



Responses to false physiological feedback in individuals with panic attacks and elevated anxiety sensitivity

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ARTICLE INFO

Article history:

Received 27 February 2008

Received in revised form 5 June 2008

Accepted 9 June 2008

Keywords:

Panic attacks
Panic disorder risk
Anxiety sensitivity
False feedback
Heart rate

ABSTRACT

Participants with elevated anxiety sensitivity and a history of panic attacks were compared to a low anxiety comparison group with respect to physiological and subjective reactivity to false heart-rate feedback and reactivity to a priming procedure. Whereas accurate heart-rate feedback elicited minimal responses, participants across groups showed significant physiological and subjective responses to false feedback. High risk and low risk participants did not differ in heart-rate responses to false feedback, though panic attack frequency did predict physiological and subjective reactions to false feedback in the high risk group. Self-reported nonspecific anxiety was significantly higher in high risk female participants than in low risk female participants, while males did not differ in general subjective anxiety. However, high risk participants reported more panic-specific symptoms during the false feedback task than low risk participants, regardless of the sex of the participant. Therefore, although the experimental paradigm appeared to trigger nonspecific anxiety in high risk female participants, panic attack symptoms in reaction to the task were specific to risk group, not sex, and consistent with hypotheses. Surprisingly, the priming procedure did not influence physiological or subjective responses to false feedback in either group. These results raise additional questions regarding the process and impact of interoception in individuals with panic attacks, and suggest that false perception of internal changes may contribute to risk for panic disorder when exposed to believable cues.

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Introduction

Advances have been made in the identification of risk factors for panic disorder. It is less understood how these factors relate specifically to the anxiety and fears of bodily sensations that are characteristic of panic disorder. The goal of this study was to examine a sample of individuals with risk factors for panic disorder within the context of emotional reactivity to false physiological feedback.

At least two factors place individuals at risk for panic disorder. The first is a history of panic attacks. In Ehlers' (1995) sample, 15% of infrequent panickers without panic disorder at baseline met criteria by 1-year follow-up, versus only 2% in a control group. In a study of emergency room patients with panic symptoms, 15% of individuals who had experienced at least one panic attack developed panic disorder within the following year (Swinson, Soulios, Cox, & Kuch, 1992). This rate far exceeds the 12-month

population estimates of 2.7% for panic disorder reported in the National Comorbidity Survey (Kessler et al., 2005).

A catastrophic belief system about physical symptoms of anxiety represents a second risk factor. Clark (1986, 1988) attributed panic attacks to interpreting objectively harmless sensations as threatening. Related to Clark's cognitive theory is the construct of *anxiety sensitivity*, or a trait-like tendency to believe that anxiety and associated physical symptoms are harmful (Reiss, Peterson, Gursky, & McNally, 1986). Anxiety sensitivity has been shown to predict initial panic attacks in a group of non-clinical controls (Ehlers, 1995; Li & Zinbarg, 2007; Schmidt, Zvolensky, & Maner, 2006), as well as spontaneous panic attacks and general anxiety in non-clinical individuals in response to the stress of military training, even after partialling the influence of prior history of panic attacks (Schmidt & Lerew, 2002; Schmidt, Lerew, & Jackson, 1999). Younger samples show similar patterns, with anxiety sensitivity predicting panic attacks and agoraphobic avoidance in a longitudinal study of adolescents (Hayward, Killen, Kraemer, & Taylor, 2000; Hayward, Killen, & Taylor, 2003; Hayward & Wilson, 2007), and a 10-year longitudinal study of college-aged samples (Plehn & Peterson, 2002). Reviews of the literature indicate that whereas anxiety sensitivity is a vulnerability to anxiety disorders in general, a more

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specific link exists with panic attacks and individuals with panic disorder (Cox, Endler, & Swinson, 1995; Schmidt et al., 2006).

The mechanisms by which these risk factors predispose individuals to panic disorder remain unclear. One proposed mechanism is enhanced emotional reactivity to physiological sensations associated with panic attacks. In support, several studies indicate that persons with panic disorder are more likely to fear procedures that elicit bodily sensations similar to those experienced during panic attacks than patients with other anxiety disorders (e.g., Perna, Gabriele, Caldirola, & Bellodi, 1995; Rapee, 1986; Rapee, Brown, Antony, & Barlow, 1992) or healthy controls (e.g., Gorman et al., 1994). These include benign cardiovascular, respiratory, and audio-vestibular exercises (Antony, Ledley, Liss, & Swinson, 2006; Jacob, Furman, Clark, & Durrant, 1992), as well as more invasive procedures such as carbon dioxide inhalation (e.g., Gorman et al., 1994; Perna et al., 1995; Rapee, 1986; Rapee et al., 1992).

False feedback studies provide additional evidence by assessing emotional reactivity to the perception of physical sensations rather than their veridical presence. Emotional responding to the mere perception of physical sensations may explain important features of panic disorder, such as the substantial minority (40%) of panic attacks reported to occur in the absence of actual elevations in heart rate (Taylor, 1986; Taylor, Telch, & Havvik, 1983) and the tendency to report arrhythmic heart rate in the absence of actual arrhythmias (Barsky, Cleary, Sarnie, & Ruskin, 1994). Using a false feedback paradigm, Ehlers, Margraf, Roth, Taylor, and Birbaumer (1988) found that panic disorder patients exhibited increased physiological activity and self-reported anxiety when given misleading information that their heart rates were increasing. Furthermore, individuals with panic disorder who suffered regular nocturnal panic attacks exhibited greater subjective and physiological arousal when awakened by signals that they were misled to believe reflected aberrant versus normal physiological activity (Craske, Lang, Tsao, Mystkowski, & Rowe, 2001). These findings highlight the relative power of perceived physiological activity, regardless of the accuracy of the source or cue.

It is unclear whether or not such emotional reactivity to perceived physiological sensations precedes and contributes to the development of panic disorder, or is simply a by-product of the disorder. That is, there has been no evaluation of reactivity to false physiological feedback in at-risk samples. A few studies have demonstrated positive associations between anxiety sensitivity and subjective emotional reactivity to physiological inductions such as carbon dioxide inhalation (Forsyth, Palav, & Duff, 1999), balloon inflation (Messenger & Shean, 1998), and hyperventilation (Rapee & Medoro, 1994; Sturges, Goetsch, Riddley, & Whittal, 1998), though these protocols involved reactivity to actual sensations versus false cues of physiological changes. Thus, the goal of the current study was to evaluate whether those at risk for panic disorder show enhanced emotional reactivity to false physiological feedback. If false cues elicit anxiety in at-risk individuals, then we have additional evidence of the power of fallible interoception in generating actual emotional responses.

At-risk individuals are unlikely to show the same breadth and severity of bodily fears as individuals with panic disorder, as suggested by data showing that subclinical participants and those in remission show lower anxiety sensitivity than patients with panic disorder (Ehlers, 1995). Anxiety-related beliefs about physiological sensations are linked with stronger emotional responses to physiological induction procedures (e.g., Forsyth et al., 1999). Hence, increasing the accessibility of anxiety-related beliefs through priming may enhance the salience of physiological feedback for those at risk (Clare & Colcombe, 2003; Schneider, Unnewehr, Florin, & Margraf, 2002), and therefore enhance their reactivity to false physiological feedback. In priming, activation of an affective concept or representation enhances subsequent

accessibility of thoughts and behaviors associated with that concept (Clare & Colcombe, 2003).

The influence of priming affective concepts using words associated with anxiety and threat (e.g., “panic attack”) has received some support in anxious (Mikulincer, Gillath, & Shaver, 2002; Schneider et al., 2002) and non-clinical samples (e.g., Mathews & MacLeod, 2002). In one study (Schneider et al.), priming led to more panic interpretations of symptom scenarios by children of panic disorder patients than by children of nonanxious parents. Mathews and MacLeod showed that a priming-like scenario could induce anxiety biases in non-clinical participants that then led to greater anxiety in response to a real life stressor (university exam). Thus, a second goal of this study was to evaluate the degree to which priming enhances emotional reactivity to false cues in at-risk samples.

We compared emotional reactivity to false cues of physiological arousal across groups of individuals at high and low risk for panic disorder, defined by anxiety sensitivity and history of panic attacks. We hypothesized that individuals at higher risk for panic disorder would exhibit more physiological and subjective reactivity to false feedback than a low risk comparison group. Also, we hypothesized that reactivity to false feedback would be enhanced by procedures that primed panic-related concepts, particularly in high risk rather than low risk individuals.

Methods

Participants

Participants were screened using two questionnaires. The first screen was the physical subscale of the Anxiety Sensitivity Index (Reiss et al., 1986) that shows greater predictive power for responses to panic-specific stimuli and biological challenge paradigms than the original 16-item ASI (Deacon & Valentiner, 2001; Keogh, Dillon, Georgiou, & Hunt, 2001; Zinbarg, Barlow, & Brown, 1997). Those who scored in the upper 25% were eligible for the high risk group (HRG), as were those scoring in the lower 25% for the low risk group (LRG; Keogh et al., 2001).

Participants were chosen at the upper and lower ends of the ASI distribution to maximize power for our novel false physiological feedback paradigm. Our goal was to define distinct risk groups (based on panic attacks and anxiety sensitivity) versus evaluating anxiety sensitivity as a risk continuum. In addition, an extreme groups' approach reduces the risk of error variance in sampling and increases the power to detect group differences. Thus, we sought to examine only those individuals who clearly endorsed or clearly denied these particular beliefs.

The second screen was a set of questions regarding panic attacks, modified from questions used in other studies of sub-clinical panic attacks (e.g., Brown & Cash, 1990). HRG participants (Ps) responded “yes” to the following two questions: “In the past 12 months, have you experienced a spell or attack when, for no apparent reason (unexpectedly), your heart suddenly began to race, you felt faint, or you couldn't catch your breath?” “In these spells or attacks over the last 12 months, did you also feel frightened, anxious, or very uneasy for no apparent reason”. LRG Ps responded “no” to these questions. During the subsequent diagnostic interview, HRG Ps who did not endorse a history of at least one panic attack (screener false positives) were excluded from the study, as were LRG Ps who endorsed a history of one or more panic attacks during the diagnostic interview (screener false negatives).

In addition, Ps were excluded from this study if they satisfied any of the following conditions: DSM-IV criteria for panic disorder or history of panic disorder; current treatment for any form of anxiety; current or past psychotic symptoms; current use of psychotropic medications; and nonfluency in English. These conditions

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