

ANXIETY SENSITIVITY AND ITS IMPLICATIONS FOR UNDERSTANDING AND TREATING PTSD

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INTRODUCTION

Empirically supported psychosocial treatments for posttraumatic stress disorder (PTSD) all entail some form of trauma-related exposure therapy (TRE). Although TRE tends to be effective, it is not effective for all PTSD sufferers. New developments in trauma treatment are needed to address this problem. This is especially important given that PTSD is a severe, prevalent, and often chronic disorder (APA, 2000). In this book there are a number of important suggestions for improving treatment outcome, such as reducing trauma-related anger, augmenting treatment with virtual reality interventions, and improving social support (Cahill, Rauch, Hembree, & Foa, 2002; Rothbaum, Ruef, Litz, Han, & Hodges, 2002; Tarrier & Humphreys, 2002).

In this chapter I would like to suggest another approach, which had some similarities to that taken by Falsetti et al. (chapter 3), but differs in its target population, conceptual basis, and treatment ingredients. Unlike the work by Falsetti and colleagues, we were interested in improving treatment outcome for PTSD sufferers in general, whereas Falsetti et al.

The work reported in this chapter was supported in part by grants from the British Columbia Health Research Foundation.

focused on PTSD with comorbid, recurrent panic attacks. Our treatment approach was derived largely from anxiety sensitivity theory (Taylor, 1999), and it emphasizes some interventions (TRE and interoceptive exposure) and deliberately omits others (cognitive restructuring and breathing retraining). In the following sections I will briefly review TRE, followed by a discussion of the theoretical and empirical reasons why anxiety sensitivity should be explicitly considered in understanding and treating PTSD.

TRAUMA-RELATED EXPOSURE THERAPY

TRE is one of the most effective treatments for PTSD (Chambless & Ollendick, 2001; van Etten & Taylor, 1998). Controlled studies, particularly the studies that do not have the methodological shortcomings of early research, indicate that TRE may be as effective or perhaps more effective than other psychosocial treatments (e.g., Foa & Meadows, 1997; Taylor et al., 2003). A recent meta-analysis further indicates that, in the short term, TRE is as effective as the most effective PTSD medications (e.g., selective serotonin reuptake inhibitors) (van Etten & Taylor, 1998). Treatment-related gains for TRE have been found to be maintained at follow-up intervals of at least 12 to 15 months (Foa et al., 1999a; van Etten & Taylor, 1998). TRE may be more effective than medications at long-term follow-up, because TRE teaches patients skills that they can continue to use, as needed, once the therapy sessions have ended. In comparison, patients treated with medications are not typically trained in symptom-management skills, and they may relapse when medication is discontinued. (Little is known about the long-term effects of medication treatment for PTSD.)

TRE entails a combination of (a) imaginal exposure to traumatic memories (e.g., writing out and rereading a description of the traumatic event), and (b) in vivo exposure to distressing but harmless reminders of the trauma (e.g., returning to the scene of a traumatizing road traffic collision) (Foa & Rothbaum, 1998; Marks, Lovella, Noshirvania, Livanou, & Thrasher, 1998; Taylor et al., 2001, 2003). TRE is thought to exert its effects by exposing the person to corrective information (Foa & Rothbaum, 1998). For example, exposure exercises can modify trauma-related beliefs (e.g., the belief that the world is a dangerous place), which in turn can reduce PTSD symptoms (e.g., hypervigilance). Physiological habituation, resulting from exposure to anxiety-provoking but harmless trauma-related stimuli, also is thought to provide the person with corrective information about the harmlessness of these stimuli.

TRE is not effective for all patients; some have a partial response and others do not respond at all. Residual symptoms are often seen even in patients who no longer meet *DSM-IV* criteria for PTSD at the end of treatment (e.g., Foa et al., 1999a; Marks et al., 1998; Taylor et al., 2003). Therefore we need to search for ways of boosting its efficacy. Several investigators have examined whether outcome is improved by combining TRE with cognitive restructuring. So far the results have been disappointing, suggesting that treatment outcome is not enhanced (Foa et al., 1999a; Marks et al., 1998).

Similarly, there is no evidence that outcome is improved by teaching patients coping exercises such as breathing retraining. Foa et al. (1999a) combined TRE with breathing retraining, whereas other studies used TRE without breathing retraining (Marks et al., 1998; Taylor, et al. 2003). Foa et al.'s treatment tended to be more effective than that of Marks et al., but less effective than that of Taylor et al. Thus, it is unclear whether breathing retraining improves outcome. A concern with breathing retraining is that it can impair the efficacy of behavioral and cognitive-behavioral treatment of panic disorder, because it encourages patients to avoid feared bodily sensations (Schmidt et al., 2000; Taylor, 2000, 2001). The same may apply to PTSD. A more promising direction for improving PTSD treatment comes from recent research on anxiety sensitivity and PTSD.

ANXIETY SENSITIVITY

Several lines of evidence suggest that anxiety sensitivity plays an important role in PTSD, and that treatment outcome may be enhanced by combining TRE with interventions that reduce anxiety sensitivity. Anxiety sensitivity is the fear of arousal-related sensations, arising from beliefs that these sensations have harmful consequences. Research suggests that there are at least three basic dimensions of anxiety sensitivity: (a) fear of publicly observable anxiety reactions (e.g., fear of trembling, arising from beliefs that trembling will attract ridicule or rejection), (b) fear of cognitive dyscontrol (e.g., fear of concentration difficulties arising from beliefs that such difficulties are the harbingers of insanity), and (c) fear of somatic sensations (e.g., fear of palpitations arising from beliefs that cardiac sensations lead to heart attacks) (Taylor, 1999).

Evidence suggests that anxiety sensitivity contributes to, or amplifies, the intensity of emotional reactions (particularly anxiety), and that it also plays a role in producing panic attacks (see Taylor, 1999, 2000, for reviews).

Table 4.1 Anxiety Sensitivity in PTSD, Panic Disorder, and Normal Controls

	PTSD (without panic disorder)			Panic disorder (without PTSD)			Normal controls		
	M	SD	N	M	SD	N	M	SD	N
Taylor et al., 1992	31.6	12.8	32	36.6	12.3	151	17.8	8.8	1,013
Taylor et al., 2001	30.5	13.8	41	—	—	—	—	—	—
Taylor et al., 2003	31.0	14.6	42	—	—	—	—	—	—

To illustrate, a person who is phobic about driving would experience anxiety when required to drive. If that person had elevated anxiety sensitivity, then he or she would also become anxious about being anxious. Thus, the fear of driving would be amplified.

Although anxiety sensitivity plays a particularly important role in panic disorder, there is growing evidence of its importance in PTSD. Table 4.1 shows the mean scores (prior to treatment) for a widely used measure of anxiety sensitivity, the Anxiety Sensitivity Index (Peterson & Reiss, 1992). As suggested by the table, the severity of anxiety sensitivity in PTSD is somewhat lower than in panic disorder ($p < .06$) but people with PTSD have much greater anxiety sensitivity than normal controls ($p < .0005$) (Taylor, Koch, & McNally, 1992). Anxiety sensitivity also tends to be higher in PTSD compared with other anxiety disorders (apart from panic disorder) (Taylor et al., 1992). Research further shows that the severity of anxiety sensitivity is correlated with the severity of PTSD symptoms (Fedoroff, Taylor, Asmundson, & Koch, 2000).

There appear to be at least two ways that elevated anxiety sensitivity and PTSD may be related. Elevated anxiety sensitivity may be a predisposing factor, predating the development of PTSD. People with elevated anxiety sensitivity, compared with people with low or normal levels of anxiety sensitivity, would tend to have more intense emotional reactions to traumatic stressors because of the amplifying nature of anxiety sensitivity. That is, the person would become alarmed by the stressor, and also alarmed by their anxiety sensations, thereby amplifying their emotional response and correspondingly increasing the risk of developing PTSD.

Another possible relationship between elevated anxiety sensitivity and PTSD is that both may arise from a traumatic stressor. Such a stressor may not only trigger PTSD but also cause the person to become frightened by all stimuli associated with the stressor, including arousal-related

bodily sensations. Through a process of associative learning (interoceptive conditioning) (Bouton, Mineka, & Barlow, 2001) anxiety sensitivity may be inflated by trauma exposure. Anxiety sensitivity may then amplify PTSD symptoms. For example, the person may become alarmed by re-experiencing symptoms, believing them to be harbingers of insanity. Similarly, the person may become alarmed by hyperarousal symptoms (e.g., palpitations), believing them to be signs of some physical catastrophe such as cardiac arrest (Fedoroff et al., 2000).

Regardless of the actual relations between elevated anxiety sensitivity and PTSD, the above-mentioned data and arguments suggest that the treatment of PTSD may be improved by incorporating treatments that reduce anxiety sensitivity, regardless of whether the person has comorbid panic disorder.

Additional suggestive evidence for the importance of these interventions comes from examining the effects of TRE on anxiety sensitivity. Although there are specialized interventions for reducing anxiety sensitivity (as described below), TRE can reduce anxiety sensitivity to some extent. This is because TRE encourages patients to be exposed to arousal-related sensations (as part of their exposure to trauma-related stimuli). Such exposure may help the patient learn that these sensations do not have harmful consequences (e.g., experiencing palpitations during TRE helps the patient learn that palpitations do not lead to heart attacks).

Two studies from our clinic show that TRE-related reductions in anxiety sensitivity are correlated with reductions in PTSD symptoms. In a study of the efficacy of cognitive-behavioral therapy for 28 patients with PTSD due to road traffic collisions, we computed pre- to posttreatment residual gain scores (change scores) for several measures, including a measure of anxiety sensitivity, a measure of PTSD symptoms, and measures of the strength of belief in the dangerousness of road travel (trauma-related beliefs). The latter were assessed because trauma-related beliefs are thought to play an important role in PTSD (Foa & Rothbaum, 1998; Foa, Ethers, Clark, Tolin, & Orsillo, 1999b). We found that the degree of pre- to posttreatment reduction in anxiety sensitivity was the strongest predictor of reductions in global severity of PTSD symptoms, even though anxiety sensitivity was not directly targeted during treatment (Fedoroff et al., 2000).

A similar finding emerged in another study of 15 PTSD patients treated with TRE, without targeting anxiety sensitivity (Taylor et al., 2003). The participants had PTSD arising from a number of traumata, commonly physical abuse, sexual assault, or road traffic collisions. We computed

Table 4.2 Correlations Among Change Scores (Pre- to Posttreatment Residual Gain Scores)

	Changes in PTSD symptoms			
	Reexperiencing	Avoidance	Numbing	Hyperarousal
<i>Changes in anxiety sensitivity</i>				
Fear of publicly observable anxiety reactions	.32	.30	.48	.34
Fear of cognitive dyscontrol	.65**	.29	.78**	.39
Fear of somatic sensations	.53*	.32	.40	.24
<i>Changes in trauma-related beliefs</i>				
Negative beliefs about self	.26	.09	.29	-.05
Negative beliefs about world	.18	.19	.05	-.17
Self-blame	.19	.01	.39	.00

* $p < .05$. ** $p < .01$. Medium-sized or larger correlations ($\geq .30$) are in boldface.

pre- to posttreatment residual gain scores for PTSD symptoms, residual gains for the three dimensions of anxiety sensitivity, and residual gains for three dimensions of trauma-related beliefs. The latter were assessed by the recently developed Posttraumatic Cognitions Inventory (Foa et al., 1999b). All PTSD symptoms were assessed by structured interview (the Clinician Administered PTSD Scale; Blake et al., 1997).

Table 4.2 lists the variables and shows the correlations among residual gain scores. Due to the small sample size, the power to detect significant correlations was limited, and so we supplemented significance tests by classifying the correlations in terms of Cohen’s (1988) criteria: Large effects are indicated by correlations of .50 and higher, medium correlations are .30 to .49, and small correlations are .10 to .29. Correlations smaller than .10 can be regarded as trivial.

Scores on all measures tended to decline from pre- to posttreatment. The table shows that changes in PTSD symptoms were significantly correlated only with changes in anxiety sensitivity. Most of the correlations with anxiety sensitivity were medium sized or larger, whereas most of the correlations among trauma-related beliefs and PTSD symptoms were small or trivial in size. These findings, like those of Fedoroff et al. (2000), are consistent with the view that reducing anxiety sensitivity may be useful in reducing PTSD. These findings are only suggestive, however, because the research was correlational, and anxiety sensitivity was not directly targeted. Nevertheless, they encourage further research to directly examine the merits of reducing anxiety sensitivity in the treatment of PTSD.

INTEROCEPTIVE EXPOSURE THERAPY (IE)

There are more powerful, direct methods than TRE for reducing anxiety sensitivity. Among the most potent is IE (Taylor, 1999, 2000). This involves deliberately inducing arousal-related bodily sensations so patients can learn that the sensations have no harmful consequences. For example, a person might be asked to hyperventilate for 2 minutes to induce palpitations and dizziness, and thereby learn that these sensations do not have catastrophic consequences such as heart attacks or insanity (see Taylor, 2000, for detailed IE protocols).

IE is widely used to reduce anxiety sensitivity in panic disorder. Little is known about whether it is useful in reducing PTSD, although the above-mentioned evidence linking PTSD and anxiety sensitivity suggests that IE may be useful. To our knowledge, only one group of investigators has considered using IE in the treatment of PTSD. Falsetti and colleagues included IE in their multicomponent treatment of patients who suffer from both PTSD and recurrent panic attacks. Their preliminary results are encouraging, although it is not known whether IE is useful in treating PTSD in general, regardless of whether the patient also suffers from recurrent panic attacks.

A potential limitation of the Falsetti protocol is its reliance on breathing retraining, which appears to undermine the efficacy of panic treatment because patients sometimes use the breathing exercises as a way of avoiding feared bodily sensations (Schmidt et al., 2000; Taylor, 2001). Another potential limitation of the Falsetti protocol is the reliance on cognitive restructuring, which does not enhance the efficacy of TRE (Foa et al., 1999a; Marks et al., 1998). A more promising protocol would be one that gives a greater emphasis to both TRE and IE.

IE may be a useful pretreatment for patients who are to undergo TRE. By first reducing the patient's anxiety sensitivity, that patient may be better able to engage in TRE, both during the treatment session and during homework assignments. That is, persons pre-treated with IE should experience less distress during TRE because their anxiety about anxiety has been reduced. As a result, the person may be more likely to complete TRE exercises, which are anxiety provoking. Clinically, it is not uncommon for people with TRE to avoid homework assignments and to limit within-session exposure exercises because the exercises are distressing. Reducing the person's anxiety sensitivity should make these exercises easier (less distressing) for the patient. Thus, TRE may be more efficacious when preceded by IE. If IE enhances the patient's adherence to, and

completion of, TRE exercises, then the speed of reduction in PTSD symptoms for TRE should also be faster when this treatment is preceded by IE, because the enhanced adherence and completion of TRE will increase its “dose.” The potential merits of this treatment protocol are currently under investigation in our research clinic.

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