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Review

Anxiety sensitivity in the association between posttraumatic stress and substance use disorders: A systematic review

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HIGHLIGHTS

- Anxiety sensitivity (AS) is a malleable cognitive-affective factor.
- AS is related to PTSD and substance use disorders (SUD).
- This systematic review summarized the published literature from 1966 – 5/1/2018.
- The review includes 35 manuscripts relevant to AS, PTSD, and SUD.
- Limitations and future directions are discussed.

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ABSTRACT

Posttraumatic stress disorder (PTSD) and substance use disorders (SUD) are complex psychiatric conditions that commonly co-occur. No evidence-based, "gold standard" treatments for PTSD/SUD comorbidity are currently available. Thus, it is imperative to better understand cognitive-affective mechanisms, targetable via cognitive-behavioral intervention (i.e., malleable), that may be related to both disorders in order to improve the theory and treatment of PTSD/SUD. Anxiety sensitivity is a malleable cognitive-affective factor with relevance to both PTSD and SUD. This systematic review focused on the published literature on anxiety sensitivity and trauma/PTSD and substance use/SUD from 1966 – May 1, 2018, and includes a total of 35 manuscripts. The state of the literature, limitations, and future research directions are discussed.

Posttraumatic stress disorder (PTSD) and substance use disorders (SUD) are complex psychiatric conditions that commonly co-occur (McCauley, Killeen, Gros, Brady, & Back, 2012), presenting an immense challenge to clinical scientists and practitioners. The development of a deeper understanding of this comorbidity is critical, as the co-occurrence of PTSD and SUD presents a severe clinical and public health concern. The comorbidity is complex, difficult-to-treat, and marked by a more costly and chronic clinical course, when compared to either disorder alone (McCauley et al., 2012; Mills, Teesson, Ross, & Peters, 2006; Schäfer & Najavits, 2007). People with PTSD/SUD comorbidity, relative to those with only one of these conditions, manifest worse treatment outcomes, more legal problems, increased risk for experiencing violence, poorer social functioning, more severe physical health problems, and higher rates of suicide attempts (e.g., Back, Brady, Jaanimagi, & Jackson, 2006; Clark, Masson, Delucchi, Hall, & Sees, 2001; McCauley et al., 2012; Mills Teesson, Ross, & Peters, 2006; Mills, Teeson, Ross, & Darke, 2007; Najavits et al., 1998; Ouimette, Brown, & Najavits, 1998; Ouimette, Finney, & Moos, 1999; Read, Brown, & Kahler, 2004). Moreover, PTSD, including subclinical PTSD symptomatology (Norman, Tate, Anderson, & Brown, 2007), is predictive of

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stronger drug cravings (Coffey et al., 2002; Saladin et al., 2003), withdrawal symptoms (Boden et al., 2013), a greater tendency to use substances to alleviate negative mood states (Back, Brady, Jaanimägi, & Jackson, 2006; Chilcoat & Breslau, 1998; Jacobsen, Southwick, & Kosten, 2001), worse response to treatment, and shorter periods of abstinence (Driessen et al., 2008).

A recent meta-analysis (Roberts, Roberts, Jones, & Bisson, 2015) reviewed randomized controlled trials (RCTs) of treatment studies for comorbid PTSD/SUD. The authors included 14 studies examining cognitive behavioral therapies that addressed both PTSD and SUD with over 1400 participants in total. Results showed that treatments that included trauma processing (e.g., exposure therapy) outperformed treatment as usual (TAU) or other control conditions in regard to both PTSD symptoms and substance use at follow-up time points, but dropout rates were higher for trauma processing treatments. These limitations of the current research landscape underscore the need to improve extant interventions and/or develop novel interventions for PTSD/SUD. One avenue toward this end includes advancing our understanding of cognitive-affective mechanisms, targetable via cognitive-behavioral intervention, that may underlie both disorders. Identifying and targeting mechanisms underlying PTSD and SUD in treatment has potential to improve outcomes for this challenging comorbidity.

One cognitive-affective factor, malleable via intervention, with relevance to both PTSD and SUD is anxiety sensitivity (AS), defined as the fear of anxiety and arousal-related sensations (McNally, 2002). A growing body of literature has conceptualized AS as a transdiagnostic risk and maintenance factor for emotional disorders (Olatunji & Wolitzky-Taylor, 2009). The AS construct is comprised of one higher order factor and three specific lower-order factors (S. Taylor et al., 2007). The three lower-order factors represent Physical Concerns (e.g., "*When I feel pain in my chest, I worry that I'm going to have a heart attack*"), Cognitive Concerns (e.g., "*When my thoughts seem to speed up, I worry that I might be going crazy*"), and Social Concerns (e.g., "*I worry that other people will notice my anxiety*"). The AS construct is measured via self-report and several iterations of AS indices have been developed and refined over time (e.g., Anxiety Sensitivity Index [ASI], Reiss, Peterson, Gursky, & McNally, 1986; ASI-Revised, Taylor & Cox, 1998; ASI-3, Taylor et al., 2007). The measures range from 16 to 38 items and all comprised of 5-point Likert-style scale item ratings. Versions of the ASI also have been developed for children and adolescents (i.e., Childhood ASI; Silverman, Fleisig, Rabian, & Peterson, 1991).

Several studies have demonstrated that AS is malleable (i.e., targetable) via intervention (Otto, Reilly-Harrington, & Taylor, 1999; Schmidt et al., 2007; Schmidt, Capron, Raines, & Allan, 2014; Schmidt et al., 2007; Smits, Berry, Tart, & Powers, 2008; Vujanovic, Bernstein, Berenz, & Zvolensky, 2012). Research to date has found that AS reduction interventions, based largely on interoceptive exposure, are efficacious in the prevention and treatment of panic-related psychopathology as well as anxiety symptoms, more generally, with large effect sizes documented in treatment-seeking samples (e.g., Gardenswartz & Craske, 2001; Schmidt, Eggleston, et al., 2007; Schmidt, Mitchell, & Richey, 2008; Smits et al., 2008).

Indeed, extant research indicates that heightened AS is related to PTSD (Keogh, Ayers, & Francis, 2002; Lang, Kennedy, & Stein, 2002). Theoretically, AS amplifies pre-existing states of anxiety in the short-term and increases the risk for anxiety symptoms (e.g., panic disorder) and PTSD in the long-term (Olatunji & Wolitzky-Taylor, 2009; S. Taylor, Koch, McNally, & Crockett, 1992). Studies indicate that AS is concurrently, incrementally, and prospectively associated with increased PTSD symptoms (Bernstein & Zvolensky, 2007; Elwood, Mott, Williams, Lohr, & Schroeder, 2009; Fedroff, Taylor, Asmundson, & Koch, 2000; Feldner, Lewis, Leen-Feldner, Schnurr, & Zvolensky, 2006; G. N. Marshall, Miles, & Stewart, 2010; Stephenson, Valentiner, Kumpula, & Orcutt, 2009). To date, there is no empirically-derived longitudinal evidence that AS mediates the relation between traumatic event exposure and subsequent PTSD symptom development. However, in cross-sectional research, AS has been found to moderate – or exacerbate - the association between number of trauma exposure event types and PTSD symptom severity in community adults (Feldner et al., 2006). In addition, preliminary work suggests clinical promise for the implementation of interoceptive exposure to targeting AS for individuals with PTSD (e.g., Teng et al., 2008; Vujanovic et al., 2012; Wald, 2008; Wald and Taylor, 2008, 2007).

An emerging literature also suggests that AS is related to SUD (DeMartini & Carey, 2011; Leventhal & Zvolensky, 2015), and AS has been most extensively studied with regard to tobacco and alcohol use. For instance, negative reinforcement models of SUD suggest that a greater sensitivity to physiological distress (i.e., high AS) may predispose individuals to catastrophically interpret symptoms of withdrawal, which may lead to continued substance use to avoid the distressing sensations and cognitions. Indeed, AS is positively associated with smoking to reduce negative affect (Farris, Leventhal, Schmidt, & Zvolensky, 2015), and it is predictive of greater positive affect post-smoking (Wong et al., 2013). AS has been associated with more intense nicotine withdrawal symptoms and greater odds of early lapse and relapse (Leventhal & Zvolensky, 2015). Furthermore, AS has been associated with increased incidence of alcohol use disorder (AUD) (Schmidt, Buckner, & Keough, 2007), increased alcohol use (Stewart, Karp, Pihl, and Peterson, 1997), and increased coping-oriented motives for alcohol use (Conrod et al., 1998; R. A. B. DeHaas et al., 2002; R. A. DeHaas et al., 2001; Sherry H. Stewart, Karp, Pihl, & Peterson, 1997), above and beyond severity of anxiety symptoms. Similarly, AS is related to coping-oriented cannabis use (Bonn-Miller, Zvolensky, & Bernstein, 2007; Comeau, Stewart, & Loba, 2001; Farris, Metrik, Bonn-Miller, Kahler, & Zvolensky, 2016; Zvolensky et al., 2009), and AS is indirectly associated with risk for cannabis dependence through coping motives of use (Johnson, Mullin, Marshall, Bonn-Miller, & Zvolensky, 2010). In terms of other SUD, AS has been associated with sedative use in women (Hearon et al., 2011), and it is predictive of treatment dropout among heroin and crack/cocaine users in residential SUD treatment (Lejuez et al., 2008). Furthermore, interventions targeting AS reduction have been found to be efficacious in the prevention and treatment of certain SUD (Castellanos & Conrod, 2006; Watt, Stewart, Birch, & Bernier, 2006; Worden, Genova, & Tolin, 2017; Zvolensky, Yartz, Gregor, Gonzalez, & Bernstein, 2008).

Based upon extant literature, AS may be related to PTSD/SUD comorbidity in at least four key ways. First, AS may mediate the association between PTSD and SUD, such that individuals with PTSD who also have a heightened tendency to fear anxiety-related sensations (e.g., trauma-related hyperarousal) may be at increased risk of developing or maintaining SUD in order to cope with the negative emotional states associated with PTSD (Berenz & Coffey, 2012; Khantzian et al., 1999). Second, AS may predispose individuals with SUD who experience potentially traumatic life events to develop PTSD due to their increased sensitivity to anxiety-related sensations that often accompany substance use (e.g., withdrawal symptoms). Such increased physiological reactivity stemming from chronic substance may combine with elevated AS to confer heightened risk for PTSD in the aftermath of trauma (Jacobsen et al., 2001; Sharkansky, Brief, Peirce, Meehan, & Mannix, 1999; Stewart, Conrod, Samoluk, Pihl, & Dongier, 2000). Third, PTSD and SUD may develop concurrently in the aftermath of exposure to potentially traumatic events due to elevated AS, which may serve as a shared vulnerability for both disorders (Breslau, Davis, Andreski, Peterson, & Schultz, 1997; Cottler et al., 2011; Fassino, Daga, Delsedime, Rogna, & Boggio, 2004; Krueger & Markon, 2006; Wolf et al., 2010). Fourth, AS may predict PTSD/SUD treatment outcome, since heightened AS may increase vulnerability for lapse or relapse to substance use following attempted abstinence (Leventhal & Zvolensky, 2015) and/or maintain PTSD symptoms (via avoidance) as a result of elevated reactivity to internal or external trauma cues due to fear of anxiety-related sensations.

Given the potentially promising role of AS in both PTSD and SUD, it