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Anxiety sensitivity: Prospective prediction of panic attacks and Axis I pathology ☆

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Abstract

Emerging evidence suggests that anxiety sensitivity (AS) predicts subsequent development of anxiety symptoms and panic attacks. However, evidence regarding whether AS serves as a premorbid risk factor for the development of clinical syndromes is lacking. The primary aim of the present study was to determine whether AS acts as a vulnerability factor in the pathogenesis of psychiatric diagnoses. A large nonclinical sample of young adults ($N = 404$) was prospectively followed over two years. The Anxiety Sensitivity Index (ASI: Reiss S, Peterson RA, Gursky DM, McNally RJ. Anxiety sensitivity, anxiety frequency, and the prediction of fearfulness. Behaviour Research and Therapy 1986; 24: 1–8.) and trait anxiety served as predictors. Consistent with prior reports, AS predicted the development of spontaneous panic attacks in those with no history of panic. Importantly, AS was found to predict the incidence of anxiety disorder diagnoses and overall Axis I diagnoses in those with no history of Axis I diagnoses at study entry. These are the first data to provide strong prospective evidence for AS as a risk factor in the development of anxiety disorders.

Keywords: Anxiety sensitivity; Anxiety diagnoses; Panic attacks; Prospective; Longitudinal

A variety of biological and psychological theories of panic have received empirical support (Clark, 1986; Klein, 1993; Wiedemann et al., 1999). One model, derived from expectancy theory (Reiss, 1991) and contemporary psychosocial accounts of anxiety psychopathology, posits that fear of anxiety (i.e., anxiety sensitivity; AS) is important in the development and maintenance of these conditions (Barlow, 2002; Clark, 1986; McNally, 1990). AS is a trait-like cognitive characteristic that predisposes individuals to the development of panic problems (Taylor, 1999); it encompasses fears of physical, mental, and publicly observable experiences (Zinbarg et al., 1997), all of which are believed to amplify preexisting anxiety (Reiss, 1991). For example, "high AS" individuals are theorized to perceive bodily sensations associated with autonomic arousal as a sign of imminent personal harm and, as a result, to experience elevated levels of anxiety and be at increased risk for a panic attack.

Individual differences in AS are hypothesized to emerge from the combined influences of genetic variation along with any number of experiences that ultimately lead to the acquisition of beliefs about the potentially aversive consequences of arousal and anxiety-related states (Reiss and Havercamp, 1998). Research conducted across diverse populations has supported the AS model, providing strong evidence of cross-cultural and developmental specificity in terms of the latent structure and stability of the construct (Chorpita and Daleiden, 2000; Muris et al., 2001; Zinbarg et al., 2001; Zvolensky et al., 2005b; Zvolensky et al., 2001a). AS also is unique from,

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and demonstrates incremental validity to, trait anxiety (Rapee and Medoro, 1994) as well as negative affectivity (Zvolensky et al., 2005a).

Unlike many other cognitive conceptualizations of anxiety, AS is believed to be a dispositional characteristic that may precede the development of clinical anxiety symptoms or diagnoses. Empirical studies provide converging evidence that AS does indeed act as a risk factor for anxiety problems. First, laboratory studies indicate that baseline AS predicts fear responses to bodily sensations (Rabian et al., 1999; Unnewehr et al., 1996; Zvolensky et al., 2001b); these effects are observed above and beyond variance accounted for by trait anxiety (Zinbarg et al., 2001). Second, AS levels are elevated among individuals with anxiety disorders compared to those without anxiety disorder (Kearney et al., 1997; Rabian et al., 1993; Taylor et al., 1992). Third, prospective studies with healthy adults (Schmidt et al., 1997, 1999) and adolescents (Hayward et al., 2000) indicate that AS predicts the future occurrence of anxiety symptoms and panic attacks, even after controlling for trait anxiety. Additionally, AS predicts the maintenance of panic disorder among untreated patients, the prospective emergence of panic attacks among infrequent (nonclinical) panickers, and the emergence of panic among individuals free from a history of panic attacks (Ehlers, 1995), thereby indicating that AS is a predictor of (future) panic-related problems over time. Although the AS literature is not entirely consistent (Struzik et al., 2004), many studies provide evidence for a link between AS and a range of anxiety psychopathology. However, almost no studies to date have shown that AS serves as a predictor of clinical diagnosis.

Although there is a clear linkage between fear of anxiety and anxiety sensations and panic vulnerability, individuals with other types of psychopathology may similarly fear, albeit to a different extent, anxiety symptoms. Not surprisingly, researchers also have documented an association between AS and other forms of psychopathology (Taylor, 2003; Watt and Stewart, 2000). In addition to panic disorder, AS appears to be elevated compared to nonclinical participants, across most of the other anxiety disorders including SAD, GAD, and PTSD (Keogh et al., 2002; Stewart et al., 2000; Taylor et al., 1992). For example, levels of AS in PTSD are equivalent to those observed in panic disorder (Taylor et al., 1992), suggesting that AS may be involved in both types of psychopathology. It also has been suggested that the ASI subscales tapping social evaluation fears and fears of cognitive dyscontrol may contribute to the risk for some of the other anxiety disorders (Cox et al., 1999). Consistent with expectancy theory, extant data suggest that AS may act as a fundamental fear that amplifies risk for many anxiety conditions (Reiss and McNally, 1985).

The relationship between AS and alcohol use disorders (AUD) also has received considerable attention, in part due to the comorbidity between anxiety and this form of drug use (Schneier et al., 1989; Thyer et al., 1986). Of particular relevance, Stewart and colleagues have outlined a specific model pertaining to AS and alcohol use (Stewart et al., 1999). According to this model, high AS persons are more likely to use alcohol for stress response dampening purposes because AS increases the risk of experiencing anxiety-related symptoms and thought processes. Consistent with this model, research suggests that in outpatient substance abusing samples, high AS men are more likely than low AS men to prefer depressants such as alcohol (Norton et al., 1997). Among individuals with panic disorder, AS is associated with increased alcohol consumption and increased belief in the efficacy of alcohol to dampen anxiety (Cox et al., 1993). Studies of undergraduate analogue samples have also found large relationships between AS and drinking behavior (Stewart et al., 1995).

In addition, several reports have documented an association between AS and depression (Otto et al., 1995; Taylor et al., 1996). Evaluation of depressed patients indicated significant elevations in AS when compared to clinical controls, as well as available norms for nonclinical samples (Peterson and Reiss, 1987). One prospective study of AS also suggested a linkage between AS and depression symptoms (Schmidt et al., 1997). In a more refined analysis of these data, it appears that the AS subfactor assessing fear of cognitive dyscontrol may account for the association between AS and depression (Schmidt et al., 1998). Thus, there is accumulating evidence to suggest that AS, or some aspect of the construct, may be a risk factor for depression.

In summary, relationships between AS and a number of different emotional disorders have been documented (Cox et al., 1999; Taylor, 2003; Watt and Stewart, 2000), though this literature is focused most centrally on panic attacks and panic disorder. Yet, the possibility that AS serves as a premorbid risk factor for the genesis of psychiatric diagnoses has not been demonstrated. That is, although cross-sectional studies indicate that AS is elevated across a number of psychiatric diagnoses and prospective studies indicate that heightened AS is predictive of increased anxiety *symptoms* and panic attacks, there has been no definitive demonstration that AS serves as a risk factor for the development of anxiety (or other AS-linked) diagnoses.

Only one study has provided some initial evidence for an association between AS and the subsequent development of anxiety diagnoses. Maller and Reiss (1992) administered the ASI to approximately 50 college students and followed them over time to evaluate future diagnostic status. Findings indicated that, compared to participants with low baseline ASI scores, participants with high ASI scores were at approximately five times greater risk for developing an anxiety disorder during a three-year follow-up period. In addition to the small sample size, however, this study suffered from a major limitation: the presence of current or previous anxiety disorders was not assessed at study entry leaving open the possibility that elevated AS scores predated the onset of the diagnoses documented during the follow-up period.

The current study provides a more definitive evaluation of whether AS acts as a premorbid risk factor for