Chapter 27

Mechanisms of Action in the Treatment of Social Anxiety Disorder

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Treatment outcome studies for social anxiety disorder have provided consistent evidence for the efficacy of two modalities of treatment: cognitive behavioral therapy (CBT) and pharmacotherapy (for reviews see Belzer, McKee, & Liebowitz, 2005; Jorstad-Stein & Heimberg, 2009; Schneier, 2011). Comparison-treatment studies (e.g., Davidson et al., 2004; Heimberg et al., 1998; Otto et al., 2000), as well as a meta-analytic review of the treatment outcome literature (Gil, Carrillo, & Meca, 2001; Gould, Buckminster, Pollack, Otto, & Yap, 1997; Roshanaei-Moghaddam et al., 2011) suggest that, on average, these treatment modalities provide equivalent outcome.

Among pharmacologic treatments, the monoamine oxidase inhibitors (MAOIs), selective serotonin reuptake inhibitors (SSRIs), and benzodiazepines have the highest estimates of efficacy (Gould et al., 1997; Van Ameringen, Mancini, Patterson, & Simpson, 2009; van der Linden, Stein, & van Balkom, 2000; de Menezes et al., 2011). Among cognitive-behavioral treatments, there is evidence that exposure-based and combined cognitive-restructuring and exposure treatments can outperform cognitive interventions alone, and that these treatments are more powerful than relaxation-based treatment and social skills training alone (for reviews, see Acarturk et al., 2009; Gould et al., 1997; Feske & Chambless, 1995; Heimberg & Juster, 1995; Jorstad-Stein & Heimberg, 2009; Taylor, 1996).

In addition to approximately equal outcome, there is evidence that cognitive behavioral and pharmacological interventions are equally tolerable to patients, at least as assessed by dropout rates, with an overall 10% dropout rate for CBT and 14% for pharmacotherapy (Fedoroff & Taylor, 2001; Gould et al., 1997). Moreover, to date there appears to be little evidence for differential predictors of response for these two modalities of treatment. For example, Otto et al. (2000) examined demographic, diagnostic, and symptom-severity predictors of

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outcome in patients undergoing treatment with either group CBT or clonazepam. Severity of the disorder, as assessed by a range of social anxiety disorder symptom measures, was a reliable predictor of poorer outcome, but there was no consistent evidence for differential prediction based on treatment modality.

The fact that two different modalities of treatment have similar rates of efficacy and similar predictors of outcome presents an interesting and difficult challenge for the identification of mechanisms of action for these treatments. Given this challenge, it is helpful to consider potential mechanisms of action within a model of the cognitive, behavioral, and physiological components of social anxiety disorder that must be modified to return an individual to more-normal social functioning. Interventions may differ in the way in which one or more of these components are targeted, but for remission of the disorder, it is these patterns that must be normalized. In addition to the material presented here, discussions of the cognitive, behavioral, and affective patterns that characterize and maintain social anxiety disorder are provided by Heimberg, Brozovich, & Rapee (Chapter 24 in this volume), Clark and Wells (1995), Heimberg & Barlow (1991), Hofmann (2007), Otto (1999), and Rapee & Heimberg (1997).

A MODEL OF SOCIAL ANXIETY DISORDER

Perhaps one of the most salient features of social anxiety disorder is the negative and catastrophic expectations that socially phobic individuals bring to social situations. Fears and expectations of poor social performance ("I won't think of anything to say" or "I will be humiliated"), negative evaluations from others ("They will think I am stupid"), and uncontrollable anxiety ("I will tremble so much I won't be able to finish the talk") predominate. Accordingly, models of social anxiety disorder attend to the emotional consequences of such cognitions and other distortions in the interpretation and processing of socially relevant information (Clark & Wells, 1995; Heimberg, 2001; McNally, 1996; Rapee & Heimberg, 1997; see also Heimberg, Brozovich, & Rapee, Chapter 24 in this volume). The natural result of these fears and negative expectations is that the socially phobic individual enters social situations with anxious apprehension: hoping that negative outcomes will not occur, but being excessively vigilant to feared negative or catastrophic outcomes. In addition to the fear of negative evaluation, recent studies also provide support for a more general fear of evaluation which would include fears of positive evaluation, suggesting that socially phobic individuals are not necessarily preoccupied by a desire for praise but rather a desire to remain inconspicuous (Weeks, Heimberg, Rodebaugh, Thomas, & Norton, 2008).

Inherent to many of these fears are the tendencies to exaggerate the perceived consequences of performing inadequately in social situations, to underestimate one's ability to cope in social situations, and to rehearse self-defeating and global attributions about oneself and future social behavior (Clark &Wells, 1995; Rapee & Heimberg, 1997). Salient negative core beliefs (see Beck,

Emery, & Greenberg, 1985) such as "I am worthless," "Others will not like me," or "Because I have anxiety, I am inadequate," are at the heart of social anxiety disorder, and they become activated when individuals with social anxiety disorder are confronted by social situations. Moreover, these beliefs may include catastrophic interpretations of the meaning of minor mistakes or anxiety in social situations that further amplify negative emotional reactions to these events. Particularly important may be beliefs about the meaning of anxiety itself. Fears that anxiety symptoms will be perceived by others form their own factor on social anxiety scales (Safren, Heimberg, & Turk, 1998), and there is evidence that socially anxious individuals overestimate the degree to which their anxiety symptoms are noticed by others (e.g., McEwan & Devins, 1983). The belief that anxiety in social situations is a sign of personal failure is one of three "amplifying cognitions" identified by Otto (1999).

In addition to increasing anxiety, these negative and catastrophic expectations also act to direct attention to signs of negative outcomes. This vigilance to perceived danger has the additional destructive effect of distracting individuals from more-relevant information processing. Instead of attending to relevant social cues (e.g., the enjoyment of a conversation, the topic at hand, additional conversation items) negative expectations and vigilance to potential negative outcomes direct attention to a wide range of "off task" thoughts and events. These include fears such as whether one is about to blush, sweat, pause too long in a conversation, or otherwise do something embarrassing or humiliating.

These patterns—negative social expectations, vigilance to negative outcomes, rising symptoms, and negative interpretations of symptoms and outcomes—motivate escape from and avoidance of social situations. Avoidance itself offers the potential of rapid reduction in anxiety at the cost of severe disruptions in quality of life (Bögels & Mansell, 2004; Safren, Heimberg, Brown, & Holle, 1997) and the maintenance of anxiety by preventing the disconfirmation of negative expectations. Consequently, fears of future social situations are enhanced, distorted cognitions are strengthened, and social anxiety disorder continues.

There is also evidence that even subtle avoidance behaviors, termed safety behaviors by Wells et al. (1995), can have similar deleterious effects with respect to the maintenance of social anxiety disorder. Safety behaviors include such strategies as holding a drink or clenching one's hands to hide trembling, and talking quickly, avoiding eye contact, or taking shallow breaths to avoid freezing up in a conversation. These safety behaviors, like other avoidance behaviors, offer anxiety reduction at the cost of maintained social fears. In an elegant study, Wells et al. found that the use of safety behaviors impaired anxiety reduction from exposure, perhaps by hampering the disconfirmation of fears. More recently, Kim (2005) found that exposures in which patients were encouraged to drop safety behaviors when a cognitive rationale was provided proved most efficacious for reducing anxiety when compared to patients who dropped safety behaviors with an extinction rationale as well as those who were not encouraged to change safety behaviors.

Accordingly, a number of targets for treatment can be translated from this model of the maintenance of social anxiety disorder. These include: (1) direct modulation of the anxiety evoked in social situations; (2) correction of the dysfunctional thoughts that create apprehension and anxiety, including core amplifying cognitions that intensify anxiety experiences in social situations; (3) elimination of failure-focused attention and the perceived social cost of these failures; (4) elimination of safety behaviors; and (5) development of accurate evaluation of performance in social situations. Direct modulation of the anxiety experience is most frequently associated with pharmacological approaches to treatment, whereas modification of patterns which create or sustain this anxiety is typically the target of cognitive-behavioral interventions.

MECHANISMS OF TREATMENT: PHARMACOTHERAPY

The absence of consistent differences between individuals with social anxiety disorder and healthy control subjects in studies using chemical and naturalistic provocations (see Tancer, Lewis, & Stein, 1995) makes a detailed biological model of social anxiety disorder difficult to construct. This difficulty is exacerbated by the wide range of agents that successfully treat the disorder. There is an abundance of evidence that patients can improve from MAOI, SSRI, and tricyclic antidepressants, as well as benzodiazepine treatment (Gould et al., 1997; Van Ameringen, Mancini, Patterson, & Simpson, 2009; van der Linden et al., 2000). Historically, the success of dopaminergic agents led to emphasis on dopaminergic pathways in the pathogenesis of social anxiety disorder (Cervenka et al., 2012; Liebowitz, Campeas, & Hollander, 1987; but see Bell et al., 2013). Likewise, the more recent success of the SSRIs has focused attention on serotonergic contributions to the disorder (Tancer et al., 1995). However, these findings do little to account for the etiology and nature of social anxiety disorder. Instead their empirical support and explanatory value appears to be limited to identification of some of the neurophysiologic pathways that may help regulate anxious affect, regardless of whether it arises in the context of social anxiety disorder or other anxiety conditions.

Both pathways, serotonergic and dopaminergic, have received attention in Gray's (l982a, 1982b) neuropsychological theory of anxiety. These neurotransmitters are hypothesized to play an important role in chemically labeling information delivered to the "behavioral inhibition system" (BIS). The BIS is hypothesized to be the core component of an anxiety system that includes the septohippocampal system, the Papez circuit, the prefrontal cortex, and ascending monoaminergic pathways enervating this cortex. The BIS is hypothesized to respond to potentially anxiety-provoking stimuli—novel stimuli, stimuli associated with punishment, and stimuli associated with nonreward—by increasing arousal, increasing attention to the environment, and inhibiting ongoing behavior. Regarding anxiety conditions such as social anxiety disorder, the BIS is assumed to be overactive as a product of conditioning experiences or

overactive serotonergic or noradrenergic inputs to the septohippocampal system. Gray hypothesizes that serotonergic afferents may be especially important for labeling stimuli as aversive and for fostering motor inhibition but notes that differentiation of the relative contributions of serotonergic and noradrenergic functions is difficult at best. Nonetheless, the action of antidepressant treatment of anxiety conditions is assumed to be a function of reductions in the intensity of serotonergic and noradrenergic signals reaching the septohippocampal system. Benzodiazepine treatment on the other hand, is assumed to have more general anti-anxiety effects by facilitating gamma-aminobutyric acid (GABA), an inhibitory neurotransmitter that may modulate the effects of the BIS through any of a number of mechanisms (Gray, 1982a, 1982b). In short, pharmacologic treatments have, as a core treatment effect for social anxiety disorder, the ability to reduce or eliminate the pathologic anxiety signal in social situations.

Elimination of the severe anxiety in social situations would, according to the model of social anxiety disorder presented previously, have a number of significant additional effects. Firstly, with the successful blockade of severe anxiety responses, patients gain control over one aspect of their social fears: the fear of uncontrolled anxiety in social situations. This should substantially reduce the fears of negative evaluations from others ("They will see me tremble and think I am weird") as well as negative self-evaluations ("I feel so nervous; I am really failing") that depend on the socially catastrophic interpretations of anxious affect and anxiety symptoms. Secondly, with anxiety under at least partial control, patients have the ability to feel more comfortable entering social situations, and potentially, to experience more-normal social interactions. With each successful social outing, patients responding to medication treatment have the potential to achieve greater confidence in their social abilities and effectiveness, as well as reductions in negative expectations and anticipatory anxiety before future social events. Failure-focused attention should therefore diminish over time, to be replaced by more-normal information processing that focuses attention on the true social demands at hand. Accordingly, it is important to note that pharmacologic treatment of social anxiety disorder, at least treatment that encourages patients to reenter social situations, tend to achieve reductions in fears of negative evaluation, at least to the levels achieved by cognitive-behavioral interventions (Mattia, Heimberg, & Hope, 1993; Otto et al., 2000). Similar evidence is available for changes in the processing of social threat information. Using an emotional Stroop color-naming task, Mattia and colleagues (1993) found that socially phobic individuals who responded to treatment achieved normalization of response latencies to social threat words, regardless of whether improvement was achieved from cognitive-behavioral group therapy or from phenelzine treatment.

According to the model presented thus far, medication treatment gains are initiated by reductions in anxious affect and are followed by broader changes in dysfunctional patterns, mediated by the effects of successful reentry and performance in social situations. This model suggests that the degree of treatment

gains would be associated with the degree of social exposure practiced while taking medications. Future research should test this hypothesis explicitly; nonetheless, tentative support is provided by a study of the treatment of panic disorder with agoraphobia. Telch, Agras, Taylor, Roth, and Gallen (1985) showed that most of the beneficial effects of imipramine treatment could be greatly attenuated by instructions from clinicians that discouraged step-by-step exposure. Without exposure, patients did not have a mechanism to translate anxiety suppression into the fuller reduction of fears and impairment that is brought by learning that feared situations are again safe.

Given this model of the efficacy of pharmacotherapy, the question arises why relaxation treatments, targeted directly to the modification of anxious arousal, are not more effective (see Gould et al., 1997; Taylor, 1996, for efficacy estimates). For example, Alstrom, Nordlund, Persson, Harding, and Ljungquist (1984) found that relaxation training was ineffective, with poorer outcome than the exposure and supportive therapy comparison conditions, and no better than a control condition. One answer to this question is that although both relaxation training and pharmacologic treatment can reduce anxiety, relaxation training requires active, in-situation effort. This effort to relax in social situations may have the untoward effects of further distracting attention from relevant social cues ("I am getting nervous, I need to relax my shoulders") and providing safety behaviors ("I will be OK, because I can relax my shoulders") that help in the moment but may maintain fears of social situations over the long term ("If not for my relaxation, it would have been a disaster"). In contrast, medication use does not require in-situation behaviors; instead, medications are taken well before the social situation, and the individual is left to focus on relevant social behaviors with an increased sense of confidence ("I will probably be OK; I took my medication").

Of course, these considerations imply that patients treated with pharmacotherapy will be entering situations under conditions of a safety cue (taking medication), and accordingly they should be at risk for relapse upon medication discontinuation. That is, patients taking medication may never learn that social situations are truly safe, but are only conditionally safe as long as medication is controlling anxious affect. These considerations have received particular attention in panic disorder (see Otto, Pollack, & Sabatino, 1996; Westra & Stewart, 1998), and they are consistent with high rates of relapse after medication discontinuation in social anxiety disorder (Davidson, Tupler, & Potts, 1994; Sutherland, Tupler, Colket, & Davidson, 1996) and tentative observations of poorer longer-term efficacy for medications over time (Heimberg et al., 1994). In fact, based on these relapse rates, a review suggests that pharmacotherapy should be continued for at least 12 months to maintain gains (Davidson, 2003).

Thus far, we have discussed the cognitive effects of medication as an indirect effect of successful exposure: When patients observe the blockade of feared anxiety responses and the success of initial social exposures, their negative expectations and self-evaluations diminish. There is also evidence that

medications may have more direct influence on cognitions, perhaps as a direct function of the modulation of affect.

There is evidence that negative affect may increase the severity of typical anxiety-related cognitions. For example, Ball, Otto, Pollack, Uccello, and Rosenbaum (1995) found that the presence of major depression was associated with increased fears of negative evaluation and lower assertiveness among patients with social anxiety disorder, and depressed mood appears to increase scores on other measures of dysfunctional attitudes characteristic of social anxiety disorder (Bruch, Mattia, Heimberg, & Holt, 1993; Ingram, 1989).

This evidence for mood state effects on anxiety-related cognitions is complemented by a wealth of evidence from the study of major depression. Successful pharmacologic treatment of major depression is associated with significant reductions in negative thoughts and dysfunctional attitudes (Dohr, Rush, & Bernstein, 1989; Fava, Bless, Otto, Pava, & Rosenbaum, 1994; Peselow, Robins, Block, Barouche, & Fieve, 1990; Szentagotai, David, Lupu, & Cosman, 2008), suggesting that some of these negative beliefs are mood-state dependent (see also Miranda, Persons, & Byers, 1990). Likewise, the presence of major depression is associated with elevations or greater fears of anxiety sensations (anxiety sensitivity), which decrease significantly after the pharmacologic treatment of depression (Otto, Pollack, Fava, Uccello, & Rosenbaum, 1995; see also Taylor, Koch, Woody, & McLean, 1996). Presumably, this finding may reflect the contribution—and subsequent elimination of the contribution—of negative affectivity to the negative and catastrophic evaluations of anxiety sensations. Extending these considerations to social anxiety disorder, pharmacotherapy may exert more-direct effects on: fears of negative evaluation and other anxiogenic cognitions by reducing negative affect. With less negative affect, catastrophic expectations for social situations may be directly reduced, further attenuating the cycle of anticipatory anxiety and negative interpretations.

Some of these hypothesized mechanisms of the action of pharmacotherapy are open to empirical testing. Given that fears of anxiety sensations appear to form their own factor on social anxiety measures (Safren et al., 1998), the time course of changes on this factor, relative to other aspects of social anxiety fears, can provide insight about the degree to which pharmacotherapy has initial, specific effects on catastrophic interpretations of anxiety sensations, and whether other changes in negative cognitions change more slowly over time. Likewise, examination of the effects of anti-exposure instruction during the early phase of pharmacologic treatment of social anxiety disorder would help elucidate which cognitive changes may be a more-direct effect of medications, and which are dependent on successful exposure to social situations for change. Finally, examination of residual levels of fears of negative evaluation, relative to residual anxious distress or avoidance, offers the potential of clarifying which changes are most important for maintenance of treatment gains from medication.

It is also important to note that there are a number of new conceptualizations of the action of antidepressant medications relevant to the treatment of

mood and anxiety disorders. One prominent new account focuses on the role of antidepressant medications on neurogenesis. Specifically, brain-derived neurotrophic factor (BDNF) levels rise significantly after antidepressant treatment (Brunoni, Lopes, & Fregni, 2008), and new theories contend that these effects, particularly the promotion of cortical plasticity and inhibition of the effects of stress, may explain antidepressant action (e.g., Castrén & Rantamäki, 2010; Masi & Brovedani, 2011).

Elucidation of potential mechanisms of action in the treatment of social anxiety disorder with antidepressants will be informed by additional research on the role of BDNF alternations on the maintenance or attenuation of fear circuitry (e.g., Giachero, Bustos, Calfa, & Molina, 2013), with the potential for modification of the overactivity in emotional processing circuits in those with social anxiety disorder (e.g., Brühl et al., 2011).

MECHANISMS OF TREATMENT: CBT

Research provides good evidence that the outcome of CBT is not dependent on expectancy or nonspecific effects alone, although positive expectancies are positively associated with treatment benefit (Safren, Heimberg, & Juster, 1997). Furthermore, CBT has been shown to offer efficacy over nonspecific treatment effects alone, such as group support and time with a caring therapist (Heimberg et al., 1990; Heimberg et al., 1994). Specific to delivery format of CBT for social anxiety, there is some debate as to whether individual or group format is most advantageous. Although research providing direct comparison of formats is limited, the majority of such studies have found equivalent effects for both individual and group treatment (Heimberg, 2001) with group format allowing for more easily simulated social situations and vicarious learning (Stangier et al., 2003) as well as superior cost-effectiveness (Heimberg, 2001). In addition, group-administered CBT showed greater effectiveness in reducing fear of negative evaluation than individual CBT (Dogaheh, Mohammedkhani, & Dolatshahi, 2011). In contrast, a recent review suggests a potential advantage for individual CBT with greater effect sizes and lower attrition rates as compared to group CBT (Aderka, 2009), highlighting the need for further investigation in this area. More recently, self-help CBT protocols for social anxiety provided in both guided and unguided formats over the Internet have increased in prevalence with more consistent positive effects seen in guided forms of self-help treatment, indicating some benefit of therapist contact (Andersson, 2009; Bisson, 2012; Titov et al., 2008). However, regardless of format, both individual and group CBT protocols for social anxiety target similar mechanisms of action.

Cognitive-behavioral treatments for social anxiety disorder focus directly on the modification of patterns hypothesized to maintain the disorder. Informational, cognitive restructuring, and exposure interventions are central to most current treatment packages. These interventions are combined, at times, with social skills training or anxiety management interventions. Informational components

are designed to provide the patient with a model of the disorder, a rationale for treatment procedures, and a guide for collaborative treatment efforts. Cognitive restructuring focuses directly on the modification of the anxiogenic cognitions and core beliefs associated with the disorder. It has at least four interrelated targets for modification: (1) the negative expectancies that are present before and during exposure to social situations, (2) failure-focused attention and overestimation of the cost of social failures, (3) amplifying cognitions and associated dysfunctional interpretations of social performance and anxiety affect, and (4) maladaptive self-evaluations following performance in social situations.

In typical cognitive restructuring, thoughts are treated as hypotheses, and emphasis is placed on the development of more accurate thinking patterns. Cognitive-restructuring strategies include guided discussions, Socratic questioning, and self-monitoring, although distinctions between cognitive and exposure procedures are blurred by the additional use of behavioral experiments to test the validity of specific beliefs using an exposure format. Indeed, the use of behavioral experiments to effect cognitive change is consistent with evidence that exposure treatments alone achieve cognitive changes in the same range as those achieved by traditional cognitive-restructuring procedures used alone (e.g., Hope, Heimberg, & Bruch, 1995; Mattia et al., 1993; Mattick, Peters, & Clark, 1989; Newman, Hofmann, Trabert, Roth, & Taylor, 1994). However, cognitive change appears to be only one component of the benefits offered by exposure. Mattick et al. (1989) found that exposure trailed cognitive-restructuring interventions in achieving cognitive changes, but it outperformed cognitive restructuring for overall improvement of social anxiety. In a meta-analysis conducted by Gil, Carrillo, and Meca (2001), no statistically significant differences in effectiveness were found between exposure techniques, cognitive restructuring, and social skills training. What then are the additional mechanisms of action of exposure?

A traditional perspective is that exposure breaks learned associations by a process of habituation to anxiety-provoking stimuli within an exposure session. Extinction of the fear response over repeated exposures (for review, see Barlow, 1988) has received central attention as a mechanism of exposure treatment. As long as patients are not resensitized by the actual occurrence of feared outcomes, fear reduction should occur fairly naturally with prolonged exposure. However, this perspective ignores the powerful role that cognitive conceptualizations play in determining whether the feared outcome occurs. Danger in social situations is not an objective phenomenon but a subjective evaluation dependent on cognitive biases.

Two case examples illustrate this point. The first is provided by Heimberg (1991) in his treatment manual for cognitive behavioral group therapy (CBGT). He describes an exposure session in which a woman was asked to pour water into a cocktail glass as part of a mock cocktail party exposure. Although the patient's performance was objectively successful, the patient was aware that she spilled a few drops of water during the exposure and concluded that she had failed and was a hopeless case. An unsuccessful suicide attempt followed.

Although such extreme reactions are rare, this example illustrates the importance of cognitive interpretations in determining whether exposures are sensitizing or anxiolytic. Likewise, a recent patient in our treatment program reported that he went to a party and started a conversation with three new people, exceeding his exposure goal of talking to one person during the evening. However, the patient considered this experience a failure because of his belief that this task would be hard only for someone who is "truly inadequate."

These examples illustrate the powerful role of cognitive interpretations on defining whether feared outcomes have occurred and how exposure treatments must take into account the "cognitive set" of patients during exposure. Indeed, simple instruction to attend to task-relevant rather than internal (anxiety) stimuli appears to increase the effectiveness of social exposure (Wells & Papageorgiou, 1998). Recently, Furukawa et al. (2009) examined the role of self-focused attention and engagement in safety behaviors; the results indicated that the degree to which patients reduced their level of self-focused attention predicted observer ratings of their visible anxiety as well as their belief in feared outcome.

In addition, research has begun to focus more closely on changes in judgmental bias as a possible cognitive mechanism (Foa, Franklin, Perry & Herbert, 1996; Foa, Huppert, & Cahill, 2006, Hofmann, 2004, McManus, Clark & Hackmann, 2000; Smits, Rosenfield, McDonald, & Telch, 2006). More specifically, judgmental bias in social anxiety disorder can be broken down into a probability bias, the tendency to associate feared stimuli or responses with an unrealistically high estimation of harm, and a social cost bias, the tendency to exaggerate the negative consequences of a harmful event. Thus far, research has been mixed regarding which of these two biases plays a more prominent role in the maintenance of social anxiety disorder. Studies conducted by Foa and colleagues (1996) as well as Hofmann (2004) support the notion that changes in cost bias account for a significant proportion of the variance in treatment outcome, while research conducted by McManus and colleagues (2000) asserts that changes in cost bias did not account for a significant proportion of variance in outcome when probability bias was statistically controlled. Additionally, in studying the contribution of each of these biases, Smits and colleagues (2006) found that reductions in probability bias lead to a reduction in fear whereas the reduction in cost bias was a consequence of fear reduction. As the data in this area is still mixed, a treatment approach which addresses both may be warranted. Traditional cognitive reappraisal can be used to correct overestimation of threat, and social cost exposure, a type of exposure in which the patient purposely causes a social mishap to occur, can allow the patient to more correctly assess the true threat when a social mishap does happen (e.g., Hofmann & Otto, 2008).

The question remains, however, whether exposure is simply the weakening of fear associations in the context of corrective experiences, or whether much more active learning of safety is involved. Recent findings in the animal learning literature support the latter view. Rather than reflecting unlearning,

exposure-based extinction appears to reflect the learning of alternative associations. As a result of exposure trials, fear cues may take on a much more ambiguous meaning (e.g., no longer signaling danger). Such extinction effects also appear to be sensitive to the context of learning, in which the presence of external or internal (e.g., emotional) contextual cues may influence whether fear or safety associations are recalled upon re-exposure to a phobic stimulus (for review, see Bouton & Nelson, 1998; Bouton & Swartzentruber, 1991). Morissette, Spiegel, and Barlow (2008) examined the role medication can play as a contextual cue in exposure procedures and found that state-dependent learning effects are possible when combining exposure and pharmacotherapy.

A useful heuristic for conceptualizing the nature of exposure treatment is provided by information-processing theories of emotion that focus on fear networks. According to Lang's (1977) bioinformational theory of emotion, fear networks consist of: (1) stimulus elements that represent sensory cues associated with the feared event; (2) response elements that include cognitive, affective, physiological, and behavioral responses to these cues; and (3) interpretive elements that include information about the meaning of the event and the nature of the association between the stimulus and response elements. Once the network is formed, cues associated with the network (e.g., either stimulus or response cues) can activate the fear network, and consequently activate anxiety and urges to avoid or escape.

Regarding treatment, Foa and Kozak (1986) have argued that two conditions are necessary for fear reduction via exposure: activation of the fear network and incorporation of new information into that network. Foa and Kozak emphasize four issues associated with accessibility to and modification of fear networks: (1) the match between the fear network and exposure cues, (2) the medium in which the exposure is delivered, (3) the duration of exposure, and (4) adequate attention to fear cues.

Fear evocation appears to be maximized by realistic exposure scenarios. These can be achieved by role-playing social situations, which are easily instrumented in group treatment settings (see Heimberg, 1991; Hope & Heimberg, 1994). In addition, to ensure realistic exposure conditions, clinicians should consider the need to include response cues in exposure sessions. For example, for patients who fear speaking because they catastrophically interpret the potential consequences of having a dry throat or feeling dizzy during the presentation ("They will think I am crazy; I will be unable to continue"), authors of exposure assignments may well want to include procedures for inducing these feared symptoms in conjunction with speaking exposures (e.g., the exposure would be conducted only after the patient induced symptoms using interoceptive exposure techniques, in this case hyperventilation).

Regarding the medium of exposure, social anxiety disorder treatments often rely on in vivo exposure formats that are likely more powerful than approximated methods, including verbal descriptions, imaginal exposure, and role-play situations that may be used in other disorders (e.g., posttraumatic stress disorder).

The duration of the exposure session has received consistent attention in the treatment of phobic conditions. A decrease in anxiety during the exposure session is thought to allow integration of new information into the fear network, in part because exposure to the cues is not accompanied by current threat or incessant anxiety. Longer exposure sessions provide greater opportunities for habituation, and, correspondingly, prolonged exposure (e.g., 50 minutes or more) has been found to be superior to short exposure for more severe phobic conditions (Foa & Kozak, 1986). In addition to duration, Berry, Rosenfeld, & Smits (2009) found that extinction retention, the extent to which fear reduction is maintained between two separate exposure sessions, is also associated with improvement in fear and avoidance.

Work by Moscovitch, Hofmann, Suvak, & In-Albon (2005) further clarified the relationship between social anxiety disorder and major depression, providing support for the notion that differential underlying treatment mechanisms are at work. Their results indicate that while 91% of the variance in decreases in depressive symptoms can be accounted for by changes in social anxiety, the reciprocal is not true. This provides support for the notion that improvement in social anxiety disorder is distinct from direct mood effects of interventions.

Finally, as discussed previously, objective presentation of phobia cues does not ensure that these cues will be processed. Patients may modulate their emotional responses to exposure by minimizing their attention to exposure stimuli or using a variety of safety cues or behaviors. In addition to those detailed by Wells et al. (1995), safety cues may include being accompanied to social events by a person who is less phobic, or by holding a drink when having a conversation. Safety behaviors may also include cognitive strategies such as "acting" the part of another while in a social situation. Accordingly, Wells et al. (p. 160) have suggested guidelines for maximizing the effectiveness of exposure by taking into account safety behaviors and cognitive biases that may insulate patients from corrective feedback and thus interfere with anxiety reduction:

1. Patients' feared catastrophes and their perceived likelihood should be assessed.
2. Safety behaviors that are rationally linked to these feared catastrophes should be identified.
3. A cognitive set focusing on active disconfirmation of negative beliefs should be established.
4. Safety behaviors should be eliminated or reversed during exposure.
5. The outcome of the assignment should be discussed in information-processing terms.

In particular, the therapist should ask whether the feared catastrophe happened. If it did not, what is the patient's explanation? Is the nonoccurrence simply attributed to residual safety behavior, or has the exercise produced a more profound change in belief?

All of these considerations suggest that exposure is an active process that must take into account information-processing biases. Moreover, the goal of exposure is more than the simple loosening of fear associations; it is the active relearning of safety in the phobic situation. Consequently, effort needs to be applied to ensure that this learning is not conditional (e.g., "I will be OK only if I do not sweat/if I use safety behaviors/if I am with my partner," etc.) by providing unambiguous exposure practice.

Given the overlap in methods between cognitive and exposure interventions, it is perhaps not surprising that combined cognitive and exposure interventions sometimes fail to produce results significantly better than exposure alone (compare Hope et al., 1995; Gil et al., 2001). Relative to the model of social anxiety disorder presented here, cognitive restructuring provides a means to challenge and reduce the negative expectations and self-defeating amplifying cognitions associated with social anxiety disorder. This process is aided by monitoring of thoughts during naturally occurring anxiety episodes, and specific practicing of cognitive restructuring during exposure procedures. The combination of exposure and cognitive restructuring also provides patients with an opportunity to develop alternative cognitive skills in the context in which these skills are needed most, including more-accurate self-evaluation of performance.

As a consequence, exposure combined with cognitive restructuring provides a context for correcting dysfunctional thoughts, redirecting failure-focused attention, and the elimination of safety behaviors. The construction of clear behavioral goals for exposure and reviews of objective goal attainment provide a context for challenging dysfunctional, subjective evaluations of performance. In particular, with repeated exposure, patients learn that they tend to meet objective performance goals despite their subjective experience of anxiety. As confidence rises with subsequent exposures, negative expectations and evocation of anxiety in social situations is further reduced. Successful exposure breeds more-positive memories and more-adaptive expectations for future performance. At the same time, patients learn not to fear minor social mishaps and to change evaluations of the "adequacy" of social performance. In short, fear memories are replaced by alternative, more-adaptive associations and beliefs.

Our own view is that cognitive interventions are especially useful in reducing negative expectancies prior to exposure, inhibiting maladaptive information processing, guiding attention to actual performance demands during exposure, and aiding the accurate evaluation of objective performance (including the noncatastrophic status of social errors) during and after exposures. Although early meta-analyses evaluating separate studies—emphasizing exposure interventions relative to cognitive interventions—have suggested a more optimal outcome for exposure techniques relative to cognitive strategies alone (see Gould et al., 1997; Heimberg & Juster, 1995; Taylor, 1996), three recent studies have shown the opposite (see Ougrin, 2011), perhaps indicating that the inclusion of social-cost interventions in more recent protocols helps aid efficacy.

COMBINED PHARMACOTHERAPY AND CBT

One implication of the models of change detailed previously is that combinations of pharmacotherapy and CBT may have the additive benefits of physiologically mediated anxiety suppression combined with direct modification of anxiogenic cognitive and behavioral patterns. Research to date in this area has been mixed with some evidence for enhanced effects of combined treatments (Blomhoff et al., 2001) and some demonstrating no increased benefit for this treatment approach (Davidson et al., 2004; Foa, Franklin, & Moser, 2002). This one-two punch may have specific advantages early in treatment, but due to the hypothesized role of medications serving as safety cues, it may interfere with unambiguous reductions in fear later in treatment. Consequently, patients continuing to take medications during the conclusion of short-term CBT would be expected to be at higher risk of relapse than patients who achieved similar treatment gains without medication. Moreover, both the animal-learning literature and treatment studies in humans (compare Bouton, Kenney, & Rosengard, 1990; Marks et al., 1993) provide evidence for the reemergence of fears upon medication discontinuation, when extinction trials were conducted while subjects were medicated.

Concerns about the combination of pharmacotherapy and CBT are also voiced by Gray (1982a, 1982b). Pharmacologic blockade of noradrenergic and serotonergic afferents to the septohippocampal system are hypothesized to provide only temporary blockade of anxiety, with a return of anxiety upon removal of medication. In contrast, exposure treatment is hypothesized by Gray to have more permanent effects on the septohippocampal system, eliminating the danger interpretation to phobic stimuli through a process of habituation. Gray emphasizes that the potential habituating effects of exposure on the septohippocampal system may be disrupted by medication use: With modulation of afferents to the septohippocampal system by medications, crucial biological effects of exposure ("biological toughening up") may not occur. One mechanism for the absence of strong additive effects between pharmacotherapy and CBT is that pharmacotherapy may block some of the natural memory-enhancing effects of cortisol release during exposure (Otto, McHugh, & Kantak, 2010), attenuating the degree to which therapeutic learning during CBT is retained. Additional research is needed to further examine these hypotheses, and to further examine the nature of failures to obtain additive effects between CBT and pharmacotherapy (e.g., Davidson et al., 2004) as well as longer term attenuation of CBT efficacy with traditional combination treatment strategies (e.g., Otto, Smits, & Reese, 2005).

There has also been a recent focus on using therapy-enhancing medications, such as D-cycloserine (DCS) to speed up the aforementioned learning process. This strategy represents a departure from typical pharmacotherapy for social anxiety which generally provides medications meant to reduce anxiety; DCS is administered in a single-dose fashion prior to exposure sessions as a means of

enhancing the learning provided by CBT. Early studies showed strong enhancement of treatment response when DCS was used as an adjunct to exposure-based treatment of social anxiety disorder (Guastella et al., 2008; Hofmann et al., 2006). However, the reliability of DCS augmentation effects appears to be attenuated when a full course of CBT is offered (Hofmann et al., 2013). Nonetheless, there is emerging evidence that the reliability of DCS enhancement effects depends on the degree of fear learning achieved during any index session where DCS is administered. Specifically, there is evidence that DCS, and other agents targeting enhancement of the retention of therapeutic learning (yohimbine; Smits et al., 2013a), enhances the consolidation of treatment gains and ultimate outcome only when low fear is achieved at the end of an exposure session (Smits et al., 2013b; Smits et al., 2013c). These studies, which show that DCS has advantages over placebo augmentation only when low fear is achieved at the conclusion of exposure, raises questions as to whether DCS may slow benefit when given in conjunction with inadequate/unsuccessful exposure sessions.

SOCIAL SKILLS TRAINING

Inadequate social skills, as differentiated from the inhibition of extant skills due to social anxiety, may require additional interventions. Social skills training is designed to help patients develop a more adaptive social repertoire, eliminating patterns that may be leading to poor social performance and consequent anxiety about future interactions. Actual skill deficits are not assumed to be a necessary feature of social anxiety disorder, and accordingly, there is some support for a treatment-matching approach. Social skills training appears to be more efficacious for patients who are socially unskilled relative to socially overanxious patients (Heimberg & Barlow, 1991; Ost, Jerremalm, & Johansson, 1981), but this is clearly not always the case (see Mersch, Emmelkamp, & Lips, 1991; Stravynski, Kyparissis, & Amado, Chapter 8 in this volume).

It is important to note that social skills training can easily incorporate exposure procedures as part of skill training and rehearsal, making it difficult to ascertain the effects of skill acquisition separate from exposure practice. Indeed, different conclusions on the efficacy of social skills training (compare Heimberg & Juster, 1995; Taylor, 1996) appear to be largely a function of inclusion of studies utilizing social skills training in the context of exposure in estimates of the efficacy of social skills training.

ACCEPTANCE AND COMMITMENT THERAPY

Other behavioral treatments do not focus on restructuring maladaptive thoughts; rather, they focus on teaching patients to conceptualize thoughts as behaviors in their own right that should be considered as useful or not useful instead of correct or incorrect. This sort of perspective, represented prominently by Acceptance and Commitment Therapy (ACT) (Hayes, 1995), focuses on increasing

adaptive behaviors by helping patients focus on the contingencies at hand rather than elevating the importance of thoughts about what they must do in response to emotions. Using rich metaphors to help patients reevaluate the nature of thoughts, ACT guides patients to accept emotions while targeting their behaviors towards relevant contingencies.

In our experience, the goals of ACT are similar to exposure therapy in many ways in our clinical and research applications of Heimberg's CBGT (Heimberg, 1991) as well as treatments emphasizing social cost exposures and traditional exposures (Hofmann & Otto, 2008). By midtreatment (e.g., sessions 5 to 7 of a 12-session treatment protocol), as patients complete exposures and objectively evaluate their performance, they begin to realize that objective goals are reliably met despite negative expectations and anxiety. In other words, patients learn that negative thoughts and the experience of anxiety do not match objective performance; goals are met despite these internal events. As treatment progresses, cognitive evaluations and affective experience come in line with these objective evaluations.

This process of change is consistent with some of the goals of ACT: to eliminate reliance on maladaptive cognitions or faulty emotional signals, to teach emotional acceptance, and to guide patients to respond to contingencies at hand. Emotional acceptance of anxiety in particular (e.g., treating anxiety as an emotional signal rather than a statement on the true danger of the situation or the personal effectiveness of the individual) is directly relevant to the modification of amplifying cognitions. With both treatments, anxiety loses its ability to signify failure and becomes a sign of emotional arousal alone. Research into ACT's effectiveness with socially phobic populations is still in the early stages; however, several pilot studies have demonstrated potential promise in this area (Dalrymple & Herbert, 2007; Ossman, Wilson, Storaasli, & McNeil, 2006).

OTHER PSYCHOSOCIAL TREATMENTS

Despite the wealth of evidence for the effectiveness of CBT for anxiety disorders, relatively few patients appear to receive these treatments in clinical practice, and psychodynamic treatments continue to be commonly applied for anxiety disorders, at least in certain locales (Goissman et al., 1993). What are the likely effects of psychodynamic psychotherapy? Any treatment that leads patients to reevaluate their negative and catastrophic interpretations of social situations and associated anxiety symptoms offers the potential to help patients enter social situations and further decrease subjective evaluations of danger (as long as performance deficits do not maintain actual poor performance). Accordingly, to the extent that psychodynamic treatments offer patients a historical explanation (narrative truth) for the anxiety elicited in social situations, new cognitions may be engendered, so patients can disattend to their catastrophic or amplifying cognitions and attend more to objective reality. Additional social confidence may be engendered by the accepting environments offered by

therapists in which the patients have the opportunity to test aspects of their social selves without social punishment. However, such indirect, in-session "exposure practice" may generalize poorly to out-of-session social situations without specific practice. Indeed, completion of out-of-session homework tends to be a significant predictor of therapeutic change in CBT for social anxiety disorder (Leung & Heimberg, 1996), and psychodynamic theories do not necessarily guide patients towards in-the-moment applications of historical insights or encourage reentry into avoided situations. This is particularly true given that psychodynamic therapeutic relationships are frequently discussed as being "unlike any other relationship." This uniqueness suggests that generalization of skills learned in the context of the therapeutic interaction may be particularly hard to generalize to out-of-session interactions.

These hypotheses await empirical evaluation, with attention to both the efficiency as well as the ultimate outcome of psychodynamic interventions and the therapeutic interventions that appear to drive beneficial change. Interestingly, investigations of this kind are under way. For example, Ablon & Jones (2002) coded session transcripts of therapists doing interpersonal therapy (IPT) or cognitive therapy for depression in the context of the multicenter treatment trial for major depression (Elkin et al., 1989). Prototypic psychodynamic and cognitive-behavioral therapist behaviors were coded, and scores were examined as predictors of treatment outcome. Despite the psychodynamic rationale for interventions in the manual-driven IPT, interventions that were coded as cognitive-behavioral within this therapy (and within CBT) were those most linked with beneficial outcome.

SUMMARY

Throughout this chapter, we argued that successful treatment of social anxiety disorder is achieved by interruption of the ongoing cycle of the negative social expectations, and vigilance to negative outcomes, rising symptoms, negative interpretations of symptoms and outcomes, and avoidance and escape behaviors that characterize the disorder. Pharmacological, cognitive, and exposure-based interventions were hypothesized to intervene at different points in this cycle, attending to different "linchpins" in disrupting the self-perpetuating cycle of social anxiety. Both pharmacological and psychosocial interventions work. Exposure is obviously not the central element of change in pharmacotherapy as it is in CBT. Nonetheless, exposure as an important element of change has earned attention in both modalities of treatment. Exposure is designated in Gray's (1982a, 1982b) neuropsychological theory of anxiety as a tool to achieve more enduring changes in neurophysiological systems maintaining anxiety. Exposure also ranks as an important context for the application of pharmacological treatment (Sutherland & Davidson, 1995), and it is given central attention in various cognitive-behavioral accounts of the disorder and its treatment (Hofmann, 2000, 2007; Hofmann & Otto, 2008).

Exposure effects were conceptualized as being far from a passive process of loosening fear associations. Instead, exposure was discussed as an active process involving the acquisition of safety in a phobic situation, which is richly dependent on the cognitive set that accompanies the processing of the exposure experience. Although the mechanisms of action presented here are consistent with the available data, the mere consistency between the model and available data does not rule out alternative accounts of the mechanisms of change. Whenever possible, we have suggested areas for future inquiry that may further clarify change mechanisms in social anxiety disorder.

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