

# Emotional Processing Theory (EPT) and Exposure Therapy for PTSD

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**Abstract** Emotional processing theory developed by E. B. Foa and M. J. Kozak (1986) has informed the conceptualization of anxiety disorders and the development of effective treatments for these disorders, including posttraumatic stress disorder (PTSD). This article presents a summary and update of emotional processing theory as it applies to the treatment of PTSD, data in support of this theory, and clinical examples of how the theory can be used to assist in the treatment of clients with PTSD. Common difficulties confronted during exposure therapy for PTSD, including underengagement and overengagement, are discussed in the context of emotional processing theory and suggestions for how to manage these difficulties are presented.

**Keywords** Exposure therapy · PTSD · Trauma · Emotional processing

## 1. Introduction

Emotional processing theory developed by Foa and Kozak (1986) has informed the conceptualization of anxiety disorders and the development of effective treatments for these disorders, including posttraumatic stress disorder (PTSD). This article presents a summary and update of emotional processing theory as it applies to the treatment of PTSD, data in support of this theory, and clinical examples of how the theory can be used to assist in the treatment of clients with PTSD.

The starting point of the theory is Lang's bioinformational theory of fear (1977, 1979). In this theory, fear is represented in memory as structures that are made up of associated stimulus, response, and meaning elements designed as a program to avoid or escape danger. For instance, a fear structure may include a gun as a stimulus element. This would be connected to various behavioral and physiological response elements (i.e., running away, hiding, heart racing, sweating, etc.). In addition, it would be connected to various meaning elements (i.e., "I am going to die"). When something in the environment matches one or more of the fear structure elements, it is activated and the activation spreads throughout the network.

Foa and Kozak (1986) proposed that specific pathological fear structures underlie the anxiety disorders. Pathological fear structures are different from normal fear structures in that they "involve excessive response elements and resistance to modification" (p. 21) and the associations among the different elements do not accurately represent reality. In applying emotional processing theory to PTSD, Foa and Rothbaum (1998) proposed that the fear structure of PTSD includes excessive stimulus and response elements as well as pathological meaning elements. For example, a survivor of a motor vehicle accident may accurately associate driving fast with danger, but also associate blue cars with danger since the car that hit him was blue. However, in reality blue cars are not more dangerous than red cars. Likewise, the rape survivor who was raped in a park may feel that all parks are dangerous and may completely avoid parks. Either of these trauma survivors may have problematic meaning elements in their fear structures, such as "I am incompetent to handle stress" or "I should have prevented the trauma."

Emotional processing theory as applied to PTSD (e.g., Foa & Riggs, 1993; Foa & Rothbaum, 1998) posits that the fear structures of trauma survivors with PTSD include two basic dysfunctional cognitions that underlie the development

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and maintenance of PTSD. First, the world is completely dangerous (e.g., it is dangerous to be alone). Second, one's self is totally incompetent (e.g., I can't handle any stress, my PTSD symptoms mean that I am going crazy).

Foa and Kozak (1986) propose that effective psychosocial intervention requires modification of the pathological elements of the fear structure. Specifically, they propose two necessary conditions for modification of a fear structure: (1) the fear structure must be activated; and (2) information incompatible with the elements of the fear structure must be presented and integrated into the fear structure to replace pathological elements with realistic ones.

Foa and Cahill (2001) propose that the processes operating in natural recovery from a trauma are similar to those occurring in successful treatment of PTSD. Specifically, they suggested that, after traumatic events the survivors come to view the world as extremely dangerous and themselves as unable to cope with stress. These views reflect fear structures that include many stimulus elements that, despite being objectively non-threatening, have become associated with a meaning of danger as well as responses that were likely adaptive at the time of the trauma and associated with the meaning of incompetence. For most survivors, these pathological elements are corrected through engagement in daily activities that disconfirm them. However, traumatized individuals who systematically avoid trauma-related thoughts and activities do not have the opportunity to incorporate disconfirming information and thus maintain these pathological elements. In this way, chronic PTSD develops and is maintained. Effective treatment involves engaging the patient with the avoided daily activities and the traumatic memory to disconfirm the pathological elements of the fear structure. Indeed, exposure therapy involves systematic repeated confrontation with the traumatic memories (imaginal exposure) and with avoided trauma-related situations (in vivo exposure). According to emotional processing theory, these exposures present patients with information that disconfirms the pathological elements of the fear structure, thereby ameliorating PTSD symptoms. Consistent with this theory, Foa and Rauch (2004) found that greater reduction in thoughts of incompetence and the dangerousness of the world are associated with greater reduction in PTSD symptoms following prolonged exposure therapy.

## 2. Clinical application

One important component of exposure therapy for PTSD is psychoeducation regarding PTSD and its treatment. Psychoeducation about the rationale for treatment provides a framework for the therapist and patient as they plan and carry out both imaginal and in vivo exposures. The rationale for exposure therapy presents the key tenets of emotional

processing theory as described above in a way that is understandable to people with PTSD. A complete description of the presentation of the rationale is beyond the scope of this paper. The reader is referred to Foa and Rothbaum (1998) and Hembree, Rauch, and Foa (2003) for a detailed description of prolonged exposure. Although a key component of any exposure therapy program, PE has not received much empirical investigation. Zoellner, Feeny, Cochran, and Pruitt (2003) found that women who rated the rationale for prolonged exposure therapy for the treatment of PTSD to be more credible and more positively reacted to it than a similar rationale for sertraline were more likely to choose prolonged exposure therapy. Additional studies are underway (e.g., Cochran et al., 2003; Feeny et al., 2004; Zoellner et al., 2004).

Emotional processing theory posits that effective treatment involves an optimal level of activation of the fear structure targeted for treatment. It follows that both too little activation (underengagement) and too much activation (overengagement) can impede the efficacy of the treatment. Both will be discussed below.

### 2.1. Underengagement

Emotional theory posits that if the fear structure is not sufficiently activated, the necessary modifications cannot occur. Underengagement can occur during in vivo or imaginal exposure.

An example of underengagement during imaginal exposure is Ms. M, a 40-year-old woman with PTSD related to a motor vehicle accident that occurred 10 years prior to seeking treatment. During imaginal exposure, she was able to recount the event but did so in a detached and unemotional voice without personal feelings and thoughts. In addition to the flat appearance and non-verbal behaviors, Ms. M reported very low levels of distress [as measured by her Subjective Units of Distress (SUDs) recorded throughout the exposure on a scale of 0 (not at all distressed) to 100 (most distressed ever)]. Her peak SUDs during the first 60 min of imaginal exposure was 50. Although she had listened to the imaginal exposure tape several times between sessions, her peak SUDs remained at 50.

Several studies lend support to the hypothesis that fear activation is necessary for exposure therapy to be successful in reducing anxiety symptoms. Lang, Melamed, and Hart (1970) found that greater initial heart rate reactivity during fear-relevant imagery and greater concordance between self-reported distress and heart rate activation predicted successful treatment in phobic patients. Similar results were reported in treatment for chronic PTSD (Jaycox, Foa, & Morral, 1998; Pitman, Orr, Altman, & Longpre, 1996). Consistent with these results is the finding in animal research that the amygdala must be activated in order for extinction to occur (Nader, Schafe, & LeDoux, 2000).

Clinical experience suggests that patients who do not display distress during recounting of their traumatic experiences do not benefit much from exposure therapy. Ms. M's trauma narrative included many details about the traumatic event but few details about her thoughts, feelings, and physical sensations. Further, Ms. M skipped over parts of the event that held important, personal meaning for her. Specifically, she omitted details about her thoughts and feelings when her car was actually hit. Similarly, she did not include a description of the moment when she regained consciousness and could not feel her legs.

In order to increase patient engagement with the trauma memory during imaginal exposure, more of the elements of the fear structure need to be activated. In cases of underengagement:

1. Remind the patient of the rationale for exposure (i.e., "By talking about the difficult moments during the trauma instead of avoiding it, you can learn that remembering and recounting the traumatic event is not dangerous.")
2. Encourage the patient to keep his/her eyes closed, speak in the present tense, and include details, thoughts, feelings, and actions.
3. Use probe questions during the imaginal exposure in order to encourage patients to include distressing details, thoughts, feelings, or sensory information.
4. If the patient continues to remain underengaged role play how you would like the patient to recount the traumatic event with eyes closed, using the present tense and including all emotional details, thoughts, feelings, and actions.

In the case of Ms. M, after realizing that Ms. M had difficulty emotionally engaging with her traumatic memory, the therapist discussed this issue in the next session prior to the implementation of imaginal exposure. The therapist first praised Ms. M for her courageous recounting of the trauma in the previous session. The therapist mentioned that she noticed there were moments during the story that must have been particularly scary, but that the patient had rushed through them and often did not include her personal thoughts and feelings. The therapist then explained that it was really important to include the scary moments during the imaginal exposure, and focus on her personal thoughts and feelings. Ms. M then related her fear that if she talked about these scary parts of the memory she would "lose it." And the therapist reminded Ms. M of the rationale for imaginal exposure and reassured her that she was safe in the therapist's office. As a result of this discussion, Ms. M included in her narrative many more thoughts and feeling and displayed significantly more fear activation in non-verbal behavior and self-report of SUDs. Indeed, she cried during this exposure. At this point, Ms. M's SUDs began to decrease over the course of treatment.

## 2.2. Overengagement

Emotional processing theory posits that overengagement prevents processing of information that disconfirms the pathological elements in the fear structure and thus impedes the modification of the fear structure.

An example of an overengager is Ms. P, a 35-year-old woman who was raped 15 years prior to seeking treatment for PTSD. During her second imaginal exposure session, Ms. P was brushing her hand past her face as if to brush her hair from her face over and over again as she described the rapist pushing her head down. She then became so anxious she jumped up from her chair and screamed. When discussing what had happened during imaginal exposure, Ms. P reported that she had a vivid flashback and actually began to feel as if she was being raped. She thought she was brushing her long hair back from her face despite the fact that she currently had quite short hair. Ms. P expressed a sense of feeling like a failure because she had lost control during the exposure. Thus, the experience reinforced her fear that she would not be able to control herself if she thought about the rape.

While the theory predicts that overengagement impedes treatment efficacy, this has not been empirically demonstrated. In a review of neurobiological research related to PTSD, Bremner, Krystal, Southwick, and Charney (1995) suggest several potential neural mechanisms for reduced memory function under stress (i.e., impaired learning following high dose epinephrine administration but enhancement at low dose; brain structural changes with stress). However, how these potential mechanisms of overengagement may relate to treatment outcome has not been examined.

Consistent with emotional processing theory and neurobiological evidence, clinical experience suggests that overengagement occurs when the patient's fear structure becomes so activated during exposure that the patient is unable to focus on new information to incorporate into the fear structure. In cases of overengagement:

1. Remind the patient of the rationale for exposure (i.e., "By going over the trauma memory again and again, here in my office, you can learn that it is a memory and it is no longer dangerous. To do that I need you to remember that this memory is in the past.")
2. Help the patient stay grounded in the present. Remind the patient during exposure that this is a memory and ask the patient to open his/her eyes and/or use past tense. Asking the patient to touch the chair that they are sitting in to ground the patient.
3. If the above do not work, have the patient write the traumatic event rather than verbally recounting it.

Ms. P had such a vivid image of the rape that she lost track of the fact that she was sitting in the therapist's office while recounting her trauma. After reiterating the rationale

for imaginal exposure and calming Ms. P, the therapist asked her to tell the story of the rape again with her eyes open. Even with her eyes open, Ms. P was quite anxious while describing the rape but she was able to recount the trauma without jumping out of the chair. She reported very high SUDs and the therapist encouraged her with comments like, “You are doing a good job. Stay with the memory” and “Remember you are with me here and this is just a memory.” The patient demonstrated some reduction in SUDs when she repeated the story. As she listened to the audiotape of the imaginal exposure between sessions, her reported anxiety dropped.

### 2.3. Lack of habituation

Emotional processing theory proposes that as the fear structure is modified, the strength of the responses will diminish (i.e., the urge to flee will be reduced, anxiety related to the feared stimulus will be reduced). Consistent with this, a systematic decline in heart rate reactivity with repeated fear-relevant imagery predicts successful treatment outcome (Lang et al., 1970). Also, greater reductions in reported anxiety during imaginal exposure are significantly related to greater reductions in PTSD symptoms (Rauch, Foa, Furr, & Filip, 2004). Although between session habituation has been consistently related to treatment outcome, the relationship between within session habituation and symptom reduction has been inconsistent (Chaplin & Levine, 1981; van Minnen & Foa, 2005; van Minnen & Hagennaars, 2002).

Thus, when between session habituation is not evident over successive exposures, the therapist should closely examine how exposure is being conducted and modify it to maximize the likelihood of habituation. For example, if a patient continually reports SUDs of 90 for repeated in vivo exposures to malls, the therapist should ensure that the patient is: (1) remaining in the situation for a long enough duration for disconfirmation of the idea that something bad will happen in the mall, (2) not engaging in safety behaviors that may interfere with habituation, and (3) repeating the exposure in a similar context.

The typical guide for in vivo exposure duration is remaining in the situation until SUDs drop to half of the peak rating or until the patient has remained in the situation for 30 min. Some patients who experience particular difficulties in habituating should be instructed to stay for longer duration or conduct more repetitions.

Safety behaviors are behaviors in which a patient engages in order to prevent anticipated negative consequences or to reduce anxiety. It is important to identify such behaviors (e.g., standing by the door, bringing along a safe person, carrying a weapon) as well thoughts that may serve as safety behaviors (e.g., I’ll do this because I have to and then I’ll never do this again; if I pray while I do this I will be safe).

Engaging in safety behaviors during exposure prevents the patients from disconfirmation of the pathological elements in their structure. Specifically, if a patient attributes the successful completion of an exposure exercise to having engaged in a safety behavior rather than to the belief that the situation is safe, the erroneous perception will persist and the symptoms will not diminish.

Finally, habituation may be impeded if the exposures are not sufficiently repeated in a similar context. Although it is important to repeat exposures in different contexts in order to promote generalization, such changes should only be introduced after habituation within the same context had already occurred. Thus, if a patient fails to show between session habituation, the same exposure should be repeated several times. Depending on the exposure, this may mean going to the exact same location, conducting the exposure during the same time of day, and/or examining the patient’s thoughts during the exposure exercise in order to detect what maintains the fear. If a patient is focusing on different elements of the exposure situation at each repetition, habituation may take more repetitions to be evident.

In conclusion, emotional processing theory provides a useful framework for understanding the processes that take place during effective exposure therapy for PTSD. This framework was used in the development of exposure therapy and can also help to conceptualize why difficulties occur and how to overcome them by instituting modification in the treatment procedures. Although research has supported some of the assertions of emotional processing theory, significant empirical questions remain to be examined.

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