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Anxiety sensitivity as a predictor of panic attacks

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Abstract

Anxiety sensitivity (AS) is the fear of physical symptoms of anxiety and related sensations believed to have harmful consequences. AS may play a central role in the nature and etiology of panic disorder (PD) and the genesis of panic attacks. We collected Anxiety Sensitivity Index (ASI) scores from PD patients and controls to determine if AS accurately predicts panic. ASIs were completed prior to panic induction using the modified Read rebreathing test in both hypoxic and hyperoxic conditions. Total scores first-order factors, and individual item ASI scores were correlated with panic presence (Spearman correlation) for each of the hypoxic and hyperoxic rebreathing tests for both study populations. Control subjects' data correlated significantly for items 4, 8, and 11 of the ASI for the hyperoxic ($n=9$; $r_S=0.63$, 0.70, and 0.63, respectively) and items 4 and 8 for the hypoxic rebreathing tests ($n=9$; $r_S=0.63$ and 0.70, respectively). Panic patients' data correlated significantly for item 1 of the ASI for hyperoxic tests ($n=8$; $r_S=0.76$) and item 5 for the hypoxic tests ($n=8$; $r_S=0.95$). Total ASI scores or first-order factors (physical, social concerns, and mental incapacitation) scores of either study group did not correlate significantly with panic presence. AS may not be a reliable predictor of panicogenic responses to CO_2-induced panic in either PD or normal control populations. AS may not be an ultimate causal element in eliciting panic attacks.

Keywords: Anxiety sensitivity; Panic attack; Anxiety Sensitivity Index; CO_2-induced panic

1. Introduction

Anxiety sensitivity (AS) is the *fear* of anxiety-related symptoms arising from beliefs that these symptoms have harmful consequences (Reiss, 1991). Unlike trait anxiety, which deals with the proneness to experience general anxiety symptoms, AS is characterized by the *fear* of those symptoms. AS is thought to amplify anxiety responses, thus facilitating fear conditioning of aversive stimuli, and is believed to be a risk factor for the development of anxiety disorders and panic disorder (PD) in particular (Reiss, 1991; Reiss and McNally, 1985; McNally, 1994). AS is thus believed to be integral to the generation of panic.

The AS theory of panic is related to Clark's (1986, 1988) cognitive model of panic, wherein panic is believed to be the result of the catastrophic misinterpretation of uncertain bodily sensations. Panic attacks (PAs) are thought to arise when the person interprets the sensations as being much more dangerous than

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they really are and as being indicative of some impending doom (e.g. heart pounding representing heart attack). Nevertheless, developers of AS theory contrast it with the cognitive model of panic, emphasizing the fact that AS is the *fear* of anxiety/arousal sensations and not the misinterpretation that the sensations are a sign of imminent catastrophe (McNally, 1994).

Although a variety of biological panic-inducing agents have been employed in the laboratory, few studies have examined the construct of AS as an integral component of panic generation. If AS theory is an inherent and independent aspect of panic development (rather than a learned fear of previous panic; Goldstein and Chambless, 1978), then it should be predictive of challenge-provoked (and theoretically unprovoked) panic. Some studies employing challenges in analogue populations attest to this aspect of AS theory (e.g. Holloway and McNally, 1987; Donnel and McNally, 1989; Rapee and Medoro, 1994; McNally and Eke, 1996; Telch et al., 1996; Sturges et al., 1998; Forsyth et al., 1999); others do not (e.g. Koszycki et al., 1993; Aluja et al., 1997; Koszycki and Bradwejn, 2001). More importantly, few studies have assessed this predictive aspect of AS theory in the PD population, with no known studies using panic attack as the dependent measure. This is of particular interest since the behavioural responses (both somatic and psychologic) and sensitivity to any particular biological challenge are marked by considerable interindividual differences. The limited literature using panicogenic agents in PD populations provides mixed support for AS theory, with some studies reporting enhanced post-challenge physiologic (Perna et al., 2003; Shipherd et al., 2001; Rassovsky et al., 2000) and psychologic responsiveness (Rassovsky et al., 2000; Brown et al., 2003), while other work does not support enhanced post-challenge behavioural responsiveness (Koszycki and Bradwejn, 2001; Van Megan et al., 1994; Veltman et al., 1998; Koszycki et al., 1996). Also, AS is reported to account for from 5% (Rapee et al., 1992) to 60% (Rassovsky et al., 2000) of the variance in the affective response to a given challenge. Furthermore, the majority of studies cited use the total score of the Anxiety Sensitivity Index (ASI) as a measure of AS. However, the hierarchical nature of the ASI (Zinbarg et al., 1997; Lilienfeld et al., 1993) suggests a focus on lower level factors of the ASI, with suggestions of item-level analysis of ASI responses, as a more valid test of the AS construct of panic (Zinbarg et al., 1999).

Our goal was twofold: (1) to test the predictive value of AS (as measured by ASI scores) as a marker of laboratory-induced panic in both PD and healthy volunteer (HV) populations using PA as an outcome measure; and (2) to determine the specific item sets of the ASI that are responsible for this predictive value. This was done as part of a larger study (Katzman et al., 2002) examining the pathophysiology of panic using a modified Read rebreathing test (Mohan and Duffin, 1997) to generate hypercapnia (elevated end-tidal CO_2) under either hyperoxic (high partial pressure of oxygen, PO_2) or relatively hypoxic (low PO_2) conditions.

2. Method

Eleven PD patients (mean age of 33.2 years ± 12.6; 5 men, 6 women), with or without agoraphobia (3 non-agoraphobics), and 10 healthy volunteers (HV; mean age 23.7 years ± 2.4; 5 men, 5 women) participated in the study. Of these, eight PD patients and nine HVs returned the AS assessment materials, constituting the sample sizes for analysis. Diagnosis was established by standard psychiatric interview and confirmed by semistructured clinical interview (SCID). All subjects were nonsmokers, free of medication, and in good physical health as part of study inclusion criteria requirements, and all gave written informed consent to study participation.

This study was part of a larger investigation of the respiratory control of panic patients, with protocol and technique details reviewed elsewhere (Katzman et al., 2002). Briefly, subjects underwent a series of three modified Read rebreathing tests: a practice test, a hyperoxic test, and a final hypoxic test. The modified Read rebreathing test has been used previously in this laboratory to induce panic in PD and HV populations (Katzman et al., 2002; Struzik et al., 2002) and uses added computer control of the partial pressure of oxygen (PO_2) in the rebreathing bag to generate hypoxia or hypoxia, superimposed onto the classic Read rebreathing method (Read, 1967) of inducing hypercapnia, which has been shown to reliably induce panic in vulnerable populations (e.g. Lousberg et al.,