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Pathological Anxiety: Emotional Processing in Etiology and Treatment, Edited by Barbara Olatov Rothbaum

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PART I

Theoretical Conceptualizations of Pathological Anxiety

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Emotional Processing Theory

An Update

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For nearly two decades, emotional processing theory (Foa & Kozak, 1985, 1986) has influenced the conceptualization of the nature of anxiety disorders and the psychological mechanisms of their effective treatment. This chapter discusses the basic premises of emotional processing theory as originally described by Foa and Kozak, as well as several hypotheses derived from the theory. We first describe the emotional processing conceptualization of the anxiety disorders and the mechanisms that are thought to underlie their recovery through effective treatment or natural recovery. We then discuss the current status of the theory by reviewing the relevant literature. In this review we focus on the evidence for the role of activation, within- and between-session habituation, and distraction and the relationship among these variables and cognitive, behavioral, and psychophysiological change. Next we discuss recent modifications to the theory, particularly in the areas of trauma/posttraumatic stress disorder (PTSD) and social phobia, which are based on empirical evidence, as well as learning and information processing theories. Finally, we identify a number of essential questions to guide future research in the nature and treatment of anxiety disorders.

HISTORY OF THE EMOTIONAL PROCESSING CONCEPT

The concept of emotional processing has its origin in Lang's (1977) analyses of fear-relevant imagery in the context of behavior therapy for fear reduction. In studying the procedure of systematic desensitization, Lang, Melamed, and Hart (1970) found three predictors of successful treatment: greater initial heart rate reactivity during fear-relevant imagery, greater concordance between self-reported distress and heart rate elevation during fear-relevant imagery, and a systematic decline in heart rate reactivity with repetition of the imagery. On the basis of his research, Lang (1977) suggested that "the psychophysiological structure of imagined scenes may be a key to the *emotional processing* [italics added] which the therapy is designed to accomplish" (p. 863). Although Lang did not elaborate on the term "emotional processing," subsequent papers advanced a bioinformational model of fear (e.g., Lang, 1984).

Lang's (1977, 1984) conceptual framework holds that a fear image is a cognitive structure containing stimulus, response, and meaning information that acts as a program to avoid or escape from danger. For example, the fear structure of a person with dog phobia includes representations of dogs, various physiological and behavioral fear responses (e.g., rapid heart rate, sweating, running away), and threat meanings associated with both dogs (e.g., "Dogs are dangerous") and the person's responses (e.g., "My rapid heart rate and sweating mean that I am afraid"); these representations are associated with one another. The fear structure is activated by information that matches some of the information represented in the structure and then spreads to other associated representations. Thus, in the dog-phobia example, confrontations with a dog not only activate the representation of a dog but also the danger meaning and the physiological responses of arousal and defensive behavioral responses. According to Lang (1977), "the aim of therapy could be described as the reorganization of the image unit in a way that modifies the affective character of its response elements" (p. 867). He further proposed that for image modification to occur, it is necessary for the fear structure to be activated to some extent. Accordingly, it is the necessary activation followed by image reorganization that accounts for the relationship between physiological measures (i.e., elevated heart rate reactivity followed by a decline in heart rate reactivity) and good outcome in exposure therapy for fear. However, Lang did not specify how activating the fear structure brings about changes in the response elements of the fear structure nor the nature of the changes that bring about a therapeutic outcome.

Rachman (1980) followed up on Lang's work by defining emotional processing as "a process whereby emotional disturbances are absorbed, and decline to the extent that other experiences and behaviors can proceed without disruption" (p. 51). Accordingly, successful processing would be

indicated by a person's confrontation with a previously distressing stimulus or event without experiencing or exhibiting signs of distress. By contrast, clinical phenomena such as persistent fears, anxiety, or obsessions; failures to benefit from exposure therapy; and the return of fear after exposure therapy would be evidence of failed or incomplete emotional processing. Rachman's analysis provides a description of the phenomena of emotional processing but does not provide a theoretical explanation of this construct. Indeed, as noted by Foa and Kozak (1986), Rachman's definition suffers from circular reasoning: The fear reduction that is attributed to successful emotional processing is the same evidence used to infer that successful emotional processing has occurred. The task taken up by Foa and Kozak (1986) was to provide a theoretical framework to explain the phenomena of emotional processing.

FOA AND KOZAK'S EMOTIONAL PROCESSING THEORY

Fear Structures

Emotional processing theory (Foa & Kozak, 1985, 1986) builds on Lang's concept of fear structure, described previously, to explain the psychopathology and treatment of anxiety and its disorders. To this end, Foa and Kozak (1986) distinguished between normal and pathological fear structures. The former is adaptive, whereas the latter is maladaptive. In an adaptive fear structure, the associations among the representations reflect reality faithfully (e.g., a car veering toward me — danger meaning ["cars coming toward me are dangerous"] and fear [heart rate acceleration, scanning the road, veering the car off the road]). Thus, when a normal fear structure is activated by a dangerous situation (e.g., a car veering toward the person), it generates fear and leads to adaptive maneuvering by the individual (e.g., moving to safety) to avoid danger. In contrast, a pathological fear structure contains associations among the stimulus, response, and meaning representations that distort reality and includes excessive response elements (e.g., avoidance of safe situations). Foa and Kozak (1986) noted that pathological fear structures are resistant to modification but did not provide an explanation for this phenomenon. We propose that the persistence of a pathological fear structure is due to behavioral and cognitive avoidance, as well as to cognitive biases in processing information at various stages (encoding, interpretation, and retrieval). The avoidance and cognitive biases interfere with the acquisition of relevant information that is inconsistent with the existing elements of the pathological fear structure, a process that, as is discussed subsequently, constitutes the essence of recovery or emotional processing.

Foa and Kozak (1985) proposed that specific pathological fear structures underlie the different anxiety disorders and that successful psychosocial

treatment modifies the pathological elements in the structures. Furthermore, each disorder contains elements common to other anxiety disorders (physiological response elements and escape or avoidance responses), as well as disorder-specific elements and associations. For example, the fear structure of patients with PTSD is characterized by a pathological association between trauma reminders, which are essentially safe situations or images, and danger or a sense of incompetence. By contrast, panic disorder is characterized by a pathological association between response elements (bodily sensations), such as shortness of breath, and threat of death or going crazy. Lang and colleagues (e.g., Lang, Davis, & Ohman, 2000; Cuthbert et al., 2003) reported differential levels of specificity and coherence of the fear structures for PTSD, panic disorder, specific phobia, and social anxiety disorder. Specifically, when imagining personally feared consequences, individuals with specific phobias had the highest magnitude of physiological reactivity, and those with panic disorder and PTSD had the lowest. Those with social phobia appeared to fall in the middle; people with generalized social phobia had somewhat less physiological reactivity, and individuals with more circumscribed fears had more physiological reactivity. These data are consistent with the contention that individuals with more discrete fears have more coherent fear structures (Foa & Kozak, 1986).

Emotional Processing: Modifying the Pathological Associations in the Fear Structures

Foa and Kozak (1985, 1986) originally defined emotional processing as the modification of the fear structure in which pathological associations among stimuli, responses, and meaning are replaced with nonpathological associations. They further suggested that this modification involves weakening erroneous associations and acquiring new associations. However, recent work on extinction and reinstatement (Bouton, 2000; Rescorla, 2001) suggests that extinction does not eliminate or replace previous associations but rather results in new learning that competes with the old information. Architecturally, it is not clear whether the new learning is best represented as the acquisition of new associations that inhibit expression of the old associations in the old structure or as the acquisition of a new fear structure that exists along with old structure (see Foa & McNally, 1996, for a discussion of the latter concept). In either case, both the old and the new information remain stored in memory. Depending on the context, either the original association/structure or the new association/structure can be activated and determine behaviors, cognitions, and emotions. Either model better accounts for spontaneous recovery in extinction paradigms and relapses after treatment than the original conceptualization by Foa and Kozak. One major therapeutic implication of this reconceptualization is that treatment should occur in multiple contexts in order to increase the likelihood that

the new nonpathological fear structure or associations will be activated instead of the original pathological ones.

Foa and Kozak (1986) proposed that two conditions are necessary for emotional processing to occur: (1) activation of the fear structure and (2) incorporation of new information that is incompatible with the pathological elements of the fear structure. Activation occurs when an individual encounters stimuli or produces responses that are represented in the fear structure and that therefore are associated with danger meaning. In general, the greater the match between the fear-evoking experience and the person's pathological fear structure, the greater the activation. Although there was some suggestion of an "optimal" level of activation in the original theory, it seems that this concept requires further elaboration. Interestingly, neurobiological evidence now suggests that the amygdala needs to be activated in order to promote extinction in animals (Nader, Schafe, & LeDoux, 2000), thus indicating that some level of activation is in fact necessary. Our clinical experience suggests that an extreme level of activation (overactivation) may interfere with emotional processing, though the animal literature does not indicate such a phenomenon. It is likely that overactivation leads to a failure to incorporate new information due to inhibited attention, which diminishes encoding of the new corrective information and biases the processing of available information. Emotional processing theory posits that although activation is a necessary condition for emotional processing, it is not a sufficient condition, and that emotional processing requires the presence of information that disconfirms the erroneous elements in the structure. When such information is unavailable because the individual avoids or escapes the situation, the fear structure remains unchanged. Moreover, if the evocative situation contains information that confirms the person's feared consequences, the fear structure does not change and may even be strengthened. Even when disconfirmatory information is present during the evocative experience, emotional processing occurs only when it is encoded and incorporated into existing knowledge, that is, when new learning has occurred.

Exposure Therapy and Cognitive Modifications

Foa and Kozak (1986) suggested that certain pathological elements characterize the fear structures of individuals with anxiety disorders. The first type of pathological elements involves erroneous stimulus–stimulus associations (bald men → gun, for an individual raped by a bald man), stimulus–response associations (supermarket → heart palpitations, for an individual with panic disorder), and, most important, erroneous associations of safe stimuli with threat meanings (floors → germs → illness → death, for a person with obsessive–compulsive disorder) and responses with threat meanings (heart palpitations → heart attacks → death or danger, for a person with panic disorder). Foa and Kozak further suggested that, in addition to

pathological associations, pathological elements in the fear structure involve erroneous evaluations. Three such evaluations received particular attention: exaggerated probability estimates of harm (e.g., everyone is critical), exaggerated cost associated with this anticipated harm (e.g., it is dreadful to be criticized), and the anticipation that in the absence of escape or avoidance, anxiety will remain forever and may itself cause psychological (e.g., going crazy) or physical harm (e.g., dying).

In discussing the mechanisms by which emotional processing of fear is achieved, Foa and Kozak (1985, 1986) suggested that *in vivo* exposure (real-life confrontations) to the feared stimulus (e.g., dog) in the absence of the anticipated harm corrects the exaggerated probability estimates of harm. Exaggerated cost, on the other hand, is achieved via habituation of fear during confrontation with the feared consequences, sometimes through imaginal exposure. We suggest that imaginal exposure to the feared consequences not only corrects the exaggerated cost but also strengthens the discrimination between “thoughts about harm” and “real harm,” thus altering the associations between threat meaning of stimulus and/or response elements in the fear structure. A clear example is the patient with obsessive–compulsive disorder who erroneously equates thinking about harm with causing harm. Imaginal exposure to causing harm in the absence of real harm may alter the association between thinking about harm and threat. In the same vein, through repeated imaginal exposure of the traumatic memory, the patient with PTSD learns the distinction between remembering the trauma and being retraumatized, thus altering the association between the traumatic memory and threat meaning.

Exposure Therapy and Habituation

Emotional processing can occur as a result of everyday experiences (e.g., natural recovery following a trauma; see the later section on PTSD) or in the context of psychosocial treatment, such as cognitive and behavioral therapies or psychodynamic therapy. Exposure therapy involves helping people to repeatedly confront safe but feared thoughts, sensations, situations, and activities in order to promote emotional processing. Thus exposure therapy exercises are explicitly designed to activate the fear structure and at the same time provide corrective information about the nonthreat value of the stimuli, responses, and meaning elements evoked during the exercise.

Typically a decrease in fear occurs during prolonged exposure exercises (within-session habituation; e.g., Chaplin & Levine, 1981; Foa & Chambless, 1978), along with a decrease in peak intensity of fear across sessions (between-session habituation; e.g., Chaplin & Levine, 1981). Foa and Kozak (1986) identified the activation of fear followed by within- and between-session habituation as indicators of emotional processing that are related to,

but conceptually distinct from, symptom reduction. In addition to being an indicator of emotional processing, habituation may be a source of disconfirming information, such as information about the *absence* of physiological responding in the presence of the targeted stimulus that is incompatible with prior response information. Habituation may also disconfirm erroneous beliefs about the consequences of intense anxiety, such as the belief that anxiety will persist unless the person escapes the fear-evoking situation.

Several studies have found a relationship of between-session habituation with symptom reduction (i.e., treatment outcome; van Minnen & Hagedaars, 2002; van Minnen & Foa, submitted; Kozak, Foa, & Steketee, 1988; Jaycox, Foa, & Morral, 1998). However, the relationship between within-session habituation and symptom reduction is more ambiguous. Within-session habituation has been positively related to longer continuous exposure (Chaplin & Levine, 1981; van Minnen & Hagedaars, 2002; van Minnen & Foa, submitted), and longer continuous exposure has been positively related to symptom reduction in some studies (Chaplin & Levine, 1981; Rabavilas, Boulougouris, & Stefanis, 1976; Stern & Marks, 1973) but not others (van Minnen & Foa, submitted). However, most studies have not found a direct relationship between within-session habituation and symptom reduction (van Minnen & Foa, submitted; Jaycox et al., 1998; Foa et al., 1983; Kozak et al., 1988; Mathews, Johnston, Shaw, & Gelder, 1974). Further evidence that within-session habituation is not a necessary condition for improvement includes the finding that people with agoraphobia who were allowed to escape from their feared situations before their anxiety decreased improved as much as those who were instructed to stay in the situations until their fear diminished (Emmelkamp, 1974; De Silva & Rachman, 1984; Rachman, Craske, Tallman, & Solyon, 1986). It is possible, then, that within-session habituation is not a reliable indicator of emotional processing. Indeed, reduction of anxiety occurring within a session may sometimes be due to factors that are hypothesized to impair emotional processing, such as distraction and cognitive avoidance. In addition, some information may take time to be processed, such that disconfirming information that had been presented during exposure is not fully incorporated until some time after the exposure exercise (i.e., between sessions) rather than within the session. In fact, this may be the case with cognitive therapy for the anxiety disorders (e.g., Clark, 2001; Rachman, 2003). That being said, it is likely that, for individuals who are fully engaged with an exposure exercise (i.e., without any avoidance) and experience within-session habituation, such habituation is still an indicator of emotional processing and may facilitate between-session habituation (Foa et al., 1983; Pitman, Orr, Altman, & Longpre, 1996). Overall, the deemphasis on the relationship between within-session habituation and outcome is not critical to emotional processing theory because the proposed mechanism underlying symptom reduction is the modification of the relevant

erroneous associations through disconfirming information, not through habituation per se. In fact, Foa and Kozak (1986) proposed that within-session habituation is mainly important for patients whose core fear is the erroneous belief that anxiety “stays forever unless escape is realized.” For these patients, within-session habituation provides the information that disconfirms their erroneous evaluation. In most cases, the encoding of new information that contradicts the pathological elements in the fear structure occurs both within and between sessions.

Exposure Therapy and Distraction

Foa and Kozak (1986) viewed distraction as a form of cognitive avoidance and therefore hypothesized that distraction would impair emotional processing. Theoretically, distraction could serve to limit activation of the fear structure, prevent the encoding of disconfirming information, or both. The empirical literature on the effects of distraction on fear reduction is inconsistent with regard to the relationship between distraction and habituation. A number of studies have presented data suggesting that distraction interferes with within- and between-session habituation (Grayson, Foa, & Steketee, 1986; Rodriguez & Craske, 1993; Telch et al., 2004), whereas two studies by Page and colleagues (Oliver & Page, 2003; Johnstone & Page, 2004) have suggested that distraction actually facilitates habituation in people with blood-injury and spider phobia. These inconsistencies may be due to methodological differences among studies. First, the only studies to date that have suggested that distraction may facilitate habituation and symptom reduction are for specific phobia. All studies on other anxiety-disordered populations have not found such effects, though none have attempted to replicate Page’s distraction technique. Perhaps distraction facilitates partial disengagement in people with specific phobia, but it could be likely to interfere more fully in other individuals with anxiety disorders who also show less coherent fear structures (see Cuthbert et al., 2003, and previous discussion). Alternatively, it is possible that distraction and decreased attention are two different concepts. Page’s method involved counting backward by various number intervals, which would still allow eye contact and some level of attentional engagement during the exposure. Many of the other studies used tasks that aimed at producing such an extensive cognitive load that virtually no attentional resources could be allocated to the threat stimuli during the exposure. If distraction is defined as complete disengagement from threat stimuli, then Page’s studies do not meet such criteria. However, questions remain as to why decreased attention would be beneficial during exposure to feared stimuli, as it would presumably interfere with activation and with encoding of corrective information.

PTSD AND SOCIAL PHOBIA: THE APPLICATION OF EMOTIONAL PROCESSING THEORY TO SPECIFIC ANXIETY DISORDERS

In the original formulation of emotional processing theory (Foa & Kozak, 1986), examples from specific phobias, panic disorder with agoraphobia, and obsessive–compulsive disorder were used to explicate the model. More recently, we have focused on the psychopathology and treatment of PTSD and social anxiety disorder and have elaborated models for both disorders within the framework of emotional processing theory (Foa, Steketee, & Rothbaum, 1989; Foa & Jaycox, 1999; Foa & Cahill, 2001; Huppert & Foa, 2004). In this section we start with an application of emotional processing theory to natural recovery from trauma and treatment of chronic PTSD. Following that discussion, we describe a model of social anxiety disorder and how it may integrate well with another current conceptualization of social anxiety (Clark & Wells, 1995).

Natural Recovery from Trauma

Longitudinal studies investigating patterns of reactions following a trauma indicate that individuals differ in their abilities to successfully recover from, and therefore process, a traumatic event. Although most trauma survivors do process the trauma successfully, a substantial minority fail to do so and consequently develop chronic PTSD (e.g., Riggs, Rothbaum, & Foa, 1995; Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992).

Foa (1997) identified three factors that are associated with both natural recovery from trauma and reduction of PTSD severity via exposure therapy. The first factor is emotional engagement with the trauma memory, which is equivalent to the concept of fear activation discussed earlier. In natural recovery, emotional engagement refers to fear activation that occurs when one encounters a trauma reminder in the natural environment. Given that fear activation has been found to be positively associated with treatment outcome (Foa, Riggs, Massie, & Yarczower, 1995; Pitman et al., 1996), we may hypothesize that a *lack* of emotional engagement would be associated with poor natural recovery following a traumatic event. Dissociative symptoms, such as feelings of derealization or depersonalization during or following a trauma or amnesia for information related to the trauma, have been conceptualized as strategies that reduce emotional engagement in order to avoid trauma-related emotions (Foa & Hearst-Ikeda, 1996). Consistent with the tenets of emotional processing theory, several studies have found that peritraumatic dissociation is associated with more severe PTSD (e.g., Cardena & Spiegel, 1993; Koopman, Classen, & Spiegel, 1994). Similarly, a delay in the peak reaction to a traumatic event may also be seen as initial interference in emotional engagement and thus is

expected to hinder emotional processing and natural recovery. Consistent with this hypothesis, individuals whose peak symptom severity occurred within 2 weeks of the traumatic event were less symptomatic 14 weeks later than were individuals whose symptoms peaked between 2 and 6 weeks after the event (Gilboa-Schechtman & Foa, 2001).

The second factor associated with both natural recovery and treatment outcome is a change in trauma-related cognitions. Two basic meaning elements are thought to be at the core of the fear structure for PTSD: that the world is completely dangerous and that the self is totally incompetent (Foa & Rothbaum, 1998; Foa & Riggs, 1993). It follows that individuals with PTSD will exhibit more severe negative cognitions about the world and themselves than individuals who have either experienced a trauma without developing PTSD or who never experienced a trauma. This hypothesis was confirmed by Foa, Ehlers, Clark, Tolin, and Orsillo (1999). Similarly, successful emotional processing of a traumatic memory requires changes in the fear structure related to these two meaning elements such that natural recovery following trauma and recovery from chronic PTSD following treatment will both be associated with reductions in those negative cognitions. In a longitudinal study of crime victims, individuals who recovered from PTSD 3 months after their assaults exhibited fewer negative cognitions than those who continued to have PTSD (Riggs, Rauch, Moser, & Foa, 2005). Similarly, cognitions about the world and themselves decreased with treatment for PTSD, and changes in cognitions were associated with changes in PTSD severity (Foa & Rauch, 2004).

The third factor hypothesized by Foa (1997) to be associated with both natural recovery and improvement with treatment is the level of organization of the trauma narratives. This factor has also gained empirical support. Specifically, a higher degree of narrative articulation shortly after the trauma was predictive of greater recovery (lower PTSD symptom severity) 3 months later (Amir, Stafford, Freshman, & Foa, 1998). Similarly, exposure therapy for PTSD was associated with increased organization and decreased disorganization of the trauma narrative over the course of treatment, and those changes were correlated with symptom improvement (Foa, Molnar, & Cashman, 1995). The ways in which exposure therapy is thought to promote fear activation and modification of erroneous cognitions were explicated earlier. In addition, repeated recounting of the trauma memory during imaginal exposure and discussing the memory with the therapist not only alters the association between remembering the trauma and threat or danger but may also help reduce the fragmented quality of the trauma narrative. Over repetitions of the narrative, the sequence of events becomes better established, and details remembered in one recounting are incorporated into subsequent recountings, thereby helping to create a coherent narrative. It may be hypothesized that a more coherent narrative reflects a more coherent fear

structure that allows more complete activation and subsequent changes in the pathological elements of the fear structure. Foa and Cahill (2001) proposed that emotional processing that leads to natural recovery occurs through repeated activation of the fear structure via the trauma memory by engaging with trauma-related thoughts and feelings, by sharing the thoughts and feelings with others, and by confronting trauma-reminder stimuli in daily life. In the absence of additional traumas, these natural exposures contain information that disconfirms the common posttrauma associations within the fear structure, such as that the world in general or specific trauma reminders are dangerous and that the person's actions during the trauma or subsequent PTSD symptoms means the person is incompetent. For example, a rape survivor may repeatedly encounter men who are physically similar to the perpetrator, thus activating the trauma memory structure. As these encounters do not lead to additional assaults, the natural initial postrape inclination to view the world (e.g., all men) as entirely dangerous is not confirmed and thus gradually subsides. In addition, repeated thinking or talking about the traumatic event promotes habituation of negative emotions and the generation of an organized trauma narrative. By contrast, individuals who avoid engaging with the traumatic memories by either suppressing related thoughts and feelings or avoiding trauma-related situations do not have the opportunity to experience the habituation of negative emotions, to disconfirm the initial posttrauma negative cognitions, or to form an organized narrative and thus develop chronic disturbances.

Consistent with the proposed conceptualization of natural recovery, Creamer, Burgess, and Pattison (1992) found, among a group of individuals who were present when a man went on a shooting spree in a crowded building, that high levels of reexperiencing shortly after the trauma were correlated with lower levels of distress 4 weeks later. Similarly, Lepore, Silver, Wortman, and Wayment (1996) found that, 18 months after the trauma of losing their babies to sudden infant death syndrome, mothers who were able to share their loss with a supportive social network had resolved their grief more than did mothers who were not able to do so. Thus avoidance of trauma memories and reminders appears to interfere with natural recovery, whereas talking about the experience with supportive others appears to facilitate natural recovery. As noted previously, a less fragmented narrative is also associated with less severe PTSD (e.g., Amir et al., 1998), but a recent study by Gray and Lombardo (2001) found that organization was no longer related to PTSD after accounting for the decreased cognitive abilities often found in patients with PTSD. To the extent that cognitive impairments seen in PTSD reflect the effects of anxiety on information processing, it is possible that increased organization is a *consequence* of emotional processing rather than a mechanism by which emotional processing occurs. More research is needed to examine the conflicting findings.

The Fear Structure of Social Anxiety Disorder

Social anxiety disorder is characterized by excessive fear of embarrassment or humiliation in interpersonal or other social situations that leads to significant distress and impairment. The core fear in social anxiety disorder is not of physical threat, but rather of criticism, isolation, ostracization, and/or rejection by others. Thus social anxiety disorder may be conceptualized as a “fear of embarrassment.” Data suggest that social anxiety disorder can be clustered into specific situational fears (e.g., a fear of public speeches) or fears that vary across a number of social situations, including interpersonal and performance realms (generalized social phobia; Kessler, Stein, & Berglund, 1998). Because individuals with generalized social anxiety disorder make up the majority of the patients who seek treatment, we focus on them here. The model we present for the fear structure of generalized social anxiety disorder draws on clinical research into the psychopathology of social anxiety disorder, research in social psychology, emotion theory on embarrassment (e.g., Keltner & Buswell, 1997), and our direct clinical experience.

Stimulus Representations

By definition, the stimuli represented in the fear structure of an individual with social anxiety disorder are circumscribed to people or social situations (e.g., peers, authority figures, or individuals of the opposite sex). However, for most of these individuals, the fear structure contains a multitude of stimuli and contexts. One particular aspect of the fear structure in social anxiety disorder that has gained recent interest is images of oneself in social interactions. For example, Hackmann, Clark, and McManus (2000) found that patients with social anxiety disorder had specific recurrent images during social interactions and that these images appeared to be related to negative social interactions retrospectively reported to be related to the onset of the disorder. Furthermore, the individual views these images from the perspective of an observer (Clark & Wells, 1995; Spurr & Stopa, 2003; Wells, Clark, & Ahmad, 1998), and a consequence of these images is that they bias information processing (for a more detailed discussion of these and other findings about imagery and social anxiety, see Hirsch & Clark, 2004).

Representations of Verbal, Physiological, and Behavioral Responses

Verbal responses in the fear structure of individuals with social anxiety can have several functions: an expression of anxiety (e.g., hesitations in speech such as “ummm” or “uhhhhh”), avoidance of poor performance in a social

situation (e.g., by asking questions, changing topics away from oneself, etc.), or attempts to distract others from signs of one's anxiety (e.g., saying "it's hot in here" if the person feels sweaty). Physiological responses include changes in heart rate, blushing, sweating, and trembling. Some of the physiological responses reflect anxiety (sweating, trembling or shaking), and others reflect embarrassment (blushing). Notably, anxiety is associated with increases in heart rate (Cuthbert et al., 2003), whereas embarrassment is often associated with decreased heart rate (Keltner & Buswell, 1997); and either of these experiences may occur in the individual with social anxiety disorder. Behavioral responses include various types of escape and avoidance maneuvers, which can be of a very subtle nature and may include cognitive strategies (e.g., distraction). Clark and Wells (1995) have labeled these subtle avoidance behaviors "safety behaviors" (cf. Salkovskis, 1991) and have emphasized their role in the maintenance of social anxiety disorder.

Several of the responses in social anxiety are associated with threat meaning because they are viewed as drawing criticism, thereby leading to a spiraling of anxiety in social situations. Two studies examined the hypothesis that the fear structure of social anxiety disorder contains pathological associations between response representations (e.g., heart racing, blushing, sweating) and threatening meaning (e.g., social incompetence). Roth, Antony, and Swinson (2001) found that individuals with social anxiety disorder were more likely than controls to interpret their symptoms of anxiety as pathological (i.e., intense anxiety or some psychiatric problem) and less likely to interpret them as normal. Furthermore, Wells and Papageorgiou (2001a) reported that false feedback regarding pulse rate (e.g., "your pulse has increased/decreased") influenced ratings of self-reported anxiety and the strength of beliefs about an idiosyncratic feared consequence in the expected direction in patients with social anxiety disorder. Thus perceived strength of responses influences the threat meaning of those responses.

Cognitive Biases and the Meaning Representations of Stimuli and Responses

As noted, an additional aspect of the original emotional processing theory is the central role played by two erroneous evaluations that can also be conceptualized as cognitive biases: overestimation of the probability of feared harm and exaggerated cost of the feared outcome. Both feature prominently in the pathological fear structure of social anxiety disorder (Foa & Kozak, 1985, 1986, 1993). Foa, Franklin, and Kozak (2001) elaborated on this model, proposing that the erroneous meanings associated with social stimuli and fear responses are influenced by interpretation and judgment biases. Huppert and Foa (2004) further elaborated these concepts

and integrated these findings with Mathews and Mackintosh's (1998) model of information processing biases in anxiety.

Several studies provide support for the hypothesized relationship between judgment biases about social stimuli and social anxiety disorder. For example, Gilboa-Schechtman, Franklin, and Foa (2000) found that patients with generalized social anxiety disorder had greater estimates of probability and cost for unambiguous negative events (e.g., a boss berating one in front of others), greater estimates of cost of positive events, and lower estimates of the probability of positive events than both anxious and nonanxious control participants. Similarly, Foa, Franklin, Perry, and Herbert (1996) found both cost and probability biases in patients with generalized social anxiety disorder, although changes in cost biases were more predictive of change in symptoms of social anxiety after cognitive-behavioral treatment than were changes in probability biases. Consistent with Foa et al. (1996), Uren, Szabo, and Lovibond (2004) reported that although both cost and probability bias appear to contribute to social anxiety, cost was a stronger predictor of severity, whereas probability and cost estimates equally predicted the severity of the fear of bodily sensations in panic disorder. Stopa and Clark's (2000) results further support the relationship between exaggerated cost for negative social events and generalized social anxiety disorder. The primacy of cost over probability could not be tested in their study because the latter was not examined. However, in a follow-up study, McManus, Clark, and Hackmann (2000) included both mild and severe negative social events and found that both probability and cost were related to severity of social anxiety and to treatment outcome. Voncken, Bogels, and de Vries (2003) also found that both probability and cost equally contributed to severity of social anxiety. However, Voncken et al. (2003) used only evaluation situations for the judgments, excluding physiological experiences that can be important for individuals with social anxiety.

In summary, Foa and Kozak (1985) proposed that exaggerated cost is the more prominent erroneous evaluation in social anxiety disorder, whereas overestimation of probabilities is more central to specific phobia and panic disorder. The research to date indicates that both cost and probability estimates contribute to social anxiety, but their relative contributions are not entirely clear. We suggest that for highly negative social events (e.g., being rejected by many people), probabilities are likely going to be more important, whereas for mild negative social events (which frequently occur), it is the overestimation of cost that will be more important in social anxiety. Many other anxiety disorders do not include mild negative events, thus making cost a more prominent feature in social anxiety than in other anxiety disorders. Perhaps, then, as suggested by Foa and Kozak (1985), specific fear structures underlying the anxiety disorders differ in the relative influence that probability and cost estimates have on the threat meaning associated with stimuli and responses.

THE TREATMENT OF SOCIAL ANXIETY DISORDER

In order to promote emotional processing via exposure therapy (correction of the pathological elements of the target fear structure), the situation that activates the fear structure should incorporate corrective information that is contradictory to the erroneous associations represented in the structure. Generally, the corrective information is embedded in the absence of harm during confrontation with the feared situation, object, or memory (e.g., giving a speech without the audience booing), thus leading to changes in the patient's evaluations. Indeed, Hope, Heimberg, and Bruch (1995) found that negative social cognitions decreased significantly after exposure therapy. However, for emotional processing to occur, it is essential that the person perceives that the feared consequences did not occur. In the case of a person with dog phobia interacting with a friendly dog, the absence of negative consequences is obvious. However, because of the nature of social interactions, information disconfirming the patient's belief that others are judgmental may be obscured. The ambiguity of a social situation stems from the fact that explicit negative feedback during social interactions is censored and that false praise may be offered in the name of politeness. Thus the absence of open criticism or the presence of some compliments cannot be interpreted as an indication that the individuals involved in a given social interaction unambiguously enjoyed it. Even when corrective information is available in a social interaction, a number of other factors may interfere with encoding this information: self-focus, engaging in safety behaviors, attentional bias, and overactivation. Safety behaviors (e.g., keeping hands in one's pockets to prevent people from seeing them shake; Clark & Wells, 1995; Salkovskis, 1991), also referred to as subtle avoidance behaviors (Foa & Kozak, 1986), may be performed in order to prevent the feared consequences, reinforcing the perception that criticism or rejection would have occurred had they not engaged in the safety behaviors. Attentional and interpretation biases lead to selective encoding of social situations as negative, thus further impeding opportunities for emotional processing in the natural environment of the individual with social phobia.

It follows from the preceding considerations that the goal of treatment is to set up social situations in sessions that will both activate the fear structure and provide unambiguous information that disconfirms the patient's negative perceptions and evaluations. In other words, successful treatment imposes task demands that are sufficiently strong to override the hypervigilance to negative feedback and forces the patient to incorporate evidence of his or her adequate social performance (e.g., through video feedback or feedback from others; cf. Harvey, Clarke, Ehlers, & Rapee, 2000). In this way, disconfirming evidence, either during or after the contrived social situation, is incorporated into the fear structure, thus reducing the estimated probability and cost of negative outcomes.

Accordingly, a number of techniques that have recently been introduced into cognitive-behavioral therapies for social anxiety disorder emphasize the elimination of safety behaviors, the encouragement of outward focus, and the presentation of incompatible information via video and confederate feedback (Clark, 2001). Treatments utilizing these techniques have shown a successful reduction of social anxiety (Clark et al., 2003; Wells & Papageorgiou, 2001b). To optimize emotional processing, we have combined these techniques with imaginal and *in vivo* exposure and social skills training in our individualized Comprehensive Cognitive Behavioral Therapy (CCBT; Huppert, Roth, & Foa, 2003).

After successful treatment of an individual with social anxiety disorder, there are a number of changes that indicate successful emotional processing. These include reduced (1) probability estimates, (2) cost estimates, (3) attentional biases, (4) interpretation bias, (5) beliefs that anxiety during social situations remains forever, and (6) beliefs about the consequence of social situations (e.g., being rejected). The first four indicators are changes in information processing that may mediate the changes in beliefs, as well as produce reduction in social anxiety symptoms.

DISCUSSION AND FUTURE DIRECTIONS

In this chapter we have presented an update of emotional processing theory and applied the theory to two disorders that were not discussed in great detail in Foa and Kozak's (1985, 1986) original formulation: PTSD and social anxiety disorder. Emotional processing theory appears to account quite well for natural recovery, as well as for treatment of anxiety disorders, whether by exposure therapy, cognitive therapy, or other alternatives. However, a number of questions require further examination.

1. Can emotional processing theory account for other emotions in the same way in which it accounts for fear and anxiety? Recent work by Hayes suggests that depression may be conceptualized within an emotional processing theory framework and that mechanisms similar to those underlying fear reduction operate during reduction of depression (see Hayes, Beavers, Feldman, Laurenceau, & Perlman, 2005). Data indicating that anger, guilt, and shame decrease with treatment for PTSD suggest that these emotions as well may share common mechanisms with anxiety (see Cahill, Rauch, Hembree, & Foa, 2003, Foa & Rauch, 2004; Rothbaum, Ruef, Litz, Han, & Hodges, 2003).

2. Can findings from neuroscience help clarify principles of emotional processing theory? For example, data from LeDoux's lab indicate that amygdala activation is necessary for extinction (Nader et al., 2000). This

finding is consistent with the proposition that activation of the fear structure is necessary for emotional processing to occur.

3. How can emotional processing theory be integrated with other information processing theories of anxiety (e.g., Mogg & Bradley, 1998)? How does it relate to the recent findings of training of attentional and interpretation biases (Mathews & MacLeod, 2002)?

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