

Preliminary data on the relationship between anxiety sensitivity and borderline personality disorder: The role of experiential avoidance

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Abstract

Although research on the temperamental vulnerabilities associated with borderline personality disorder (BPD) has focused primarily on the role of impulsive-aggression, affective instability, and emotional vulnerability, growing evidence suggests that anxiety sensitivity (AS) also may increase vulnerability for BPD. This study provides preliminary data on the relationship between AS and BPD, examining whether AS distinguishes outpatients with BPD from outpatients without a personality disorder (non-PD), and whether the relationship between AS and BPD is mediated by experiential avoidance (i.e., attempts to avoid unwanted internal experiences, such as anxiety). Findings indicate that BPD outpatients reported higher levels of AS than non-PD outpatients and AS reliably distinguished between these two groups. Furthermore, the relationship between AS and BPD was mediated by experiential avoidance. Finally, results indicate that AS (and experiential avoidance as a mediator) accounted for a significant amount of additional variance in BPD status above and beyond both negative affect and two well-established temperamental vulnerabilities for BPD (affect intensity/reactivity and impulsivity). Findings suggest the need to further explore the role of AS in the pathogenesis of BPD.

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1. Introduction

Borderline personality disorder (BPD) is associated with severe dysfunction across emotional, behavioral, interpersonal, and cognitive domains (Gunderson, 2001; Linehan, 1993; Skodol et al., 2002). There is a general consensus that BPD is best accounted for by a diathesis-stress model, developing as a result of a combination of temperamental vulnerabilities and distressing childhood experiences (Linehan, 1993; Paris, 1997; Zanarini and Frankenburg, 1997). Although research on the temperamental vulnerabilities associated with BPD has focused primarily on the role of impulsive-aggression (Siever and Davis, 1991; Skodol et al., 2002), affective instability (Siever and Davis, 1991; Siever et al., 2002; Skodol et al., 2002), or emotional vul-

nerability more generally (Linehan, 1993; Livesley et al., 1998; Zanarini and Frankenburg, 1997), other temperamental factors also may increase the risk for BPD.

One such factor is anxiety sensitivity (AS). AS is considered to be a stable individual difference characteristic that involves a propensity for developing beliefs that anxiety-related symptoms will have negative somatic, cognitive, and/or social consequences (Reiss, 1991). Specifically, individuals high in AS are thought to be more likely than those low in AS to regard anxiety-related symptoms (e.g., increased heart rate, shortness of breath) as having the potential for some kind of negative outcome. For example, an individual with low AS may simply interpret her or his heart beating rapidly as a natural (albeit uncomfortable) consequence of experiencing anxiety; however, an individual with high AS may interpret this rapid heart beat as a sign that she or he is at risk for a heart attack, fainting, losing control, or some other negative consequence. This kind

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of interpretation of essentially harmless anxiety sensations tends to further increase anxiety, eventually resulting in a fear of anxiety-related sensations. As such, AS is thought to be a vulnerability factor for the development of anxiety disorders.

Although AS is often described as a *learned* tendency (e.g., McNally, 1990; Schmidt et al., 1997), it has recently been suggested that a cognitive bias toward the evaluation of anxiety-related sensations as aversive or threatening may be an inherited quality (Stein and Rapee, 1999). Providing support for the conceptualization of AS as an inherited temperamental vulnerability, AS has a heritability coefficient of .45 (Stein et al., 1999), which is comparable to the heritability coefficients of more well-established temperamental vulnerabilities for BPD (e.g., affective instability and impulsivity; see Jang et al., 1996). Furthermore, AS is considered to be distinct from trait anxiety (see Lilienfeld, 1999), reflecting an individual's characteristic way of evaluating and responding to the experience of anxiety, rather than the *frequency* or intensity of the anxiety experience per se (Cox et al., 1999). Research supports this distinction. For example, although AS is associated with trait anxiety, studies have consistently demonstrated that AS exhibits incremental validity above and beyond measures of trait anxiety in predicting a variety of anxiety disorder-relevant outcomes, including fearful responding to biological challenge tasks (e.g., hyperventilation or CO₂ inhalation; Rapee and Medoro, 1994; Zvolensky et al., 2001; for a review, see McNally, 1990) and the development of spontaneous panic attacks (Schmidt et al., 1997).

Consistent with the conceptualization of AS as a vulnerability factor for the development of anxiety disorders, cross-sectional studies have found elevated AS among individuals with anxiety disorders compared to normal controls (Cox et al., 1999; Taylor and Cox, 1998; Taylor et al., 1992), and prospective studies have found that heightened AS predicts the development of spontaneous panic attacks (Schmidt et al., 1997, 1999). Moreover, there is growing evidence for the role of AS in the development of psychopathology more broadly, as studies have found heightened AS in other clinical disorders as well (e.g., major depression; see Otto et al., 1995; Taylor et al., 1996).

Although the role of AS in BPD has not been examined extensively, converging evidence suggests its potential relevance to this disorder. For example, patients with BPD have been found to exhibit elevated symptoms of anxiety (e.g., Gunderson and Singer, 1975; Snyder and Pitts, 1988), and to have high rates of co-occurring anxiety disorders (McGlashan et al., 2000; Skodol et al., 1995; Zanarini et al., 1998, 1989; Zimmerman and Mattia, 1999). In addition, the trait of anxiousness has the highest loading on the emotion dysregulation factor of the Dimensional Assessment of Personality Pathology (Livesley and Jackson, *in press*), proposed by Livesley et al. (1998) to be the core aspect of BPD. Moreover, family history data indicate a high level of anxiety disorders in the relatives of BPD pro-

bands (Zanarini et al., 1988). Finally, evidence for a potential relationship between BPD and AS per se is provided by Lilienfeld and Penna (2001), who found a significant positive association between AS and BPD traits within a non-clinical sample.

Given the conceptualization of AS as only a *vulnerability* factor in the development of psychopathology, the relationship between AS and BPD is likely not direct. Research on the role of AS in the development of anxiety disorders speaks to this, as this relationship is thought to be mediated by other factors (Zinbarg et al., 2001; Zvolensky and Forsyth, 2002), such as experiential avoidance (i.e., attempts to avoid unwanted internal experiences, including emotions, thoughts, and bodily sensations; Hayes et al., 1996). That is, researchers have suggested that it is not heightened AS per se that leads to the development of an anxiety disorder, but rather its associated maladaptive attempts to avoid anxiety-related symptoms (motivated by beliefs regarding their negative consequences; see Taylor and Fedoroff, 1999; Zvolensky and Forsyth, 2002). Consistent with this premise, clinical researchers have suggested that experiential avoidance underlies, and contributes to, many forms of psychopathology (Hayes et al., 1996, 1999; for reviews, see Hayes et al., 2004; Salters-Pedneault et al., 2004). Specifically, although efforts to avoid unwanted internal experiences may initially result in reduced distress (thereby negatively reinforcing these avoidance behaviors), the chronic use of experiential avoidance can have paradoxical effects, resulting in increased distress and dysregulation (Hayes et al., 1996). Indeed, research indicates that attempts to avoid internal experiences (e.g., through inhibition, suppression, or concealment) have paradoxical effects, increasing the frequency, severity, and accessibility of these experiences (e.g., Clark et al., 1991, 1993; Lavy and van den Hout, 1990; Roemer and Borkovec, 1994; Wegner and Erber, 1992; Wegner et al., 1993, 1987), as well as physiological arousal in general (e.g., Campbell-Sills et al., 2006; Gross and Levenson, 1993, 1997).

Furthermore, the literature on the relationship between AS and experiential avoidance in the anxiety disorders is consistent with the literature on the relationship between emotional vulnerability and experiential avoidance in BPD, which suggests that a temperamental emotional vulnerability increases the risk for BPD *indirectly*, through its association with the increased use of avoidant emotion regulation strategies (see Linehan, 1993). Consistent with this, there is preliminary evidence for heightened experiential avoidance tendencies in BPD (Bijttebier and Vertommen, 1999; Cheavens et al., 2005; Kruegelbach et al., 1993; Rosenthal et al., 2004; Trull, 1995).

The present study provides preliminary data on the relationship between AS and BPD, examining whether AS distinguishes outpatients with BPD from outpatients without a personality disorder (non-PD), and whether the relationship between AS and BPD is mediated by experiential avoidance. Further, this study examines whether AS predicts BPD status above and beyond both negative affect

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