791.

Quinlan, T. (2014). *Mechanisms of change in cognitive behavioral social skills training for schizophrenia* (Doctoral thesis). Retrieved May 4, 2017, from University of California, San Diego *Electronic Theses and Dissertations*, https://search.proquest.com/openview/4c53261957b7d0122f90a3ef6547b5d9/1?pq-origsite=gscholar&cbl=18750&diss=y

Rector, N. A., Seeman, M. V., & Segal, Z. V. (2003). Cognitive therapy for schizophrenia: A preliminary randomized controlled trial. *Schizophrenia Research*, *63*(1), 1–11. doi:10.1016/s0920-9964(02)00308-0

Resnick, S. G., Bond, G. R., & Mueser, K. T. (2003). Trauma and posttraumatic stress disorder in people with schizophrenia. *Journal of Abnormal Psychology*, *112*(3), 415–423. doi:10.1037/0021-843X.112.3.415

Rus-Calafell, M., Gutiérrez-Maldonado, J., Ortega-Bravo, M., Ribas-Sabaté, J., & Caqueo Urízar, A. (2013). A brief cognitive–behavioural social skills training for stabilized outpatients with schizophrenia: A preliminary study. *Schizophrenia Research*, *143*(2), 327–336. doi:10.1016/j.schres.2012.11.014

Salkever, D., Domino, M. E., Burns, B. J., Santos, A. B., Deci, P. A., Dias, J., … Paolone, J. (1999). Assertive community treatment for people with severe mental illness: The effect on hospital use and costs. *Health Services Research*, *34*(2), 577–601.

Sarin, F., Wallin, L., & Widerlöv, B. (2011). Cognitive behavior therapy for schizophrenia: A meta-analytical review of randomized controlled trials. *Nordic Journal of Psychiatry*, *65*(3), 162–174. doi:10.3109/08039488.2011.577188

Schmidt, S. J., Mueller, D. R., & Roder, V. (2011). Social cognition as a mediator variable between neurocognition and functional outcome in schizophrenia: Empirical review and new results by structural equation modeling. *Schizophrenia Bulletin*, *37*(Suppl. 2), S41–S54.

Schooler, N., Hogarty, G., & Weissman, M (1979). Social Adjustment Scale II (SAS-II). In W. A. Hargreaves, C. C. Atkisson, & J. E. Sorenson (Eds.), *Resource materials for community mental health program evaluations* (pp. 290–303). Rockville, MD, National Institute of Mental Health.

Scott, H., Johnson, S., Menezes, P., Thornicroft, G., Marshall, J., Bindman, J., … Kuipers, E. (1998). Substance misuse and risk of aggression and offending among the severely mentally ill. *British Journal of Psychiatry*, *172*(4), 345–350. doi:10.1192/bjp.172.4.345

Selten, J. P., Van der Ven, E., Rutten, B. P., & Cantor-Graae, E. (2013). The social defeat hypothesis of schizophrenia: An update. *Schizophrenia Bulletin*, *39*(6), 1180–1186. doi:10.1093/schbul/sbt134

Sensky, T., Turkington, D., Kingdon, D., Scott, J. L., Scott, J., Siddle, R., … Barnes, T. R. (2000). A randomized controlled trial of cognitive-behavioral therapy for persistent symptoms in schizophrenia resistant to medication. *Archives of General Psychiatry*, *57*(2), 165–172. doi:10.1001/archpsyc.57.2.165

Slade, P. D. (1972). The effects of systematic desensitization on auditory hallucinations. *Behaviour Research and Therapy*, *10*, 85–91. doi:10.1016/0005-7967(72)90013-7

Slade, P. D. (1974). The external control of auditory hallucinations: An information theory analysis. *British Journal of Social and Clinical Psychology*, *13*, 73–79. doi:10.1111/j.2044-8260.1974.tb00880.x

Slade, P. D. (1976). Towards a theory of auditory hallucinations: Outline of an hypothetical four factor model. *British Journal of Social and Clinical Psychology*, *15*, 415–423. doi:10.1111/j.2044-8260.1976.tb00053.x

So, S. H. W., Chan, A. P., Chong, C. S. Y., Wong, M. H. M., Lo, W. T. L., Chung, D. W. S., & Chan, S. S. (2015). Metacognitive training for delusions (MCTd): Effectiveness on data gathering and belief flexibility in a Chinese sample. *Frontiers in Psychology*, *6*, 1–15. doi:10.3389/fpsyg.2015.00730

Strauss, G. P., Harrow, M., Grossman, L. S., & Rosen, C. (2010). Periods of recovery in deficit syndrome schizophrenia: A 20-year multifollowup longitudinal study. *Schizophrenia Bulletin*, *36*, 788–799. doi:10.1093/schbul/sbn167

Sundquist, K., Frank, G., & Sundquist, J. A. N. (2004). Urbanisation and incidence of psychosis and depression. *British Journal of Psychiatry*, *184*(4), 293–298. doi:10.1192/bjp.184.4.293

Tarrier, N. (1987). An investigation of residual psychotic symptoms in discharged schizophrenic patients. *British Journal of Clinical Psychology*, *26*, 141–143. doi:10.1111/j.2044-8260.1987.tb00740.x

Tarrier, N., Harwood, S., Yusupoff, L., Beckett, R., & Baker, A. (1990). Coping strategy enhancement (CSE): A method of treating residual schizophrenic symptoms. *Behavioral Psychotherapy*, *18*, 283–293. doi:10.1017/s0141347300010387

Turkington, D., Kingdon, D., & Weiden, P. J. (2006). Cognitive behavior therapy for schizophrenia. *American Journal of Psychiatry*, *163*(3), 365–373. doi:10.1176/appi.ajp.163.3.365

Turkington, D., Sensky, T., Scott, J., Barnes, T. R., Nur, U., Siddle, R., … Kingdon, D. (2008). A randomized controlled trial of cognitive behavior therapy for persistent symptoms in schizophrenia: A five-year follow-up. *Schizophrenia Research*, *98*(1), 1–7. doi:10.1016/j.schres.2007.09.026

Turnbull, G., & Bebbington, P. (2001). Anxiety and the schizophrenic process: Clinical and epidemiological evidence. *Social Psychiatry and Psychiatric Epidemiology*, *36*(5), 235–243. doi:10.1007/s001270170054

Turner, D. T., Van der Gaag, M., Karyotaki, E., & Cuijpers, P. (2014). Psychological interventions for psychosis: A meta-analysis of comparative outcome studies. *American Journal of Psychiatry*, *171*(5), 523–538. doi:10.1176/appi.ajp.2013.13081159

Valmaggia, L. R., Van der Gaag, M., Tarrier, N., Pijnenborg, M., & Slooff, C. J. (2005). Cognitive–behavioural therapy for refractory psychotic symptoms of schizophrenia resistant to atypical antipsychotic medication. *British Journal of Psychiatry*, *186*(4), 324–330. doi:10.1192/bjp.186.4.324

Van Donkersgoed, R., De Jong, S., Van der Gaag, M., Aleman, A., Lysaker, P., Wunderink, L., & Pijnenborg, G. (2014). A manual-based individual therapy to improve metacognition in schizophrenia: Protocol of a multi-center RCT. *BMC Psychiatry*, *14*(27), 1–8. doi:10.1186/1471-244X-14-27

Van Oosterhout, B., Krabbendam, L., De Boer, K., Ferwerda, J., Van der Helm, M., Stant, A., & Van der Gaag, M. (2014). Metacognitive group training for schizophrenia-spectrum patients with delusions: A randomized controlled trial. *Psychological Medicine*, *44*(14), 3025–3035. doi:10.1017/s0033291714000555

Veling, W., Susser, E., Van Os, J., Mackenbach, J. P., Selten, J. P., & Hoek, H. W. (2008). Ethnic density of neighborhoods and incidence of psychotic disorders among immigrants. *American Journal of Psychiatry*, *165*(1), 66–73. doi:10.1176/appi.ajp.2007.07030423

Velligan, D. I., Tai, S., Roberts, D. L., Maples-Aguilar, N., Brown, M., Mintz, J., & Turkington, D. (2014). A randomized controlled trial comparing cognitive behavior therapy, cognitive adaptation training, their combination and treatment as usual in chronic schizophrenia. *Schizophrenia Bulletin*, *41*(3), 597–603. doi:10.1093/schbul/sbu127

Wells, A. (1995). Meta-cognition and worry: A cognitive model of generalised anxiety disorder. *Behavioural and Cognitive Psychotherapy*, *23*, 301–320. doi:10.1017/s1352465800015897

Wells, A., & Cartwright-Hatton, S. (2004). A short form of the metacognitions questionnaire: Properties of the MCQ-30. *Behaviour Research and Therapy*, *42*(4), 385–396. doi:10.1016/s0005-7967(03)00147-5

Wykes, T., Reeder, C., Landau, S., Everitt, B., Knapp, M., Patel, A., & Romeo, R. (2007). Cognitive remediation therapy in schizophrenia. *British Journal of Psychiatry*, *190*(5), 421–427. doi:10.1192/bjp.bp.106.026575

Wykes, T., Steel, C., Everitt, B., & Tarrier, N. (2008). Cognitive behavior therapy for schizophrenia: Effect sizes, clinical models, and methodological rigor. *Schizophrenia Bulletin*, *34*(3), 523–537. doi:10.1093/schbul/sbm114

15

Psychotherapy and Autism Spectrum Disorder

Conceptual and Pragmatic Challenges

Raymond G. Romanczyk, Rachel N. S. Cavalari, and Jennifer M. Gillis
Department of Psychology, Binghamton University, Binghamton, USA

The conceptualization of autism has evolved significantly over the eight decades since it was first describe by Kanner (1943). While there are earlier writings that laid some conceptual groundwork, Kanner was the first to describe cases in detail and propose the syndrome of early infantile autism. It is important to note that, while historically the word "autism" is associated with "withdrawal," Kanner emphasized the term "aloof" to describe his observations of the detachment in social interactions of individuals with autism (Romanczyk & Callahan, 2012).

As part of his description, in addition to the emphasis on aloofness, Kanner identified a number of additional specific characteristics, some examples of which are:

- an intense desire to maintain sameness;
- looks "normal" and has an alert expression;
- avoids eye contact (gaze aversion);
- may appear deaf and/or blind;
- no anticipatory reaching out from infancy;
- does not initiate sounds or gestures for communication;
- does not use speech for social communication.

15.1 History of the Diagnosis of Autism

Autism was first codified in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) in 1980, using the term "infantile autism" (American Psychiatric Association, 1980). Although the term "autistic" was used in the second edition of the DSM (American Psychiatric Association, 1968), it was used in the context of the diagnosis of schizophrenia (Grinker, 2007). Significant changes occurred between the third edition (DSM-III; released in 1980) and the revised third edition (DSM-III-R; in 1987), and again with the fourth edition (DSM-IV; in 1994) and the text revision of the fourth edition (DSM-IV-TR; in 2000). In May of 2013, the DSM-5 diagnostic criteria

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

were published and reversed several decades of clinical and research efforts to subcategorize autism and related disorders (American Psychiatric Association, 2013). Whereas there had been multiple disorders in the DSM-IV-TR, the DSM-5 now contains a single disorder, termed "autism spectrum disorder."

The change in diagnostic approach heralded by the DSM-5 cannot be overstated (for a more detailed description, see Romanczyk, Turner, Sevlever, & Gillis, 2014). The DSM-IV-TR used the organizational structure of "pervasive developmental disorders," which contained five discrete disorders, including autistic disorder, Asperger's disorder, and pervasive developmental disorder not otherwise specified. This class of specific disorders was dropped from the DSM-5, and there remains only autism spectrum disorder, which is under "neurodevelopmental disorders." While research indicated variability in the distribution of use of the diagnoses within the DSM-IV-TR's pervasive develodpmental disorders (e.g., Lord et al., 2011), there was also research indicating clear differences in important clinical characteristics between these DSM-IV-TR diagnoses (e.g., Eagle, Romanczyk, & Lenzenweger, 2010; Turner & Romanczyk, 2012). Moving forward, it will be difficult to compare research conducted under previous DSM editions to research conducted under the DSM-5 criteria.

15.2 Heterogeneity

The diagnosis of autism spectrum disorder (ASD)[1] is orthogonal to intellectual impairment and spoken language development, is considered a lifespan disorder, and is remarkable for the heterogeneity of its symptom presentation. Additionally, currently comorbid conditions are routinely diagnosed, in a clear break from past diagnostic processes. The results of a study by Simonoff et al. (2008) indicated that 70% of a sample of 112 children and adolescents aged between 10 and 14 years with an ASD had at least one comorbid psychiatric disorder and 41% had at least two. The more common comorbid diagnoses include several anxiety disorders, depression, attention-deficit hyperactivity disorder, sleep disorders, and conduct and oppositional defiant disorders (Bradley, Summers, Wood, & Bryson, 2004; Ghaziuddin, Weidmer-Mikhail, & Ghaziuddin, 1998; Simonoff et al., 2008). Approximately 75% of individuals with an ASD show some level of intellectual disability (Croen, Grether, Hoogstrate, & Selvin, 2002).[2]

Further, the extant literature on ASD is highly skewed with respect to age, wherein the bulk of the research concerns young children, followed by adolescents and adults. These factors present significant challenge in collating and synthesizing the clinical and research literature.

15.3 Core Characteristics

For the purposes of this chapter, we focus upon the core characteristics of ASD presented in the DSM-5: (1) persistent deficits in social communication and social interaction across contexts, not accounted for by general developmental delays, and (2) restricted, repetitive patterns of behavior, interests, or activities.

There are thousands of published research, descriptive, and case studies concerning treatment for ASD, but the majority focus upon specific characteristics and/or

symptoms that are not core deficits and often are simply associated or collateral behaviors or symptoms. Thus the focus of this chapter is to examine the clinical impact upon the core characteristics, rather than cataloging an expansive list of individual clinical interventions for highly specific behaviors or symptoms. That is, the focus is not upon providing services for individuals with an ASD with respect to addressing narrow behaviors or symptoms but rather on providing effective services to address the core characteristics of individuals with ASD.

15.4 Prevalence

As can be inferred from the information already provided, statements concerning prevalence cannot be definitive, as too many variables have changed over time. That is, there is not a consistent base with which to compare current assessments of prevalence.

After Kanner's publication in 1943, autism was considered a rare disorder for several decades, affecting approximately 4 in 10,000 people (Lotter, 1966). It was not until the 1990s that a significant change in the long-standing prevalence rate estimate occurred. The past two decades in particular present a trend in prevalence rates that is striking. Bertrand et al. (2001) reported a prevalence of 40.5 per 10,000. In 2007, the Centers for Disease Control and Prevention (CDC) reported a prevalence rate of 67 per 10,000 (CDC, 2007). More recently, the CDC reported 1 in 88 for the target age of 8 years (CDC, 2012) and 1 in 50 for children aged 6–17 (Blumberg et al., 2013).

Several explanations have been offered for this increase (King & Bearman, 2009). The explanation that has perhaps received the most attention is the changing diagnostic criteria, as the prevalence rate of autism has markedly increased since the publication of the DSM-IV and has continued to increase since the publication of the DSM-IV-TR. It is unclear what if any changes in prevalence will result from the changes in the DSM-5 (as described earlier).

Increasing public awareness of ASD may also play a role along with the increasing availability of services. Romanczyk and Callahan (2012) examined a popular press outlet, *Time* magazine. In the 1980s, 3 articles were published on ASD; in the 1990s, there were 11 articles and 2 cover stories; and in the 2000s, there were 73 articles and 6 cover stories. Such press coverage, along with frequent public service announcements and combined with organizations such as the American Academy of Pediatrics mandating multiple early identification screenings for every child during the first 3 years of age (Myers & Johnson, 2007), have all contributed to the prevalence rate increase.

15.5 Etiology and Theoretical Approach: The Context of Etiological Complexity

Because of the difficulty of specifying the disorder(s) and the resultant heterogeneity of the population of individuals with ASD, the causes of ASD are not yet known. Hypotheses come from research in neurobiology, genetics, and psychology, as well as sheer speculation. Given the heterogeneity of autism, there are probably multiple contributors to its development.

Research focused on genetic and neurobiological factors (e.g., Santangelo & Tsatsanis, 2005) has produced positive results. More than 15 genes have been associated with ASD

and twin studies provide support for the role of genetic factors. The current research in this area supports a biological link in the development of ASD, but the specific mechanism is not clear. Multiple genes have been implicated in the development of ASD, including chromosomes, 2, 7, 13, 15, 16, 17, and the X chromosome, with neuroanatomical features hypothesized to be linked to ASD including the brain stem, cortex, cerebellum, hippocampus, and amygdala.

However, drawing conclusions from these promising research areas is problematic in that sample sizes are often quite small. Also, participants are often skewed to the "higher-functioning" individuals with ASD. While understandable from the perspective of participant compliance requirements in relation to intensive assessment procedures, failure to include the full range of expression of ASD in the proportions proper to the ASD population makes generalizing the results of such studies difficult.

The current lack of understanding of specific etiological pathways, as well as multiple likely pathways, supports radical and at times pseudoscientific explanations. A prime example is the widespread assumption that the MMR (measles, mumps, and rubella) vaccine causes autism. It is a complex topic as it relates to the MMR vaccine itself, the timing and spacing of administration of other vaccinations, and the use of mercury-based thimerosal preservative in vaccines. The CDC (2015a) has been clear on this topic, indicating that the research does not support a link, as also illustrated by DeStefano, Price, and Weintraub (2013), whose recent epidemiological research suggests no link between vaccines and autism.

The number of poorly conceived theories of etiology and in turn treatment derived from such inadequate theories is unfortunately much greater than the number of intervention approaches derived from sound theory that has a basic foundation of research.

15.6 Defining Psychotherapy in the Context of Autism Spectrum Disorder

Because ASD represents a disorder or disorders characterized by a pervasive impact on the individual's functioning and because it occurs early in the individual's development and thus is a developmental disorder, a traditional definition of psychotherapy would exclude many individuals with ASD. An example of a dictionary definition of psychotherapy is: "Any alteration in an individual's interpersonal environment, relationships, or life situation brought about especially by a qualified therapist and intended to have the effect of alleviating symptoms of mental or emotional disturbance" (Merriam-Webster, 2017).

Issues of definition become more complex with regard to federally mandated education requirements and the concept of "medical necessity" as applied particularly to children with ASD (Romanczyk, Callahan, Turner, & Cavalari, 2014). For example, some definitions of "medical necessity" would appear to be quite inclusive. The American Academy of Pediatrics defines medical necessity for children as "health care interventions that are evidence-based, evidence-informed, or based on consensus advisory opinion and that are recommended by recognized health care professionals, such as the [American Academy of Pediatrics], to promote optimal growth and development in a child and to prevent, detect, diagnose, treat, ameliorate, or palliate the effects of physical, genetic, congenital, developmental, behavioral, or mental conditions, injuries, or disabilities" (Long, 2013, p. 400).

For the purposes of this chapter, given the context of ASD regarding heterogeneity in expression and given that ASD is an early onset disorder affecting many areas of functioning and development, the rather broad definition of psychotherapy offered by the American Psychological Association in August of 2012 appears appropriate:

> WHEREAS: evidence-based practice in psychology is "the integration of the best available research with clinical expertise in the context of patient characteristics, culture and preferences" (APA Task Force on Evidence Based Practice, 2006, p. 273);
>
> WHEREAS: a working definition for Psychotherapy is as follows: "Psychotherapy is the informed and intentional application of clinical methods and interpersonal stances derived from established psychological principles for the purpose of assisting people to modify their behaviors, cognitions, emotions, and/or other personal characteristics in directions that the participants deem desirable" (Norcross, 1990, p. 218–220);
>
> WHEREAS: a working definition for Treatment is as follows: Treatments when used in the context of health care, refer to any process in which a trained health-care provider offers assistance based upon his or her professional expertise to a person who has a problem that is defined as related to "health" or 'illness." In the case of "mental" or "behavioral" health, the conditions for which one may seek "treatment" include problems in living, conditions with discrete symptoms that are identified as or as related to illness or disease, and problems of interpersonal adjustment. The treatment consists of any act or services provided by a bonafide health provider intended to correct, change or ameliorate these conditions or problems (Beutler, 1983; Frank, 1973). (American Psychological Association, 2012)

Within this overall context, we differentiate types of intervention for ASD into two groups: (1) those that fit the American Psychological Association's definition and (2) the larger group that lies beyond and reflects the reality of the range of interventions provided to individuals with ASD.

There are no definitive surveys that address the prevalence of utilization of methods for ASD intervention. Sources of information come primarily from websites that receive comments from consumers as to the intervention choices they make. For example, the site About.com (2014) lists the "top 10" treatments:

1. applied behavior analysis;
2. speech therapy;
3. occupational therapy;
4. social skills therapy;
5. physical therapy;
6. play therapy;
7. behavior therapy;
8. developmental therapies;
9. visually based therapies;
10. biomedical therapies.

The Autism Speaks website gives a list of the top eight therapies (Peacock, 2017):

1. occupational therapy: 39%;
2. speech therapy: 27%;
3. applied behavior analysis (ABA) therapy: 15%;
4. social skills classes: 8%;
5. hippotherapy (occupational therapy through horseback riding): 2%;
6. the gluten-free casein-free diet: 2%;
7. psychiatrist/psychologist sessions: 2%;
8. Floor Time, relationship development intervention, picture exchange communication, swimming, and pivotal response training: five-way tie, each with 1%.

The CDC (2015b) lists 10 common treatments:

1. ABA;
2. developmental, individual-difference, relationship-based therapy, also called Floor Time;
3. Treatment and Education of Autistic and Related Communication-Handicapped Children (TEACCH);
4. occupational therapy;
5. sensory integration therapy;
6. speech therapy;
7. the Picture Exchange Communication System (PECS);
8. dietary approaches;
9. medication;
10. complementary and alternative treatments.

Finally, in a comprehensive review of websites, Romanczyk, Gillis, White, & DiGennaro (2008) categorized various treatment "types" according to website descriptions and attempted to classify them by strength of research support, as shown in Table 15.1.

As mentioned, all such compilations based on reviews of websites must be viewed cautiously as they reflect the limited available information about actual prevalence of use. However, the influence of website information on the decision-making processes of consumers, as well as professionals, should not be underestimated. Only a minority of consumers and service providers in the area of autism routinely rely on primary research sources. It is understandable, albeit undesirable, how the abundance of web-based information can be perceived as authoritative. This is further compounded by the lack of clarity and often intertwining contradictory information commonly observed in web-based writings. As an example, the following quote from the WebMD (2017) website illustrates this point: "There are a number of [sensory] therapies that have been successful at treating children with autism spectrum disorder. Although these therapies can help, there is no scientific documentation that sensory therapies are effective in treating the disorder." This type of juxtaposition—stating that a given therapy or approach is useful yet also acknowledging that there is not supporting research—is not unusual. Even more disturbing are simple noncritical lists, such as this from the CDC (2015b): "There are many different types of treatments available. For example, auditory training, discrete trial

Table 15.1 Autism spectrum disorder treatment "types" according to website descriptions, as classified by Romanczyk, Gillis, White, and DiGennaro (2008) according to strength of research of support.

Strength of Research Support	Treatment Types
Strong	• Discrete trial instruction (applied behavior analysis) • UCLA Young Autism Project (applied behavior analysis: Lovaas Institute)
Moderate	• Applied behavior analysis • Denver model • Douglass Developmental Disabilities Center • Lifeskills and Education for Students with Autism and other Pervasive Developmental Disorders (LEAP) • Pivotal Response Model (University of California at Santa Barbara) • Stimulants • Walden Early Childhood Programs (Emory University)
Limited	• Antihypertensive medication • Atypical antipsychotics • Autism Preschool Program • Child's Talk • Floor Time/Developmental Individual Difference • Incidental teaching • Oxytocin infusion • Princeton Child Development Institute (PCDI) • Scottish Centre for Autism • Touch therapy • Treatment and Education of Autism and Related Communication Handicapped Children (TEACCH) • Vitamin B
Limited with single subject only	• Exercise • Hyperbaric oxygen treatment
Mixed	• Anticonvulsants • Cyproheptadine • Gluten-free casein-free diet • Immunotherapy • Ketogenic diet • Sensory integration training • Vision therapy
Pragmatic or developmentally based interventions	• Haloperidol • Naltrexone • Risperdal • Speech therapy
Not recommended	• Antibiotics • Antifungal medication • Antiviral medication • Auditory integration training • Chelation • Dimethylglycine • Facilitated communication • Fenfluramine • Holding therapy • Megavitamin therapy • Music therapy • Secretin • Selective serotonin reuptake inhibitors • Tricyclic antibiotics • Vitamin B

Table 15.2 David and Montgomery's (2011) evaluative framework.

	Theory		
Therapeutic Package	**Well Supported**	**Equivocal: No, Preliminary, or Mixed Data**	**Strong Contradictory Evidence**
Well Supported	Category I	Category II	Category V
Equivocal: No, Preliminary, or Mixed Data	Category III	Category IV	Category VII
Strong Contradictory Evidence	Category VI	Category VIII	Category IX

training, vitamin therapy, anti-yeast therapy, facilitated communication, music therapy, occupational therapy, physical therapy, and sensory integration." This list from a well-known and generally trusted source of health-related information mixes together the full spectrum of interventions, including those that have very strong research support for efficacy and those that have very strong research evidence for lack of efficacy. The general climate within the broad area of ASD in both the public and government arenas seems to reflect a desire to be neutral on matters of efficacy research.

When large-scale research is conducted, variations in treatment category labels and operational definitions also cloud definitive conclusions. For example, a comparison chart generated by the Autism Society of America shows marked differences between intervention categories utilized in evidence-based intervention analyses.[3] According to our interpretation of their aggregated information, the Centers for Medicare and Medicaid Services, the National Autism Center, and the National Professional Development Center on ASD used similar intervention category labels for only 5 of the 34 listed treatment approaches. Even with those similarities, the operational definitions of what qualified as treatment under those categories differed for each analysis.

The purpose of this chapter is to utilize the framework developed by David and Montgomery (2011) to categorize treatment approaches along the two dimensions of theoretical support versus treatment efficacy support (see Table 15.2).

In order to provide an objective evaluation of the research base concerning treatments for ASD (of the type described in the introduction), an in-depth literature review was deemed necessary. Not surprisingly, there is a direct relationship between the quantity of methodologically sound research publications and a given treatment's efficacy. Treatments with a substantial research literature tend to fall in Categories I–IV, whereas numerous treatments summarized in the previous sections are dispersed among Categories VI, VII, VIII, and IX and are described primarily via web-based reports rather than peer-reviewed journal articles. In the field of ASD treatment, there are no treatments that have strong contradictory theory and well-supported treatment, and therefore Category V is not represented in our analysis.

15.7 Evidence-Based Treatment Review

An initial literature search was conducted using PsycINFO, Google Scholar, and the Cochrane Library with the search terms "autism treatment" and "review." Articles not in the English language were excluded.

A total of 2,452 articles were found in the initial search. Article titles and abstracts were then reviewed to exclude any clear mismatches with the psychosocial treatment

of ASD, including medical treatments, complementary and alternative medicine, single treatment studies, conceptual or theoretical reviews, book reviews, and reviews that focus on interventions targeting symptoms other than core symptoms of ASD. A total of 58 articles were identified for further screening.

Subsequent detailed screening of article content indicated that seven of the articles were not appropriate due to an emphasis on clinical process, due to their inclusion of a recommendation for the application of certain techniques, or due to a notation that the review was out of date and an updated edition was available. This screening process resulted in a total of 51 papers appropriate for analysis, two of which were large-scale focused review reports from organizations: the National Standards Project (National Autism Center [NAC], 2009) and Educating Children With Autism (Committee on Educational Interventions for Children With Autism: National Research Council, 2001).

The National Standards Project (NAC, 2009) is one of the most comprehensive reviews of treatment for individuals with ASD from birth through age 21. Due to the thoroughness of the NAC review, we chose to use the NAC's treatment category labels and definitions to categorize the content of the other 50 reviews. Since the National Standards Project covered a range of treatment targets that affect both core symptoms and associated features of ASD, treatment categories were maintained in the present analysis if the National Standards Project report indicated an emphasis on any one category addressing core symptoms of ASD. Specifically, all treatments described as skill-development interventions (with a focus on communication, interpersonal skills, personal responsibility, play, or self-regulation) were included. Further, treatments that purported to reduce restricted, repetitive, and nonfunctional behaviors, interests, and activities or those that emphasized sensory and emotional regulation were also included. Treatments focused exclusively on academic skills or that were clear nonpsychotherapeutic interventions (i.e., peer-mediated academic instructional intervention and massage therapy) were excluded. See Table 15.3 for all NAC categories included in this review. Readers are referred to the National Standards Project (NAC, 2009) for a full listing of treatment categories and definitions.

Table 15.3 National Standards Project treatment categories and definitions.

Treatment Category	Definition
Evidence Level: Established	
Antecedent package	This package involves modification of situational events that typically precede the occurrence of a target behavior. These alterations are made to increase the likelihood of success or reduce the likelihood of problems occurring. Treatments falling into this category reflect research representing the fields of applied behavior analysis, behavioral psychology, and positive behavior supports.
Behavioral package	These interventions are designed to reduce problem behavior and teach functional alterative behaviors or skills through the application of basic principles of behavior change. Treatments falling into this category reflect research representing the fields of applied behavior analysis, behavioral psychology, and positive behavior supports.

(*continued*)

Table 15.3 (*Continued*)

Treatment Category	Definition
Comprehensive behavioral treatment for young children	This treatment reflects research from comprehensive treatment programs that involve a combination of applied behavior analytic procedures, which are delivered to young children (generally under the age of 8). These treatments may be delivered in a variety of settings. All of the studies falling into this category met the strict criteria of (1) targeting the defining symptoms of ASD, (b) having treatment manuals, (c) providing treatment with a high degree of intensity, and (d) measuring the overall effectiveness of the program. These treatments may be referred to as ABA programs, behavioral inclusive programs, or early intensive behavioral interventions.
Joint attention intervention	These interventions involve building foundational skills involved in regulating the behaviors of others. Joint attention often involves teaching a child to respond to the nonverbal social bids of others or to initiate joint attention interactions. Examples include pointing to objects, showing items/activities to another person, and following eye gaze.
Modeling	These interventions rely on an adult or peer providing a demonstration of the target behavior that should result in an imitation of the target behavior by the individual with ASD. Modeling can include simple and complex behaviors. These interventions are often combined with other strategies, such as prompting and reinforcement. Examples include live modeling and video modeling.
Naturalistic teaching strategies	These interventions involve using primarily child-directed interactions to teach functional skills in the natural environment. These interventions often involve providing a stimulating environment, modeling how to play, encouraging conversation, providing choices and direct/natural reinforcers, and rewarding reasonable attempts. Examples of this type of approach include but are not limited to focused stimulation, incidental teaching, milieu teaching, embedded teaching, and response education and prelinguistic milieu teaching.
Peer training package	These interventions involve teaching children without disabilities strategies for facilitating play and social interactions with children on the autism spectrum. Peers often include classmates or siblings. When both initiation training and peer training were components of treatment in a given study, the study was coded as "peer training package." These interventions may include components of other treatment packages (e.g., self-management for peers, prompting, reinforcement). Common names for intervention strategies include peer networks, circle of friends, buddy skills package, Integrated Play Groups[M], peer initiation training, and peer-mediated social interactions.
Pivotal response treatment	This treatment is also referred to as pivotal response teaching and pivotal response training (PRT). It focuses on targeting "pivotal" behavioral areas, such as motivation to engage in social communication, self-initiation, self-management, and responsiveness to multiple cues, with the development of these areas having the goal of very widespread and fluently integrated

Table 15.3 (*Continued*)

Treatment Category	Definition
	collateral improvements. Key aspects of PRT intervention delivery also focus on parental involvement in the intervention delivery and on intervention in the natural environment (such as homes and schools) with the goal of producing naturalized behavioral improvements.
Schedules	These interventions involve the presentation of a task list that communicates a series of activities or steps required to complete a specific activity. Schedules are often supplemented by other interventions, such as reinforcement. Schedules can take several forms, including written words, pictures or photographs, or work stations.
Self-management	These interventions involve promoting independence by teaching individuals with ASD to regulate their behavior by recording the occurrence/nonoccurrence of the target behavior, and securing reinforcement for doing so. Initial skills development may involve other strategies and may include the task of setting one's own goals. In addition, reinforcement is a component of this intervention, with the individual with ASD independently seeking and/or delivering reinforcers. Examples include the use of checklists (using checks, smiley/frowning faces), wrist counters, visual prompts, and tokens.
Story-based intervention package	These treatments involve a written description of the situations under which specific behaviors are expected to occur. Stories may be supplemented with additional components (e.g., prompting, reinforcement, discussion). Social Stories are the most well-known story-based interventions and they seek to answer the "who," "what," "when," "where," and "why" in order to improve perspective-taking.
Evidence Level: Emerging	
Augmentative and alternative communication	These interventions involve the use of high or low technologically sophisticated devices to facilitate communication. Examples include but are not restricted to pictures, photographs, symbols, communication books, computers, and other electronic devices.
Cognitive–behavioral intervention packages	These interventions focus on changing everyday negative or unrealistic thought patterns and behaviors with the aim of positively influencing emotions and/or life functioning.
Developmental relationship-based treatments	These treatments involve a combination of procedures that are based on developmental theory and emphasize the importance of building social relationships. These treatments may be delivered in a variety of settings (e.g., home, classroom, community). All of the studies falling into this category met the strict criteria of (1) targeting the defining symptoms of ASD, (2) having treatment manuals, (3) providing treatment with a high degree of intensity, and (4) measuring the overall effectiveness of the program (i.e., studies that measure subcomponents of the program are listed elsewhere in this report). These treatment programs may also be referred to as the Denver model, DIR (Developmental, Individual Differences, Relationship-based)/Floortime, the Relationship Development Intervention, or responsive teaching.

(*continued*)

Table 15.3 (*Continued*)

Treatment Category	Definition
Exercise	These interventions involve an increase in physical exertion as a means of reducing problem behaviors or increasing appropriate behavior.
Imitation-based interaction	These interventions rely on adults imitating the actions of the child.
Initiation training	These interventions involve directly teaching individuals with ASD to initiate interactions with their peers.
Language training (production)	These interventions have as their primary goal to increase speech production. Examples include but are not restricted to echo-relevant word training, oral communication training, oral verbal communication training, structured discourse, simultaneous communication, and individualized language remediation.
Language training (production and understanding)	These interventions have as their primary goals to increase both speech production and understanding of communicative acts. Examples include but are not restricted to total communication training, position object training, position self-training, and language programming strategies.
Multicomponent package	These interventions involve a combination of multiple treatment procedures that are derived from different fields of interest or different theoretical orientations. These treatments do not better fit one of the other treatment "packages" in this list, nor are they associated with specific treatment programs.
Picture Exchange Communication System	This treatment involves the application of a specific augmentative and alternative communication system based on behavioral principles that are designed to teach functional communication to children with limited verbal and/or communication skills.
Reductive package	These interventions rely on strategies designed to reduce problem behaviors in the absence of increasing alternative appropriate behaviors. Examples include but are not restricted to water mist, behavior chain interruption (without attempting to increase an appropriate behavior), protective equipment, and ammonia.
Scripting	These interventions involve developing a verbal and/or written script about a specific skill or situation that serves as a model for the child with ASD. Scripts are usually practiced repeatedly before the skill is used in the actual situation.
Sign instruction	These interventions involve the direct teaching of sign language as a means of communicating with other individuals in the environment.
Social communication intervention	These psychosocial interventions involve targeting some combination of social communication impairments, such as pragmatic communication skills, and the inability to successfully read social situations. These treatments may also be referred to as social pragmatic interventions.
Social skills package	These interventions seek to build social interaction skills in children with ASD by targeting basic responses (e.g., eye contact, name response) up to complex social skills (e.g., how to initiate or maintain a conversation).

Table 15.3 (*Continued*)

Treatment Category	Definition
Structured teaching	Based on neuropsychological characteristics of individuals with autism, this intervention involves a combination of procedures that rely heavily on the physical organization of a setting, predictable schedules, and individualized use of teaching methods. These procedures assume that modifications in the environment, materials, and presentation of information can make thinking, learning, and understanding easier for people with ASD if they are adapted to individual learning styles of autism and individual learning characteristics. All of the studies falling into this category meet the strict criteria of (a) targeting the defining symptoms of ASD; (2) having treatment manuals; (3) providing treatment with a high degree of intensity; and (4) measuring the overall effectiveness of the program (i.e., studies that measure subcomponents of the program are listed elsewhere in this report). These treatment programs may also be referred to as TEACCH (Treatment and Education of Autistic and Related Communication-Handicapped Children).
Technology-based treatments	These interventions require the presentation of instructional materials using the medium of computers or related technologies. Examples include but are not restricted to Alpha Program, Delta Messages, the Emotion Trainer Computer Program, pager, robot, or a PDA (personal digital assistant). The theories behind technology-based treatments may vary but they are unique in their use of technology.

ABA = applied behavior analysis; ASD = autism spectrum disorder.

An umbrella review was conducted on the systematic review papers by reviewing the content and findings and categorizing the support evidenced for various treatments in each review. The focus of the umbrella review was on established levels of evidence for psychosocial therapeutic interventions, as absence of evidence at any level would imply both a lack of theoretical and clinical significance given the status of the field. It is important to note that the National Standards Project identified academic interventions, auditory integration training, facilitated communication, gluten-free and casein-free diet, and sensory integrative packages as having unestablished levels of evidence (NAC, 2009). More importantly, it specified that its evaluation of these treatments was accompanied with the caution that several professional organizations and a limited amount of available research have indicated that the treatments either are harmful and should not be used or have no documented benefit.

For the purposes of the present review, formulaic values were applied to the findings to evaluate the overall efficacy of each reviewed treatment as follows:

- *Support (established).* Rating of 1; the majority of studies summarized within the review for that treatment demonstrated a positive outcome (more than three studies required, with 70% or more studies with positive outcome).
- *Mixed (emerging).* Rating of 0.5; both positive and negative outcomes were reported for a treatment within the same review (at least three studies required).
- *Minimal evidence (unclear).* Rating of 0.25; positive outcome reported, but for two or fewer studies.

The formulaic values were then summed across all reviews to generate a total evaluative score for each treatment. The treatments were then ordered in descending score order (see Table 15.4). Although our method precludes an in-depth analysis of each treatment area, it does permit evaluation of the most frequently cited treatment types with extensive literature coverage and related effectiveness in Categories I–IV in the evidence matrix.

15.7.1 Results

It is important to note that the existing reviews were not designed to match the treatment categories used by the NAC. Therefore, exact alignment of the reviews with the available categories might not fully represent the treatment mode used for delivery. For example, it is common for technology-based treatments and video-modeling approaches to be based on the principles of behaviorism and they may also be delivered using behavioral methodology; however, computer-based programs and modeling approaches were categorized separately from behavioral approaches for the purposes of the current review. In some cases, the identified categories do overlap with a particular theoretical background and so we have combined these categories under their various larger headings in Table 15.5 to aid the interpretation of the findings.

It is clear that behavioral approaches are overwhelmingly more frequently utilized, with a great deal of support for their effectiveness in the treatment of the core symptoms of ASD. Speech and language training and naturalistic teaching approaches are the next most commonly reviewed methodology with support for intervention, although these interventions typically focus on either communication or play and interpersonal skills separately.

15.7.2 Framework Categorization

15.7.2.1 Worst evaluation category

With respect to Category IX (see Table 15.2), the intersection of strongly negative efficacy research and strongly negative support of theory or conceptual rationale, one treatment for ASD that has existed for over 30 years and remains "popular" has unique status. The American Psychological Association (2017) has issued the statement that "facilitated communication is a controversial and unproved communication procedure with no scientifically demonstrated support for its efficacy." Similarly, and again quite uniquely, much the same policies have been adopted by the American Academy of Child and Adolescent Psychiatry, the American Association on Intellectual and Developmental Disabilities, and the American Academy of Pediatrics ("Facilitated Communication: Courts Say 'No,'" 1992).

Although they have received less attention from professional organizations, procedures such as auditory integration, dolphin-assisted therapy, chiropractic manipulation, and holding therapy are also appropriately positioned in Category IX.

15.7.2.2 Best evaluation category

The best therapeutic packages with respect to efficacy and outcome (i.e., those in Category I; see Table 15.2) are limited in number. Concerning these treatments, there is a very strong convergence of the research evidence base and there have been large-scale

Table 15.4 Treatment categories with analysis ratings and distribution of support.

National Autism Center Categories: Core ASD Symptoms	*N*	Evaluative Score	% Support	% Mixed	% Minimal
Behavioral package	19	18.50	94.7	5.3	0.0
Social skills package	16	13.00	68.8	18.8	12.5
Modeling	14	12.00	78.6	14.3	7.1
Comprehensive behavioral treatment	14	11.50	78.6	21.4	0.0
Peer training package	10	7.50	60.0	20.0	20.0
Antecedent package	8	7.25	87.5	0.0	12.5
Naturalistic teaching strategies	8	7.25	87.5	0.0	12.5
Structured teaching	8	5.75	50.0	37.5	12.5
Cognitive–behavioral package	9	5.50	33.3	44.4	22.2
Story-based intervention package	7	4.75	42.9	42.9	14.3
Technology-based/computer-assisted	6	4.75	66.7	16.7	16.7
Pivotal response treatment	6	4.50	66.7	0.0	33.3
Picture Exchange Communication System	7	4.25	42.9	14.3	42.9
Self-management	6	3.75	50.0	0.0	50.0
Augmentative and alternative communication	5	3.75	60.0	20.0	20.0
Schedules	5	3.50	60.0	0.0	40.0
Scripting	4	3.50	75.0	25.0	0.0
Language training (production and understanding)	4	2.75	50.0	25.0	25.0
Developmental relationship-based treatments	4	2.50	25.0	75.0	0.0
Multicomponent package	3	2.50	66.7	33.3	0.0
Reductive package	3	2.50	66.7	33.3	0.0
Sign instruction	3	2.50	66.7	33.3	0.0
Social communication intervention	3	2.50	66.7	33.3	0.0
Joint attention intervention	2	2.00	100.0	0.0	0.0
Exercise	1	0.50	0.0	100.0	0.0
Imitation-based interaction	1	0.50	0.0	100.0	0.0
Initiation training	1	0.50	0.0	100.0	0.0
Language training (production)	1	0.50	0.0	100.0	0.0

Treatment categories are organized by evaluative score in descending order. Evidence categories are defined as follows: support = more than three studies within a review with 70% or more positive outcome reported; mixed = at least three studies within a review with both positive and negative outcomes reported; minimal = two or fewer studies within a review that report some evidence of positive outcome. ASD = autism spectrum disorder.

Table 15.5 Treatment categories as organized by major theoretical approach.

Theoretical Domain	*N*	Overall Score	% Support	% Mixed	% Minimal
Behavioral Approaches • Behavioral package • Social skills package • Modeling • Comprehensive behavioral treatment • Peer training package • Antecedent package • Structured teaching • Pivotal response treatment • Picture Exchange Communication System • Self-management • Augmentative and alternative communication • Schedules • Scripting • Reductive package • Joint attention intervention • Imitation-based interaction • Initiation training	129	104.25	70.54	15.50	14.73
Speech and Language Training • Language training (production) • Language training (production and understanding) • Sign instruction • Social communication intervention	11	8.25	55.00	36.00	9
Naturalistic teaching approach	8	7.25	87.50	0.00	12.50
Cognitive–behavioral approach	9	5.50	33.30	44.40	22.20
Story-based intervention approach	7	4.75	42.90	42.90	14.30
Technology/computer-assisted approach	6	4.75	66.70	16.70	16.70
Developmental relationship-based approach	4	2.50	25.00	75.00	0.00
Multicomponent package approach	3	2.50	66.70	33.30	0.00
Exercise approach	1	0.50	0.00	100.00	0.00

Domains are organized by overall evaluative score in descending order. Evidence categories are defined as follows: support = more than three studies within a review with 70% or more positive outcome reported; mixed = at least three studies within a review with both positive and negative outcomes reported; minimal = two or fewer studies within a review that report some evidence of positive outcome.

literature and policy reviews. Examples of such large-scale reviews, representing, professional, state, and federal organizations are Missouri Department of Mental Health (2012), Mudford et al. (2009), Myers and Johnson (2007), NAC (2009), National Research Council (2001), and New York State Department of Health, Early Intervention Program (1999).

Based upon the literature review already described and these literature and policy reviews, the therapeutic approach applied behavior analysis falls solidly in Category I due to the unique convergence of an extensive well-researched theoretical base and the most extensive treatment outcome research literature. It is the quality, quantity, and highly replicated nature of the research base for applied behavior analysis that places it in Category I.

15.7.2.3 Mixed evaluation categories

Because therapeutic packages for ASD are often composed of multiple elements with varying degrees of efficacy research for the components and varying degrees of theoretical support for the conceptual elements, Categories II, III, and IV are difficult to discriminate in the context of ASD. This is where there is interaction between age of individual, severity of expression, verbal versus nonverbal, cognitive development level, and so on, and the breadth of the conceptual basis for the intervention approach. Given these limitations and complicating factors, the treatments that fall into these categories are:

- speech and language training (Category II);
- naturalistic teaching (Category III);
- story-based, computer-assisted, developmental relationship-based, and exercise approaches (Category IV).

15.7.2.3.1 Category II: Speech and language training

Speech and language training—including language training (production only, and production and understanding), sign instruction, and social communication interventions—is assigned to Category II. The highest evaluative score in this theoretical approach was found for language training involving both speech production and communicative understanding. Although the score was higher compared to the scores for other speech and language approaches, only 4 of the 11 studies addressed this type of intervention and only 2 of those studies fell within the category of "support" according to the present analysis. Nevertheless, 6 of the 11 studies under the speech and language training approach fell within the "support" category and four fell within the "mixed" category, indicating that the majority of available studies evidence some level of support based on positive outcomes documented in the literature.

15.7.2.3.2 Category III: Naturalistic teaching

Naturalistic teaching involves interventions that are primarily child-directed and target teaching functional skills in the natural environment (see Table 15.3 for a full description of this category). According to the present analysis, this treatment approach was associated with positive outcomes in seven of eight of the studies reviewed, but, given that there were fewer than ten studies evaluating this approach, it was assigned to Category III. Naturalistic teaching strategies, while typically packaged as different from behavioral approaches, tend to involve components of behavioral strategies, such as reinforcement and teaching in the target environment. Therefore, although it is often designated as a separate treatment category, this approach does utilize numerous techniques based on the strong theoretical foundation of behavioral approaches. With that

said, the limited number of published research studies precludes our ability to establish firm conclusions about the therapeutic package's effectiveness, indicating an equivocal level of support until additional research is conducted.

15.7.2.3.3 Category IV: Story-based, computer-assisted, developmental relationship-based, and exercise approaches

Category IV includes the remaining treatment approaches summarized in Table 15.4 and Table 15.5 story-based, computer-assisted, developmental relationship-based, and exercise approaches. These intervention types had fewer than 10 published studies and, with the exception of computer-assisted approaches, had a larger distribution of "mixed" or "minimal" support than studies in Categories II and III. The approaches that are classified in Category IV do not have a strong basis of theory for their use and the presently limited research evidence suggests mostly tentative positive outcomes at this time. Given this, the published evidence falls in the equivocal category until further research is conducted to substantiate a stronger base of therapeutic effectiveness. Therefore, the most appropriate classification for these approaches is Category IV, which represents both an equivocal theoretical basis and equivocal therapeutic effectiveness.

15.7.2.3.4 Cognitive–behavioral therapy (Category III)

Because cognitive–behavioral approaches represent one of the most clearly traditional forms of psychotherapeutic treatment used for individuals with ASD, cognitive–behavioral therapy (CBT) is presented separately and a more in-depth review and discussion of the extant literature is offered. In addition to examining review articles, we conducted a secondary literature search using the terms "autism" and "cognitive–behavioral therapy" in PsycINFO to obtain a subset of CBT studies that addressed core symptoms of ASD. A total of 472 publications were initially identified. Due to the large number of publications that targeted comorbid anxiety disorder symptoms, operands of "not anxiety" and "not obsessive compulsive disorder" were added to the search parameters, which resulted in 363 publications. When the search was limited to peer-reviewed journal articles, there were 271 publications. Article titles and abstracts were then reviewed to exclude any clear mismatches with the topic of focused research on CBT for ASD core symptoms.

Eleven papers were identified that addressed core symptoms of ASD. Five papers were kept for further review as the other six studies were already summarized in obtained systematic reviews. One study was excluded because it referenced using "behavioral and cognitive techniques" but did not provide sufficient detailed information about those techniques to establish that the described intervention was in fact CBT. Another paper was excluded because it was an overview of the Program for the Education and Enrichment of Relational Skills and did not contain an explicit review of supporting evidence. The remaining three studies are included with the three available review papers and are described in more detail below.

In terms of existing review papers, Binnie and Blainey (2013) outline seven studies that utilized CBT in the context of case studies and quasi-experiments (nonrandomized, nonblind controlled trials). Their findings suggest that CBT may be effective in treating comorbid mental health issues in individuals with ASD but that their effectiveness in treating core symptoms is unclear. A more in-depth review by Danial

and Wood (2013) covered a separate set of papers and specifically divided studies into those that addressed comorbid anxiety ($n = 11$), disruptive behavior ($n = 2$), and core symptoms of ASD ($n = 10$). Their conclusions indicate that CBT may be a useful method of treatment for anxiety in high-functioning individuals with ASD aged between 7 and 14 years old, but that none of the 11 studies reviewed would meet the American Psychological Association's Division 12 criteria for "well-established" treatments. With regard to disruptive behavior, the two studies reviewed focused on CBT-based treatment of anger management for 45 children with Asperger's disorder between the ages of 10 and 14 years (Sofronoff, Attwood, Hinton, & Levin, 2007) and mindfulness treatment for three adolescents with high levels of aggression (Singh et al., 2011). Both studies cited marked improvements in reducing problematic outbursts, but the limited number of studies available regarding disruptive behavior limits the possibility of drawing strong conclusions about effectiveness. Although externalizing behaviors are not core features of ASD, they are evidence of the difficulties in emotional self-regulation that are related to core social deficits, and further research in this area is needed. Finally, with regard to CBT for core ASD symptoms, the 10 reviewed studies for children with ASD between 6 and 17 years of age indicate promising findings. CBT approaches to treat core ASD symptoms primarily emphasize social responsiveness training to build a better understanding of one's own thoughts, goals, and intentions, with the assumption that such an understanding will allow for improved social–emotional reciprocity with others. In their review, Danial and Wood (2013) indicate that, although positive outcomes were reported across studies, the treatment approaches, research designs, treatment settings, and outcome measurements varied widely. The authors argue that these vast differences between studies make comparisons between treatment packages difficult and strong conclusions regarding effectiveness tentative at best.

Danial and Wood's (2013) findings overlap substantially with an earlier review conducted by Rotheram-Fuller and MacMullen (2011) on CBT for children with ASD. In this earlier review, the authors found that CBT, when delivered in school and clinic settings, was effective in reducing comorbid anxiety symptoms and increasing social interactions. Their review included only five articles because they focused on studies with a sample size of more than 20 individuals with ASD and the emphasis of the review was to recommend adaptations of CBT for implementation in the school environment. Epp (2008) also describes the success of a school-based social skills group model (SuperKids) that capitalizes on emotional insight techniques generated from cognitive–behavioral strategies. The SuperKids afterschool program assigns children between the ages of 6 and 12 years to groups of six who meet with a master's-level lead therapist for weekly 1-hour sessions throughout the school year (September–May). In a pre–post analysis of 44 children, parent-reported scores on the Social Skills Rating System (Gresham & Elliott, 1990) showed significant improvements in the areas of assertion, internalizing problems, hyperactivity, and overall problem behaviors. These results suggest that school-based group CBT interventions can be effective and can offer a much-needed service within the child's familiar school setting, but that overall improvements with regard to core social deficits are still minimal at best.

The two more recent individual studies included in our literature review show a clear effort by researchers to address the absence of evidence for the treatment of core symptoms of ASD with CBT. Andrews, Attwood, and Sofronoff (2013) implemented

a cognitive–behavioral intervention targeting appropriate displays of affection with children between the ages of 7 and 12 years who has been diagnosed with Asperger's disorder. The participants were randomly assigned to either an intervention ($n = 29$) or a waitlist ($n = 29$) condition. In the intervention group, children were assigned to nine groups consisting of three to four children and each child was assigned two to three therapists. Parents also attended large group parent sessions with two therapists. The intervention was conducted for 2 hours per session over the course of five consecutive Saturdays. Results indicated that participants in the CBT group were able to demonstrate more appropriate expression of affection to others, but acceptance of affection from others showed no gains. Twenty-three of the children in the intervention condition were available for follow-up assessment, and data indicated that gains were maintained 3 months after treatment concluded. Given these findings, a focused intervention targeting a subset of socioemotional skills for the equivalent of at least 10 hourly sessions shows some promise in addressing social deficits in ASD.

Wood, Fujii, Renno, and Van Dyke (2014) summarize successful implementation of 90-minute weekly parent-and-child sessions of the Building Confidence CBT program delivered over the course of 32 weeks (n =7) compared to community mental health (CMH; $n = 6$; 16 weeks of treatment). Child age ranged from 6 to 10 years. The researchers' findings indicated that children in the CBT group were observed to engage in significantly less solitary behavior and more positive and appropriate peer interactions when observed on two separate days at baseline and posttreatment during school recess periods. These gains were substantially greater than those in the CMH group. Despite these positive outcomes, the authors caution that the small sample size, the fact the observations were conducted only in school playgrounds, the disparity in treatment length between the CBT and CMH groups, and the absence of follow-up assessments limit the strengths of their study.

Although the reviews and individual studies of CBT implementation we have summarized show some promise for addressing core symptoms and associated features of ASD, it is clear that remediation of ASD symptoms requires more long-term and intensive treatment than is typically considered standard for CBT intervention. Many authors cite that improvements were noted in comorbid conditions but not necessarily in core symptoms. When core symptoms were impacted, the changes were not global and only a focused skill set was improved without consistent, clear substantiation that gains were maintained posttreatment. Therefore, although CBT is well grounded in theory and has shown excellent evidence of efficacy for other psychological disorders, it remains in the equivocal category due to mixed evidence and unclear gains in the core symptom domains of ASD.

15.7.2.4 Empty categories

The matrix evaluating the intersection of theoretical support and therapeutic package support had several categories empty in the context of ASD. Specifically, Categories V, VI, VII, and VIII (see Table 15.2) did not have appropriate entries.

This is reflective of the state of the field wherein there are a plethora of "theories" but a paucity of positive evidence-based outcome research or any well-controlled outcome research for the theories, and a similar lack of research specifically on the theories themselves. This results in the present analyses falling into Categories I, II, III, IV, and IX.

15.8 Discussion

Because ASD is a complex, heterogeneous, neurodevelopmental cluster of disorders that primarily expresses as dysfunction in social relationships and is associated with a variety of comorbid disorders, discussion of effective treatment is complex. This complexity provides fertile ground for pseudoscientific approaches that offer simple "solutions" and for approaches that, while perhaps well-intentioned, lack appropriate outcome research support.

15.8.1 Implications for Practice and Research

The purpose of this chapter was to conduct a review of the psychosocial therapeutic interventions for individuals with ASD. It is our hope that the chapter will inform practitioners, students, and researchers about current available effective treatments for individuals with ASD. The information contained in this chapter is intended to increase familiarity with the broad array of treatments specifically in order to assist the practicing clinician in the referral process. It is likely that clinicians will see an increase in the number of clients who are diagnosed with ASD as the importance of comorbid conditions receives improved recognition and attention. Therefore, the content of this chapter adds valuable information about effective treatments that should be considered in providing clinical services.

The reader can readily observe several treatments as being poorly substantiated in the literature. Facilitated communication and auditory integration training are two examples. There are several references available to help clinicians become increasingly familiar with pseudoscientific treatments and the field of ASD. Because new treatments for ASD are continuously developed and popularized, for better or worse, it is imperative that clinicians, students, and researchers continue to critically evaluate the research literature for these treatments for support or lack thereof in order to guide families in the process of selecting treatment and making decisions.

The treatment with the most support and the longest history of effectiveness, with respect to both theory and treatment effectiveness, is applied behavior analysis. Clinicians wishing to serve this population with this type of treatment are strongly urged to consider furthering their training in applied behavior analysis. This is not a small task, as most families seek, and agencies and insurance companies typically require, national certification credentials to indicate a specialization in applied behavior analysis. Becoming certified requires a number of additional courses taught at the graduate level as well as approximately 1 year of supervised clinical experience. There is also a certification exam and several additional requirements along with ongoing requirements to maintain certification. Most practicing clinicians understandably do not choose to become board-certified behavior analysts. As an alternative, it is important to develop relationships with individuals who are board-certified behavior analysts if one is providing services to individuals with ASD. To find board-certified behavior analysts, one can utilize the Behavior Analyst Certification Board's website (https://bacb.com) or contact and become familiar with organizations such as the Association of Professional Behavior Analysts. Forming such professional relationships is crucial for assisting families with treatment options and treatment coordination, especially in the period soon after a young child is diagnosed with ASD.

As the evidence presented in this chapter indicates, speech and language training treatments also yield some positive support for consideration. Both board-certified behavior analysts and speech-language pathologists conduct or provide interventions to improve communication and language skills. Part of being an effective clinician for the ASD population is developing a network of providers who are active in implementing evidence-based treatments for ASD. It is our hope that this chapter will increase familiarity with these treatments in order to help clinicians to identify other professionals who are conducting appropriately supportive treatments.

Given the intended audience of this book, a discussion of more traditional psychotherapy for ASD is warranted. As one can conclude from the evidence presented, there is increased interest in the adaptation or modification of CBT for individuals with ASD. The current available evidence is mixed for CBT treatment. However, this is a growing field and future research may yield new and exciting clinical implications for clinicians. Most likely this will predominately be in the area of comorbid conditions. It is noteworthy that many individuals with ASD develop comorbid disorders throughout their lifetime, starting at a young age. Although the review for this chapter was focused on interventions that address core ASD symptoms, treatment of comorbid conditions can be essential to overall positive outcome for the individual. Comorbid conditions such as high rates of anxiety and depression, as well as relationship dysfunction (friendships and intimate relationships), are of particular concern. These and other areas of comorbidity are sources of struggle for individuals with ASD, and the development of evidence-based interventions is needed.

While there is clear research support for the many interventions that have been developed for ASD based on the theory and principles of ABA, and while (importantly) ABA-based interventions can address core symptoms of ASD, additional evidence-based approaches are needed. The relatively recent increase in research on CBT treatment—both for the core ASD symptom of social development, which may be promising for individuals at specific developmental levels, and for the comorbid conditions commonly associated with ASD—is encouraging. The research on CBT is important and has implications for specific individuals in the ASD population as well as for clinicians.

In general, there are numerous psychosocial therapeutic interventions for the treatment of core symptoms and young children with ASD. Areas where there is little research and where the research needs to expand include psychosocial therapeutic interventions for adolescents and both younger and older adults with ASD, as well as health and medical issues across the lifespan. Given the success of ABA and the increasing success of CBT, collaboration between these two fields might facilitate the development and establishment of empirically supported treatments for both core and associated symptoms of ASD. Regardless, this is a growing and aging population requiring additional treatment research to meet many areas of need.

15.9 Conclusion

Clinicians have central roles in the assessment and treatment of individuals with ASD. The importance of the clinician's role as a diagnostician and referral or information source should not be underestimated. The information presented in this chapter will

hopefully increase the awareness of common but unsupported treatments versus well-supported treatments for the core symptoms of ASD, to help guide the decision-making process and thereby improve the quality of life of individuals with ASD. There is a need for increased research on the role of the clinician in providing direct care services for comorbid problems and core problems that many people with ASD experience throughout their lifespan. However, it is important to stress that the currently available CBT interventions do not fully address the core symptoms of ASD. Based on the information presented, it is imperative that CBT clinicians and other clinicians reach out to other professionals who can implement empirically supported interventions such as those in the field of applied behavior analysis. Collaborations for scientific research between the fields of ABA and CBT will likely improve the overall quality of life of individuals with ASD, their families, and significant others.

Notes

1. From this point forward, the term ASD will be used to reflect the current DSM-5 designation, while acknowledging that it is not fully synonymous with previous diagnostic terms.
2. An important cautionary note is that, as diagnostic criteria are changing, interpretation of prevalence of comorbid disorders must be interpreted in that context.
3. Chart was accessed August 20, 2014, but is no longer available at the time of publication.

References

About.com. (2014). Autism treatment options. Retrieved August 20, 2014, from http://autism.about.com/od/treatmentoptions/tp/topdevandbehav.htm

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd rev. ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed. TR). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.

American Psychological Association. (2012). "Recognition of Psychotherapy Effectiveness." Retrieved September 29, 2017, from http://www.apa.org/about/policy/resolution-psychotherapy.aspx

American Psychological Association. (2017). "Chapter XI: Scientific affairs." Retrieved October 2, 2017, from http://www.apa.org/about/policy/chapter-11.aspx

American Psychological Association Task Force on Evidence-Based Practice. (2006). Evidence-based practice in psychology. *American Psychologist*, *61*, 271–285. doi:10.1037/0003-066X.61.4.271

Andrews, L., Attwood, T., & Sofronoff, K. (2013). Increasing the appropriate demonstration of affectionate behavior, in children with Asperger syndrome, high functioning autism, and PDD-NOS: A randomized controlled trial. *Research in Autism Spectrum Disorders*, *7*, 1568–1578. doi:10.1016/j.rasd.2013.09.010

Bertrand, J., Mars, A., Boyle, C., Bove, F., Yeargin-Allsopp, M., & Decoufle, P. (2001). Prevalence of autism in a United States population: The Brick Township, New Jersey, investigations. *Pediatrics*, *108*, 1155–1161. doi:10.1542/peds.108.5.1155

Beutler, L. E. (1983). *Eclectic psychotherapy: A systematic approach*. New York, NY: Pergamon Press.

Binnie, J., & Blainey, S. (2013). The use of cognitive behavioural therapy for adults with autism spectrum disorders: A review of the evidence. *Mental Health Review Journal*, *18*(2), 93–104. doi:10.1108/MHRJ-05-2013-0017

Blumberg, S. J., Bramlett, M. D., Kogan, M. D., Schieve, L. A., Jones, J. R., & Lu, M. C. (2013). *Changes in prevalence of parent-reported autism spectrum disorder in school-aged US children: 2007 to 2011–2012*. Washington, DC: Centers for Disease Control and Prevention.

Bradley, E. A., Summers, J. A., Wood, H. L., & Bryson, S. E. (2004). Comparing rates of psychiatric and behavior disorders in adolescents and young adults with severe intellectual disability with and without autism. *Journal of Autism and Developmental Disorders*, *34*(2), 151–161.

Centers for Disease Control and Prevention. (2007). Prevalence of autism spectrum disorders: Autism and Developmental Disabilities Monitoring Network, 14 sites, United States, 2002. *Morbidity and Mortality Weekly Report*, *56*(1), 12–28.

Centers for Disease Control and Prevention. (2012). Prevalence of autism spectrum disorders: Autism and Developmental Disabilities Monitoring Network, 14 sites, United States, 2008. *Morbidity and Mortality Weekly Report*, *61*(3), 1–19.

Centers for Disease Control and Prevention. (2015a). Vaccines do not cause autism. Retrieved October 3, 2017, from https://www.cdc.gov/vaccinesafety/concerns/autism.html

Centers for Disease Control and Prevention. (2015b). "Autism spectrum disorder (ASD): Treatment." Retrieved September 29, 2017, from https://www.cdc.gov/ncbddd/autism/treatment.html

Croen, L. A., Grether, J. K., Hoogstrate, J., & Selvin, S. (2002). The changing prevalence of autism in California. *Journal of Autism and Developmental Disorders*, *32*(3), 207–215. doi:10.1023/a:1015453830880

Danial, J. T., & Wood, J. J. (2013). Cognitive behavioral therapy for children with autism: Review and considerations for future research. *Journal of Developmental & Behavioral Pediatrics*, *34*(9), 702–715. doi:10.1097/dbp.0b013e31829f676c

David, D., & Montgomery, G. (2011). The scientific status of psychotherapies: A new evaluative framework for evidence-based psychosocial interventions. *Clinical Psychology: Science and Practice*, *18*, 88–99. doi:10.1111/j.1468-2850.2011.01239.x

DeStefano, F., Price, C. S., & Weintraub, E. S. (2013). Increasing exposure to antibody-stimulating proteins and polysaccharides in vaccines is not associated with risk of autism. *Journal of Pediatrics*, *163*(2), 561–567. doi:10.1016/j.jpeds.2013.02.001

Eagle, R., Romanczyk, R. G., & Lenzenweger, M. (2010). Classification of children with autism spectrum disorders: A finite mixture modeling approach to heterogeneity. *Research in Autism Spectrum Disorders*, *4*(4), 772–781. doi:10.1016/j.rasd.2010.02.001

Epp, K. M. (2008). Outcome-based evaluation of a social skills program using art therapy and group therapy for children on the autism spectrum. *Children & Schools*, *30*(1), 27–36. doi:10.1093/cs/30.1.27

Facilitated communication: Courts say "no." (1992). *Autism Research Review International*, *6*(3). Retrieved September 25, 2017, from http://www.srmhp.org/archives/facilitated-communication.html

Frank, J. D. (1973). *Persuasion and healing: A comparative study of psychotherapy* (rev. ed.). Baltimore, MS: The Johns Hopkins University Press.

Ghaziuddin, M., Weidmer-Mikhail, E., & Ghaziuddin, N. (1998). Comorbidity of asperger syndrome: A preliminary report. *Journal of Intellectual Disability Research*, *42*(4), 279–283. doi:10.1111/j.1365-2788.1998.tb01647.x

Gresham, F. M., & Elliott, S. N. (1990). *The Social Skills Rating System*. Circle Pines, MN: American Guidance Service.

Grinker, R. R. (2007). *Unstrange minds: Remapping the world of autism*. New York, NY: Basic Books.

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, *2*, 217–250.

King, M., & Bearman, P. (2009). Diagnostic change and the increased prevalence of autism. *International Journal of Epidemiology*, *38*, 1224–1234. doi:10.1093/ije/dyp261

Long, T. F. (2013). Essential contractual language for medical necessity in children, Committee on Child Health Financing. *Pediatrics*, *132*, 398–401. doi:10.1542/peds.2013-1637

Lord, C., Petkova, E., Hus, V., Gan, W., Lu, F., Martin, D., … Risi, S. (2011). A multisite study of the clinical diagnosis of different autism spectrum disorders. *Archives of General Psychiatry*, *69*(3), 306–313. doi:10.1001/archgenpsychiatry.2011.148

Lotter, V. (1966). Epidemiology of autistic conditions in young children: I. Prevalence. *Social Psychiatry*, *1*, 124–135. doi:10.1007/bf00584048

Merriam-Webster. (2017). "Psychotherapy." Retrieved September 29, 2017, from http://www.merriam-webster.com/medical/psychotherapy

Missouri Department of Mental Health. (2012). *Autism spectrum disorders: Guide to evidence-based interventions*. Retrieved October 2, 2017, from https://www.autismguidelines.dmh.mo.gov/documents/Interventions.pdf

Mudford, O., Blampied, N., Phillips, K., Harper, D., Foster, M., Church, J., … Wilczynski, S. (2009). *Technical review of published research on applied behaviour analysis interventions for people with autism spectrum disorders*. Wellington, New Zealand: Ministry of Education.

Myers, S. M., & Johnson, C. P. (2007). Management of children with autism spectrum disorders. *Pediatrics*, *120*(5), 1162–1182. doi:10.1542/peds.2007-2362

National Autism Center. (2009). *National standards report*. Randolph, MA: National Autism Center.

National Research Council. (2001). *Educating children with autism*. Washington, DC: National Academy Press.

New York State Department of Health, Early Intervention Program. (1999). *Clinical practice guideline: Guideline technical report—Autism/pervasive developmental*

disorders, assessment and intervention for young children (ages 0–3 years). Albany, NY: New York State Department of Health.

Norcross, J. C. (1990). An eclectic definition of psychotherapy. In J. K. Zeig & W. M. Munion (Eds.), *What is psychotherapy? Contemporary perspectives* (pp. 218–220). San Francisco, CA: Jossey-Bass.

Peacock, E. (2017). "Top 8 Autism Therapies—Reported by Parents." *Autism Speaks*. Retrieved September 29, 2017, from https://www.autismspeaks.org/blog/2012/09/25/top-8-autism-therapies-%E2%80%93-reported-parents

Romanczyk, R. G., & Callahan, E. H. (2012). Autism and pervasive developmental disorders. In V. Ramachandran (Ed.), *The encyclopedia of human behavior* (pp. 243–250). Waltham, MA: Academic Press.

Romanczyk, R. G., Callahan, E. H., Turner, L. B., & Cavalari, R. N. S. (2014). Efficacy of behavioral interventions for young children with autism spectrum disorders: Public policy, the evidence base, and implementation parameters. *Journal of Autism and Developmental Disorders*, *1*(4), 276–326. doi:10.1007/s40489-014-0025-6

Romanczyk, R. G., Gillis, J. M., White, S., & DiGennaro, F. (2008). Comprehensive treatment packages for ASD: Perceived vs proven effectiveness. In J. Matson (Ed.), *Autism spectrum disorder: Evidence based assessment and treatment across the lifespan* (pp. 351–382). Cambridge, MA: Elsevier.

Romanczyk, R. G., Turner, L. B., Sevlever, M., & Gillis, J. M. (2014). The status of treatment for autism spectrum disorders: The weak relationship of science to interventions. In L. Lilienfeld & J. Lynn (Eds.), *Science and pseudoscience in contemporary clinical psychology* (2nd ed., pp. 431–465). New York, NY: Guilford Press.

Rotheram-Fuller, E., & MacMullen, L. (2011). Cognitive-behavioral therapy for children with autism spectrum disorders. *Psychology in the Schools*, *48*(3), 263–271. doi:10.1002/pits.20552

Santangelo, S. L., & Tsatsanis, K. (2005). What is known about autism: Genes, brain and behavior. *American Journal of Pharmacogenomics*, *5*, 71–92. doi:10.2165/00129785-200505020-00001

Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child & Adolescent Psychiatry*, *47*(8), 921–929. doi:10.1097/chi.0b013e318179964f

Singh, N. N., Lancioni, G. E., Manikam, R., Winton, A. S. W., Singh, A. N. A., Singh, J., & Singh A. D. A. (2011). A mindfulness-based strategy for self-management of aggressive behavior in adolescents with autism. *Research in Autism Spectrum Disorders*, *5*, 1153–1158. doi:10.1016/j.rasd.2010.12.012

Sofronoff, K., Attwood, T., Hinton, S., & Levin, I. (2007). A randomized controlled trial of a cognitive behavioural intervention for anger management in children diagnosed with Asperger syndrome. *Journal of Autism and Developmental Disorders*, *37*, 1203–1214. doi:10.1007/s10803-006-0262-3

Turner, L. B., & Romanczyk, R. G. (2012). Assessment of fears and phobias in children with an autism spectrum disorder. *Research in Autism Spectrum Disorders*, *6*, 1203–1210. doi:10.1016/j.rasd.2012.03.010

WebMD. (2017). Therapies for autism spectrum disorder. Retrieved September 29, 2017, from http://www.webmd.com/brain/autism/autism-therapies-aba-rdi-and-sensory-therapies?page%20=%203#2-5

Wood, J. J., Fujii, C., Renno, P., & Van Dyke, M. (2014). Impact of cognitive behavioral therapy on observed autism symptom severity during school recess: A preliminary randomized, controlled trial. *Journal of Autism and Developmental Disorders*, *44*(9), 2264–2276. doi:10.1007/s10803-014-2097-7

16

Varieties of Psychotherapy for Attention-Deficit Hyperactivity Disorder

An Evidence-Based Evaluation

Anca Dobrean,[1] Costina R. Păsărelu,[1] and Manfred Döpfner[2]

[1] *Department of Clinical Psychology and Psychotherapy, International Institute for the Advanced Studies of Psychotherapy and Applied Mental Health, Babeş-Bolyai University, Cluj-Napoca, Romania*

[2] *Department of Child and Adolescent Psychiatry and Psychotherapy, University of Cologne, Cologne, Germany*

16.1 Description of the Disorder

Attention-deficit hyperactivity disorder (ADHD) is a neurodevelopmental disorder characterized by attention deficits and hyperactive behaviors. According to the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2013), ADHD can be divided into three subtypes based on the inattention and hyperactivity dimensions: a predominantly inattentive type, a predominantly hyperactive–impulsive type, and a combined hyperactive–inattentive type. The worldwide prevalence of ADHD is approximately 3.4% for children and adolescents (Polanczyk, Salum, Sugaya, Caye, & Rohde, 2015) and 5% for adults (Willcutt, 2012). More males tend to be affected than females. ADHD interferes with academic performance (Loe & Feldman, 2007) and is associated with a high risk for comorbid conditions, such as conduct disorders (Waschbusch, 2002), anxiety (Jarrett & Ollendick, 2008), depression (Blackman, Ostrander, & Herman, 2005), accidental injuries (Nigg, 2013), and suicidality (Barbaresi et al., 2013).

ADHD is a major public health concern as the disease is burdensome on both individuals and society (Pelham, Foster, & Robb, 2007; Robb et al., 2011). The annual costs related to education, treatment, and juvenile justice for ADHD children and adolescents are estimated to be around $43 billion. For adults, ADHD results in approximately 121 million missed work days, with $20 billion lost in salary alone (Pelham et al., 2007). Costs associated with ADHD are comparable to those associated with major depression and stroke (Pelham et al., 2007). Therefore, the development of effective treatments for ADHD is important on both the individual and the societal levels.

Guidelines from the National Institute for Health and Care Excellence (previously the National Institute for Health and Clinical Excellence [NICE], 2008) recommend pharmacotherapy as a first-line treatment in adults with ADHD and in children with severe

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

cases of ADHD. Pharmacotherapy for ADHD, in the form of stimulant and nonstimulant medications, is recommended, either alone or in combination with psychosocial interventions. Pharmacotherapy is effective in only 70% of cases (Spencer et al., 2005) and is associated with several side effects in the short term (e.g., delayed sleep, reduced appetite, headaches, abdominal pain); however, the long-term safety of pharmacotherapy for ADHD is insufficiently investigated (Clavenna & Bonati, 2014). Another important issue related to the use of medications for ADHD is that medication adherence rates decrease throughout adolescence; up to 70% of teenagers stop taking their ADHD medications by the age of 15 (Wolraich et al., 2005). An active debate also remains regarding whether pharmacotherapy improves functional impairments associated with ADHD, as studies have shown that medications are less effective for improving functional deficits (e.g., positive social behaviors, peer rejection) as compared to their effects on core ADHD symptoms (Langberg & Becker, 2012; Nijmeijer et al., 2008).

Another type of treatment for ADHD is dietary interventions. Three main types of dietary interventions that have been investigated in the treatment of ADHD are increased omega-3 fatty acids, few foods diet, and artificial food color elimination. However, according to a recent systematic review, more research needs to be conducted in order to establish the efficacy and mechanisms of change of dietary interventions for ADHD (Pelsser, Frankena, Toorman, & Rodrigues Pereira, 2017).

Regarding psychological treatments recommended for people with ADHD, clinical guidelines have been established that take into account patient age and ADHD severity. For example, for preschool and school-aged children with moderate ADHD severity, the NICE (2008) guidelines recommend parent training programs as the first-line treatment. For adolescents, NICE recommends individual or group CBT and skills training. For adults with ADHD, pharmacotherapy (methylphenidate; e.g., Ritalin) is recommended first, except in cases in which the patient actively requests another form of treatment (e.g., psychotherapy). Still, not all governing bodies and professional associations make the same recommendations. While NICE (2008) recommends medication as the first-line treatment for adults with ADHD, based on strong research support, the American Psychological Association Division 12 recommends CBT.

Overall, there are less-than-consistent efficacy data and inconsistent treatment guidelines for ADHD at this time. This state of affairs could lead to confusion on the part of both clinicians and patients. Therefore, a review of the psychological interventions for ADHD is needed in order to help clarify recommendations for patients. David and Montgomery (2011) have proposed a new evaluative framework for evidence-based psychotherapies. The approach classifies psychotherapies on two dimensions: their underlying theory (mechanisms of change) and the therapeutic package derived from that theory. Each of these two dimensions is further organized by three levels: empirically well supported, moderate support (preliminary, equivocal, or mixed data), and no supportive data or findings indicating the treatment is harmful. This organizational scheme results in nine categories. The scheme considers both absolute efficacy (i.e., therapeutic package fares better than a comparison condition) and relative efficacy (i.e., relative to another evidence-based intervention). Furthermore, the authors state that there should also be a clear relationship between a guiding theoretical base and the empirical data collected.

The focus of this chapter is to review psychological interventions for ADHD based on David and Montgomery's (2011) evaluative framework. Clinical guidelines,

Table 16.1 A graphical representation of the classification of psychotherapies for ADHD using David and Montgomery's (2011) evaluative framework.

	Theory		
Therapeutic Package	**Well Supported**	**Equivocal: No, Preliminary, or Mixed Data**	**Strong Contradictory Evidence**
Well Supported	Category I: Evidence-based psychotherapies Combined treatment	Category II: Intervention-driven psychotherapies CBT; OST; psychoeducation	Category V: Good-intervention- and bad-theory-driven psychotherapies None
Equivocal: No, Preliminary, or Mixed Data	Category III: Theory-driven psychotherapies BPT	Category IV: Investigational psychotherapies CT; mindfulness; NF; SST	Category VII: Bad-theory-driven psychotherapies None
Strong Contradictory Evidence	Category VI: Good-theory- and bad-intervention-driven psychotherapies None	Category VIII: Bad-intervention-driven psychotherapies None	Category IX: Bad-theory- and bad-intervention-driven psychotherapies None

BPT = behavioral parent training; CBT = cognitive–behavioral therapy; CT = cognitive training; NF = neurofeedback; OST = organizational skills training; SST = social skills training. Lighter backgrounds indicate scientifically oriented psychotherapies (the core of these therapies is represented by Category I), while darker backgrounds indicate pseudoscientifically oriented psychotherapies (the core of this category is represented by Category IX).

meta-analyses, literature reviews, and randomized controlled trials (RCTs) were reviewed in order to evaluate both the underlying theory and the specific psychological intervention packages for treating ADHD. In order to evaluate the underlying psychological theory, we searched for data from component analysis, mediation and moderation analyses from complex controlled trials, and experimental and cross-sectional studies that tested the theory independently. Table 16.1 summarizes our findings.

16.2 Classification of Psychotherapies

16.2.1 Category I: Evidence-Based Psychotherapies

Category I includes psychological interventions for which there is strong supporting evidence for both the therapeutic package and the theory of change underlying the clinical protocol. In our review of psychological interventions for ADHD, combined treatment interventions fit this category.

16.2.1.1 Combined treatment

The assumption behind combining pharmacology with psychotherapy is based on the premise that neither form of treatment alone is sufficient to manage ADHD symptoms and resultant impairment. Recent evidence has shown that combined treatments were associated with better clinical results, even with lower doses of both treatments, compared with each intervention alone (Pelham et al., 2014). ADHD treatment guidelines (American Academy of Pediatrics Subcommittee on Attention-Deficit/Hyperactivity

Disorder et al., 2011; NICE, 2008) recommend combined treatment for adults, based on the assumption that behavioral and pharmacological interventions could have additive effects for the reduction of ADHD symptoms (see Klein, Abikoff, Hechtman, & Weiss, 2004; Klein et al., 1997). According to the NICE guidelines (2008), combined psychological and pharmacological treatment is recommended in adults, as well as in children with severe ADHD (group-based behavior parent training plus pharmacological treatment for children).

In the following sections, we consider types of combined treatments (psychotherapy plus medication) that allow direct comparisons between conditions only, as established by the study protocol.

16.2.1.1.1 Absolute efficacy

We were not able to find any studies comparing combined treatment for ADHD with waitlist control. Therefore, the results concerning relative efficacy (see Section 16.2.1.1.2) should be interpreted cautiously (Temple & Ellenberg, 2000).

16.2.1.1.2 Relative efficacy

In the multimodal treatment study of children with ADHD (MTA study; MTA Cooperative Group, 1999), combined treatment (behavioral management plus pharmacology) for ADHD was superior to medication alone, based on parent ratings of conduct problems following treatment. A second RCT shows that medication (methylphenidate) plus behavioral treatment is superior to medication alone (*better than standard treatment*) in the reduction of ADHD symptoms (So, Leung, & Hung, 2008).

16.2.1.1.3 Specific efficacy

The first criterion, regarding equivalence (*equivalent to standard therapies*) or superiority to standard treatment (*better than standard treatment*), is met. We conclude that there are well-supported data regarding the efficacy of the combined treatment therapeutic package.

The second criterion, regarding the empirical support for the underlying theory for specific mechanisms of change, is met. Regarding the underlying theory of the therapeutic package, we found several analyses or secondary analyses based on the MTA study (MTA Cooperative Group, 1999) regarding possible mediators (e.g., adherence to treatment) and moderators (e.g., comorbidity, socioeconomic status, parental depression) of ADHD treatment. An important mechanism of change in combined treatment is parenting, and evidence comes from several RCTs. Reductions in negative and ineffective discipline significantly mediated the efficacy of combined treatment on child outcomes as reported by teachers in the MTA study (Hinshaw et al., 2000). Other evidence on the mechanisms of change comes from a RCT that compared the efficacy of a multicomponent psychosocial intervention with parent-focused treatment and treatment as usual (TAU) in a sample of children with ADHD–inattentive type. Improvements in positive and negative parenting were found to be significant mediators of treatment efficacy on child outcomes (impairment), even after controlling for child inattention symptoms on both parent and teacher ratings (Haack, Villodas, McBurnett, Hinshaw, & Pfiffner, 2016). Another study comparing a multicomponent psychosocial intervention with a parent support and education program reported that reductions in negative parenting significantly mediated the efficacy of the intervention on child outcomes (homework

performance) according to parent (indirect mediation) and teacher reports (total mediation) (Booster, Mautone, Nissley-Tsiopinis, Van Dyke, & Power, 2016). Therefore, we conclude that there are *well-supported data* on the mechanisms of change for combined treatments for ADHD.

16.2.2 Category II: Intervention-Driven Psychotherapies

Category II includes psychological treatments for which there is strong supporting evidence for the efficacy/effectiveness of the therapeutic package but where the results are equivocal (i.e., missing, preliminary, or mixed) for the underlying theory from which the package is derived. In the following sections, we present the psychological interventions for ADHD that meet Category II criteria, namely CBT, psychoeducation, and organizational skills training (OST).

16.2.2.1 Cognitive–behavioral therapy

According to the CBT model of adult ADHD (Safren et al., 2005; Young & Bramham, 2006), repeated negative life experiences of failure and underachievement, mainly due to neurobiological deficits, influence self-esteem and self-concept, giving rise to negative self-beliefs that, consequently, cause dysfunctional emotions (e.g., depression, anxiety). These negative self-beliefs could also be associated with maladaptive behavioral strategies (e.g., negation, procrastination, avoidance) when an individual is confronted with a difficult or attention-consuming task (Ramsay & Rostain, 2008). Even though the dysfunctional belief system does not contribute to the etiology of primary neurocognitive symptoms of ADHD, it does contribute to secondary comorbid symptoms and emotional maladjustment.

NICE (2008) recommends individual psychological interventions (i.e., CBT or social skills training) for older adolescents with ADHD and moderate impairment, while individual or group CBT adjunctive to medication is recommended for adults. The APA Division of Clinical Psychology also describes CBT interventions for adults as having strong research support. CBT has typically been tested on adults whose ADHD symptoms persist despite treatment with medication.

The CBT interventions tested so far were delivered in several formats: cognitive rehabilitation programs, dialectical behavioral training, meta-cognitive therapy, and coaching. However, in most of the RCTs, CBT interventions for ADHD were generally used as an adjunct to stimulant medication. CBT sessions have used the following formats: motivational interview, ADHD psychoeducation, behavioral training in organization and planning skills, behavioral skills training to reduce distractibility, and cognitive restructuring.

Several RCTs have supported the efficacy of CBT for ADHD.

16.2.2.1.1 Absolute efficacy

An RCT comparing group CBT in medicated adolescents with a waitlist control group showed that CBT reduced ADHD symptoms significantly (Vidal et al., 2015). CBT was superior to waitlist based on self-report (Cohen's $d = 7.50$), parental report ($d = 8.38$), and blind evaluator ($d = 7.71$) ratings. In addition to reduced ADHD symptoms, there were significant decreases in functional impairment (parental report: $d = 2.29$; blinded evaluator report: $d = 7.71$). In a pilot RCT, Virta et al. (2010) found that group CBT in

medicated adult patients was more effective than waitlist based on self-reported ADHD total symptoms, attention, memory, and quality of life. Also, Bramham et al. (2009) provided an intensive 1-day intervention (1 day per month for 3 months) to medicated adult patients and compared it with waitlist controls. While significant effects of CBT were found on psychoeducation, self-efficacy, and self-esteem measures, there was no significant effect of CBT on anxiety and depression. In two RCTs conducted by the same team (Stevenson, Stevenson, & Whitmont, 2003; Stevenson, Whitmont, Bornholt, Livesey, & Stevenson, 2002), which investigated the efficacy of a cognitive remediation program in both medicated and nonmedicated ADHD adults as compared to waitlist, the results favored the intervention group in terms of reduced ADHD symptomatology ($d = 1.4$), improved organizational skills ($d = 1.2$), and reduced levels of anger ($d = 0.5$). There were also significant improvements in ADHD symptoms ($d = 1.4$) and organizational skills ($d = 1.3$) at the 1-year follow-up assessment. Additionally, CBT interventions were effective when they were delivered in different formats. For example, in an RCT (Pettersson, Söderström, Edlund-Söderström, & Nilsson, 2014) involving adult ADHD patients on medication, two types of CBT were compared with waitlist. The first was an internet-delivered self-help CBT (iCBT) intervention, while the second intervention was iCBT plus weekly group sessions. There was a significant difference between the iCBT and waitlist ($d = 1.07$), with no significant difference between the two formats of iCBT. Furthermore, in another RCT, a smartphone-delivered CBT intervention for medicated adult patients was better that waitlist. Participants in the CBT group showed significant improvements in inattention and organizational skills on both self-reported and blind assessor measures, as well as significant decreases in hyperactivity and depression. However, no effect was found on anxiety, stress, or overall functioning (Moëll, Kollberg, Nasri, Lindefors, & Kaldo, 2015). Based on these studies, we conclude that CBT is superior to a waitlist control.

16.2.2.1.2 Relative efficacy

Emilsson et al. (2011) found that a group CBT intervention in medicated adult ADHD patients with persistent symptoms was better than TAU control conditions. CBT had a significant effect on ADHD symptoms, with effect sizes in the medium to large range. Safren et al. (2005) compared individual CBT in medicated adult patients with persistent symptoms with continued pharmacotherapy alone. There were superior results for the CBT group in terms of ADHD symptoms ($d = 1.20$ for self-rated), ADHD global severity ($d = 1.40$ for independent assessor rated), anxiety, and depression. Young et al. (2015) compared CBT (both individual and group sessions) with TAU in a sample of medicated ADHD adults and found that CBT was superior to TAU in terms of ADHD symptoms, with medium effect sizes from both blinded assessors and self-ratings (*better than standard treatment*). Namely, this means that the treatment is better than another evidence-based psychological intervention, and both treatments are better than waitlist control conditions.

16.2.2.1.3 Specific efficacy

Based on our review, we argue that there is strong evidence to support CBT as a well-supported treatment package for ADHD, as the first criterion, involving superiority to placebo or equivalence to other standard therapies, is met.

However, the second criterion, regarding empirical support for the underlying theory and mechanisms of change, does not appear to be met. We were able to find only one independent study investigating the validity of the CBT model of ADHD in adults (Torrente et al., 2014). The research documented that dysfunctional cognitions were strongly associated with emotional symptoms and that ADHD participants presented elevated avoidant coping strategies, but these findings were not clearly associated with dysfunctional cognitions or emotions. Several additional correlational studies show that ADHD is associated with negative patterns of thinking. For example, researchers have found a significant positive association between self-reported cognitive distortions and ADHD, with perfectionism being the most prevalent cognitive distortion found in ADHD adult patients (Strohmeier, Rosenfield, DiTomasso, & Ramsay, 2016). Further evidence sustaining the CBT model in ADHD patients comes from a study comparing adults diagnosed with ADHD (archival chart review) with nonclinical controls (undergraduate students) (Mitchell, Benson, Knouse, Kimbrel, & Anastopoulos, 2013). Results indicated a positive association between inattention and negative automatic thoughts on self-, other- and clinician ratings; however, no significant association was found between hyperactivity and negative automatic thoughts. Moreover, for all three types of raters, the association between ADHD–inattentive symptoms and negative automatic thoughts remained stable even after controlling for depression associated with ADHD. An ongoing RCT aims to examine the mechanisms of change of CBT for ADHD (Dittner, Rimes, Russell, & Chalder, 2014), but the results are not yet available. According to David and Montgomery's (2011) framework, we would view these studies as providing *preliminary data* in support of the underlying theory of CBT. Future RCTs investigating the mediator/moderator role of proposed mechanisms of change (e.g., negative automatic thoughts, worry, cognitive distortions) in the relationship between the efficacy of CBT interventions and ADHD outcomes are needed to establish that the theory on which CBT interventions are based is well supported.

16.2.2.2 Psychoeducation

Psychoeducation that includes offering information about the illness and its treatment, skills development, and patient empowerment is considered a well-established evidence-based practice for some severe psychiatric disorders in adulthood. Psychoeducation sessions can be delivered in several formats, including to parents alone or to both parents and their children. Sessions can also be specifically designed for teachers. These sessions are structured as didactic presentations, discussions, role-playing, homework role-playing, token reinforcement systems, contingency management techniques, and problem-solving plans. European and NICE guidelines on ADHD have suggested that psychoeducation programs might be useful in the management of ADHD (Ferrin & Taylor, 2011; NICE, 2008).

In terms of treatment package, we will refer to several RCTs regarding the efficacy of psychoeducation for ADHD.

16.2.2.2.1 Absolute efficacy

In an RCT, psychoeducation delivered for school teachers was better than waitlist control, with moderate to large effect sizes (*better than waitlist*) on outcomes related to ADHD knowledge, attitudes, and behavioral treatment interventions (Lasisi, Ani, Lasebikan, Sheikh, & Omigbodun, 2017).

16.2.2.2.2 Relative efficacy

In an RCT, a psychoeducational program for families of ADHD children was better than TAU with respect to ADHD symptoms (total symptoms, inattention, and hyperactivity), with medium to large effect sizes on parent ratings, no differences on teacher ratings, and improvements on clinician ratings (Ferrin et al., 2016). In one pilot RCT comparing psychoeducation with a standard intervention, Vidal et al. (2013) found psychoeducation to be as effective as CBT in ADHD-medicated adult patients (*equivalent to standard therapies*), with significant improvements on inattention, hyperactivity, impulsivity, self-esteem, anxiety symptoms, and depression in both groups. Furthermore, improved quality of life and lower global severity of symptoms were reported by both patients and clinicians.

16.2.2.2.3 Specific efficacy

The first criterion, involving superiority to placebo or equivalence to other standard therapies, was met. Based on the data already presented, we conclude that the therapeutic package is *well supported*.

The second criterion, regarding the empirical support for the underlying theory for specific mechanisms of change in the case of the therapeutic package, is not met, as we were not able to find mediation/moderation analyses or component analyses from complex controlled trials investigating mechanisms of change. There is only preliminary evidence on the mechanisms of change of psychoeducation. In a systematic review of the effects of psychoeducation interventions on ADHD and Tourette syndrome, researchers proposed several mechanisms explaining how psychoeducation can influence ADHD outcomes (Nussey, Pistrang, & Murphy, 2013). One mechanism is through parents' knowledge related to ADHD, which presumably influences their attitudes, intentions, and behaviors regarding ADHD treatment, which is congruent with the theory of planned behavior (Ajzen, 1991). One study conducted with a sample of parents of children diagnosed with ADHD referred for treatment found that parental knowledge influences parents' probability to enroll their children in ADHD treatments (both pharmacological and nonpharmacological) (Corkum, Rimer, & Schachar, 1999). Psychoeducation can improve ADHD outcomes via another important mechanism, namely adherence to treatment. In one study, in which parents of children diagnosed with ADHD were randomized to either psychoeducation or clinical counseling, participants in the former condition had greater improvement in ADHD symptoms, increased parental knowledge about ADHD, and better adherence to medication (Bai, Wang, Yang, & Niu, 2015). We conclude that there are *preliminary data* regarding mechanisms of change in the effectiveness of psychoeducation. More evidence from complex controlled trials in which the proposed mechanisms (e.g., ADHD knowledge) mediate the efficacy of psychoeducation interventions in ADHD outcomes is needed to validate theories for this therapeutic package.

16.2.2.3 Organizational skills training

OST helps children develop the ability to organize materials and to plan. Specifically, children learn new tools and routines to record assignments and organize school papers, and they learn to use checklists for materials needed, for time management strategies, and to break tasks into graded steps. Moreover, skills training is combined with rewards to encourage the child to use these skills in the school setting. Parents and teachers are trained to use contingency management appropriately in order to reward children's

planning skills. An example of such an intervention is Homework, Organization, and Planning Skills (Langberg, 2011), which focuses on improving participants' physical organization and homework management.

In terms of treatment package, we will refer to several RCTs and meta-analyses regarding the efficacy of organizational skills interventions for ADHD.

16.2.2.3.1 Absolute efficacy

A recent meta-analysis including 12 studies indicated that OST produces changes associated with large effect sizes on parent-reported organizational skills and medium effect sizes on teacher-reported organizational skills (Bikic, Reichow, McCauley, Ibrahim, & Sukhodolsky, 2017). With regard to inattention symptoms, the same meta-analysis indicated small (teacher) to medium (parent) effect sizes, while for academic performance the effect sizes were small for both teacher and parent ratings. An RCT (Abikoff et al., 2013) comparing an organizational skills intervention, a performance-based intervention, and a waitlist condition in elementary-school-aged children (8–11 years) found that the organizational skills training produced superior outcomes, according to self-, parent, and teacher ratings of organization, academic functioning, homework completion, and family functioning as compared with a waitlist condition; when compared with the performance-based intervention, the only significant difference was on parent-reported child organizational skills. These results were replicated by another RCT (Langberg, Epstein, Becker, Girio-Herrera, & Vaughn, 2012) involving adolescents (11–14 years) who showed significant parent-rated improvements for organized actions, task-planning, and homework completion that were maintained at 3-month follow-up.

16.2.2.3.2 Relative efficacy

An RCT that compared an OST intervention (Child Life and Attention Skills) with parent training and TAU indicated that OST was superior to both conditions via teacher and parent reports on measures of inattention, organizational and social skills, and global functioning (Pfiffner et al., 2014). In another RCT conducted with adolescents, OST, namely the program Supporting Teens' Autonomy Daily combined with motivational interviewing, was better than TAU with respect to parent-reported outcomes (symptom severity, disruptive behavior, organizational problems, homework, home privileges, parent–adolescent contracting, parenting stress) (Sibley et al., 2016). OST in medicated children (ages 8–13) was better than placebo (Abikoff et al., 2009); however, the organizational functioning of the majority of children still remained in the clinical range.

16.2.2.3.3 Specific efficacy

The first criterion, involving superiority to other standard therapies, is met.

The second criterion, regarding the empirical support for the underlying theory for specific mechanisms of change pertinent to the therapeutic package, is not met. Regarding theory, researchers investigated therapeutic alliance and organization skills as mechanisms of change in a cross-sectional study involving children aged 11–14. Changes in organizational skills were significant predictors of parent-reported outcomes (organization, planning, homework problems), even after controlling for the effect of the therapeutic alliance (Langberg, Becker, Epstein, Vaughn, & Girio-Herrera, 2013). Another study conducted by the same team found that the working alliance between therapist and adolescent, parenting stress, and conflict between parent and adolescent are important predictors of OST treatment efficacy (Langberg et al., 2016). We were not able

to find other studies investigating mechanisms of change in OST using component or mediation/moderation analyses based on complex controlled trials. Accordingly, there are *preliminary data* for the theory of change behind OST.

16.2.3 Category III: Theory-Driven Psychotherapies

Category III includes psychological treatments for which there is strong supporting evidence for the underlying theory but where the treatment package is insufficiently supported (equivocal results). In this category we include behavioral parent training (BPT) for ADHD.

16.2.3.1 Behavioral parenting training

BPT is listed as having strong support by NICE (2008). BPT is recommended for children with mild to moderate ADHD. BPT is based on learning theory, and it is grounded in principles of classical conditioning, operant conditioning, cognitive–behavioral theory, and social learning theory. In this approach, parents manipulate the antecedents and consequences of child behaviors in order to increase desired behaviors and decrease undesirable ones. Recent developments in this type of intervention came with an enhancement of traditional interventions: telephone-based BPT (McGrath et al., 2011) and BPT for single mothers (Chacko et al., 2008) or fathers (Fabiano et al., 2009, 2012). The aim of "behavioral classroom management" interventions is to help teachers and students gain control over behavior at school (Miranda, Presentación, & Soriano, 2002). Consequently, behavior modification techniques are applied in the school setting in line with NICE (2008) recommendations, which state that teachers should receive training about ADHD and its management, and teachers should provide behavioral interventions in the classroom in order to help children and young people with ADHD. Examples of such interventions are summer treatment programs (Pelham & Hoza, 1996), which engage children in a controlled camp-like setting, or the use of daily report cards (Volpe & Fabiano, 2013) intended to facilitate parent–teacher communication regarding the assessment of behavior throughout a day. The rationale for "behavioral peer interventions" is that, through staff being trained to manipulate contingencies in several settings where children and peers are present, ADHD children will demonstrate improvements in social functioning across settings. More recently, telephone-assisted self-help interventions for parents of children with ADHD have been developed based on principles of parent management training. Written booklets and additional telephone calls with parents were used to help the parents change their parenting behavior and to reduce the ADHD symptoms of the child. Several studies found effects of these kinds of interventions in preschool children (e.g., Kierfeld, Ise, Hanisch, Görtz-Dorten, & Döpfner, 2013), with effects maintained at a 1-year follow-up (Ise, Kierfeld, & Döpfner, 2015). Moreover, this intervention enhances the effects of methylphenidate treatment in families who complete the intervention (Dose et al., 2016). A meta-analysis also found substantial effects of self-help behavioral interventions (O'Brien & Daley, 2011).

16.2.3.1.1 Absolute efficacy

Several meta-analyses have reported weak support for parental training. For example, Sonuga-Barke et al.'s (2013) meta-analysis found that effect sizes for behavioral interventions dropped to nearly zero when data were collected from blinded informants.

Hodgson, Hutchinson, and Denson's (2014) meta-analysis documented that behavior modification, school-based behavior therapy, behaviorally based parent training, and behavioral self-monitoring treatments each had negative effect sizes when compared with comparison conditions. A meta-analysis of RCTs comparing parent training with no treatment, waitlist, or TAU, in children aged 5–18 (Zwi, Jones, Thorgaard, York, & Dennis, 2011), indicated significant changes on parent-related outcomes (e.g., parental stress, parental confidence); however, there were many methodological problems in the studies, which raise questions regarding their quality. Abikoff et al. (2014) investigated specialized and generic programs of parent training compared with waitlist and found that the parent-reported improvements were not corroborated with teacher ratings or objective observations. In a RCT that compared the Incredible Years parent and child training program in preschoolers (ages 4–6) with waitlist, there were significant treatment effects for children's externalizing (on both parent and teacher reports), hyperactivity, inattentive and oppositional behaviors, emotion regulation (on parent report only), social competence (on both parent and peer observations), emotion vocabulary, and problem-solving ability (on parent report only) (Webster-Stratton, Reid, & Beauchaine, 2011). However, for decades, many RCTs have supported behavioral parenting therapies. For example, Sonuga-Barke, Daley, Thompson, Laver-Bradbury, & Weeks, 2001) compared BPT with parental counseling and support, and with waitlist and found that BPT was more effective than waitlist (*better than waitlist*). The effect sizes of the impact of the New Forest Parenting Package on ADHD versus waitlist control were 0.87 (parent reports) and 0.43 for direct observations of attention (Sonuga-Barke, Thompson, Abikoff, Klein, & Brotman, 2006). Another RCT found that, compared with waitlist, BPT was better at reducing ADHD symptoms and associated problems in preschoolers (Herbert, Harvey, Roberts, Wichowski, & Lugo-Candelas, 2013).

16.2.3.1.2 Relative efficacy

In the MTA study (MTA Cooperative Group, 1999), the behavioral training group was inferior to both the methylphenidate intervention group and the combined treatment group in terms of reducing core ADHD symptoms. Thompson et al. (2009) compared BPT with TAU in a small-scale RCT (the Revised New Forest Parenting Program) involving preschoolers with ADHD and reported a large effect size for the BPT group ($d = 1.36$) on ADHD symptoms with significant effects persisting 9 weeks postintervention. A recent study investigating treatment sequencing shows that BPT was superior to medication on outcomes such as observed classroom violation rules and disciplinary events (Pelham et al., 2016).

16.2.3.1.3 Specific efficacy

The first criterion, involving superiority to placebo or equivalence to other standard therapies, is not met (see Section 16.2.3.1.2). Based on the data presented above, we would argue that there are equivocal, *mixed data* regarding the superiority of the intervention over placebo or standard treatment due to different reported outcomes from blinded and unblinded raters.

The second criterion, regarding the empirical support for the underlying theory for specific mechanisms of change in the case of the therapeutic package, is met. Parenting is the active ingredient of BPT interventions, and the RCTs that supported the mediator

role of parenting in the relationship between treatment and outcomes have already been presented in Section 16.2.1.1 (see Booster et al., 2016; Haack et al., 2016). Data that support the theoretical rationale for the treatment package come from a pilot study (Van den Hoofdakker et al., 2012) based on the data from a RCT comparing BPT plus ongoing routine clinical care with routine clinical care alone in children aged 4–12 with ADHD. The study found that genetic differences in the dopamine transporter gene moderate responses to behavioral parent training in children with ADHD. Given the aforementioned evidence, we assume that the theory on which the BPT therapeutic package is based is *well supported*.

16.2.4 Category IV: Investigational Psychotherapies

Category IV includes psychological treatments for which the evidence for both components (treatment package and underlying theory) is insufficiently tested: There is neither strong supporting nor contradictory evidence for the efficacy/effectiveness of the therapeutic package and for the theory of change underlying it. In addition, the evidence for both components is equivocal (i.e., missing, preliminary, or mixed). Next, we present the intervention-driven psychotherapies for ADHD. In this category, we include social skills training (SST), cognitive training interventions, neurofeedback training, and mindfulness-based interventions.

16.2.4.1 Social skills training

SST helps ADHD children learn useful strategies for improved interactions with peers. According to de Boo and Prins (2007), ADHD is not a knowledge deficit as much as it is a performance disorder. In fact, ADHD children's problems in social behaviors appear to stem from their impulsivity or poor emotional regulation, which prevents them from putting what they know into practice. Therefore, new approaches to SST are based on a shift in researchers' perspective: Children with ADHD do not lack social knowledge; rather, they experience difficulties applying that knowledge in real-world situations with peers. In these interventions, parents and teachers provide contingencies, reminders, and reinforcements to children with ADHD in order to help them perform socially skilled behaviors in naturalistic settings with peers. The ultimate goal is to help the children internalize these skills and to be able to implement them in new contexts. Therefore, the main aim of these interventions is not to teach social skills but rather to help children use social skills in everyday settings. SST for children with ADHD includes sharing, making conversation, joining new groups of peers, following rules when playing games, taking turns, calming down when upset, and identifying emotions. Several SST programs exist, and one example is Parental Friendship Coaching (Mikami, Lerner, Griggs, McGrath, & Calhoun, 2010), which trains parents to offer instructions in social skills knowledge in order to provide environmental contingencies and structure needed to facilitate children's generalization of skills to real-world peer situations.

In terms of the treatment package, we refer to several RCTs and meta-analyses regarding the efficacy of social SST interventions for ADHD.

16.2.4.1.1 Absolute efficacy

A Cochrane Review (Storebø, Gluud, Winkel, & Simonsen, 2012) on SST for children aged 5–12 versus either no intervention or waitlist control reported that there were no statistically significant treatment effects on social skills competences, teacher-rated

general behavior, or ADHD symptoms. However, these results should be interpreted cautiously as there were only a few RCTs included, and they were associated with a high risk of bias. In an RCT of medicated children (aged 8–12) with inattentive and combined type ADHD, researchers compared SST (8 weeks of SST treatment) with a no-intervention control group. SST produced improvements in cooperation, empathy, and assertion skills, but no improvement was seen in social competence (Antshel & Remer, 2003). Empirical evidence suggests that intensive involvement of parents or teachers in SST may increase the likelihood that children will generalize positive social behaviors to real-world situations with peers. Pfiffner and McBurnett (1997) showed that an intervention combining SST with group treatment for parents, in which parents learned to reinforce their child's display of competent social behaviors outside sessions, is better than SST only involving children or a no-intervention control group. According to teacher reports, improvements generalized to school settings as well. In an RCT conducted by the same team and involving only children with ADHD–inattentive type, a program involving simultaneous child SST and parent groups to encourage the child's generalization of social skills was *better than a no-treatment* control group on both parent and teacher reports of social behaviors (Pfiffner et al., 2007). In a pilot randomized study (Mikami et al., 2010), an intervention to train parents to be social coaches (Parental Friendship Coaching) was *better than no treatment* on both parent and teachers reports on social behaviors and showed more acceptance and less rejection from peers compared with a control.

16.2.4.1.2 Relative efficacy

An RCT comparing a social skills intervention (combined with parent training and standard treatment) with standard treatment found no significant effect of the former intervention (Storebø et al., 2011).

16.2.4.1.3 Specific efficacy

The first criterion, involving superiority to placebo or equivalence to other evidence-based therapy, is not met. Based on the evidence presented, we assume that there are equivocal, *mixed data* regarding the therapeutic package.

The second criterion, regarding the empirical support for the underlying theory for specific mechanisms of change in the case of the therapeutic package, is not met. In an RCT (Mikami et al., 2010), changes in some parenting behaviors during peer interactions, namely parent facilitation of successful behaviors, correction of child behavior, and reductions in criticisms, mediated the effect of training on child peer functioning. A recent meta-analysis investigating the relationship between ADHD and social functioning indicated a significant association between ADHD and peer functioning, social skills, and information-processing (Ros & Graziano, 2017). We would qualify the findings as providing *preliminary data* for the theory behind SST.

16.2.4.2 Cognitive training interventions

As executive function deficits are frequently included in most of the current ADHD models (Barkley, 1997; Rapport et al., 2008; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005), cognitive training interventions aim to improve ADHD symptoms by targeting deficient neuropsychological functions associated with ADHD pathology. According to a neuropsychological theory, ADHD symptoms are the result of a primary deficit in executive functions (Barkley, 1997). In most of the studies on executive

functioning, the targeted deficits are attention control, working memory, and inhibitory control. Cognitive training interventions consist of repeated and graded exposure to cognitive stimuli (Rabipour & Raz, 2012) in a game format in which difficulty is adjusted for the child on a trial-by-trial basis, becoming more difficult as the training advances. Cogmed training is probably the most investigated cognitive training program, and it has been adapted for both preschoolers and older children (Cogmed JM and RM) as well as for adults (Cogmed QM). The intervention is delivered in a videogame format in which the participant responds to several visuospatial and verbal memory tasks.

In terms of the treatment package, we refer to recent meta-analyses and several RCTs regarding the efficacy of cognitive training interventions for ADHD.

16.2.4.2.1 Absolute efficacy

A meta-analysis (Cortese et al., 2015) based on data from 16 RCTs comparing cognitive training interventions with a control (waitlist, TAU, placebo, or sham) reported significant effects on total ADHD and inattentive symptoms for unblinded raters, proximal to the treatment setting (parents). Working memory interventions implemented alone had no effect on key measures, while multiprocess training approaches produced large effect sizes on total ADHD based on proximal assessments. However, when blinded raters were considered, there were small effect sizes on total ADHD. Finally, when compared with active training, placebo conditions, or sham conditions, which involved other computer or alternative interventions, cognitive training had nonsignificant effects on total ADHD for both blinded and unblinded raters. These results have been supported in other meta-analyses (see Sonuga-Barke et al., 2013; Rapport, Orban, Kofler, & Friedman, 2013). Given the mixed results, we will not consider investigating relative or specific efficiency further. Therefore, regarding the *therapeutic package*, the data are *equivocal and mixed data*. Regarding the *underlying theory*, a longitudinal study (Coghill, Hayward, Rhodes, Grimmer, & Matthews, 2014) found that changes in executive functions are nonsignificant predictors of changes in ADHD symptoms. An RCT investigated predictors and moderators of cognitive training for children with ADHD and indicated that the effect of cognitive training on ADHD is moderated by several variables, such as type of ADHD, comorbid conditions presented, medication, and initial verbal and visual working memory skills (Van der Donk, Hiemstra-Beernink, Tjeenk-Kalff, Van der Leij, & Lindauer, 2016). Therefore, we consider this evidence to be *preliminary data* related to the mechanism of change for cognitive training.

16.2.4.3 Neurofeedback training

The aim of neurofeedback training is to achieve control of particular brain activity patterns using electroencephalographic (EEG) technology. The two most common neurofeedback protocols that have been applied are EEG frequency-band (i.e., theta/beta) training and the training of slow cortical potentials. In theta/beta frequency-band training, children learn to decrease activity in the theta band of the EEG (4–8 Hz) and to increase activity in the beta band (13–20 Hz), whereas in the training of slow cortical potentials the aim is to regulate cortical excitability (Gevensleben, Holl, Albrecht, Schlamp et al., 2009). The intervention is delivered as a computer game in which participants receive visual and auditory rewards each time brain activity patterns change in the desired direction (Gevensleben, Rothenberger, Moll, & Heinrich, 2012).

In terms of treatment package, we refer to several randomized trials and meta-analyses regarding the efficacy of neurofeedback for ADHD.

16.2.4.3.1 Absolute efficacy

In a meta-analytic review (Hodgson et al., 2014) compared various types of interventions to no treatment, waitlist, placebo, or alternative treatment. Neurofeedback training had an average weighted effect size ($d = 0.21$) across 20 outcomes, resulting in statistically significant improvements in inattention symptoms, neuropsychological test performance, and impulsivity behavior (*better than waitlist*).

16.2.4.3.2 Relative efficacy

In a meta-analysis comparing neurofeedback with active interventions and sham (placebo) (Micoulaud-Franchi et al., 2014), results revealed significant reductions in ADHD symptoms using unblinded assessments (parents) but not when teacher assessments were used. When compared with standard pharmacological treatment, neurofeedback interventions for children aged 7–14 (Meisel, Servera, Garcia-Banda, Cardo, & Moreno, 2014) produced similar effects on both ADHD symptoms and functional impairments, with maintained gains at both follow-up assessments. In one RCT on ADHD participants (aged 6–18), neurofeedback alone was as effective as stimulation drug treatment alone or combined treatment (Duric, Assmus, Gundersen, & Elgen, 2012). However, this study used blinded raters. In one RCT, neurofeedback was inferior to pharmacological treatment in children and adolescents aged 7–16 (Ogrim & Hestad, 2013) based on parent and teacher ratings, with no blinded raters.

16.2.4.3.3 Specific efficacy

The first criterion, involving superiority to placebo or equivalence to other standard therapies, is not met (see Section 16.2.4.3.2). Based on the data presented, we conclude that there are equivocal, *mixed data*.

The second criterion, regarding the empirical support for the underlying theory for specific mechanisms of change in the therapeutic package, is not met, as there are only *preliminary data*. An experimental study comparing neurofeedback with waitlist provides evidence regarding neurofeedback mechanisms of change, as the results of this study indicated significant changes in the medial frontal cortices involved in response inhibition in the neurofeedback group only (Bluschke, Broschwitz, Kohl, Roessner, & Beste, 2016). Despite a few studies that found certain normalization of EEG and event-related potential after neurofeedback training (Bakhshayesh, Hänsch, Wyschkon, Rezai, & Esser, 2011; Doehnert, Brandeis, Straub, Steinhausen, & Drechsler, 2008; Monastra, Monastra, & George, 2002) and a reduction of theta activity (Gevensleben, Holl, Albrecht, Vogel et al., 2009), further evidence is needed in order to establish the underlying theory of neurofeedback interventions.

Given the available evidence on neurofeedback, recent technological developments, such as functional magnetic resonance imaging (fMRI), present new, promising interventions for patients with ADHD, such as real-time fMRI neurofeedback (rt-fMRI neurofeedback). In this intervention, participants learn to regulate their brain activity voluntarily by receiving information on how to do so while in the scanner. However, research on such new interventions is still in its infancy, with few proof-of-concept

RCTs conducted on a small number of participants, without comparisons with standard treatments. For example, the efficacy of rt-fMRI neurofeedback in a sample of ADHD adolescents was investigated in an RCT (Alegria et al., 2017). Youth in the experimental group received rt-fMRI neurofeedback of the right inferior prefrontal cortex (one region affected in ADHD), whereas participants in the control group received active placebo (rt-fMRI neurofeedback of the left parahippocampal gyrus). Results indicated that both groups evidenced significant improvements in symptoms; however, only the experimental group proved transfer effects and brain changes that correlated with symptom improvement. Another RCT on rt-fMRI neurofeedback was conducted with a sample of adults with ADHD (Zilverstand et al., 2017). Participants in the experimental group learned to regulate the activation of the dorsal anterior cingulate cortex (dACC) based on the feedback they received regarding their activation level of dACC during a mental calculation task, whereas participants in the control group performed the task without any feedback. Results of this study showed similar activation increases in dACC; however, participants in the rt-fMRI neurofeedback condition had superior performance on cognitive functioning (sustained attention, working memory). In conclusion, additional research is needed in order to establish the evidence-based status of neurofeedback interventions for ADHD.

16.2.4.4 Mindfulness-based interventions

Mindfulness-based interventions are part of a "third wave" or "third generation" of CBT (Hayes, Follette, & Linehan, 2004; Hayes, Luoma, Bond, Masuda, & Lillis, 2006). They often involve meditation training, which is based on Buddhist tradition and Western psychology, in which awareness of the present moment and nonjudgmental observation are increased and automatic responding is reduced (Kabat-Zinn, 2003). The proposed mechanisms of change in mindfulness training are body awareness, the enhancement of attention regulation involving processes relating to executive functions (Hölzel et al., 2011; Keng, Smoski, & Robins, 2011) and emotion regulation (Shapiro, Carlson, Astin, & Freedman, 2006), and change in perspective on the self (for a detailed presentation of the proposed mechanisms, see Hölzel et al., 2011).

In terms of the treatment package, we refer to several randomized trials and meta-analyses regarding the efficacy of mindfulness for ADHD.

16.2.4.4.1 Absolute efficacy

An RCT conducted with college students with ADHD compared the efficacy of a mindfulness-based cognitive therapy with waitlist control. The researchers found that mindfulness was superior in terms of ADHD total symptoms, inattention, and hyperactivity, as well as anxiety and mindful attention and awareness (Gu, Xu, & Zhu, 2016). Schoenberg et al. (2014) compared a mindfulness intervention for ADHD adults (aged 18–65) with waitlist and found that mindfulness was *better than waitlist*, as it was associated with reduced hyperactivity/impulsivity and inattention, as well as improvements in mindfulness and quality of life. In a pilot trial comparing mindfulness with waitlist in adult patients (aged 18–50) with ADHD stratified by medication (Mitchell, McIntyre et al., 2013), ADHD symptoms and executive functions improved on both self- and clinician assessments, whereas emotion dysregulation improved for the treatment group (self-assessment only) over time with a large effect (*better than waitlist*).

16.2.4.4.2 Relative efficiency

An RCT comparing mindfulness with methylphenidate is currently being conducted in the Netherlands; however, results are not yet available (Meppelink, de Bruin, & Bögels, 2016).

16.2.4.4.3 Specific efficiency

The first criterion, involving superiority to placebo or equivalence to other standard therapies, is not met (see Section 16.2.4.4.2). Based on the data presented, we conclude that there are *preliminary data*.

The second criterion, regarding the empirical support for the underlying theory for specific mechanisms of change in the case of the therapeutic package, is not met, as we were not able to find either mediation/moderation analyses from complex controlled trials investigating mechanisms of change or component analyses. Data from an RCT comparing mindfulness with waitlist in a sample of adults with ADHD indicated that a significant partial mediator in the relationship between mindfulness efficacy and outcomes (ADHD symptoms, executive functioning) is mindful awareness (Hepark et al., 2015). Furthermore, correlational studies show that ADHD symptoms are negatively associated with self-reported mindfulness (Keith, Blackwood, Mathew, & Lecci, 2016; Smalley et al., 2009). Changes in parenting behaviors following mindfulness could be another mechanisms of change of mindfulness for ADHD children, as there is evidence from experimental studies reporting that, after mindful parenting (Haydicky, Shecter, Wiener, & Ducharme, 2015), parental mindful awareness (Van der Oord, Bögels, & Peijnenburg, 2012) increased. Additionally, preliminary data exist on the associated neuronal mechanisms of mindfulness (Schoenberg et al., 2014; Tang, Hölzel, & Posner, 2015). We conclude that there are *preliminary data* regarding mechanisms of change associated with mindfulness interventions, and future rigorous studies should clarify the validity of these proposed mechanisms of change.

16.2.5 Categories V, VI, VII, VIII, and IX

Categories V, VI, VII, VIII, and IX are constituted by good-intervention- and bad-theory-driven psychotherapies (Category V), good-theory- and bad-intervention-driven psychotherapies (Category VI), bad-theory-driven psychotherapies (Category VII), bad-intervention-driven psychotherapies (Category VIII), and bad-theory- and bad-intervention-driven psychotherapies (Category IX). The common factor for all of these categories is that each requires data invalidating (*strong contradictory evidence*) either the therapeutic package or the underlying theory based on two rigorous trials conducted by at least two different investigating teams. We were not able to identify any ADHD studies in these categories.

However, in the absence of empirical data, any claims regarding the validity of a psychotherapeutical approach should be stated cautiously. For example, let us consider the case of psychodynamic therapies. There are two main perspectives regarding the psychoanalytic conceptualizations of ADHD diagnosis: ego psychology and object relations. According to the first perspective, ADHD symptoms in children indicate the ego's difficulties in abilities such as synthesizing, organizing, and integrating experiences (Gilmore, 2000). According to the second perspective, ADHD symptoms result from

difficulties in interacting with others. Through the transference occurring in the therapeutic relationship, the therapist may intervene at the level of the child's object relations, which, in turn, leads to therapeutic change (Cione, Coleburn, Fertuck, & Fraenkel, 2011). Self-regulatory capacities develop when the patient applies the experiences learned in therapy to daily experiences and relationships with others (Leuzinger-Bohleber et al., 2011). We were only able to find case studies and one larger prevention and intervention study for ADHD based on this perspective. We identified no RCTs that tested the therapeutic package or the underlying theory. Therefore, in the absence of RCTs investigating the efficacy of both the therapeutic package and the underlying theory, we cannot assume that this type of psychotherapy is either efficacious or not. We can only say that there is no evidence for classifying it into one of the nine categories proposed by David and Montgomery (2011).

16.3 Conclusions and Discussion

16.3.1 Implications for Research

According to David and Montgomery's (2011) evaluative framework, current treatments for ADHD fall into four main categories: Category I (evidence-based psychotherapies), Category II (intervention-driven psychotherapies), Category III (theory-driven psychotherapies), and Category IV (investigational psychotherapies). Importantly, we were not able to identify strong contradictory evidence. Even though we found equivocal data (preliminary or mixed findings) for most of the therapeutic packages, no study investigated possible harmful effects. Consequently, as this chapter did not aim to investigate ADHD interventions exhaustively, pseudoscientific approaches to ADHD treatment could exist, but we might not have identified them as such.

Our classification of ADHD evidence-based psychotherapies (David & Montgomery, 2011) differs from current guidelines and suggests that far more research is needed in order to establish evidence-based psychotherapies for ADHD. Let us consider the case of behavioral parent training, currently recommended by NICE guidelines as an evidence-based psychotherapy for a child with ADHD. According to David and Montgomery's classification system, this intervention falls in Category III (theory-driven psychotherapies), which means that it has a well-supported theory and an insufficiently tested treatment package. Due to mixed data regarding its equivalence or superiority to standard treatment as a function of unblinded versus blinded assessors' ratings (e.g., parents, teachers, or clinicians), further research is necessary to control for possible biases. By testing these biases in RCTs, ADHD interventions could move from one category to another, either in the direction of scientifically oriented psychotherapies (if there is evidence for a well-supported treatment package or theory) or to pseudoscientifically oriented psychotherapies (if there is strong contradictory evidence showing absence of benefit or evidence of harm). Research should also bring evidence regarding the maintenance of gains after treatment (e.g., consider the scarcity of studies that investigated the long-term effects of neurofeedback interventions) and investigate not only the clinical efficacy of an available treatment option but also its cost-efficiency.

16.3.2 Implications for Practice

ADHD is a prevalent and debilitating condition that calls for more research on evidence-supported treatments. In the past decade, there has been an explosion of cognitive training interventions (e.g., Cogmed, Lumosity, Mindsparke, Jungle Memory) promising evidence-based training associated with outcomes such as intelligence, creativity, academic improvement, and improved attention. Further investigation of their underlying theory and treatment package could prevent the large-scale dissemination of pseudoscientific interventions. In addition to nonclinical implications of psychotherapeutic interventions, the development of interventions that target both core symptoms of ADHD and the functional impairments associated with this condition could have major implications for clinical practice. Given the resources that patients and their families invest in treatment (costs, time), it is highly important to offer in practice the best treatment options according to the existent research, which have also proved to be cost-efficient.

In conclusion, it is highly important to investigate both the therapeutic package and its active ingredients in future RCTs in order to establish the evidence-based status of ADHD psychotherapies. Only after rigorous testing of both components instrumental to psychotherapy should the findings be incorporated into clinical guidelines.

References

Abikoff, H., Gallagher, R., Wells, K. C., Murray, D. W., Huang, L., Lu, F., & Petkova, E. (2013). Remediating organizational functioning in children with ADHD: Immediate and long-term effects from a randomized controlled trial. *Journal of Consulting and Clinical Psychology*, *81*(1), 113–128. doi:10.1037/a0029648

Abikoff, H., Nissley-Tsiopinis, J., Gallagher, R., Zambenedetti, M., Seyffert, M., Boorady, R., & McCarthy, J. (2009). Effects of MPH-OROS on the organizational, time management, and planning behaviors of children with ADHD. *Journal of the American Academy of Child & Adolescent Psychiatry*, *48*(2), 166–175. doi:10.1097/CHI.0b013e3181930626

Abikoff, H. B., Thompson, M., Laver-Bradbury, C., Long, N., Forehand, R. L., Miller Brotman, L., … Sonuga-Barke, E. (2014). Parent training for preschool ADHD: A randomized controlled trial of specialized and generic programs. *Journal of Child Psychology and Psychiatry*, *56*(6), 618–631. doi:10.1111/jcpp.12346

Ajzen, I. (1991). The theory of planned behavior. *Organizational Behavior and Human Decision Processes*, *50*(2), 179–211. doi:10.1016/0749-5978(91)90020-T

Alegria, A. A., Wulff, M., Brinson, H., Barker, G. J., Norman, L. J., Brandeis, D., … Rubia, K. (2017). Real-time fMRI neurofeedback in adolescents with attention deficit hyperactivity disorder. *Human Brain Mapping*, *38*(6), 3190–3209. doi:10.1002/hbm.23584

Antshel, K. M., & Remer, R. (2003). Social skills training in children with attention deficit hyperactivity disorder: A randomized-controlled clinical trial. *Journal of Clinical Child & Adolescent Psychology*, *32*(1), 153–165. doi:10.1207/S15374424JCCP3201_14

American Academy of Pediatrics Subcommittee on Attention-Deficit/Hyperactivity Disorder, Steering Committee on Quality Improvement and Management, Wolraich, M., Brown, L., Brown, R. T., DuPaul, G., … Visser, S. (2011). ADHD: Clinical practice

guideline for the diagnosis, evaluation, and treatment of attention-deficit/hyperactivity disorder in children and adolescents. *Pediatrics*, *128*(5), 1007–1022. doi:10.1542/peds.2011-2654

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.

Bai, G.-N., Wang, Y.-F., Yang, L., & Niu, W.-Y. (2015). Effectiveness of a focused, brief psychoeducation program for parents of ADHD children: Improvement of medication adherence and symptoms. *Neuropsychiatric Disease and Treatment*, *11*, 2721–2735. doi:10.2147/NDT.S88625

Bakhshayesh, A. R., Hänsch, S., Wyschkon, A., Rezai, M. J., & Esser, G. (2011). Neurofeedback in ADHD: A single-blind randomized controlled trial. *European Child & Adolescent Psychiatry*, *20*(9), 481–491. doi:10.1007/s00787-011-0208-y

Barbaresi, W. J., Colligan, R. C., Weaver, A. L., Voigt, R. G., Killian, J. M., & Katusic, S. K. (2013). Mortality, ADHD, & psychosocial adversity in adults with childhood ADHD: A prospective study. *Pediatrics*, *131*(4), 637–644. doi:10.1542/peds.2012-2354

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin*, *121*(1), 65–94. doi:10.1037/0033-2909.121.1.65

Bikic, A., Reichow, B., McCauley, S. A., Ibrahim, K., & Sukhodolsky, D. G. (2017). Meta-analysis of organizational skills interventions for children and adolescents with attention-deficit/hyperactivity disorder. *Clinical Psychology Review*, *52*, 108–123. doi:10.1016/j.cpr.2016.12.004

Blackman, G. L., Ostrander, R., & Herman, K. C. (2005). Children with ADHD and depression: A multisource, multimethod assessment of clinical, social, and academic functioning. *Journal of Attention Disorders*, *8*(4), 195–207. doi:10.1177/1087054705278777

Bluschke, A., Broschwitz, F., Kohl, S., Roessner, V., & Beste, C. (2016). The neuronal mechanisms underlying improvement of impulsivity in ADHD by theta/beta neurofeedback. *Scientific Reports*, *6*, 31178. doi:10.1038/srep31178

Booster, G. D., Mautone, J. A., Nissley-Tsiopinis, J., Van Dyke, D., & Power, T. J. (2016). Reductions in negative parenting practices mediate the effect of a family–school intervention for children with attention deficit hyperactivity disorder. *School Psychology Review*, *45*(2), 192–208. doi:10.17105/SPR45-2.192-208

Bramham, J., Young, S., Bickerdike, A., Spain, D., McCartan, D., & Xenitidis, K. (2009). Evaluation of group cognitive behavioral therapy for adults with ADHD. *Journal of Attention Disorders*, *12*(5), 434–441. doi:10.1177/1087054708314596

Chacko, A., Wymbs, B. T., Flammer-Rivera, L. M., Pelham, W. E., Walker, K. S., Arnold, F. W., … Herbst, L. (2008). A pilot study of the feasibility and efficacy of the Strategies to Enhance Positive Parenting (STEPP) program for single mothers of children with ADHD. *Journal of Attention Disorders*, *12*(3), 270–280. doi:10.1177/1087054707306119

Cione, G. F., Coleburn, L. A., Fertuck, E. A., & Fraenkel, P. (2011). Psychodynamic play therapy with a six-year-old African American boy diagnosed with ADHD. *Journal of Infant, Child, and Adolescent Psychotherapy*, *10*(1), 130–143. doi:10.1080/15289168.2011.576593

Clavenna, A., & Bonati, M. (2014). Safety of medicines used for ADHD in children: A review of published prospective clinical trials. *Archives of Disease in Childhood*, *99*(9), 866–872. doi:10.1136/archdischild-2013-304170

Coghill, D. R., Hayward, D., Rhodes, S. M., Grimmer, C., & Matthews, K. (2014). A longitudinal examination of neuropsychological and clinical functioning in boys with attention deficit hyperactivity disorder (ADHD): Improvements in executive functioning do not explain clinical improvement. *Psychological Medicine*, *44*(5), 1087–1099. doi:10.1017/S0033291713001761

Corkum, P., Rimer, P., & Schachar, R. (1999). Parental knowledge of attention-deficit hyperactivity disorder and opinions of treatment options: Impact on enrolment and adherence to a 12-month treatment trial. *Canadian Journal of Psychiatry/Revue Canadienne de Psychiatrie*, *44*(10), 1043–1048. doi:10.1177/070674379904401011

Cortese, S., Ferrin, M., Brandeis, D., Buitelaar, J., Daley, D., Dittmann, R. W., … European ADHD Guidelines Group. (2015). Cognitive training for attention-deficit/hyperactivity disorder: Meta-analysis of clinical and neuropsychological outcomes from randomized controlled trials. *Journal of the American Academy of Child & Adolescent Psychiatry*, *54*(3), 164–174. doi:10.1016/j.jaac.2014.12.010

David, D., & Montgomery, G. H. (2011). The scientific status of psychotherapies: A new evaluative framework for evidence-based psychosocial interventions. *Clinical Psychology: Science and Practice*, *18*(2), 89–99. doi:10.1111/j.1468-2850.2011.01239.x

De Boo, G. M., & Prins, P. J. (2007). Social incompetence in children with ADHD: Possible moderators and mediators in social-skills training. *Clinical Psychology Review*, *27*(1), 78–97. doi:10.1016/j.cpr.2006.03.006

Dittner, A. J., Rimes, K. A., Russell, A. J., & Chalder, T. (2014). Protocol for a proof of concept randomized controlled trial of cognitive-behavioural therapy for adult ADHD as a supplement to treatment as usual, compared with treatment as usual alone. *BMC Psychiatry*, *14*(1), 248. doi:10.1186/s12888-014-0248-1

Doehnert, M., Brandeis, D., Straub, M., Steinhausen, H.-C., & Drechsler, R. (2008). Slow cortical potential neurofeedback in attention deficit hyperactivity disorder: Is there neurophysiological evidence for specific effects? *Journal of Neural Transmission*, *115*(10), 1445–1456. doi:10.1007/s00702-008-0104-x

Dose, C., Hautmann, C., Buerger, M., Schuermann, S., Woitecki, K., & Doepfner, M. (2016). Telephone-assisted self-help for parents of children with attention-deficit/hyperactivity disorder who have residual functional impairment despite methylphenidate treatment: A randomized controlled trial. *Journal of Child Psychology and Psychiatry*, *58*(6), 682–690. doi:10.1111/jcpp.12661

Duric, N. S., Assmus, J., Gundersen, D., & Elgen, I. B. (2012). Neurofeedback for the treatment of children and adolescents with ADHD: A randomized and controlled clinical trial using parental reports. *BMC Psychiatry*, *12*(1), 107. doi:10.1186/1471-244X-12-107

Emilsson, B., Gudjonsson, G., Sigurdsson, J. F., Baldursson, G., Einarsson, E., Olafsdottir, H., & Young, S. (2011). Cognitive behaviour therapy in medication-treated adults with ADHD and persistent symptoms: A randomized controlled trial. *BMC Psychiatry*, *11*(1), 116. doi:10.1186/1471-244X-11-116

Fabiano, G. A., Pelham, W. E., Coles, E. K., Gnagy, E. M., Chronis-Tuscano, A., & O'Connor, B. C. (2009). A meta-analysis of behavioral treatments for attention-deficit/hyperactivity disorder. *Clinical Psychology Review*, *29*(2), 129–140. doi:10.1016/j.cpr.2008.11.001

Fabiano, G. A., Pelham, W. E., Cunningham, C. E., Yu, J., Gangloff, B., Buck, M., … Gera, S. (2012). A waitlist-controlled trial of behavioral parent training for fathers of children with ADHD. *Journal of Clinical Child & Adolescent Psychology*, *41*(3), 337–345. doi:10.1080/15374416.2012.654464

Ferrin, M., Perez-Ayala, V., El-Abd, S., Lax-Pericall, T., Jacobs, B., Bilbow, A., & Taylor, E. (2016). A randomized controlled trial evaluating the efficacy of a psychoeducation program for families of children and adolescents with ADHD in the United Kingdom: Results after a 6-month follow-up. *Journal of Attention Disorders*. doi:10.1177/1087054715626509

Ferrin, M., & Taylor, E. (2011). Child and caregiver issues in the treatment of attention deficit-hyperactivity disorder: Education, adherence and treatment choice. *Future Neurology*, *6*(3), 399–413. doi:10.2217/fnl.11.5

Gevensleben, H., Holl, B., Albrecht, B., Schlamp, D., Kratz, O., Studer, P., … Heinrich, H. (2009). Distinct EEG effects related to neurofeedback training in children with ADHD: A randomized controlled trial. *International Journal of Psychophysiology*, *74*(2), 149–157. doi:10.1016/j.ijpsycho.2009.08.005

Gevensleben, H., Holl, B., Albrecht, B., Vogel, C., Schlamp, D., Kratz, O., … Heinrich, H. (2009). Is neurofeedback an efficacious treatment for ADHD? A randomised controlled clinical trial. *Journal of Child Psychology and Psychiatry*, *50*(7), 780–789. doi:10.1111/j.1469-7610.2008.02033.x

Gevensleben, H., Rothenberger, A., Moll, G. H., & Heinrich, H. (2012). Neurofeedback in children with ADHD: Validation and challenges. *Expert Review of Neurotherapeutics*, *12*(4), 447–460. doi:10.1586/ern.12.22

Gilmore, K. (2000). A psychoanalytic perspective on attention-deficit/hyperactivity disorder. *Journal of the American Psychoanalytic Association*, *48*(4), 1259–1293. doi:10.1177/00030651000480040901

Gu, Y., Xu, G., & Zhu, Y. (2016). A randomized controlled trial of mindfulness-based cognitive therapy for college students with ADHD. *Journal of Attention Disorders*. doi:10.1177/1087054716686183

Haack, L. M., Villodas, M., McBurnett, K., Hinshaw, S., & Pfiffner, L. J. (2016). Parenting as a mechanism of change in psychosocial treatment for youth with ADHD, predominantly inattentive presentation. *Journal of Abnormal Child Psychology*, *45*(5), 841–855. doi:10.1007/s10802-016-0199-8

Haydicky, J., Shecter, C., Wiener, J., & Ducharme, J. M. (2015). Evaluation of MBCT for adolescents with ADHD and their parents: Impact on individual and family functioning. *Journal of Child and Family Studies*, *24*(1), 76–94. doi:10.1007/s10826-013-9815-1

Hayes, S. C., Follette, V. M., & Linehan, M. M. (Eds.). (2004). *Mindfulness and acceptance: Expanding the cognitive-behavioral tradition*. New York, NY: Guilford Press.

Hayes, S. C., Luoma, J. B., Bond, F. W., Masuda, A., & Lillis, J. (2006). Acceptance and commitment therapy: Model, processes and outcomes. *Behaviour Research and Therapy*, *44*(1), 1–25. doi:10.1016/j.brat.2005.06.006

Herbert, S. D., Harvey, E. A., Roberts, J. L., Wichowski, K., & Lugo-Candelas, C. I. (2013). A randomized controlled trial of a parent training and emotion socialization program for families of hyperactive preschool-aged children. *Behavior Therapy*, *44*(2), 302–316. doi:10.1016/j.beth.2012.10.004

Hepark, S., Janssen, L., de Vries, A., Schoenberg, P. L. A., Donders, R., Kan, C. C., & Speckens, A. E. M. (2015). The efficacy of adapted MBCT on core symptoms and executive functioning in adults with ADHD: A preliminary randomized controlled trial. *Journal of Attention Disorders*. doi:10.1177/1087054715613587

Hinshaw, S. P., Owens, E. B., Wells, K. C., Kraemer, H. C., Abikoff, H. B., Arnold, L. E., … Wigal, T. (2000). Family processes and treatment outcome in the MTA:

Negative/ineffective parenting practices in relation to multimodal treatment. *Journal of Abnormal Child Psychology*, *28*(6), 555–568.

Hodgson, K., Hutchinson, A. D., & Denson, L. (2014). Nonpharmacological treatments for ADHD: A meta-analytic review. *Journal of Attention Disorders*, *18*(4), 275–282. doi:10.1177/1087054712444732

Hölzel, B. K., Lazar, S. W., Gard, T., Schuman-Olivier, Z., Vago, D. R., & Ott, U. (2011). How does mindfulness meditation work? Proposing mechanisms of action from a conceptual and neural perspective. *Perspectives on Psychological Science*, *6*(6), 537–559. doi:10.1177/1745691611419671

Ise, E., Kierfeld, F., & Döpfner, M. (2015). One-year follow-up of guided self-help for parents of preschool children with externalizing behavior. *Journal of Primary Prevention*, *36*(1), 33–40. doi:10.1007/s10935-014-0374-z

Jarrett, M. A., & Ollendick, T. H. (2008). A conceptual review of the comorbidity of attention-deficit/hyperactivity disorder and anxiety: Implications for future research and practice. *Clinical Psychology Review*, *28*(7), 1266–1280. doi:10.1016/j.cpr.2008.05.004

Kabat-Zinn, J. (2003). Mindfulness-based interventions in context: Past, present, and future. *Clinical Psychology: Science and Practice*, *10*(2), 144–156. doi:10.1093/clipsy.bpg016

Keith, J. R., Blackwood, M. E., Mathew, R. T., & Lecci, L. B. (2016). Self-reported mindful attention and awareness, go/no-go response-time variability, and attention-deficit hyperactivity disorder. *Mindfulness*, *8*(3), 765–744. doi:10.1007/s12671-016-0655-0

Keng, S.-L., Smoski, M. J., & Robins, C. J. (2011). Effects of mindfulness on psychological health: A review of empirical studies. *Clinical Psychology Review*, *31*(6), 1041–1056. doi:10.1016/j.cpr.2011.04.006

Kierfeld, F., Ise, E., Hanisch, C., Görtz-Dorten, A., & Döpfner, M. (2013). Effectiveness of telephone-assisted parent-administered behavioural family intervention for preschool children with externalizing problem behaviour: A randomized controlled trial. *European Child & Adolescent Psychiatry*, *22*(9), 553–565. doi:10.1007/s00787-013-0397-7

Klein, R. G., Abikoff, H., Hechtman, L., & Weiss, G. (2004). Design and rationale of controlled study of long-term methylphenidate and multimodal psychosocial treatment in children with ADHD. *Journal of the American Academy of Child & Adolescent Psychiatry*, *43*(7), 792–801. doi:10.1097/01.chi.0000128798.91601.fe

Klein, R. G., Abikoff, H., Klass, E., Ganeles, D., Seese, L. M., & Pollack, S. (1997). Clinical efficacy of methylphenidate in conduct disorder with and without attention deficit hyperactivity disorder. *Archives of General Psychiatry*, *54*(12), 1073–1080. doi:10.1001/archpsyc.1997.01830240023003

Langberg, J. M. (2011). *Homework, Organization, and Planning Skills (HOPS) interventions: A treatment manual*. Bethesda, MD: National Association of School Psychologists.

Langberg, J. M., & Becker, S. P. (2012). Does long-term medication use improve the academic outcomes of youth with attention-deficit/hyperactivity disorder? *Clinical Child and Family Psychology Review*, *15*(3), 215–233. doi:10.1007/s10567-012-0117-8

Langberg, J. M., Becker, S. P., Epstein, J. N., Vaughn, A. J., & Girio-Herrera, E. (2013). Predictors of response and mechanisms of change in an organizational skills intervention for students with ADHD. *Journal of Child and Family Studies*, *22*(7), 1000–1012. doi:10.1007/s10826-012-9662-5

Langberg, J. M., Epstein, J. N., Becker, S. P., Girio-Herrera, E., & Vaughn, A. J. (2012). Evaluation of the Homework, Organization, and Planning Skills (HOPS) intervention for

middle school students with ADHD as implemented by school mental health providers. *School Psychology Review*, *41*(3), 342–364.

Langberg, J. M., Evans, S. W., Schultz, B. K., Becker, S. P., Altaye, M., & Girio-Herrera, E. (2016). Trajectories and predictors of response to the challenging horizons program for adolescents with ADHD. *Behavior Therapy*, *47*(3), 339–354. doi:10.1016/j.beth.2016.01.001

Lasisi, D., Ani, C., Lasebikan, V., Sheikh, L., & Omigbodun, O. (2017). Effect of attention-deficit-hyperactivity-disorder training program on the knowledge and attitudes of primary school teachers in Kaduna, North West Nigeria. *Child and Adolescent Psychiatry and Mental Health*, *11*, 15. doi:10.1186/s13034-017-0153-8

Leuzinger-Bohleber, M., Laezer, K. L., Pfenning-Meerkoetter, N., Fischmann, T., Wolff, A., & Green, J. (2011). Psychoanalytic treatment of ADHD children in the frame of two extraclinical studies: The Frankfurt Prevention Study and the EVA Study. *Journal of Infant, Child, and Adolescent Psychotherapy*, *10*(1), 32–50. doi:10.1080/15289168.2011.575703

Loe, I. M., & Feldman, H. M. (2007). Academic and educational outcomes of children with ADHD. *Journal of Pediatric Psychology*, *32*(6), 643–654. doi:10.1093/jpepsy/jsl054

McGrath, P. J., Lingley-Pottie, P., Thurston, C., MacLean, C., Cunningham, C., Waschbusch, D. A., … & W. Chaplin. (2011). Telephone-based mental health interventions for child disruptive behavior or anxiety disorders: Randomized trials and overall analysis. *Journal of the American Academy of Child & Adolescent Psychiatry*, *50*(11), 1162–1172. doi:10.1016/j.jaac.2011.07.013

Meisel, V., Servera, M., Garcia-Banda, G., Cardo, E., & Moreno, I. (2014). Reprint of "Neurofeedback and standard pharmacological intervention in ADHD: A randomized controlled trial with six-month follow-up." *Biological Psychology*, *95*, 116–125. doi:10.1016/j.biopsycho.2013.09.009

Meppelink, R., de Bruin, E. I., & Bögels, S. M. (2016). Meditation or medication? Mindfulness training versus medication in the treatment of childhood ADHD: A randomized controlled trial. *BMC Psychiatry*, *16*, 267. doi:10.1186/s12888-016-0978-3

Micoulaud-Franchi, J.-A., Geoffroy, P. A., Fond, G., Lopez, R., Bioulac, S., & Philip, P. (2014). EEG neurofeedback treatments in children with ADHD: An updated meta-analysis of randomized controlled trials. *Frontiers in Human Neuroscience*, *8*, 906. doi:10.3389/fnhum.2014.00906

Mikami, A. Y., Lerner, M. D., Griggs, M. S., McGrath, A., & Calhoun, C. D. (2010). Parental influence on children with attention-deficit/hyperactivity disorder: II. Results of a pilot intervention training parents as friendship coaches for children. *Journal of Abnormal Child Psychology*, *38*(6), 737–749. doi:10.1007/s10802-010-9403-4

Miranda, A., Presentación, M. J., & Soriano, M. (2002). Effectiveness of a school-based multicomponent program for the treatment of children with ADHD. *Journal of Learning Disabilities*, *35*(6), 546–562. doi:10.1177/00222194020350060601

Mitchell, J. T., Benson, J. W., Knouse, L. E., Kimbrel, N. A., & Anastopoulos, A. D. (2013). Are negative automatic thoughts associated with ADHD in adulthood? *Cognitive Therapy and Research*, *37*(4), 851–859. doi:10.1007/s10608-013-9525-4

Mitchell, J. T., McIntyre, E. M., English, J. S., Dennis, M. F., Beckham, J. C., & Kollins, S. H. (2013). A pilot trial of mindfulness meditation training for ADHD in adulthood: Impact on core symptoms, executive functioning, and emotion dysregulation. *Journal of Attention Disorders*. doi:10.1177/1087054713513328

Moëll, B., Kollberg, L., Nasri, B., Lindefors, N., & Kaldo, V. (2015). Living SMART: A randomized controlled trial of a guided online course teaching adults with ADHD or sub-clinical ADHD to use smartphones to structure their everyday life. *Internet Interventions*, *2*(1), 24–31. doi:10.1016/j.invent.2014.11.004

Monastra, V. J., Monastra, D. M., & George, S. (2002). The effects of stimulant therapy, EEG biofeedback, and parenting style on the primary symptoms of attention-deficit/hyperactivity disorder. *Applied Psychophysiology and Biofeedback*, *27*(4), 231–249. doi:10.1023/A:1021018700609

MTA Cooperative Group. (1999). A 14-month randomized clinical trial of treatment strategies for attention-deficit/hyperactivity disorder. *Archives of General Psychiatry*, *56*(12), 1073–1086. doi:10.1001/archpsyc.56.12.1073

National Institute for Health and Clinical Excellence. (2008). *Attention deficit hyperactivity disorder: Diagnosis and management of ADHD in children, young people and adults*. Retrieved October 6, 2017, from https://www.nice.org.uk/guidance/cg72

Nigg, J. T. (2013). Attention-deficit/hyperactivity disorder and adverse health outcomes. *Clinical Psychology Review*, *33*(2), 215–228. doi:10.1016/j.cpr.2012.11.005

Nijmeijer, J. S., Minderaa, R. B., Buitelaar, J. K., Mulligan, A., Hartman, C. A., & Hoekstra, P. J. (2008). Attention-deficit/hyperactivity disorder and social dysfunctioning. *Clinical Psychology Review*, *28*(4), 692–708. doi:10.1016/j.cpr.2007.10.003

Nussey, C., Pistrang, N., & Murphy, T. (2013). How does psychoeducation help? A review of the effects of providing information about Tourette syndrome and attention-deficit/hyperactivity disorder. *Child: Care, Health and Development*, *39*(5), 617–627. doi:10.1111/cch.12039

O'Brien, M., & Daley, D. (2011). Self-help parenting interventions for childhood behaviour disorders: A review of the evidence. *Child: Care, Health and Development*, *37*(5), 623–637. doi:10.1111/j.1365-2214.2011.01231.x

Ogrim, G., & Hestad, K. A. (2013). Effects of neurofeedback versus stimulant medication in attention-deficit/hyperactivity disorder: A randomized pilot study. *Journal of Child and Adolescent Psychopharmacology*, *23*(7), 448–457. doi:10.1089/cap.2012.0090

Pelham, W. E., Burrows-MacLean, L., Gnagy, E. M., Fabiano, G. A., Coles, E. K., Wymbs, B. T., … Waschbusch, D. A. (2014). A dose-ranging study of behavioral and pharmacological treatment in social settings for children with ADHD. *Journal of Abnormal Child Psychology*, *42*(6), 1019–1031. doi:10.1007/s10802-013-9843-8

Pelham, W. E., Fabiano, G. A., Waxmonsky, J. G., Greiner, A. R., Gnagy, E. M., Pelham, W. E., … Murphy, S. A. (2016). Treatment sequencing for childhood ADHD: A multiple-randomization study of adaptive medication and behavioral interventions. *Journal of Clinical Child & Adolescent Psychology*, *45*(4), 396–415. doi:10.1080/15374416.2015.1105138

Pelham, W. E., Foster, E. M., & Robb, J. A. (2007). The economic impact of attention-deficit/hyperactivity disorder in children and adolescents. *Ambulatory Pediatrics*, *7*(1), 121–131. doi:10.1016/j.ambp.2006.08.002

Pelham, W. E., Jr., & Hoza, B. (1996). Intensive treatment: A summer treatment program for children with ADHD. In A. Summer, D. E. D. Hibbs, & P. S. Jensen (Eds.), *Psychosocial treatments for child and adolescent disorders: Empirically based strategies for clinical practice* (pp. 311–340). Washington, DC: American Psychological Association.

Pelsser, L. M., Frankena, K., Toorman, J., & Rodrigues Pereira, R. (2017). Diet and ADHD, reviewing the evidence: A systematic review of meta-analyses of double-blind

placebo-controlled trials evaluating the efficacy of diet interventions on the behavior of children with ADHD. *PLoS ONE*, *12*(1), e0169277. doi:10.1371/journal.pone.0169277

Pettersson, R., Söderström, S., Edlund-Söderström, K., & Nilsson, K. W. (2014). Internet-based cognitive behavioral therapy for adults with ADHD in outpatient psychiatric care: A randomized trial. *Journal of Attention Disorders*, *21*(6), 508–521. doi:10.1177/1087054714539998

Pfiffner, L. J., Hinshaw, S. P., Owens, E., Zalecki, C., Kaiser, N. M., Villodas, M., & McBurnett, K. (2014). A two-site randomized clinical trial of integrated psychosocial treatment for ADHD-inattentive type. *Journal of Consulting and Clinical Psychology*, *82*(6), 1115–1127. doi:10.1037/a0036887

Pfiffner, L. J., & McBurnett, K. (1997). Social skills training with parent generalization: Treatment effects for children with attention deficit disorder. *Journal of Consulting and Clinical Psychology*, *65*(5), 749–757. doi:10.1037/0022-006X.65.5.749

Pfiffner, L. J., Mikami, A. Y., Huang-Pollock, C., Easterlin, B., Zalecki, C., & McBurnett, K. (2007). A randomized, controlled trial of integrated home-school behavioral treatment for ADHD, predominantly inattentive type. *Journal of the American Academy of Child & Adolescent Psychiatry*, *46*(8), 1041–1050. doi:10.1097/chi.0b013e318064675f

Polanczyk, G. V., Salum, G. A., Sugaya, L. S., Caye, A., & Rohde, L. A. (2015). Annual research review: A meta-analysis of the worldwide prevalence of mental disorders in children and adolescents. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *56*(3), 345–365. doi:10.1111/jcpp.12381

Rabipour, S., & Raz, A. (2012). Training the brain: Fact and fad in cognitive and behavioral remediation. *Brain and Cognition*, *79*(2), 159–179. doi:10.1016/j.bandc.2012.02.006

Ramsay, J. R., & Rostain, A. L. (2008). Adult ADHD research: Current status and future directions. *Journal of Attention Disorders*, *11*(6), 624–627. doi:10.1177/1087054708314590

Rapport, M. D., Alderson, R. M., Kofler, M. J., Sarver, D. E., Bolden, J., & Sims, V. (2008). Working memory deficits in boys with attention-deficit/hyperactivity disorder (ADHD): The contribution of central executive and subsystem processes. *Journal of Abnormal Child Psychology*, *36*(6), 825–837. doi:10.1007/s10802-008-9215-y

Rapport, M. D., Orban, S. A., Kofler, M. J., & Friedman, L. M. (2013). Do programs designed to train working memory, other executive functions, and attention benefit children with ADHD? A meta-analytic review of cognitive, academic, and behavioral outcomes. *Clinical Psychology Review*, *33*(8), 1237–1252. doi:10.1016/j.cpr.2013.08.005

Robb, J. A., Sibley, M. H., Pelham, W. E., Jr., Foster, E. M., Molina, B. S., Gnagy, E. M., & Kuriyan, A. B. (2011). The estimated annual cost of ADHD to the US education system. *School Mental Health*, *3*(3), 169–177. doi:10.1007/s12310-011-9057-6

Ros, R., & Graziano, P. A. (2017). Social functioning in children with or at risk for attention deficit/hyperactivity disorder: A meta-analytic review. *Journal of Clinical Child & Adolescent Psychology*, 1–23. doi:10.1080/15374416.2016.1266644

Safren, S. A., Otto, M. W., Sprich, S., Winett, C. L., Wilens, T. E., & Biederman, J. (2005). Cognitive-behavioral therapy for ADHD in medication-treated adults with continued symptoms. *Behaviour Research and Therapy*, *43*(7), 831–842. doi:10.1016/j.brat.2004.07.001

Schoenberg, P. L., Hepark, S., Kan, C. C., Barendregt, H. P., Buitelaar, J. K., & Speckens, A. E. (2014). Effects of mindfulness-based cognitive therapy on neurophysiological

correlates of performance monitoring in adult attention-deficit/hyperactivity disorder. *Clinical Neurophysiology*, *125*(7), 1407–1416. doi:10.1016/j.clinph.2013.11.031

Shapiro, S. L., Carlson, L. E., Astin, J. A., & Freedman, B. (2006). Mechanisms of mindfulness. *Journal of Clinical Psychology*, *62*(3), 373–386. doi:10.1002/jclp.20237

Sibley, M. H., Graziano, P. A., Kuriyan, A. B., Coxe, S., Pelham, W. E., Rodriguez, L., … Ward, A. (2016). Parent-teen behavior therapy + motivational interviewing for adolescents with ADHD. *Journal of Consulting and Clinical Psychology*, *84*(8), 699–712. doi:10.1037/ccp0000106

Smalley, S. L., Loo, S. K., Hale, T. S., Shrestha, A., McGough, J., Flook, L., & Reise, S. (2009). Mindfulness and attention deficit hyperactivity disorder. *Journal of Clinical Psychology*, *65*(10), 1087–1098. doi:10.1002/jclp.20618

So, C. Y. C., Leung, P. W. L., & Hung, S.-F. (2008). Treatment effectiveness of combined medication/behavioural treatment with Chinese ADHD children in routine practice. *Behaviour Research and Therapy*, *46*(9), 983–992. doi:10.1016/j.brat.2008.06.007

Sonuga-Barke, E. J., Brandeis, D., Cortese, S., Daley, D., Ferrin, M., Holtmann, M., … & European ADHD Guidelines Group. (2013). Nonpharmacological interventions for ADHD: Systematic review and meta-analyses of randomized controlled trials of dietary and psychological treatments. *American Journal of Psychiatry*, *170*(3), 275–289. doi.org/10.1176/appi.ajp.2012.12070991

Sonuga-Barke, E. J., Daley, D., Thompson, M., Laver-Bradbury, C., & Weeks, A. (2001). Parent-based therapies for preschool attention-deficit/hyperactivity disorder: A randomized, controlled trial with a community sample. *Journal of the American Academy of Child & Adolescent Psychiatry*, *40*(4), 402–408. doi:10.1097/00004583-200104000-00008

Sonuga-Barke, E. J., Thompson, M., Abikoff, H., Klein, R., & Brotman, L. M. (2006). Nonpharmacological interventions for preschoolers with ADHD: The case for specialized parent training. *Infants & Young Children*, *19*(2), 142–153. doi:10.1097/00001163-200604000-00007

Spencer, T., Biederman, J., Wilens, T., Doyle, R., Surman, C., Prince, J., … Faraone, S. (2005). A large, double-blind, randomized clinical trial of methylphenidate in the treatment of adults with attention-deficit/hyperactivity disorder. *Biological Psychiatry*, *57*(5), 456–463. doi:10.1016/j.biopsych.2004.11.043

Stevenson, C. S., Stevenson, R. J., & Whitmont, S. (2003). A self-directed psychosocial intervention with minimal therapist contact for adults with attention deficit hyperactivity disorder. *Clinical Psychology & Psychotherapy*, *10*(2), 93–101. doi:10.1002/cpp.356

Stevenson, C. S., Whitmont, S., Bornholt, L., Livesey, D., & Stevenson, R. J. (2002). A cognitive remediation programme for adults with attention deficit hyperactivity disorder. *Australian & New Zealand Journal of Psychiatry*, *36*(5), 610–616. doi:10.1046/j.1440-1614.2002.01052.x

Storebø, O. J., Gluud, C., Winkel, P., & Simonsen, E. (2012). Social-skills and parental training plus standard treatment versus standard treatment for children with ADHD: The randomised SOSTRA trial. *PLoS ONE*, *7*(6), e37280. doi:10.1371/journal.pone.0037280

Storebø, O. J., Skoog, M., Damm, D., Thomsen, P. H., Simonsen, E., & Gluud, C. (2011). Social skills training for attention deficit hyperactivity disorder (ADHD) in children aged 5 to 18 years. *Cochrane Database of Systematic Reviews*, *12*, CD008223. doi:10.1002/14651858.CD008223.pub2

Strohmeier, C. W., Rosenfield, B., DiTomasso, R. A., & Ramsay, J. R. (2016). Assessment of the relationship between self-reported cognitive distortions and adult ADHD, anxiety, depression, and hopelessness. *Psychiatry Research*, *238*, 153–158. doi:10.1016/j.psychres.2016.02.034

Tang, Y.-Y., Hölzel, B. K., & Posner, M. I. (2015). The neuroscience of mindfulness meditation. *Nature Reviews Neuroscience*, *16*(4), 213–225. doi:10.1038/nrn3916

Temple, R., & Ellenberg, S. S. (2000). Placebo-controlled trials and active-control trials in the evaluation of new treatments: Part 1. Ethical and scientific issues. *Annals of Internal Medicine*, *133*(6), 455–463. doi:10.7326/0003-4819-133-6-200009190-00014

Thompson, M. J., Laver-Bradbury, C., Ayres, M., Le Poidevin, E., Mead, S., Dodds, C., … & Sonuga-Barke, J. S. (2009). A small-scale randomized controlled trial of the revised New Forest parenting programme for preschoolers with attention deficit hyperactivity disorder. *European Child & Adolescent Psychiatry*, *18*(10), 605–616. doi:10.1007/s00787-009-0020-0

Torrente, F., López, P., Prado, D. A., Kichic, R., Cetkovich-Bakmas, M., Lischinsky, A., & Manes, F. (2014). Dysfunctional cognitions and their emotional, behavioral, and functional correlates in adults with attention deficit hyperactivity disorder (ADHD): Is the cognitive-behavioral model valid? *Journal of Attention Disorders*, *18*(5), 412–424. doi:10.1177/1087054712443153

Van den Hoofdakker, B. J., Nauta, M. H., Dijck-Brouwer, D. A., Van der Veen-Mulders, L., Sytema, S., Emmelkamp, P. M., … Hoekstra, P. J. (2012). Dopamine transporter gene moderates response to behavioral parent training in children with ADHD: A pilot study. *Developmental Psychology*, *48*(2), 567–574. doi:10.1037/a0026564

Van der Donk, M. L. A., Hiemstra-Beernink, A.-C., Tjeenk-Kalff, A. C., Van der Leij, A., & Lindauer, R. J. L. (2016). Predictors and moderators of treatment outcome in cognitive training for children with ADHD. *Journal of Attention Disorders*. doi:10.1177/1087054716632876

Van der Oord, S., Bögels, S. M., & Peijnenburg, D. (2012). The effectiveness of mindfulness training for children with ADHD and mindful parenting for their parents. *Journal of Child and Family Studies*, *21*(1), 139–147. doi:10.1007/s10826-011-9457-0

Vidal, R., Bosch, R., Nogueira, M., Gómez-Barros, N., Valero, S., Palomar, G., … & Ramos-Quiroga, J. A. (2013). Psychoeducation for adults with attention deficit hyperactivity disorder vs. cognitive behavioral group therapy: A randomized controlled pilot study. *Journal of Nervous and Mental Disease*, *201*(10), 894–900. doi:10.1097/NMD.0b013e3182a5c2c5

Vidal, R., Castells, J., Richarte, V., Palomar, G., García, M., Nicolau, R., … Ramos-Quiroga, J. A. (2015). Group therapy for adolescents with attention-deficit/hyperactivity disorder: A randomized controlled trial. *Journal of the American Academy of Child & Adolescent Psychiatry*, *54*(4), 275–282. doi:10.1016/j.jaac.2014.12.016

Virta, M., Salakari, A., Antila, M., Chydenius, E., Partinen, M., Kaski, M., … Iivanainen, M. (2010). Short cognitive behavioral therapy and cognitive training for adults with ADHD: A randomized controlled pilot study. *Neuropsychiatric Disease and Treatment*, *6*, 443–453.

Volpe, R. J., & Fabiano, G. A. (2013). *Daily behavior report cards: An evidence-based system of assessment and intervention*. New York, NY: Guilford Press.

Waschbusch, D. A. (2002). A meta-analytic examination of comorbid

hyperactive-impulsive-attention problems and conduct problems. *Psychological Bulletin*, *128*(1), 118–150. doi:10.1037/0033-2909.128.1.118

Webster-Stratton, C. H., Reid, M. J., & Beauchaine, T. (2011). Combining parent and child training for young children with ADHD. *Journal of Clinical Child & Adolescent Psychology*, *40*(2), 191–203. doi:10.1080/15374416.2011.546044

Willcutt, E. G. (2012). The prevalence of DSM-IV attention-deficit/hyperactivity disorder: A meta-analytic review. *Neurotherapeutics*, *9*(3), 490–499. doi:10.1007/s13311-012-0135-8

Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity of the executive function theory of attention-deficit/hyperactivity disorder: A meta-analytic review. *Biological Psychiatry*, *57*(11), 1336–1346. doi:10.1016/j.biopsych.2005.02.006

Wolraich, M. L., Wibbelsman, C. J., Brown, T. E., Evans, S. W., Gotlieb, E. M., Knight, J. R., … Wilens, T. (2005). Attention-deficit/hyperactivity disorder among adolescents: A review of the diagnosis, treatment, and clinical implications. *Pediatrics*, *115*(6), 1734–1746. doi:10.1542/peds.2004-1959

Young, S., & Bramham, J. (2006). *ADHD in adults: A psychological guide to practice*. Chichester, UK: John Wiley & Sons.

Young, S., Khondoker, M., Emilsson, B., Sigurdsson, J. F., Philipp-Wiegmann, F., Baldursson, G., … Gudjonsson, G. (2015). Cognitive-behavioural therapy in medication-treated adults with attention-deficit/hyperactivity disorder and co-morbid psychopathology: A randomized controlled trial using multi-level analysis. *Psychological Medicine*, *45*(13), 2793–2804. doi:10.1017/S0033291715000756

Zilverstand, A., Sorger, B., Slaats-Willemse, D., Kan, C. C., Goebel, R., & Buitelaar, J. K. (2017). fMRI neurofeedback training for increasing anterior cingulate cortex activation in adult attention deficit hyperactivity disorder: An exploratory randomized, single-blinded study. *PLOS One*, *12*(1), e0170795. doi:10.1371/journal.pone.0170795

Zwi, M., Jones, H., Thorgaard, C., York, A., & Dennis, J. A. (2011). Parent training interventions for attention deficit hyperactivity disorder (ADHD) in children aged 5 to 18 years. *Cochrane Database of Systematic Reviews*, *12*, CD003018. doi:10.1002/14651858.CD003018.pub3

17

The Treatment of Insomnia

Elisabeth Hertenstein,[1] Bogdan Voinescu,[2,3] and Dieter Riemann[1]

[1] *Department of Clinical Psychology and Psychophysiology, Center for Mental Disorders, University Medical Center Freiburg, Germany*

[2] *Department of Clinical Psychology and Psychotherapy, Babeş-Bolyai University, Cluj-Napoca, Romania*

[3] *Institute of Psychiatry, Psychology and Neuroscience, King's College London, London, UK*

Sleep is a central aspect of physical and mental health. Dissatisfaction with sleep is highly frequent in the general population. In a representative epidemiological study in Europe, Ohayon and Reynolds (2009) found that 37% of the participants had a sleep complaint. Insomnia is associated with long-term adverse health consequences, such as cardiovascular events and depression (Baglioni et al., 2011; Chien et al., 2010). The present chapter presents recent diagnostic criteria, central psychological theories, and psychological and behavioral treatments for insomnia. The primary aim of the chapter is to provide a review of existing treatments in terms of both empirical support for their efficacy and the mechanisms hypothesized to mediate or moderate their effects. For this review, we refer to the framework proposed by David and Montgomery (2011). They suggest rating the state of evidence for each psychological treatment on a scale with three proficiencies: well supported, equivocal data, or strong contradictory evidence. This rating is provided for both efficacy and mechanisms, resulting in a framework with nine categories (see Chapter 1).

17.1 Description of the Disorder

17.1.1 Definitions

The term "insomnia" can describe both a symptom and a disorder. Moreover, workers in the field have used a variety of definitions and diagnostic categories to describe insomnia. The three current central classification systems, namely the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013), the tenth edition of the International Classification of Diseases (ICD-10; World Health Organization, 1993), and the third edition of the International Classification of Sleep Disorders (ICSD; American Academy of Sleep Medicine, 2014), provide different definitions of insomnia. All of them require, beyond the sleep complaint, significant distress and/or impairment in daytime functioning, although no sleep-time-related cut-offs are postulated.

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

The ICSD provides a categorization of sleep disorders for diagnostic and research purposes. According to the ICD-10, nonorganic insomnia is defined as quantitatively or qualitatively disrupted sleep at least three times per week for a duration of at least 1 month, resulting in distress and psychosocial or occupational impairment. In contrast to 4 weeks in the ICD-10, a diagnosis of insomnia disorder according to the DSM-5 criteria requires a duration of the complaint for at least 3 months. Whereas nonorganic insomnia according to the ICD-10 requires the presence of an identifiable stressor related to the onset, antecedent factors are not mentioned in the DSM-5.

17.1.2 Subtypes

According to the DSM-5, insomnia disorder can be episodic (i.e., symptoms are present for at least 1 month but for less than 3 months), persistent (i.e., symptoms last for at least 3 months), and recurrent (i.e., two or more episodes within 1 year). Insomnia disorder can present as a single disorder or comorbidly with other sleep disorders, other medical illnesses, or a psychiatric disorder.

17.1.3 Assessment

The diagnosis of insomnia is usually made after a detailed clinical interview, including current sleep and wake behaviors, sleep environment, daytime consequences of insomnia, sleep history, treatment history, history of mental and medical disorders, and substance use. Sleep diaries (Carney et al., 2012) and questionnaires such as the Insomnia Severity Index (Morin, 1993) and the Pittsburgh Sleep Quality Index (Buysse, Reynolds, Monk, & Berman, 1989) complement the diagnostic process. Sleep diaries are widely recognized as a main outcome in clinical trials on insomnia because they provide a detailed assessment of subjective sleep over a period of 1 or 2 weeks. They are often preferred over objective measures such as polysomnography (PSG) or actigraphy because they are closer to patients' own perceptions of their complaint (Carney et al., 2012). If symptoms of other sleep disorders, such as sleep-related breathing disorders, sleep-related movement disorders, other parasomnias, or narcolepsy, are reported by the patient or a bed partner, a thorough diagnostic clarification in a sleep laboratory including PSG should be performed, while in some cases actigraphy can be useful to provide additional information about sleep and wakefulness (Schutte-Rodin, Broch, Buysse, Dorsey, & Sateia, 2008).

17.1.4 Prevalence and Risk Factors

According to a cross-sectional study involving 25,579 individuals aged 15 years and over and representative of the general population of Finland, France, Germany, Italy, Portugal, Spain, and the United Kingdom, chronic insomnia is a prevalent health problem afflicting about 7–10% of the general population, depending on the rigor of the applied diagnostic criteria (Ohayon & Reynolds, 2009). The prevalence of primary insomnia, defined as persistent insomnia without comorbid mental or somatic disorders accounting for the sleep problems, is estimated at 2–4% (Ohayon, 2002). The prevalence generally increases with age, although this increase is more prominent in women (Sivertsen, Krokstad, Øverland, & Mykletun, 2009). The risk ratio of women is

estimated at 1.41 compared with men (Zhang & Wing, 2006). In addition to female gender, menopause has also been linked to insomnia (Ohayon, 2002). Longitudinal studies indicate that insomnia is mostly a persistent condition, particularly in those with more severe insomnia (Morin et al., 2009). While often left unrecognized and untreated in clinical practice (Hamblin, 2007), chronic insomnia is associated with a reduced health-related quality of life (Kyle, Morgan, & Espie, 2010), enhanced absenteeism and reduced job performance (Simon & VonKorff, 1997), cognitive impairments (Fortier-Brochu, Beaulieu-Bonneau, Ivers, & Morin, 2012), difficulties with emotional functioning and social participation, and limited life aspirations (Kyle, Espie, & Morgan, 2010). Furthermore, chronic insomnia has been identified as a risk factor for the first onset of a major depressive episode (Baglioni et al., 2011).

Psychiatric disorders that are often comorbid with insomnia are anxiety disorders, adjustment disorders, and bipolar disorder (Ohayon & Reynolds, 2009). Musculoskeletal and articular diseases (e.g., back pain, arthritis, fibromyalgia) are the most common medical comorbidities of insomnia, followed by heart diseases, gastrointestinal diseases, and pulmonary diseases (Budhiraja, Roth, Hudgel, Budhiraja, & Drake, 2011; Ohayon & Reynolds, 2009). Insomnia can be comorbid with other sleep disorders, most often circadian rhythm disorders (Ohayon, Lemoine, Arnaud-Briant, & Dreyfus, 2002). Insomnia is also a common comorbidity of alcohol abuse and dependence (Brower, Krentzman, & Robinson, 2011) and is often reported by smokers (Moreno-Coutiño, Calderón-Ezquerro, & Drucker-Colín, 2007) or as a side-effect of various medications (Lichstein, Taylor, McCrae, & Ruiter, 2011). Insomnia leads to very high costs for societies due to a reduced ability to work and increased health care expenses (Kessler et al., 2011).

17.2 Psychological Theories and Mechanisms of Change

17.2.1 Classical Conditioning

Sleep can be viewed as a classically conditioned reflex response that is triggered by sleepiness (Ribordy & Denney, 1977). Likewise, falling asleep can be understood as an example of operant conditioning—that is, the act of falling asleep is reinforced by sleep (Bootzin & Perlis, 1992). In healthy sleepers, the sleeping environment is robustly paired with sleepiness and sleep. However, in chronic insomniacs, the conditioned stimulus (bedroom) is often also associated with wakefulness, worry, and anxiety. The rationale of stimulus control therapy is to reestablish a strong connection between the sleeping environment, sleepiness, and sleep.

Empirical investigations of classical conditioning of sleep have been conducted in two older case studies in which sleep was induced by the intravenous administration of a barbiturate in patients with severe insomnia. Sleep onset, which occurs several seconds after the injection, was paired with a neutral stimulus, such as counting or the noise of a metronome, which was then used to facilitate sleep onset at home (without an injection) (Evans & Bond, 1969; Poser, Fenton, & Scotton, 1965). Whereas the classical conditioning paradigm per se is empirically well supported, evidence for the hypothesis that sleep disturbances and chronic insomnia are primarily driven by conditioned learning must be classified as weak. The reported two investigations are case studies that are insufficiently documented: Whereas Evans and Bond (1969) state that their client's sleep

improved after the conditioning procedure, they do not report whether he used counting as a conditioned stimulus when sleeping at home.

In a different, interesting approach, researchers not only investigated the classical conditioning paradigm per se but also aimed at isolating different components of a stimulus control intervention. In a study of 47 undergraduate students with sleep problems, Zwart and Lisman (1979) randomized the participants to five groups: stimulus control as usual, an intervention lacking the critical contingency aspect (arising a fixed number of times within a 20-minute resting period), a countercontrol in which subjects were required to stay in bed and read or watch TV when not able to sleep, a time-related control that only included the aspects of stimulus control that overlap with sleep restriction treatment, and a waitlist. They observed that the regular stimulus control group and the countercontrol group improved to a similar extent (which contradicts the classical conditioning paradigm). Subjects were told to expect improvements 4 weeks after treatment. Each treated group showed more improvement than the waitlist group at this time. In contrast, 3 weeks after treatment, when subjects were told not to expect any improvements, only the regular stimulus control group and the counter control group improved. The findings of this study support the hypothesis that sleep can be improved with stimulus control therapy; however, they challenge the classical conditioning paradigm as a mechanism of action of this intervention. Similarly, Davies, Lacks, Storandt, and Bertelson (1986) found that a countercontrol treatment reduced sleep maintenance insomnia by 30% in a sample of 34 insomniacs.

17.2.2 Behavioral Model

The behavioral model is also known as the "3P" model or diathesis–stress model, and explains the interaction between *p*redisposing, *p*recipitating, and *p*erpetuating factors (Spielman, Caruso, & Glovinsky, 1987). Predisposing factors (e.g., genetics, early trauma, learning history) render a person vulnerable to the experience of sleeplessness when exposed to stressors. Precipitating factors can be all kinds of stressors strong enough to exceed an individual's threshold for disturbing the sleep–wake rhythm. In an attempt to resolve their sleep problem, affected persons may develop behaviors that in fact worsen and perpetuate insomnia: extending bedtimes, napping during the day, trying to compensate for sleep loss the next day or at weekends, or using alcohol and/or other drugs in order to fall asleep faster. Contrary to the patient's intentions, however, a reduced sleep pressure and an irregularity of the sleep–wake rhythm consolidate the sleep problem. Alcohol, which is sometimes used to fall asleep faster, may actually decrease SOL (sleep onset latency, or time needed to fall asleep), but it often interferes with the person's natural sleep–wake rhythm and induces nighttime awakenings (Roehrs & Roth, 2001).

A population-based twin cohort study ($N = 1{,}782$) provides evidence for the hypothesis that sleep reactivity to stress constitutes a genetic vulnerability for the onset of insomnia (Drake, Friedman, Wright, & Roth, 2011). In this study, the heritability of sleep reactivity was found to be 29% in females and 43% in males. The heritability of insomnia was 55% in females and 43% in males. Comparable results (moderate heritability estimates for insomnia symptoms, 34–45%) were found in the Finnish Twin Cohort Study ($N = 12{,}502$) (Hublin, Partinen, Koskenvuo, & Kaprio, 2011). Concerning precipitating factors, a Swedish population-based study with a combined cross-sectional and prospective design ($N = 1{,}936$) found that anxiety (compared with depression) plays a

key role in the development of insomnia over the course of 1 year (Jansson & Linton, 2006). In another analysis in a different subsample ($N = 1{,}873$, aged 50–60 years) of the original population-based sample, the authors found that perceived work stressors were significantly but weakly (explained variance < 10%) related to the onset and maintenance of insomnia (Jansson-Fröjmark, Lundqvist, Lundqvist, & Linton, 2007).

Interestingly, Morin, Rodrigue, and Ivers (2003) found in a longitudinal comparison study ($N = 67$) that, while insomniacs and good sleepers reported a comparable number of stressful events over a period of 21 consecutive days, both the impact of daily minor stressors and the intensity of major negative life events were rated higher by the insomniacs, while the patients experienced less control over stressful events. This suggests that not the occurrence of stressful events per se but rather their appraisal and a perceived lack of control increase vulnerability to insomnia.

Haario, Rahkonen, Laaksonen, Lahelma, and Lallukka (2013) found that sleep disturbances were associated with subsequent alcohol abuse and physical inactivity. Alcohol abuse was also associated with subsequent insomnia symptoms, suggesting a bidirectional relationship between adverse health behaviors and sleep disturbances. Two studies suggest that the frequent use of safety behaviors such as clock watching and avoiding emotional strains in the evening is associated with persistent insomnia (Norell-Clarke, Jansson-Fröjmark, Tillfors, Harvey, & Linton, 2014; Ree & Harvey, 2004). Other evidence, however, indicates that, while the frequency of safety behavior use is correlated with the severity of insomnia symptoms, safety behaviors do not predict persistent insomnia (Yang, Lin, & Cheng, 2013). This outcome is in line with the findings of another study, which found that only the perceived utility, and not the frequency of use, of safety behaviors predicted the severity of insomnia (Hood, Carney, & Harris, 2011).

Perpetuating factors are the part of the 3P model most relevant for the conceptualization of therapeutic interventions. Whereas predisposing factors explain why some individuals develop insomnia and others do not, and precipitating factors help us to understand why insomnia occurs at a certain point in one's life, perpetuating factors provide a rationale for the development of chronic as opposed to acute and transient insomnia. Extended bedtimes and daytime napping (common perpetuating behaviors) are addressed by sleep restriction therapy. Complex clinical trials have demonstrated that the effects of sleep restriction therapy are indeed mediated by reduced time in bed, supporting the role of maladaptive behaviors in the maintenance of insomnia (see Section 17.2.7 for more details).

17.2.3 Hyperarousal Model

The hyperarousal model of insomnia provides an integrative perspective, shedding light on psychological as well as physical aspects of the disorder (Riemann et al., 2010). The model postulates that patients with chronic insomnia are characterized by a heightened arousal during nighttime and daytime that manifests itself on various levels, including cognitive and motor activity, the autonomic nervous system, neuroendocrinology, neuroimmunology, and measures of brain activity. Conditioned arousal (instead of relaxation and sleepiness) associated with bedtime is assumed to be one important perpetuating factor of chronic insomnia besides other factors (e.g., extended bedtimes). Hyperarousal may contribute to extended SOL and WASO (wake time after sleep onset, or time spent awake after initially falling asleep) as well as the perception of unrestful

sleep, sleep state misperception, or paradoxical insomnia (Riemann et al., 2010). The hyperarousal model explains the discrepancy between the subjective perception of severe sleep loss and minor objective disruptions of sleep as a result of a dysbalance between activating and sleep-promoting brain systems. The model assumes a malfunction of the "key switch" between activation/arousal and deactivation/promotion of sleep. Whereas activating pathways (e.g., orexin neurons in the hypothalamus) are normally deactivated during sleep, they are hyperactive for 24 hours in patients with insomnia. This results in an experience of fatigue due to sleep loss accompanied by an inability to sleep due to the heightened arousal.

Empirical evidence supporting the hyperarousal model predominantly stems from cross-sectional studies comparing patients with chronic insomnia to healthy controls. A wide basis of evidence using various methods supports the hypothesis of hyperarousal in patients with insomnia (Riemann et al., 2010). However, due to the cross-sectional nature of the existing evidence, it remains to be elucidated whether hyperarousal is a primary predisposing factor for insomnia, whether hyperarousal is a consequence of chronic sleep loss, or whether there is a bidirectional relationship between sleep and arousal. Thus, concerning implications for treatment, it can be suspected that interventions that are appropriate to reduce arousal may be effective to improve sleep in patients with insomnia; however, this conclusion cannot be considered as confirmed because it is possible to imagine many different causal pathways in the development of arousal, disturbances of the sleep–wake rhythm, and insomnia.

Addressing somatic hyperarousal as a rationale for relaxation interventions for insomnia, Lichstein and colleagues included measures of subjective and objective arousal/relaxation as adherence ratings into two clinical trials (Lichstein, Riedel, Wilson, Lester, & Aguillard, 2001; Lichstein, Wilson, & Johnson, 2000). In the first study, the authors randomized 44 older patients with insomnia secondary to medical or psychiatric conditions to a combination of relaxation and stimulus control treatment or a no-treatment control condition. They calculated an adherence index consisting of minutes of relaxation practice, change in relaxation ratings from before to after practice, change in pulse rate from before to after practice, and the proportion of stimulus control instructions followed. The adherence index was related to improvements in WASO, sleep efficiency, and sleep quality. In the second study, 89 older patients with primary insomnia were randomized to relaxation, sleep restriction, or a placebo desensitization control group. In the relaxation group, a higher duration of practice per week predicted better sleep-related treatment outcome at posttreatment and at follow-up. The two studies provide preliminary evidence that arousal can be reduced in patients with insomnia and that increased relaxation practice may be associated with sleep improvements.

17.2.4 Attention Intention Effort Model

The attention intention effort (AIE) model by Espie, Broomfield, MacMahon, Macphee, and Taylor (2006) explains the development and maintenance of psychophysiological insomnia starting from sleep-related processes in healthy sleepers. In contrast to many activities in daily life, sleep is an automatic process that is inhibited rather than improved by explicit attention, intention, and effort. Whereas good sleepers are essentially passive, patients with insomnia are typically very concerned about their sleep. Compared to

healthy individuals, they show heightened attention to sleep-related stimuli, an explicit intention to fall asleep, and increased sleep effort, which paradoxically results in prolonged sleep latencies and may foster behaviors that are in the long term incompatible with sleep (e.g., extended bedtimes and substance use).

As the proposed model consists of three components, experimental studies on each of the components are needed in order to verify or falsify the model. Espie and colleagues (2006) reviewed and summarized relevant studies. The attention component can be classified as well supported in real-world experiments and computerized experiments, whereby, in the latter case, well-established paradigms such as the Stroop task, the dot-probe task, and the flicker task have been used. Patients with primary insomnia show an attentional bias toward sleep-related stimuli compared to good sleepers. However, there are fewer studies on the intention and effort components. Indirect evidence for a potentially paradoxical role of explicit intention and effort stems from observations during the multiple sleep latency test (MSLT; Richardson et al., 1978). The MSLT is a diagnostic test consisting of four 20-minute daytime nap opportunities that is based on the assumption that the sleepier people are, the faster they will fall asleep. Surprisingly, patients with paradoxical intention do not show sleepiness on the MSLT. They even show increased sleep latencies compared with healthy controls (Friedman et al., 2000). Considering the reported sleep deficits and daytime fatigue, the opposite could be expected. A possible explanation of this finding is that the MSLT includes an explicit demand to fall asleep, which in patients with paradoxical intention could lead to a kind of "performance pressure" and consequently prolonged sleep latencies.

In a study by Ansfield, Wegner, and Bowser (1996) with 110 undergraduates, two factors were experimentally manipulated: sleep intention (instruction to fall asleep as fast as possible vs. whenever you want) and "cognitive load" (sleep-disturbing marching band music vs. sleep-conducive music). The results showed that explicit intention to fall asleep fast was sleep disturbing only under high cognitive load. This experimental condition could possibly mirror the situation of patients with insomnia who experience both a high intention to sleep and high mental load—for example, in the form of stress and anxiety related to insomnia. Harvey (2003) found that thought suppression inhibited sleep in patients with insomnia and good sleepers. It is conceivable that thought suppression with the aim of calming the mind is what happens when someone intentionally tries to fall asleep (Harvey, 2001). In a study on 34 individuals with sleep onset insomnia, Broomfield and Espie (2003) showed that a paradoxical intention protocol was effective to reduce sleep effort and sleep performance anxiety as measured by the Glasgow Sleep Effort Scale but not subjective and objective SOL. In a study on 33 healthy sleepers, Rasskazova, Zavalko, Tkhostov, and Dorohov (2014) found that an instruction to fall asleep as fast as possible combined with a financial reward, compared to a neutral instruction, lead to increased WASO, number of awakenings, and arousal index during daytime napping as measured by PSG. Of note, this study found an effect of sleep effort on objectively measured sleep fragmentation; however, contrary to the hypotheses of the authors, the instruction did not have any effect on sleep latency or subjective appraisals of sleep quality.

In summary, an attentional bias toward sleep-related cues is well documented in patients with insomnia, and preliminary evidence supports the concept of sleep intention and effort as perpetuating factors. More research, particularly on patients

with clinical insomnia, is desirable. The AIE model provides a rationale for the efficacy of paradoxical intention treatment and is furthermore linked to acceptance-based treatments teaching an open, nonintentional attitude toward sleep.

17.2.5 Cognitive Model

The cognitive model explains the persistence and maintenance of insomnia with the help of typical thought patterns distinguishing patients with insomnia from healthy sleepers. A core assumption of the model is that altered thought patterns in insomnia patients exist both in the daytime and at night. Various authors have concentrated on cognitive processes in insomnia and evolved theoretical models; the most prominent cognitive model was published by Harvey (2005). She identified five interrelated processes distinguishing patients with chronic insomnia from healthy persons:

1. excessive worry about sleep and negative consequences of poor sleep, resulting in increased autonomic arousal, anxiousness, and emotional distress;
2. selective attention to and monitoring of internal and external sleep-related stimuli as a consequence of the association between sleep or loss of sleep and threat;
3. preoccupation with sleep-related cognitive content, resulting in an overestimation of sleep loss and its daytime consequences;
4. unhelpful beliefs about sleep;
5. safety behaviors (e.g., extended bedtimes, daytime napping, substance use), which are intended to improve sleep but actually reduce sleep pressure and lead to an irregular sleep–wake schedule.

Harvey (2005) reviewed empirical research on the five components of the cognitive model. Tang and Harvey (2004) experimentally manipulated cognitive arousal by randomizing 54 healthy sleepers to three conditions prior to a daytime nap. Anxious and neutral cognitive arousal led to an increased discrepancy between subjectively and objectively (actigraphy) measured sleep relative to a control group. Anxious cognitive arousal and physiological arousal were associated with greater SOL and reduced TST (total sleep time or sleep duration). Tang, Schmidt, and Harvey (2007) found that pre-sleep worry triggered by clock-monitoring was associated with increased SOL and greater overestimation of sleep disturbance in a sample of 38 patients with primary insomnia. Sunnhed and Jansson-Fröjmark (2013) reported that reductions in sleep-related worry were associated with improvements in WASO and TST, but not SOL, after cognitive–behavioral therapy for insomnia (CBT-I). The findings suggest that reductions of sleep-related worry may be an operating mechanism of CBT-I. However, as a multicomponent intervention was used, it is not known whether these changes were effects of cognitive therapy, of the other interventions, or of the treatment package as a whole. An alternative explanation for the findings is that reductions in worry occurred as a by-product of symptom reductions.

In a large randomly selected sample from the Swedish general population ($N = 3{,}600$), Jansson and Linton (2007) found that anxiety, depression, arousal, and the belief that insomnia has long-term negative consequences (as assessed with subscales of the Dysfuntional Beliefs and Attitudes About Sleep Scale [DBAS]; Morin, Vallières, & Ivers, 2007) were significantly related to the maintenance of insomnia. These variables explained 50% of the variance when the normal sleep group and the group with

insomnia were compared. The belief in the long-term consequences of insomnia explained the largest amount of variance, followed by arousal. Research supporting the concept of worry in insomnia also comes from studies using correlational measures, interviews, and audiotapes of pre-sleep mentation (Harvey, 2005).

However, Sanavio (1988) found that a tailored cognitive intervention did not have an advantage in treatment outcome for patients with high cognitive arousal. The authors divided 24 patients with psychophysiological insomnia into two groups with high and low cognitive arousal according to a questionnaire score. Within these groups, patients were randomized to either a cognitive intervention or electromyography (EMG) biofeedback. Both treatments were effective, though biofeedback led to greater reductions in pre-sleep tension whereas cognitive therapy was associated with greater reductions in pre-sleep intrusions. There was no added benefit if treatment matched the patient's complaint of cognitive hyperarousal (Sanavio, 1988).

As already outlined, many studies have demonstrated an attentional bias toward internal and external sleep-related cues in patients with insomnia using experimental paradigms such as the dot-probe task or the flicker task (Espie et al., 2006). A misperception of sleep, defined as a discrepancy between subjective estimates of sleep time (e.g., in a sleep diary) and objectively measured sleep (e.g., PSG, actigraphy), is also well documented. In a small study ($N = 21$) in which objective sleep was measured by PSG and subjective sleep was assessed with sleep diaries, patients with insomnia underestimated their total sleep time and overestimated their wakefulness (Mercer, Bootzin, & Lack, 2002). Likewise, another small study ($N = 20$) found that insomniacs and healthy sleepers were quite similar concerning sleep as measured by PSG; however, retrospective evaluations of total sleep time and reports of habitual sleep and daytime performance were markedly worse in patients with insomnia (Mendelson, James, Garnett, & Sack, 1986).

Both the cognitive model and the behavioral model point to the role of maladaptive coping strategies and safety behaviors in the maintenance of insomnia. Whereas the behavioral model stresses the dysregulation of the sleep–wake rhythm as a consequence of dysfunctional sleep-related behavior, the cognitive model emphasizes distorted beliefs about sleep that are never falsified due to the constant use of safety behaviors. Patients who regularly exert safety strategies in order to prevent insomnia and its feared daytime consequences will never experience that their beliefs about sleep are untrue or that a feared outcome will not occur, even if safety behaviors are omitted. In a cross-sectional online survey on a sample of 376 undergraduate students, Hood and colleagues (2011) investigated the link between cognitive and behavioral aspects of safety behaviors. Interestingly, they found that the perceived utility (i.e., underlying maladaptive beliefs), but not the actual frequency, of safety behavior use was associated with the severity of insomnia symptoms. In line with these results, Woodley and Smith (2006) found that depressive symptoms and dysfunctional beliefs, but not insomnia severity, predicted dysfunctional safety behaviors in a sample of 40 students with and without clinical insomnia. The results challenge the cognitive model, which predicts that the use of safety behaviors maintains or worsens insomnia and thus shows that the frequency of safety behavior use should be closely linked to insomnia severity. However, due to the cross-sectional nature of these studies, causal conclusions cannot be drawn, and it remains to be further elucidated whether maladaptive beliefs and safety behaviors actually play a causal role in the maintenance of insomnia symptoms.

The cognitive model is quite complex, consisting of five components that purportedly interact with each other. Several studies have demonstrated that changes in dysfunctional beliefs and attitudes mediate the effect of CBT (e.g., Schwartz & Carney, 2012). However, as pointed out by Harvey (2005), the aim of cognitive therapy is to address all five components of the model at night and during the day. Further research is needed in order to work out whether all five postulated components are related to the severity and maintenance of insomnia, whether they can be influenced by (cognitive) treatment, and whether these potential changes are related to symptom improvements. In particular, it would be interesting to clarify whether cognitive therapy as a stand-alone treatment specifically targets cognitive processes and whether or not cognitions are also influenced by other forms of treatment.

17.2.6 Metacognitive Model

Ong and colleagues (Ong & Sholtes, 2010; Ong, Ulmer, & Manber, 2012) proposed a metacognitive model of insomnia that can be considered an extension of the cognitive model and also encompasses parts of the AIE model concerning the role of awareness, intention, and effort in the maintenance of insomnia symptoms. Metacognition means becoming aware of one's own mental activity, of the way one relates to one's thoughts, or, in simplified terms, "thinking about thinking." The authors suggest that, whereas "primary arousal" is caused by the content of a thought, "secondary arousal" stems from a rigid attachment to these thoughts (Ong et al., 2012). Ong and Sholtes (2010) propose a mindfulness-based therapeutic approach to insomnia with the aim of increasing awareness of mental and physical states associated with insomnia and its daytime symptoms. Participants are taught to distinguish sleepiness from fatigue and to estimate their degree of arousal. The aim is that patients learn to adaptively respond to their current state instead of reacting automatically—for example, not trying to sleep if fatigued but not sleepy and in a state of high arousal.

Research on the metacognitive model of insomnia as a theoretical background of mindfulness- and acceptance-based therapies is still in a relatively early stage. In a study on patients with chronic pain and sleep complaints, it was found that psychological inflexibility (i.e., a nonaccepting attitude, experiential avoidance, and low mindfulness scores) were associated with poor sleep (McCracken, Williams, & Tang, 2011). To date, the mechanisms of action of mindfulness and acceptance for insomnia are not sufficiently specified and thus are still poorly understood. Measuring abstract constructs such as secondary arousal or the degree of fusion with sleep-related thoughts is still a challenge for further research.

17.2.7 Mediators of Treatment Effects

Besides research explicitly testing the validity of psychological theories and explanation models in experimental trials and correlational studies, theoretical assumptions behind therapeutic approaches are evaluated with the help of mediator analyses embedded into clinical trials. In 2012, Schwartz and Carney conducted a review of mediators of CBT-I, which are, according to the respective models, hypothesized to be associated with treatment effects: decreased time in bed for the behavioral model, decreased maladaptive beliefs about sleep and increased self-efficacy for the cognitive model, decreased

sleep effort for the AIE model, and reduced cognitive and physical arousal for the hyperarousal model (Schwartz & Carney, 2012). Eleven studies including secondary analyses on mediators were identified. In three randomized controlled trials (RCTs) investigating time in bed as a mediating factor, patients spent less time in bed after CBT-I than before treatment and bedtime reductions were greater in the CBT-I groups than the control groups. Eight RCTs included the DBAS, measuring maladaptive beliefs about sleep. The findings were consistent with the cognitive model: Greater reductions in DBAS scores were consistently found after CBT-I compared to the control conditions, and greater reductions in DBAS were associated with greater sleep improvement. No study examined sleep effort. One RCT studied subjectively reported physical and cognitive arousal as measured by the Pre-sleep Arousal Scale as a mediator, without any significant findings. No study assessed whether objective measures of arousal, such as pulse rate, heart rate, or muscle tension, were related to CBT-I outcomes. Taken together, the results of this review indicate that CBT-I is associated with changes in its proposed mediating variables. The evidence was strongest for time in bed as a behavioral factor and dysfunctional beliefs and attitudes as a cognitive variable, whereas arousal was less well studied and studies on the mediating role of sleep effort were lacking.

17.3 Therapeutic Packages

CBT-I has been classified as a well-established treatment for chronic insomnia by the American Academy of Sleep Medicine (Morgenthaler et al., 2006). The level of recommendation for CBT-I was classified as "standard" in this evidence-based review, meaning that its effectiveness has been demonstrated in "randomized well-designed trials with low alpha and beta error" (p. 1416). Whereas CBT-I and medication with benzodiazepines are classified as effective short-term insomnia treatments ($\leq$ 4 weeks), studies with follow-up comparisons (follow-up periods of 6 to 24 months) favor CBT (Mitchell, Gehrman, Perlis, & Umscheid, 2012).

CBT-I is a multicomponent therapeutic package. The studied packages include various combinations of cognitive and behavioral interventions such as cognitive restructuring, stimulus control, sleep restriction, relaxation training, and sleep hygiene education. Due to the considerable heterogeneity within the term "CBT-I" and a lack of clarity concerning the role of single treatment components within the whole package, this chapter will separately address CBT-I as a multicomponent treatment package as well as addressing the single interventions. Beyond CBT-I as a gold-standard treatment for insomnia, other interventions will be reviewed in this section.

17.3.1 Cognitive–Behavioral Therapy

Perlis, Jungquist, Smith, and Posner (2005) provide a session-by-session treatment manual for CBT-I. They describe eight individual sessions covering sleep hygiene education, stimulus control, sleep restriction, and cognitive therapy. The content of each intervention is briefly described in the following sections addressing single interventions as stand-alone treatments.

A number of RCTs have demonstrated the superiority of CBT-I over waitlist control groups and over sleep hygiene education alone. In this section, several recent studies

that serve as salient examples will be described in detail. Jansson and Linton (2005) randomized 165 patients with a mild level of chronification (duration of insomnia of 3 to 12 months) to six sessions of CBT-I in a group setting or a control group (self-help information package). Compared to the control group, at 1-year follow-up, CBT-I more effectively improved sleep parameters (as measured by sleep diaries), dysfunctional beliefs and attitudes about sleep, and daytime symptoms, such as performance impairment at work. These results suggest that CBT-I is applicable in an early stage of insomnia and can be an effective treatment to prevent its persistence.

Edinger et al. (2009) randomized 40 patients with primary insomnia and 41 patients suffering from insomnia associated predominantly with mixed psychiatric disorders to CBT-I or a sleep hygiene control group. CBT-I consisted of four biweekly individual outpatient sessions of sleep education, stimulus control, and bedtime restrictions, whereas the control group received the same "dose" of sleep hygiene only. CBT-I outperformed the control group in sleep diary measures of SOL and sleep efficiency as well as actigraphy measures of WASO. The effects were still present but smaller after 6 months. Most effect sizes were within the medium range. The results indicate that patients with primary and comorbid insomnia equally benefit from CBT-I. A randomized trial provided an indication that CBT-I is also superior to a waitlist when delivered in a "natural" setting in Sweden (Bothelius, Kyhle, Espie, & Broman, 2013). In this trial, 66 unselected primary care patients with chronic insomnia were randomized to five manual-guided CBT-I group sessions or a waitlist. The treatment was provided by primary health care nurses and a social worker who had received 2 days of training. In this trial, CBT-I was significantly superior (high effect size) to waitlist on the primary outcome, the Insomnia Severity Index. However, the treated group had deteriorated on all measures at 18-month follow-up.

A meta-analysis identified 14 RCTs of CBT-I for primary insomnia (Okajima, Komada, & Inoue, 2011). The comparison groups were placebo controls (6 studies), waitlists (5), treatment as usual (TAU) (1), and information about CBT-I techniques (2). For within-subject changes, mean effect sizes for subjective sleep parameters (sleep diaries) were medium to large and were stable at follow-up with the exception of time in bed. Between-group comparisons with subjective sleep parameters as an outcome clearly favored CBT-I with moderate to large effect sizes (Cohen's *d*) directly after treatment and small to moderate effects at follow-ups of 1–12 months. Another meta-analysis supported these results, demonstrating improvements in SOL, WASO, TST, and sleep efficiency that were stable at follow-up (Trauer, Qian, Doyle, Rajaratnam, & Cunnington, 2015). Importantly, CBT-I also yields medium to large effects in patients with insomnia and comorbid mental and psychiatric conditions (Wu, Appleman, Salazar, & Ong, 2015).

17.3.2 Brief Behavioral Treatment

In an RCT investigating dose–response effects in the treatment of insomnia, Edinger, Wohlgemuth, Radtke, Coffman, and Carney (2007) found that 58.3% of patients receiving four biweekly sessions improved significantly, compared with 43.8% receiving only one session, 22.2% receiving two sessions, and 35.3% receiving eight sessions. Brief behavioral treatment for insomnia (BBT-I) is an intervention with two face-to-face sessions (first and third week) and two additional sessions that can be delivered via

telephone (second and fourth week). The first session lasts 45 to 75 minutes and starts with information about healthy sleep and mechanisms of sleep regulation. Next, based on data from sleep diaries, average sleep parameters are calculated and the main rules to improve sleep are presented. This session concludes with a tailored "sleep prescription" that includes recommended bed and rising times, activities to engage in prior to bedtime or during the night if awake, and medication timing if needed. Potential side effects of the intervention are also presented. The second session lasts 20 minutes, reviews sleep behavior during the past week, and addresses problems related to treatment adherence. The third session (30 minutes) addresses the difficulties in implementing treatment recommendations, monitors and reinforces adherence to treatment, and provides information to titrate the individual sleep schedule. The fourth and last session (20 minutes) reviews rules for a better sleep (i.e., instructions for sleep restriction and stimulus control) and for adjusting time in bed.

Buysse et al. (2011) tested the efficacy of BBT-I in a sample of 79 older adults with chronic insomnia who were randomized to BBT-I or an information control group receiving printed information, both delivered by a nurse clinician. BBT-I produced significantly higher rates of responders (67% vs. 25%) and remitters (55% vs. 13%). The effects appeared comparable to those found for full-length CBT-I in older adults. Watanabe et al. (2011) used BBT-I (in this study, consisting of 1-hour individual sessions held once a week over 4 weeks) as a complementary or aftercare treatment for patients with depression who were adequately medically adjusted but still presented with refractory insomnia and mild to moderate depressive symptoms. They conducted an RCT on 37 outpatients who were randomly assigned to TAU or TAU plus BBT-I. BBT-I was significantly more efficacious than the control condition on the main outcome (insomnia severity) and secondary outcomes such as sleep efficiency and depression. Additional support for the efficacy of BBT-I comes from an RCT of a modified BBT-I delivered in a pharmacy setting by trained pharmacists (Fuller, Wong, Hoyos, Krass, & Saini, 2016). Wang, Wei, Wu, Zhong, and Li (2016) found moderate to large between-group effects in their RCT comparing BBT-I to sleep hygiene education in 79 patients with insomnia. Importantly, BBT-I is also more effective than TAU for the improvement of quality of life (Shimodera et al., 2014). Research on BBT-I thus shows that the efficacy of this low-threshold intervention is well supported.

17.3.3 Computerized Cognitive–Behavioral Therapy

With the increasing popularity and dissemination of the internet and internet-ready devices (e.g., computers and smartphones), CBT has increasingly been offered via electronic devices or through the internet. Computerized cognitive behavior therapy (CCBT; or CCBT-I for CCBT for insomnia) refers to a number of methods of delivering CBT via an interactive computer interface or a personal computer. The use of CCBT has some obvious advantages, mainly related to its cost-efficiency (Thiart et al., 2016) and its wide and easy access; still, there are disadvantages, such as those related to therapeutic alliance and confidentiality. The structured nature of CBT-I makes it ideal to be adapted as a computer or online program and to be delivered via the internet.

One of the first attempts to deliver CBT-I via the internet was conducted by researchers from Sweden (Ström, Pettersson, & Andersson, 2004). They conducted an RCT with 109 participants. The intervention consisted of a self-help manual that was

delivered in sections by email once a week over 5 weeks. Though the control group only received information about the study, researchers found that sleep improved both in the intervention and in the control group. The only significant difference was found in dysfunctional attitudes and beliefs about sleep, in which the control group showed no improvements. Of note, the use of sleep aids was permitted in both branches. The high dropout rate in the intervention arm (32 of 54 completed the study) also recommends caution in interpreting the results. Relatively similar results were reported by another RCT, conducted in Japan (Suzuki et al., 2008). The authors included 43 participants, of whom only 12 from the intervention arm were included in the final analysis. The intervention consisted of basic education on sleep and ways to improve sleep, daily personalized automatic messages aiming to improve adherence, a sleep diary, and weekly self-monitoring of the effects of sleep-related behavior changes. The duration of the intervention was 2 weeks. Although significant changes in sleep-related behaviors were found between pre- and postintervention, the quality of sleep did not improve significantly in the intervention group compared with the control group. Nevertheless, sleep quality continued to improve in the intervention group at the follow-up, 3 weeks later.

A more structured approach, called SHUTi (Sleep Healthy Using the Internet), was designed by researchers from the United States, who evaluated the efficacy of the program compared with a waitlist control in an RCT (Ritterband et al., 2009; Thorndike et al., 2008). The intervention was divided into six weekly "cores": one that introduced the intervention, two that promoted behavioral interventions (sleep restriction and stimulus control), one that focused on improving sleep hygiene, one that aimed to change unhelpful sleep beliefs and thoughts, and a last one for relapse prevention. Results revealed significant and clinically relevant improvements in the severity of insomnia, both posttreatment and at 6-month follow-up, compared to the waitlist control. Though the number of participants was rather small (45, with 18 completing the follow-up) and individuals with comorbid insomnia were excluded, this was the first study to provide clear support for using the internet as an efficacious method for delivering CBT-I. Several years later, the same method was used in a sample of patients suffering from insomnia comorbid with cancer, with promising results (Ritterband et al., 2012). Recently, Ritterband et al. (2017) published the outcomes of a larger study using the SHUTi program on a larger sample (303 participants) that included participants with comorbid insomnia (medical or psychiatric). Compared with those who only accessed a patient education website, those who used SHUTi reported significant improvements in their sleep (SOL, WASO, and the Insomnia Severity Index) both posttreatment and at the 1-year follow-up. Two of the three secondary sleep outcomes (sleep efficiency and sleep quality but not total sleep time) showed significant overall group and time interactions favoring SHUTi. The intervention has also been demonstrated to be effective in reducing depression severity at the population level (Christensen et al., 2016).

In Canada, Vincent and Lewycky (2009) conducted a two-armed RCT (CCBT-I vs. waitlist) called Return2Sleep in patients with primary insomnia. The intervention consisted of five weekly modules including psychoeducation, sleep hygiene, relaxation, sleep restriction, cognitive restructuring, and medication tapering. The results showed significant improvements in sleep quality, insomnia severity, and fatigue, as well as a reduction in maladaptive beliefs about sleep and pre-sleep mental activity. A later study, comparing the effectiveness of CCBT-I with a similar telehealth treatment intervention, revealed

that the interventions were equivalent in terms of sleep outcomes and use of sleep aids (Holmqvist, Vincent, and Walsh, 2014).

Using a non-internet-based approach, researchers from the United States tested whether a device originally designed for real-time monitoring and scheduled prompting of smoking behavior could help in delivering sleep restriction and stimulus control interventions in real time (Riley, Mihm, Behar, & Morin, 2010). The authors recruited 90 participants and randomly assigned them to the treatment or a self-help manual program. The results supported the feasibility of the real-time computerized delivery of behavioral interventions for insomnia (85% of the participants reported using the device on most or all nights), but the computer-supported intervention failed to provide significant benefits compared with the control group. The authors' explanation was that the control group received the guide and tape with information about sleep hygiene, relaxation, and cognitive restructuring as well. They also encountered software errors that led to failures in delivering the behavioral interventions and supposedly reduced efficacy. Overall, 80% of the participants completed both the program and the follow-up.

Another large trial was run in the Netherlands (Lancee, Van den Bout, Van Straten, & Spoormaker, 2012) with 623 participants randomly assigned to three arms: electronic self-help CBT, paper-and-pencil self-help CBT, and waitlist. The authors found that self-help CBT improved global insomnia symptoms and daily sleep measures 4 and 48 weeks after the intervention. This study suggested that some patients may not need face-to-face support because self-help is also efficacious, no matter whether it is provided in an electronic or a paper-and-pencil format. The main issue raised in this article was that almost half of the participants did not adhere to the intervention. In a recent study of equivalence ($N = 90$), contrary to the researchers' expectations, the same electronic intervention (slightly modified, as feedback was provided) was found to be inferior to the face-to-face treatment of insomnia (Lancee, Van Straten, Morina, Kaldo, & Kamphuis, 2016).

In 2012, Espie and colleagues compared a CCBT-I intervention to an online placebo intervention (imagery relief therapy). The study randomly assigned 164 participants to a web-based CCBT-I course delivered by an automated virtual therapist online, imagery relief therapy delivered the same way, or TAU. The first two arms consisted of six sessions over a minimum of 6 weeks. The results revealed significant improvements in the main outcome (sleep efficiency) in the CCBT-I arm compared to both controls at post-treatment and at follow-up. The increase in sleep efficiency in the CCBT-I arm was 20% at both time points.

More recently, two RCTs from Sweden have compared CCBT-I to active treatments. The largest of them ($N = 148$) tested whether an 8-week-long CBT intervention, delivered over the internet, was more effective than an active intervention that did not include sleep restriction and stimulus control and that aimed to increase the credibility of the CBT and to avoid nocebo effects (Kaldo et al., 2015). In the first arm, the intervention consisted of psychoeducation about sleep and sleep medication, sleep hygiene, sleep restriction, stimulus control, stress management, and how to manage fatigue and handle negative thoughts about sleep. Written feedback was provided by a trained clinical psychologist and reminders or encouragements were sent after 7 days of inactivity. In the second arm, no support was offered and the intervention consisted of psychoeducation. The researchers found that CCBT-I was superior immediately as well as 6 and

12 months afterwards, while the effect sizes were large and very large. Another important finding was that CCBT-I had a positive effect in reducing sleep medication use. The other Swedish study ($N = 48$) aimed to test whether CCBT-I was equivalent to group CBT-I (Blom, Tillgren et al., 2015). The internet-delivered intervention was similar to the one used by Kaldo and colleagues (2015). Both treatment groups improved significantly, with small to large effect sizes for the outcome measures. The authors concluded that there was no clinically meaningful difference between the two treatment modalities and that there were no statistically significant differences between the groups on any outcome measure at posttreatment or 6-month follow-up assessments. The same intervention, delivered over 9 weeks, was also shown to be more effective in alleviating insomnia symptoms than an internet-delivered CBT for depression in a study involving patients ($N = 43$) diagnosed with both insomnia disorder and major depressive disorder (Blom, Jernelöv et al., 2015). CCBT-I was also tested in adolescents ($N = 116$) in a study in the Netherlands with encouraging findings (de Bruin, Oort, Bögels, & Meijer, 2014). CCBT- I was delivered over the internet over six weekly sessions. Both group therapy and CCBT-I were found to be effective in treating insomnia in adolescents, both in the short and longer term, with medium to large effect sizes and with minimal differences between the two.

A recent systematic review and meta-analysis (Seyffert et al., 2016), which identified 15 trials that used CCBT-I delivered over the internet, concluded that CCBT-I is effective in improving sleep efficiency, insomnia severity, TST, SOL, WASO, and depressive symptoms and that it has a similar efficacy to face-to-face interventions.

Along with issues raised concerning internet-supported therapies in general, the studies presented here reveal an important challenge CCBT-I deals with: high attrition rates. As shown in Table 17.1, the rates vary from 2.5% to 35.9% and do not appear to be related to duration of intervention, insomnia type, receiving remuneration or support, or type of recruitment. Overall, CCBT-I seems to be a promising approach in the treatment of chronic insomnia that addresses the problem of dissemination of CBT-I into the supply system.

17.3.4 Sleep Hygiene Education

Sleep hygiene aims at the identification and correction of adverse health behaviors interfering with healthy sleep. Common sleep hygiene advice includes the following recommendations:

- Establish fixed bedtimes and getting-up times, including for weekends.
- Avoid napping.
- Eat a healthy diet, avoid heavy evening meals, and exercise on a regular basis.
- Do not consume stimulants (e.g., caffeine, black or green tea, or nicotine) after noon.
- Keep abstinent from drugs.
- Minimize sleep-disturbing factors (e.g., heat, cold, noise, and light) in the sleeping environment.
- Do not watch the clock at night.
- Establish a personal bedtime ritual.

In a randomized trial ($N = 155$ patients with insomnia), written sleep hygiene advice was compared to a self-help book (Bjorvatn, Fiske, & Pallesen, 2011).

Table 17.1 Published studies on computerized cognitive–behavioral therapy for insomnia.

Study	Location	Length of the Intervention	Sample Size (Number of Arms)	Recruitment Type	Insomnia Diagnosis	Insomnia Type	Payment Provided?	Support Provided?	Effect Sizes for SOL / WASO / TST	Attrition Rate (%)
Blom, Tillgren et al. (2015)	Sweden	8 weeks	48 (2)	Self-referred	Battery of scales and phone interview	Primary and comorbid	No	Yes	−0.88 / — / 0.88	6.25
De Bruin, Oort, Bögels, & Meijer (2014)	Netherlands	6 weeks	116 (3)	Self-referred	Battery of scales	Primary	No	Yes	−1.03 / −0.06 / 0.12	2.5
Espie et al. (2012)	United Kingdom	6 weeks	164 (3)	Self-referred	Battery of scales and face-to-face interview	Primary	No	Yes	−0.71 / −1.03 / 0.63	15.0
Ho, Chung, Yeung, Ng, & Cheng (2014)	Hong Kong	6 weeks	312 (3)	Self-referred	Battery of scales	Primary and comorbid	No	Yes	−0.40 / −0.27 / 0.05	35.9
Holmqvist, Vincent, & Walsh (2014)	Canada	6 weeks	73 (2)	Self- and physician-referred	Interview	Primary and comorbid	No	No	−0.68 / −0.59 / 0.48	15.3
Kaldo et al. (2015)	Sweden	8 weeks	626 (2)	Self-referred	Phone interview	Primary and comorbid	No	No	−0.35 / — / 0.03	11.4
Lancee, Van den Bout, Van Straten, & Spoormaker (2012)	Netherlands	6 weeks	623 (3)	Self-referred	Battery of scales	Primary	No	No	−0.62 / −0.82 / 0.50	22.2

(*continued*)

Table 17.1 (*Continued*)

Study	Location	Length of the Intervention	Sample Size (Number of Arms)	Recruitment Type	Insomnia Diagnosis	Insomnia Type	Payment Provided?	Support Provided?	Effect Sizes for SOL / WASO / TST	Attrition Rate (%)
Lancee, Van Straten, Morina, Kaldo, & Kamphuis (2016)	Netherlands	6 weeks	90 (3)	Self-referred	Battery of scales and phone interview	Primary	No	Yes	−0.4 / −0.7 / 0.3	13.3
Riley, Mihm, Behar, & Morin (2010)	USA	6 weeks	90 (2)	Self-referred	Interview	Primary	Yes, cash	No	−0.43 / −0.82 / 0.42	16.0
Ritterband et al. (2009)	USA	9 weeks	45 (2)	Self- and physician-referred	Interview	Primary	Yes, cash	No	−0.63 / −1.14 / 0.72	4.5
Ritterband et al. (2017)	USA	9 weeks	303 (2)	Self- and physician-referred	Interview	Primary and comorbid	Yes, gift certificate	No	−0.95 / −1.41 / 0.38	9.2
Ström, Pettersson, & Andersson (2004)	Sweden	5 weeks	109 (2)	Self-referred	Battery of scales	Primary	Yes, gift certificate	Yes	−0.44 / −0.34 / 0.45	33.0
Suzuki et al. (2008)	Japan	2 weeks	43 (2)	Self-referred	Battery of scales	Primary	No	Yes	−0.69 / — / 0.05	4.7
Thiart, Lehr, Ebert, Berking, & Riper (2015)	Germany	6 weeks	128 (2)	Self-referred	Battery of scales	Primary	No	No	— / — / —	4.6
Van Straten et al. (2014)	Netherlands	6 weeks	118 (2)	Self-referred	Battery of scales	Primary	No	Yes	−0.54 / — / 0.59	27.1
Vincent & Lewycky (2009)	Canada	5 weeks	118 (2)	Self- and physician-referred	Interview	Primary and comorbid	No	No	−0.39 / −0.34 / 0.52	32.2

SOL = sleep onset latency; TST = total sleep time; WASO = wake time after sleep onset.
Source: Updated from Voinescu, Szentagotai, and David (2013).

Pittsburgh Sleep Quality Index scores were significantly improved after sleep hygiene education. However, DBAS scores deteriorated after this intervention, the patients increased their medication intake, and sleep hygiene advice was outperformed by the self-help book concerning sleep improvement. In another randomized trial ($N = 79$) comparing the effectiveness of intensive sleep retraining (ISR), stimulus control, and the combination of ISR and stimulus control as a treatment for chronic sleep onset insomnia, sleep hygiene education was implemented as a control condition (Harris, Lack, Kemp, Wright, & Bootzin, 2012). The treatment was delivered in five weekly sessions of 30 minutes. Only minimal, predominantly nonsignificant improvements of sleep parameters (as measured by sleep diaries and actigraphy) were observed for sleep hygiene education. It was clearly outperformed by the three other treatments in this trial. This was not the case for a trial of four sessions of adjuvant sleep education (compared to pharmacotherapy [lorazepam] and a combination of sleep education and pharmacotherapy) delivered to 120 patients suffering from depression and treated with a selective serotonin reuptake inhibitor (Rahimi et al., 2016). All of the three methods improved sleep, with no advantages for any one method, while lorazepam alone was outperformed by sleep education alone in treating depression.

Current evidence indicates that this low-threshold intervention may be of benefit for some patients but is not sufficient as a stand-alone treatment for chronic insomnia (Morgenthaler et al., 2006). One potential reason for the relatively poor effects of sleep hygiene education is that patients with insomnia are not significantly more likely than healthy sleepers to have poor sleep hygiene, as demonstrated in a comparison study (Harvey, 2000).

17.3.5 Stimulus Control

The rationale of stimulus control therapy is the assumption that sleep disturbance is attributable to classical and/or operant conditioning between the sleeping environment and wakefulness, arousal, or anxiety, instead of deactivation and sleep. In order to weaken the association between the sleeping environment and wakefulness and at the same time reestablish a stable association with sleep, patients are instructed to go to bed only when they feel sleepy, leave the bed only if they are not able to sleep for 20 minutes or longer, and use the bed only for sleeping (and sexual activity).

In a pilot study ($N = 20$ patients with sleep onset insomnia), stimulus control therapy alone was compared to a combined intervention including stimulus control and relaxation training (Jacobs et al., 1993). The mean improvement of SOL, as assessed with a sleep diary, was 47 minutes in the stimulus control group, compared to 59 minutes for the combined intervention. The results of this small study indicate that stimulus control therapy alone is a highly effective treatment; however, the combined treatment tended to achieve a better effect in this study. The effect of stimulus control therapy alone was also found in another pilot study ($N = 16$ older adults with insomnia): SOL was strongly reduced, from around 70 minutes to around 30 minutes (Puder, Lacks, Bertelson, & Storandt, 1983). No improvement was observed during a waiting period in a group that received stimulus control treatment after a 10-week delay.

Another study of the same workgroup ($N = 65$) compared four 1-hour sessions of stimulus control to relaxation, paradoxical intention, and a placebo group. Stimulus control

treatment was superior to placebo and resulted in the greatest reductions in SOL (Lacks, Bertelson, Gans, & Kunkel, 1983).

Turner and Ascher (1979) conducted an RCT ($N = 50$) comparing stimulus control, progressive muscle relaxation, paradoxical intention, a placebo intervention (quasi-desensitization), and a waitlist control. All active treatments were significantly superior to the two control groups on five different sleep diary variables; however, the treatment effects in the three active groups were comparable, suggesting that stimulus control was not superior to relaxation and paradoxical intention treatment. Other small studies support the finding that stimulus control is effective for the treatment of insomnia, among them a small RCT ($N = 27$) that found that stimulus control and paradoxical intention were better than information on sleep and self-monitoring (Ladouceur & Gros-Louis, 1986) and two others that observed that stimulus control was superior to imagery training (Morin & Azrin, 1987, 1988). Engle-Friedman and Hazlewood (1992) found that, in a sample of 53 older adults suffering from primary insomnia, stimulus control performed best immediately posttreatment and at 2-year follow-up compared with support and sleep hygiene alone, progressive muscle relaxation, and a measurement control group. However, Pallesen et al. (2003) found that, in their sample of 55 adults above the age of 60 years with insomnia, stimulus control therapy was effective (effect sizes 0.49 for SOL, 0.36 for WASO, and 0.39 for TST) but not superior to a relaxation tape.

Epstein, Sidani, Bootzin, and Belyea (2012) conducted a dismantling study on behavior therapy for older adults with insomnia. They randomized 179 adults (mean age: 69 years) with primary insomnia to sleep restriction therapy alone, stimulus control therapy alone, the combination of both, or a waitlist control. The outcome measures were sleep diaries and actigraphy. The researchers found equal treatment effects without significant differences between the three active treatment arms, indicating that stimulus control is effective as a stand-alone treatment. Immediate posttreatment effect sizes (Cohen's *d*) for stimulus control were 1.44 for SOL, 1.04 for WASO, and 1.08 for TST as assessed by sleep diaries. Effects on actigraphy, in contrast, were small to medium in size.

As outlined in Section 17.3.6, the existing research on sleep restriction therapy largely relies on smaller studies with potentially biased results, whereas larger RCTs have mostly investigated CBT-I as a multicomponent treatment package. However, taken together, the data support stimulus control treatment over placebo.

17.3.6 Sleep Restriction

At first sight, restricting the bedtimes of patients with insomnia seems counterintuitive. This treatment approach is based on the behavioral model of insomnia, which assumes that increasing sleep pressure will consolidate sleep, because patients with insomnia tend to extend their bedtimes and nap during the day in an attempt to catch up on sleep. Patients are usually asked to fill in a sleep diary prior to the first session. In the first session, sleep diaries are reviewed and the recommended time in bed per night is set at approximately the mean total sleep time per night. For example, if a patient sleeps approximately 5 hours per night according to the sleep diary, the patient will agree on a regular bedtime and rising time together with the therapist that results in 5 or 5.5 hours in bed. If sleep efficiency improves with treatment, time in bed can be prolonged step by step.

Friedman, Bliwise, Yesavage, and Salom (1991) conducted a comparison of sleep restriction and relaxation in 22 elderly insomniacs. Subjects were alternately allocated to one of the two conditions and matched for age and insomnia severity. Sleep diaries were used as outcome measures. The authors found that, after the sleep restriction intervention, SOL was reduced at posttreatment and at 3-month follow-up. WASO was reduced at immediate posttreatment but increased at follow-up. TST remained relatively unchanged at posttreatment but significantly increased at follow-up. Sleep restriction was clearly superior to the relaxation intervention. Interestingly, the same workgroup conducted another comparison study in a similar sample of 39 older adults with insomnia, using actigraphy as an outcome measure. Contrary to the hypothesis, sleep restriction was not superior to a sleep hygiene control condition (Friedman et al., 2000). In the previously cited dismantling study by Epstein et al. (2012), the effect sizes (Cohen's *d*) for sleep restriction therapy were 1.1 for SOL, 1.2 for WASO, and 0.6 for TST.

A recent evidence-based review by Miller et al. (2014) investigated the efficacy of sleep restriction as a stand-alone treatment for adults with insomnia disorder. The authors found four studies of adequate methodological quality to examine the efficacy of sleep restriction treatment for chronic insomnia, among them three RCTs of which the previously cited trial by Epstein et al. is the largest and best designed study on the topic. The authors found moderate to large pre- to posttreatment effect sizes (within group) for sleep diary measures of SOL, WASO, and sleep efficiency, and small effects on TST. They conclude that sleep restriction is efficacious as a stand-alone treatment for chronic insomnia in adults.

Several other recent studies have investigated adverse side effects of bedtime restriction, which had previously been neglected in behavior therapy for insomnia (contrary to efficacy studies in the area of pharmacotherapy that routinely assess undesired side effects). Bedtime restriction and consequent loss of sleep beyond extant sleeping difficulties is associated with sleepiness, fatigue, depressed mood, headache, and reduced performance, especially in the first weeks of the treatment (Kyle, Morgan, Spiegelhalder, & Espie, 2011; Miller, Kyle, Marshall, & Espie, 2013). Importantly, a simplified version of sleep restriction can effectively be applied in common primary care (Falloon, Elley, Fernando, Lee, & Arroll, 2015).

17.3.7 Relaxation

The use of relaxation training in the treatment of insomnia is based on the assumption that patients with sleep problems suffer from heightened arousal, which inhibits the onset and maintenance of sleep. Interventions that focus on physical relaxation are most commonly used, especially progressive muscle relaxation, developed by Jacobson (1938).

In 2006, a task force of the American Academy of Sleep Medicine published a review on the recent evidence on psychological treatments for insomnia. The authors classified relaxation as one of five therapies meeting the criteria for empirically supported treatments for insomnia (Morin et al., 2006). Other therapies meeting these criteria were stimulus control, sleep restriction, paradoxical intention, and CBT-I. Well-designed randomized trials with adequate power demonstrating the superiority of relaxation therapy over placebo were conducted by Edinger, Wohlgemuth, Radtke, Marsh, and Quillian (2001; RCT on 70 patients with persistent primary insomnia) and Lichstein

et al. (2001; RCT on 72 patients with psychophysiological insomnia). Edinger et al. (2001) found that patients treated with relaxation achieved a reduction of their WASO of 16%, TST was increased by 25 minutes, and sleep efficiency increased from 72% at baseline to 79% after relaxation treatment. The authors concluded that relaxation is superior to a placebo intervention but was clearly outperformed by CBT-I. Lichstein et al. (2001) found that, after relaxation, WASO was reduced from 67 to 43 minutes at immediate posttreatment and 52 minutes at follow-up. The authors reported no significant improvements of daytime impairment. Similarly to Edinger et al. (2001), they found that relaxation was superior to placebo but less effective than a behavioral intervention (sleep restriction). Empirical evidence clearly shows that relaxation is more effective than a placebo intervention but produces considerably smaller effects than behavioral therapy.

17.3.8 Cognitive Therapy

Developed from the cognitive model by Harvey (2005; see Section 17.2.5), cognitive therapy for insomnia aims at restructuring unrealistic and unhelpful thoughts about sleep, sleeplessness, and the consequences of sleeplessness. Therapeutic strategies used in cognitive interventions are, for instance, the Socratic dialogue, behavioral experiments, and the replacement of unhelpful thoughts with more adaptive thoughts.

In a randomized trial, Pech and O'Kearney (2013) compared classical cognitive therapy with a problem-solving therapy focusing on the reduction of worry and the enhancement of self-efficacy in a sample of 47 adult insomniacs. Both interventions were delivered as adjuncts to sleep hygiene education, stimulus control, and relaxation. Sleep efficiency as measured by sleep diaries was significantly increased after treatment in both groups (Cohen's $d = 1.42$ for problem-solving and 1.26 for cognitive therapy). Improvements were maintained at follow-up 1 month after treatment. The authors found the two treatments to be equally efficacious. To date, the effects of problem-solving therapy on symptoms of insomnia are poorly evaluated. Thus, it remains unclear whether problem-solving as a comparison is to be classified as an efficacious treatment or rather a kind of placebo intervention. In addition, the design of the study does not allow for a real comparison between problem-solving and cognitive therapy, as the effects in both groups could be driven by the established behavioral interventions alone.

In a recent RCT, Harvey and colleagues (2014) aimed at systematically investigating the relative efficacy of cognitive therapy and behavior therapy as stand-alone treatments compared to multicomponent CBT-I. They included 188 adults with persistent insomnia in their study. They found that the effects of cognitive therapy (rates of responders and remissions) were slower than the effects in the other two groups, but the effects were comparable in size and were sustained at a 6-month follow-up. Of note, treatment was delivered in an individual setting in this trial, whereas other trials often favor group settings.

17.3.9 Paradoxical Intention

Paradoxical intention therapy aims to reduce sleep effort and thus relates to the AIE model by Espie et al. (2006; see Section 17.2.4). A paradoxical instruction to try to stay awake as long as possible is supposed to reduce the fear of sleeplessness and the pressure to fall asleep fast.

Researchers have compared paradoxical intention with established interventions in two small RCTs. In the first study, Turner and Ascher (1979) compared five treatment arms in a sample of 50 individuals with clinically relevant sleep disturbances: paradoxical intention, sleep restriction, relaxation, placebo, and no treatment. No significant differences between the two active treatment groups were observed (which may, however, be due to the relatively small sample size). All active treatments were superior to the control conditions. Espie, Lindsay, Brooks, Hood, and Turvey (1989) allocated 70 patients with chronic sleep onset insomnia to relaxation, stimulus control, paradoxical intention, placebo (imagery relief), or no treatment. Stimulus control and paradoxical intention were significantly superior to no treatment in reducing SOL. Paradoxical intention achieved a final outcome that was similar to stimulus control, but improvements occurred more slowly. Interestingly, in one-third of the patients in the paradoxical intention group, SOL deteriorated in the first week of treatment with an improvement starting later on. SOL was reduced from 72 to 36 minutes in this group.

Broomfield and Espie (2003) conducted a study aimed at investigating putative mechanisms underlying paradoxical intention. The authors randomly allocated 34 non-treatment-seeking patients with clinically relevant sleep-onset insomnia to paradoxical intention or a waitlist. Patients treated with paradoxical intention demonstrated reduced sleep effort, reduced sleep performance anxiety, and reduced SOL relative to controls. The treatment effect size was moderate (41.85% reduction of SOL).

17.3.10 Intensive Sleep Retraining

ISR is a newer behavioral intervention designed to generate high sleep pressure with the help of sleep restriction and subsequently realize a sequence of sleep onsets with short SOL within the following night (due to high sleep pressure). The theory behind the intervention is based on classical conditioning: It is assumed that patients have learned to show sleeplessness as a response to attempts to fall asleep. The second night of the ISR intervention, in which subjects are supposed to fall asleep fast several times in a row within one night, is intended to dissolve the conditioned insomnia response and reestablish sleep as a conditioned response. In the first night of ISR, sleep duration is typically restricted to 5 hours. In the second night, patients are awakened after 3 minutes of sleep if they are able to fall asleep within a trial period of 20 minutes. Patients receive detailed feedback regarding their sleep and wake behavior after the second night.

Harris et al. (2012) conducted an RCT ($N = 79$ patients with chronic sleep-onset insomnia) in which patients were randomized to ISR, a stimulus control, ISR plus stimulus control, or sleep hygiene education (control condition). All treatment conditions were superior to the control group, with few differences among the active treatments. An advantage of ISR appears to be a faster treatment response. The posttreatment effect sizes for ISR were 0.61 for SOL, 0.53 for TST, 0.26 for WASO, and 0.65 for sleep efficiency.

17.3.11 Mindfulness

Mindfulness meditation, a practice characterized by nonjudgmental, focused awareness in the present moment, has recently been integrated into the treatment of primary and comorbid insomnia. Mindfulness Based Stress Reduction (MBSR) is an 8-week program

designed to teach formal mindfulness practice and a mindful attitude in everyday life. In weekly sessions of 2 hours, patients are taught formal meditation exercises such as the bodyscan, yoga, and a sitting meditation. Mindfulness instructions consist of attentively observing the present moment, being open to all inner and outer experiences, and being nonjudgmental. Participants are often instructed to mindfully observe their breath or their body.

The efficacy of mindfulness-based interventions, in slightly different modes of application and patient samples, has been evaluated in several RCTs. MBSR, combined with sleep hygiene advice, is similarly effective to eszopiclone (3 mg) combined with sleep hygiene (Gross et al., 2011). A short mindfulness-based intervention as an add-on to four sessions of CBT-I is similarly effective as an add-on based on cognitive therapy (Wong, Ree, & Lee, 2016). However, a limitation is that, whereas these studies show that mindfulness-based therapy is not more effective than CBT-I or eszopiclone, they are not adequately powered to show noninferiority. Garland, Rouleau, Campbell, Samuels, and Carlson (2015) published a randomized noninferiority trial on MBSR compared with CBT-I for 111 patients with insomnia comorbid with cancer. Whereas CBT-I was superior immediately posttreatment, treatment effects between the two groups were equal at follow-up.

Ong and colleagues developed a treatment combining mindfulness meditation with classical behavior therapy for insomnia, including sleep restriction and stimulus control (mindfulness-based therapy for insomnia [MBTI]; Ong, Shapiro, & Manber, 2008). In an uncontrolled study of 30 patients diagnosed with psychophysiological insomnia, the authors found promising results concerning sleep improvements. In a subsequent RCT comparing three treatment groups, MBTI and MBSR both were more effective than self-monitoring (filling in sleep diaries), but MBTI was not more effective than MBSR.

In a meta-analysis summarizing the effects of mindfulness meditation for insomnia (including six RCTs), Gong et al. (2016) found that mindfulness is significantly more effective than control interventions for total wake time, SOL, sleep quality, and sleep efficiency, but not for total sleep time. They concluded that mindfulness is effective but that the effects are quite small. Thus, in summary, whereas it has been clearly demonstrated that mindfulness improves insomnia, it remains to be further investigated how effective mindfulness is compared with CBT-I. In addition, further research is needed to clarify the role of mindfulness in the treatment process—for example, as an alternative to CBT-I, as an add-on treatment combined with elements of CBT-I, or as a treatment for specific populations (e.g., patients who do not wish to participate in CBT-I).

17.3.12 Biofeedback

EMG biofeedback provides moment-to-moment feedback on levels of tension and relaxation via sensors placed on the patient's skin. This allows for online monitoring of the effects of a relaxation intervention. Patients learn how to adapt their behavior in order to induce a desired effect. EMG biofeedback is used in insomnia to reduce somatic tension and arousal. EMG biofeedback met the American Psychological Association's criteria for "probably efficacious treatments" in 2006 (Morin et al., 2006).

Sanavio, Vidotto, Bettinardi, Rolletto, and Zorzi (1990) randomized 40 adults to EMG biofeedback, cognitive treatment, stimulus control, relaxation, or a waitlist. They found improvements in SOL, WASO, and subjective sleep quality that were sustained at 1- and

3-year follow-ups in the active treatment groups. There were no clear differences among the active treatments. The finding of reduced SOL compared with a waitlist control after EMG biofeedback had previously been found in another small trial ($N = 36$; female undergraduates with insomnia; VanderPlate & Eno, 1983). However, EMG biofeedback was not superior to a placebo biofeedback group in this study.

Freedman and Papsdorf (1976) allocated 18 patients with insomnia to EMG biofeedback, progressive muscle relaxation, and a placebo (pseudo-relaxation). The reduction of SOL was 30 minutes in the biofeedback group, which was significantly greater than the reduction in the placebo control group. The two experimental groups did not significantly differ from each other. Haynes, Sides, and Lockwood (1977) similarly found a superiority of EMG biofeedback over a placebo relaxation intervention in the reduction of SOL in a study on 24 subjects. Hauri (1981), however, did not find any effect of EMG biofeedback compared with a no-treatment control group. He randomized 48 patients with insomnia to EMG biofeedback, a combination of EMG and theta feedback, sensorimotor rhythm feedback, and a no-treatment control. Sleep logs were used as a main outcome directly after treatment and 9 months later. None of the feedback groups improved significantly more than the controls. The lack of effect may, however, be attributable to the lack of power inherent in the design, which consisted of four groups and a small sample. Nicassio, Boylan, and McCabe (1982) randomized 40 adults with severe chronic insomnia to EMG biofeedback, progressive relaxation, or a placebo biofeedback control. They found a significant reduction of SOL after EMG biofeedback; however, the improvements were not significantly greater than in the two control interventions.

Taken together, the literature on EMG biofeedback primarily consists of relatively old studies with small sample sizes. Based on existing data, one can assume that, for some insomniacs, EMG biofeedback is more effective than no intervention or a placebo relaxation intervention. However, there is no evidence supporting the superiority of the treatment over other less elaborate forms of relaxation training, such as progressive muscle relaxation. In addition, the degree of sleep improvement was not related to the amount of reduction in muscle tension during EMG biofeedback treatment in several studies, which argues against the rationale of biofeedback (Freedman & Papsdorf, 1976; Haynes et al., 1977; Nicassio et al., 1982).

17.4 Implications for Research and Practice

The present chapter summarizes psychological treatments for insomnia in terms of both their efficacy and their treatment mechanisms. Table 17.2 includes a rating of the level of evidence according to the nine categories proposed by David and Montgomery (2011) and moreover provides an overview of the theories and treatment packages. Overall, the strongest empirical evidence exists for CBT-I, which is currently considered the gold-standard treatment for primary and comorbid insomnia. Numerous RCTs have demonstrated its relatively fast (immediate effects after approximately six therapy sessions) and long-lasting (sustained treatment effects at follow-up) effects on subjective sleep parameters. Newer conceptualizations of CBT-I address the issue of implementation and dissemination of efficacious treatments into the supply system. Lower-threshold interventions, such as computerized CBT-I and short-term interventions, have the potential to facilitate this process.

Table 17.2 Treatment approaches for insomnia with empirical evidence for theoretical background and therapeutic package.

	Theoretical Basis		Therapeutic Package		
Approach	**Classification**	**Important Studies**	**Classification**	**Important Studies**	**Category**
Cognitive–behavioral therapy (CBT-I)	Preliminary data	See single components	Well supported	Okajima et al., 2011; Trauer et al., 2015; Wu et al., 2015 (meta-analyses; comorbid psychiatric and medical conditions): CBT-I > control (mixed control groups, including waitlist, placebo, and SH)	II
Brief behavioral treatment (BBT-I)	Preliminary data	See single components	Well supported	Buysse et al., 2011: BBT-I > information control Watanabe et al., 2011: BBT-I + TAU > TAU Shimodera et al., 2014: BBT-I + TAU > TAU Wang et al., 2016: BBT-I > SH Fuller et al., 2016: BBT-I > TAU	II
Computerized cognitive behavior therapy (CCBT-I)	Preliminary data	See single components	Well supported	Blom, Tillgren et al., 2015: CCBT-I = group therapy Holmqvist et al., 2014: CCBT-I = telehealth Ritterband et al., 2017: CCBT-I > Patient education Seyffert et al., 2016 (meta-analysis and systematic review): similar efficacy to in-person interventions and printed materials	III

Sleep hygiene (SH)	Sleep-incompatible behavior in insomniacs; preliminary data against theory	Harvey, 2000: sleep hygiene not a significant predictor of sleep quality; sleep quality not poorer in insomniacs than healthy controls	Strong contradictory evidence for SH as a stand-alone treatment	Bjorvatn et al., 2011: self-help book > SH Harris et al., 2012: minimal effects, SH < SC and ISR	VIII
Stimulus control (SC)	Classical conditioning, conditioned hyperarousal; preliminary data with methodological flaws	Evans & Bond, 1969; Poser et al., 1965: classical conditioning of sleep *Limitation: case studies with methodological flaws* Zwart & Lisman, 1979: data contradict theory Davies et al., 1986: countercontrol treatment reduces sleep maintenance insomnia	Well supported	Lacks et al., 1983: SC > placebo Morin & Azrin, 1987, 1988: SC > imagery training Epstein et al., 2012: SC > waitlist; SC = SR	II
Sleep restriction (SR)	Behavioral model: extended bedtimes as perpetuating factor; preliminary data	Schwartz & Carney, 2012: time in bed is mediator of treatment effects in CBT-I Hood et al., 2011; Yang et al., 2013: safety behaviors do not predict insomnia	Well supported	Friedman et al., 1991: SR > REL Epstein et al., 2012: SR > WL Miller et al., 2014 (review): SR > SH (three RCTs)	II

(*continued*)

Table 17.2 (*Continued*)

Approach	Theoretical Basis Classification	Important Studies	Therapeutic Package Classification	Important Studies	Category
Relaxation (REL)	(Somatic) hyperarousal model; preliminary data	Lichstein et al., 2001: "adherence index" including measures of relaxation is associated with treatment effect; duration of relaxation practice is related to outcome	Well supported, but less effective than behavioral therapy	Edinger et al., 2001: REL > placebo; REL < CBT-I Lichstein et al., 2001: REL > placebo; REL < SR	II
Cognitive therapy (CT)	Cognitive theory: preliminary data predominantly supporting theory	Harvey, 2005; Tang & Harvey, 2004; Tang et al., 2007: worry and arousal associated with sleep difficulties and misperception of sleep Sunnhed & Jansson-Fröjmark, 2013: reductions of sleep-related worry are associated with improvements after CBT-I Espie et al., 2006 (review): attentional bias toward sleep-related cues in insomnia Mendelson et al., 1986; Mercer et al., 2002: misperception of sleep in insomnia	Preliminary	Harvey et al., 2014: longer latency of treatment effect in CT compared to behavioral therapy and CBT-I; comparable effect sizes	III

		Hood et al., 2011; Woodley & Smith, 2006; Yang et al., 2013: safety behaviors not associated with insomnia severity Schwartz & Carney, 2012: dysfunctional beliefs and attitudes are mediators of outcome of CBT-I Jansson & Linton, 2007: belief in long-term consequences of insomnia is associated with maintenance of insomnia			
Paradoxical intention (PI)	Sleep intention and effort inhibit sleep; preliminary data	Fogle & Dyal, 1983: giving up trying to sleep reduces SOL Ansfield et al., 1996: sleep intention disturbs sleep under high cognitive load Broomfield & Espie, 2003: PI reduces sleep effort and sleep-related performance anxiety Rasskazova et al., 2014: sleep effort impairs objective, but not subjective, measures of sleep	Well supported	Turner & Ascher, 1979: PI > placebo Espie et al., 1989: PI > placebo	II
Intensive sleep retraining (ISR)	Classical conditioning; preliminary data with methodological flaws; see SC		Preliminary	Harris et al., 2012: ISR > SH	IV

(*continued*)

Table 17.2 (*Continued*)

Approach	Theoretical Basis Classification	Important Studies	Therapeutic Package Classification	Important Studies	Category
Mindfulness (MBSR, MBCT)	Metacognitive model; preliminary data	McCracken et al., 2011: psychological inflexibility is associated with poor sleep	Preliminary	Gong et al., 2016 (meta-analysis, six RCTs): mindfulness > waitlist / attention control, but efficacy compared to established interventions unclear	IV
EMG biofeedback (BF)	Preliminary data against theory	Freedman & Papsdorf, 1976; Haynes et al., 1977; Nicassio et al., 1982: reduction in muscle tension with treatment is not related to sleep improvement	Mixed data: BF is probably superior to no treatment and placebo, but not superior to simple relaxation	Hauri, 1981: BF not superior to no treatment Nicassio et al., 1982: BF > no treatment; BF not superior to BF placebo VanderPlate & Eno 1983: BF > waitlist; BF not superior to BF placebo Sanavio et al., 1990: BF > waitlist Freedman & Papsdorf, 1976: BF > placebo Haynes et al. 1977: BF > placebo; BF not superior to relaxation	IV

EMG = electromyography; MBCT = mindfulness-based cognitive therapy; MBSR = mindfulness-based stress reduction; SOL = sleep onset latency; TAU = treatment as usual. Limitations of selected studies are presented to give reasons for the classification of the evidence as "preliminary." The table provides an overview of relevant studies on the empirical evidence for the theoretical basis and the efficacy of therapeutic packages for the most common treatment approaches for insomnia. No complete representation of the available evidence is provided. Please see the text for more details.

As CBT-I is a multicomponent treatment typically combining several distinct interventions (such as sleep restriction, stimulus control, relaxation, and cognitive therapy), the question emerges whether all these components or their combination and potential interaction effects provide a relevant contribution to CBT-I's efficacy. This question is linked to the theoretical basis of the treatment and its mechanisms of action. As CBT-I consists of many elements (which are also sometimes used as single interventions), there is no single theory behind this approach. The present chapter attempted to address this issue by investigating theoretical backgrounds and efficacy data for all common components of CBT-I as single interventions. However, it must be said that this approach does not account for the possibility that the effects of single interventions do not simply add up in a combined treatment but instead have interaction effects and complement each other. If a single intervention is not effective when used alone, this does not necessarily mean that it is not an active component of a treatment package. However, to our knowledge, no theory on the interaction of the components of CBT-I has been published to date.

Among the single interventions, behavioral therapy (sleep restriction and stimulus control; both Category II) was identified as the insomnia treatment supported by the strongest base of empirical evidence. Mediational analyses in three complex clinical trials demonstrated that reductions of time in bed were associated with symptom improvements after sleep restriction treatment, supporting the rationale of behavior therapy. It is assumed that a shortened time in bed increases sleep pressure and that in this way sleep latencies reduce, sleep becomes more consolidated, and sleep is experienced as more restful. Other studies, however, found that sleep-related safety behaviors were also relatively common in healthy sleepers. The frequency of safety behaviors did not predict the severity of insomnia in two studies, suggesting that the interactions between cognitions, behavior, and symptoms of insomnia may be more complex.

The efficacy of sleep restriction for the improvement of subjective sleep parameters is well established. Recent research, however, has shed light on adverse effects of sleep restriction therapy, such as reduced performance level, sleepiness, and fatigue in the first weeks of the treatment. Due to these undesired effects, sleep restriction treatment is perceived as aversive by many patients. In clinical practice, patients should be informed of potential positive and negative effects of the various treatment options, should be warned that their ability to drive a vehicle safely and to operate machinery may be impaired during sleep restriction treatment, and should be educated about the various treatment options. Stimulus control is another behavioral intervention that shows effects comparable to sleep restriction on subjective sleep and is equally well supported in RCTs. However, it is questionable whether stimulus control actually works through its proposed mechanisms of action. An alternative explanation is that patients who follow stimulus control instructions reduce their time in bed; thus, hypothetically, stimulus control therapy might actually work very similarly to sleep restriction.

The efficacy of progressive muscle relaxation over placebo is also well established, so it can be an alternative for patients who do not wish to engage in behavioral treatment. However, relaxation has often been outperformed by behavioral treatments in clinical trials. It is therefore a valuable treatment that can be effective for some patients, but its efficacy falls short of behavioral treatments.

A strong potential for new developments in the field lies, for example, in the more detailed evaluation of cognitive therapy, which has only been investigated as a

stand-alone treatment in one RCT so far. Furthermore, mindfulness- and acceptance-based therapies are increasingly recognized as potential complementary or alternative interventions in the treatment of insomnia. They have the potential to broaden the relatively narrow therapeutic view on sleep regulation and move new aspects into focus, such as daytime performance, dealing with difficult thoughts and emotions, and quality of life. Of note, none of the reviewed theories can be classified as well supported, highlighting the need for additional well-designed research into the putative mechanisms of action of insomnia treatment.

17.5 Conclusions

Following the current state of empirical evidence, behavioral therapy is the first-line treatment for primary and comorbid insomnia. Computerized CBT-I and brief behavioral treatment can be helpful to overcome barriers of treatment implementation in clinical practice. Behavioral therapy is superior to pharmacological treatment with benzodiazepines and benzodiazepine receptor agonists regarding efficacy at follow-up. Whereas insomnia often reoccurs after the discontinuation of medication, the effects of behavior therapy are stable for up to 2 years after treatment. Paradoxical intention and relaxation are additional well-supported treatments for insomnia. Other psychological treatments, such as cognitive therapy, mindfulness- and acceptance-based interventions, ISR, and biofeedback, can also be considered; however, more research into their theoretical background and efficacy is needed. In clinical practice, patients should be included in the process of making decisions for or against a specific form of therapy. Potential positive and negative effects of pharmacotherapy and psychotherapy should be discussed.

References

American Academy of Sleep Medicine. (2014). *International classification of sleep disorders* (3rd ed.). Darien, IL: American Academy of Sleep Medicine.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.

Ansfield, M. E., Wegner, D. M., & Bowser, R. (1996). Ironic effects of sleep urgency. *Behaviour Research and Therapy*, *34*(7), 523–531. doi:10.1016/0005-7967(96)00031-9

Baglioni, C., Battagliese, G., Feige, B., Spiegelhalder, K., Nissen, C., Voderholzer, U., & Riemann, D. (2011). Insomnia as a predictor of depression: A meta-analytic evaluation of longitudinal epidemiological studies. *Journal of Affective Disorders*, *135*(1/3), 10–19. doi:10.1016/j.jad.2011.01.011

Bjorvatn, B., Fiske, E., & Pallesen, S. (2011). A self-help book is better than sleep hygiene advice for insomnia: A randomized controlled comparative study. *Scandinavian Journal of Psychology*, *52*(6), 580–585. doi:10.1111/j.1467-9450.2011.00902.x

Blom, K., Jernelöv, S., Kraepelien, M., Bergdahl, M. O., Jungmarker, K., Ankartjärn, L., … Kaldo, V. (2015). Internet treatment addressing either insomnia or depression, for patients with both diagnoses: A randomized trial. *Sleep*, *38*(2), 267–277. doi:10.5665/sleep.4412

Blom, K., Tillgren, H. T., Wiklund, T., Danlycke, E., Forssen, M., Söderström, A., … Andersson, G. (2015). Internet- vs. group-delivered cognitive behavior therapy for insomnia: A randomized controlled non-inferiority trial. *Behaviour Research and Therapy*, *70*, 47–55. doi:10.1016/j.brat.2015.05.002

Bootzin, R., & Perlis, M. (1992). Nonpharmacological treatments of insomnia. *Journal of Clinical Psychiatry*, *53*, 37–41.

Bothelius, K., Kyhle, K., Espie, C. A., & Broman, J.-E. (2013). Manual-guided cognitive–behavioural therapy for insomnia delivered by ordinary primary care personnel in general medical practice: A randomized controlled effectiveness trial. *Journal of Sleep Research*, *22*(6), 688–696. doi:10.1111/jsr.12067

Broomfield, N. M., & Espie, C. A. (2003). Initial insomnia and paradoxical intention: An experimental investigation of putative mechanisms using subjective and actigraphic measurement of sleep. *Behavioural and Cognitive Psychotherapy*, *31*(3), 313–324. doi:10.1017/S1352465803003060

Brower, K. J., Krentzman, A., & Robinson, E. A. R. (2011). Persistent insomnia, abstinence, and moderate drinking in alcohol-dependent individuals. *American Journal on Addictions*, *20*(5), 435–440. doi:10.1111/j.1521-0391.2011.00152.x

Budhiraja, R., Roth, T., Hudgel, D. W., Budhiraja, P., & Drake, C. L. (2011). Prevalence and polysomnographic correlates of insomnia comorbid with medical disorders. *Sleep*, *34*(7), 859–867. doi:10.5665/sleep.1114

Buysse, D. J., Germain, A., Moul, D. E., Franzen, P. L., Brar, L. K., Fletcher, M. E., & Reynolds, C. F. (2011). Efficacy of brief behavioral treatment for chronic insomnia in older adults. *Archives of Internal Medicine*, *171*(10), 887–895. doi:10.1001/archinternmed.2010.535

Buysse, D. J., Reynolds, C. F., Monk, T. H., & Berman, S. R. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research*, *28*(2), 193–213. doi:10.1016/0165-1781(89)90047-4

Carney, C. E., Buysse, D. J., Ancoli-Israel, S., Edinger, J. D., Krystal, A. D., Lichstein, K. L., & Morin, C. M. (2012). The Consensus Sleep Diary: Standardizing prospective sleep self-monitoring. *Sleep*, *35*(2), 287–302. doi:10.5665/sleep.1642

Chien, K.-L., Che, P.-C., Hsu, H.-C., Su, T.-C., Sung, F.-C., Chen, M.-F., & Lee, Y.-T. (2010). Habitual sleep duration and insomnia and the risk of cardiovascular events and all-cause death: Report from a community-based cohort. *Sleep*, *33*(2), 177–184. doi:10.1093/sleep/33.2.177

Christensen, H., Batterham, P. J., Gosling, J. A., Ritterband, L. M., Griffiths, K. M., Thorndike, F. P., … Mackinnon, A. J. (2016). Effectiveness of an online insomnia program (SHUTi) for prevention of depressive episodes (the GoodNight Study): A randomised controlled trial. *Lancet Psychiatry*, *3*(4), 333–341. doi:10.1016/s2215-0366(15)00536-2

David, D., & Montgomery, G. (2011). The scientific status of psychotherapies: A new evaluative framework for evidence-based psychosocial interventions. *Clinical Psychology: Science and Practice*, *18*, 89–99. doi:10.1111/j.1468-2850.2011.01239.x

Davies, R., Lacks, P., Storandt, M., & Bertelson, A. D. (1986). Countercontrol treatment of sleep-maintenance insomnia in relation to age. *Psychology and Aging*, *1*(3), 233–238. doi:10.1037/0882-7974.1.3.233

de Bruin, E. J., Oort, F. J., Bögels, S. M., & Meijer, A. M. (2014). Efficacy of internet and group-administered cognitive behavioral therapy for insomnia in adolescents: A pilot study. *Behavioral Sleep Medicine*, *12*(3), 235–254. doi:10.1080/15402002.2013.784703

Drake, C. L., Friedman, N. P., Wright, K. P., & Roth, T. (2011). Sleep reactivity and insomnia: Genetic and environmental influences. *Sleep*, *34*(9), 1179–1188. doi:10.5665/sleep.1234

Edinger, J. D., Olsen, M. K., Stechuchak, K. M., Means, M. K., Lineberger, M. D., Kirby, A., & Carney, C. E. (2009). Cognitive behavioral therapy for patients with primary insomnia or insomnia associated predominantly with mixed psychiatric disorders: A randomized clinical trial. *Sleep*, *32*(4), 499–510. doi:10.1093/sleep/32.4.499

Edinger, J. D., Wohlgemuth, W. K., Radtke, R. A., Coffman, C. J., & Carney, C. E. (2007). Dose-response effects of cognitive-behavioral insomnia therapy: A randomized clinical trial. *Sleep*, *30*(2), 203–212. doi:10.1093/sleep/30.2.203

Edinger, J. D., Wohlgemuth, W. K., Radtke, R. A., Marsh, G. R., & Quillian, R. E. (2001). Cognitive behavioral therapy for treatment of chronic primary insomnia: A randomized controlled trial. *JAMA*, *285*(14), 1856–1864.

Engle-Friedman, M., & Hazlewood, L. (1992). An evaluation of behavioral treatments for insomnia in the older adult. *Journal of Clinical Psychology*, *48*(1), 77–90. doi:10.1002/1097-4679(199201)48:1<77::aid-jclp2270480112>3.0.co;2-j

Epstein, D. R., Sidani, S., Bootzin, R. R., & Belyea, M. J. (2012). Dismantling multicomponent behavioral treatment for insomnia in older adults: A randomized controlled trial. *Sleep*, *35*(6), 797–805. doi:10.5665/sleep.1878

Espie, C. A., Broomfield, N. M., MacMahon, K., Macphee, L. M., & Taylor, L. M. (2006). The attention–intention–effort pathway in the development of psychophysiologic insomnia: A theoretical review. *Sleep Medicine Reviews*, *10*(4), 215–245. doi:10.1016/j.smrv.2006.03.002

Espie, C. A., Kyle, S. D., Williams, C., Ong, J. C., Douglas, N. J., Hames, P., & Brown, J. S. L. (2012). A randomized, placebo-controlled trial of online cognitive behavioral therapy for chronic insomnia disorder delivered via an automated media-rich web application. *Sleep*, *35*(6), 769–781. doi:10.5665/sleep.1872

Espie, C. A., Lindsay, W. R., Brooks, D. N., Hood, E. M., & Turvey, T. (1989). A controlled comparative investigation of psychological treatments for chronic sleep-onset insomnia. *Behaviour Research and Therapy*, *27*(1), 79–88. doi:10.1016/0005-7967(89)90123-x

Evans, D. R., & Bond, I. K. (1969). Reciprocal inhibition therapy and classical conditioning in the treatment of insomnia. *Behaviour Research and Therapy*, *7*(3), 323–325. doi:10.1016/0005-7967(69)90015-1

Falloon, K., Elley, C. R., Fernando, A., Lee, A. C., & Arroll, B. (2015). Simplified sleep restriction for insomnia in general practice: A randomised controlled trial. *British Journal of General Practice*, *65*, e508–e515. doi:10.3399/bjgp15x686137

Fogle, D. O., & Dyal, J. A. (1983). Paradoxical giving up and the reduction of sleep performance anxiety in chronic insomniacs. *Psychotherapy: Theory, Research & Practice*, *20*(1), 21–30. doi:10.1037/h0088473

Fortier-Brochu, É., Beaulieu-Bonneau, S., Ivers, H., & Morin, C. M. (2012). Insomnia and daytime cognitive performance: A meta-analysis. *Sleep Medicine Reviews*, *16*(1), 83–94. doi:10.1016/j.smrv.2011.03.008

Freedman, R. R., & Papsdorf, J. D. (1976). Biofeedback and progressive relaxation treatment of sleep-onset insomnia. *Biofeedback and Self-Regulation*, *1*(3), 253–271. doi:10.1007/BF01001167

Friedman, L., Benson, K., Noda, A., Zarcone, V., Wicks, D. A., O'Connell, K., & Yesavage, J. A. (2000). An actigraphic comparison of sleep restriction and sleep hygiene treatments

for insomnia in older adults. *Journal of Geriatric Psychiatry and Neurology*, *13*(1), 17–27. doi:10.1177/089198870001300103

Friedman, L., Bliwise, D. L., Yesavage, J. A., & Salom, S. R. (1991). A preliminary study comparing sleep restriction and relaxation treatments for insomnia in older adults. *Journal of Gerontology*, *46*(1), P1–P8. doi:10.1093/geronj/46.1.p1

Fuller, J. M., Wong, K. K., Hoyos, C., Krass, I., & Saini, B. (2016). Dispensing good sleep health behaviours not pills: A cluster-randomized controlled trial to test the feasibility and efficacy of pharmacist-provided brief behavioural treatment for insomnia. *Journal of Sleep Research*, *25*(1), 104–115. doi:10.1111/jsr.12328

Garland, S. N., Rouleau, C. R., Campbell, T., Samuels, C., & Carlson, L. E. (2015). The comparative impact of Mindfulness-Based Cancer Recovery (MBCR) and Cognitive Behavior Therapy for Insomnia (CBT-I) on sleep and mindfulness in cancer patients. *Explore*, *11*, 445–454. doi:10.1016/j.explore.2015.08.004

Gong, H., Ni, C. X., Liu, Y. Z., Zhang, Y., Su, W. J., Lian, Y. J., Peng, W., & Jiang, C. L. (2016). Mindfulness meditation for insomnia: A meta-analysis of randomized controlled trials. *Journal of Psychosomatic Research*, *89*, 1–6. doi:10.1016/j.jpsychores.2016.07.016

Gross, C. R., Kreitzer, M. J., Reilly-Spong, M., Wall, M., Winbush, N. Y., Patterson, R., & Cramer-Bornemann, M. (2011). Mindfulness-based stress reduction versus pharmacotherapy for chronic primary insomnia: A randomized controlled clinical trial. *Explore: Journal of Science and Healing*, *7*(2), 76–87. doi:10.1016/j.explore.2010.12.003

Haario, P., Rahkonen, O., Laaksonen, M., Lahelma, E., & Lallukka, T. (2013). Bidirectional associations between insomnia symptoms and unhealthy behaviours. *Journal of Sleep Research*, *22*(1), 89–95. doi:10.1111/j.1365-2869.2012.01043.x

Hamblin, J. E. (2007). Insomnia: An ignored health problem. *Primary Care*, *34*(3), 659–674, viii. doi:10.1016/j.pop.2007.05.009

Harris, J., Lack, L., Kemp, K., Wright, H., & Bootzin, R. (2012). A randomized controlled trial of intensive sleep retraining (ISR): A brief conditioning treatment for chronic insomnia. *Sleep*, *35*(1), 49–60. doi:10.5665/sleep.1584

Harvey, A. G. (2000). Sleep hygiene and sleep-onset insomnia. *Journal of Nervous and Mental Disease*, *188*(1), 53–55. doi:10.1097/00005053-200001000-00011

Harvey, A. G. (2001). I can't sleep, my mind is racing! An investigation of strategies of thought control in insomnia. *Behavioural and Cognitive Psychotherapy*, *29*(1), 3–11. doi:10.1017/S1352465801001023

Harvey, A. G. (2003). The attempted suppression of presleep cognitive activity in insomnia. *Cognitive Therapy and Research*, *27*(6), 593–602. doi:10.1023/A:1026322310019

Harvey, A. G. (2005). A cognitive theory and therapy for chronic insomnia. *Journal of Cognitive Psychotherapy*, *19*(1), 41–59. doi:10.1891/jcop.19.1.41.66332

Harvey, A. G., Bélanger, L., Talbot, L., Eidelman, P., Beaulieu-Bonneau, S., Fortier-Brochu, E., … Morin, C. M. (2014). Comparative efficacy of behavior therapy, cognitive therapy, and cognitive behavior therapy for chronic insomnia: A randomized controlled trial. *Journal of Consulting and Clinical Psychology*, *82*(4), 670–683. doi:10.1037/a0036606

Hauri, P. (1981). Treating psychophysiologic insomnia with biofeedback. *Archives of General Psychiatry*, *38*(7), 752–758. doi:10.1001/archpsyc.1981.01780320032002

Haynes, S. N., Sides, H., & Lockwood, G. (1977). Relaxation instructions and frontalis electromyographic feedback intervention with sleep-onset insomnia. *Behavior Therapy*, *8*(4), 644–652. doi:10.1016/S0005-7894(77)80194-9

Ho, F. Y. Y., Chung, K. F., Yeung, W. F., Ng, T. H. Y., & Cheng, S. K. W. (2014). Weekly brief phone support in self-help cognitive behavioral therapy for insomnia disorder: Relevance to adherence and efficacy. *Behaviour Research and Therapy*, *63*, 147–156. doi:10.1016/j.brat.2014.10.002

Holmqvist, M., Vincent, N., & Walsh, K. (2014). Web- vs telehealth-based delivery of cognitive behavioral therapy for insomnia: A randomized controlled trial. *Sleep Medicine*, *15*(2), 187–195. doi:10.1016/j.sleep.2013.10.013

Hood, H. K., Carney, C. E., & Harris, A. L. (2011). Rethinking safety behaviors in insomnia: Examining the perceived utility of sleep-related safety behaviors. *Behavior Therapy*, *42*(4), 644–654. doi:10.1016/j.beth.2011.02.004

Hublin, C., Partinen, M., Koskenvuo, M., & Kaprio, J. (2011). Heritability and mortality risk of insomnia-related symptoms: A genetic epidemiologic study in a population-based twin cohort. *Sleep*, *34*(7), 957–964. doi:10.5665/sleep.1136

Jacobs, G. D., Rosenberg, P. A., Friedman, R., Matheson, J., Peavy, G. M., Domar, A. D., & Benson, H. (1993). Multifactor behavioral treatment of chronic sleep-onset insomnia using stimulus control and the relaxation response: A preliminary study. *Behavior Modification*, *17*(4), 498–509. doi:10.1177/01454455930174005

Jacobson, E. (1938). *Progressive relaxation* (2nd ed.). Chicago, IL: University of Chicago Press.

Jansson, M., & Linton, S. J. (2005). Cognitive-behavioral group therapy as an early intervention for insomnia: A randomized controlled trial. *Journal of Occupational Rehabilitation*, *15*(2), 177–190. doi:10.1007/s10926-005-1217-9

Jansson, M., & Linton, S. J. (2006). The role of anxiety and depression in the development of insomnia: Cross-sectional and prospective analyses. *Psychology & Health*, *21*(3), 383–397. doi:10.1080/14768320500129015

Jansson, M., & Linton, S. J. (2007). Psychological mechanisms in the maintenance of insomnia: Arousal, distress, and sleep-related beliefs. *Behaviour Research and Therapy*, *45*(3), 511–521. doi:10.1016/j.brat.2006.04.003

Jansson-Fröjmark, M., Lundqvist, D., Lundqvist, N., & Linton, S. J. (2007). Psychosocial work stressors for insomnia: A prospective study on 50–60-year-old adults in the working population. *International Journal of Behavioral Medicine*, *14*(4), 222–228. doi:10.1007/BF03002996

Kaldo, V., Jernelöv, S., Blom, K., Ljótsson, B., Brodin, M., Jörgensen, M., … Lindefors, N. (2015). Guided internet cognitive behavioral therapy for insomnia compared to a control treatment: A randomized trial. *Behaviour Research and Therapy*, *71*, 90–100. doi:10.1016/j.brat.2015.06.001

Kessler, R. C., Berglund, P. A., Coulouvrat, C., Hajak, G., Roth, T., Shahly, V., & Walsh, J. K. (2011). Insomnia and the performance of US workers: Results from the America Insomnia Survey. *Sleep*, *34*(9), 1161–1171. doi:10.5665/sleep.1230

Kyle, S. D., Espie, C. A., & Morgan, K. (2010). "Not just a minor thing, it is something major, which stops you from functioning daily": Quality of life and daytime functioning in insomnia. *Behavioral Sleep Medicine*, *8*(3), 123–140. doi:10.1080/15402002.2010.487450

Kyle, S. D., Morgan, K., & Espie, C. A. (2010). Insomnia and health-related quality of life. *Sleep Medicine Reviews*, *14*(1), 69–82. doi:10.1016/j.smrv.2009.07.004

Kyle, S. D., Morgan, K., Spiegelhalder, K., & Espie, C. A. (2011). No pain, no gain: An exploratory within-subjects mixed-methods evaluation of the patient experience of sleep

restriction therapy (SRT) for insomnia. *Sleep Medicine*, *12*(8), 735–747. doi:10.1016/j.sleep.2011.03.016

Lacks, P., Bertelson, A. D., Gans, L., & Kunkel, J. (1983). The effectiveness of three behavioral treatments for different degrees of sleep onset insomnia. *Behavior Therapy*, *14*(5), 593–605. doi:10.1016/s0005-7894(83)80052-5

Ladouceur, R., & Gros-Louis, Y. (1986). Paradoxical intention vs stimulus control in the treatment of severe insomia. *Journal of Behavior Therapy and Experimental Psychiatry*, *17*(4), 267–269. doi:10.1016/0005-7916(86)90062-5

Lancee, J., Van den Bout, J., Van Straten, A., & Spoormaker, V. I. (2012). Internet-delivered or mailed self-help treatment for insomnia? A randomized waiting-list controlled trial. *Behaviour Research and Therapy*, *50*(1), 22–29. doi:10.1016/j.brat.2011.09.012

Lancee, J., Van Straten, A., Morina, N., Kaldo, V., & Kamphuis, J. H. (2016). Guided online or face-to-face cognitive behavioral treatment for insomnia: A randomized wait-list controlled trial. *Sleep*, *39*(1), 183–191. doi:10.5665/sleep.5344

Lichstein, K., Riedel, B. W., Wilson, N. M., Lester, K. W., & Aguillard, R. N. (2001). Relaxation and sleep compression for late-life insomnia: A placebo-controlled trial. *Journal of Consulting and Clinical Psychology*, *69*(2), 227–239. doi:10.1037//0022-006x.69.2.227

Lichstein, K. L., Taylor, D. J., McCrae, C. S., & Ruiter, M. E. (2011). Insomnia: Epidemiology and risk factors. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practices of sleep medicine* (5th ed., pp. 827–837). St. Louis, MO: Elsevier.

Lichstein, K. L., Wilson, N. M., & Johnson, C. T. (2000). Psychological treatment of secondary insomnia. *Psychology and Aging*, *15*(2), 232–240. doi:10.1037//0882-7974.15.2.232

McCracken, L. M., Williams, J. L., & Tang, N. K. Y. (2011). Psychological flexibility may reduce insomnia in persons with chronic pain: A preliminary retrospective study. *Pain Medicine*, *12*(6), 904–912. doi:10.1111/j.1526-4637.2011.01115.x

Mendelson, W. B., James, S. P., Garnett, D., & Sack, D. A. (1986). A psychophysiological study of insomnia. *Psychiatry Research*, *19*(4), 267–284. doi:10.1016/0165-1781(86)90120-4

Mercer, J. D., Bootzin, R. R., & Lack, L. C. (2002). Insomniacs' perception of wake instead of sleep. *Sleep*, *25*(5), 559–566. doi:10.1093/sleep/25.5.559

Miller, C. B., Espie, C. A., Epstein, D. R., Friedman, L., Morin, C. M., Pigeon, W. R., … Kyle, S. D. (2014). The evidence base of sleep restriction therapy for treating insomnia disorder. *Sleep Medicine Reviews*, *18*(5), 415–424. doi:10.1016/j.smrv.2014.01.006

Miller, C. B., Kyle, S. D., Marshall, N. S., & Espie, C. A. (2013). Ecological momentary assessment of daytime symptoms during sleep restriction therapy for insomnia. *Journal of Sleep Research*, *22*(3), 266–272. doi:10.1111/jsr.12024

Mitchell, M. D., Gehrman, P., Perlis, M., & Umscheid, C. A. (2012). Comparative effectiveness of cognitive behavioral therapy for insomnia: A systematic review. *BMC Family Practice*, *13*, 40. doi:10.1186/1471-2296-13-40

Moreno-Coutiño, A., Calderón-Ezquerro, C., & Drucker-Colín, R. (2007). Long-term changes in sleep and depressive symptoms of smokers in abstinence. *Nicotine & Tobacco Research*, *9*(3), 389–396. doi:10.1080/14622200701188901

Morgenthaler, T., Kramer, M., Alessi, C., Friedman, L., Boehlecke, B., Brown, T., & Swick, T. (2006). Practice parameters for the psychological and behavioral treatment of

insomnia: An update—An American Academy of Sleep Medicine report. *Sleep*, *29*(11), 1415–1419. doi:10.1093/sleep/29.11.1415

Morin, C. M. (1993). *Insomnia: Psychological assessment and management*. New York, NY: Guilford Press.

Morin, C. M., & Azrin, N. H. (1987). Stimulus control and imagery training in treating sleep-maintenance insomnia. *Journal of Consulting and Clinical Psychology*, *55*(2), 260–262. doi:10.1037//0022-006x.55.2.260

Morin, C. M., & Azrin, N. H. (1988). Behavioral and cognitive treatments of geriatric insomnia. *Journal of Consulting and Clinical Psychology*, *56*(5), 748–753. doi:10.1037/0022-006x.56.5.748

Morin, C. M., Bélanger, L., LeBlanc, M., Ivers, H., Savard, J., Espie, C. A., & Grégoire, J.-P. (2009). The natural history of insomnia: A population-based 3-year longitudinal study. *Archives of Internal Medicine*, *169*(5), 447–453. doi:10.1001/archinternmed.2008.610

Morin, C. M., Bootzin, R. R., Buysse, D. J., Edinger, J. D., Espie, C. A., & Lichstein, K. L. (2006). Psychological and behavioral treatment of insomnia: Update of the recent evidence (1998–2004). *Sleep*, *29*(11), 1398–1414. doi:10.1093/sleep/29.11.1398

Morin, C. M., Rodrigue, S., & Ivers, H. (2003). Role of stress, arousal, and coping skills in primary insomnia. *Psychosomatic Medicine*, *65*(2), 259–267. doi:10.1097/01.PSY.0000030391.09558.A3

Morin, C. M., Vallières, A., & Ivers, H. (2007). Dysfunctional Beliefs and Attitudes about Sleep (DBAS): Validation of a brief version (DBAS-16). *Sleep*, *30*(11), 1547–1554.

Nicassio, P. M., Boylan, M. B., & McCabe, T. G. (1982). Progressive relaxation, EMG biofeedback and biofeedback placebo in the treatment of sleep-onset insomnia. *British Journal of Medical Psychology*, *55*(2), 159–166. doi:10.1111/j.2044-8341.1982.tb01494.x

Norell-Clarke, A., Jansson-Fröjmark, M., Tillfors, M., Harvey, A. G., & Linton, S. J. (2014). Cognitive processes and their association with persistence and remission of insomnia: Findings from a longitudinal study in the general population. *Behaviour Research and Therapy*, *54*, 38–48. doi:10.1016/j.brat.2014.01.002

Ohayon, M. M. (2002). Epidemiology of insomnia: What we know and what we still need to learn. *Sleep Medicine Reviews*, *6*(2), 97–111. doi:10.1053/smrv.2002.0186

Ohayon, M. M., Lemoine, P., Arnaud-Briant, V., & Dreyfus, M. (2002). Prevalence and consequences of sleep disorders in a shift worker population. *Journal of Psychosomatic Research*, *53*(1), 577–583. doi:10.1016/s0022-3999(02)00438-5

Ohayon, M. M., & Reynolds, C. F. (2009). Epidemiological and clinical relevance of insomnia diagnosis algorithms according to the DSM-IV & the International Classification of Sleep Disorders (ICSD). *Sleep Medicine*, *10*(9), 952–960. doi:10.1016/j.sleep.2009.07.008

Okajima, I., Komada, Y., & Inoue, Y. (2011). A meta-analysis on the treatment effectiveness of cognitive behavioral therapy for primary insomnia. *Sleep and Biological Rhythms*, *9*(1), 24–34. doi:10.1111/j.1479-8425.2010.00481.x

Ong, J., Shapiro, S., & Manber, R. (2008). Combining mindfulness meditation with cognitive-behavior therapy for insomnia: A treatment-development study. *Behavior Therapy*, *39*, 171–182. doi:10.1016/j.beth.2007.07.002

Ong, J., & Sholtes, D. (2010). A mindfulness-based approach to the treatment of insomnia. *Journal of Clinical Psychology*, *66*(11), 1175–1184. doi:10.1002/jclp.20736

Ong, J. C., Ulmer, C. S., & Manber, R. (2012). Improving sleep with mindfulness and

acceptance: A metacognitive model of insomnia. *Behaviour Research and Therapy*, *50*(11), 651–660. doi:10.1016/j.brat.2012.08.001

Pallesen, S., Nordhus, I. H., Kvale, G., Nielsen, G. H., Havik, O. E., Johnsen, B. H., & Skjøtskift, S. (2003). Behavioral treatment of insomnia in older adults: An open clinical trial comparing two interventions. *Behaviour Research and Therapy*, *41*(1), 31–48. doi:10.1016/s0005-7967(01)00122-x

Pech, M., & O'Kearney, R. (2013). A randomized controlled trial of problem-solving therapy compared to cognitive therapy for the treatment of insomnia in adults. *Sleep*, *36*(5), 739–749. doi:10.5665/sleep.2640

Perlis, M. L., Jungquist, C., Smith, M. T., & Posner, D. A. (2005). *Cognitive behavioral treatment of insomnia: A session-by-session guide*. New York, NY: Springer.

Poser, E. G., Fenton, G. W., & Scotton, L. (1965). The classical conditioning of sleep and wakefulness. *Behaviour Research and Therapy*, *3*(4), 259–264. doi:10.1016/0005-7967(65)90035-5

Puder, R., Lacks, P., Bertelson, A. D., & Storandt, M. (1983). Short-term stimulus control treatment of insomnia in older adults. *Behavior Therapy*, *14*(3), 424–429. doi:10.1016/s0005-7894(83)80104-x

Rahimi, A., Ahmadpanah, M., Shamsaei, F., Cheraghi, F., Bahmani, D. S., Holsboer-Trachsler, E., & Brand, S. (2016). Effect of adjuvant sleep hygiene psychoeducation and lorazepam on depression and sleep quality in patients with major depressive disorders: Results from a randomized three-arm intervention. *Neuropsychiatric Disease and Treatment*, *12*, 1507–1515. doi:10.2147/NDT.S110978

Rasskazova, E., Zavalko, I., Tkhostov, A., & Dorohov, V. (2014). High intention to fall asleep causes sleep fragmentation. *Journal of Sleep Research*, *23*(3), 297–303. doi:10.1111/jsr.12120

Ree, M., & Harvey, A. (2004). Investigating safety behaviours in insomnia: The development of the Sleep-Related Behaviours Questionnaire (SRBQ). *Behaviour Change*, *21*(1), 26–36. doi:10.1375/bech.21.1.26.35971

Ribordy, S. C., & Denney, D. R. (1977). The behavioral treatment of insomnia: An alternative to drug therapy. *Behaviour Research and Therapy*, *15*(1), 39–50. doi:10.1016/0005-7967(77)90086-9

Richardson, G. S., Carskadon, M. A., Flagg, W., Van den Hoed, J., Dement, W. C., & Mitler, M. M. (1978). Excessive daytime sleepiness in man: Multiple sleep latency measurement in narcoleptic and control subjects. *Electroencephalography and Clinical Neurophysiology*, *45*(5), 621–627. doi:10.1016/0013-4694(78)90162-1

Riemann, D., Spiegelhalder, K., Feige, B., Voderholzer, U., Berger, M., Perlis, M., & Nissen, C. (2010). The hyperarousal model of insomnia: A review of the concept and its evidence. *Sleep Medicine Reviews*, *14*(1), 19–31. doi:10.1016/j.smrv.2009.04.002

Riley, W. T., Mihm, P., Behar, A., & Morin, C. M. (2010). A computer device to deliver behavioral interventions for insomnia. *Behavioral Sleep Medicine*, *8*(1), 2–15. doi:10.1080/15402000903425314

Ritterband, L. M., Bailey, E. T., Thorndike, F. P., Lord, H. R., Farrell-Carnahan, L., & Baum, L. D. (2012). Initial evaluation of an internet intervention to improve the sleep of cancer survivors with insomnia. *Psycho-Oncology*, *21*(7), 695–705. doi:10.1002/pon.1969

Ritterband, L. M., Thorndike, F. P., Gonder-Frederick, L. A., Magee, J. C., Bailey, E. T., Saylor, D. K., & Morin, C. M. (2009). Efficacy of an internet-based behavioral

intervention for adults with insomnia. *Archives of General Psychiatry*, *66*(7), 692–698. doi:10.1001/archgenpsychiatry.2009.66

Ritterband, L. M., Thorndike, F. P., Ingersoll, K. S., Lord, H. R., Gonder-Frederick, L., Frederick, C., … Morin, C. M. (2017). Effect of a web-based cognitive behavior therapy for insomnia intervention with 1-year follow-up: A randomized clinical trial. *JAMA Psychiatry*, *74*(1), 68–75. doi:10.1001/jamapsychiatry.2016.3249

Roehrs, T., & Roth, T. (2001). Sleep, sleepiness, sleep disorders and alcohol use and abuse. *Sleep Medicine Reviews*, *5*(4), 287–297. doi:10.1053/smrv.2001.0162

Sanavio, E. (1988). Pre-sleep cognitive intrusions and treatment of onset-insomnia. *Behaviour Research and Therapy*, *26*(6), 451–459. doi:10.1016/0005-7967(88)90139-8

Sanavio, E., Vidotto, G., Bettinardi, O., Rolletto, T., & Zorzi, M. (1990). Behaviour therapy for DIMS: Comparison of three treatment procedures with follow-up. *Behavioural Psychotherapy*, *18*(3), 151–167. doi:10.1017/s0141347300009654

Schutte-Rodin, S., Broch, L., Buysse, D., Dorsey, C., & Sateia, M. (2008). Clinical guideline for the evaluation and management of chronic insomnia in adults. *Journal of Clinical Sleep Medicine*, *4*(5), 487–504.

Schwartz, D. R., & Carney, C. E. (2012). Mediators of cognitive-behavioral therapy for insomnia: A review of randomized controlled trials and secondary analysis studies. *Clinical Psychology Review*, *32*(7), 664–675. doi:10.1016/j.cpr.2012.06.006

Shimodera, S., Watanabe, N., Furukawa, T. A., Katsuki, F., Fujita, H., Sasaki, M., & Perlis, M. (2014). Change in quality of life after brief behavioral therapy for insomnia in concurrent depression: Analysis of the effects of a randomized controlled trial. *Journal of Clinical Sleep Medicine*, *10*, 433–439. doi:10.5664/jcsm.3624

Seyffert, M., Lagisetty, P., Landgraf, J., Chopra, V., Pfeiffer, P. N., Conte, M. L., & Rogers, M. A. (2016). Internet-delivered cognitive behavioral therapy to treat insomnia: A systematic review and meta-analysis. *PLOS One*, *11*(2), e0149139. doi:10.1371/journal.pone.0149139

Simon, G. E., & VonKorff, M. (1997). Prevalence, burden, and treatment of insomnia in primary care. *American Journal of Psychiatry*, *154*(10), 1417–1423. doi:10.1176/ajp.154.10.1417

Sivertsen, B., Krokstad, S., Øverland, S., & Mykletun, A. (2009). The epidemiology of insomnia: Associations with physical and mental health—The HUNT-2 study. *Journal of Psychosomatic Research*, *67*(2), 109–116. doi:10.1016/j.jpsychores.2009.05.001

Spielman, A. J., Caruso, L. S., & Glovinsky, P. B. (1987). A behavioral perspective on insomnia treatment. *Psychiatric Clinics of North America*, *10*(4), 541–553.

Ström, L., Pettersson, R., & Andersson, G. (2004). Internet-based treatment for insomnia: A controlled evaluation. *Journal of Consulting and Clinical Psychology*, *72*(1), 113–120. doi:10.1037/0022-006X.72.1.113

Sunnhed, R., & Jansson-Fröjmark, M. (2013). Pre-sleep arousal, unhelpful beliefs and maladaptive sleep behaviors as mediators in cognitive behavior therapy for insomnia. *Sleep Medicine*, *14*, e158–e159. doi:10.1016/j.sleep.2013.11.365

Suzuki, E., Tsuchiya, M., Hirokawa, K., Taniguchi, T., Mitsuhashi, T., & Kawakami, N. (2008). Evaluation of an internet-based self-help program for better quality of sleep among Japanese workers: A randomized controlled trial. *Journal of Occupational Health*, *50*(5), 387–399. doi:10.1539/joh.l7154

Tang, N. K. Y., & Harvey, A. G. (2004). Effects of cognitive arousal and physiological arousal on sleep perception. *Sleep*, *27*(1), 69–78. doi:10.1093/sleep/27.1.69

Tang, N. K. Y., Schmidt, D. A., & Harvey, A. G. (2007). Sleeping with the enemy: Clock monitoring in the maintenance of insomnia. *Journal of Behavior Therapy and Experimental Psychiatry*, *38*(1), 40–55. doi:10.1016/j.jbtep.2005.07.004

Thiart, H., Ebert, D. D., Lehr, D., Nobis, S., Buntrock, C., Berking, M., … Riper, H. (2016). Internet-based cognitive behavioral therapy for insomnia: A health economic evaluation. *Sleep*, *39*(10), 1769–1778. doi:10.5665/sleep.6152

Thiart, H., Lehr, D., Ebert, D. D., Berking, M., & Riper, H. (2015). Log in and breathe out: Internet-based recovery training for sleepless employees with work-related strain—Results of a randomized controlled trial. *Scandinavian Journal of Work, Environment & Health*, *41*(2), 164–174. doi:10.5271/sjweh.3478

Thorndike, F. P., Saylor, D. K., Bailey, E. T., Gonder-Frederick, L., Morin, C. M., & Ritterband, L. M. (2008). Development and perceived utility and impact of an internet intervention for insomnia. *E-Journal of Applied Psychology*, *4*(2), 32–42. doi:10.7790/ejap.v4i2.133

Trauer, J., Qian, M., Doyle, J., Rajaratnam, S., & Cunnington, D. (2015). Cognitive behavioral therapy for chronic insomnia: A systematic review and meta-analysis. *Annals of Internal Medicine*, *163*, 191–204. doi:10.7326/m14-2841

Turner, R. M., & Ascher, L. M. (1979). Controlled comparison of progressive relaxation, stimulus control, and paradoxical intention therapies for insomnia. *Journal of Consulting and Clinical Psychology*, *47*(3), 500–508. doi:10.1037/0022-006x.47.3.500

VanderPlate, C., & Eno, E. N. (1983). Electromyograph biofeedback and sleep onset insomnia: Comparison of treatment and placebo. *Behavioral Engineering*, *8*(4), 146–153.

Van Straten, A., Emmelkamp, J., De Wit, J., Lancee, J., Andersson, G., Van Someren, E. J. W., & Cuijpers, P. (2014). Guided internet-delivered cognitive behavioural treatment for insomnia: A randomized trial. *Psychological Medicine*, *44*(7), 1521–1532.

Vincent, N., & Lewycky, S. (2009). Logging on for better sleep: RCT of the effectiveness of online treatment for insomnia. *Sleep*, *32*(6), 807–815. doi:10.1093/sleep/32.6.807

Voinescu, B. I., Szentagotai, A., & David, D. (2013). Internet-administered cognitive-behavioral therapy for insomnia. *Journal of Cognitive and Behavioral Psychotherapies*, *13*(1a), 225–237.

Wang, J., Wei, Q., Wu, X., Zhong, Z., & Li, G. (2016). Brief behavioral treatment for patients with treatment-resistant insomnia. *Neuropsychiatric Disease and Treatment*, *4*, 1967–1975. doi:10.2147/ndt.s110571

Watanabe, N., Furukawa, T. A., Shimodera, S., Morokuma, I., Katsuki, F., Fujita, H., … Perlis, M. L. (2011). Brief behavioral therapy for refractory insomnia in residual depression: An assessor-blind, randomized controlled trial. *Journal of Clinical Psychiatry*, *72*(12), 1651–1658. doi:10.4088/jcp.10m06130gry

Wong, M., Ree, M., & Lee, C. (2016). Enhancing CBT for chronic insomnia: A randomised clinical trial of additive components of mindfulness or cognitive therapy. *Clinical Psychology and Psychotherapy*, *23*, 377–385. doi:10.1002/cpp.1980

Woodley, J., & Smith, S. (2006). Safety behaviors and dysfunctional beliefs about sleep: Testing a cognitive model of the maintenance of insomnia. *Journal of Psychosomatic Research*, *60*(6), 551–557. doi:10.1016/j.jpsychores.2006.03.002

World Health Organization. (1993). *ICD-10, the ICD-10 classification of mental and behavioural disorders: Diagnostic criteria for research*. Geneva, Switzerland: World Health Organization.

Wu, J., Appleman, E., Salazar, R., & Ong, J. (2015). Cognitive behavioral therapy for insomnia comorbid with psychiatric and medical conditions: A meta-analysis. *JAMA Internal Medicine*, *175*, 1461–1472. doi:10.1001/jamainternmed.2015.3006

Yang, C., Lin, S., & Cheng, C. (2013). Transient insomnia versus chronic insomnia: A comparison study of sleep-related psychological/behavioral characteristics. *Journal of Clinical Psychology*, *69*(10), 1094–1107. doi:10.1002/jclp.22000

Zhang, B., & Wing, Y.-K. (2006). Sex differences in insomnia: A meta-analysis. *Sleep*, *29*(1), 85–93. doi:10.1093/sleep/29.1.85

Zwart, C. A., & Lisman, S. A. (1979). Analysis of stimulus control treatment of sleep-onset insomnia. *Journal of Consulting and Clinical Psychology*, *47*(1), 113–118. doi:10.1037/0022-006x.47.1.113

18

The Scientific Status of Evidence-Based Psychotherapies

Concluding Thoughts

Daniel David,[1] Steven Jay Lynn,[2] and Guy H. Montgomery[3]

[1] *Department of Clinical Psychology and Psychotherapy, Babeş-Bolyai University, Cluj-Napoca, Romania*

[2] *Psychology Department, Binghamton University, Binghamton, USA*

[3] *Department of Population Health Science and Policy, Icahn School of Medicine at Mount Sinai, New York, USA*

Our book stands at the forefront of a burgeoning movement to evaluate the scientific status of diverse psychotherapies and to encourage the widespread practice of evidence-based psychotherapies. We defined the highest standard of evidence-based practice herein as (1) the use of psychological interventions determined to be effective in rigorous clinical trials and comprehensive qualitative and quantitative reviews of the literature and (2) based on well-established scientific theories. Each contributor to this volume has clearly distinguished interventions that produce successful outcomes and are grounded in scientifically supported mechanisms of action from clinical practices that bear the patina of science yet lack crucial empirical support.

To facilitate our confidence that a psychotherapy can be considered to be well supported, we requested that contributors use David and Montgomery's (2011) framework, which categorizes interventions based not only on the level of support for outcomes associated with the treatment package but also on the level of support for mechanisms hypothesized to mediate or moderate treatment effects. This categorical scheme generates nine categories ranging from I (well-supported effectiveness and mechanisms) to IX (not effective or harmful, or a "pseudoscientifically oriented psychotherapy"; David & Montgomery, 2011, p. 94). We hope that our contributors' efforts have succeeded in educating the scientific community and the public about the important difference between scientific and pseudoscientific practices in psychotherapy. Our book is thus geared not only to identifying psychological treatments with a strong evidential base but also to educating readers about *how* to identify such interventions for a variety of conditions and disorders.

As research progresses, the list of evidence-based psychological treatments for clinical conditions will no doubt change, as new treatments gain legitimacy and acceptance in the psychotherapy community and some old treatments relinquish their scientific status

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

as empirically supported. Although our book provides a snapshot of the current status of evidence-based psychological treatments, we contend that:

1. What we identified as evidence-based psychological treatments in Category I (well-supported theory, well-supported treatment package) will most likely remain in that category, as substantial evidence has already accrued regarding their effectiveness. Nevertheless, we predict with confidence that future research will warrant that more treatments be included in Category I. The knowledge of what "works" and what does not will steer the field toward refining and enhancing extant treatments and developing new and exciting approaches to treating psychological conditions.
2. The interventions we identified as belonging to Category IX, which are marked by strong contradictory evidence and can rightly be considered pseudoscientific practices, will probably languish as part of the history of the field and not evolve into empirically supported practices. We suggest that responsible therapists, concerned about ethical professional practices, would do well to eschew such interventions, as they may harm rather than help patients (see also Lilienfeld, 2007).
3. Psychological treatments ranked in the middle categories (II–VIII), which are marked by mixed support for their effectiveness and hypothesized mechanisms, will probably shift in category placement as the research base expands and the empirical status of each therapy becomes increasingly clear and solidified. For example, some psychotherapies currently designated as Category IV, an early investigational stage in which most psychological treatments are likely to "start," might ultimately warrant reassignment to Category I as researchers rise to the challenge of rigorously evaluating treatment effectiveness and mechanisms. On the contrary, other psychological treatments may come eventually to be recognized as lacking in empirical support and consequently drop in the rankings. If changes in empirical status categorization *do not* occur, then it would suggest that psychotherapy research is stagnant or, worse, regressive. Such circumstances should be viewed as a warning bell for the psychotherapy community.

Various authors (e.g., Barlow, 2004) have argued that we should distinguish between psychotherapy and psychological treatments. Psychotherapy refers to a more general system that includes theoretical principles, mechanisms, and techniques. In contrast, psychological treatment refers to well-defined and articulated therapeutic packages or clinical protocols that often are derived from a general system of psychotherapy and are ready to be evaluated (e.g., by randomized clinical trials) or implemented in clinical practice. The contributors to this book focused on identifying evidence-based psychological treatments for various clinical conditions. However, starting from the findings presented in this compendium, several questions are important for future researchers, scholars, and clinicians to explore, in order to enhance and expand the conclusions presented in each chapter:

1. Do we focus limited resources directed toward training new generations of psychotherapists mainly on individual evidence-based psychological treatments or on more general or encompassing psychotherapy systems? If the answer is to focus on evidence-based treatments, will future practitioners be able to work successfully in a creative yet scientifically oriented manner when well-supported treatments are not

yet available? For example, if clinical practitioners are trained in specific cognitive–behavioral therapy (CBT) protocols but not in CBT as a general system, will they be able to devise effective treatment plans in the absence of an empirically supported and well-articulated evidence-based CBT protocol?

2. If clinical practitioners are trained primarily in psychoanalysis, for example, but also acquire training in specific CBT protocols but not in CBT as a general system, can we consider their expertise in CBT to be equivalent to that of practitioners trained in CBT both as a general system and as specific protocols? Most readers would probably agree that an optimal strategy is to train psychotherapists in both general systems and allied evidence-based treatments. Yet we might still ask: On what basis do clinicians or training programs choose a general system to champion, when knowledge of active treatment mechanisms is not as well developed as knowledge of treatment efficacy, as is typically the case in the psychotherapies reviewed in this volume? Moreover, how do we address the possibility that a general system of psychotherapy is theoretically incompatible with a promising evidence-based psychological treatment?
3. If two out of three CBT clinical protocols are validated for various anxiety disorders, can we consider CBT to be an evidence-based system for those anxiety disorders? This issue cropped up in many chapters, in which mixed yet generally strong support was described for a particular psychological disorder or condition. We suggest that further discussion in the psychotherapy field be devoted to analysis of adequate criteria for extending the label of evidence-based from psychological treatments to the more general system from which they are derived.

We propose further questions and topics to strengthen the theoretical and methodological impact of the chapters:

4. Is an evidence-based psychological treatment in which underlying mechanisms of change correspond to the etiopathogenetic mechanisms or theory of the clinical condition (i.e., etiopathogenetic psychological treatment) more important than an evidence-based treatment in which mechanisms of change are not directly or demonstrably related to the etiopathogenetic mechanisms or theory of the clinical condition (i.e., symptomatic treatment)? For example, is a clinical protocol that focuses on changing catastrophizing in the context of anxiety problems (i.e., etiopathogenetic psychological treatment), which is theoretically related to the origin of excessive anxiety, more or equally important compared with an intervention that targets anxiety reduction via relaxation (e.g., symptomatic treatment)? Of course, assessing the efficacy/effectiveness of the treatments in randomized trials, including follow-up analyses (e.g., an etiopathogenetic treatment is expected to better prevent relapse than a symptomatic treatment), is the final way to answer this question, but the implication of the question is more general. Indeed, framing the issue another way, we might ask: What is the proper focus of practice and research? More specifically, should we focus primarily on symptoms or on addressing the hypothesized mechanisms that mediate or moderate symptom expression? If we address both, which is arguably an ideal strategy, is there a particular order in which we should proceed in tackling symptoms versus mechanisms? Should this question be considered in terms of individual disorders, or can we identify general principles for how to proceed that can be applied on a transdiagnostic basis? Currently, this fundamental question

related to etiopathogenetic versus symptomatic psychological treatments—so important in the medical field—is challenging to answer, as psychological problems might be (a) less dependent on their original causes, (b) less related to specific psychological or medical causes (e.g., "meaning-making" could be a general sanogenetic or health-related mechanism, no matter the specific content of the meaning), and/or (c) more difficult to conceptualize in the medical terms of the etiopathogenetic mechanisms generating symptoms or signs.

5. We suggest that more attention should be devoted to the type of clinical trial (i.e., superiority vs. noninferiority vs. equivalence trial) used to support evidence-based treatments. A lack of significant findings in a superiority clinical trial in which treatment A is designed to be *superior* to treatment B does not support the same conclusions and does not have the same implications as findings derived from a nonsuperiority trial in which treatment A is designed to be *not much worse* than treatment B (but treatment A could be more cost-effective or advantageous in some respect) or an *equivalent* clinical trial (e.g., in which treatment A is designed to be similar in efficacy to treatment B; but treatment A could be more cost-effective or advantageous in some respect) (Lesaffre, 2008).
6. How can we relate the evidence-based psychological treatment paradigm our book touts to the specific versus common factor debate in psychotherapy (for details see Wampold & Imel, 2015) and/or to other evidence-based approaches, such as those focused more narrowly on the therapeutic relationship, rather than on the treatment package in its entirety (e.g., evidence-based psychotherapy relationships: Norcross, 2015)? Our approach, which considers both efficacy and theory, may provide a resolution to the debate by assessing general as well as specific mechanisms of change and serve as a platform for integrating various practices and mechanisms (e.g., therapeutic alliance with specific factors). In other words, our proposed framework can accommodate and integrate various debates in the psychotherapy field: (a) the theory component can assimilate, for example, the general versus specific factor approach, and (b) the practical approach (e.g., evaluating the effectiveness of a therapy and its components—global treatment package vs. componential analysis of treatment package) can accommodate various evidence-based practices (e.g., evidence-based psychotherapy relationship as a component in the global evidence-based treatment package).
7. What is the relationship between findings derived from evidence-based research on psychotherapy that is based on classical diagnostic schemes, such as that of the DSM-5 (American Psychiatric Association, 2013), versus findings derived from studies in which symptoms are conceptualized and treated in a transdiagnostic framework? Some scholars argue that there should not be a sole focus on clinical disorders per se (e.g., generalized anxiety disorder) in evaluating outcome, because of the heterogeneity of such clinical categories, but also on specific symptoms (e.g., fear) that cut across different clinical conditions (e.g., generalized anxiety disorder, panic attack). The conclusions of research may thus vary widely with the nature of the measures chosen to gauge treatment success (e.g., symptomatic change vs. shift in diagnostic status vs. both diagnosis and specific symptoms assayed).
8. How can we extend the concept of evidence-based psychological treatments from the clinical domain (e.g., clinical and subclinical problems) to facilitate human

development and optimize organizations, promote health in the general population, and prevent clinical conditions and disorders? Should we change the label of "psychological treatment" to "psychological intervention" or "psychological modification" to avoid the problem of using clinical constructs, such as "treatment," for nonclinical conditions?

We have posed many questions, yet at least one outcome is probable: Some treatments that are viewed as the state of the science today might be replaced by interventions that are even more cost-effective and/or better supported empirically, as knowledge about psychotherapy accrues. We plan to monitor shifts in the landscape of psychotherapy as they develop in order to provide readers with an ongoing picture of the empirical status of psychotherapy. As a follow-up to this book, we intend to develop an evidence-based psychological treatment website that readers can consult as the field evolves.

We are pleased that the contributors to this book have identified crucial gaps in the research literature on psychotherapy. We are optimistic that our understanding of the scientific status of psychotherapies will be advanced by: (1) attention to the role of nonspecific factors in accounting for treatment effects, including the role of expectancies, the treatment alliance, and allegiance effects in research (i.e., the impact of implementing a treatment one is closely allied with); (2) dismantling treatments and identifying the effective components of diverse psychotherapies; and (3) determining not only which treatments are superior to no treatment or treatment as usual but also which treatments are superior to competing interventions that vie for empirical support. Additionally, it will be important for researchers to carefully stipulate hypothesized mechanisms in advance rather than on a post hoc basis so that mechanistic studies can be operationalized, conducted, and interpreted with confidence. We encourage large-scale collaborations across laboratories that are aimed not only at replicating findings across testing contexts but also at conducting sufficiently powered studies to draw definitive conclusions regarding treatment outcome on a short- and long-term basis. Finally, we encourage researchers to specify in advance criteria for adjudging treatment success, such as what would be considered a clinically significant versus a statistically significant treatment gain and over what time period. In closing, we hope that our book plays a pivotal role in educating scholars and clients about the modern concept of evidence-based psychological treatments and in stimulating critical thinking in identifying, analyzing, implementing, disseminating, and further developing evidence-based psychological treatments.

References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.

Barlow, D. (2004). Psychological treatments. *American Psychologist*, *59*(9), 869–878. doi:10.1037/0003-066x.59.9.869

David, D., & Montgomery, G. (2011). The scientific status of psychotherapies: A new evaluative framework for evidence-based psychosocial interventions. *Clinical Psychology: Science and Practice*, *18*, 88–99. doi:10.1111/j.1468-2850.2011.01239.x

Lesaffre, E. (2008). Superiority, equivalence, and non-inferiority trials. *Bulletin of the NYU Hospital for Joint Diseases*, *66*(2), 150–154.

Lilienfeld, S. O. (2007). Psychological treatments that cause harm. *Perspectives in Psychological Sciences*, *2*(1), 53–70. doi:10.1111/j.1745-6916.2007.00029.x

Norcross, J. (2015). Evidence-Based Therapy Relationships Task Force conclusions. *Society for the Advancement of Psychotherapy*. Retrieved September 25, 2017, from http://societyforpsychotherapy.org/evidence-based-therapy-relationships

Wampold, B., & Imel, Z. E. (2015). *The great psychotherapy debate* (2nd ed.). New York, NY: Routledge.

Index

Page numbers in *italics* refer to tables.

a

"absolute efficacy"
  definition 12
  see also specific therapies
acceptance and commitment therapy (ACT)
  depression 24
  generalized anxiety disorder (GAD) 102
  obsessive–compulsive disorder (OCD) 132–134
  schizophrenia-spectrum disorders 279, 370
acceptance-based theory/therapy
  generalized anxiety disorder (GAD) 100–102
  insomnia 474, 496
action-oriented therapy: psychopathy 286–287
adolescents/youths
  bipolar disorder (BD) 46–47, 52–53
  borderline personality disorder (BPD) 318
    dialectical behavior therapy (DBT-A) 303, 305
    mentalization-based therapy (MBT-A) 307, 308
    schema-focused therapy (SFT) 310
    transference-focused psychotherapy (TFP-A) 314
  delinquency/psychopathy 280, 281, 287
  see also anorexia nervosa; attention-deficit hyperactivity disorder (ADHD)
Adult Attachment Inventory 315
affective disturbance model of schizophrenia 376, 377
Alcoholics Anonymous (AA) 220, 235–237, 240
alcohol use disorders (AUDs) 219–220
  classification and diagnosis 221, *222*
  epidemiology 222–224
  evaluation of treatments
    Alcoholics Anonymous (AA) (Category II) 235–237, 240
    behavioral couples therapy (BCT) (Category I) 231–235
    cognitive–behavioral therapy (CBT) (Category I) 229–230, 281–282
    confrontational interventions (Category IX) 237–239
    motivational interviewing (MI/MET) (Category I) 227–229
    reinforcement-based treatment (Category I) 244–247
  evaluative framework *225*, 240
  history of treatment 220–221
  implications for research and practice 239–240
  and insomnia 467, 469
  see also substance abuse
alexithymia 349–350

Evidence-Based Psychotherapy: The State of the Science and Practice, First Edition.
Edited by Daniel David, Steven Jay Lynn, and Guy H. Montgomery.

Ambiguous Intentions Hostility Questionnaire 384
"ambiguous scenario training": obsessive–compulsive disorder (OCD) 139–140
American Academy of Pediatrics 409, 410, 420
  Subcommittee on ADHD 437–438
American Psychiatric Association 3
  generalized anxiety disorder (GAD) 95
  obsessive–compulsive disorder (OCD) 123
  panic disorder (PD) 63
  social anxiety disorder (SAD) 71
  see also Diagnostic and Statistical Manual (DSM)
American Psychological Association
  definition of psychotherapy 411
  Division of Clinical Psychology 3, 14, 19, 22, 24, 330
  facilitated communication 420
  Presidential Taskforce 190, 192, 193, 197, 200, 329, 368, 381
anorexia nervosa 190–196
  Category I: evidence-based psychotherapies 190
  Category II: intervention-driven psychotherapies 190–191
  Category III: theory-driven psychotherapies 191
  Category IV: investigational psychotherapies 191–196
  Categories V–IX 196
  evaluative framework *191*
  see also eating disorders
antisocial personality disorder (ASPD) 273–274
anxiety
  and attention bias 137
  and depression 1–2
  and "meta-worry" 376
  see also fear; generalized anxiety disorder (GAD); panic disorder (PD)
Anxiety Disorders Interview Schedule 129, 161
applied behavior analysis (ABA): autism 427, 428, 429
applied relaxation (AR) *see* relaxation techniques
Assertive Community Treatment: schizophrenia 392
attachment *see* childhood trauma and attachment issues
attention bias
  anxiety 137
  social anxiety disorder (SAD) 72–73
  specific phobia 80
attention bias modification/training (ABM/ABMT)
  obsessive–compulsive disorder (OCD) 136–139, 143
  social anxiety disorder (SAD) 74–75
  specific phobia 82
attention-deficit hyperactivity disorder (ADHD)
  Category I: evidence-based psychotherapies 437–439
  Category II: intervention-driven psychotherapies 439–444
  Category III: theory-driven psychotherapies 444–446
  Category IV: investigational psychotherapies 446–451
  Categories V—IX 451–452
  description 435–437
  evaluative framework *437*
  implications for practice 453
  implications for research 452
attention intention effort (AIE) model of insomnia 470–472
auditory hallucinations 363, 367–368, 371, 374, 377
auditory integration: autism 419, 420, 427
Autism Society of America 414
autism spectrum disorder (ASD) 407
  core characteristics 408–409
  defining psychotherapy in context of 410–411
  etiology and theoretical approach 409–410
  evaluative framework *414*, 420–426
    Category I 420–423
    Category II 423
    Category III 423–426

Category IV 424
Categories V–VIII 426
Category IX 420
heterogeneity 408
history of diagnosis 407–408
implications for practice and research 427–428
prevalence 409
avoidance/safety behaviors
panic disorder (PD) 65–66
posttraumatic stress disorder (PTSD) 158, 159, 176–177
social anxiety disorder (SAD) 72
see also exposure exercises/therapy

b

Beck, A. 170, 254–255, 308, 366
Beck Anxiety Inventory 256
Beck Depression Inventory II 129, 256
behavioral activation (BA): depression 19–20
"behavioral classroom management": attention-deficit hyperactivity disorder (ADHD) 444
behavioral couples therapy (BCT)
alcohol use disorders (AUDs) 231–235
depression 24–25
behavioral family systems therapy (BFST): anorexia 192
behavioral model of insomnia 468–469, 473
behavioral parenting training (BPT): attention-deficit hyperactivity disorder (ADHD) 444–446
"behavioral peer interventions": attention-deficit hyperactivity disorder (ADHD) 444
behavioral therapies (BT)
anorexia 196
bulimia 206
depression 18–20
sexual dysfunction 262–264
behavioral weight control (BWC): binge eating 200–201
belief modification: schizophrenia 367
binge eating disorder 197–202
Category I: evidence-based psychotherapies 197–199
Category II: intervention-driven psychotherapies 199
Category III: theory-driven psychotherapies 199
Category IV: investigational psychotherapies 200–202
Categories V–IX 202
evaluative framework *197*
see also eating disorders
biofeedback
attention-deficit hyperactivity disorder (ADHD) 448–450
insomnia 488–489
panic disorder (PD) 68
biosocial theory of borderline personality disorder (BPD) 303–304
bipolar disorder (BD) 37–38
Categories I–III 40
Category IV: investigational psychotherapies 40–50
Categories V–IX 50
evaluative framework 39–40
implications for practice and research 50–53
bodily sensations, fear of: panic disorder (PD) 64–65
borderline personality disorder (BPD) 299–300
Category II: intervention-driven psychotherapies 301–308
Category IV: investigational psychotherapies 308–315
evaluative framework 300–301
implications for practice 317–318
implications for research 316–317
other therapies 315–316
summary and conclusions 319
brief Adlerian psychodynamic psychotherapy (B-APP): generalized anxiety disorder (GAD) 110
brief behavioral treatment for insomnia (BBT-I) 476–477
bulimia nervosa 202–208
Category I: evidence-based psychotherapies 202–204

bulimia nervosa (*Continued*)
Category II: intervention-driven psychotherapies 205
Category III: theory-driven psychotherapies 205
Category IV: investigational psychotherapies 205–208
Categories V–IX 208
evaluative framework *202*
see also eating disorders

C

case studies
depersonalization/derealization disorder (DP/DR) 332–336, 337
dissociative identity disorder (DID) 339–340, 341–342, 344, 349
obsessive–compulsive disorder (OCD) 135
psychopathy 272, 285–286
catastrophic misinterpretations: panic disorder (PD) 65
Centers for Disease Control (CDC): autism 409, 412–414
change, theory and mechanisms of 3–5
anorexia: cognitive–behavioral therapy (CBT) 194
attention-deficit hyperactivity disorder (ADHD)
behavioral parent training (BPT) 445–446
mindfulness 451
neurofeedback training 449
organizational skills training 443–444
psychoeducation 442
social skills training 447
binge eating: cognitive–behavioral therapy (CBT) 198, 199
bipolar disorder (BD) 43, 45, 50, 51
borderline personality disorder (BPD)
cognitive–behavioral therapy (CBT) 313
dialectical behavior therapy (DBT) 304–305
mentalization-based therapy (MBT) 308
schema-focused therapy (SFT) 311
transference-focused psychotherapy (TFP) 315
depersonalization/derealization (DP/DR) 334–335
depression (MDD) 28, 29
behavioral therapy 20
cognitive therapy (CT) 15–17
interpersonal psychotherapy (IPT) 18
problem-solving therapy (PST) 20
rational emotive behavior therapy (REBT) 22
third-wave cognitive–behavioral therapy (CBT) 24
dissociative identity disorder (DID)
cognitive analytical therapy (CAT) 344
cognitive approaches 340, 341
hypnotherapy 343
narrative approach 342
schizophrenia-spectrum disorders 392–393
cognitive–behavioral therapy (CBT) 378–380
mentalization-based therapy (MBT) 390–391
social skills training (SST) 384–385
change talk: motivational interviewing (MI) 227, 228
childhood trauma and attachment issues
borderline personality disorder (BPD) 306, 307–308, 309, 310–311, 315, 318
dissociative identity disorder (DID) 340–342
generalized anxiety disorder (GAD) 107
model of schizophrenia 377
posttraumatic stress disorder (PTSD) 162, 163
classical conditioning: sleep and insomnia 467–468
classification of psychotherapies 3–5
new evaluative framework 5, 6
see also evaluative framework/categories *under specific disorders*
client-centered therapy (CCT)/humanistic approach
alcohol use disorders (AUD) 227, 228
panic disorder (PD) 70

Clinician-Administered PTSD Scale (CAPS) 161
Cochrane Reviews 3, 22, 23–24, 45–46, 300–301, 302, 370–371, 382, 446–447
Cogmed training 448
cognitive and affective model of schizophrenia 376
cognitive analytical therapy (CAT)
  anorexia nervosa 194
  dissociative identity disorder (DID) 344, 345
  schizophrenia-spectrum disorders 375
cognitive avoidance theory of generalized anxiety disorder (GAD) 106–107
cognitive–behavioral therapy (CBT)
  alcohol use disorders (AUDs) 229–230
    vs AA approach 236, 237
  attention-deficit hyperactivity disorder (ADHD) 439–441
  autism spectrum disorder 424–426, 428, 429
  bipolar disorder (BD) 40–43
  borderline personality disorder (BPD) 312–313
  depersonalization/derealization (DP/DR) 336–337
  eating disorders 208, 209–210
    anorexia 193–194
    binge eating 197–198
    bulimia 202–203
  generalized anxiety disorder (GAD) 97–98
    vs other therapies 102–103, 108, 109, 110
  insomnia
    CBT-I 475–476, 489–495
    computerized (CCBT) 477–480, *481–482*
    mediators 474–475
  obsessive–compulsive disorder (OCD) 131–132, 144–145
  panic disorder (PD) 64–66
    vs other therapies 67, 68, 69, 70, 81–83
  posttraumatic stress disorder (PTSD) 164, 170, 172
  psychopathy 279–283
  schizophrenia 366–380
  sexual dysfunction disorders 254–260
  social anxiety disorder (SAD) 72–73
    vs other therapies 74, 76, 77, 81–83
  specific phobia 79–80, 81–83
cognitive bias
  and delusions: schizophrenia 386, 390
  panic disorder (PD) 65
  social anxiety disorder (SAD) 72–73
  specific phobia 79–80
cognitive bias modification *see* concreteness training (CNT)
cognitive dissonance theory 227, 228
cognitive model
  borderline personality disorder (BPD) 312, 313
  depersonalization/derealization (DP/DR) 336, 337
  dissociative identity disorder (DID) 340–341
  insomnia 472–474
cognitive processing therapy (CPT): posttraumatic stress disorder (PTSD) 166, 171–172
cognitive remediation: schizophrenia 387–389, 392
cognitive restructuring
  dissociative identity disorder (DID) 340, 341
  posttraumatic stress disorder (PTSD) 165
  social anxiety disorder (SAD) 73
  specific phobia 80
cognitive therapy (CT)
  depression 14–17
  insomnia 486
  obsessive–compulsive disorder (OCD) 128–130, 144
  posttraumatic stress disorder (PTSD) 170, 174
cognitive training interventions: attention-deficit hyperactivity disorder (ADHD) 447–448, 453
community reinforcement approach (CRA): alcohol use disorders (AUDs) 225–226
  Family Training (CRAFT) 226, 238–239

comorbidity
attention-deficit hyperactivity disorder (ADHD) 435
borderline personality disorder (BPD) 300, 316
dissociative disorders 332, 345–346, 349
insomnia 467, 469
posttraumatic stress disorder (PTSD) 176–177
schizophrenia 365
Comprehensive Alcohol Abuse and Alcoholism Prevention, Treatment, and Rehabilitation Act (1970) 220
computer-assisted approaches
autism spectrum disorder (ASD) 424
erectile dysfunction: ICBT 255, 256
insomnia: CCBT 477–480, *481–482*
posttraumatic stress disorder (PTSD): telemedicine 177–178
concreteness training (CNT): depression 21, 26–27, 28
conditioned fear responses 80, 168, 263, 264
confrontational interventions: alcohol use disorders (AUDs) 237–239
contextual approach: dissociative identity disorder (DID) 342, 345
contingency management (CM)
alcohol use disorders (AUDs) 225–227
attention-deficit hyperactivity disorder (ADHD) 442–443
contrast avoidance theory: generalized anxiety disorder (GAD) 107–108
coping skills
alcohol use disorders (AUDs) 229–230
psychopathy 281–282
schizophrenia 367–368
cortisol 162, 163, 304
couples
sex therapy 260–262
see also behavioral couples therapy (BCT)
cultural differences: sexual dysfunction 254, 267

d

David, D. and Montgomery, G. 3–4, 5, 6, 507–508
see also evaluative framework/categories *under specific disorders*
daydreaming 349
defeatist beliefs 378
defusion exercises/thought–action fusion 132–133, 134–135
delusions 363, 367–368, 369, 371, 375, 376, 379, 386, 393
depersonalization/derealization (DP/DR) 331, 332
posttraumatic stress disorder (PTSD) 158–159
treatments 332–337
sleep hygiene and daydream content interventions 349
depression
and anxiety 1–2
bipolar disorder (BD) 37–38, 41, 42
see also major depressive disorder (MDD)
Diagnostic and Statistical Manual (DSM)
alcohol use disorders (AUDs) 221, 224
autism spectrum disorder (ASD) 407–408, 409
bipolar disorder (BD) 37–38
borderline personality disorder (BPD) 299
dissociative disorders 331–332
eating disorders 189
insomnia 465, 466
major depressive disorder (MDD) 11
posttraumatic stress disorder (PTSD) 157–159
psychopathy 272–274
schizophrenic-spectrum disorders 363–365
sexual dysfunction 253
specific phobias 78
see also American Psychiatric Association
dialectical behavior therapy (DBT)
binge eating 201–202
borderline personality disorder (BPD) 301–305
bulimia 206
diathesis–stress (3P) model 41, 468, 469
dietary counseling *see* nutritional counseling (NC)
dietary interventions: attention-deficit hyperactivity disorder (ADHD) 436

dissociation 329–331, 350–351
innovative treatments 347–350
dissociative amnesia (DA) 346–347
dissociative fugue (DF) 346–347
dissociative identity disorder (DID) 331, 332
treatments 337–346, 350
dose–response relationship
Alcoholics Anonymous (AA) 237
brief behavioral treatment for insomnia 476–477
obsessive–compulsive disorder (OCD) interventions 144
trauma exposure and posttraumatic stress disorder (PTSD) 163
dot-probe task 75, 137–138, 473
dynamic deconstructive psychotherapy (DDP): borderline personality disorder (BPD) 316

e

"early maladaptive schema" (EMS) 310–311
eating disorders
description 189–190
discussion and conclusions 208–210
evaluative framework 190
implications for research and practice 209–210
see also anorexia nervosa; binge eating disorder; bulimia nervosa
EEG biofeedback: attention-deficit hyperactivity disorder (ADHD) 448–450
efficacy and mechanisms: evaluative framework 5, 6
see also evaluative framework/categories *under specific disorders*
Ego Identity Index 287
ego-oriented individual therapy (EOIT): anorexia 195–196
Ellis, A. 170, 366
EMG biofeedback: insomnia 488–489
emotional dysregulation model/regulation therapy
binge eating 201–202
borderline personality disorder (BPD) 303–305
dissociative symptoms 349–350
generalized anxiety disorder (GAD) 102, 104–105
emotional Stroop task 137
emotion-focused therapy (EFT)
female sexual dysfunction 266
panic disorder (PD) 70
empirically supported studies (ESTs) 329–330, 331
energy therapies: posttraumatic stress disorder (PTSD) 173–174
"equivocal evidence", definition 13
erectile dysfunction 253, 255–256, 261–262
exercise, physical 66–67, 200–201
exorcism: dissociative identity disorder (DID) 344–345
exposure exercises/therapy 82
generalized anxiety disorder (GAD) 105
posttraumatic stress disorder (PTSD) 165, 166–170, 174
social anxiety disorder (SAD) 73
specific phobia 80–81
exposure and ritual prevention (ERP): obsessive–compulsive disorder (OCD) 125–127, 129, 143, 144
eye movement desensitization and reprocessing (EMDR)
obsessive–compulsive disorder (OCD) 140–142, 143–144
panic disorder (PD) 70
posttraumatic stress disorder (PTSD) 173

f

facilitated communication: autism 419, 420, 427
family interventions
alcohol use disorders (AUDs) (CRAFT) 226, 238–239
anorexia 191–193
bipolar disorder (BD) 45–49, 52–53
bulimia 206–207
schizophrenia 392
fear
acquisition models 79
of bodily sensations 64–65
conditioned responses 80, 168, 263, 264

fear (*Continued*)
extinction learning 66, 67, 80, 168
normal and maladaptive 125
female sexual dysfunction 253–254, 257–259, 259–260, 264
5-HTTLPR polymorphism 162
FK506 binding protein gene 162
flooding treatments 335
fMRI studies 305, 449–450
focal psychodynamic therapy (FPT): anorexia 196
focal supportive psychotherapy (FSP): bulimia 205

g

generalized anxiety disorder (GAD)
Category I: evidence-based psychotherapies 96–102
Category II: intervention-driven psychotherapies 102–104
Category III: theory-driven psychotherapies 104–109
Category IV: investigational psychotherapies 109–110
features and prevalence 95–96
implications for research and practice 111
genetics
autism spectrum disorder (ASD) 409–410
posttraumatic stress disorder (PTSD) 161–162
grief therapy 25
guided self-help CBT (GSH-CBT)
binge eating 198–199
bulimia 203–204

h

hallucinations 363, 367–368, 369, 371, 375, 376, 377–378, 379
humanistic approach *see* client-centered therapy (CCT)
hyperarousal model
insomnia 469–470
panic disorder (PD) 64–65
hypersensitive attachment system 307–308
hypervigilance 65, 73
hypnotherapy
depersonalization/derealization (DP/DR) 335
dissociative amnesia/fugue (DA/DF) 347
dissociative identity disorder (DID) 342–343
female sexual dysfunction 264, 266
panic disorder (PD) 71
hypoactive sexual desire disorder (HSD) 253–254, 257–259
hypocapnia 68
hypomania 37–38

i

iatrogenic harm: dissociative identity disorder (DID) 339
identity
"identity diffusion" model of BPD 314–315
see also dissociative identity disorder (DID)
image-based *vs* verbal–linguistic quality of worry 106–107
imagery rescripting (ImRs): social anxiety disorder (SAD) 77
information-processing
posttraumatic stress disorder (PTSD) 171–172
social anxiety disorder (SAD) 72–73
specific phobia 79–80
insomnia 465
assessment 466
definitions 465–466
implications for research and practice 489–496
prevalence and risk factors 466–467
psychological theories and mechanisms of change 467–474
mediators in treatment effects 474–475
subtypes 466
therapeutic packages 475–489, *490–494*
sleep-related interventions for dissociation 348–349
intensive sleep retraining (ISR) 487

International Classification of Diseases (ICD) 221, 465, 466
International Index of Erectile Function (IIEF) 255, 256
internet-based CBT (ICBT)
  erectile dysfunction 255, 256
  see also computer-assisted approaches
interpersonal/emotional processing (I/EP) therapy: generalized anxiety disorder (GAD) 108–109
interpersonal psychotherapy (IPT)
  anorexia 195
  binge eating 200
  borderline personality disorder (BPD) 316
  bulimia 205–206
  depression 17–18
  social anxiety disorder (SAD) 75–76
interpersonal and social rhythm therapy (IPTSR): bipolar disorder (BD) 49–50
interpretation bias modification training (IBMT): obsessive–compulsive disorder (OCD) 139–140
intolerance of uncertainty (IU) model/therapy (IUT): generalized anxiety disorder (GAD) 96–98
intrusion symptoms: posttraumatic stress disorder (PTSD) 158

j

"jumping to conclusions" (JTC): schizophrenia 376, 379, 387

l

"limited reparenting" 309

m

major depressive disorder (MDD)
  Category I: evidence-based psychotherapies 13–17
  Category II: intervention-driven psychotherapies 17–20
  Category III: theory-driven psychotherapies 20–22
  Category IV: investigational psychotherapies 22–25
  Categories V–IX 25
  description 11–12
  evaluative framework 12–13
  implications for practice 27–29
  implications for research 26–27
maladaptive schemas/cognitions
  borderline personality disorder (BPD) 308–309, 310–311
  posttraumatic stress disorder (PTSD) 163–164, 170, 171–172
  sexual dysfunction 254–255, 259
male sexual dysfunction 253, 255–256, 259, 263–264
mania 37–38
MATRICS Consensus Cognitive Battery 384
Maudsley model of anorexia nervosa treatment for adults (MANTRA) 195
mechanisms of change *see* change, theory and mechanisms of
memory
  dissociative amnesia (DA) 346–347
  dissociative identity disorder (DID) 341–342
  imagery rescripting (ImRs) 77
  posttraumatic stress disorder (PTSD) 158, 163, 168
  recovery techniques 339, 350
  and saccadic eye movements 141
mentalization-based therapy (MBT)
  alexithymia 349–350
  borderline personality disorder (BPD) 305–308, 350
metacognitive model/therapy (MCT)
  generalized anxiety disorder (GAD) 98–100
  insomnia 474
  obsessive–compulsive disorder (OCD) 134–136
  schizophrenia-spectrum disorders 385–391
"meta-worry" 376
military veterans
  alcohol use disorders (AUDs) 237
  posttraumatic stress disorder (PTSD) 161–162, 163, 165, 166, 172, 176

mindfulness
generalized anxiety disorder (GAD) 100, 105–106
insomnia 474, 487–488, 496
panic disorder (PD) 67–69
sexual dysfunction 266
specific phobia 82
mindfulness-based cognitive therapy (MBCT)
attention-deficit hyperactivity disorder (ADHD) 450–451
bipolar disorder (BD) 43–44
generalized anxiety disorder (GAD) 105–106
mindfulness-based stress reduction (MBSR)
generalized anxiety disorder (GAD) 105–106
insomnia 487–488
social anxiety disorder (SAD) 76
Minnesota Multiphasic Personality Inventory (MMPI) Psychopathic Deviate scale 280
motivational interviewing (MI/MET): alcohol use disorders (AUDs) 227–229
multiple sleep latency test (MSLT) 471

n

narrative/story-based approaches 341–342, 424
National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) 299–300
National Institute for Health and Care Excellence (NICE) 3
attention-deficit hyperactivity disorder (ADHD) 435–436, 438, 444, 452
bipolar disorder (BD) 37, 46, 49, 51, 52
depression 14, 19, 22, 24–25
eating disorders 190, 192, 197, 204, 205–206
posttraumatic stress disorder (PTSD) 164, 166
National Standards Project (NAC): autism spectrum disorder (ASD) treatment reviews 415, *415–419*, 419
naturalistic teaching: autism 423–424
negative automatic thoughts: attention-deficit hyperactivity disorder (ADHD) 441
neurobiological perspective
autism spectrum disorder (ASD) 409–410
borderline personality disorder (BPD) 304, 305
posttraumatic stress disorder (PTSD) 162–163, 175–176
psychopathy 288
schizophrenia 365, 379–380
sleep 348
neurocognitive assessments: alcohol use disorders (AUDs) 239
neurofeedback training: attention-deficit hyperactivity disorder (ADHD) 448–450
neuroimaging (fMRI) 305, 449–450
nocturnal panic attacks 64
nonfalsifiable therapies 27, 29
nutritional counseling (NC)
anorexia 196
bulimia 207

o

object relations model of borderline personality disorder (BPD) 314–315
obsessive–compulsive disorder (OCD)
Category II: intervention-driven psychotherapies
acceptance and commitment therapy (ACT) 132–134
cognitive–behavioral therapy (CBT) 131–132, 144–145
cognitive therapy (CT) 128–130, 144
exposure and ritual prevention (ERP) 125–127, 129, 143, 144
metacognitive therapy (MCT) 134–136
Category IV: investigational psychotherapies
attention bias modification training (ABMT) 136–139, 143

eye movement desensitization and reprocessing therapy (EMDR) 140–142, 143–144
interpretation bias modification training (IBMT) 139–140
features 123–124
implications for practice 145–146
implications for research 142–145
summary and conclusions 146
operant learning theory 224, 259–260
organizational skills training (OST) 442–444
overgeneralization 21
oxytocin nasal spray 384

p

panic attacks 63, 64
panic disorder (PD)
Category I: evidence-based psychotherapies 64–66
Category II: intervention-driven psychotherapies 66
Category III: theory-driven psychotherapies 66–69
Category IV: investigational psychotherapies 69–70
Categories V–IX 70–71
features 63–64
implications for research and practice 81–83
panic-focused psychodynamic psychotherapy (PFPP) 69–70
paradoxical intention: insomnia 486–487
Personality Belief Questionnaire (PBQ) 312, 313
pharmacological treatments
attention-deficit hyperactivity disorder (ADHD) 436, 437–439
posttraumatic stress disorder (PTSD) 165–166, 175–176
phobias *see* social anxiety disorder (SAD); specific phobia
physical exercise 66–67, 200–201
PORT Review 381, 386, 391–392
Positive and Negative Syndrome Scale (PANSS) 371, 374–375, 384, 387–389, 390, 391
posttraumatic stress disorder (PTSD) 157–159
assessment 161
clinical features 159–160
diagnostic considerations 160
epidemiology 160–161
etiological pathways and causal mechanisms 161–166
behavioral and molecular genetics 161–162
cognitive influences 163–164
course, prognosis, and treatment 164–166
learning, modeling, and life events 163
neuroanatomy and neurobiology 162–163
sex and racial/ethnic considerations 164
panic attacks 64
treatments
comorbid disorders 176–177
pharmacological 165–166, 175–176
psychosocial 166–176
telemedicine 177–178
Present State Examination 336
problem-solving therapy (PST): depression 19, 20
progressive muscular relaxation *see* relaxation techniques
Project MATCH 228–229, 235, 236, 237
prolonged exposure (PE) therapy: posttraumatic stress disorder (PTSD) 168–169
psychodrama 286–287
psychodynamic/psychoanalytical model and therapies
attention-deficit hyperactivity disorder (ADHD) 451–452
borderline personality disorder (BPD) 313–315
bulimia 207–208
dissociative amnesia/fugue (DA/DF) 332–334, 337, 347
dissociative identity disorder (DID) 343–344

psychodynamic/psychoanalytical model and therapies (*Continued*)
generalized anxiety disorder (GAD) 109–110
panic disorder (PD) 69–70
psychopathy 285–287
social anxiety disorder (SAD) 76–77
psychoeducation
attention-deficit hyperactivity disorder (ADHD) 441–442
bipolar disorder (BD) 44–45
family 47–48
generalized anxiety disorder (GAD) 105
sexual dysfunction disorders 265
social anxiety disorder (SAD) 73
psychopathy 271–272
conceptualizing and measuring 272–275
evaluation of treatments 278–279
cognitive–behavioral therapy (CBT) 279–283
psychodynamic approaches 285–287
therapeutic communities 283–285
implications for practice 289–290
implications for research 287–289
methodological issues in treatment investigations 276–278
lack of consensus regarding conceptualization and assessment 278
lack of contemporary, empirically rigorous studies 276–277
lack of theoretically grounded treatments 277
prevalence 275
summary and conclusions 290
treatability debate 275–276
Psychopathy Checklist (PCL) 274, 275, 280, 281, 282–283, 284
psychosis *see* schizophrenic-spectrum disorders
psychotherapies
classification 3–5
efficacy and mechanisms: new evaluative framework 5, 6
evidence-based
and clinical practice 1–3
scientific status and future research 507–512
Psychotic Symptom Rating Scale (PSYRATS) 371, 387, 389–390, 391
pure self-help CBT (PSH-CBT)
binge eating 199
bulimia 204

r

randomized controlled trials (RCTs) 4
schizophrenia
cognitive–behavioral therapy (CBT) 370–375
metacognitive therapy (MCT) 387–389
social skills training (SST) 382–384
see also specific disorders/therapies
rational emotive behavioral therapy (REBT)
depression 21–22
schizophrenia 366
reality-testing: schizophrenia 367
reinforcement-based treatment: alcohol use disorders (AUDs) 244–247
"relative efficacy"
definition 12
see also specific therapies
relaxation techniques
generalized anxiety disorder (GAD) 102–103, 105
insomnia 470, 485–486, 495
obsessive–compulsive disorder (OCD) 133, 134
panic disorder (PD) 68–69
respiratory training with biofeedback: panic disorder (PD) 68
Rogers, C. 70, 227, 228
rumination 21

s

safety behaviors *see* avoidance/safety behaviors
schema-focused therapy (SFT): borderline personality disorder (BPD) 308–311

schizophrenic-spectrum disorders 363–365
evaluation of treatments 365–366
Category II 366–385
Category IV 385–391
other 391–392
implications for practice 393
implications for research 392–393
Seeking Safety (SS): posttraumatic stress disorder (PTSD) and substance abuse 176–177
"seepage" theory of hallucinations 377
self-help groups: Alcoholics Anonymous (AA) 220, 235–237
self-injury (NSSI)/suicidality 300–301, 302, 304–305, 307, 317, 318
self-perception theory 227, 228
sex offenders 283
sex therapy 260–262
sexual dysfunction disorders 253–254
Category I: evidence-based psychotherapies 254–260
Category III: theory-driven psychotherapies 260–262
Category IV: investigational psychotherapies 262–264
Category VIII 265
implications for practice 267
implications for research 266–267
other therapies 266
short-term psychodynamic psychotherapy (STPP): depression 22–23
skills training
borderline personality disorder (BPD) 301–302, 304–305
organizational (OST): attention-deficit hyperactivity disorder (ADHD) 442–444
see also social skills training (SST)
sleep hygiene education 480–483
sleep-related interventions: dissociation 348–349
sleep restriction therapy 469, 484–485, 495
social anxiety disorder (SAD) 71–78
Category I: evidence-based psychotherapies 72–74
Category II: intervention-driven psychotherapies 74
Category III: theory-driven psychotherapies 74–75
Category IV: investigational psychotherapies 75–77
Categories V–IX 77–78
defining features 71–72
and generalized anxiety disorder (GAD) 102
implications for research and practice 81–83
panic attacks 64
social cognitive deficits 364, 384
"social defeat hypothesis" 377
Social Functioning Scale 384
social isolation 376, 377
social network effects: Alcoholics Anonymous (AA) 236
social skills training (SST)
attention-deficit hyperactivity disorder (ADHD) 446–447
schizophrenia-spectrum disorders 380–385
social anxiety disorder (SAD) 74
"specific efficacy"
definition 12
see also change, theory and mechanisms of; *specific therapies*
specific phobia 78–81
Category I: evidence-based psychotherapies 79–81
Categories II–IX 81
defining features 78–79
implications for research and practice 81–83
speech and language training: autism 423, 428
spirituality
Alcoholics Anonymous (AA) 235
exorcism 344–345
stimulus control treatment: insomnia 470, 483–484, 495

story/narrative-based approaches 341–342, 424
"stress-induced false-positive identification of threats" 377
stress inoculation training (SIT) 172
stress-vulnerability model 46, 375–376
"strong contradictory evidence", definition 13
Stroop task, emotional 137
Structured Clinical Interview for DSM-IV
  dissociative disorders 335
  posttraumatic stress disorder (PTSD) 161
substance abuse
  and borderline personality disorder (BPD) 316
  and posttraumatic stress disorder (PTSD) 176–177
  see also alcohol use disorders (AUDs)
suicidality
  and posttraumatic stress disorder (PTSD) 159–160
  and self-injury (NSSI) 300–301, 302, 304–305, 307, 317, 318
Supported Employment: schizophrenia 392
supportive–expressive (SE) psychodynamic model of anxiety (GAD) 109–110

t

task concentration training (TCT): social anxiety disorder (SAD) 73–74
telemedicine: posttraumatic stress disorder (PTSD) 177–178
theory of change *see* change, theory and mechanisms of
therapeutic communities 283–285
therapeutic relationship/alliance 28, 367, 452
  borderline personality disorder (BPD) 309, 313, 318
  dissociation disorders 329, 339–340, 343–344, 345
"third-wave" cognitive–behavioral therapies
  depression 23–24
  see also specific therapies
thought–action fusion/defusion exercises 132–133, 134–135
thought suppression 99, 106
token economy interventions 392
transdiagnostic model of generalized anxiety disorder (GAD) 103–104
transference-focused psychotherapy (TFP): borderline personality disorder (BPD) 313–315
trauma, childhood *see* childhood trauma and attachment issues
trauma management therapy (TMT): posttraumatic stress disorder (PTSD) 169
traumatic memory *see* memory
12-step facilitation (TSF) (Alcoholics Anonymous) 235, 236, 237

v

vaginismus 257, 264
verbal–linguistic *vs* image-based quality of worry 106–107
veterans *see* military veterans
violence reduction program: cognitive–behavioral therapy (CBT) 282
virtual reality (VR) and exposure therapy: posttraumatic stress disorder (PTSD) 169–170

w

"well-supported" psychotherapy, definition 13
"White Christmas" test 377
World Health Organization
  Global Burden of Disease study: depression 11
  International Classification of Diseases (ICD) 221, 465, 466
  Quality of Life outcome measure 256

y

Yale–Brown Obsessive–Compulsive Scale (Y-BOCS) 126, 129, 131, 144
Young's Schema Questionnaire (YSQ) 311
youths *see* adolescents/youths