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

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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 (Pfehn & Peterson, 2002). Reviews of the literature indicate that whereas anxiety sensitivity is a vulnerability to anxiety disorders in general, a more
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, Thee, Taylor, Tutch, & 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, Myskowski, & 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 reaction 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 interception 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 subclinical 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 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
were established through administration of a diagnostic interview, the Mini-International Neuropsychiatric Interview (MINI; Sheehan et al., 1998) conducted by trained research assistants. The MINI is a fully structured diagnostic measure designed for use by trained lay interviewers. Undergraduate research assistants were trained to administer the MINI by a graduate-level clinician with supervision from the second author (MGC). Training involved observed mock assessments with the MINI until the measure was consistently administered and scored according to instructions. This instrument was chosen for its utility within research settings and high inter-rater reliability (Sheehan et al., 1998).

Screening questionnaires were distributed to 1273 undergraduate psychology students over four academic quarters, with 176 meeting initial eligibility criteria for the HRG group, and 258 for the LRG group. Eligible participants were contacted by phone and email; approximately 50% replied. Of these, 101 students, aged 18–26 years, agreed to participate and were available to complete the diagnostic assessment and experiment, which resulted in a final sample size of 97 (high risk n = 44, low risk n = 53).

Design

This study consisted of a 2 x 2 between subjects design, with group consisting of HRG and LRG, and condition including negative priming and neutral priming. HRG and LRG Ps were randomized to priming condition. Dependent variables included heart-rate response to false feedback, skin conductance response to false feedback, subjective anxiety following false feedback, and panic symptoms following false feedback.

Priming

The priming procedure consisted of an adapted sentence unscrambling task based on research in conceptual priming (Bargh & Chartrand, 2000; Bargh, Chen, & Burrows, 1996; Innes-Ker & Niedenthal, 2002). Ps were presented with six words in random order, five of which could be used to construct a grammatically correct sentence, while one word would not fit with the others. Thirty sentences were constructed for the procedure, with 60% (18 sentences) assigned a panic-specific word for the negative priming condition; each panic word (18 total) was used only once. The following words were selected because of their common use in describing panic attacks and feared consequences: breathlessness, dizzy, heart, breathe, dead, weak, died, sweating, nauseous, numbed, fainted, shaking, lightheaded, disoriented, sick, uncontrollable, fear, and choked. In the neutral condition, panic-specific words were removed while attempting to maintain the general context of the sentence (e.g., negative = “Rides make Julie feel dizzy”; neutral = “Julie enjoys riding roller coasters”). Sentences in the negative priming condition that did not contain panic-specific words were replicated in the neutral priming condition. The negative priming procedure was expected to elicit greater responses from Ps during physiological feedback by activating anxiety-based biases about physiological sensations like dizziness and nausea.

Each sentence unscrambling trial was presented on a 17-inch computer monitor using Labview 7.0 software. Ps were asked to form a grammatically correct sentence with five out of six words as quickly as possible by writing this sentence on a numbered form. They were notified by a tone when each item was presented and specifically told not to work on previous items. Each group of words was presented for 20 s.

False feedback

The false feedback task involved cues of increasing heart rate (HR) at specific intervals regardless of actual HR changes. Ps were instructed to hold their breath when they heard the command “hold” and exhale when they heard the command “release”. Following the “release” command, one of three feedback lights illuminated to indicate HR changes relative to the period prior to holding their breath. Ps were told to remain still while the feedback light remained illuminated (10 s) to prevent movement artifacts during the latency period (.5–4 s following feedback). Ps then heard the recorded command “rate”, at which time they marked on a response form whether this change in HR was expected or unexpected in a yes/no format. Each trial was followed by a 20-s rest period before the next “hold” command was given. The breath-holding exercise and ratings of expected change were designed to mask the experimental hypotheses. Over the course of 16 trials, true feedback was interspersed at a ratio of 1:3 (true: false) to increase credibility. Each participant received the same order of true and false feedback (T-F-F-F-F-F-F-F-F-F-F-F-F-F-F). Each experiment started with accurate feedback and then proceeded with relatively equally interspersed true trials throughout the remainder of the experiment, without establishing a clear alternating pattern, such as alternating every three trials. False feedback trials always indicated an increasing HR.

Physiological and subjective measures

HR responses to false feedback were measured using standard trunk electrocardiogram leads connected to a Coulbourn hi-gain bioamplifier (S75-01), inputted to a tachometer (S77-26). Eight-millimeter Ag–AgCl electrodes with Sigma conducting gel were placed below the midline of the right clavicle, below the lowest left rib, and in the center of the Ps forehead (ground) after gently abrading each region with a cleansing gel. Heart-rate responses were measured as the average HR for the pre-stimulus baseline subtracted from the peak heart-rate value within 4500 ms post-feedback on every trial.

Skin conductance was recorded with 8-mm Ag–AgCl electrodes and Unibase (.15 sodium chloride) conducting paste. Electrodes were connected to the thenar and hypothenar eminences of the P’s non-dominant hand. Skin conductance responses (SCR) were measured as the minimum value between 500 ms and 3500 ms post-feedback subtracted from the peak conductance value recorded between 1000 ms and 7000 ms post-feedback, on every trial (Dawson, Schell, & Filion, 2001).

Subjective anxiety was measured in subjective units of distress (SUDS) using a standard analog scale (100 mm line) anchored at not at all distressed to very much distressed (Wolpe, 1958). Four emotions, anxiety, sadness, anger, and fatigue, were assessed prior to and following the false feedback task, though only subjective anxiety was included in analyses. The other three emotional ratings were intended to mask the variable of interest. Ps also recorded symptoms of a panic attack experienced during the false feedback task on the Acute Panic Inventory (API; Dillon, Gorman, Liebowitz, Fyer, & Klein, 1987). The API includes 17-items assessing the symptoms of a panic attack and has been used for self-reported anxiety responses to laboratory protocols (e.g., Antony, 2001). Also, Ps estimated the number of panic attacks experienced over the previous four-weeks using a Panic Attack Frequency Calendar, one of the more reliable methods of retrospective reporting for attacks (PAFC; Nelson & Clum, 2002). This variable was obtained for secondary analyses of predictors of response to the false feedback task.

Procedure

Following mass screening in an undergraduate psychology course, Ps were contacted by email and telephone and informed of their provisional eligibility status, with final eligibility determined...
by a diagnostic interview. All Ps underwent informed consent prior to completing the diagnostic assessment. A research assistant described the project as a study measuring physiological changes in the body following specific cognitive and physical tasks that might or might not influence heart rate. A trained research assistant administered the MINI (Sheehan et al., 1998) to assess for the presence of panic disorder and psychosis as exclusion criteria. Diagnostic status was determined using the criteria outlined for each disorder in the Diagnostic and Statistical Manual for Mental Disorders – 4th Ed. (American Psychiatric Association, 1994). Following the diagnostic assessment, eligible participants in the high and low risk groups were randomly assigned to either the negative priming or neutral priming group.

In the laboratory, trained research assistants attached electrodes to each P's trunk, non-dominant hand, and forehead. Ps were asked to sit quietly for a 10-min acclimation period, during which time the skin conductance and HR equipment were adjusted for optimal data acquisition. A 5-min baseline recording was taken immediately following the acclimation period while Ps read magazines.

Following the baseline recording period, the experimenter described the purpose of the “cognitive challenge” task (i.e., priming) as a way of measuring heart-rate responses to a task that required mental effort. Ps then began the priming task (30 sentence unscrambling trials), with each trial lasting 20 s regardless of completion time or accuracy. After priming, participants completed a SUDS rating of anxiety, sadness, anger, and fatigue.

Next, the experimenter provided instructions for the false feedback task, stating that the purpose was to measure whether or not they noticed changes in their heart rate following a brief breath-holding exercise. Ps were told that the breath-holding exercise may change their heart rate, and a light of different colors would indicate whether their heart rate increased, decreased or remained stable after each exercise. Following each trial, the P was asked to record whether or not this change in heart rate was expected or not expected. Ps were led to believe that all heart-rate feedback was accurate. Each P initially completed three practice trials, during which the research assistant corrected Ps who did not follow protocol or appeared to misunderstand the directions. All breath-holding instructions and HR feedback during the false feedback task were automated and presented with the computer program, LabView 7.0, to minimize experimenter bias and demand characteristics. Following the false feedback task, Ps again rated anxiety, sadness, anger, and fatigue on SUDS scales, and panic attack symptoms on the API. After completing a final questionnaire packet, each P was asked to describe any concerns with procedures and speculate on the hypotheses of the study. No Ps correctly guessed the study hypotheses or questioned the veracity of the heart-rate feedback prior to debriefing.

Analyses

First, physiological and subjective differences between groups (HRG and LRG) and conditions (positive and negative priming) were examined using Analysis of Covariance. Second, panic attack frequency and physical anxiety sensitivity were entered into hierarchical regression models to predict physiological and subjective responses to false feedback in the high risk group only.

Results

Sample characteristics

Demographic data are displayed in Table 1. Ps did not differ in age or education, though there was a trend for a difference in the male-to-female ratios for the HRG (M = 12, F = 32) and LRG (M = 25, F = 28) groups (χ²(1) = 3.41, p = .064). Therefore, subsequent analyses included sex as a covariate or predictor. There were no differences by priming conditions (χ² = .751, p = .356). Table 2 displays descriptive statistics for each dependent variable for the sample.

Heart rate

A mixed Group (2) × Priming (2) × Accuracy (2) ANCOVA yielded a significant within subjects effect for Accuracy (F(1, 91) = 17.79, MSE = 5.77, p = .000, η² = .164), due to false feedback trials eliciting a higher HR response than true feedback trials. Accuracy represented the within subjects variable comparing mean HR response to true versus false feedback trials. There were no main effects for Group (F(1, 91) = .431, MSE = 31.25, p = .513, η² = .005) or Priming (F(1, 80) = .000, MSE = 31.25, p = .990, η² = .000), and no significant interactions between factors. Independent variable effects also did not vary across trials (F(3, 240) = .276, MSE = 29.085, p = .842, η² = .003).¹

Skin conductance

SCR difference scores were log transformed to normalize data. A mixed Group (2) × Priming (2) × Accuracy (2) ANCOVA analyses revealed a trend for a main effect of Group (F(1, 93) = 3.421, MSE = .022, p = .068, η² = .035), where LRG Ps tended to exhibit overall greater skin conductance responses than HRG Ps. There was no main effect for Accuracy (F(1, 93) = .478, MSE = .003, p = .491, η² = .005), or Priming (F(1, 93) = .040, MSE = .022, p = .841, η² = .000), and no significant interaction between factors.

Subjective anxiety

A 2 × 2 × 2 ANCOVA of subjective anxiety (SUDS) following the priming procedure was significant (F(7, 88) = 2.77, p < .05, η² = .18), and revealed a significant main effect for Group (F(1, 88) = 5.965, MSE = 368.48, p < .05, η² = .06), where the HRG participants (M = 25.17, SD = 3.27) reported significantly greater anxiety than the LRG (M = 14.87, SD = 2.87); no interactions were

Table 1

Demographic data for study sample

<table>
<thead>
<tr>
<th></th>
<th>High risk</th>
<th>Low risk</th>
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<tbody>
<tr>
<td></td>
<td>Negative prime</td>
<td>Neutral prime</td>
</tr>
<tr>
<td>Mean age (SD)</td>
<td>18.9 (1.20)</td>
<td>19.1 (1.57)</td>
</tr>
<tr>
<td>Males</td>
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</tr>
<tr>
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<td>African American</td>
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<td>1</td>
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<td>Hawaiian/Pacific Islander</td>
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<td>5</td>
</tr>
<tr>
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<td>0</td>
<td>1</td>
</tr>
<tr>
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</tr>
<tr>
<td>Hispanic/Latino</td>
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</tr>
<tr>
<td>Non-Hispanic/Latino</td>
<td>18</td>
<td>19</td>
</tr>
</tbody>
</table>

¹ Pre-stimulus HR was higher on average in the high risk group than the low risk group, though this difference was only marginally significant (F(3, 92) = 3.16, MSE = 119.48, p = .072, η² = .033). To control for potential effects of restricted variance, peak HR responses were submitted to a range correction by converting each response to a proportion of heart-rate change over range in HR (Lykken, 1972), though groups differences in proportional heart-rate response did not emerge.
significant. Differences in anxiety following the priming procedure were not initially hypothesized, though these results suggest that HRG Ps experienced greater anxiety prior to starting the false feedback task, potentially as a result of the priming protocol or general experimental context.

All participants showed a significant decrease in SUDS between priming and the false feedback task \( F(1, 91) = 11.01, \text{MSE} = 321.631, p = .001, \eta^2 = .108 \), with no interactions between independent variables.

A \( 2 \times 2 \times 2 \) ANCOVA of SUDS to the false feedback task reflected a significant Group by Sex interaction \( F(1, 89) = 5.46, \text{MSE} = 369.11, p = .02, \eta^2 = .058 \). Simple effect analyses showed the difference between HRG women \( (M = 25.43, \text{SD} = 3.22) \) and LRG women \( (M = 8.40, \text{SD} = 3.33) \) to be significant \( F(1, 88) = 13.49, \text{MSE} = 318.76, p = .001, \eta^2 = .194 \). In contrast, HRG males \( (M = 10.75, \text{SD} = 4.99) \) and LRG males \( (M = 11.76, \text{SD} = 3.48) \) did not differ significantly in subjective responses to the false feedback task \( F(1, 87) = .028, \text{MSE} = 298.59, p = .869, \eta^2 = .001 \). However, when anxiety following the priming task was covaried in these analyses, the Group by Sex interaction was no longer significant \( (p = .073, \eta^2 = .04) \).

A \( 2 \times 2 \times 2 \) ANCOVA of API scores revealed a significant main effect for Group \( (F(1, 87) = 27.76, \text{MSE} = 15.11, p < .01, \eta^2 = .24) \), though there were no effects for Priming \( (F(1, 91) = 806, p = .372, \eta^2 = .009) \) or Sex \( (F(1, 87) = 673, p = .414) \), or significant interactions. As expected, the HRG group reported significantly more panic attack symptoms following the false feedback task than the LRG group (see Fig. 1). Also, group differences in panic symptoms remained significant after covarying the effects of anxiety following the priming task \( F(8, 85) = 5.128, \text{MSE} = 14.720, p < .01, \eta^2 = .33 \), indicating that these symptoms did not represent carryover effects from previously reported anxiety.

### Predictors: panic attack history and anxiety sensitivity in high risk participants

Panic attack frequency (PAF), physical anxiety sensitivity (PAS), and sex were entered into a hierarchical linear regression model predicting physiological and subjective responses to each phase of the experiment in the HRG group. PAF significantly predicted heart-rate (HR) responses to false feedback \( R^2 = .131, B = .125, p < .05 \), though neither PAS \( (p = .68) \) nor sex \( (p = .10) \) significantly improved the model. As expected, PAF \( (p = .46) \) and PAS \( (p = .26) \) did not predict HR response to true physiological feedback, though sex was a significant predictor \( R^2 = .143, B = 3.307, p < .05 \). PAF \( (p = .11) \) and sex \( (p = .14) \) did not predict skin conductance response to false feedback, though PAS was significant \( R^2 = .142, B = .013, p < .05 \). PAS also predicted subjective anxiety (SUDS) following the priming task \( R^2 = .156, B = 2.38, p < .01 \), while sex \( (p = .08) \) and PAF \( (p = .98) \) were not significant in this model. PAF \( (B = 1.65) \) and sex \( (B = 13.86) \) predicted subjective anxiety (SUDS) following the false feedback task \( R^2 = .253, p < .01 \), while PAS did not \( (p = .36) \). Finally, PAF predicted self-reported panic symptoms (API total) following the false feedback task \( R^2 = .357, B = .658, p < .01 \), while sex \( (p = .32) \) and PAS \( (p = .51) \) were not significant.

### Discussion

This study showed that high risk participants reported more subjective anxiety following false physiological feedback than low risk participants, though these differences were specific to high and low risk female participants only. Also, the difference in subjective anxiety may have represented a carryover effect from the priming task, which elicited more anxiety in high risk female participants. However, when asked about panic-specific symptoms, high risk participants reported more symptoms in response to false feedback than low risk participants, regardless of sex or subjective anxiety during the priming task. This pattern suggests that false HR feedback elicited subjective experiences of mild panic for high risk participants as anticipated, while the experimental paradigm in general may have triggered more nonspecific anxiety particularly in high risk females.

As secondary findings, panic attack frequency in the high risk group predicted HR responses to false physiological feedback trials, and self-reported anxiety and panic responses to the false feedback task, regardless of sex; physical anxiety sensitivity did not predict

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**Table 2**

<table>
<thead>
<tr>
<th></th>
<th>High risk</th>
<th>Neutral priming</th>
<th>Low risk</th>
<th>Neutral priming</th>
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</thead>
<tbody>
<tr>
<td>Priming response</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Subjective distress</td>
<td>33.57 (24.62)</td>
<td>22.30 (17.75)</td>
<td>15.20 (18.24)</td>
<td>14.29 (15.39)</td>
</tr>
<tr>
<td>False feedback task</td>
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<tr>
<td>Overall HR response</td>
<td>9.21 (5.57)</td>
<td>9.34 (3.79)</td>
<td>8.60 (3.93)</td>
<td>9.13 (3.70)</td>
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<tr>
<td>False HR response</td>
<td>9.43 (5.53)</td>
<td>9.58 (3.93)</td>
<td>9.02 (4.05)</td>
<td>9.64 (3.59)</td>
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<tr>
<td>True SCL</td>
<td>8.61 (4.48)</td>
<td>8.16 (3.30)</td>
<td>7.26 (4.17)</td>
<td>7.35 (6.30)</td>
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<tr>
<td>Overall SCL</td>
<td>.33 (14)</td>
<td>.30 (12)</td>
<td>.34 (8)</td>
<td>.36 (9)</td>
</tr>
<tr>
<td>Subjective distress</td>
<td>23.96 (22.11)</td>
<td>18.80 (16.68)</td>
<td>8.46 (13.77)</td>
<td>10.89 (18.81)</td>
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<td>Panic symptoms</td>
<td>6.23 (4.46)</td>
<td>7.5 (5.84)</td>
<td>2.08 (2.21)</td>
<td>2.22 (2.33)</td>
</tr>
</tbody>
</table>

*Log transformation of SCL responses.*
responses in the high risk group. Also, regardless of risk status, participants exhibited larger average HR in response to false feedback trials than true feedback trials. Interestingly, the priming procedure failed to produce observable differences across all measured variables, though it appeared to elicit subjective anxiety in the high risk group.

Overall, results indicated that individuals with elevated anxiety sensitivity and a history of panic attacks reported more panic symptoms in reaction to false signals of increasing heart rate compared to low risk individuals. In addition, there was a direct relationship between history of panic attacks and the magnitude of both physiological and subjective responses to this false feedback.

Prior research has established that elevations on risk factors for panic disorder (i.e., anxiety sensitivity) are associated with greater emotional reactivity to actual physiological cues (e.g., Rapee & Medoro, 1994). The current findings indicate an association between elevations on risk factors and greater self-reported emotional reactivity to the misperception of physiological cues, and suggest that perceptions of physiological change may be sufficient to elicit symptoms of panic in at-risk individuals. In addition, these results remain consistent with Clark’s (1988) model that cognitive interpretations of physical symptoms play an important role in risk for panic disorder. Although somewhat indirectly, this study helped characterize this process because participants were responding to cues about physiological changes that were not occurring. Therefore, participants’ reactions in the absence of actual physiological triggers suggested that information alone (feedback) is potentially threatening enough to elicit a response in some individuals.

False feedback in general elicited larger HR responses than true HR feedback to the same degree across groups. There may be several explanations for this effect. First, HR responses to false feedback trials may have functioned differently in the high and low risk groups, especially given the heightened anxiety reported by high risk participants. Conceivably, during false feedback trials, the low risk group exhibited a basic “awareness” of incongruity between the feedback and the subjective experience of their HR, such that their increased HR reflected a shift in attention or an orienting response. The high risk group, in contrast, panic symptoms were greater in the high risk than the low risk group regardless of sex, remaining more consistent with panic attacks as well (e.g., Barlow, Brown, & Craske, 1994), thereby possibly illuminating one pathway through which panic attacks increase risk for future panic attacks. As noted above, however, whether this process represents a cognitively mediated or a pre-conscious conditioning process is not clear. Nonetheless, the result is consistent with several studies suggesting that panic frequency may be a strong predictor of developing panic disorder (Ehlers, 1995; Swinson et al., 1992) in subclinical samples.

Previous research has claimed a strong relationship between physical anxiety sensitivity and panic attacks (Deacon & Valentinier, 2001; Keogh et al., 2001) and subjective responses to stressors associated with physiological arousal (e.g., Keogh & Cochrane, 2002). In the current study, physical anxiety sensitivity was unrelated to physical and subjective reactions to false feedback in the high risk group. More recent work has shown that physical anxiety sensitivity does not fully represent cognitive biases in non-clinical samples (Lang & Sarantos, 2004), or that perhaps the ASI is not an adequate measure of this construct (Schmidt, Keough, Timpano, & Richey, in press). It remains to be seen, however, whether more sensitive measures of cognitive biases in non-clinical samples predict physiological and subjective responding to false feedback above and beyond a history of experience with panic attacks.

There were limitations to this study. It is difficult to interpret the lack of effects with respect to skin conductance responses. Discordances between physiological variables are not uncommon, especially given the varying influences that modulate each response system, with skin conductance response a measure of sympathetic arousal, and heart rate a reflection of both sympathetic and parasympathetic activity (Bertelson, Cacioppo, & Quigley, 1993; Öhman, Hamm, & Hugdahl, 2000). The study was also limited by an ineffective priming procedure, despite research and theoretical support for the chosen protocol (e.g., Bargh & Chartrand, 2000). The priming procedure may have been contaminated by performance anxiety due to the time-constraints of the task, particularly in high risk participants. This unintended effect was suggested by higher anxiety ratings in the high risk than the low risk group following the priming task, and this reaction to the task potentially masked effects of the specific content of the primes. Additionally, priming effects may have been diluted unintentionally by the materials that participants read during the acclimation period of the study. It is possible that participants were exposed to several anxiety and panic relevant words since these materials were not carefully selected for the absence of such potential confounds.

The external validity of these findings was also limited by the use of extreme scorers on the physical subscale of the ASI. We used this selection criterion because of the exploratory nature of this study and the novel experimental paradigms. Recruiting across the broader range of scores would likely be more representative of the underlying construct of anxiety sensitivity and should be considered in follow-up research. Finally, risk groups were not balanced by sex, though this confound was controlled in all relevant analyses. While this was a potential methodological limitation, it did result in some interesting findings regarding sex differences with respect to general or nonspecific subjective anxiety, which was experienced primarily by high risk females. In contrast, panic symptoms were greater in the high risk than the low risk group regardless of sex, remaining more consistent with hypotheses that false HR feedback would elicit panic-related responses from those participants.
Finally, we did not formally assess the degree to which participants believed the heart-rate feedback. At the end of the experiment, we asked participants to report any concerns that they had with this study (e.g., that the equipment was not accurate), and we asked them to guess the study hypotheses or purposes of the tasks. No participants indicated that the equipment was inaccurate, and none correctly guessed the hypotheses of the study or the experimental manipulations. While this suggests that participants believed the feedback and masking procedures, it provides less information than a more formal assessment of believability.

Future directions

The current study was exploratory in nature and utilized several novel paradigms. As a result, these results laid the groundwork for future research on cognitive processes involved in the perception of physiological arousal and how misperceptions might be related to the development of panic disorder. At the same time, some aspects of the design signaled areas for methodological improvement.

The selection of an appropriate sample is critical to testing hypotheses of cognitive bias and susceptibility to panic disorder. Our ASI cut-off scores were quite close to the physical anxiety sensitivity mean score of panic disorder (M = 19.65) and nonanxious controls (M = 2.88) in Zinbarg et al.’s (1997) sample of adults presenting to an outpatient clinic. Nevertheless, it may be important for future studies to recruit participants with a broader range of anxiety sensitivity, versus using either end of a distribution. In addition, it may be important to include other measures of anxiety and sensitivity to physical symptoms, such as the Bodily Sensations Questionnaire and the Agoraphobic Cognitions Questionnaire (Chambless, Caputo, Bright, & Gallagher, 1984). These additional tools would allow examination of the specificity of this task to risk for panic disorder versus other forms of anxiety. Moreover, it may be valuable to include a biological challenge task (e.g., CO₂ inhalation) for comparison to false feedback.

With respect to affective priming, our study provided some useful information regarding the challenges to implementing such procedures in non-clinical participants. The use of priming paradigms that involve a speeded component is confounded by the potential anxiety induced by performance expectations, particularly in anxious participants. Our sentence unscrambling task was susceptible to this confound, as are other tasks that could serve a similar priming role (e.g., dot-probe paradigm and the Stroop task). An alternative procedure might involve exposure to priming words in a more casual experimental setting; for example, one could ask participants to read passages from an essay while equipment is calibrated, with priming passages containing panic relevant words in somewhat neutral contexts (e.g., July felt dizzy after the amusement park ride).

Validation of alternative priming methods could advance research in this area, though it may be important to study hypotheses about responses to false feedback and affective priming separately. Validation of the false feedback procedure is a necessary step, and it should be compared to alternative methods of eliciting anxiety with physiological information, such as auditory feedback procedures (e.g., beating heart), visual feedback with indication of actual heart rate, and/or tactile feedback. There may be specific procedural effects that are difficult to assess without such comparisons. Testing whether or not it is possible to activate or enhance cognitive biases through priming might be an important second step in this research, but perhaps it should be withheld until more is understood about susceptibility to false feedback.

The discrepancy between groups in subjective anxiety deserves additional follow-up as well, particularly since both groups showed increased heart rate in response to false feedback. As stated, this difference may reflect an orienting response in the LRG and an anxious response in the HRC. Future studies might examine orienting responses in feedback paradigms that present non-threatening, but inaccurate information to participants. Heart-rate increases in response to non-threatening feedback that was incongruent with expectations would support the hypothesis of an orienting response. Testing this hypothesis in a high risk sample by comparing responses to threatening (e.g., physiological data) and non-threatening feedback (e.g., changing temperature, humidity, and ambient light data in the laboratory environment) might differentiate responses to unexpected information from anxious responses to feared stimuli.

Finally, our findings offer some clinical implications, as they suggest that individuals who suffer panic attacks react to their own beliefs about bodily functioning regardless of accuracy. Very simply, clients may benefit from the understanding that interoceptive accuracy is limited for many people, and it therefore may be worthwhile to question whether or not their perception of physiological cues is accurate. Biofeedback might represent one method of helping clients observe their inaccuracies in perceiving physiologic functioning, as well as training them to increase their accuracy when actual physical symptoms occur.

Conclusions

These results highlight the potential role of perception in the detection and interpretation of physical symptoms. The experience of panic attacks appears to increase at-risk individuals’ sensitivity to cues about their physical functioning, regardless of the veracity of these signals. If the response to these cues includes panic symptoms, then we may be observing a precipitant to future panic attacks. Determining whether this reflects an active appraisal process, a conditioned response, or some interaction between these two processes requires further study.

References
