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Effects of threat context and cardiac sensitivity on fear responding to a 35% CO_2 challenge: A test of the context-sensitivity panic vulnerability model

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ABSTRACT

The present study tested several predictions of a context-sensitivity panic vulnerability model emphasizing the interaction between threat context and threat sensitivities. Participants without a history of panic ($N = 47$) completed both global and domain-specific panic relevant sensitivity measures and were then randomized to undergo a 35% CO_2 inhalation challenge in the presence or absence of a cardiac defibrillator (threat context). As predicted by the model, cardiac sensitivity (but not trait anxiety or anxiety sensitivity) potentiated the effects of the presence of the defibrillator on CO_2 fear responding. Moreover, as predicted by the model, the observed potentiation effects of cardiac sensitivity on CO_2 fear responding were mediated by participants' threat appraisals connected to the presence of the defibrillator. Theoretical and clinical implications are discussed.

The past two decades have witnessed a proliferation of psychological theories of panic disorder (Barlow, 1988; Beck, Emery, & Greenberg, 1985; Bouton, Mineka, & Barlow, 2001; Chambless & Gracely, 1989; Clark, 1986; McNally, 1990; Wolpe & Rowan, 1988). Fear of anxiety sensations appears as a common thread throughout many of these models. Evidence supporting the link between fear of anxiety sensations and panic comes from several lines of research including (a) descriptive studies showing elevations in anxiety sensitivity (AS) among panic disorder patients (Taylor, Koch, & McNally, 1992; Telch, Jacquin, Smits, & Powers, 2003); (b) laboratory studies demonstrating heightened subjective fear and panic in response to biological challenges such as CO_2 inhalation among panic patients (Gorman et al., 1994; Perna, Barbini, Cocchi, Bertani, & Gasperini, 1995); (c) studies showing that patients undergoing cognitive behavioral treatment for panic display significant reductions in anxiety sensitivity (Penava, Otto, Maki, & Pollack, 1998; Telch et al., 1993); (d) evidence suggesting that panic disorder symptom improvement following CBT is mediated by changes in fear of bodily sensations (Smits, Powers, Cho, & Telch, 2004); and (e) prospective studies suggesting that those scoring high on measures of anxiety sensitivity have a significant increased risk for developing subsequent panic attacks (Maller & Reiss, 1992; Schmidt, Lerew, & Jackson, 1997, 1999), panic symptoms (Grant, Beck, & Davila, 2007), or other anxiety disorders (Schmidt & Zvolensky, 2007; Schmidt, Zvolensky, & Maner, 2006).

Although the evidence linking anxiety sensitivity to panic is substantial, some negative findings have emerged (e.g., Koszycki & Bradwejn, 2001; Struzik, Vermani, Duffin, & Katzman, 2004) and even among the positive findings, anxiety sensitivity explains only a small proportion of the variance in panic attack occurrence. These data highlight the importance of identifying additional causal factors implicated in panic. Research examining contextual factors during biological challenge has provided important data on the psychology of panic. For example, Rapee, Mattick, and Murrell (1986) observed greater anxious responding to 50% CO_2/air challenge among panic disorder participants who received an explanation for their symptoms compared to panic disorder participants who did not receive such explanation. Similarly, panic patients undergoing a CO_2 challenge without a safe person present responded with greater fear, and a greater number of catastrophic

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cognitions than panic patients who did have a safe person present (Carter, Hollon, Carson, & Shelton, 1995). In addition to perceived safety, perceived control has also shown to be predictive of panicking in response to CO_2. Panic disorder patients who were led to believe that they could decrease the concentration of CO_2 during inhalation were significantly less likely to display CO_2-induced panic relative to patients who were not offered this option (Sanderson, Rapee, & Barlow, 1989). However, an attempt to replicate this finding was not successful (Welkowitz, Papp, Martinez, Browne, & Gorman, 1999). Finally, there is some evidence that attentional focus to internal cues moderated the relationship between panic disorder status and fearful responding to CO_2 (Schmidt & Trakowski, 1999).

Taken together, results suggest that psychological variables such as perceived safety, perceived control, and perceived predictability influence fearful responding to biological challenge among panic disorder patients. However, it is not clear from these studies whether these variables function as contextual risk factors for heightened fear responding to challenge or whether they become panicogenic only after the emergence of panic disorder. Experiments manipulating these potential threat-enhancing contextual factors in non-clinical participants provide some evidence to suggest that they increase emotional responding to challenge in participants with no history of panic disorder. For example, the effects of perceived control on non-clinical subjects' emotional response to a 450 mg. Caffeine challenge was examined through an experimental manipulation in which a caffeine antidote pill placebo was made available to half of non-clinical participants with instructions that they could ingest the pill if their caffeine-induced sensations became too uncomfortable (Telch, Silverman, & Schmidt, 1996). Results showed a significant interaction between participants' anxiety sensitivity and their perceived control assignment. Specifically, those high in anxiety sensitivity showed higher fear when they underwent the caffeine challenge without the placebo caffeine antidote (no perceived control); whereas low anxiety sensitive participants were not affected by the perceived control manipulation.

Additional support for the interactive effects of contextual factors and dispositional factors comes from a study manipulating offset control during 20% CO_2 challenge (Zvolensky, Eifert, & Lejuez, 2001). As predicted, only those high in anxiety sensitivity showed heightened fear in the no offset control condition. Finally, using an instructional set manipulation, Telch, Harrington, Smits, and Powers (2005) provided non-clinical participants scoring high or low in anxiety sensitivity with instructions to expect either arousal or relaxation during a single 35% CO_2 inhalation. Consistent with the hypothesis that unexpected arousal would be perceived as more threatening, those receiving relaxation instructions were significantly more likely to panic in response to CO_2 relative to those receiving arousal instructions. However, consistent with earlier findings, this effect was observed only for those high in anxiety sensitivity.

Taken together these findings suggest that person and contextual factors may interact to influence challenge-induced fear responding. Telch (1995) offered a context-sensitivity vulnerability model to account for the often-observed interaction between anxiety sensitivity and contextual factors in laboratory studies of panic provocation. This formulation proposed that contextual factors, which increase either the anticipated threat of the challenge or the perceived threat of the consequent reactions (somatic, cognitive, or affective) during the challenge will result in greater fear responding. Unlike other psychological formulations of panic that emphasize the threatening misinterpretation of bodily sensations (Clark, 1986) or the enduring tendency to perceive anxiety as threatening (McNally, 1990), the current formulation places central importance on the *interaction* of context and dispositional tendencies (sensitivities) in predicting panic. It should be emphasized that this model is not a theoretical formulation of panic disorder but rather an explanatory model for the propensity to experience panic in situations that pose no objective threat to the individual (i.e., false alarms). Given the high prevalence of false alarms across the full spectrum of anxiety disorders as well as the general population, the proposed panic vulnerability model has applicability across a broad range of anxiety disorders and non-clinical cases in which panic reactions occur.

The model makes several specific predictions. First, it proposes that certain dispositional tendencies potentiate the likelihood of experiencing panic in certain contexts. The use of the term potentiate here is meant to imply that the combined effects of the specific sensitivity and the specific context are greater than the sum of the individual contributions of the presence of the sensitivity alone or the context alone. Context is defined here as a stimulus that may be internal as in the case of a somatic cue (e.g., chest tightness), threatening thought (I'm going to lose control), or experienced emotion (e.g., anger); or external (e.g., being in a densely crowded place with a small exit). The threat potentiating dispositional sensitivities can be quite broad as in the case of trait anxiety or anxiety sensitivity or may be more narrow or domain-specific as in the case of cardiac or respiratory sensitivity.

The model also proposes that the strength of the panic potentiation brought about by a specific dispositional sensitivity will be directly related to the conceptual match between the dispositional variable under investigation and the threat-relevant context (context-sensitivity matching hypothesis). Indirect support for this hypothesis comes from biological challenge studies showing that more narrow-band sensitivities such as physical concerns (Carter, Suchday, & Gore, 2001; Zinbarg et al, 2001) or suffocation concerns (Eke & McNally, 1996; McNally & Eke, 1996; Shipherd, Beck, & Ohtake, 1996) outperform anxiety sensitivity in predicting fear response to provocations that elicit a strong physical/respiratory reaction i.e., CO_2 inhalation and voluntary hyperventilation.

A final prediction of the model is that dispositional tendencies such as anxiety sensitivity or rejection sensitivity potentiate the panicogenic effects of certain internal or external contextual cues by increasing the likelihood that an objectively non-threatening context will be perceived as threatening. For example, those displaying high dissociation sensitivity are more likely to perceive the effects of hyperventilation or marijuana ingestion to be threatening and consequently panic. Similarly, possessing high vestibular sensitivity may contribute to panic when confronting a high platform due to an exaggerated concern of losing one's balance.

The aim of the present experiment was to perform a preliminary test of the aforementioned context-sensitivity panic vulnerability theory by experimentally examining the singular and interactive effects of a putative threat-enhancing contextual cue (i.e., presence or absence of a cardiac defibrillator) and several putative threat-enhancing dispositional variables (i.e., trait anxiety, anxiety sensitivity, respiratory sensitivity and cardiac sensitivity) among young adults undergoing a 35% CO_2 challenge.

Our rationale for using a cardiac defibrillator to manipulate threat context was two-fold. First, cardiac concerns are extremely common among panic patients (Sheehan, 1983), hence it seemed appropriate to use a contextual manipulation that was directly related to the threat-relevant concerns of panic attack sufferers. Second, because the presence of a cardiac defibrillator is not inherently threatening, any fear-enhancing effects are more easily attributable to how participants appraise its presence. This was an important feature because one of the major assumptions of the proposed theory is that dispositional factors such as trait anxiety and cardiac sensitivity potentiate the panicogenic effects of