

# Part VII

## Treatment of Anxiety Disorders: State of the Art

# Exposure Therapy

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## Introduction

Exposure therapy represents a collection of potent therapeutic strategies based on an evolving science of fear attenuation. Having people confront feared objects, situations, and activities dates back to 1924 when Mary Cover Jones first helped 3-year-old Peter overcome his fear of white rabbits through the repeated, graduated presentation of a white rabbit while simultaneously presenting Peter's favorite food. Thirty years later, Joseph Wolpe published his seminal work describing remarkable success using a similar technique, coined systematic desensitization, in the treatment of neurosis (Wolpe, 1958). From their early beginnings, exposure-based treatments have expanded procedurally to accommodate the full range of clinical presentations of pathological fear ranging from circumscribed fears to complex, debilitating clinical syndromes.

An observer not familiar with exposure therapy might be surprised to learn that administering repeated inhalations of CO<sub>2</sub> gas to a panic patient, having a patient with obsessive-compulsive disorder (OCD) listen to an audiotape of frightening thoughts, having a social anxiety patient intentionally seek out repeated rejection from members of the opposite sex, or encouraging a trauma patient to repeatedly recount a traumatic memory, are all examples of exposure therapy. Further, one might wonder what these divergent strategies have in common to warrant their categorization as exemplars of this potent set of therapeutic techniques.

We have organized this chapter around a series of key questions to address the nature, clinical application, efficacy and effectiveness, and change mechanisms of exposure therapy. We further address whether changing procedural parameters of exposure therapy influence its efficacy, and whether exposure therapy can be enhanced by combining it with other psychological or pharmacological strategies. We conclude by offering several recommendations for future research.

## **What is Exposure Therapy and How Does It Differ from Other Treatments?**

### **Central features of exposure therapy**

Exposure-based treatments share both an overarching strategy and set of therapeutic goals. The strategy, of course, is encouraging the patient to confront fear-eliciting stimuli. The application of this general strategy varies as a function of the nature of the feared stimulus (i.e., external object or situation vs. internal thought, image, memory, or somatic reaction) and how confrontation is achieved (e.g., in vivo vs. imaginal).

In its many variations, exposure therapy shares the following common goals: (a) to reduce emotional distress, (b) to eliminate anxiety-promoting phobic behavior, (c) to correct faulty threat appraisals, (d) to enhance patients' capacity to tolerate anxiety, and (e) to improve patients' quality of life. Note that other anxiety disorder treatments (e.g., pharmacotherapy, insight-oriented psychotherapy) target several of these same goals despite using techniques that bear little resemblance to those employed in exposure therapy.

### **How does exposure therapy differ from cognitive-behavioral therapy?**

This is a common question asked by clinicians in training. Students often embrace the misconception that exposure therapy is exclusively "behavioral," cognitive therapy (CT) is exclusively "cognitive," and that the treatment techniques are procedurally distinct. In reality, there is significant procedural overlap, which is why these therapies are commonly subsumed under the broader rubric of cognitive-behavioral therapy (CBT). Importantly, exposure therapy and CT/CBT share the core therapeutic strategy of encouraging patients to systematically confront their feared targets, but how this general strategy is executed differs between the two approaches. The CBT therapist will often have the patient conduct planned confrontations with her feared targets as a means for correcting her dysfunctional anxiety-maintaining beliefs (e.g., having a cardiac anxiety patient run up and down stairs to disconfirm the faulty belief that an increased heart rate will bring on a heart attack). In contrast, the exposure therapist is more likely to provide the patient a treatment rationale based on principles of extinction and habituation, emphasizing how repeated confrontations with the feared target will lead to a gradual reduction in fear.

The two approaches also differ in their use of cognitive interventions. For example, in the course of conducting exposure therapy, the clinician is likely to also employ ancillary cognitive procedural elements (e.g., discussing the patient's perceived threats in the process of identifying exposure targets). In contrast, the CT/CBT therapist, as illustrated in the treatment manuals developed by David Barlow and his colleagues (Barlow, 2000, 2004; Craske & Barlow, 2006), or Edna Foa and her colleagues (Foa, Hembree, & Rothbaum, 2007; Foa & Kozak, 2004), may engage the patient in a more formal process of cognitive restructuring and utilize one or more anxiety management strategies such as relaxation training or breathing retraining. Still, exposure

therapy may be viewed as the central procedural technique with these other therapeutic strategies aimed at enhancing the effects of exposure. Thus, the procedural differences between exposure therapy and CT/CBT may be more a matter of emphasis.

## Variations of Exposure Therapy

As mentioned, exposure therapy for anxiety disorders is comprised of a set of related techniques based on an overarching strategic principle and common set of treatment goals. In this section, we present a taxonomy of exposure therapy procedures organized by the stimulus class of the exposure target. We decided against organizing the various exposure techniques by anxiety disorder, because some exposure procedures are routinely used in the treatment of more than one anxiety disorder and, conversely, multiple exposure techniques are often used in treating one specific anxiety disorder. So, for the sake of conceptual clarity and efficiency, we describe the most common exposure procedures based on the nature of the feared stimulus (i.e., external object or situation, somatic sensation or reaction, senseless thought or image, worry, or traumatic memories; see Table 35.1).

### Exposure to external objects and situations

Over the years, many procedural variations with a myriad of labels have appeared from research centers in the United States and Europe. Although these treatments differ on

**Table 35.1** Variations of exposure therapy

| <i>Stimulus target</i>   | <i>Examples of specific treatments</i>  | <i>Anxiety disorder applications</i>  |
|--|---|---------------------------------------|
| External objects, activities, and situations (including social situations)                               | In vivo exposure<br>Prolonged exposure<br>Exposure and response prevention<br>Imaginal exposure<br>Virtual reality exposure | SP, AG, SAD, PTSD<br>OCD<br>PTSD, OCD |
| Bodily sensation or reaction (e.g., heart racing or pounding, lightheadedness, breathlessness, blushing) | Interoceptive exposure  | PD/PDA, SAD                           |
| Threatening thought or image (e.g., harming others, contamination)                                       | Imaginal exposure with therapist<br>Thought exposure via loop tape  | OCD, GAD                              |
| Anxiety-eliciting worries  | Worry exposure  | GAD                                   |
| Traumatic memories   | Prolonged exposure EMDR<br>Emotional writing  | PTSD                                  |

*Note.* SP = specific phobia; AG = agoraphobia; SAD = social anxiety disorder; PTSD = posttraumatic stress disorder; OCD = obsessive-compulsive disorder; PD = panic disorder; PDA = panic disorder with agoraphobia; GAD = generalized anxiety disorder; EMDR = eye movement desensitization and reprocessing.

one or more exposure parameters, such as presentation (i.e., in vivo vs. imaginal), use of therapist modeling of exposure tasks, spacing of exposure sessions, intensity (i.e., graded vs. ungraded), assistance (i.e., therapist-assisted vs. self-directed), and use of anxiety control strategies such as relaxation or cognitive techniques, they each have in common the core strategy of having the patient repeatedly confront objectively harmless fear-eliciting cues.

### Exposure to social evaluative situations

Social anxiety disorder (i.e., social phobia; SAD) is the most prevalent of all anxiety disorders (Ruscio et al., 2008). Although patients presenting with social anxiety differ greatly with respect to both the number and type of fear- and avoidance-eliciting situations, they share the central feature of exaggerating the likelihood and/or severity of being judged negatively by others (Clark & Wells, 1995; Foa, Franklin, Perry, & Herbert, 1996). Conducting exposure therapy for social anxiety presents several challenges for clinicians. One challenge is the unpredictability of other human beings. Because exposure therapy for SAD often involves engaging the patient in social situations, the clinician has little control over how other people (i.e., the exposure targets) will respond to their patient as they attempt to carry out their exposure exercises. In dealing with this issue, the experienced clinician will prepare the patient in advance for the varying responses that others may have to them. Exposure simulations are often used early in treatment prior to introducing real-life exposures as a way for the therapist to gain greater control over the patient's experience during exposure (Heimberg & Becker, 2002).

Patients presenting with significant social skill deficits (e.g., poor eye contact, poor conversational skills) pose another significant challenge to clinicians. Because these deficits are likely to evoke negative reactions from others, clinicians should perform a careful assessment of social skills prior to introducing social exposures in the patient's natural environment, and provide social skills training prior to initiating in vivo exposure to social situations. This approach has the benefit of enhancing patients' social skills while simultaneously providing controlled exposure exercises in the context of that training.

Finally, it has been our experience that most clinicians focus their exposure therapy efforts on providing opportunities for correcting the patient's threat probability overestimation bias (e.g., perceived likelihood of being rejected at a party) while neglecting the patient's threat severity (cost) overestimation bias (e.g., "It would be horrible if someone didn't like me"). We have found that feigning techniques (e.g., having the patient intentionally act in a manner designed to bring on negative attention from others) are helpful in targeting this important appraisal bias dimension.

### Exposure to internal fear cues

So far, we have described exposure therapy procedures that encourage patients to confront fear cues that reside outside of the individual. However, some presentations of pathological fear are linked not to external environmental cues but, rather, to cues

residing within the person. These internal fear cues may take several forms, including somatic sensations and reactions, thoughts, images, worries, and memories. In this section we describe some common exposure procedures for helping patients who present with pathological fears of these various internal cues.

*Exposure to somatic reactions (interoceptive exposure)* Dating back to the early 1970s, fear researchers theorized that somatic cues associated with arousal could become conditioned stimuli for anxiety due to the repetitive pairing of arousal cues with anxiety states (Evans, 1972; Razran, 1961). Coming from a more clinical perspective, Goldstein and Chambless (1978) outlined a theory based on the earlier work on interoceptive conditioning and their clinical observations that PD/PDA patients tend to perceive certain bodily cues as signals of an impending panic attack. They asserted that this observed “fear of fear” pattern was the central feature underlying agoraphobia.

Early efforts to have anxiety patients intentionally elicit somatic cues of arousal with the goal of fear attenuation were first reported by Bonn and colleagues (Bonn, Harrison, & Rees, 1973), who treated 33 patients presenting with “free-floating” anxiety by administering the panic provocation agent sodium lactate (5 ml/kg body weight) over a 20-minute period twice per week for 3 weeks. During the lactate challenges, patients were also encouraged to recognize the somatic reactions while tolerating them. Results of their “psychological flooding” technique resulted in significant improvements in all but 5 of the 33 patients.

Using a different panicogenic agent, Griez and van den Hout (1983) reported the results of a case study in which a patient with PDA was administered repeated inhalations of 35% CO<sub>2</sub>/65% O<sub>2</sub> in an effort to expose the patient to an intense respiratory perturbation with the goal of attenuating the patient’s conditioned fear to these respiratory cues. With the support of other early proof-of-concept work and demonstrations of therapeutic effects (e.g., Griez, Lousberg, van den Hout, & van der Molen, 1987; van den Hout & Griez, 1984), inhalation of CO<sub>2</sub>-enriched air has become a commonly used interoceptive exposure procedure in a number of centers in the United States and Europe.

Using less dramatic somatic perturbation strategies (e.g., voluntary hyperventilation, running in place, and breathing through a straw), Barlow and Cerny (1988) more formally introduced interoceptive exposure as a central procedural element in the treatment of PD/PDA. In clinical practice, the application of interoceptive exposure follows three sequential stages: (1) careful assessment of the patient’s idiosyncratic somatic fear cues; (2) individually tailored graduated exercises designed to repeatedly elicit the patient’s feared somatic cues under the supervision of the therapist; and (3) homework implementing the specific interoceptive exercises in a graduated fashion. Table 35.2 presents the more common interoceptive exposure strategies used in the treatment of panic.

*Exposure to threatening thoughts, images, and memories* For many forms of pathological anxiety, the primary fear-eliciting cue involves a threatening thought, image, or memory. Examples include the OCD patient who experiences crippling anxiety and phobic behavior in response to the thought or image of strangling his 4-year-old

**Table 35.2** Examples of interoceptive exposure (IE) strategies

| <i>Fear domain</i> | <i>Specific IE task</i>   | <i>Specific cues elicited</i>                          |
|--------------------|---|--|
| Respiratory        | Breathing through a straw, voluntary hyperventilation, CO <sub>2</sub> inhalation, stair climbing       | Breathlessness, air hunger                             |
| Cardiac            | Running in place, stair climbing, ingestion of caffeine and herbal stimulants                           | Heart racing, heart pounding, breathlessness, sweating |
| Vestibular         | Head shaking side to side, 30 seconds<br>spinning in chair, 15 seconds<br>twirling in place             | Dizziness  |
| Dissociation       | Stare at door on wall, stare at oneself in mirror, audio-photoc stimulation, voluntary hyperventilation | Derealization, dissociation                            |
| Gastrointestinal   | Eat spicy foods, smell rotten eggs, place finger on the back of tongue                                  | Nausea   |

daughter, the PTSD patient who continues to experience debilitating emotional disturbance in response to unrelenting, intrusive memories of a horrific physical assault, or the GAD patient who spends much of their waking day engaged in needless, uncontrollable worry about their job, interpersonal relationships, and health. In each of these examples, the primary fear-eliciting cue is an internal “mental event” that is only directly observable by the patient.

Fortunately, several variants of exposure therapy have been developed for helping anxiety patients whose primary threat disturbance involves one or more fear-eliciting mental threat cues. These include prolonged exposure therapy for PTSD (Foa et al., 2007), imaginal exposure for OCD (Foa & Kozak, 2004), and worry exposure for GAD (Craske, Barlow, & O’Leary, 1992). The interested reader should consult these sources for a detailed description of each of these interventions.

Successful implementation of any of these exposure protocols to internal mental fear cues involves several important procedural elements. These include: (a) providing the patient a compelling rationale for exposure; (b) careful assessment of the fear-eliciting target (e.g., trauma memory, worry scenario); (c) in-session therapist guidance in helping the patient perform the exercise; (d) monitoring the patient’s level of emotional engagement in the thought, image, worry, or memory; and (e) assigning and monitoring self-directed home practice of the prescribed exposure procedures.

*Presenting the rationale for exposure* Most anxiety patients use avoidance to cope with their threatening internal fear cues with the underlying misassumption that avoidance of aversive mental material will reduce their anxiety. The skilled therapist works with the patient to help them appreciate the connection between mental avoidance and anxiety (e.g., how not thinking about “X” actually works against them). This discussion often begins with examples of how avoidance of external feared situations maintains

or even increases anxiety, since patients seem to more readily grasp how avoidance of external situations ultimately leads to greater fear (e.g., avoiding high places ultimately increases fear of heights). Exposure is then presented, with examples, as a powerful “anti-avoidance” strategy that has been shown to be effective across all types of anxiety problems. An additional strategy for enhancing the patient’s appreciation for the maladaptive role of mental avoidance is to have her attempt to intentionally suppress a thought, image, or worry and report back to the therapist on the outcome of the experiment. Patients are often immediately struck with the near impossibility of the task, and even those who are momentarily successful experience rebound of suppressed material once their mental effort is relinquished (Wenzlaff & Wegner, 2000).

*Assessment of the patient’s threatening mental fear targets* A thorough assessment of internal fear cues is a critically important step prior to initiating actual imaginal exposure. Skilled clinicians understand that anxiety patients differ significantly in their insight, capacity, and willingness to communicate about their feared thoughts, images, memories, and worries. An effective and sensitive assessor remains flexible and collaborative with the patient with respect to the pace and depth of assessment, with the goal of soliciting a candid portrayal and obtaining an accurate understanding of the feared mental material. Perhaps most importantly, they convey to the patient understanding of and appreciation for the patient’s distress and helps the patient see the huge potential payoff for moving forward in treatment.

*Conducting in-session therapist-guided exposure* There is mounting evidence that therapist modeling and guidance can play an important role in the success of exposure treatment (Gloster et al., 2011; Williams & Zane, 1989). Although not surprising, therapists often underestimate the level of guidance and supervision needed for some patients to successfully execute the prescribed exposure procedures. In part, this may be due to questionable assumptions on the part of the therapist (e.g., “If I explain a procedure clearly, my patient should understand the procedure,” or “If my patient understands my instructions, they should be able to successfully execute them”).

*Assisting the patient to optimally engage in imaginal exposure* Based on what we know about emotional processing of feared material, failure to emotionally engage the patient during exposure is likely to render the exposure less effective (Foa, Huppert, & Cahill, 2006). Consequently, the clinician should be vigilant to signs that the fear network is activated during exposure. Several strategies can be helpful in this regard. First, obtaining fear or distress ratings during the exposure trial can be used as one source of evidence that the patient is achieving a sufficient level of emotional activation. A second strategy is to observe the patient’s postural movements and facial expressions during the exposure trial. This strategy can be particularly helpful in cases where the patient appears to be overestimating their subjective emotional distress. In cases where patients are not sufficiently emotionally engaged during exposure, the therapist should explore with the patient possible causes for the insufficient activation such as unintentional distraction or intentional mental avoidance. An alternative strategy is to offer supportive coaching during the trial by instructing the patient to focus complete attention on the feared stimuli using all their sensory modalities.



Although rare, patients may sometimes become emotionally overengaged during imaginal exposure and achieve a level of emotional activation that is too high, thus preventing the processing of corrective disconfirming information. Should emotional overengagement occur, the therapist should facilitate distancing, for instance, by gently reminding the patient that the stimulus is just in her mind and therefore not dangerous, and that she is safe in the room with the therapist. If the level of activation remains too high, the therapist should have the patient take a brief time-out and perhaps repeat the exposure with a stimulus target slightly lower on the patient's fear hierarchy.

*Assigning and monitoring exposure homework* Once the patient has provided evidence that they have mastered the procedure with the therapist, the patient should be provided specific instructions for home practice between sessions. These instructions should outline the specific procedures to be practiced along with the expected duration and frequency of exposure. A recording form to monitor their practice should also be provided. At our center, we use an online web survey service that facilitates the design and data monitoring of patients' exposure practice between sessions.

## How Does Exposure Therapy Work?

This question has captured the interest of clinicians and researchers alike. Not surprisingly, the various theories of how exposure works are integrally linked to theories of pathological fear. In this section, we review several of the major theories relevant to the question of how exposure therapy works. We begin with early conditioning theories, followed by contemporary conditioning theories, cognitive appraisal theories, and emotional processing theory. Note that all the theories share the basic assumption that the change mechanisms governing the effects of exposure-based therapies operate equally across the full spectrum of pathological fear conditions.

### Early conditioning theories

*Reciprocal inhibition theory* In 1958, Joseph Wolpe published his seminal book entitled *Psychotherapy by Reciprocal Inhibition* in which he laid out a theory for how systematic desensitization works. The central tenet of his theory was that pathological fear could be eliminated by pairing a patient's fear response with a physiological response that was incompatible (i.e., antagonistic) to the fear response. Wolpe believed that repeated pairings of the patient's fear response with the incompatible response (i.e., relaxation) would lead to a gradual weakening (i.e., inhibition) of the patient's fear response and a gradual strengthening of the antagonistic response. He referred to this process as reciprocal inhibition. Not surprisingly, others put Wolpe's theory to the test by conducting experiments in which the patient confronted their feared situation, but without using relaxation as an incompatible response. Results of these experiments showed that relaxation was not necessary to achieve meaningful fear reduction, and

thus called into question Wolpe's reciprocal inhibition theory (see Kazdin & Wilcoxon, 1976, for review).

*Habituation theory* Others have argued that exposure therapy works through a process of emotional habituation (e.g., Marks, 1978). Habituation is a form of nonassociative learning that is characterized by a "*temporary*" decrease in the reaction to a stimulus in response to repeated presentations of the stimulus. Habituation is not exclusive to humans; rather, it is ubiquitous across species. Decades of research have illuminated factors influencing the speed and durability of habituation. Some of these include: (a) frequency of stimulus presentation (e.g., increased frequency leads to a greater decrement in emotional response); (b) intensity of the stimulus presented (e.g., greater stimulus intensity leads to greater habituation); and (c) presentation of a new stimulus during the latter stages of habituation training (e.g., introduction of a new stimulus can lead to increased responding to the previously habituated stimulus).

Several prominent anxiety theorists have criticized the use of habituation as an explanatory mechanism to account for the fear reduction observed during exposure therapy. For example, Bandura (1977) has argued that habituation cannot be an explanatory mechanism of fear reduction because of the circularity inherent in inferring that habituation has caused a reduction in fear when the reduction in fear is the index that habituation has taken place. Rachman (1990) offered several limitations of habituation as a theory of how exposure therapy works. He noted that habituation theory has difficulty accounting for the persistence of some pathological forms of fear since one might expect that naturally occurring habituation would take place as a result of repeated confrontation with the feared target. He also pointed to the challenge of explaining the often-observed persistence of fear reduction over months or years following exposure therapy, or fear reduction occurring by the mere provision of information.

### Contemporary learning theory

*Fear extinction as new context-dependent learning* Over the last few decades, scientists investigating fear extinction learning have come to the general consensus that fear extinction training (i.e., exposure therapy) does not result in an erasure of the original fear memory, but rather results in the formation of a new memory that interferes with the original fear memory and its expression (Bouton, 2000; Bouton & Bolles, 1979; Bouton & Swartzentruber, 1991; Chelonis, Calton, Hart, & Schachtman, 1999; Craske et al., 2008; Dickinson, 1980).

Woods and Bouton (2008) outline six fear recovery effects observed in research on fear attenuation in animals supporting the view that fear extinction learning does not erase the original fear memory. These include: (a) return of fear following extinction training due to the mere passage of time (spontaneous recovery; Pavlov, 1927); (b) return of fear following a change in context (renewal; Bouton & Bolles, 1979); (c) return of fear following the presentation of the unconditioned stimulus (reinstatement; Rescorla & Heth, 1975); (d) faster reacquisition of fear after extinction training

(Bouton & Swartzentruber, 1989); (e) summation of residual excitation during a compound test probe following extinction training of two stimuli independently (Reberg, 1972); and (f) data suggesting that memories for extinction, like other episodic memories, are vulnerable to retrograde amnesia (Briggs & Riccio, 2007).

*Strategies for enhancing fear extinction learning* Based on the assumption that context-dependent inhibitory learning drives the effects of exposure therapy in humans, Craske and colleagues (2008) provides a thoughtful set of suggested strategies for enhancing the effects of exposure therapy. The strategies are divided into those that might assist in the creation of a robust inhibitory memory versus those aimed at facilitating the retrieval of a new inhibitory memory. With respect to the first aim, they highlight the importance of maximizing the mismatch between patients' expected versus actual outcomes. Increasing the frequency or duration of exposures until the feared consequences are no longer seen as likely outcomes may help achieve greater mismatch. This is sometimes referred to in the animal fear extinction literature as conducting "massive extinction." The expectation of feared consequences may also be strengthened (thus creating greater mismatch) by employing multiple conditioned excitors (i.e., feared stimuli) during exposure. They also suggest that eliminating safety signals and safety behaviors will facilitate the formation of an inhibitory fear memory. The importance of fading safety aids/behaviors is discussed later in this chapter (see section entitled "Exposure Augmentation Strategies in the Treatment of Anxiety Disorders"). Finally, they suggest that cognitive enhancers (e.g., methylene blue, d-cycloserine) and the enhancement of inhibitory regulation (e.g., enhancing the functioning of brain areas implicated in fear learning, including the amygdala, hippocampus, and prefrontal cortex) might improve the formation of inhibitory fear memories (see section entitled "Augmenting Exposure Therapy with Pharmacological Agents").

Several additional strategies have been suggested to facilitate the retrieval of inhibitory memory after exposure. These strategies include: (a) increasing the number of fear stimuli during exposure (Rowe & Craske, 1998a); (b) wider spacing of treatment sessions (Rowe & Craske, 1998b); (c) conducting exposure therapy in multiple contexts (Mystkowski, Craske, & Echiverri, 2002); (d) intentionally recalling the exposure therapy context (Mystkowski, Craske, Echiverri, & Labus, 2006); and (e) providing environmental cues that were present during extinction (Collins & Brandon, 2002).

*Strategies for enhancing reconsolidation of fear memory* Exciting new research with both rodents (Monfils, Cowansage, Klann, & LeDoux, 2009) and humans (Schiller et al., 2010) suggests we may be able to facilitate attenuation of pathological fear by augmenting exposure therapy with behavioral procedures that capitalize on fear memory reconsolidation mechanisms. Researchers have found that a brief, isolated retrieval of a fear memory can destabilize associated protein structures for approximately 6 hours, after which the memory restabilizes, or reconsolidates. Extinction trials conducted within this 6-hour window could afford an opportunity to actually alter the original fear memory – a process quite distinct from the putative neural mechanisms underlying the formation of a new inhibitory memory.

In a series of well-designed experiments with rodents, Monfils and colleagues (2009) tested whether conducting extinction trials after first administering a brief retrieval trial would destabilize the original fear memory and thus provide a window of opportunity for altering the original memory through the reconsolidation update mechanism. Their procedure, called the “retrieval-extinction” procedure, consisted of (1) conducting a fear retrieval trial, (2) disengaging from the feared stimulus for a period of at least 10 minutes, and (3) conducting fear extinction trials. Results demonstrated that rats that received extinction alone exhibited classic spontaneous recovery, renewal, and reinstatement of fear, whereas rats that received the retrieval-extinction procedure prior to extinction training did not. Schiller et al. (2010) replicated these findings using a human fear-conditioning paradigm. Participants who received extinction alone (or extinction training outside the 6-hour reconsolidation window) showed classic spontaneous recovery and reinstatement of fear, whereas individuals who underwent the fear retrieval procedure followed by extinction training within the reconsolidation window showed no significant evidence of return of fear 12 months after extinction training. Replications with anxiety-disordered populations would constitute a significant breakthrough for enhancing the durability of exposure therapy for treating pathological fear.

### Cognitive appraisal theories

*Self-efficacy theory* First introduced by Albert Bandura, self-efficacy theory proposes that a person’s appraisal of his or her ability to exercise control over potential threats plays an influential role in human agency (Bandura, 1977). As applied to treatment for anxiety disorders, Bandura (1988) argues that it is enhancement of one’s sense of coping self-efficacy that governs therapeutic change in treatment – not the mere habituation of anxiety brought about by repeated exposure to feared stimuli. In a series of elegant experiments, Bandura and colleagues demonstrated that treatments that target enhancing participants’ perceived mastery to cope with phobic threats led to significantly greater reductions in phobic behavior and anxiety even after carefully controlling for the total amount of exposure to the feared target stimuli (Bandura, Jeffery, & Wright, 1974; Williams, Dooseman, & Kleinfield, 1984; Williams, Turner, & Peer, 1985). Moreover, coping self-efficacy outperforms expectations of anxiety or danger in predicting changes in phobic behavior during confrontations with phobic threats (Valentiner, Telch, Petruzzini, & Bolte, 1996).

*Expectancy theories* Several theoretical accounts of pathological fear highlight the important role played by faulty threat appraisals in the onset and maintenance of anxiety disorders. For example, Beck and Emery (1985) state, “The main problem in the anxiety disorders is not in the generation of anxiety, but in the overactive cognitive patterns (schemas) relevant to danger that are continually structuring external and/or internal experiences as a sign of danger” (p. 15). From this theoretical perspective, elimination of pathological fear is achieved by correcting *faulty* threat expectancies (e.g., “I might suffocate if I get stuck in that elevator”). Although the change mechanism is presumed to be cognitive, one of the most potent strategies for achieving that

cognitive change is to provide the patient threat-disconfirming experiences via direct encounters with the feared target. A recent meta-analysis has provided some support that changes in threat appraisal mediate the effects of exposure-based treatments on treatment outcome (Smits, Julian, Rosenfield, & Powers, 2012).

In addition to expectations of threat or danger, other theorists have highlighted the importance of anxiety expectancies in pathological fear (Kirsch, Tennen, Wickless, Saccone, & Cody, 1983; Reiss, 1980). For instance, Reiss (1980) has asserted that anxiety patients have learned to anticipate becoming anxious in specific fear-eliciting situations and because the experience of anxiety is aversive, the expectation of its occurrence can be self-fulfilling. Extending this argument to fear reduction, Kirsch (1990) suggested that exposure therapy or any therapeutic intervention that successfully reduces expectations of anxiety should lead to fear reduction through its effects on reducing patients' expectation of fear. Indeed, data from our laboratory investigating potential mediators of in vivo exposure treatment for SAD were consistent with fear expectancy theory, demonstrating that anxiety expectancies, but not perceived consequences of anxiety symptoms, mediated the changes observed during in vivo exposure treatment (Smits, Rosenfield, McDonald, & Telch, 2006).

*Emotional processing theories* Unlike cognitive appraisal theories, emotional processing theories of fear reduction conceptualize anxiety pathology as structures in memory that are responsible for the subjective, behavioral, and physiological manifestations of fear. According to Lang (1977, 1984), these structures comprise stimulus, response, and meaning propositions which subserve coping with danger and perceived threat. Activation of the fear structure occurs when information is received that matches one or more of the stimulus, response, or meaning elements contained in the fear network. Building on Lang's bio-information theory of fear, Rachman (1980) defined emotional processing as "a process whereby emotional disturbances are absorbed, and decline to the extent that other experiences and behavior can proceed without disruption" (p. 51). He recommends using test probes (i.e., presenting material consistent with the original fear structure) after the emotional disturbance has declined to index the degree to which emotional processing has occurred. Rachman also described factors that either promote (e.g., high perceived self-efficacy) or impede (e.g., fatigue) emotional processing.

Embracing Lang's propositional network model of fear, Foa and Kozak (1986) expanded on Rachman's description of emotional processing by providing greater specificity on (a) what constitutes pathological fear and (b) the processes governing its modification. Unlike adaptive fear states, pathological anxiety is characterized by associations between stimulus, response, or meaning propositions that fail to reflect reality. Consequently, in information-processing terms, the danger "program" is activated needlessly. Accordingly, exposure therapy exerts its beneficial effects on pathological fear by both assisting in the activation of the fear structure and the integration of new corrective information that is incompatible with the pathological elements of the fear structure. In their original theoretical account, Foa and Kozak proposed three indicators of emotional processing during exposure therapy: (1) physiological activation during the early phase of exposure to the feared target, (2) within-session habituation of fear, and (3) between-session habituation in initial reactions to feared targets.

However, in a subsequent reworking of the theory, Foa, Huppert, and Cahill (2006) removed within-session habituation as an index of emotional processing based on a growing body of evidence suggesting that within-session habituation during exposure therapy does not predict treatment outcome (Jaycox, Foa, & Morral, 1998; Kamphuis & Telch, 2000; Telch, Valentiner, Ilai, Petrucci, & Hehmsoth, 2000).

## **Brief Overview of the Evidence Supporting the Efficacy of Exposure Therapy**

A vast number of studies have demonstrated the efficacy of exposure-based treatments across the full spectrum of anxiety disorders. This has led some to proclaim it as a major success story in clinical psychology (McNally, 2007). Although a full consideration of the efficacy and effectiveness of exposure therapy is beyond the scope of this chapter, here we provide a brief overview of the evidence pertaining to the clinical efficacy of exposure therapy.

### **How effective is exposure therapy relative to psychological “placebo”?**

Several disorder-specific meta-analyses have provided useful data for evaluating how exposure therapy stacks up to placebo treatments (e.g., Bisson & Andrew, 2009; Mitte, 2005a, 2005b; Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010; Taylor, 1996; van Balkom et al., 1997; Wolitzky-Taylor, Horowitz, Powers, & Telch, 2008), but perhaps the best available data come from a transdiagnostic meta-analysis of 27 randomized clinical trials comparing exposure-based CBT to a psychological or pharmacological placebo (Smits & Hofmann, 2008). Between-group effect sizes based on posttreatment completer analyses of anxiety disorder severity were in the medium to large range ( $ES = .73$ ) in favor of CBT over placebo. Moderator analyses revealed that effect sizes for symptom severity varied significantly across disorders, with OCD showing the largest effect (1.37), followed by acute stress disorder (ASD) (1.31), SAD and PTSD (.62), GAD (.51), and PD/PDA (.35). These findings demonstrate that CBT interventions that include an exposure component significantly outperform placebo treatments, suggesting that their clinical efficacy cannot be accounted for by nonspecific treatment effects.

### **How effective is exposure therapy relative to multicomponent CBT?**

Manualized CBT protocols have become the most widely researched and disseminated psychological approaches in the treatment of anxiety (e.g., Craske, Antony, & Barlow, 2006; Craske & Barlow, 2006; Foa et al., 2007; Foa & Kozak, 2004; Heimberg & Becker, 2002). Although exposure is a core common element, additional strategies are included (e.g., cognitive restructuring, breathing retraining) based on the premise that multiple components will produce additive gains. Yet, a number of clinical trials have shown no appreciable difference in efficacy between exposure therapy and multicomponent CBT in the treatment of OCD (Cottraux et al., 2001; McLean et al., 2001; van Oppen et al., 1995; Vogel, Stiles, & Götestam, 2004; Whittal, Thordarson,

& McLean, 2005), panic disorder with agoraphobia (Burke, Drummond, & Johnston, 1997; Williams & Falbo, 1996) and without agoraphobia (Arntz, 2002), and PTSD (Foa, Hembree, et al., 2005; Marks, Lovell, Noshirvani, Livanou, & Thrasher, 1998; Resick, Nishith, Weaver, Astin, & Feuer, 2002). These findings converge with meta-analytic reports of negligible between-treatment differences (e.g., Abramowitz, Taylor, & McKay, 2005; Mitte, 2005a).

These conclusions should be qualified, however, considering certain conditions may be specifically and better treated with exposure techniques (e.g., panic with severe agoraphobia; Williams & Falbo, 1996), and there are clear benefits of multicomponent interventions. For instance, a few high-quality trials have shown clear superiority for CBT over exposure alone (e.g., Bryant et al., 2008; Bryant, Moulds, Guthrie, Dang, & Nixon, 2003), attrition rates may be reduced for patients receiving CBT over exposure alone (e.g., Abramowitz et al., 2005), and cognitive interventions may be more appropriate for patients with certain subtypical presentations (e.g., OCD with minimal or no overt compulsions; Whittal, Woody, McLean, Rachman, & Robichaud, 2010).

### How effective is exposure therapy relative to pharmacotherapy?

Both pharmacotherapy and exposure therapy have amassed impressive empirical support for their respective clinical efficacy.<sup>1</sup> Several disorder-specific meta-analyses have addressed this question (e.g., Clum, Clum, & Surls, 1993; Eddy, Dutra, Bradley, & Westen, 2004; Fedoroff & Taylor, 2001; Gould, Otto, & Pollack, 1995; Mitte, 2005a, 2005b), but there is a general lack of consensus, partly due to disorder-specific receptiveness to various treatments, use of different analytic strategies, study inclusion criteria, and follow-up periods. Results of a transdiagnostic meta-analysis of 24 clinical trials directly comparing CBT and pharmacotherapy (Bandelow, Seidler-Brandler, Becker, Wedekind, & Rüther, 2007) found negligible differences in short-term efficacy between the two modes of treatment ( $ES = .15$ ). However, results of other meta-analyses (Gould, Buckminster, Pollack, Otto, & Yap, 1997; Gould et al., 1995) and several high-impact clinical trials (e.g., Barlow, Gorman, Shear, & Woods, 2000; Clark et al., 1994; Foa, Liebowitz, et al., 2005) suggest that when follow-up periods are considered, relative to the more durable effects of psychological treatments, the benefits of pharmacotherapy tend to attenuate over time, and relapse is common. Further, considering the high rates of attrition associated with pharmacotherapy, drug treatment may be generally less tolerable (e.g., Gould et al., 1997; Mitte, 2005a). This is particularly problematic as successful pharmacotherapy is best implemented as a longer-term intervention due to the observed high rates of relapse following discontinuation and clear benefits of continuing treatment (Donovan, Glue, Kolluri, & Emir, 2010). Also, many patients receive drug treatments that are grossly inadequate in both dose and duration (e.g., Cowley, Ha, & Roy-Byrne, 1997; Weilburg, O'Leary, Meigs, Hennen, & Stafford, 2003).

### How effective is exposure therapy in clinically representative settings?

As evidence began to mount supporting the clinical efficacy of exposure-based therapies from tightly controlled randomized controlled trials (RCTs), skeptics questioned

whether the methodological features inherent in RCTs (e.g., use of homogeneous patient samples, strict training and monitoring of therapists) greatly limit conclusions about how effective these treatments are when transported to outpatient clinics in the community (Persons & Silberschatz, 1998). Fortunately, over the past decade, significant progress has been made in the number and quality of anxiety disorder treatment effectiveness studies (for an excellent review see Stewart & Chambless, 2009). Note that these studies do not evaluate the effectiveness of exposure therapy in isolation but rather in the context of a broader, multicomponent CBT treatment that includes exposure as an important therapeutic element.

Overall, effectiveness studies of CBT have demonstrated comparable (e.g., Deacon & Abramowitz, 2006; Gaston, Abbott, Rapee, & Neary, 2006; McEvoy, Nathan, Rapee, & Campbell, 2012; Sharp, Power, & Swanson, 2000) and durable effects (e.g., Foa, Liebowitz, et al., 2005), with successful administration to challenging clinical populations and disadvantaged minority groups (e.g., Feske, 2001; Friedman, Braunstein, & Halpern, 2006). These data are even more impressive when one considers the practical constraints on community clinicians, and the minimal supervision and training they receive (e.g., Gillepsie, Duffy, Hackmann, & Clark, 2002; Foa, Liebowitz, et al., 2005; Levitt, Malta, Martin, Davis, & Cloitre, 2007).

In their meta-analysis of 56 effectiveness studies of CBT across anxiety disorders, Stewart and Chambless (2009) found large pre- to post- effect sizes (.83 to 2.59) across studies. Moreover, large controlled effect sizes (.76 to 1.83) were obtained across the six studies that included a control group. Further, benchmarking against selected efficacy studies showed that exposure-based treatments for anxiety disorders are about as effective when delivered in the real world as they are when delivered in the context of more well-controlled efficacy studies conducted in research settings.

Unfortunately, despite the evidence that exposure therapies work in the real world, most anxiety disorder patients do not receive them. The use of supportive counseling is common, whereas exposure therapy and other evidenced-based interventions are less common (e.g., Becker, Zayfert, & Anderson, 2004). Few clinicians are trained in the implementation of exposure-based treatment, many prefer individualized over manualized treatments, and concerns of adverse reactions to exposure treatments persist (Becker et al., 2004; Cahill, Foa, Hembree, Marshall, & Nacash, 2006). Accordingly, many have called for greater dissemination of exposure therapy (Barlow, Levitt, & Bufka, 1999; Shafraan et al., 2009), which ultimately requires clinical psychology and psychiatry training programs to incorporate training in exposure therapy as an integral component for student clinicians.

## **Investigation of Exposure Parameters**

Given the many procedural variations of exposure therapies, it makes sense to ask whether certain variations of exposure administration are more effective than others. In this section, we provide a brief overview of research examining five distinct exposure therapy parameters: (1) the spacing of exposure therapy sessions, (2) the degree to which feared cues are introduced gradually, (3) whether exposure is administered



in groups or individually, (4) whether feared cues are confronted in vivo or in imagination, and (5) the level of therapist involvement.

### Massed vs. spaced

What's the optimal spacing of exposure therapy sessions? Some researchers have suggested that spacing exposure sessions closer together (massed exposure) may be superior to exposure sessions spaced further apart (spaced exposure) because smaller inter-trial intervals reduce the opportunities for avoidance (Foa, Jameson, Turner, & Payne, 1980). Alternatively, others have proposed that time serves as a context across which extinction learning may generalize (Bouton, 1993; Bouton & Swartzentruber, 1991). In this view, spacing sessions further apart may increase generalization, thereby enhancing the effectiveness of treatment.

A number of studies have investigated the effects of manipulating the spacing of exposure sessions. For instance, two studies of fear of animals (Ramsay, Barends, Breuker, & Kruseman, 1966; Rowe & Craske, 1998b) and one study of fear of public speaking (Tsao & Craske, 2000) found that wider spacing of sessions confers an advantage, whereas one agoraphobia study found an advantage of massed practice (Foa et al., 1980). However, most studies comparing various intervals of spacing sessions of exposure (or CBT with an exposure component) have produced null results. These include studies of specific phobia (e.g., Lang & Craske, 1999; Ning & Liddell, 1991; Öst, Alm, Brandberg, & Breitholtz, 2001; Öst, Brandberg, & Alm, 1997; Öst, Hellstrom, & Kaver, 1992), OCD (Abramowitz, Foa, & Franklin, 2003; Emmelkamp, van den Heuvel, Ruphan, & Sanderman, 1989), and PDA (Bohni, Spindler, Arendt, Hougaard, & Rosenberg, 2009; Chambless, 1990). Thus, the bulk of the evidence suggests the spacing of exposure sessions makes little difference in treatment outcome. However, because of the small sample sizes and the wide range of spacing intervals in the studies published to date, strong conclusions are premature.

### Graduated vs. nongraduated

In the early research of the 1960s and 1970s, researchers tackled the question of whether exposure treatment was more effective if conducted using a less fear-provoking, graduated approach versus a more intense fear-provoking, nongraduated approach. Several variants of graduated exposure appeared with different labels (e.g., systematic desensitization, reinforced practice, successive approximation, self-observation), which all shared the feature of having the patient gradually and progressively confront more challenging fear-provoking targets. In contrast, nongraduated exposure approaches, including implosion therapy and flooding, encouraged the patient to confront maximally provocative fear-eliciting cues for the full duration of treatment.

Studies directly comparing graduated and nongraduated exposure have shown an advantage of graduated treatment in specific phobia (De Moor, 1970; Rachman, 1966; Willis & Edwards, 1969) and OCD (Boersma, Hengst, Dekker, & Emmelkamp, 1976). In contrast, one study with speech phobia (Kirsch, Wolpin, & Knutson, 1975) and one study with mixed phobias (i.e., specific phobia and agoraphobia;

Boulougouris, Marks, & Marset, 1971) found flooding to be superior to graduated treatment. However, other research with mixed anxiety disorders (Crowe, Marks, Agras, & Leitenberg, 1972; Gelder et al., 1973), OCD (Abramowitz, 1996), and agoraphobia (Emmelkamp, 1974; Everaerd, Rijken, & Emmelkamp, 1973) has shown little to no difference in treatment efficacy as a function of this exposure parameter.

Drawing strong conclusions from the available research is complicated by the use of small sample sizes, crossover designs, and reliance on completer analyses (e.g., Boulougouris et al., 1971; Crowe et al., 1972; Everaerd et al., 1973). Findings across disorders are mixed, although there seems to be a benefit of graduated treatment for specific phobia. Furthermore, graduated exposure offers significant practical advantages over nongraduated treatment, which include reduced treatment refusals and dropouts, minimization of sensitization during exposure, and ease of implementation using a self-directed format. It is likely that these advantages have led to the adoption of graduated exposure in most contemporary, evidenced-based treatments for anxiety disorders.

### Group vs. individual

Another parameter of exposure that might influence treatment outcome is whether treatment is conducted individually or in groups. While individual formats may offer more focused attention on the individual patient, group formats provide a number of benefits, most notably, improved cost-effectiveness. Over a dozen studies have investigated the relative efficacy of individual vs. group exposure-based treatments, with diverging results.

For instance, several OCD treatment studies suggest that individually administered exposure therapy alone or in the context of CBT offers a slight short-term advantage over group treatment (Anderson & Rees, 2007; Cabedo et al., 2010; Jaurrieta, Jiménez-Murcia, Menchón, et al., 2008; Jónsson, Hougaard, & Bennedsen, 2011; O'Connor et al., 2005). Similarly, one open trial (Néron, Lacroix, & Chaput, 1995) and one RCT in panic disorder (Sharp, Power, & Swanson, 2004) found an advantage of individual over group formats. In contrast, other OCD trials have shown negligible differences between the two exposure modalities in both the short term (Barrett, Healy-Farrell, & March, 2004; Fals-Stewart, Marks, & Schafer, 1993) and at follow-ups ranging from 1 to 7 years (Barrett, Farrell, Dadds, & Boulter, 2005; Jaurrieta, Jiménez-Murcia, Alonso, et al., 2008; Jónsson et al., 2011; O'Leary, Barrett, & Fjermestad, 2009; Whittal, Robichaud, Thordarson, & McLean, 2008). Importantly, studies in social phobia suggest that the efficacy of a particular treatment format may depend, in part, on the strategies emphasized in treatment. For instance, CBT protocols with a greater emphasis on exposure seem to be slightly more effective when delivered using a group format (Dogaheh, Mohammadkhani, & Dolatshahi, 2011), whereas protocols with a greater emphasis on cognitive restructuring seem to be more effective when delivered individually (Mörtberg, Clark, Sundin, & Åberg Wistedt, 2007; Stangier, Heidenreich, Peitz, Lauterbach, & Clark, 2003).

In sum, the extant literature suggests there is a slight advantage of individual over group exposure-based treatment for OCD and panic disorder, whereas there may be an advantage of group treatment for social phobia. However, group treatment offers a

number of ancillary benefits, such as improved cost-effectiveness and enhanced compliance with homework. Further, simply attending group sessions provides additional exposure for patients with significant social anxiety. It should be noted, however, that some patients may be unwilling to enter treatment or drop out prematurely due to the social evaluative demands inherent in group-administered treatments.

### Imaginal vs. in vivo

In practical terms, conducting in vivo exposure (IVE) can place a significant burden on the therapist in terms of the time and resources required for therapy. In vivo exposure (if conducted during the session) will often require leaving the therapist's office and seeking out stimuli suited to the client's idiosyncratic fears. Imaginal exposure (IE), on the other hand, is simple to conduct in the therapist's office and can easily fit within the standard 50-minute session. Comparisons of IE and IVE have been conducted in several patient populations, including specific phobia, OCD, agoraphobia, and PTSD.

Research directly comparing IE and IVE suggests they are equally effective in treating specific phobia (Hecker, 1990; Minor, Leone, & Baldwin, 1984; Rentz, Powers, Smits, Coughle, & Telch, 2003), OCD (Chambless, Foa, Groves, & Goldstein, 1982; Foa, Steketee, & Grayson, 1985), agoraphobia (James, Hampton, & Larsen, 1983; Mathews et al., 1976), and PTSD (Bryant et al., 2008). However, other studies suggest that IVE is superior to IE in the treatment of specific phobias (Bandura, Blanchard, & Ritter, 1969; Barlow, Agras, Leitenberg, & Wincze, 1970; Barlow, Leitenberg, Agras, & Wincze, 1969), OCD (Rabavilas, Boulougouris, & Stefanis, 1976), agoraphobia (Emmelkamp & Wessels, 1975), PTSD (Richards, Lovell, & Marks, 1994), and mixed anxiety disorders (including agoraphobia, social phobia, and specific phobia; Crowe et al., 1972).

Several studies have compared the combination of IE and IVE (IE+IVE) to each treatment modality alone. In comparing IVE+IE to IVE alone, some studies found the two treatments equally effective for OCD (De Araujo, Ito, Marks, & Deale, 1995), PTSD (Bryant et al., 2008), and agoraphobia (Emmelkamp, 1974; Everaerd et al., 1973; Mathews et al., 1976). However, other studies found IVE+IE superior to IVE alone in treating specific phobia (Kaloupek, 1983), OCD (Foa, Steketee, Turner, & Fischer, 1980), and secondary symptoms of OCD, such as general anxiety (Abramowitz, 1996) and depression (Rosa-Alcázar, Sánchez-Meca, Gómez-Conesa, & Marín-Martínez, 2008). Additionally, several studies have compared IVE+IE to IE alone. One agoraphobia study found an advantage for the combined treatment (Emmelkamp & Wessels, 1975). However, two other studies (Mathews et al., 1976; Bryant et al., 2008) found that combined treatment was no more effective than imaginal exposure alone.

Overall, the literature suggests that IVE alone is either equivalent or superior to IE alone. Low statistical power and the inclusion of in vivo exposure homework for patients assigned to IE may have contributed to some of the null findings observed in the studies reviewed. However, when tailoring treatment to an individual patient, the decision to use imaginal exposure, in vivo exposure, or both should take into consideration the nature of the patient's clinical presentation. For instance, OCD

patients with vivid, intrusive images may benefit most from imaginal exposure to descriptions of these images, whereas OCD patients with contamination obsessions provoked by particular situations may benefit most from in vivo exposure to these situations. Our overall recommendation from the research findings is to use in vivo exposure either alone or in combination with imaginal exposure when practically possible in order to increase the likelihood that treatment gains will generalize outside the therapist's office.

### Therapist-assisted vs. self-directed

One of the central issues in the delivery of exposure treatments for anxiety disorders is whether patients benefit more when the therapist takes a more active role in the field with the patient (i.e., therapist-assisted exposure), as opposed to providing instructions and guidance in the confines of the therapist's office (i.e., self-directed exposure). Therapist-assisted exposure may provide the necessary motivation to confront fear-provoking targets (Holden, O'Brien, Barlow, Stetson, & Infantino, 1983). Furthermore, therapist presence in the field offers the opportunity to provide instructions and modeling to ensure that exposures are conducted correctly with respect to exposure target selection, duration of exposure, and fading of safety behaviors. In contrast, self-directed exposure is more cost-efficient. Furthermore, the presence of the therapist has the potential to slow improvement by inadvertently serving as a safety cue, thereby undermining the patient's self-efficacy to manage fear-proving situations without the therapist.

Direct comparisons of self-directed or manual-directed exposure to therapist-assisted exposure have shown the latter to be more effective for specific phobia (Barlow et al., 1970; Hellström & Öst, 1995; O'Brien & Kelley, 1980; Öst, Salkovskis, & Hellström, 1991). These findings were replicated for OCD in three studies (Abramowitz, 1996; Rosa-Alcázar et al., 2008; Tolin et al., 2007), but not in a large study (van Oppen et al., 2010). Studies comparing the combination of therapist-assisted and self-directed exposure to self-exposure alone have generally provided strong support for the enhanced efficacy of the combined exposure approach. This finding has been demonstrated in social phobia (Al-Kubaisy et al., 1992; Alström, Nordlund, Persson, Harding, & Ljungqvist, 1984) and agoraphobia (Marks et al., 1983; Mavissakalian & Michelson, 1983), including a large ( $N = 369$ ) state-of-the-art randomized trial (Gloster et al., 2011). Furthermore, two studies of specific phobia comparing the combination of therapist-assisted and self-directed exposure to therapist-assisted exposure alone suggest that the addition of self-directed treatment might improve outcomes (Bandura, Jeffery, & Gajdos, 1975; Smith & Coleman, 1977).

In summary, there is overwhelming evidence that therapist-assisted exposure is more effective than self-directed exposure alone, and that the combination also outperforms self-directed exposure alone. We would like to underscore several points with respect to self-directed exposure. First, self-directed exposure instructions provided by a therapist have been shown to be no more effective than self-directed exposure guided by a computer (Ghosh, Marks, & Carr, 1988; Marks, Kenwright, McDonough, Whittaker, & Mataix-Cols, 2004) or book (Ghosh et al., 1988). Second, despite being

less potent than true therapist-assisted exposure, self-directed exposure, whether delivered via book, computer, or therapist, is highly cost-effective and does lead to significant reductions in anxiety and phobic disability for some patients. Identification of factors that predict which patients require the addition of therapist-assisted exposure should be a high priority for future research.

## **Exposure Augmentation Strategies in the Treatment of Anxiety Disorders**

Given the centrality of exposure treatment across the full spectrum of anxiety disorders, it makes sense to ask whether other treatment strategies can be added to exposure therapy to enhance its overall efficacy. In this section we address this question by briefly reviewing the following exposure augmentation strategies: (a) anxiety control strategies; (b) pharmacological strategies; (c) cognitive strategies; (d) guided mastery; and (e) fading of safety behaviors. For each type of augmentation approach, we limit our review to evidence from randomized clinical trials comparing exposure treatment alone to exposure in combination with the augmentation strategy or findings from meta-analyses that provide controlled effect sizes comparing an exposure treatment to that same exposure treatment in combination with one of the augmentation approaches listed above.

### **Augmenting exposure with anxiety management**

Anxiety management strategies vary widely, ranging from relaxation skills (e.g., diaphragmatic breathing, progressive muscle relaxation, pleasant imagery) to cognitive skills (e.g., thought stopping, cognitive restructuring) to distraction. The acquisition of skills to manage anxiety might enhance exposure therapy by instilling clients with a sense of self-efficacy or mastery (Murphy, Michelson, Marchione, Marchione, & Testa, 1998). Anxiety management might also reduce the aversiveness of exposure, potentially enhancing compliance (Meuret, Wilhelm, Ritz, & Roth, 2003). Alternatively, anxiety management techniques may detract from the efficacy of exposure if they are used as a safety behavior (e.g., using controlled breathing to avoid feared sensations of anxiety in panic disorder; Schmidt et al., 2000).

Research in the treatment of social anxiety has suggested that the addition of anxiety management techniques can enhance the outcome of exposure therapy (Borkovec & Sides, 1979; Butler, Cullington, Munby, Amies, & Gelder, 1984). In contrast, research testing the addition of stress inoculation training to prolonged exposure therapy (PE) for PTSD suggests that PE alone is superior to the combined treatment (Foa et al., 1999). The bulk of the research on the use of anxiety management strategies to augment exposure has tested the use of breathing retraining to enhance treatment for PD/PDA. Early theorists (e.g., Ley, 1985) posited that the addition of breathing retraining (BR) to reduce hyperventilation would enhance treatment. They suggested that increased hyperventilation in response to feared sensations of anxiety played a central role in causing panic attacks. However, research suggests that

exposure (or multicomponent CBT) with and without breathing retraining produces equivalent outcomes for panic disorder patients (e.g., de Ruiter, Rijken, Garssen, & Kraaimaat, 1989; Schmidt et al., 2000) and for patients high in anxiety sensitivity (Deacon et al., 2012). Furthermore, the addition of a multicomponent relaxation-training package (which included BR) does not appear to enhance exposure treatment for panic disorder with agoraphobia (Michelson, Marchione, Greenwald, Testa, & Marchione, 1996).

In summary, most of the research testing whether the addition of anxiety management enhances exposure treatments has been done with PDA/agoraphobia patients and strongly suggests that the addition of anxiety management training does not enhance the efficacy of treatment. Conclusions with respect to anxiety management augmentation for other anxiety disorders await future research.

### Augmenting exposure therapy with pharmacological agents

*Augmentation of exposure therapy with traditional pharmacological agents* Exposure and traditional pharmacological therapies are effective first-line treatments for anxiety disorders, but there is much room for improvement (Hofmann & Smits, 2008; Baldwin, Waldman, & Allgulander, 2011). An obvious enhancement strategy that has garnered mixed empirical support (e.g., Hohagen et al., 1998; Furukawa, Watanabe, & Churchill, 2006) is to combine these treatments, by administering the other when either fails, or using an initial combined approach (Smits, Reese, Powers, & Otto, 2010; Telch, 1988; Telch, Agras, Taylor, Roth, & Gallen, 1985; Telch, Teannan, & Taylor, 1983). While it is reasonable to presume this approach may produce synergistic effects, many question whether such an approach truly leads to superior outcomes (e.g., Pontoski & Heimberg, 2010), a growing literature suggests little enduring advantage (Hofmann, Sawyer, Korte, & Smits, 2009), and others caution that concomitant drug treatment may preclude receiving the full benefit from exposure therapy (e.g., Otto, Behar, Smits, & Hofmann, 2009; Otto, McHugh, & Kantak, 2010). Several reviews of combined treatments across anxiety disorders (e.g., Deacon, 2006; Pull, 2007; Choi, Rothbaum, Gerardi, & Ressler, 2010) have found no reliable conditions under which traditional pharmacotherapy may augment exposure-based treatments for anxiety disorders. Further, the trials that demonstrate superiority of combined treatments often also demonstrate the flimsy, ephemeral nature of their effects (e.g., Barlow et al., 2000), perhaps due to medication-induced, state-dependent learning, external attributions of treatment gains, undermining of coping self-efficacy, and reliance on medication as a safety behavior (Mitte, 2005a).

*Augmentation of exposure therapy with alternative pharmacological agents* Unlike traditional augmentation strategies that directly target an anxiolytic effect, an alternative augmentation approach is to use specific “cognitive enhancement” drugs that have no direct anxiety-reducing properties when administered alone, but when combined with exposure therapy facilitate the learning mechanisms governing exposure’s therapeutic effects (see Hofmann, Smits, Asnaani, Gutner, & Otto, 2011, for review). The most widely studied is the partial agonist of the N-methyl-aspartate (NMDA)

receptor, d-cycloserine (DCS). Preliminary results with DCS have shown promise (De Kleine, Hendriks, Kusters, Broekman, & van Minnen 2012; Guastella et al., 2008; Hofmann et al., 2006; Otto, Tolin, et al., 2010; Ressler et al., 2004), but findings have generally been inconsistent (cf. Kushner et al., 2007; Storch et al., 2007; Wilhelm et al., 2008).

Other agents of interest include methylene blue, yohimbine, caffeine, glucocorticoids, modafinil, endocannabinoids, and certain natural supplements (see Hofmann et al. 2011, and Farach et al., 2012, for review). Investigations of these agents are under way, but few have been published (Guastella, Howard, Dadds, Mitchell, & Carson, 2009; Meyerbröker, Powers, van Stegeren, & Emmelkamp, 2012; Mystkowski, Mineka, Vernon, & Zinbarg, 2003; Powers, Smits, Otto, Sanders, & Emmelkamp, 2009; Soravia et al., 2006), so strong conclusions cannot yet be drawn.

As results are replicated and other treatment-augmenting agents are applied, these novel combined approaches may offer benefits to both patients and providers by accelerating treatment response, reducing the number of treatment sessions needed to achieve clinical response, and freeing clinical resources so that more patients can benefit from treatment.

### Augmenting exposure therapy with cognitive strategies

Several studies have investigated whether the addition of cognitive restructuring to exposure therapy enhances treatment efficacy. This is an important area of investigation relevant to improving the efficiency and cost-effectiveness of treatment, considering that administering cognitive techniques requires both time and considerable skill. Overall, the available evidence suggests a number of factors may influence whether enhancement effects are observed, including the expertise of investigators and certain methodological considerations.

Two high-impact RCTs investigating exposure-based and cognitive therapy for PTSD suggest there is no enhancement effect of adding cognitive restructuring to exposure (Marks et al., 1998; Foa, Hembree, et al., 2005). In contrast, two other investigations support a facilitative effect. In the first of these (Bryant et al. 2003), prolonged exposure with cognitive restructuring significantly outperformed imaginal exposure that explicitly excluded cognitive interventions in reducing trauma-related symptoms and maladaptive cognitions. In a comment about this study, Foa, Hembree, et al. (2005) suggested the Bryant et al.'s results might be due to their decision to provide only imaginal exposure, whereas other studies (and evidenced-based practice) commonly employ both imaginal and *in vivo* exposure in the treatment of PTSD. This concern was addressed in a follow-up four-arm RCT in which PTSD participants were randomized to imaginal exposure (IE), *in vivo* exposure (IVE), combined IE and IVE, and combined IE, IVE, and cognitive restructuring (Bryant et al., 2008). At 6-month follow-up, those receiving the combination of exposure plus cognitive restructuring showed markedly higher rates of remission (69%) relative to the three exposure alone conditions, IE (25%), IVE (31%), and IE/IVE (27%). Particularly striking was the marked lower response rates for the exposure only treatment arms

in this study relative to the trials reported by Foa, Hembree, et al. (2005). As noted by Bryant et al. (2008), one possible factor that might have accounted for the significantly lower treatment response rates relative to those reported by Foa, Hembree, et al. (2005) was the intentional removal of the discussion component (i.e., “processing”) following exposure sessions. Thus, one reasonable conclusion from the results of these studies is that the processing component of Foa, Hembree, et al.’s original protocol, or some alternative element that provides for an exploration of the patient’s perspective on his or her trauma (e.g., formal cognitive restructuring), seems to significantly enhance treatment outcome.

Collectively, the relevant studies investigating treatments for PDA, SAD, and OCD are inconclusive as to whether exposure may be enhanced by cognitive techniques. For instance, treatment studies of PDA largely suggest that the combination of cognitive and exposure-based strategies confers no benefits beyond exposure therapy alone (Öst, Thulin, & Ramnerö, 2004; van den Hout, Arntz, & Hoekstra, 1994; Williams & Falbo, 1996), although one study supported an enhancement effect of including cognitive interventions (Michelson et al., 1996). One possible explanation for this discrepant finding is the relatively larger sample size ( $N = 92$ ) used in the Michelson et al. (1996) trial, which may have sufficiently increased statistical power to detect a “true” enhancement effect; however, a more likely explanation is the markedly increased “dose” of cognitive therapy, which constituted at least a threefold increase in therapy hours relative to the other studies. Likewise, two treatment studies of SAD provide evidence of a facilitative effect of cognitive techniques when combined with exposure therapy based on observed treatment gains on behavioral tests of avoidance and measures of phobia (Mattick & Peters, 1988; Mattick, Peters, & Clarke, 1989). However, other studies found no evidence of enhanced outcome for SAD patients assigned to exposure plus cognitive therapy relative to exposure therapy alone (Salaberria & Echeburua, 1998; Scholing & Emmelkamp, 1993a, 1993b), which is consistent with conclusions of a recent meta-analysis of 34 RCTs investigating treatments for SAD (Powers, Sigmarsson, & Emmelkamp, 2008). Finally, we identified only one small-scale treatment study for OCD that directly compared exposure alone vs. combined with a cognitive intervention (Emmelkamp & Beens, 1991). Although both treatments led to significant improvement in OCD symptoms, there was no evidence supporting an enhancement effect for the combined treatment.

In sum, the studies reviewed in this section provide somewhat of a mixed picture as to whether cognitive strategies enhance exposure treatments. Surely, the most reasonable conclusion is that under certain conditions the combination of exposure treatment and cognitive techniques shows a clear advantage over either exposure treatment or cognitive therapy alone. The conditions that contribute to this enhancement effect are not fully understood; however, it is our view that three factors, when present, may increase the likelihood of observing cognitive enhancement of exposure therapy. These are: (1) a trial with sufficient statistical power to detect a modest but still clinically significant effect size; (2) a relatively “pure” exposure treatment condition that is stripped of cognitive techniques; and (3) a principal investigator who has significant expertise in the direct application of cognitive interventions.



### Augmenting exposure therapy with guided mastery techniques

Participant modeling was first introduced by Bandura and his colleagues in a series of well-crafted experiments investigating cognitive change mechanisms governing the reduction of pathological fear (Bandura et al., 1974, 1975). As in other exposure-based treatments, participant modeling (later renamed guided mastery) encourages the patient to confront their fear-provoking situation in vivo. However, in guided mastery, the therapist plays a very active role in incorporating specific mastery-enhancing strategies to help the patient overcome their fear. These coping enhancement elements include: (a) therapist modeling coping behavior in the feared situation; (b) systematic introduction and subsequent fading of performance aids (e.g., the therapist sits next to the driving-phobic patient and then gradually fades their presence); (c) setting proximal goals and mastering subtasks to help patients manage challenging tasks (e.g., having the driving phobic drive only one exit on the highway prior to tackling multiple exits); (d) identification and elimination of defensive maneuvers (e.g., having the patient loosen their vice grip on the steering wheel); and (e) encouraging the patient to vary their performance (e.g., venture into different grocery stores).

One of the first controlled investigations of guided mastery was reported by Williams and colleagues (1984). Thirty-two patients displaying severe driving and height phobias were randomly assigned to one of three conditions: (1) guided mastery; (2) in vivo exposure alone; and (3) no-treatment control. Total amount of exposure time in the two active treatments was carefully controlled. At posttreatment, both active treatments outperformed no treatment; however, participants receiving guided mastery showed significantly greater improvement than those receiving in vivo exposure alone on multiple indices of outcome, including performance on behavioral approach tests and patient ratings of anxiety and coping self-efficacy. In a subsequent study (Williams & Zane, 1989), 26 patients with agoraphobia were randomized to receive in vivo exposure alone, exposure with guided mastery, or delayed treatment. Results again showed that after controlling for the amount of exposure, those receiving guided mastery evidenced significantly greater gains across multiple indices of agoraphobic avoidance, panic, anxiety, and self-efficacy. Subsequent studies of guided mastery have provided consistent support for its efficacy in the treatment of agoraphobia (Hoffart, 1995, 1998).

### Augmenting exposure therapy with safety behavior fading

Human beings are hardwired to engage in protective actions in the face of physical danger or nonphysical threats. Examples of such actions include wearing warm clothing when venturing outside on a winter's day, regular use of seatbelts, and the use of condoms to prevent contracting a sexually transmitted disease. However, engaging in such protective actions in the absence of any real threat seems to be a significant factor in the onset (Olatunji, Etzel, Tomarken, Ciesielski, & Deacon, 2011) and maintenance of anxiety pathology (Telch & Lancaster, 2012). Evidence that safety behaviors are conceptually linked to the idiosyncratic threats perceived by anxiety disorder patients was first reported by Salkovskis (1991). Examples of safety behaviors commonly observed in anxiety patients include the repeated checking of one's pulse

**Table 35.3** Studies examining the effects of fading safety behaviors during exposure therapy

| <i>Study</i>  | <i>Anxiety problem</i> | <i>Outcome</i>   |
|---|------------------------|------------------|
| Wells et al. (1995)                                 | Social phobia          | Enhanced outcome |
| Morgan & Raffle (1999)                              | Social phobia          | Enhanced outcome |
| Salkovskis, Clark, Hackmann, Wells, & Gelder (1999) | Agoraphobia            | Enhanced outcome |
| Kim (2005)  | Social phobia          | Enhanced outcome |
| Salkovskis, Hackmann, Wells, Gelder, & Clark (2006) | Agoraphobia            | Enhanced outcome |
| Okajima & Sakano (2008)                             | Social phobia          | Enhanced outcome |
| Taylor & Alden (2010)                               | Social phobia          | Enhanced outcome |
| Taylor & Alden (2011)                               | Social phobia          | Enhanced outcome |

for a patient with health anxiety, carrying rescue medication or other safety aids for a panic patient, or various forms of impression management tactics for the socially phobic patient (Clark & Wells, 1995).

The clinical observation that anxiety patients frequently use safety aids/safety behaviors when facing fear-provoking situations (Salkovskis, 1991; Telch, 1991), and evidence that safety behaviors may undermine the effects of exposure therapy (Powers, Smits, & Telch, 2004; Sloan & Telch, 2002) led anxiety researchers to investigate whether the fading of safety behaviors would enhance the effects of exposure therapy. Eight independent treatment studies across multiple anxiety problems have directly tested whether the systematic fading of patients' safety behaviors during exposure treatment enhances treatment outcome. As seen in Table 35.3, the studies spanned a number of anxiety problems including agoraphobia, SAD, and specific phobia. Results across all eight studies were consistent in showing that fading safety behaviors lead to significantly better outcomes than exposure without safety behavior fading (Telch & Lancaster, 2012).

Overall, the findings with respect to safety behavior fading are strikingly consistent in demonstrating that the systematic fading of safety behaviors during exposure therapy exerts a powerful facilitative effect on treatment outcome. No other exposure augmentation strategy (behavioral or pharmacological) has shown such promise.

## Conclusions and Recommendations for Future Research

Exposure therapy for anxiety disorders remains one of the major success stories in the history of psychotherapy. Despite this success, there remain significant gaps in our knowledge. We conclude this chapter with several specific recommendations for filling these gaps.

- 1 *Identification of patient characteristics that predict treatment response and dropout.* Despite decades of research investigating exposure therapy, we know very little about the characteristics that predict who will undergo exposure therapy, who

will drop out, who will respond to acute treatment, and who will maintain their clinical gains in the long term. Answers to these questions are paramount in formulating targeted and effective treatments, but require treatment studies specifically designed with these questions in mind. Careful attention to methodological requirements (e.g., *a priori* designation of putative moderator variables, attention to statistical power) and utilization of recent advances in analytic strategies for testing treatment moderators are necessary to achieve this important research objective.

- 2 *Targeting barriers to dissemination.* Recent evidence suggests that most individuals with anxiety disorders do not receive exposure therapy, despite its proven efficacy. Identifying and understanding the many factors contributing to this state of affairs should be given high research priority. In this regard, important lines of research include (1) targeting strategies for fortifying clinical training programs which provide the skills necessary for implementing best evidence-based practices, and (2) improving patients' and the public's understanding of, and receptivity to, exposure-based treatments.
- 3 *Additional research on mechanisms of change.* Although not a new idea, continued research on the processes governing the reduction of pathological fear should remain a high priority. Greater understanding of both the psychological and neurobiological change mechanisms underlying exposure's therapeutic effects will inform the development of future refinements which may serve to enhance the efficacy of this already potent set of therapeutic techniques.

## Note

1. While exposure-based therapies have well-established clinical efficacy in the treatment of specific phobias (see Wolitzky-Taylor et al., 2008), the same cannot be said for pharmacological treatments.

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