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Anxiety sensitivity and post-traumatic stress reactions: Evidence for intrusions and physiological arousal as mediating and moderating mechanisms

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ABSTRACT

A growing body of research has implicated anxiety sensitivity (AS) and its dimensions in the development of post-traumatic stress disorder (PTSD). However, the mechanism(s) that may account for the association between AS and PTSD remains unclear. Using the "trauma film paradigm," which provides a prospective experimental tool for investigating analog intrusion development, the present study examines the extent to which intrusions mediate the association between AS and the development of posttraumatic stress reactions. After completing a measure of AS and state mood, unselected participants ($n = 45$) viewed a 10 min film of graphic scenes of fatal traffic accidents and then completed a second assessment of state mood. Participants then kept a daily diary to record intrusions about the film for a one-week period. Post-traumatic stress reactions about the film were then assessed after the one-week period. The results showed that general AS and physical and cognitive concerns AS predicted greater post-traumatic stress reactions about the film a week later. Furthermore, the number of intrusions the day after viewing the traumatic film, but not fear and disgust in response to the trauma film, mediated the association between general AS (and AS specifically for physical and cognitive concerns) and post-traumatic stress reactions a week later. Subsequent analysis also showed that physiological arousal during initial exposure to the traumatic film moderated the association between general AS and the number of intrusions reported the day after viewing the film. The implications of these analog findings for conceptualizing the mechanism(s) that may interact to explain the role of AS in the development of PTSD and its effective treatment are discussed.

1. Introduction

Anxiety sensitivity (AS) refers to the fear of anxiety-related bodily sensations derived from beliefs that these symptoms have harmful physical, psychological, or social consequences (Reiss & McNally, 1985). AS is a trait-like cognitive vulnerability that amplifies preexisting anxiety levels such that those high in AS misinterpret physical sensations as danger signals and, as a result, experience elevated levels of anxiety. AS is thought to arise from a combination of genetic predispositions (Stein, Jang, & Livesley, 1999) and learning experiences that result in the acquisition of beliefs about potential harmful effects of autonomic arousal (e.g., Stewart et al., 2001). AS is thought to amplify fearful reactions, thereby placing individuals at risk for the development of anxiety-related conditions, especially panic disorder (Olatunji & Wolitzky-Taylor, 2009; Schmidt, Lerew, & Jackson, 1997; Schmidt, Zvolensky, & Maner, 2006). However, a growing body of research has also implicated AS in the development of post-traumatic stress disorder (PTSD; Olatunji, Armstrong, Fan, & Zhao, 2014; Taylor, 2003). For example, Lang, Kennedy, and Stein (2002) found significantly higher AS among women who developed PTSD in response to intimate partner violence compared to both those experiencing such violence who did not develop PTSD and women with no trauma history. Research has also shown that AS predicts PTSD symptoms even when controlling for negative affect, trait anxiety, and the number of types of trauma exposures (Kilic, Kilic, & Yilmaz, 2008; Vujanovic, Zvolensky, & Bernstein, 2008).

Prospective research has also implicated AS in PTSD (Keogh, Ayers, & Francis, 2002). For example, Marshall, Miles, and Stewart (2010) employed cross-lagged panel analysis of interview data collected from survivors of traumatic physical injury within days of physical injury, at 6-month follow-up, and at 12-month follow-up.

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The results indicated that AS predicted subsequent PTSD symptom severity. AS may contribute to symptoms of PTSD because individuals high in AS catastrophize their own physical sensations during a traumatic event. Indeed, physical sensations are heightened in panic disorder and PTSD relative to other anxiety disorders (Brown & McNiff, 2009). In fact, Taylor, Koch, and McNally (1992) found that AS scores in panic disorder were significantly higher than those of the other anxiety disorders, with the exception of PTSD. Those with preexisting high levels of AS may be more physiologically reactive during a trauma, which may contribute to the peritraumatic distress that has been shown to be a strong predictor of symptoms of PTSD (e.g., Brunet et al., 2001; Ozer, Best, Lipsey, & Weiss, 2003). As in panic disorder, AS may function as a cognitive diathesis that, in interaction with exposure to trauma, confers risk for PTSD. AS may also develop concurrently with PTSD as a direct result of exposure to a traumatic event and in this context amplify negative reactions to the trauma. For example, those high in AS may avoid reminders of a traumatic event, thereby preventing extinction of learned trauma-related fear (Lang et al., 2002). Accordingly, targeting AS may facilitate symptom reduction during treatment of PTSD (Fedoroff, Taylor, Asmundson, & Koch, 2000).

AS has been conceptualized as consisting of three lower-order dimensions (Taylor, 1999): (a) a fear that physiological symptoms of anxiety (e.g., palpitating heart) are physically dangerous (physical concerns), (b) a fear that cognitive symptoms of anxiety (e.g., difficulty concentrating) are precursors to mental illness (cognitive concerns), and (c) a fear that observable anxiety symptoms (e.g., shaking) will result in negative evaluation (social concerns). Recent research supports a bifactor model of AS where a general factor reflects common variance (e.g., general AS factor) and multiple group factors (e.g., physical, social, and cognitive concerns) reflect additional shared variance that cannot be due to the general factor (Ebesutani, McLeish, Luberto, Young, & Maack, 2014; Osman et al., 2010). There is some evidence to suggest that AS dimensions differ in the extent to which they confer risk for PTSD (Zahradnik, Stewart, Marshall, Schell, & Jaycox, 2009). For example, research has shown that the physical (Asmundson & Stapleton, 2008) and psychological (Feldner, Lewis, Leen-Feldner, Schnurr, & Zvolensky, 2006; Lang et al., 2002) dimensions of AS may be the most predictive of PTSD. However, the bifactor model highlights the importance of examining the extent to which a general AS dimension and distinct physical, cognitive, and social concerns dimensions may be differentially related to PTSD-relevant processes (Allan, Albanese, Short, Raines, & Schmidt, 2015).

Delineating the mechanism(s) by which AS and its dimensions confer risk for the development of PTSD may also require consideration of other cognitive factors (Ehlers & Clark, 2000). For example, AS may exert its influence on PTSD symptoms of avoidance and hyperarousal through negative intrusive thoughts about the trauma. Although no study to date has examined the extent to which intrusive thoughts mediate the association between AS and symptoms of PTSD, intrusive thoughts have been implicated in the development of PTSD (Ehlers et al., 2002). Such intrusions generally include thoughts about threat and danger, negative thoughts about the self, and thoughts about the meaning of the event (De Silva & Marks, 1999). Research has also shown that reducing the frequency, vividness, distress, and nowness of intrusions may contribute to effective treatment of PTSD (Hackmann, Ehlers, Speckens, & Clark, 2004). The hypothesis that initial intrusive thoughts may mediate the association between AS and PTSD symptoms is also consistent with existing theoretical models. For example, Horowitz (1982) has observed that the psychological sequelae of trauma can be summarized in two major sequential manifestations: intrusion and avoidance. In this model, intrusion refers to the penetration of consciousness by thoughts, images, feelings, and nightmares about the trauma. This model specifies that intrusions represent the initial phase of the psychological sequelae of trauma that then gives rise to avoidance (Horowitz, 1982). Consistent with this view, research has shown that the experience of intrusions is the more dominant reaction shortly after a traumatic event, which then leads to greater utilization of avoidance responses (Horowitz, Wilner, & Alvarez, 1979). The frequency of intrusive thoughts may increase among individuals with high AS because both the event and subsequent recollections are experienced as more aversive. As a result of the intrusive thoughts, those with PTSD may then engage in maladaptive avoidance, including suppressing intrusive thoughts (Ehlers, Mayou, & Bryant, 1998).

Although the available literature suggests that AS may be an important vulnerability factor in the development of PTSD, the mechanism(s) that may account for this relationship remains unclear. The purpose of the present study is to examine the extent to which intrusive thoughts mediate the association between AS and its dimensions and post-traumatic stress reactions after exposure to a trauma film. The trauma film paradigm has been used extensively in previous research (Holmes, James, Coode-Bate, & Deeprose, 2009; Holmes, James, Kilford, & Deeprose, 2010) and provides a prospective experimental tool for investigating analog peritraumatic mechanisms underlying intrusion development (Weidmann, Conradi, Gröger, Fehm, & Fydrich, 2009). As described by Holmes and Bourne (2008), the trauma film paradigm involves showing participants short films that contain scenes depicting traumatic events. A review of the literature suggests that the trauma film paradigm is effective in inducing intrusions in line with predictions (Holmes & Bourne, 2008).

To enhance causal inference, it is critical to control for as many relevant confounders as is practical. Such relevant confounders should include other mediators that may be related to the proposed mediator (Smits, Julian, Rosenfield, & Powers, 2012). Anxiety sensitive individuals have been hypothesized to be more distressed not only by the trauma, but also by their own reactions to the trauma (Watt & Stewart, 2008). Accordingly, the present study examined trauma-related emotional distress as a rival mediator to intrusive thoughts because both have been implicated in the development of PTSD. This approach allows for the determination of the extent to which intrusive thoughts are related to symptoms of PTSD over and above their relation with trauma-related distress.

The existing literature has highlighted the utility of a diathesis-stress model in understanding the role of AS in the development of PTSD (Bernstein et al., 2005; Elwood, Mott, Williams, Lohr, & Schroeder, 2009b). Research along these lines highlights the importance of examining putative moderators that may influence the relationship between AS and the mechanism(s) by which it confers risk for the development of PTSD. The underlying mechanism of AS, fear of physical sensations, points to physiological arousal as a potential moderator of interest. Indeed, PTSD is robustly associated with physiological reactivity to trauma cues (Pole, 2007). An extensive body of research suggests that AS may contribute to panic through interoceptive conditioning (Bouton, Mineka, & Barlow, 2001). That is, early physiological changes during a panic attack may become signals for more intense and aversive physiological arousal (e.g., a panic attack or intense fear; Craske, 1991) and thus elicit a panic attack on their own. Such learned relations then alter the function of formerly benign physiological events such that they become significant fear-evoking events in their own right. Among those high in AS, heightened physiological reactions during exposure to a traumatic event may result in more negative intrusive thoughts, which then contribute to the development of PTSD symptoms. Accordingly, the present study also examines the extent to which the association between AS and negative intrusive thoughts