The Cutting Edge

PROLONGED EXPOSURE THERAPY: PAST, PRESENT, AND FUTURE

Edna B. Foa, Ph.D., is a professor of Clinical Psychology in Psychiatry at the University of Pennsylvania and Director of the Center for the Treatment and Study of Anxiety. Dr. Foa devoted her academic career to study the psychopathology and treatment of anxiety disorders, primarily obsessive–compulsive disorder (OCD) and posttraumatic stress disorder (PTSD). Her research, aimed at delineating theoretical frameworks, targeted treatments, and treatment mechanisms of pathological anxiety has been highly influential. She is currently one of the leading experts in the areas of PTSD OCD. The treatment program she has developed for PTSD sufferers has received the most evidence for its efficacy and has been disseminated in the United States and around the world.

Dr. Foa has published 18 books and more than 350 articles and book chapters and has lectured extensively around the world. Her work has been recognized with numerous awards and honors, among them the *Distinguished Scientific Contributions to Clinical Psychology Award* from the American Psychological Association; *Lifetime Achievement Award* presented by the International Society for Traumatic Stress Studies; *Lifetime Achievement Award* presented by the Association for Behavior and Cognitive Therapies; TIME 100 most influential people of the world; 2010 *Lifetime Achievement in the Field of Trauma Psychology Award* from the American Psychological Association; and the Inaugural International Obsessive–Compulsive Disorder Foundation Outstanding Career Achievement Award.

As is apparent from its title, I will focus in this article on prolonged exposure therapy (PE), a treatment program developed specifically for posttraumatic stress disorder (PTSD).[1] It is important to note, however, that PE has its roots in the long tradition of exposure therapy for anxiety disorders, and in the conceptualization of these disorders and their treatment within emotional processing theory (EPT).[2,3] Therefore, I will first discuss briefly how EPT views the psychopathology underlying anxiety disorders and the mechanisms that are involved in exposure therapy for these disorders. I will then describe how EPT explains the psychopathology of PTSD and how PE addresses this psychopathology. Next, I will describe the treatment program and summarize the empirical literature that supports its efficacy and effectiveness. Finally, I will discuss novel ways of using exposure therapy for PTSD.

EXPOSURE THERAPY AND ITS THEORETICAL FOUNDATION

Exposure therapy is a set of treatment programs that are commonly used to reduce pathological fear and related emotions, such as guilt, common in posttraumatic stress disorder (PTSD) and other anxiety disorders (e.g., obsessive–compulsive disorder). During exposure for pathological anxiety, patients intentionally confront feared, but otherwise safe, objects, situations, thoughts, sensations, and memories with the goal of reducing fear and other negative reactions to the same or similar stimuli in the future.[4]

Exposure procedures are divided into three primary types: in vivo ("real life"), imaginal, and interoceptive. The selection of the type of exposure is dictated by the pathological characteristics of a given disorder. It is also often the case that several types of exposure are concurrently used in exposure programs.

The conceptualization of anxiety disorders was greatly influenced by Mowrer's two-factor model,[5] which explained the acquisition of fear as involving classical conditioning, and the maintenance of the conditioned fear avoidance as involving and operant conditioning. Accordingly, avoidance prevents the organism from extinction learning; that is, from learning that the CS no longer predicts harm. Mowrer's two-factor model implies that therapy must not only promote extinction through confrontation with erroneously feared objects, but also eliminate avoidances that would impede extinction learning.

In 1986, Foa and Kozak[3] developed emotional processing theory (EPT) in which they expanded on

*Correspondence to: Edna B. Foa, Center for the Treatment and Study of Anxiety, University of Pennsylvania, 3535 Market Street, 6th Floor, Philadelphia, PA 19104. E-mail: foa@mail.med.upenn.edu

No conflict of interest was declared.

DOI 10.1002/da.20907
Published online in Wiley Online Library (wileyonlinelibrary.com).

these early learning accounts of anxiety and its treatment by adopting Lang's concept of the fear (emotional) structure,[6,7] to create a comprehensive model for understanding pathological anxiety and the mechanisms involved in exposure therapy for anxiety disorders. According to EPT, a fear (emotional) structure is a program for escaping or avoiding danger that includes representations of feared stimuli, responses, and the meaning of stimuli and responses. This structure is activated by input that matches the information stored in the structure. Foa and Kozak[3] emphasized the importance of meaning representations as distinguishing between normal and pathological fear structures which underlie the different anxiety disorders.

Foa and Kozak[3] assert that psychological interventions known to reduce fear, such as exposure, achieve their effects by "emotional processing," the process by which accurate information is incorporated into the fear structure and modifies the pathological elements in the structure. Influenced by modern learning theories that conceptualize extinction as creating new associations rather than modifying old associations,[8] Foa and McNally[9] proposed that exposure therapy does not alter the existing pathological structure, but rather forms competing structures that do not include pathological associations among stimulus, response, and meaning representations. For therapy to be successful, the new emotional structure needs to be more easily retrievable when shared elements between the old and the new structures are present; conversely, when the old pathological structure is activated, relapse occurs.

EPT specifies two conditions that are necessary for therapeutic fear reduction to occur. First, the fear (emotional) structure must be activated in order for it to be available for modification. Second, new information that is incompatible with the pathological elements of the fear structure must be available and incorporated into the pathological memory structure (or form a new nonpathological competing structure). Exposure is a convenient and efficient way to satisfy these two conditions: approaching feared, distressing but safe stimuli is likely to activate the fear (emotional) structure and at the same time provide corrective information about consequences of approaching the feared stimuli (e.g., "The small dog did not bite me," "When facing the dog, my anxiety did not last forever"). The incorporation of realistic information into the fear structure is the essence of emotional processing and explains why exposure effectively reduces pathological emotions. EPT's emphasis on integration of disconfirming information as the mechanism of change is consistent with Rescorla and Wagner's[10] mathematical model of classical conditioning in which learning results from the discrepancy between what is expected to happen and what actually happens. This learning then modifies future expectations, according to the Rescorla–Wagner model, or meaning elements according to EPT. For a detailed review of research in support of EPT, see Gillihan and Foa.[11]

EMOTIONAL PROCESSING THEORY OF PTSD

Although individual anxiety disorders share some common elements, such as the belief that anxiety will last forever in a feared situation, these disorders are distinguished with respect to the specific pathological elements of the underlying fear structure.[2] Because activation of the fear structure depends on the match between the information that is presented and elements of the fear structure, exposure interventions are tailored to the pathological elements that are characteristic of each disorder. Matching the exposure to the disorder-specific fear structure should lead to greater fear reduction by promoting activation of the structure and providing information that is incompatible with the key pathological elements of the structure. Exposure techniques are also designed to eliminate characteristic avoidance behaviors, such as situational avoidance, safety behaviors, ritualizing, or distraction, which would prevent emotional processing from occurring.

In PTSD, the fear (emotional) structure associated with the traumatic memory is conceived as a specific pathological fear structure that include erroneous associations among stimuli and response that were present at the time of the trauma and their meaning. First, the traumatic memory structure is thought to be characterized by a particularly large number of stimulus elements that are erroneously associated with danger (e.g., "All men are rapists"), which renders the fear easily activated and is reflected in the perception that the world is entirely dangerous. Second, the representations of how the person behaved during and after the trauma as well as the presence of PTSD symptoms become associated with the meaning of self-incompetence and inability to cope. These two erroneous perceptions, "The world is entirely dangerous" and "I am completely incompetent," promote avoidance of trauma-related thoughts, images, and situations, which in turn prevents emotional processing and the resultant recovery.

PROLONGED EXPOSURE FOR PTSD: ITS THEORETICAL FOUNDATION, PRACTICE, EFFICACY, AND EFFECTIVENESS

PE for PTSD illustrates the way in which the particular exposure program developed for a specific anxiety disorder is informed by the nature of the psychopathology underlying the disorder.[1,12] As noted above, EPT conceptualizes chronic PTSD as a failure to adequately process the trauma memory due to extensive avoidance of thoughts and situations that are trauma reminders. These behaviors maintain individuals' erroneous negative beliefs about themselves and

the world and prevent emotional processing from occurring. Accordingly, the goal of PE is to promote emotional processing through deliberate systematic confrontation with trauma-related stimuli. In vivo exposure to trauma reminders and imaginal exposure (repeated revisiting and recounting the trauma aloud) followed by discussion of the revisiting experience (processing) are used concurrently in order to disconfirm the erroneous beliefs that underlie PTSD. Typically, PE is conducted over 8–15 sessions, with each session lasting 60–90 min.

In vivo exposures are designed to target PTSD patients' erroneous perceptions that safe stimuli are harmful and therefore should be avoided, that their anxiety will last forever if they remain in trauma-related situations rather than escape them, and that they are incapable of coping with stress and distress. Consequently, exposure exercises typically include systematically and gradually approaching safe situations that the person perceives to be dangerous (e.g., going out in the evening with friends), as well as situations that the individual avoids not because they are perceived as dangerous but because they are trauma reminders and cause high distress that they would not be able to tolerate (e.g., watching news about the war in Afghanistan). Any safety behaviors that interfere with activation of the trauma memory structure and/or prevent disconfirmation of danger are detected and eliminated when conducting in vivo exposure. The in vivo exposures are designed to ensure successfully achieving the two necessary conditions for emotional processing: activation of the trauma memory and disconfirmation of the expected "disasters." Unlike protocols in other anxiety disorders, in PE in vivo exposures are rarely practiced in session; rather, they are assigned as homework in order to allow sufficient time for imaginal exposure in session.

Imaginal exposure comprises a large part of the treatment session. Individuals with PTSD frequently hold erroneous beliefs that recalling the trauma memory is dangerous or harmful (e.g., will cause them to go crazy or lose control), that anxiety will last forever when thinking about the trauma, and that remembering is like being back in the trauma. Revisiting and recounting the most distressing traumatic memory in imagination is designed to help patients organize the memory, reexamination of negative perceptions about their conduct during the trauma, regain new perspectives about themselves and others, distinguish between thinking about the trauma and reexperiencing the trauma, generate habituation to the trauma memory so that the trauma can be remembered without causing undue anxiety, and foster the realization that engaging in the trauma memory does not result in harm. An important consideration is to ensure that patients are appropriately emotionally engaged (being activated) with the trauma memory during imaginal exposure.

Imaginal exposure creates a powerful opportunity for new learning by activating the trauma memory structure and bringing to the surface both unhelpful or unrealistic beliefs that maintain the symptoms of PTSD as well as new insights or awareness about evidence that contradicts the erroneous beliefs. However, patients may have difficulty identifying and integrating disconfirming information that emerges from imaginal exposure on their own. "Processing" is conducted immediately after imaginal exposure in order to explore contradictions between the patient's erroneous perceptions and what they had recounted during the imaginal exposure. Encouraging patients to elaborate on new insights and making them explicit is likely to facilitate emotional processing and modification of the pathological emotional structure.

PE is an exposure therapy for PTSD that received the most empirical evidence for its efficacy. It has been shown to be highly effective for patients with a wide variety of traumatic experiences. In a series of randomized controlled trials, PE demonstrated large treatment effects compared to waitlist (WL) control groups and similar effects compared to other active treatments, such as stress inoculation training, cognitive processing therapy, and eye movement desensitization and reprocessing.[13–17] A recent meta-analysis found a large-effect size for PE compared to WL or control active treatments at posttreatment and follow-up.[18] Research also indicates that PE can be effectively disseminated to community therapists in the United States and elsewhere.[13,19–21] Importantly, PE is well tolerated by patients[22] and does not cause long-term exacerbation of symptoms.[23]

FUTURE DIRECTION IN EXPOSURE THERAPY FOR PTSD

Although exposure therapy has been studied extensively in the treatment of anxiety disorders, including PTSD, there continue to be important areas of ongoing investigation. One pressing question is how to increase the immediate and long-term efficacy of PE. Despite its success, some patients do not improve and among the patients who experience improvement, many remain somewhat symptomatic. Foa and Kozak[24] suggest that the progress of CBT might be slow due to "alienation from experimental psychology and psychopathology research" (p 601). Recently, investigators have begun integrating research from basic science in an attempt to enhance exposure treatment so that it may be effectively delivered in fewer sessions. Specifically, researchers have been testing clinical applications of pharmacological agents that may enhance inhibitory learning during exposure. For example, D-cycloserine has been shown to enhance the effects of exposure therapy for height phobia,[25] but its effects on exposure therapy for PTSD is as yet unknown. Other agents, such as yohimbine and methylene blue, are also being investigated as

possible adjuncts to exposure therapies to enhance fear extinction.

Researchers have also been challenged by the fact that extinction learning does not appear to eliminate or modify pathological fear responses, but rather create new learning that inhibits activation of pathological fear structures. Research shows that extinguished fear response (CS) can return when the unconditioned stimulus (US) is presented in the absence of the CS (reinstatement,)[26] when the CS is presented in contexts other than the extinction setting (renewal),[27] and spontaneously (spontaneous recovery) [e.g., [28]]. These phenomena indicate that extinction procedures do not eradicate the original conditioning learning. In therapy, the return of pathological fear constitutes a relapse. Although return of symptoms is rare among patients with PTSD,[26] some patients do relapse after successfully benefiting from PE.

Recent evidence suggests that pathological fear responses may in fact be changed if corrective information is presented during the reconsolidation period when retrieved information is liable.[27] Individuals who received a reminder of the conditioned stimulus in the reconsolidation window failed to demonstrate spontaneous recovery[30] and reinstatement.[31] These remarkable findings suggest that new information can be incorporated into old memory structures when the memory is malleable during the reconsolidation window. Although this hypothesis requires further investigation, an important challenge for researchers is to explore whether this basic science research can be translated to enhance exposure in treatment protocols for PTSD.

A variant of PE was found helpful with complicated grief where individuals recount the story of the death using imaginal exposure and conduct grief-related in vivo exposures in a fashion similar to PE. This treatment has been found more effective than interpersonal therapy in the reduction of grief-related symptoms.[28] Exposure has also been used in the treatment of depression to target experiential avoidance. In Adele Hayes' exposure-based cognitive therapy (EBCT) for depression, patients are encouraged to approach negative thoughts and emotions associated with their depression through activities, such as writing essays about their depression and recounting these essays in therapy sessions. In a preliminary open treatment trial, EBCT effectively reduced depressive symptoms and symptom reduction was negatively associated with avoidance.[29] Although this work is still in its early stages, these research literatures illustrate how exposure techniques can have therapeutic applications beyond the reduction of pathological fear. Specifically, exposure may be effective when pathological emotions that arise from erroneous perceptions are maintained through cognitive and behavioral avoidances that prevent access to and integration of disconfirming information in the memory structure. Indeed, prolonged exposure reduces not only PTSD symptoms but also depression, anger, guilt, and general anxiety.

Although exposure therapy has been used in psychotherapy for more than 50 years, there continues to be exciting frontiers for exploration. Recent advances in our understanding of basic learning processes and the links between biological and behavioral learning mechanisms have the potential to break the "efficacy ceiling"[24] of cognitive behavior therapy by refining and augmenting exposure treatment techniques to maximize therapeutic outcomes.

REFERENCES

1. Foa EB, Hembree EA, Rothbaum BO. Prolonged Exposure Therapy for PTSD: Emotional Processing of Traumatic Experiences, Therapist Guide. New York: Oxford University Press; 2007.
2. Foa EB, Kozak MJ. Treatment of anxiety disorders: implications for psychopathology. In: Tuma EH, Maser JD, editors. Anixety and the Anxiety Disorders. Hillsdale, NJ: Erlbaum; 1985: 421–452.
3. Foa EB, Kozak MJ. Emotional processing of fear: exposure to corrective information. Psychol Bull 1986;99:20–35.
4. Marks IM. Reduction of fear: towards a unifying theory. J Can Psychiatr Assoc 1973;18:9–12.
5. Mowrer OH. On the dual nature of learning—a reinterpretation of conditioning and problem solving. Harvard Educ Rev 1947;17:102–148.
6. Lang PJ. Imagery in therapy: an information processing analysis of fear. Behav Ther 1977;8:862-866.
7. Lang PJ. A bio-informational theory of emotional imagery. Psychophysiology 1979;16:495–512.
8. Bouton ME, Swartzentruber D. Sources of relapse after extinction in Pavlovian and instrumental learning. Clin Psychol Rev 1991;11:123–140.
9. Foa EB, McNally RJ. Mechanisms of change in exposure therapy. In: Rapee RM, editor. Current Controversies in the Anxiety Disorders. New York: Guilford Press; 1996:329–343.
10. Rescorla RA, Wagner AR. A theory of Pavlovian conditioning: variations in the effectiveness of reinforcement and nonreinforcement. In: Black AH, Prokasy WF, editors. Classical Conditioning II: Current Research and Theory. New York: Appleton Century Crofts; 1972:64–99.
11. Gillihan SJ, Foa, EB, in press. Fear extinction and emotional processing theory: a critical review. In: Schachtman TR, Reilly S, editors. Conditioning and Animal Learning: Human and Non-Human Animal Applications. Oxford, UK: Oxford University Press.
12. Foa EB, Cahill SP. Psychological therapies: Emotional Processing. In: Smelser NJ, Bates PB. editors. International Encyclopedia of the Social and Behavioral Sciences. Oxford: Elsevier; 2001:12363–12369.
13. Foa EB, Hembree EA, Cahill SP, et al. Randomized trial of prolonged exposure for posttraumatic stress disorder with and without cognitive restructuring: outcome at academic and community clinics. J Consult Clin Psychol 2005;73:953–964.
14. Marks I, Lovell K, Noshirvani H, Livanou M, Thrasher S. Treatment of posttraumatic stress disorder by exposure and/or cognitive restructuring: a controlled study. Arch Gen Psychiatry 1998;55:317–325.

15. Paunovic N, Öst LG. Cognitive-behavior therapy vs exposure therapy in the treatment of PTSD in refugees. Behav Res Ther 2001;39:1183–1197.
16. Resick PA, Nishith P, Weaver TL, Astin MC, Feuer CA. A comparison of cognitive-processing therapy with prolonged exposure and a waiting condition for the treatment of chronic posttraumatic stress disorder in female rape victims. J Consult Clin Psychol 2002;70:867–879.
17. Rothbaum BO, Astin MC, Marsteller F. Prolonged exposure versus eye movement desensitization and reprocessing (EMDR) for PTSD rape victims. J Trauma Stress 2005;18: 607–616.
18. Powers MB, Halpern JM, Ferenschak MP, Gillihan SJ, Foa EB. A meta-analytic review of prolonged exposure for posttraumatic stress disorder. Clin Psychol Rev 2010;30:635–641.
19. Asukai N, Saito A, Tsuruta N, Kishimoto J, Nishikawa T. Efficacy of exposure therapy for Japanese patients with posttraumatic stress disorder due to mixed traumatic events: a randomized controlled study. J Trauma Stress 2010;21:340–343.
20. Karlin BE, Ruzek JI, Chard KM, et al. Dissemination of evidence-based psychological treatments for posttraumatic stress disorder in the Veterans Health Administration. J Trauma Stress 2010;23:663–673.
21. Nacasch N, Foa EB, Huppert JD, et al. The efficacy of prolonged exposure therapy for combat and terror-related PTSD: a randomized control comparison with treatment as usual. J Clin Psychiatry 2010;72:1174–1180.
22. Hembree EA, Foa EB, Dorfan NM, Street GP, Kowalski J, Tu X. Do patients drop out prematurely from exposure therapy for PTSD? J Trauma Stress 2003;16:555–562.
23. Foa EB, Zoellner LA, Feeny NC, Hembree EA, Alvarez-Conrad J. Does imaginal exposure exacerbate PTSD symptoms? J Consult Clin Psychol 2002;70:1022–1028.
24. Foa EB, Kozak MJ. Beyond the efficacy ceiling? Cognitive behavior therapy in search of theory. Behav Ther 1997;28:601–611.
25. Hofmann SG, Pollack MH, Otto MW. Augmentation treatment of psychotherapy for anxiety disorders with d-cycloserine. CNS Drug Rev 2006;12:208–217.
26. Foa EB, Dancu CV, Hembree EA, Jaycox LH, Meadows EA, Street GP. A comparison of exposure therapy, stress inoculation training, and their combination for reducing posttraumatic stress disorder in female assault survivors. J Consult Clin Psychol 1999;67:194–200.
27. Schiller D, Monfils MH, Raio CM, Johnson DC, Ledoux JE, Phelps EA. Preventing the return of fear in humans using reconsolidation update mechanisms. Nature 2009;463:49-53.
28. Shear K, Frank E, Houck PR, Reynolds3rd CF. Treatment of complicated grief: a randomized controlled trial. J Am Med Assoc 2005;293:2601–2608.
29. Hayes AM, Beevers CG, Feldman GC, Laurenceau JP, Perlman C. Avoidance and processing as predictors of symptom change and positive growth in an integrative therapy for depression. Int J Behav Med 2005;12:111–122.
30. Leung HT, Westbrook RF. Spontaneous recovery of extinguished fear responses deepens their extinction: a role for error-correction mechanisms. J Exp Psychol Anim Behav Process 2008;34:461–474.
31. Rescorla RA, Heth CD. Reinstatement of fear to an extinguished conditioned stimulus. J Exp Psychol Anim Behav Process 1975;1:88–96.