TREATMENTS FOR PSYCHOLOGICAL PROBLEMS AND SYNDROMES

EDITED BY DEAN MCKAY, JONATHAN S. ABRAMOWITZ, AND ERIC A. STORCH

WILEY Blackwell
Treatments for Psychological Problems and Syndromes
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Edited by

Dean McKay, Jonathan S. Abramowitz, and Eric A. Storch
For Jack Rosenberg. Your memory will always be a blessing.  
Dean McKay

Dedicated, with love, to Stacy, Emily, and Miriam.  
Jonathan S. Abramowitz

To Jill, Ellie, Noah, and Maya with love. And, to my wonderful students over the years.  
Eric A. Storch
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Notes on Editors

**Dean McKay**, PhD, is Professor in the Department of Psychology at Fordham University and past president (2013–2014) of the Association for Behavioral and Cognitive Therapies. He is President-elect (2017) of the Society for a Science of Clinical Psychology. He currently serves on the editorial boards of *Behavior Therapy, Behaviour Research and Therapy, Journal of Clinical Psychology, Journal of Anxiety Disorders, Psychiatry Research, Journal of Experimental Psychopathology, and International Journal of Clinical and Health Psychology*, and is associate editor of *Behavior Therapy* and *Journal of Obsessive-Compulsive and Related Disorders*. He has edited or coedited 16 books dealing with treatment of complex cases in children and adults, obsessive–compulsive disorder, disgust in psychopathology, and research methodology; published over 200 journal articles and book chapters; and has delivered over 250 conference presentations. He is board-certified in cognitive behavioral and clinical psychology by the American Board of Professional Psychology. Dr. McKay serves on the Scientific Council of the Anxiety and Depression Association of America, as well as on the Scientific Advisory Board of the International Obsessive Compulsive Disorder Foundation. His research has focused primarily on obsessive–compulsive disorder, the role of disgust in psychopathology, and most recently selective sound sensitivity (also known as misophonia). Dr. McKay is also director and founder of the Institute for Cognitive Behavior Therapy and Research, a private treatment and research center in Westchester County, New York.

**Jonathan S. Abramowitz**, PhD, is Professor and Associate Chair of Psychology, and Research Professor of Psychiatry, at the University of North Carolina (UNC) at Chapel Hill. He is Director of the UNC Anxiety and Stress Disorders Clinic, and a North Carolina-licensed psychologist with a diploma from the American Board of Professional Psychology. He is an internationally recognized expert on obsessive–compulsive disorder and anxiety, and has published over 250 research articles, books, and book chapters on these subjects. He is a past president of the Association for Behavioral and Cognitive Therapies and currently serves as editor of the *Journal of Obsessive-Compulsive and Related Disorders*, which he founded in 2011. Dr. Abramowitz is a regular presenter at professional conferences and has received numerous awards for his contributions to the field.

**Eric A. Storch**, PhD, is Professor and All Children’s Hospital Guild Endowed Chair in the departments of Pediatrics, Health Policy and Management, Psychiatry and Behavioral Neurosciences, and Psychology at the University of South Florida. He serves as the Director of Research for Developmental Pediatrics at Johns Hopkins All Children’s Hospital, and is the Clinical Director of Rogers Behavioral Health–Tampa Bay, which is a partial hospitalization program oriented to individuals with significant obsessive–compulsive disorder, anxiety, and/or eating disorders. Dr. Storch has received multiple grants from federal agencies for his research (i.e., National Institutes of Health, Centers for Disease Control and Prevention), is a Fulbright Scholar, and has published over 10 books and over 500 articles and chapters. He specializes in the nature and treatment of childhood and adult obsessive–compulsive disorder and related conditions, anxiety disorders, and anxiety among youth with autism.
## List of Contributors

**Jonathan S. Abramowitz**  
University of North Carolina at Chapel Hill, United States

**Samantha Adelsberg**  
Fordham University, United States

**Margaret S. Andover**  
Fordham University, United States

**Erland Axelsson**  
Karolinska Institutet, Sweden

**Catherine R. Ayers**  
VA San Diego Healthcare System, University of California, San Diego

**Daniel M. Bagner**  
Florida International University, United States

**Nicole E. Barroso**  
Florida International University, United States

**Donald H. Baucom**  
University of North Carolina at Chapel Hill, United States

**Natalie Bennett**  
University of Nevada, Reno, United States

**Randi Bennett**  
Fordham University, United States

**Lorraine Benuto**  
University of Nevada, Reno, United States

**Charmaine Borg**  
University of Groningen, The Netherlands

**Emily H. Brackman**  
Fordham University, United States

**Lauren Breithaupt**  
George Mason University, United States

**Elle Brennan**  
Kent State University, United States

**Lily A. Brown**  
University of California, Los Angeles, United States

**Jacqueline R. Bullis**  
Boston University, United States

**Joseph K. Carpenter**  
Boston University, United States

**Olga Cirlugea**  
University of Nevada, Reno, United States

**Sarah W. Clark**  
Virginia Commonwealth University, United States

**Dennis R. Combs**  
University of Texas at Tyler, United States

**Christine A. Conelea**  
Bradley Hasbro Children’s Research Center, United States
List of Contributors

Laren R. Conklin  
Boston University, United States

Jesse R. Cougle  
Florida State University, United States

Michelle G. Craske  
University of California, Los Angeles, United States

Joshua Curtiss  
Boston University, United States

Kendra Davis  
University of Georgia, United States

Thompson E. Davis III  
Louisiana State University, United States

Peter J. de Jong  
University of Groningen, The Netherlands

Helen F. Dodd  
University of Reading, United Kingdom

Todd J. Farchione  
Boston University, United States

Sarah Fischer  
Potomac Behavioral Solutions, United States

Christopher A. Flessner  
Kent State University, United States

Evan M. Forman  
Drexel University, United States

Sarah K. Francazio  
Kent State University, United States

Hannah E. Frank  
Temple University, United States

Dainelys Garcia  
Florida International University, United States

Natalia M. Garcia  
University of Washington, United States

Diana Gaydusek  
American University, United States

Jonathan D. Green  
Boston University School of Medicine, United States

Shelby Harris  
Montefiore Medical Center, United States

Erik Hedman  
Karolinska Institutet, Sweden

James D. Herbert  
Drexel University, United States

Jonathan Hoffman  
Neurobehavioral Institute, United States

Stefan G. Hofmann  
Boston University, United States

Melanie A. Hom  
Florida State University, United States

Jennifer L. Hudson  
Macquarie University, Australia

Alissa B. Jerud  
University of Washington, United States

Julie Kahler  
University of Nevada, Reno, United States

Maysa M. Kaskas  
Louisiana State University, United States

Terence M. Keane  
Boston University School of Medicine, United States

Lucas S. LaFreniere  
Pennsylvania State University, United States

Michael E. Levin  
Utah State University, United States
Joanna Marino  
Potomac Behavioral Solutions, United States

Elizabeth H. Marks  
University of Washington, United States

Brian P. Marx  
Boston University School of Medicine, United States

Natalie L. Matheny  
Florida State University, United States

Tina L. Mayes  
VA San Diego Healthcare System, United States

Barbara S. McCrady  
University of New Mexico, United States

James P. McCullough Jr.  
Virginia Commonwealth University, United States

Eleanor McGlinchey  
New York State Psychiatric Institute, United States

Dean McKay  
Fordham University, United States

Kim T. Mueser  
Boston University, United States

Yolanda E. Murphy  
Kent State University, United States

Michelle G. Newman  
Pennsylvania State University, United States

William T. O’Donohue  
University of Nevada, Reno, United States

Rachel Ojserkis  
Fordham University, United States

Christine Paprocki  
University of North Carolina at Chapel Hill, United States

Ronald M. Rapee  
Macquarie University, Australia

Shireen L. Rizvi  
Rutgers University, United States

Amy K. Roy  
Fordham University, United States

Paige M. Ryan  
Louisiana State University, United States

Shannon Sauer-Zavala  
Boston University, United States

Ki Eun Shin  
Pennsylvania State University, United States

Eric A. Storch  
University of South Florida, United States

William Taboas  
Fordham University, United States

Margot C. Thomas  
Rutgers University, United States

Warren W. Tryon  
Fordham University, United States

Anna Van Meter  
Yeshiva University, United States

Michael R. Walther  
Alpert Medical School–Brown University, United States

Eric Youngstrom  
University of North Carolina at Chapel Hill, United States

Lori A. Zoellner  
Florida International University, United States
The field of mental health treatment has reached a point of maturity such that most major behavioral and psychological problems now have empirically supported interventions available for application. These treatment packages have been derived from conceptual models of psychopathology that draw on basic experimental and clinical research. Available treatment packages, usually made available through treatment manuals developed and tested for particular disorders listed in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013), typically include multiple specific interventions. Yet, it is not always clear which components are essential and which are potentially less critical to good outcomes. Moreover, it might not be clear which components target which mechanisms of psychopathology. Accordingly, there might be insufficient guidance for clinicians when it comes to choices in treatment delivery; for example, when time constraints require use of the most essential components of an existing protocol, or when the presentation of a certain condition is more complicated than, or deviates from, descriptions and illustrations in treatment manuals. Further, it is conceivable that incorporating less effective treatment elements may actually hinder individual progress toward achieving wellness. Addressing these and other clinical conundrums can be challenging without clear and concise guidance that is based on the latest empirical research.

Accordingly, we have assembled this book to help the practicing clinician to more easily identify mechanisms that best explain observed psychopathology and then apply the appropriate empirically supported processes of change. Such an approach allows the clinician to practice as an evidence-based practitioner even when they may need to deviate from disorder-based treatment manuals. This approach also raises the question of whether traditional psychiatric diagnosis (i.e., based on the DSM-5) is necessary—a growing controversy in the mental health field. That is, if one conceptualizes psychopathology at the level of the individual mechanisms and processes, and then applies empirically supported techniques to reverse such mechanisms, what advantage is there to using diagnostic labels such as those in the DSM? It is our opinion that empirically supported practice begs a critical discussion of (a) mechanisms of psychopathology, (b) mechanisms of psychological change, and (c) a means for conceptualizing presenting behavioral and psychological problems and developing treatment plans that rely on valid perspectives unmoored from the current nosology.

This book was developed at an interesting time in the evidence-based practice movement. It has been just about 20 years since the standards for...
determining what counts as an empirically supported treatment were developed (Chambless & Hollon, 1998). These criteria stipulated that a minimum of two randomized controlled trials (RCTs) be conducted by two different research teams, and show efficacy for a treatment, compared to a placebo intervention, in order for the protocol to be declared empirically supported. The full set of guidelines was considered path-breaking at the time, as this was the first time any set of standards was articulated to guide practitioners in making treatment decisions. At the time these standards were developed, RCTs were comparably rare, with few studies comparing to attention-placebo control conditions.

Now, close to 20 years later, RCTs are conducted with far greater frequency. Online registries have been developed where investigators can register their trials *a priori*, with primary and secondary variables of interests declared. Many journals require that RCTs submitted for publication be registered in order to be considered. The virtue of these registries is that it allows other investigators to evaluate the full corpus of available research, including those that might be null findings that never made it to publication, in order to have a complete account of the efficacy of a treatment protocol. Given that the criteria for empirically supported treatments were silent on the matter of unpublished or null findings, a protocol could be declared empirically supported if it met the two RCT criteria, even if there were numerous failed prior trials. This problem has been addressed in the newly crafted criteria for empirically supported treatments (ESTs; Tolin, McKay et al., 2015; Tolin, Forman, et al., 2015). There was an incremental movement already underway to deal with this as evidenced by the ubiquity of meta-analyses for specific treatment protocols, and the advent of the Cochrane reviews, which surveys in comprehensive detail the effects of specific treatment programs. As a result, we are now at the point where many treatments are fairly well understood with respect to their benefits and limitations and the components that are essential ingredients.

Understanding what treatment elements are essential ingredients is the essence of evidence-based practice, whereby direct service clinicians can select components of treatment that are deemed scientifically supported for specific problems faced by their clients.

The aims of this volume are therefore threefold. The first is to shed light on both the empirically supported and the unsupported components of conceptual models of psychopathology. Second, the volume aims to identify empirically supported components of existing psychological interventions and the rationales for how multicomponent treatments are sequenced. Thus, this text provides clinicians with an understanding of the sequential nature of interventions, and the criteria for moving from one intervention to the next, particularly for seemingly disparate treatment procedures that form multicomponent treatment packages. The third aim is to illustrate specific ways of identifying mechanisms of psychopathology that might attenuate treatment outcome with established protocols, and help the clinician use empirically supported methods to address these obstacles.

All chapters in the book draw on available research evidence to make clear the connection between science and practice; and these chapters are organized into five sections. The first section offers an overview, and outlines the aims and scope of the text, as well as a brief history of the empirically supported practice movement. The second section addresses the three aims of the book as they relate to conditions for which there is extensive support for mechanisms of psychopathology and empirically supported psychological treatment procedures and processes of change. Given the unique complexities and extensive research base, two chapters cover treatments for psychopathology emerging from traumatic events. This is an important aspect of the book given the various controversies around the possible risk for dropout with evidence-based therapy for trauma (Imel et al., 2013). The third section covers areas of psychopathology and treatment for which there is emerging empirical support. The fourth section covers
domains of psychopathology for which there is only preliminary—or perhaps the potential for—evidence-based approaches to psychopathology and treatment. The fifth and final section focuses on mechanisms of psychopathology and change across the age span.

To further orient the reader, each chapter follows a general format in which the nature of the psychopathology is first discussed. The focus is on components of relevant conceptual models, including an appraisal of their scientific support, rather than a review of major etiological theories. Next, each chapter turns to a discussion of empirically supported treatment components, including the sequencing of multicomponent interventions and the factors that can interfere with implementing these procedures. Finally, each chapter includes a discussion of how mechanisms of the psychopathology itself might interfere with treatment outcome, and how clinicians might adapt therapy to address these mechanisms and optimize treatment effectiveness. It is our hope that this edited text provides the field with a handbook for understanding the nature and treatment of psychopathology at the level of psychological mechanisms, with the broader aim of helping the field evolve from a focus on multicomponent treatment manuals for “mental disorders” to a more conceptually oriented process-based approach.

References


Cognitive behavioral therapy (CBT) has shown efficacy for a range of psychiatric disorders across age groups and populations (Butler, Chapman, Forman, & Beck, 2006; Hofmann, Asnaani, Vonk, Sawyer, & Fang, 2012; Hollon, Stewart, & Strunk, 2006; Reinecke, Ryan, & DuBois, 1998; Stewart & Chambless, 2009). CBT can generally be administered over a limited number of sessions, leads to broad improvements in functioning, and does not come with the side effects of many medications or the high relapse rates associated with their discontinuation. Yet, despite its established efficacy, the best available evidence indicates that most individuals with a psychiatric disorder do not receive CBT (Wang et al., 2005). Also troubling is the fact that, among those who receive professional psychotherapy, CBT or other evidence-based treatments (EBTs) are rarely used (Wang et al., 2005).

Given this gap between science and practice, researchers have increasingly turned their attention toward the promotion and dissemination of CBT (Shafran et al., 2009). Organizations such as the Association for Behavioral and Cognitive Therapies (ABCT) have made the advocacy of EBTs, including CBT, one of their primary missions. Further, a handful of efforts have been made to disseminate CBT broadly through top-down institutional policies, including the Improving Access to Psychological Therapies program in England (Clark, 2011) and evidence-based training initiatives by the Veterans Health Administration (VHA; Karlin, Brown, et al., 2012; Karlin, Ruzek, et al., 2010).

The purpose of this chapter is to review issues related to the dissemination of CBT, including barriers and potential solutions. Because barriers occur on multiple levels, the possible leverage points are many and diverse (Harvey & Gumport, 2015). Dozens of essays have been written proffering strategies for the dissemination of CBT; we will assess these as well as propose some solutions toward this aim.

Barriers to the Use of Cognitive Behavioral Therapy in Psychotherapy Practice

Therapist Barriers

Despite the strong empirical support for its efficacy, many therapists do not use CBT. The prevalence of its reported use varies widely across studies, with some studies painting a bleaker picture of the situation than others. For example, one study found that, among those with bulimia nervosa who had received previous psychotherapy, only 6.9% indicated they received CBT (Crow, Mussell, Peterson, Knopke, & Mitchell, 1999). In contrast, a survey of psychologists who treat eating disorders found that 39% endorsed CBT as their primary treatment
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approach (Mussell et al., 2000), while a majority (65%) of the sample indicated that they used CBT techniques “always” or “often.” Another investigation found that fewer than 20% of psychologists reported using exposure therapy for post-traumatic stress disorder (PTSD; Becker, Zayfert, & Anderson, 2004), and supportive counseling was cited as the therapy most often used for PTSD in a separate study (Ehlers, Gene-Cos, & Perrin, 2009).

Even among those who receive CBT, its delivery is often suboptimal. For example, one study examined self-reported treatment history in a small sample of individuals with obsessive–compulsive disorder (OCD). Among those who reported they had previously received CBT (40% of the sample), the procedures used met minimal criteria for adequacy in only a minority of cases (Stobie, Taylor, Quigley, Ewing, & Salkovskis, 2007). Additionally, though surveys have found that the vast majority of therapists report using CBT to treat anxiety disorders (Freiheit, Vye, Swan, & Cady, 2004; Hipol & Deacon, 2013), these same respondents indicated they rarely used therapist-assisted exposure. For instance, only 22% were found to use interoceptive exposure “sometimes” or “frequently” to treat panic disorder (Freiheit et al., 2004). An analysis of therapists who reported using CBT for eating disorders found that less than 50% of the sample used at least one core CBT technique consistently (Waller, Stringer, & Meyer, 2012).

Many reasons have been proposed for the non-use of CBT by therapists in the community. Perhaps the most important is that many therapists have not received adequate training in CBT. For example, one study found that only 20% of PsyD and 21% of social work programs required supervision in CBT, though most (PsyD 96%; social work 80%) required didactics in CBT (Weissman et al., 2006). It is noteworthy that these programs produce far more therapists that ultimately practice in the community than clinical psychology PhD programs.

Therapists also appear to hold negative beliefs about CBT and other EBTs that are typically unjustified. For instance, some report skepticism regarding the findings of randomized controlled trials of CBT and believe such trials do not include the types of “real-world” patients they see in their practice (Shafran et al., 2009). Others view EBTs as potentially harmful to the therapeutic relationship and believe that therapy manuals ignore and are inflexible to individual client needs (Addis, Wade, & Hatgis, 1999). Some therapists also prefer a more eclectic approach to CBT, where only select CBT skills or principles are integrated into their therapeutic framework. Additionally, therapists generally believe that they are already using effective treatment methods and are less open to using CBT for this reason (Shafran et al., 2009; Stewart, Stirman, & Chambless, 2012). Lastly, many report not having the time or financial resources to receive additional training in CBT or other EBTs (Stewart et al., 2012).

Institutional Barriers

The Diversity of Therapists Delivering Services in the Community

Much of the research reviewed on therapist attitudes toward CBT and its use surveyed PhD-level psychologists; however, master’s-level counselors are more likely to be delivering therapy. These counselors tend to use CBT less often and are less equipped to do so (Addis, 2006; Addis et al., 1999). The diversity of service providers responsible for therapy delivered in the community challenges the dissemination of a single set of therapy techniques (e.g., CBT).

Within universities, psychology departments are generally CBT-friendly (Weissman et al., 2006), though more practicing therapists are trained in schools of education or social work. Unfortunately, there is little incentive for cross-area collaborations, let alone the promotion of CBT across departments. Psychology departments that offer courses and supervision in CBT often limit these courses to students in their department. Other departments are more likely to emphasize training and supervision in non-EBTs (Weissman et al., 2006). As a result, many graduate students who wish to receive training in CBT may not have access to it.
Accreditation Bodies and Funding Agencies
Therapist accreditation typically occurs on the state level in the United States. To date, there has been little willingness on the part of state licensing agencies and larger organizations such as the American Psychological Association to embrace evidence-based standards of care (Baker, McFall, & Shoham, 2008). Further, insurance companies tend to prioritize the cost of care over its quality; thus, many have increasingly looked to master’s-level therapists—who may not have received training in EBTs—to administer treatment at a lower cost relative to doctoral-level providers. Consequently, there has been little incentive for mental health service providers to learn or practice CBT.

Patient Barriers
Harvey and Gumport (2015) recently listed multiple patient barriers that inhibit the dissemination of CBT in the community. Some barriers involved practical challenges, including lack of financial resources or childcare, which would be required to attend sessions. Furthermore, many who need treatment live in remote areas with few or no therapists, and many are unaware of the existence of EBTs for psychiatric disorders. Additionally, it may be difficult for patients to identify therapists who are truly proficient in delivering EBTs, including CBT. This can be burdensome to patients, since, as reviewed earlier, many therapists indicate that they practice CBT, though they often provide only suboptimal delivery (Freiheit et al., 2004; Stobie et al., 2007; Waller et al., 2012). How can patients discern adequate from inadequate CBT? Some additional patient barriers exist, which are reviewed below.

Client Outcomes May Extend beyond Symptom Reduction
Clinical psychologists and advocates of evidence-based practice focus primarily on symptoms of DSM-5 disorders as the criteria by which therapy must be evaluated via randomized controlled trials. This is a reasonable focus, given the impairment, disability, and other severe consequences (e.g., suicide, unemployment) associated with symptoms of psychiatric disorders, as well as the limited resources available to fund medical care. However, many individuals, including those with psychiatric disorders, see therapists for reasons other than the alleviation of symptoms. Indeed, in one study, one-third of respondents who sought mental health treatment over the past year did not meet diagnostic criteria for any psychiatric disorder (Wang et al., 2005).

Patients may seek therapy to address dysfunctional relationship patterns or chronic procrastination. Some may feel as if they are in a “rut” or that their lives are in need of direction. Others may seek a therapist because they feel that their lives lack meaning and their jobs and relationships are unfulfilling. Many see therapists because they lack social support and someone who cares for and listens to them. They may enjoy the support and sounding board provided by many therapists who may not practice CBT. These topics are not necessarily outside the boundaries of CBT, though they are not generally the focus of CBT-oriented training. Consequently, researchers and practitioners focused on delivery of EBTs may fail to appreciate the importance of these outcomes to the clients they treat. Further, though some recent efforts by positive psychologists show promise for improving outcomes related to happiness and sense of purpose (Duckworth, Steen, & Seligman, 2005), specific interventions in this area currently lack a strong evidence base (Bolier et al., 2013).

Non-evidence-based treatments, including many insight-oriented therapies, may be attractive to patients for the sense of meaning and coherence they provide, even if they rest on pseudo-scientific or false premises. There is evidence that nostalgia and thinking about childhood memories facilitates social bonding (Wildschut, Sedikides, Arndt, & Routledge, 2006), instills feelings of moral purity, and encourages prosocial behavior (Gino & Desai, 2012). Given that CBT is present-focused and
primarily concerned with symptom reduction, it may lack this advantage. Despite the many potential negative consequences associated with non-EBTs and long-term therapies, including their lack of efficacy with regard to symptom reduction and the dependency they may foster, it is important to appreciate what they might offer to understand why patients continue to select and receive them.

Consumers Are Generally Satisfied with the Therapy They Are Given
An additional patient-level barrier to the adoption of evidence-based practice is that patients have reported high degrees of satisfaction with the therapy they receive in the community, despite the theoretical orientation of the therapist who delivers it. The oft-cited Consumer Reports Survey for 1995 (Consumer Reports, 1995) on the effectiveness of psychotherapy has several important methodological shortcomings, though its overall conclusion—that consumers are largely satisfied with the psychotherapy they receive—has not generally been disputed. A more recent poll found that, of those who received treatment for a mental health problem in the past two years, 85% were satisfied with the care they received, and 80% found it effective (Harris Interactive, 2004). Though many patients are obviously dissatisfied with the therapy they receive, and treatment that is ineffective can be harmful to those who receive it (by, at the very least, preventing them from receiving effective treatment), it is important to acknowledge that the public on the whole is mostly content (and, on some metrics, quite pleased) with the quality of therapy administered at present. This represents a significant challenge for those seeking to implement changes in standards of care at government and organizational levels, since we may lack support at the grassroots level for such changes.

Patient Preferences for Non-distressing, Easy Treatments
Standard CBT for anxiety and mood disorders typically requires a certain level of effort from patients and a willingness on their part to confront distressing thoughts and situations. Homework often involves completing worksheets or conducting different exercises. For example, repeated fear confrontation via exposure therapy is integrated into most CBT protocols for anxiety disorders. Though exposure therapy might involve a greater degree of distress than other treatments, evidence suggests they do not lead to greater attrition rates than these treatments (Hembree et al., 2003). Even so, the work and distress required by many CBT protocols may make them less attractive to many patients.

Research Barriers
Much of the push toward evidence-based psychotherapy has come from CBT researchers, and much of their focus has been on effecting change at the administrative level. These changes involve persuading many of those in power (e.g., politicians, licensure boards) to adopt certain standards for psychotherapy and provide monetary support for evidence-based training and care. While these efforts are certainly worthwhile, they also involve many factors that are largely out of these researchers’ control. However, there are multiple other areas that are more closely related to the responsibilities and work of researchers and that fall under their domains of influence.

There Are Too Many Empirically Supported Therapies
A few authors have commented on problems associated with the overabundance of therapies (Cougle, 2012; Harvey & Gumport, 2015; Weisz, Ng, & Bearman, 2014). For example, Harvey and Gumport (2015) note that this issue might make it difficult to identify which EBT to use or receive training in. They support the development and use of centralized resources, such as the American Psychological Association’s Division 12 website (www.psychologicaltreatments.org), to provide therapists and consumers with information regarding which therapies are effective. This website currently lists approximately 80 general therapies that the organization considers to be evidence-based.
Resources that provide accurate information on the many EBTs available are certainly helpful; however, the high number of existing EBTs is problematic for a few reasons. First, it can make it difficult for training programs and clinicians to determine which specific therapies to learn and use in clinical practice. Second, the vast number of therapies, along with their many different corresponding components, can also lead to quality control problems (Cougle, 2012). As a general rule, it seems more likely that we can ensure whether one specific treatment procedure is being administered adequately than that 20 different procedures (which may have been drawn from 10 different EBTs) are being administered adequately. Third, EBTs also typically come in packages with many different components, including some that have shown little to no efficacy. Indeed, one could conceivably add an EBT to the list by adding a single inert component to an established EBT and demonstrating the efficacy of this “new” therapy. Lastly, the existence of this overwhelming number of EBTs ignores real differences between EBTs that might make one better than another, including complexity, ease of implementation, and efficiency (Cougle, 2012).

Harvey and Gumport (2015) suggest that transdiagnostic or modularized therapies could help address issues related to the “too many therapies” problem, as one therapy or modularized protocol could potentially be used for many different clients with different diagnoses and clinical presentations. Although a handful of transdiagnostic treatment protocols have been developed (e.g., Barlow et al., 2010), it is not yet clear what the active ingredients of these treatment packages are or whether they represent an advance over existing treatments (Norton, 2012). Additionally, it is quite possible that a large list of disorder-specific therapies could similarly be replaced by a large list of transdiagnostic therapies.

Cognitive Behavioral Therapy Is Not Effective Enough

When studied in randomized controlled trials, CBT generally outperforms other treatments, especially for anxiety disorders (Hofmann & Smits, 2008; Tolin, 2010). Effectiveness studies that examined CBT in real-world, less controlled community settings have also demonstrated impressive outcomes in favor of CBT (Stewart & Chambless, 2009). However, some evidence indicates that CBT may decrease in efficacy as it moves from research settings to community practice (Weisz et al., 2014). For example, one recent meta-analysis found EBTs, including CBT, had only modest benefits (mean effect size = 0.29) over usual care for youths (Weisz, Ugueto, Cheron, & Herren, 2013). It is noteworthy that effectiveness studies typically involve extensive training and continuous supervision of community therapists by CBT experts. The dose and efficacy of CBT may drop substantially once these therapists are no longer receiving supervision and the interventions are removed from the control of its developers.

Despite encouraging evidence for the efficacy of CBT for anxiety disorders, research suggests that CBT is not more effective than other psychotherapies for depression. This is reflected in the British government’s evidence-based guidelines for the treatment of depression (National Institute for Health and Clinical Excellence, 2009), which included CBT alongside a range of other psychotherapies (e.g., brief dynamic therapy, interpersonal therapy, counseling) as recommended treatment modalities. Effectiveness studies on psychotherapy for depression have found no advantage of CBT over usual care with regard to symptom reduction (e.g., Weisz et al., 2009). Additionally, a meta-analytic review found that peer support was as effective as CBT for the treatment of depression (Pfeiffer, Heisler, Piette, Rogers, & Valenstein, 2011).

The fact that many therapies show equivalent efficacy for depression is both comforting and challenging. It is comforting in that it suggests that many depressed patients who are in therapy are receiving adequate, effective care, even if it is not from an evidence-based practitioner. It is challenging in that it makes it more difficult to argue for widespread adoption of certain evidence-based standards of care. Indeed, given
that clients very often present with depression as a primary or secondary complaint, many if not most therapists in the community—even those who are not amenable to CBT and EBTs—can already claim to be delivering “evidence-based” treatment.

The Absence of Dissemination Research
Researchers have bemoaned the lack of knowledge regarding the most effective methods for the dissemination of EBTs (McHugh & Barlow, 2010; Weisz et al., 2014). Training in CBT is often completed haphazardly and lacks a clear evidence base (Rakovshik & McManus, 2010). Little is known regarding the most effective and efficient methods for training therapists to achieve proficiency in CBT. To date, clinical researchers have focused primarily on improving the understanding and treatment of mental illness and have been less concerned with training methods and dissemination research.

Weisz et al. (2014) recently proposed several possible strategies for the advancement of dissemination research. Among their suggestions were: (a) shifting intervention research toward resembling the context of community practice; (b) resolving the problems of “too many therapies” by applying more stringent standards for what constitutes evidence-based treatment; (c) developing more efficient and accessible models of evidence-based care (e.g., using paraprofessionals, telehealth, self-help books); and (d) creating systems to monitor client responses to treatment and to provide feedback to clinicians.

Psychologists may be reluctant to conduct dissemination research because they see it as outside their purview. They may lack adequate training on research methods related to these topics. Additionally, research in this area, such as identification of the best training methods for therapists, is likely quite costly and labor-intensive. Even so, dissemination-oriented research holds much promise and is an important next step for advancing the widespread adoption of CBT and other EBTs.

Potential Solutions

Top-Down Solutions: Lessons from the United Kingdom
Worldwide, important efforts to disseminate EBTs have been made at the government level. Among the most noteworthy is the aforementioned Improving Access to Psychological Therapies (IAPT) program in the United Kingdom (Clark et al., 2009). The generously funded IAPT program trained nearly 3,600 therapists in EBTs for anxiety disorders and depression, and then employed them in services devoted to EBTs (see Clark, 2011). Early evaluations of this program have been encouraging, with 40.3% of patients reaching reliable recovered status at post-treatment and 63.7% achieving reliable improvement (Gyani, Shafran, Layard, & Clark, 2013). Unfortunately, outcomes were assessed without a control group comparison (e.g., usual care); thus, improvements due to natural recovery or non-specific therapy effects could not be ruled out. Interestingly, researchers were able to compare outcomes for IAPT-trained therapists who used CBT with those for counseling, and found that CBT was associated with better outcomes for generalized anxiety disorder, though both treatments produced comparable recovery rates for depression (Gyani et al., 2013).

While it would be difficult to implement an IAPT-style program in the United States because differences in these two countries’ healthcare systems, several important lessons regarding pathways to dissemination of EBTs can be drawn from England’s example. For instance, proponents of IAPT argued that EBTs were not currently available to a majority of the population, thereby underscoring a need for increased training of providers. Additionally, economists and psychologists maintained that the costs associated with improving access to EBTs would be recovered in savings from the reductions in disability and unemployment that would be achieved through the initiative. Indeed, recent data suggest that the initiative
led to improvements in these outcomes (Community and Mental Health Team, 2014). Furthermore, though the scope of IAPT was on dissemination of EBTs for anxiety disorders and depression, data from Gyani et al. (2013) suggest that efforts could have focused only on EBTs for anxiety disorders to achieve desired outcomes (though the broader focus may have made more sense politically). As more data emerge from the IAPT program, new dissemination efforts will be able to build on these and other lessons.

Focus Dissemination Efforts on Treatment Components rather than Treatment Packages

As reviewed earlier, while many therapists in the community report that they administer CBT, its delivery is often suboptimal (Freiheit et al., 2004; Stobie et al., 2007; Waller et al., 2012). Treatment packages typically have many components that have not been tested in isolation and whose use has little to no empirical support. The number and complexity of components included in a treatment can negatively impact dissemination efforts, making it more difficult to train therapists and ensure that they are administering the treatment as intended (Cougle, 2012). From a patient’s perspective, it is also difficult to be confident that therapists who claim to be proficient in CBT or other EBTs are administering them optimally. An increased focus on specific, simpler, effective treatment components, such as exposure therapy for anxiety disorders or behavioral activation for depression (Mazzucchelli, Kane, & Rees, 2009), could increase the likelihood that EBTs are administered adequately. Such efforts could also assist consumers in determining whether they are receiving acceptable treatment by simplifying and decreasing the overwhelming number of options available to them.

Direct-to-Consumer Strategies

Direct-to-consumer marketing research for psychotherapy has received recent attention (Gallo, Comer, & Barlow, 2013; Gallo, Comer, Barlow, Clarke, & Antony, 2015) and could be beneficial for increasing awareness of EBTs. Given the difficulties inherent in changing therapist behavior, as well as problems associated with therapist claims regarding CBT that they administer, empowering patients through education on effective treatments for psychological disorders represents an attractive option. Some organizations, including the International OCD Foundation (www.ocfoundation.com), have worked to increase consumer awareness about EBTs (Szymanski, 2012). Furthermore, the promotion of self-help books and popular lectures incorporating descriptions of EBTs may also aid these efforts. Many resources on effective treatments are available online. We were pleased to find that current Wikipedia entries for several psychiatric disorders (e.g., panic disorder, OCD) provided accurate, fairly detailed descriptions of CBT for these disorders. Additionally, instructors should take advantage of the fact that a large portion of the public attends general psychology and abnormal psychology classes at colleges and universities; these classes represent ideal settings in which to educate the public on what is and is not an EBT. Education on EBTs could also be incorporated into high school psychology classes and mandatory health instruction. While these strategies will not directly address problems associated with the lack of EBTs in the community, increasing awareness and knowledge of EBTs will give assurances to patients that the care they are receiving is adequate and may increase the demand for EBTs (and consequently their supply).

Exploring Other Mechanisms of Treatment Delivery

Developing and evaluating novel methods of treatment delivery may also help increase access to evidence-based care (Kazdin & Blase, 2011). A number of computerized treatments for depression and anxiety have yielded promising findings (e.g., Amir & Taylor, 2012; Williams, Blackwell, Mackenzie, Holmes, & Andrews, 2013).
Computer-assisted programs for CBT (e.g., Craske et al., 2009) have the potential not only to increase access to care but also to ensure the fidelity of CBT being provided. Self-help books, Internet sites, smartphone and tablet applications, and paraprofessional-administered EBTs could also help disseminate EBTs. Continued exploration of these and other intervention methods could significantly expand the possibilities for innovation and growth in this area.

Concluding Remarks

We have discussed many barriers to the dissemination of CBT and other EBTs. Others have discussed many additional barriers that we were not able to touch on, including pseudo-scientific beliefs held by both therapists and patients (Lilienfeld, Ritschel, Lynn, Cautin, & Latzman, 2013). In closing, we aim to impress that, although some degree of movement toward dissemination of evidence-based care may occur through changes made to government and accreditation board policies, we suspect that widespread adoption of EBTs in clinical practice is unlikely to come through top-down policies alone. Rather, changes must be made at multiple levels and will require investment from various stakeholders, including national organizations, researchers, therapists, and patients. While there are many challenges associated with the movement toward widespread adoption of evidence-based practice, we hope that the strategies discussed here might help to advance the field’s efforts in the dissemination of EBTs.

References


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Fear is a part of healthy development, and psychological mechanisms are in place beginning at birth to allow individuals to detect and avoid threat. Human beings have evolved to attend to frightening stimuli or environments that may impact survival through thousands of years of repeated classical conditioning paradigms (Ohman & Mineka, 2001). Although some level of fear can aid in survival or motivate individuals to prepare and perform, not all fears are healthy. Some fears can be intense and irrational; they may become chronic in nature and limit an individual’s ability to function. For instance, a child with a severe fear of storms may experience concentration difficulties at school when it rains or may miss out on important social opportunities if he or she is afraid to leave the house in case of a storm. What differentiates healthy, adaptive fear from detrimental, maladaptive phobia?

The Nature of the Problem

Diagnostic Criteria

The Diagnostic and Statistical Manual of Mental Disorders (5th edition [DSM-5]; American Psychiatric Association, 2013) describes specific phobia as an anxiety disorder that involves a characteristic, unreasonable fear response which has a significant negative impact on daily living. In order to be diagnosed with a specific phobia, an individual must exhibit a marked fear or anxiety about a specific stimulus, situation, or environment. When encountering the feared stimulus, the individual must respond in an excessive manner compared to the posed risk, situation, or socio-cultural context and often avoid or endure the stimulus with severe distress. This fear must almost always occur in the context of the stimulus, occur for six or more months, and cause impairment in the individual’s functioning (DSM-5). The diagnostic criteria allow for some differences in children; for example, children may respond to a feared stimulus by throwing a tantrum, crying, freezing, or clinging to a caregiver. Additionally, children may not be able to understand the irrationality of their fears or beliefs while adults are typically able to recognize the excessive nature of their response. The DSM-5 recognizes five subtypes of specific phobia: animal, natural environment, situational, blood–injury–injection (BII), and other. Interestingly, each phobia subtype has a slightly different mean age of onset, prevalence rate, and epidemiology (Fredrikson, Annas, Fischer, & Wik, 1996; Öst, 1987; Van Houtem et al., 2013).

Age of Onset

The majority of specific phobias begin in middle childhood. As children develop, their most common fears often change to mirror their cognitive
development. For example, fear of a tangible, concrete stimulus, such as a dog, may transform into abstract concepts, like performance fears, later in life (Davis, Ollendick, & Öst, 2009). An influential study by Öst (1987) gathered a sample of 370 adults with specific phobias and determined the mean age of onset for particular phobia subtypes. Consistent with theories of child development, animal phobia had the earliest age of onset (7 years), followed by BII phobia (9 years), dental phobia (12 years), and social phobia (16 years). Claustrophobia, the fear of not being able to escape from confined spaces, had the latest age of onset (20 years). The prevalence rates of these specific phobias also vary by culture, age, gender, and subtype.

Prevalence

Specific phobias are relatively common, affecting an estimated 15% of the population. Ten percent of individuals suffer from a specific phobia for their entire lifetime; however, less than 10% of these individuals ever seek services for their intense, impairing fears (Curtis, Magee, Eaton, Wittchen, & Kessler, 1998; Davis, Ollendick, & Muris, 2004; LeBeau et al., 2010; Öst, 1987). Evaluating the prevalence of specific phobia in different demographic groups yields further insights into the course of this disorder. According to the American Psychiatric Association (2013), overall prevalence rates are reportedly higher among European and American countries (6%) and lower in Asian, African, and Latin American countries (2–4%). About 5% of children worldwide (i.e., under the age of 13 years) meet criteria for a specific phobia, with a 12-month prevalence of 16% (American Psychiatric Association, 2013). Kessler, Petukhova, Sampson, Zaslavsky, and Wittchen (2012) recently estimated the prevalence of specific phobia to be 20% in adolescents (aged 13–17 years), with the rates being higher in females than in males (23% and 17%, respectively). A study on college-age students found that approximately 5% to 20% reported suffering from significant fears, varying by subtype (Seim & Spates, 2009). The prevalence in adults is lower, at approximately 13.8% (17.5% in females, 9.9% in males); however, this may be an underestimation, as many adults underreport their fears (Kessler et al., 2012).

Prevalence rates also differ with phobia subtypes and gender. Approximately 75% to 90% of individuals presenting with an animal, natural environment, or situational phobia are female. Additionally, animal-type phobias have been found to be the most common specific phobia in women, while situational phobias (i.e., heights) have been found to be the most common specific phobia in men (second most common in women; Muris, Schmidt, & Merckelbach, 1999). The gender difference is not as pronounced in the BII phobia subtype, with 55% to 70% of reporters being female (Curtis, Magee, Eaton, Wittchen, & Kessler, 1998). While the literature suggests that females suffer from phobias at greater rates than males, it is important to consider the effect of cultural expectations and the tendency of males to underreport symptomology (Muris, Schmidt, & Merckelbach, 1999). These findings collectively suggest that phobias are one of the most common psychological disorders (if not the most common), making it imperative that mental health professionals understand empirically supported components and treatments designed to combat this chronic and impairing fear (Van Houtem et al., 2013).

Comorbidity

Specific phobias are highly comorbid, with rates of additional diagnoses ranging from 60% to 75% (American Psychiatric Association, 2013; Kim et al., 2010; Silverman et al., 1999), and garnering the disorder status as a “gateway” disorder to other anxiety problems (Davis, White, & Ollendick, 2014). Overall, the most common comorbid conditions include additional phobias (19%–75%; while women are overall more likely to have more than one specific phobia, the range depends on subtype), separation anxiety disorder in children (16%), and attention deficit hyperactivity disorder (ADHD; 6%) (American Psychiatric Association, 2013; Silverman et al., 1999).
Furthermore, comorbidity differs by subtype; the natural environment and animal phobia subtypes are more strongly associated with other anxiety disorders, the animal subtype is most associated with oppositional defiant disorder, and the BII subtype is most associated with ADHD (Kim et al., 2010).

The Origins of Specific Phobias

Tripartite Model of Fear

Fear is often associated with specific, physiological responses that people later consciously label as fear (Lang, 1979). These physiological responses often include increases in gland activity (e.g., sweating, release of norepinephrine, epinephrine, or adrenaline), heart rate (e.g., feeling like one’s heart is racing or pounding), and breathing rate (e.g., taking quicker, shallower breaths) or what may otherwise be labeled a panic attack. However, the BII subtype is often accompanied by a very different physiological response. Individuals with this specific phobia may display vasovagal syncope, which is characterized by an initial increase in blood pressure (similar to that experienced with other stimuli) followed by a decrease in blood pressure and a slowed heart rate; this response can lead to fainting (Ayala, Meuret, & Ritz, 2010; Öst, 1992; Vossbeck-Elsebusch, Steinigeweg, Vögele, & Gerlach, 2012).

Fear is further marked by behaviors such as avoidance; avoidance maintains phobias by preventing the extinction of fear responses (Lang, 1979). Once individuals develop specific phobia, they either endure the feared stimuli with great distress or avoid the stimuli entirely. Avoidance prevents individuals from having positive or neutral experiences with the object of their fear, allowing for negative experiences or misconceptions to persist (Davis et al., 2012). These behaviors maintain specific phobias through negative reinforcement, as individuals are rewarded with the removal of the aversive or feared stimuli through avoidance (e.g., Mineka & Zinbarg, 2006; Mowrer, 1960).

Lastly, fear is experienced cognitively, as individuals access memories, thoughts, and cognitions associated with the stimuli they experience (Lang, 1979). Often, this experience involves activation of catastrophic cognitions—thoughts about the worst outcomes of the feared stimuli, environment, or situation (e.g., “That dog is going to bite my arm off if it gets any closer”; “This storm is going to turn into a tornado, and it will rip my house apart”; “This elevator is going to break, and we will all plummet to our deaths”). According to the tripartite model, physiological responses, behaviors, and cognitions all interact to produce the fear response. These components may occur together with the same intensity or in varied intensities. Recent research is attempting to understand the characteristic differences between individuals with distinct patterns in physiological, behavioral, and cognitive experiences of fear (Davis, Hurley, & Ollendick, 2003; Ollendick, Allen, Benoit, & Cowart, 2011).

Heritability of Specific Phobias

The genetic components of fear and phobias have been documented in the literature for decades, though largely based on animal research. For example, Saavedra-Rodriguez and Feig (2013) bred rats that were exposed to a stressful event in their youth with healthy rats. The resulting offspring for the following three generations exhibited significant limitations in functioning, including social instability and elevated anxiety. This phenomenon was particularly prominent in the female offspring. These results suggest that the experiences of the individuals, as well as their parents’ and (even) grandparents’ early experiences, can affect symptoms of fear and anxiety.

A recent meta-analysis captures variances in the heritability of phobias (Van Houtem et al., 2013). The variance was separated into two distinct components: the additive component is a summation of dominant genes present, while the non-additive component involves epigenetics (gene–environment interaction) when assessing the strength of alleles that are passed on. Recent
research has divided the variance in prevalence to shared familial environment and general, non-shared environment. Overall variance estimates of strictly additive genetic contribution have ranged from .02 to .71, depending on the type, with the average around 30% (Czajkowski, Kendler, Tambs, Røysamb, & Reichborn-Kjennerud, 2011; Distel et al., 2008; Van Houtem et al., 2013). While psychological difficulties are understood to run in families, many people do not realize that the physiological responses of psychopathology are also highly heritable. One example is the previously discussed vasovagal syncope (seen in the BII subtype). Evidence suggests it is more common in people with close relatives with the same response to needles (Ellinwood & Hamilton, 1991; Kleinknecht & Lenz, 1989; Öst, 1991).

Environmental Attributes of Fear Acquisition

As previously discussed, much of the literature has concluded that specific phobias (and most anxiety disorders) run in families, partly as a result of genetics. However, fear can also be acquired via environmental factors. Theories of fear acquisition initially postulated that fear comes from a learned response, such as a classical conditioning paradigm. This explanation was popularized from the Little Albert study, in which a stimulus was linked to a frightening stimulus, conditioning a fear response to occur with each presentation of the previously neutral stimulus (Watson & Rayner, 1920). However, many people experience traumatic events (e.g., spider bites) and do not develop phobias, while many others have an intense fear of something they have never experienced before. This may be, in part, explained by biological preparedness models which theorize that some stimuli (e.g., snakes, lions) are more easily conditioned because of their historically threatening nature (Seligman, 1971). Still, many people report that they do not know the origins of their fears (i.e., “non-associative” fears; Menzies & Clark, 1993). However, many believe that non-associative frameworks should be renamed “non-specific” because the fear’s basis is unclear (Davey, 2002). Additionally, the degree to which fears are acquired by both learning paradigms and biological components may be determined by other risk factors (Marks, 2002), and it has been suggested that it is the additive effects of multiple experiences that lead to a phobia in these instances.

Direct Experience

Classical conditioning, or the formation of fears and phobias from direct experience with stimuli, has been heavily researched. One recent meta-analysis compared components of fear conditioning in participants with anxiety disorders to participants without psychological disorders (i.e., controls). During the learning phase, results suggested an increased fear response to conditioned neutral stimuli in participants with anxiety disorders compared to controls. The authors hypothesized that the difficulty of inhibiting fear and easier attribution of stimuli to fear may be characteristic of people with anxiety disorders. During the extinction phase, participants had increased discrimination compared to controls, suggesting that people with anxiety disorders are slower to extinguish fear responses (Duits et al., 2015). Taken together, it is less likely that individuals are “hard-wired” for a particular phobia or fear, but rather they inherit a tendency to more easily associate certain stimuli with fear (Mineka & Zinbarg, 2006). As a result, the issue may be how much direct experience with a feared stimulus is necessary to condition a phobic response (cf. Marks, 2002).

Modeling

Furthermore, the learned experience of fear can also extend to the family environment. For example, parents with phobias may model fear responses to their children at a young age (Bandura, 1977). Liberman and Öst (2015) observed a positive correlation between mothers’ expressed fears and “trait” anxiety in their children. Trait anxiety is the general, stable level of anxiety characteristic of the child’s personality (rather than anxiety induced by situations
or stimuli). This level of trait anxiety was a significant predictor of the child’s fearfulness. The study also found that over half (54%) of the children in the study had at least one parent suffering from a mood or psychological disorder, 46% of the children had a parent with an anxiety disorder, and 32% had a parent with a specific phobia. The prevalence of parental psychopathology further illustrates the applicability of social learning theory to specific phobias.

**Negative Information**

Similarly, a specific phobia can be learned simply through the transmission of negative information. Parents or caregivers may directly instruct a child to avoid a specific object or situation, or may provide information to the child that is overly negative. For example, a parent might tell a child to be careful around dogs because they often bite and hurt people. A recent study examined the consequences of negative information by presenting a group of children with information about a novel animal, nicknamed “the beast” (Muris, Bodden, Merckelbach, Ollendick, & King, 2003). Children either received positive or negative information about this imaginary creature, and researchers measured the level of their fear at three time points (before receiving any information about the beast, after receiving information, and at one-week follow-up). Unsurprisingly, children who received negative information about the imaginary creature had higher levels of fear than children who received positive information at the second time point. Notably, however, this level of fear was maintained through the follow-up period, suggesting that even small amounts of negative information can create and maintain fear in the absence of experience. Furthermore, this crafted fear generalized to other contexts and animals, as children who were given negative information about the dog-like beast also became more fearful of dogs and similar creatures.

Field (2006) investigated negative information by monitoring attentional biases in children. He gave children negative information about novel animals before using a probe task to see how this negative information affected their attention. Findings demonstrated that negative, frightening information about a stimulus contributes to attentional biases toward that stimulus (e.g., demonstrating hypervigilance toward threat-related cues). Field and Lawson (2008) also demonstrated that the type of verbal information (positive or negative) impacted the acquisition speed of outcomes of situations involving animals. For example, children who received no information about the animals took significantly more trials to accurately estimate the probabilities of set outcomes. At the same time, children given negative information learned the contingencies much quicker; these children also overestimated the number of negative outcomes when outcomes were consistent with the negative information received earlier. This research demonstrates how learned information might lead to cognitive biases that contribute to fear.

**Mechanisms of Change in Specific Phobias**

**Cognitions: Information Processing and Biases**

Anxious beliefs, predictions, and expectations about the feared stimulus are important factors in both creating and maintaining specific phobia. Some studies have demonstrated links between biased information processing (i.e., attentional and interpretive biases for threat-relevant information) and intensity of fear; for example, Mogg and Bradley (2006) assessed individuals with high or low levels of spider fear and found greater attentional bias in individuals with more fear of the stimulus. Notably, the same study found that attentional bias decreased with longer exposures to photographs of spiders, even in high-fear individuals; this suggests that exposure alone is effective in treating phobia (as measured by reductions in attentional bias). Research using fear-based implicit association tests (IATs; brief assessments of reaction time which measure implicit associations and biases)
further supports the efficacy of exposure to treat cognitive aspects of phobia; individuals who received a group-based exposure treatment showed marked differences after treatment and at a two-month follow-up (Teachman & Woody, 2003). However, the literature is mixed on the evidence for attentional bias; a recent study examined attentional hypervigilance in individuals with high and low injection fear and found that individuals with greater levels of fear did not orient to injection images more often than other emotional images (Armstrong, Hemminger, & Olatunji, 2013). Conversely, individuals with high fear displayed marked patterns of attentional avoidance, which was significantly related to their performance on behavioral avoidance tasks (BATs—a well-supported method of assessing phobia severity and developing treatment plans).

There is a great body of literature supporting the idea that individuals with specific phobias exhibit interpretive biases, misconstruing threat when viewing ambiguous stimuli and magnifying expectations of the likelihood and consequences of harm (Clerkin, Cody, Stefanucci, Proffitt, & Teachman, 2009; de Jongh, Muris, Schoenmakers, & Ter Horst, 1995; Jones & Menzies, 2000; Kolassa et al., 2007; Teachman, Stefanucci, Clerkin, Cody, & Proffitt, 2008; Teachman & Woody, 2003). For example, Teachman et al. (2008) found that individuals with a fear of heights grossly overestimated the height of a balcony, displayed greater implicit fear associations on IATs, and indicated more frequent and more severe fearful height-related cognitions. Similarly, individuals with spider phobia amplified the probability and consequences of being bitten by a spider (Jones & Menzies, 2000). These cognitive distortions predict avoidance, which maintains specific phobias. Therefore, researchers and clinicians should identify, monitor, and combat interpretive biases. These cognitive mechanisms may serve both as targets for intervention and as ways to assess progress through the course of treatment.

Evidence-Based Components of Treatment

Several meta-analyses and reviews on treatments for specific phobia have found significant differences in the efficacy and maintenance of treatment gains (Choy, Fyer, & Lipsitz, 2007; Davis, Jenkins, & Rudy, 2012; Davis, May, & Whiting, 2011; Wolitzky-Taylor, Horowitz, Powers, & Telch, 2008). This section reviews the components of treatments shown to be the most efficacious. A number of treatment components have been advanced, but they generally include exposure, psychoeducation, cognitive restructuring, modeling, relaxation, coping skills training, therapist praise, rewards (e.g., tangible, social), and self-monitoring (Chorpita, 2007; Hood & Antony, 2012; Ollendick & Davis, 2013). According to Chorpita (2007), effective treatment of specific phobias always includes exposure; occasionally uses therapist praise, modeling, psychoeducation, and/or cognitive strategies; and rarely entails other strategies.

Exposure

A substantial body of literature indicates that specific phobias are most effectively and efficiently treated with exposure (Choy et al., 2007; Davis et al., 2009, 2011; Ollendick & Davis, 2013; Ollendick & Muris, 2015; Wolitzky-Taylor et al., 2008). As previously discussed, specific phobias are largely maintained by subjective physiological experiences of fear, avoidance behaviors, and catastrophic cognitions. Exposure-based treatments combat these major maintaining factors through a systematic and controlled approach, as the phobic individual repeatedly experiences the feared object or situation. With repeated experiences of the feared stimulus, individuals’ physiological responses decline and stabilize while they become habituated to and eventually extinguish their fear toward the phobic stimuli. Additionally, exposure serves as a means of restructuring cognitive biases such as the over-estimation of probability and extent of harm
Fears and Specific Phobias (Choy et al., 2007). The clinician’s role in exposure-based treatment is to structure a controlled experience for the client, who should remain in the situation long enough to discover that the predicted consequences do not occur and that the individual has the ability to manage the fear and anxiety. Exposure treatments significantly outperform placebo and control conditions as well as other active treatment conditions such as cognitive restructuring, threat reappraisal, and relaxation techniques (Choy et al., 2007; Davis et al., 2012; Wolitzky-Taylor et al., 2008). Adding cognitive restructuring to exposure-based treatments does not increase efficacy; however, some evidence suggests adding this component of therapy may match client preferences, making treatment more palatable (Botella et al., 2014). Furthermore, psychoeducation (i.e., learning about anxiety, fear, and specific phobia) does not promote treatment gains in isolation; this is demonstrated by studies which examine mid-treatment (i.e., after psychoeducation and before exposure) progress (e.g., Kendall et al., 1997), suggesting that exposure is the most essential component of treatment.

Variations on Exposure-Based Treatments

Virtual Reality
Exposure can take many forms, depending on the focus of the individual’s fear. A recent meta-analysis found that treatments involving direct (in vivo) exposure to the feared stimulus outperformed alternative forms of exposure (e.g., virtual reality exposure, imaginal exposure); however, this incongruity was significant directly after treatment but not at follow-up, suggesting that alternative modes of exposure are effective in maintaining treatment gains (Wolitzky-Taylor et al., 2008). Certain feared stimuli may be difficult to simulate and control in treatment settings, making in vivo exposure challenging or impossible. Research supports the efficacy of exposure using virtual reality and, additionally, suggests high tolerability of this method (Pachana, Woodward, & Byrne, 2007; Parsons & Rizzo, 2008; Wrzesien et al., 2015). Dismantling studies looking at the treatment components in exposure therapies suggest that it is the exposure to the visual and auditory aspects of the feared stimuli that causes the change in fear immediately after treatment and at follow-up; for example, adding motion simulation to virtual reality exposure did not change the efficacy of treatment for flying phobia (Mühlberger, Wiedemann, & Pauli, 2003).

Variants on Session Structure and Spacing
Variants of exposure therapy include manipulations of the pacing of exposure within sessions and spacing of treatment sessions. An analysis by Craske et al. (2008) found no evidence supporting a difference between gradual exposure and flooding (i.e., rapid exposure to feared stimuli); however, gradual exposure is generally considered more tolerable and ethical (Davis, Ollendick, & Öst, 2009). Research suggests that the most essential component of timing is allowing the individual sufficient time to consolidate the learned experience, regardless of the number of sessions or spacing between them (Moscovitch, Antony, & Swinson, 2009). In fact, there is a growing body of evidence supporting the efficacy of a single exposure-based treatment session; this efficacy has been well documented across phobia subtypes and with youth (Davis et al., 2009, 2012; Ollendick et al., 2009, 2015; Ollendick, Öst, Reuterskiöld, & Costa, 2010; Waters et al., 2014) and adults (Choy et al., 2007; Davis et al., 2012; Öst, 1989; Zlomke & Davis, 2008).

Eye Movement Desensitization and Reprocessing (EMDR)
As exposure is a critical component of effective treatment of specific phobia, it is not surprising that many types of treatment include some form of exposure. Therefore, it is important to consider the most essential mechanisms in order to preserve treatment efficacy and increase efficiency. One treatment modality which includes exposure exercises is EMDR; clients are instructed to
engage in brief imaginal exposures while participating in therapist-guided rapid eye movements. Existing literature suggests that EMDR is less effective than in vivo exposure (Cahill, Carrigan, & Frueh, 1999; de Jongh & Broeke, 2009; Muris & Merckelbach, 1999). Also, a recent meta-analysis found no incremental effect of adding eye movements, concluding that any efficacy in the treatment of specific phobias with EMDR can be attributed to exposure (Davidson & Parker, 2013), and previous reviews and studies have found that other, more direct, forms of exposure therapies outperform EMDR (e.g., Davis & Ollendick, 2005).

Applied Tension (AT) and Applied Relaxation (AR)
AT and AR are also variations of exposure-based treatment, designed to address the vasovagal response seen in some individuals with BII. With AT clients are taught to contract their muscles when exposed to the target of their fear; this muscle tension elevates blood pressure, which in turn reduces the likelihood of syncope. AR takes a slightly different approach, with phobic individuals instructed to use progressive muscle relaxation during exposures, bypassing the vasovagal response. A recent analysis compared several treatment conditions in order to evaluate the additive effects of tension and relaxation: exposure alone, tension alone, AR, and a combination of AR and AT. Researchers found different effects for different outcome variables, with AT outperforming other conditions on measures of self-reported anxiety and observations of in-session avoidance and fainting, while exposure alone surpassed other conditions in terms of effect sizes on phobia-specific self-report questionnaires. Contrary to expectation, BII-phobic individuals with and without a history of fainting appeared to respond similarly to AT (Ayala, Meuret, & Ritz, 2009). Notably, Öst, Fellenius, and Sterner (1991) found that the practice of muscle tension alone (i.e., even without sustained exposure to feared stimuli) was effective in significantly reducing symptoms and maintaining treatment gains at follow-up, suggesting that AT should be considered an efficacious treatment for BII phobias.

Additional Components of Treatments for Specific Phobias
Psychoeducation
Psychoeducation is a common component of many psychological interventions and can take on many forms. Psychoeducation often serves as a therapeutic tool, as clients learn about their symptomatology (e.g., how fear is the body’s reaction), develop expectations about treatment, and build rapport with their clinicians. When treating specific phobia, psychoeducation can double as a mechanism for correcting problematic beliefs, assumptions, and expectancies; for example, a client with a phobia of dogs may hold the false belief that a dog’s jaw can lock or “latch on” if biting. Psychoeducation may also include the skills training needed to prepare the client for exposure sessions (e.g., how to hold a snake). In other words, clinicians conducting treatment for specific phobia should be prepared beyond knowledge of the disorder; clinicians should also be familiar with the details of the feared stimulus and be prepared to combat misinformation about the stimulus (Davis et al., 2009). In this sense, psychoeducation appears to be an essential component of treatment, as it lays the foundation for intervention. Ollendick et al. (2009) evaluated the empirical support for psychoeducation by randomizing 196 youth to either a one-session exposure treatment (3 hours of exposure treatment), a psychoeducational package (3 hours of psychoeducation without exposure exercises), or a waitlist. Both active treatment conditions were superior to the waitlist control condition (though active exposure was superior to psychoeducation). A similar study on panic disorder indicates that psychoeducation alone is sufficient for clients with mild symptomatology, but clients with more severe conditions required additional components of intervention (Baillie & Rapee, 2004). Taken together, the
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Evidence suggests that psychoeducation should be considered a recommended component of treatment.

Cognitive Restructuring
Anxious beliefs, predictions, and expectations about the feared stimulus are important factors in maintaining specific phobias. Clinicians may elicit catastrophic cognitions in assessment of specific phobia and/or during exposure exercises. Before beginning an exposure exercise, the clinician may ask the client to predict what will happen around the feared stimulus; for example, a client with a fear of snakes may predict that the snake will slither toward the client and bite him/her. During exposure, the clinician may ask the client to describe what is happening (e.g., “The snake is just staying in the corner”). After exposure, the client may be asked to describe what actually happened, testing his/her anxious expectations and drawing his/her own conclusions (Davis et al., 2009). Cognitive restructuring (or cognitive challenging) may occur in this manner, or it may occur through more explicit discussions of automatic thoughts, biases, and cognitive distortions. Adding cognitive restructuring did not enhance treatment outcomes in one study which involved an exposure-only group, exposure plus cognitive restructuring group, and waitlist group (in individuals with chronic post-traumatic stress disorder; Foa et al., 2005) and generally cognitive therapy alone has been found to be equivalent to exposure alone (e.g., Öst, Alm, Brandberg, & Breitholtz, 2001).

Modeling
Modeling may be used in combination with or independent of exposure exercises. According to Zlomke and Davis (2008), participant modeling serves as an important technique by increasing social support and guidance and by making tasks appear more manageable and structured. Participant modeling begins when a clinician physically demonstrates a step on the client’s fear hierarchy (e.g., touching a snake) while displaying appropriate coping behaviors and engaging the client in challenging misperceptions and catastrophic cognitions (e.g., “Do you think the snake will bite me if I touch it?”). Next, the clinician should ask the client to complete the modeled step with some added physical support or assistance (e.g., the clinician may be directly touching the snake while the client touches the clinician’s hand). Clinicians should gradually withdraw physical support until the client can independently complete the step. Participant modeling has demonstrated success in treatment with both children and adults (Davis & Ollendick, 2005; Davis et al., 2009; Zlomke & Davis, 2008). Participant modeling has been found to be more effective than no-treatment control conditions, live modeling, and filmed modeling (Davis & Ollendick, 2005).

Praise and Rewards
Clinicians often integrate praise into treatment; many may do so naturally, as a way to build and maintain rapport with clients. Exposure exercises lend themselves well to praise and rewards; in fact, reinforcement of an individual’s effort and successes may be essential with some clients. Reinforcers such as verbal praise and rewards may be a potent dose of social support and may increase therapeutic rapport, particularly when used during exposure sessions that can be difficult for clients. However, the contingent use of praise and rewards is important, as reinforcement functions as one of the potential mechanisms for treatment success (Davis et al., 2009). Clinicians may use reinforcement while treating specific phobia; however, it is important that caution is exercised to avoid unintentionally reinforcing fearful behavior. While reinforcement has strong theoretical support, it is important to note that few dismantling studies have been conducted to evaluate the independent impact of praise and rewards in the treatment of specific phobia, and existing research on therapist praise is largely limited to case studies (e.g., Leitenberg, Agras, Thompson, & Wright, 1968) and studies using older versions of the DSM. However, one study that evaluated the impact of praise on approach behaviors did
not find a significant relative contribution of verbal reinforcement during systematic desensitization (Oliveau, Agras, Leitenberg, Moore, & Wright, 1969). While there is a paucity of empirical research, praise and rewards may be considered a recommended component of treating specific phobia because of strong theoretical support and utility in increasing therapeutic rapport.

**Additional Considerations When Implementing Treatment**

**Enhancing Treatment**

**Pharmacotherapy**

According to Birk (2004), pharmacotherapy is contraindicated in treatment of specific phobia: adding medications (e.g., SSRIs, tricyclic antidepressant [TCAs], SNRIs, benzodiazepines) to treatment interferes with the exposure and inhibits the extinction of fear responses. The use of medications may aid in the acute treatment of severe phobias by reducing the subjective and physiological symptoms of fear; however, rates of relapse at follow-up with medications are greater than behavioral treatments such as exposure. This effect may be explained by the tendency of individuals to credit treatment gains to the medication, leading to an increase in relapse on pharmacological cessation (Choy et al., 2007). The addition of pharmaceuticals may additionally interfere with the extinction learning and generalization across contexts that create progress in specific phobia treatment (Moscovitch et al., 2009). At this time, pharmacological components do not appear to demonstrate incremental or independent validity in the treatment of specific phobia.

Preliminary research does suggest benefits of adding d-cycloserine (DCS; a form of a common antibiotic) to augment exposure-based treatments. DCS is indicated to facilitate memory consolidation, allowing for treatment gains to occur more quickly while maintaining gains after cessation of the drug (Ressler et al., 2004). However, the effects of DCS are inconsistent across studies (Birk, 2004); Smits et al. (2013) attribute this discrepancy to the individual participant’s level of fear at the end of the exposure session, suggesting that DCS is only as effective as the exposure treatment itself. Smits et al. (2013) evaluated two groups of participants with height phobia: both groups received equivalent amounts of virtual reality exposure, but one group received DCS while the other was assigned to the pill placebo condition. Individuals who had taken DCS and ended the exposure session with a low level of fear exhibited significant symptom decline relative to their placebo counterparts; in contrast, individuals who ended their exposure session with high fear levels did not improve significantly more than their placebo counterparts.

**Attention Training**

Cognitive models of specific phobia implicate attentional biases toward threat-based stimuli as maintaining factors. Some evidence supports attention bias modification training (ABMT), which aims to redirect an individual’s attention away from threat-related cues toward neutral, non-threatening stimuli. This attention modification procedure may lead to reductions in self-reported and clinically observed anxiety symptoms (Hallion & Ruscio, 2011). However, the research literature is mixed on the efficacy of ABMT in adults and children, as a number of studies have failed to show significant effects of ABMT (e.g., Choy et al., 2007; Rapee et al., 2013). A variation on ABMT examines the effect of adding attention training toward positive stimuli (ATP) rather than neutral cues. One study assessed the impact of ATP on children with phobias, evaluating clinical outcomes (i.e., report measures) and cognitive outcomes (i.e., changes in attentional bias). While significant differences were observed in cognitive outcomes, there was no corresponding difference in clinical outcomes, suggesting that gains in ABMT and its derivatives may not be generalizable (Waters et al., 2014). Therefore,
ABMT cannot be recommended as an evidence-based component of treatment at this time (see Table 3.1).

### Treatment Appraisal and Applications

#### Recognizing Comorbidity

As previously discussed, specific phobias are highly comorbid; it is important to recognize comorbidity in treatment planning, as some diagnoses can interfere with treatment success. One example to consider is ADHD, a fairly common comorbidity, as inattention, hyperactivity, and impulsivity can make treatment more difficult. For example, failing to attend to information or being overly active during psychoeducational components of sessions may interfere with the vital learning aspect of treatment or even disrupt/distress stimuli (e.g., antagonizing dogs with exciting, jerky movements). Additionally, long, massed exposure sessions may be impossible for individuals who cannot sustain attention or engage in hyperactivity or impulsivity. In children, oppositional defiant disorder (ODD) may interfere with treatment planning and progress. For example, children may refuse to complete fear hierarchies, share cognitive biases, or exhibit defiant behavior during exposures that limit treatment progress. Additionally, parental psychopathology (e.g., depression, hostility, paranoia) can thwart treatment progress (Berman, Weems, Silverman, & Kurtines, 2000). If interference with treatment is detected, modification may be warranted. For example, children with ODD may benefit from increased reinforcement, beginning with small, frequent rewards, and moving to larger, less frequent ones (Chorpita, Becker, & Daleiden, 2007). Notably, however, recent research suggests that comorbidity may have no detrimental effect on treatment outcome. Furthermore, one study demonstrated reductions in symptoms of anxiety related to comorbid disorders in individuals who received treatment for their phobias (Ollendick et al., 2010).

#### Cultural Considerations

Treatment seeking is dependent on several factors including culture, race, and socioeconomic status. It is easy for therapists to have less experience treating one culture versus another. For example,
stigma against seeking treatment services from a mental health professional may be even more prominent in minority cultural and ethnic populations. Reasons for this stigma can include fear of discrimination, fear of social exclusion, and other social factors (e.g., pride, trust, collectivism; Green, Hayes, Dickinson, Whittaker, & Gilheany, 2003). For example, African Americans in one study expressed concerns with trusting the therapist and with the therapist’s own biases. Furthermore, many chose not to seek services because of fear or lack of understanding as to what treatment will entail (Thompson, Bazile, & Akbar, 2004).

It is also important to consider culture during the initial assessment. Social and cultural factors can direct attention to specific stimuli. For example, a condition first described in Japan, jikoshu-kyofu, is described as fear of omitting a bad odor and offending others. Many people with jikoshu-kyofu exhibit impairment in social functioning or social isolation. Asking culturally relevant questions during assessment and treatment to gauge whether the fear is out of proportion in relation to the person’s cultural beliefs may be helpful in assessing and treating fear responses (Lim & Wan, 2015). Social factors, such as the level of individualism versus collectivism within the patient’s culture, may also change the course of treatment. For example, individuals from a collectivistic culture may request that extended family members be more integrated in their treatment (National Institute of Mental Health, 1999). Matching a therapist to a client with the same language or background may also be helpful in maintaining effective treatment. Clients with culturally matched therapists were significantly less likely to prematurely terminate treatment and had higher treatment satisfaction ratings compared to clients and therapists with different native languages (Sue, 1998). In any case, evidence suggests that an active, engaged therapeutic style, coupled with cultural competence, is most effective in differentiating between satisfied and unsatisfied clients (Chang & Berk, 2009).

Clinician Reservations

While exposure-based treatments of phobias are supported by evidence and are well tolerated by the public, clinicians may have reservations about the implementation of exposure therapy. These concerns may include the possibility of lawsuits, symptom worsening, and premature cessation of treatment; in some cases, such reservations lead to clinicians choosing alternative treatments which are less substantiated by evidence or implementing exposure poorly (e.g., choosing less challenging exposures than necessary; Deacon et al., 2013). Deacon et al. (2013) measured these maladaptive therapist beliefs systematically before and after clinicians completed a workshop on exposure-based treatment. Findings demonstrated that the workshop decreased negative beliefs about exposure therapy by 50%. Clinicians wary of this type of treatment for their anxious patients may find didactic trainings helpful in improving treatment of their patients through limiting their own biases. Recommendations for treatment implementation follow (see Figure 3.1).

Troubleshooting Treatment

Clients do not always respond to treatment, and even the most efficacious therapies can fail because of external factors such as low motivation, disruptive or defiant behaviors, pessimism about the likelihood of treatment gains, lack of practice, or skills deficits. Clinicians should be flexible and prepared to implement supplemental strategies in order to increase the likelihood of treatment success and maintenance of gains. For example, a client with low motivation may not understand how his/her specific phobia interferes with his life. In this case, it is worthwhile to take some extra time to re-evaluate treatment expectations and goals with the client (e.g., quality of life); the client may have been misdiagnosed or may be ignoring evidence of interference. Clients with low motivation may also benefit from the implementation of a reward system—these rewards may be tangible (e.g., stickers for children, movie tickets, snacks
or age-appropriate special rewards) or social (e.g., attention, praise, extra time spent with a parent or loved one) in nature. It is important to note that rewards should be earned for desired behaviors (i.e., should serve as contingent reinforcers). Supplemental strategies such as parent training may be needed for children with disruptive or defiant behaviors if these behaviors interfere with treatment. Individuals who exhibit pessimistic behaviors (e.g., suggesting that treatment will not work for them) may benefit from additional psychoeducation about the empirical support and theoretical rationale behind treatments for specific phobia or additional assessment to check for comorbid depressive conditions. Skills deficits may interfere with the proper assessment of specific phobias or may complicate treatment progress. For example, an individual may exhibit significant fear and avoidance of deep water because of an inability to swim; this skill deficit may need to be addressed in the process of treatment. In this case, a clinician may incorporate swimming skills training into the educational component and exposure exercises (Chorpita, 2007). Throughout treatment, clinicians should strive to stick to the most essential evidence-based components. In other words, troubleshooting should not distract from treating the client’s specific phobia through exposure.

**References**


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**Figure 3.1** Recommended treatment sequencing.


Maysa M. Kaskas, Paige M. Ryan, and Thompson E. Davis III


This chapter provides a review of the empirically validated explanatory models of panic disorder and agoraphobia. It also discusses the latest research on the efficacy of treatments for these disorders. Finally, common barriers to success in treatment for panic disorder and agoraphobia are discussed, with an emphasis on the application of experimental findings to clinical populations. In accordance with the most recent publication of the *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition (DSM-5; American Psychiatric Association, 2013), panic disorder and agoraphobia are discussed as separate disorders. However, research prior to DSM-5 typically included agoraphobia as a classifier of panic disorder, leading to much overlap in the literature between these constructs in terms of their etiology and treatment.

**The Nature of the Problem**

**Vulnerabilities**

**Anxiety Sensitivity**

Anxiety sensitivity (AS), or the belief that anxiety symptoms are harmful (Reiss & McNally, 1985), plays a prominent role in the development of panic disorder and agoraphobia. Both of these disorders involve the fear of acute disruptions in physiological sensations and changes in behavior that serve to reduce those physiological sensations. AS is multidimensional in nature and includes three lower-order factors, comprising physical concerns, mental incapacitation concerns, and social concerns, and a higher-order general factor which continues to account for the majority of variance in AS (Zinbarg, Barlow, & Brown, 1997).

Cross-sectional research demonstrates that AS is a correlate of panic attacks across the developmental spectrum (for reviews, see Olatunju & Wolitzky-Taylor, 2009; Zvolensky, Schmidt, Bernstein, & Keough, 2006), and longitudinal research demonstrates that AS predicts panic attacks up to two years later (Schmidt, Zvolensky, & Maker, 2006). Furthermore, the physical symptom lower-order factor is significantly higher in patients with panic disorder with or without agoraphobia compared to those with specific phobia or generalized anxiety disorder, even when depression and anxiety scores are accounted for (Rector, Szacun-Shimizu, & Leybman, 2007).

Experimentally manipulated beliefs about the risk of physical sensations predict subsequent levels of anxiety. This was demonstrated in an experiment that compared signaled changes in physiological signals in patients with nocturnal panic (Craske & Freed, 1995). Some of the participants were told that significant changes in physiology are safe, while others were not. Those who were instructed on the safety of significant physiological signals reported significantly less anxiety and had lower physiological ratings.
Panic Disorder and Agoraphobia

(Craske & Freed, 1995). Similar effects have been found in panic disorder patients who complete a hyperventilation challenge, a method for experimentally instilling acute physiological disturbances (Brown, Smits, Powers, & Telch, 2003).

A meta-analysis of the relation between AS and all of the internalizing disorders found that the strongest association was between AS and panic disorder, with the physical sensations lower-order factor having the strongest correlation (Naragon-Gainey, 2010). A similar finding was reported for agoraphobia. Furthermore, when the internalizing disorders were categorized as fear (including agoraphobia and panic, but also social anxiety and specific phobia) versus distress disorders (including generalized anxiety, depression, and post-traumatic stress disorder), AS had a strong relation to both panic and agoraphobia over and above the shared variance with the other disorders (Naragon-Gainey, 2010). However, caution must be applied in the interpretation of the findings related to agoraphobia, as there were only moderate zero-order correlations with AS.

The introduction of AS into the framework of conceptualizing panic disorder and agoraphobia was essential to the development of etiological as well as treatment models of both of these disorders. However, despite the wealth of research on AS as a predictor of panic disorder and agoraphobia, some research demonstrates that it is not a specific predictor of panic, and that neuroticism is a more robust predictor (see Bouton, Mineka, & Barlow, 2001, for a review).

Neuroticism/Negative Affect

Neuroticism is defined as one’s propensity toward experiencing intense and frequent emotions or negative affect in response to stressful events (Barlow, Sauer-Zavala, Carl, Bullis, & Ellard, 2013; Clark & Watson, 1991). Broadly speaking, neuroticism confers risk for several negative health outcomes, including increased risk of stressful life events, mood, anxiety, and substance use disorders (Kendler, Gardner, & Prescott, 2003; Kotov, Gamez, Schmidt, & Watson, 2010; Malouff, Thorsteinsson, & Schutte, 2005). It is a common factor between all anxiety and mood disorders for adolescents and adults (Griffith et al., 2010; Krueger, McGue, & Iacono, 2001; Weinstock & Whisman, 2006; Zinbarg et al., 2010). A meta-analysis of genetic studies revealed that neuroticism and anxiety disorders share genetic factors (Webb et al., 2012). Despite the symptom overlap between internalizing disorders and neuroticism, a significant association between neuroticism and these disorders remains even after covarying the symptom overlap (for a review see Barlow et al., 2013; Griffith et al., 2010; Lahey, 2009). This suggests that neuroticism confers to disorder and is theoretically distinct from these disorders.

Like AS, neuroticism has strong associations with panic disorder and agoraphobia in particular. It is higher in patients with panic disorder (PD) regardless of agoraphobic status when compared to healthy controls (Carrera et al., 2006; Vohma, Aluoja, Vasar, Shlik, & Maron, 2010), and in particular is higher when PD is comorbid with depression (Freire et al., 2006; Weinstock & Whisman, 2006). A recent meta-analysis of 37 studies that include PD and 21 studies that include agoraphobia found a large effect size for the association between these disorders and neuroticism ($d = 1.92$ for panic and $d = 1.61$ for agoraphobia; Kotov et al., 2010). About one-half of the genetic variance between neuroticism and both panic disorder and agoraphobia is shared (Hettema, Neale, Myers, Prescott, & Kendler, 2006). Accordingly, there is a well-established relation between neuroticism and both panic disorder and agoraphobia.

A variety of methodologies have confirmed the predictive power of neuroticism for panic disorder and agoraphobia. In a longitudinal study, higher levels of neuroticism at baseline predicted panic attacks over an extended observation window of up to four years (Coryell, Dindo, Fyer, & Pine, 2006). The relation between neuroticism and panic disorder may be moderated by gender such that greater neuroticism at baseline in females is significantly predictive of the onset of panic disorder three years later, whereas this is not found in males (Zinbarg et al., 2016). On daily diary studies, neuroticism is significantly predictive of daily AS-related thoughts.
(e.g., “It scared me when I experienced these anxious feelings and bodily sensations” and “I worried that I may have a serious illness when I experienced these anxious feelings and bodily sensations”), and the relationship between daily levels of anxiety and AS varies as function of neuroticism (Hong, 2009). Specifically, on days with high anxiety, those with high neuroticism (compared to low neuroticism) report an increase in AS-related thoughts. During experimental manipulations, such as carbon dioxide challenges, which temporarily elevate CO₂ levels and produce symptoms similar to panic attacks, those with neuroticism exhibit greater reactivity in terms of self-reported ratings of discomfort, but not necessarily on physiological symptoms (Coryell et al., 2006; Verschoor & Markus, 2012). Therefore, neuroticism reliably predicts panic disorder and agoraphobia symptoms in a variety of contexts.

**Interoceptive Sensitivity**

The interoceptive conditioning model of panic disorder suggests that minor changes in physiology become associated with more extreme, innately aversive surges of autonomic arousal, such that benign physical sensations evoke a conditioned fear response or a full-blown panic attack (Bouton et al., 2001; Craske, 1991). The interpretation of benign physical sensations as threatening makes it more likely that those sensations will increase in intensity (Barlow, 2002). The interoceptive conditioning model is discussed in more detail later in this chapter, but this section discusses variability in the detection of interoceptive cues.

There are naturally occurring differences in the ability to detect changes in physiology. Higher levels of arousal, but not anhedonia or general distress, is related to improved accuracy in heart rate detection (Dunn et al., 2010), as are more intense emotions during emotion provocation tasks (Wiens, Mezzacappa, & Katkin, 2000). Healthy patients with higher-state and trait anxiety scores as well as AS have greater accuracy at detecting heartbeats, and their interoceptive accuracy is mediated by neuronal activation in the right insula (Craig, 2002; for a review on the role of the insula on interoception, see Critchley, Wiens, Rotshtein, Ohman, & Dolan, 2004; Domshke, Stevens, Pfeiderer, & Gerlach, 2010; Eley, Stirling, Ehlers, Greg, & Clark, 2004). Individuals who detect changes in their physiology more readily experience more arousal as a result of those changes (Dunn et al., 2010). Patients with panic disorder tend to be more accurate in detecting their heartbeat compared to healthy controls (see Ehlers & Breuer, 1996, for a review); however, they also tend to misperceive arrhythmic heart activity in the absence of irregularities (Barsky, Cleary, Sarnie, & Ruskin, 1994). This likelihood to overdetect irregular physiology may contribute to increased anxiety and panic. Therefore, sensitivity to alterations in heartbeat detection and breathing is correlated with experiencing emotions as more intense, as well as with diagnoses of panic disorder; and the insula may play a role in interoception.

Another method of testing sensitivity to interoceptive cues involves manipulating the duration of CO₂ inhalations as a conditioned stimulus (CS) or unconditioned stimulus (US), to mimic the association between physical sensations of a mild (CS) and extreme degree (US; for a complete description see the “Conditioning Models” section below) in panic disorder: When a short-duration inhalation of CO₂, which elicits mild sensations of difficulty breathing, is paired with a long-duration inhalation of CO₂, which elicits stronger sensations of difficulty breathing, increased anxiety to the short-duration inhalation is reported in comparison to participants who never had the mild and strong sensations paired together (Acheson, Forsyth, & Moses, 2012). Similar results have been found when comparing a paired association between a period of labored breathing (CS) with a period of obstructed breathing (US) to unpaired presentations of both stimuli, with more conditioned fear to the paired CS (Pappens, Smets, Vansteenwegen, Van den Bergh, & Van Diest, 2012). In addition, low levels of loaded breathing, or the addition of resistance to breathing, has been supported as a
conditional stimulus for a paired high level of loaded breathing, supporting the notion that benign physiological sensations can trigger a fear response in terms of startle electromyography (EMG) and fear ratings (Pappens et al., 2013). These findings provide evidence that internal sensations can become fear-eliciting CSs, a crucial tenet of the interoceptive sensitivity risk model of panic.

Some contexts predispose individuals toward greater sensitivity to interoceptive cues. Presenting an auditory script detailing suffocation that is intended to evoke physical sensations during a carbon dioxide challenge (i.e., 35% CO₂-enriched air) results in a conditioned response to the script compared to a valence-matched control script (De Cort, Griez, Büchler, & Schruers, 2012). Overt awareness of the contingency between the suffocation script and the CO₂-enriched air is not required to observe this effect (De Cort et al., 2012). This demonstrates the capability of some biologically relevant stimuli, such as those internal sensations evoked while listening to the suffocation script, to become a source of fear, whereas other stimuli are not as easily conditioned. Thus, patients with panic disorder may develop a fear of interoceptive cues because of their prior experiences with those cues in situations where they perceived danger, such as while standing in a closed elevator without the ability to escape.

Environmental Risk Factors
In addition to variations in temperament and interoceptive sensitivity, life experiences contribute to the development of panic disorder and agoraphobia. Support for the familial environmental impact of panic disorder comes from genetic studies which report high correlations between environmental risk factors and incidences of panic disorder after accounting for shared genetic variance with neuroticism, but low correlations for other anxiety disorders (Hettema et al., 2006). Nineteen percent of the variance in anxiety disorders is accounted for by genetic factors, whereas that value increases to 34% and 44% when one or two stressful life events is included in the explanatory model (Silberg, Rutter, Neale, & Eaves, 2001).

Childhood Adversity
Childhood adverse life events that involve loss and separation, particularly of parents and caregivers, confer a risk for the development of panic disorder or agoraphobia during adulthood (Bandelow et al., 2001; Faravelli, Webb, Ambonetti, Fonnesu, & Sessarego, 1985). Particularly risky life events include loss, threat, adjustments, marital separation, and interpersonal conflict (see Klauke, Deckert, Reif, Pauli, & Domschke, 2010, for a review). Childhood physical, as well as sexual, abuse confers unique risk to the development of panic disorder (Goodwin, Fergusson, & Horwood, 2005). The relationship between stressful life events and the development of panic disorder may be moderated by comorbid disorders like depression, though more research is needed on this area. It is also not clear if the occurrence of stressful life events gives rise to a unique risk in the development of panic disorder or all anxiety disorders.

Parental Health Anxiety
Parenting behaviors around illness may influence the development of panic disorder, although most of this research is correlational rather than causational in nature. For instance, patients with panic disorder reported more somatic symptoms prior to age 18 compared to healthy controls, those with infrequent attacks, or those with other anxiety disorders (Ehlers, 1993). Importantly, panic disordered patients report a history of receiving more attention for sick-role behaviors, observing more sick-role behaviors in their parents, and having more family members with chronic medical conditions (Ehlers, 1993). However, it is unclear if the associations between parental health anxiety and panic disorder are caused by a third variable, such as degree of somatization or patient health anxiety. Higher rates of health anxiety, disease phobia, and somatization are reported in adults with agoraphobia and panic disorder (Fava, Porcelli, Rafanelli, & Mangelli, 2010;
Rudaz, Craske, Becker, Ledermann, & Margraf, 2010). However, somatization and health anxiety, but not panic disorder alone, are predictive of work absence due to sickness (Hoedeman, Blankenstein, Krol, Koopmans, & Groothoff, 2010), possibly suggesting that panic disorder symptoms relate to sick-role behaviors through other mechanisms (i.e., somatization and health anxiety). Therefore, it is unclear if parental health anxiety without hereditary tendencies toward somatization and health anxiety would increase the risk of panic disorder development. Future research should explore this area of research.

**Physical Illness**

A number of medical conditions are associated with panic disorder including cardiac disorders, gastrointestinal illness, migraines, and genitourinary disorders, regardless of agoraphobia comorbidity or depression status (Harter, Conway, & Merikangas, 2003), as well as asthma (Katon, Richardson, Lozano, & McCauley, 2004). However, there is dissent over whether these conditions predict the development of panic disorder or are epiphenomenal (Klauke et al., 2010). The direction of causality may depend on the type of illness. Patients with short-duration panic disorder, for instance, report that their comorbid panic attacks began following coronary artery disease, whereas patients who have a longer history of panic disorder report later development of coronary artery disease (Goldberg et al., 1990).

Like the link between panic and cardiovascular disturbances, the relation between panic disorder and asthma has been well established. Poor respiratory health is a specific predictor of panic disorder and agoraphobia versus other anxiety disorders (Craske, Poulton, Tsao, & Plotkin, 2001), and patients with asthma are more likely to have panic disorder (Goodwin, Pagura, Cox, & Sareen, 2010). This association is most likely driven by hypnocapnia, or low carbon dioxide in the bloodstream, which is present in some patients with asthmatic patients and panic disorder and can have adverse effects on symptoms of asthma that may be exacerbated by emotional reactions to hyperventilation (for a thorough review, see Meuret & Ritz, 2010). Children with a history of asthma attacks are more likely to have parents with *ataque de nervios*, which is a culturally bound condition in Caribbean Latinos, and particularly Puerto Ricans, that resembles panic attacks (Ortega, Goodwin, McQuaid, & Canino, 2004). Finally, emerging research supports the notion that dysregulated breathing that naturally occurs in some patients with panic disorder may affect brain pH levels in regions implicated in fear responding such as the brainstem, amygdala, and hypothalamus, but more research must be done in this area before any firm conclusions are drawn (Wemmie, 2011).

**Conditioning Models**

A review of the etiology of panic disorder would be incomplete without a thorough discussion of the conditioning models that explain the development of this problem. The models do not operate independently of the risk factors previously described, but instead act in concert with them. Conditioning models posit that previously neutral stimuli, such as innocuous bodily sensations in the case of panic, or walking in a crowd in the case of agoraphobia, become associated with biologically threatening stimuli, such as high levels of bodily sensations. Consequently, previously neutral stimuli become conditioned through their association with the threatening stimulus, or the unconditioned stimulus. Following this excitatory learning (CS → US), the CS induces a conditioned response (CR) that includes fear, anxiety, and distress (Bouton et al., 2001; Craske, 1991). Thus, low-intensity bodily sensations come to elicit high fear and anxiety because of their prior associations with high-intensity bodily sensations.

Not all associations are created equal, and there are a variety of circumstances that make CS–US pairings more likely to result in panic disorder or agoraphobia. It is believed that initial panic attacks are typically prompted by stress or some other biologically driven factor, and
subsequent attacks occur through the encountering of stimuli that were associated with the first attack (Barlow, Chorpita, & Turovsky, 1996). These may include mild physiological sensations but also places or situations in which the initial attack occurred. The quality of initial panic attacks can determine the severity of subsequent anxiety about future panic attacks. Those attacks that occur in situations where escape is particularly difficult (e.g., in an elevator, when traveling on a bus, etc.) confer risk to greater anxiety severity, possibly because of an increased sense of uncontrollability (Bouton et al., 2001; Craske, Miller, Rotunda, & Barlow, 1990). Furthermore, patients who experience both expected and unexpected panic attacks report greater levels of anxiety following unexpected attacks, which suggests that predictability is also an important factor (Craske, Glover, & DeCola, 1995). Thus, while conditioning models lay the framework for understanding how panic and agoraphobia develop, it is important to consider the individual patient’s experience when formulating his/her treatment.

Another aspect of the patient’s experience that can shape his/her disorder is through vicarious conditioning. Experimental research has demonstrated that if neutral images are paired with a mother’s fear or disgust reaction, toddlers display greater fear and avoidance of those images compared to images followed by positive maternal reactions (Gerull & Rapee, 2002). This phenomenon of an individual acquiring fear via observation is termed vicarious conditioning, and it suggests that parental modeling of fear early in development can play an important role in the expression of fear. Similar findings have been reported in adults who had neutral cues paired with panicked reactions from confederates, in that participants displayed greater levels of physiological reactions to the neutral cues compared to an unpaired cued (Kelly & Forsyth, 2007). Vicarious conditioning models have important explanatory power for all of the anxiety disorders, including panic disorder and agoraphobia. However, the vulnerability factors associated with panic disorder and agoraphobia described above, including parental health anxiety, indicate that vicarious conditioning is particularly important to consider for these disorders.

Empirically Supported Treatment Components

Cognitive behavioral therapies (CBT) represent a number of empirically supported treatment strategies that are effective in treating patients with panic disorder and agoraphobia. This section will provide a brief overview of the components of cognitive behavior therapy; the evidence base for CBT as a package and individual treatment components are then presented, followed by the prescription of a possible sequence in which the methods may be implemented. Finally, this section will explore factors that interfere with the dissemination and adoption of CBT. The last section of this chapter provides more detail about nuances of panic and agoraphobia presentation that can hinder the success of traditional CBT.

Brief Overview of Cognitive Behavioral Treatment Components for Panic Disorder and Agoraphobia

Published manuals for treating panic disorder and agoraphobia largely include comparable treatment components, although there is variability in the emphasis on different techniques and in methods for delivering these techniques. Treatment typically begins with a functional analysis of the patient’s unique presentation, including their behavioral, cognitive, and emotional responses to their environment, as well as psychoeducation about the intersection between these three components (Craske, 2010). Psychoeducation is also provided about the fight/flight model and the evolutionary significance of anxiety and panic (Dannon, Iancu, & Grunhaus, 2002). Downward arrow techniques are employed to identify maladaptive cognitions, and cognitive
Research Supporting the Efficacy and Effectiveness of Cognitive Behavioral Treatment

CBT has been listed as an empirically supported treatment (EST) since 1993 by Division 12 of the American Psychological Association. This indicates that the efficacy of this treatment over a placebo, or equivalent performance with another EST, has been reported by at least two independent groups of researchers (Chambless et al., 1998). Across a wide range of disorders, CBT is more effective at post-treatment and follow-up compared to psychodynamic therapy (Tolin, 2010). In community managed-care settings, CBT is more effective than treatment as usual for panic disorder with or without agoraphobia (Addis et al., 2004; Craske et al., 2011; Roy-Byrne et al., 2005). A meta-analysis of CBT for anxiety disorders revealed a significant effect of treatment for panic disorder (Cohen's $d = .351$), however, given that so few of the available studies parsed apart panic from agoraphobia, separate estimates were not available for agoraphobia (Hofmann & Smits, 2008). Effects sizes are large for group CBT for panic disorder (Hedges’ $g = 1.39$) and agoraphobia (Hedges’ $g = .92$; Soares, Camargo, & Pizzinato, 2013), and for computer-delivered CBT for panic/agoraphobia (Andrews, Cuijpers, Craske, McEvoy, & Titov, 2010), but many of the comparison conditions in these meta-analytic reviews were waitlist controls. Therefore, CBT as a treatment package is a highly efficacious option for panic and agoraphobia, but these reviews do not indicate which components of the package are essential. A review of the efficacy of each component of CBT is provided below.

Psychoeducation

The evidence base for psychoeducation alone as a treatment for panic or agoraphobia is scant. A meta-analysis of psychoeducation for combined depression and psychological distress (including panic symptomatology in some studies) found a small yet significant effect size ($d = .20$), although effects were not analyzed separately by disorder (Donker, Griffiths, Cuijpers, & Christensen, 2009). Therefore, psychoeducation alone may confer a small benefit for panic and agoraphobia, though the effect sizes are lower than those for the full package of CBT, and much more research is needed before firm conclusions can be drawn.

Exposure

A review of the active components of CBT revealed that the most commonly used component of CBT for panic disorder/agoraphobia in published randomized controlled trials is exposure (Sánchez-Meca, Rosa-Alcázar, Martín-Martínez, & Gómez-Conesa, 2010). Within exposure, in vivo (which includes interoceptive exposure, or exposure to physiological sensations) is the most commonly reported, and imaginal exposure is rarely reported (Sánchez-Meca et al., 2010). Of the three most commonly reported components of CBT (exposure, cognitive therapy, and relaxation/breathing retraining), the highest effect size is for...
combined exposure and breathing retraining in panic disorder (Sánchez-Meca et al., 2010). However, Sánchez-Meca et al. (2010) combined in vivo exposure to real-life situations and interoceptive exposure. A comparison of exposure to cognitive techniques for panic disorder revealed no significant differences, though this meta-analysis did not parse apart the various types of exposure (Ougrin, 2011). When interoceptive, in vivo, or the combination of these exposure techniques have been compared to a waitlist control condition, all of the exposure conditions appear superior to control, but there are no differences between them (Ito, de Araujo, Tess, de Barros-Neto, & Asbahr, 2001). Higher levels of cortisol may predict improved exposure response rates for patients with panic disorder and agoraphobia (Meuret et al., 2015). In sum, exposure is a commonly used and well-supported component of CBT for panic disorder and agoraphobia, although individual differences may confer differential response rates. A new understanding of the mechanisms behind exposure have revealed variants in the implementation of this technique, which is discussed in below (“Methods for Enhancing Inhibitory Learning and Reducing Return of Fear”), that are likely to improve outcomes even further.

Cognitive Techniques
The second most commonly reported treatment component of CBT in meta-analytic studies is cognitive restructuring (Sánchez-Meca et al., 2010). However, there is some evidence that adding cognitive restructuring to exposure therapy does not enhance outcomes (Deacon et al., 2012; Öst, Thulin, & Ramnero, 2004). At the same time, one meta-analysis demonstrated no advantage to pure cognitive or pure exposure techniques when the two are directly compared for panic disorder with or without agoraphobia both in overall outcome levels (Ougrin, 2011). Specifically, both strategies significantly reduce symptom levels of both panic and agoraphobia (Bouchard et al., 1996). Symptom reductions following cognitive restructuring for panic disorder may be achieved via changes in symptom reappraisal as well as changes in perceptions of the ability to control symptoms, although the relation between symptoms, appraisals, and perceived control may be bidirectional in nature (Meuret, Rosenfield, Seidel, Bhaskara, & Hofmann, 2010). Therefore, cognitive restructuring may be an effective tool for reducing panic symptomatology, but it does not necessarily improve outcomes compared to exposure alone.

Acceptance
Acceptance techniques, such as those used in acceptance and commitment therapy (ACT; Hayes, Strosahl, & Wilson, 1999) and dialectical behavior therapy (Linehan, 1993), have demonstrated some efficacy in patients with panic disorder. When combined with exposure, training in acceptance techniques results in a reduction in panic symptom severity and avoidance, although there was no active comparison in this study (Meuret, Twohig, Hayes, Rosenfield, & Craske, 2012). In addition, panic disorder patients with or without agoraphobia were randomized to view video demonstrations of acceptance, cognitive distraction, or neutral scenes prior to entering a CO₂ challenge, and those who viewed the acceptance video demonstrated less anxiety and avoidance of a subsequent challenge compared to the other groups (Levitt, Brown, Orsillo, & Barlow, 2004). The benefits of acceptance over suppression were replicated in a mood induction manipulation (Campbell-Sills, Barlow, Brown, & Hofmann, 2006), though this sample was a mixed anxiety and mood-disorder sample, and thus it is not clear how the results apply to panic and agoraphobia specifically. However, there are no other studies directly examining acceptance strategies on panic or agoraphobic symptoms, and thus more research is needed on this topic.

Relaxation and Breathing Retraining
The third most commonly reported component of CBT is relaxation and breathing retraining (Sánchez-Meca et al., 2010). Although these components are actually quite distinct, they are often collapsed together in meta-analyses.
Traditional diaphragmatic breathing retraining does not improve outcomes when added to interoceptive exposure (Deacon et al., 2012), and some data suggest a worsening of outcomes with its inclusion possibly due to the tool operating as a safety behavior (Schmidt et al., 2000). However, capnometry-assisted respiration training (Meuret et al., 2008), an intervention to correct hypnocapnic breathing through biofeedback, results in changes in partial pressure of carbon dioxide (PCO2) that significantly mediate the relation between changes in panic disorder severity over time (Meuret et al., 2010). Unfortunately, CART has rarely been tested in isolation from other therapeutic techniques, and thus additional research is needed to determine its incremental utility (see Meuret, Wolitzky-Taylor, Twohig, & Craske, 2012, for a review).

Progressive muscle relaxation (PMR; Bernstein & Borkovec, 1973; Wolpe, 1958) involves the intentional tensing and relaxing of muscle groups and has been applied for the treatment of a variety of anxiety disorders. However, as with traditional breathing retraining, there is not sufficient evidence that the addition of PMR improves outcomes beyond those obtained from exposure (for a review, see Meuret, Wolitzky-Taylor et al., 2012). More research is warranted to investigate PMR in panic disordered populations prior before any firm conclusions can be drawn.

**Proposed Sequence of Treatment Components**

Given the research outlined above, recommendations can be made for which treatment components should be included in the delivery of CBT for panic disorder and agoraphobia. However, no research has investigated the sequence of individual CBT components. One study found that the majority of treatment gains were made in the first month of treatment, which included psychoeducation, diaphragmatic breathing, interoceptive exposure, cognitive restructuring, and progressive muscle relaxation, with the introduction of *in vivo* exposure after these gains were made (Peneva, Otto, Maki, & Pollack, 1998). However, this study did not directly compare the listed order of components to an alternate presentation. Therefore, it remains unclear whether, for example, exposure should precede cognitive restructuring, or vice versa. Thus, recommendations provided herein are based on the sequences provided in available empirically supported treatment manuals (see Barlow & Craske, 2006; Craske & Barlow, 2006, for examples) and will likely change with future research.

Initial sessions of CBT for panic and agoraphobia include psychoeducation about anxiety and panic, as well as a functional analysis of cognitions, behaviors, and emotions. Combining these two elements may allow patients to develop a formal understanding of anxiety and panic, broadly speaking, while also relating common presentations of anxiety and panic to their own experiences. Self-monitoring follows to reinforce the principals taught in the functional analysis and to provide additional evidence to formulate the patient’s conceptualization (Craske & Tsao, 1999). On the basis of this information, therapists can present information about behavioral principles, including conditioning and avoidance models, so that the groundwork for exposure is provided. Around this time, breathing retraining strategies typically are provided, ideally through the use of biofeedback when possible. Then, cognitive restructuring is taught to provide the patient with strategies for decatastrophizing and modifying overestimations of threat. Next, interoceptive exposure to the patient’s feared internal sensations is introduced. Exposure to these sensations begins in session and is subsequently assigned for practice between sessions. *In vivo* exposures begin with the generation of a fear hierarchy, such that the patient clearly identifies situations that he/she avoids, and then proceed with exposures conducted both within and between therapy sessions. Further recommendations about the nature of exposures based on the latest research are discussed below. Lastly, the
therapist makes a relapse prevention plan prior to treatment termination, which includes strategies to help patients continue to implement approach behaviors in their daily life, as well as cognitive restructuring of beliefs and thoughts that tend to result in avoidance. The relapse prevention plan includes normalization around the high rates of return of fear, as well as a plan for which tools to implement when or if fear returns. Treatment may be tapered down as termination approaches, or termination of in-person sessions may be followed by monthly check-in phone calls as appropriate.

**Factors that May Interfere with the Implementation of Existing Treatment Manuals and Protocols**

One set of obstacles to the implementation of available empirically supported treatments for panic disorder and agoraphobia are therapist factors. In a study of organizational and client barriers to the dissemination of exposure therapy, the most commonly reported barriers are the lack of supervision opportunities for therapists, an inability to conduct exposures outside of the therapist office, and an inability to have sessions longer than one hour (Harned, Dimeff, Woodcock, & Contreras, 2013). However, cost-effective Internet interventions that provide psychoeducation about the use of exposure result in increased adoption of this technique, with those therapists who have greater self-efficacy around exposure reporting higher rates of use (Harned et al., 2013). With the proliferation of online trainings in practically every disorder and treatment modality, it is possible that more and more clinicians will feel confident in their ability to deliver CBT. There are surprisingly few training sites providing training in CBT (Weissman et al., 2006), and an increase in CBT training opportunities will reduce the number of clinicians who enter private practice without this competency. Regarding concerns about conducting exposure outside of the therapist office, the literature overwhelmingly demonstrates that content learned during exposure therapy is context-dependent, meaning that the learning does not necessarily generalize once outside of the therapy context (Bouton et al., 2001). Exposure outside of the therapy context (e.g., office setting) has the potential to improve the generalizability of exposure therapy. However, institutional limitations clearly present unique challenges to the implementation of exposure outside of the therapist office in certain organizations. Therefore, therapists should collaboratively design homework assignments that allow patients to practice exposure in as many contexts as possible (this is discussed in more detail below). Finally, regarding the concern about length of exposures, one method for obviating this realistic challenge is to begin exposures early in session in a setting in which the patient can remain following the therapy hour. This can allow the patient to not only complete longer exposures but also to gain a sense of self-efficacy in their ability to conduct exposures independently.

Other factors that may interfere with the implementation of CBT include the cost of large-scale roll-outs of mandated treatment models. Dissemination of evidence-based practices on a large scale, such as the Improving Access to Psychological Therapies in the United Kingdom (IAPT; Clark, 2011) and the US Department of Veterans’ Affairs (VA), can cost hundreds of millions of dollars as a result of the allocation of funds toward initial therapist and supervisor training, ongoing consultation, and methods for ensuring adherence and competence (McHugh & Barlow, 2010). While this major initial expense is prohibitively large for many organizations, the long-term cost-effectiveness of implementing evidence-based practices for panic disorder and agoraphobia should be measured in terms of reduction in inpatient services (Katon et al., 2006), medication use (Michael, 1993), and utilization of healthcare services broadly (Myhr & Payne, 2006). Future research should continue to focus on these outcome variables to provide further justification for the widespread adoption of CBT for panic and agoraphobia.
Appraisal and Applications of Cognitive Behavioral Treatment for Panic and Agoraphobia

Panic disorder and agoraphobia is highly susceptible to the return of fear following exposure therapy (Craske & Mystkowski, 2006). This suggests that extinction learning involves not the erasure of the original fear memory, but instead the development of a competing inhibitory association (Bouton et al., 2001). The various ways in which the retrieval of inhibitory memories is disrupted are described below, including spontaneous recovery, renewal, reinstatement, and rapid reacquisition. Given these challenges, techniques meant to supplement traditional exposure therapy are also provided below, including variability in exposures, deepened extinction, reducing safety signals, expectation violation, retrieval cues, affect labeling, and the use of the pharmacological agent d-cycloserine.

Ways in which Retrieval of Inhibitory Memories Can Be Disrupted

Spontaneous Recovery
Spontaneous recovery involves the return of previously extinguished responding, or in the case of panic disorder and agoraphobia the return of fear, after the passage of time alone (Rescorla, 2004). In other words, after a patient with panic disorder has successfully completed exposure therapy, fear of internal sensations may return over time. Animal research demonstrates that the degree of expressed fear following extinction is a function of the amount of time that has passed, with greater intervals resulting in higher fear levels (i.e., less inhibitory memory; Quirk, 2002). However, animals that do not receive extinction trials do not demonstrate a significant increase in their fear over time, suggesting that the gradual return of fear following extinction is due to a weakening of the extinction memory rather than to an incubation of the fear memory (Quirk, 2002). Fortunately, when participants are given a short or long duration between phases of extinction, those with more time between extinction phases experience spontaneous recovery in the short run, but improved long-term outcomes (Leuong & Westbrook, 2008). This suggests that patients who experience a return of fear during therapy may actually fare better than those who do not, provided they are willing to complete subsequent exposures. More research is needed to confirm these findings in humans and in the context of panic disorder and agoraphobia specifically.

Renewal
Whereas fear learning is context-independent (i.e., the memory can be recalled in contexts outside of where it was initially learned), extinction learning is context-dependent (Bouton & Bolles, 1979; Hermans, Craske, Mineka, & Lovibond, 2006). Renewal effects are robust to even extensive extinction learning (Tamai & Nakajima, 2000) and to multiple changes in context (Neumann, Lipp, & Cory, 2007). Thus, when a patient experiences fear reduction through exposure in one setting, the fear is likely to return when the setting is changed. For example, if a patient with panic disorder engages only in interoceptive exposure to hyperventilation in the therapist’s office, minor breathing dysregulation outside of the therapist office may result in a return of fear of this sensation. Important, the definition of context is not specific to only the physical environment of the patient, but also to the effects of time of day and internal states, including drug-induced states, as well as mood-altered states (Bouton, 2002). Suggestions are provided below (under “Methods for Enhancing Inhibitory Learning and Reducing Return of Fear”) to optimize exposure given the role of the renewal of fear following changes in context.

Reinstatement
Another method through which fear may return is reinstatement. Following extinction learning, an aversive event not explicitly paired with the previously feared stimuli will result in return of fear (Rescorla & Heth, 1975). For example, if a patient with agoraphobia avoids driving for fear of having a panic attack, exposures to driving