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Does Anxiety Sensitivity Cause Panic Symptoms? An Experimental Investigation

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Abstract

Anxiety sensitivity (AS) refers to the fear of anxiety-related sensations based on beliefs about their harmful consequences. Despite a wealth of research demonstrating an association between AS and panic-related psychopathology, direct experimental evidence that AS causes panic symptoms, as hypothesized in cognitive models of panic disorder, is lacking. The present study aimed to fill this gap by testing the causal relationship between AS and panic symptoms using a false physiological feedback paradigm. Undergraduate participants without a history of panic underwent a prolonged hyperventilation challenge either with (n = 33) or without (n = 30) receiving false physiological feedback suggesting an impending loss of consciousness. As hypothesized, participants in the false feedback condition had higher peak anxiety, engaged in more avoidance, and had more catastrophic thoughts and higher body vigilance specific to fainting compared to participants in the control condition. Between-group differences on most outcome measures were fully mediated by higher concerns about fainting in the experimental condition. Baseline AS did not potentiate the effects of the experimental manipulation on fear responding. This study provides direct experimental evidence in support of the causal role of AS in panic symptoms as informed by Cox's (1996) interactional model of AS and catastrophic cognitions.

Keywords: anxiety sensitivity, panic, interoceptive exposure, cognitive

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Introduction

Cognitive models of panic (e.g., Beck & Emery, 1985; Clark, 1986) posit that panic attacks are the result of the catastrophic misinterpretation of anxiety-related body sensations. The interpretation of ambiguous body sensations as threatening is hypothesized to increase anxious arousal and intensify feared sensations, leading to further misinterpretation and sensations, and eventually culminating in a panic attack. Individuals who tend to catastrophically misinterpret their anxiety-related symptoms likely have enduring beliefs about the dangerousness of such experiences. Anxiety sensitivity (AS) refers to the fear of anxiety-related sensations based on beliefs about their harmful physical, social or cognitive consequences (Reiss & McNally, 1985). Individuals who believe that certain anxiety-related sensations are dangerous may engage in catastrophic misinterpretation, and subsequently experience panic attacks, when experiencing feared sensations (Cox, 1996). For example, an individual who believes that rapid heart rate will cause a heart attack might misinterpret palpitations induced by physical exercise as signifying an impending catastrophe, thereby triggering the vicious circle described in cognitive conceptualizations of panic. Although theorists have noted that individuals with high AS do not necessarily catastrophically misinterpret anxiety-related symptoms (e.g., McNally, 1990, 2002), cognitive models suggest that individuals who catastrophically misinterpret panic symptoms possess enduring beliefs about the dangerousness of such symptoms.

A large body of research has demonstrated a specific and robust association between AS and panic-related psychopathology (McNally, 2002). Elevated AS is associated with an increased likelihood of experiencing panic attacks, and people who have panic attacks have higher levels of AS (Asmundson & Norton, 1993; Cox, Endler, Norton, & Swinson, 1991; Donnell & McNally, 1990). Individuals with panic disorder exhibit higher AS in general, and greater fears of physical anxiety-related symptoms in particular, than those with other anxiety disorders (Deacon & Abramowitz, 2006; Rector, Szacun-Shimizu, & Leybman, 2007; Taylor, Koch, & McNally, 1992; Zinbarg, Barlow, & Brown, 1997). Moreover, AS decreases after patients with panic disorder are treated with cognitive-

behavioral therapy and reductions in AS are correlated with symptom improvement (McNally & Lorenz, 1987; Otto & Reilly-Harrington, 1999; Penava, Otto, Maki, & Pollack, 1998). Indeed, improvement in panic symptoms following cognitive-behavioral therapy appears to be mediated by the reduction of AS (Smits, Powers, Cho, & Telch, 2004).

Elevated AS reliably predicts fear and panic in reaction to laboratory biological challenge tasks (e.g., carbon dioxide inhalation, hyperventilation) in which anxiety-related body sensations are induced (Donnell & McNally, 1989; Holloway & McNally, 1987; Messenger & Shean, 1998; Rapee, Brown, Antony, & Barlow, 1992). Among the physical, cognitive, and social domains of AS, the fear of physical symptoms best predicts fearful responding to sensation induction procedures (Brown, Smits, Powers, & Telch, 2003; Carter, Suchday, & Gore, 2001; Rapee et al., 1992; Zinbarg, Brown, Barlow, & Rapee, 2001). Prospective studies have demonstrated that elevated AS is a risk factor for the subsequent development of panic attacks (Hayward, Killen, Kraemer, & Taylor, 2000; Maller & Reiss, 1992; Plehn & Peterson, 2002; Schmidt, Lerew, & Jackson, 1997, 1999; Schmidt & Zvolensky, 2007). Further, AS has been shown to predict the course of panic disorder (Ehlers, 1995). For example, Benítez and colleagues (2009) demonstrated that scores on the physical concerns subscale of the Anxiety Sensitivity Index (Reiss, Peterson, Gursky, & McNally, 1986) significantly predicted the duration of panic disorder episodes. Taken together, the results of correlational studies, laboratory challenge paradigms, intervention research, and prospective investigations suggest that the fear of anxiety-related body sensations plays an important role in the psychopathology of panic.

Despite consistent research demonstrating a positive association between AS and panic, the cause and effect relationship between these variables highlighted in cognitive models of panic has been difficult to establish. Correlational research indicating that AS is elevated among panic disorder patients, for example, cannot be used to infer that AS causes panic symptoms. Similarly, although biological challenge and prospective research indicates that high pre-existing levels of AS serve as a vulnerability factor for the development of panic, these quasi-experimental paradigms do not control for third variables that might be associated with high levels of AS and also contribute to panic symptoms, such as learning history (Watt & Stewart, 2000; Watt, Stewart, & Cox, 1998), genetics (Stein, Jang, & Livesley, 1999; Taylor, Jang, Steward, & Stein, 2008), and stressful life events (McLaughlin & Hatzenbuehler, 2009). This limitation also applies to panic challenge studies in which individuals with high AS and/or panic attacks receive false physiological feedback (Ehlers, Margraf, Roth, Taylor, & Birbaumer, 1988; Sanderson, Rapee & Barlow,1989; Story & Craske, 2008).

An additional challenge in establishing the causal relationship between appraisals of body sensations and panic concerns the rapid and possibly unconscious nature of catastrophic cognitions. Clark (1988) suggested that “catastrophic misinterpretations may be so fast and automatic that patients may not always be aware of the interpretive process” (p. 76). The notion of unconscious misinterpretation poses problems for the testability of the cognitive account (McNally, 1999) and creates a challenge for researchers seeking to tease apart the temporal and causal relationship between the misinterpretation of panic symptoms and the experience of panic itself. Studies that manipulate concerns about physical catastrophe prior to a panic challenge task are useful in testing the prediction, derived from cognitive accounts of panic (e.g., Clark, 1986), that the catastrophic misinterpretation of arousal-related body sensations causes the subsequent development of panic symptoms. Further, the experimental manipulation of beliefs about the dangerousness of arousal-related body sensations in nonclinical, panic-naïve sample avoids the potential confounds introduced by quasi-experimental approaches.

Salkovskis and Clark (1990) evaluated the cognitive model of panic by experimentally manipulating participants’ interpretations of body sensations induced by hyperventilation. This study provided preliminary evidence supporting the cognitive model of panic by demonstrating that the experimentally-induced misinterpretation of arousal-related body sensations increases emotional reactivity to panic cues. Yet, there are a number of limitations of this study, including use of a relatively benign manipulation (i.e., instructional set prior to hyperventilation as opposed to direct, alarming feedback during hyperventilation), and failure to assess panic-related cognitions, attentional biases, and safety behaviors. The present study was conducted to build upon previous research by utilizing an intensive manipulation and assessing a broad array of panic-related outcomes.

Multiple investigations have demonstrated that dispositional factors (e.g., AS) and experimental context (e.g., perception of control, increased threat) interact to predict fearful responses to panic challenge tasks (e.g.,

Sanderson et al., 1989; Telch, Harrington, Smits & Powers, 2011; Telch et al., 2010). Telch and colleagues (2010) postulated a context-sensitivity panic vulnerability model wherein the features of the context interact with an individual's predisposition and subsequently contribute to greater panic responding. In support of this model, Telch et al. (2010) found that individuals with higher fears of cardiac symptoms reported greater fear in response to a carbon dioxide inhalation challenge when a defibrillator was present (i.e., threat context), whereas fear of cardiac symptoms was unrelated to fearful responding in the absence of the defibrillator. Accordingly, the present study examined whether the interaction between baseline AS and experimental context predicted increased panic-related symptoms, as predicted by the context-sensitivity panic model.

The aim of the present study was to examine the causal relationship between AS and panic symptoms posited by Cox's interactional model (1996). This model hypothesizes that the interaction between pre-existing beliefs regarding the dangerousness of physical sensations and a congruent somatic trigger produces in-situation catastrophic cognitions which cause panic symptoms. A false physiological feedback paradigm was utilized to induce the belief among participants in the experimental condition that the body sensations associated with prolonged, voluntary hyperventilation (e.g., dizziness, shortness of breath, heart palpitations) are likely to result in a loss of consciousness. This belief manipulation served as an experimental analogue of somatically-focused anxiety sensitivity. The effects of experimentally-induced anxiety sensitivity were examined by subjecting participants to a second hyperventilation task intended to trigger in-situation catastrophic concerns about fainting. It was hypothesized that relative to a control group who did not receive false physiological feedback, participants with experimentally-induced anxiety sensitivity would evidence significantly higher anxiety symptoms, more avoidance and utilization of safety behaviors, more catastrophic cognitions, and greater hypervigilance to bodily sensations during the second hyperventilation task. Further, it was predicted that experimentally-induced catastrophic cognitions regarding fainting during the second hyperventilation task would mediate between-group differences in these outcomes. Finally, consistent with the context-sensitivity panic vulnerability model, it was hypothesized that the interaction of baseline AS and experimental condition would predict anxious responding to the second hyperventilation challenge. Specifically, it was predicted that AS specific to physical sensations would potentiate fearful responding in the experimental condition.

Method

Participants

Undergraduate students enrolled in psychology courses (N = 391) completed a web-based mass testing questionnaire packet for course credit which included a medical screener assessing the presence of seizures, hypertension, diabetes, heart problems, current pregnancy, any other condition that may be exacerbated by exercise, and a history of panic attacks. Individuals (n = 271) who had not experienced any of these conditions were invited to participate via e-mail. An a priori power analysis was conducted using G*Power Version 3.1.3 (Faul, Erdfelder, Lang, & Buchner, 2007). It was determined that a minimum sample of 42 participants would be needed to detect, with 95 percent confidence, a large effect size (i.e., d = 0.8) with alpha set at .05 (two-tailed). Thus, consistent with previous experimental designs (e.g., Salkovskis & Clark, 1990; Wild, Clark, Ehlers & McManus, 2008), approximately 30 individuals were estimated for each condition. The final sample consisted of 63 participants (68.3% women), with 33 in the experimental condition and 30 in the control condition. Participants ranged in age from 18 to 41, with a mean of 20.4 years old (SD = 3.29). The majority of participants were European American (92.1%).

Experimental Design and Manipulation

Design.

Eligible participants were randomly assigned to the control condition or the experimental condition. All participants completed a series of three voluntary hyperventilation phases during which participants were instructed to breathe deeply at a rate of 3 breaths per 4 seconds, which was paced to a voice recording instructing when to breathe in and out. The first two phases of hyperventilation were 5 minutes long and composed of 5 individual hyperventilation

trials. Each individual trial was comprised of 60 seconds of hyperventilation and then a 15 second break. Immediately following the first 5-minute phase, participants in the experimental condition were provided with false physiological feedback suggestive of an impending loss of consciousness; control participants were informed that the biofeedback equipment had malfunctioned. Next, all participants engaged in a second 5 minute phase of hyperventilation. Lastly, the third phase of hyperventilation was conducted for the purposes of testing participants' behavioral avoidance by allowing them to determine when they should stop the continuous deep breathing task. Measures were collected at baseline, immediately following the manipulation, during the second hyperventilation phase, and immediately following the second hyperventilation phase. This research was reviewed and approved by the University of Wyoming institutional review board.

Manipulation.

All participants completed the hyperventilation phases while purportedly hooked up to biofeedback equipment. Specifically, two electrodes were placed on the back of the participant's neck. The equipment used to deliver false feedback consisted of two biofeedback control boxes, two electrodes with adhesive pads, conductivity gel, speakers, and a computer with a monitor facing away from (and not visible to) the participant. The electrodes were connected to the control boxes, which appeared to be connected to the computer. The conductivity gel was applied to the electrodes, and then electrodes were placed on the back of participants' necks. As per Wild et al. (2008), the experimenter informed the participant that the electrodes were placed over a blood vessel (the vertebral artery) in the back of the neck and would measure changes in heart rate and blood pressure. After the participant was connected, the experimenter examined the computer and informed the participant that the program was properly working.

After the first phase of hyperventilation, a computer alarm emitted a beeping sound, and participants were told to stop. Participants in the experimental condition were told with urgency and marked concern that they needed to stop immediately and sit down because an increase in their heart rate (198) and a drop in their blood pressure (60/40) indicated that they were at high risk of fainting. Participants in the control condition were told that there was a slight malfunction with the equipment and the computer needed to be restarted.

Measures

Modified Panic History Form (PHF).

The PHF (Schmidt, Lerew, & Trakowski, 1997) is a four-item measure that assesses history of spontaneous panic attacks, panic attack frequency, panic-related worry, and general history of psychological and psychiatric treatment. The PHF reliably detects a history of spontaneous panic in nonclinical samples (Schmidt & Telch, 1994; Telch, Silverman, & Schmidt, 1996). In this study, participants were considered to have a history of panic attacks and were deemed ineligible for the study if they answered "Yes" to the item "Have you ever experienced a sudden and intense surge of fear or anxiety [e.g., a panic attack] in a situation for no apparent reason?"

Anxiety Sensitivity Index-3 (ASI-3).

The ASI-3 (Taylor et al., 2007) measures fear of anxious arousal on a 0 (*very little*) to 4 (*very much*) scale. This 18 item questionnaire contains three subscales: physical concerns (e.g., "It scares me when my heart beats rapidly"), cognitive concerns (e.g., "When my thoughts seem to speed up, I worry that I might be going crazy"), and social concerns (e.g., "It scares me when I blush in front of other people"). Each subscale has been shown to possess good internal consistency in past research, and the ASI-3 has demonstrated excellent convergent, discriminant and criterion-related validity (Taylor et al., 2007). ASI-3 total scores were computed by summing responses to each item. The physical concerns, cognitive concerns and social concerns subscale scores were computed by summing the responses for the items that correspond with each subscale. In this study, internal consistency was adequate (total α = .87; physical concerns α = .78; cognitive concerns α = .84; social concerns α = .75). The ASI-3 was administered to compare baseline differences in AS between participants in the two conditions.

Modified Agoraphobic Cognitions Questionnaire (ACQ).

This scale measures the frequency of 14 different catastrophic thoughts such as “I am going to pass out” and “I will have a heart attack” (Chambless, Caputo, Bright, & Gallagher, 1984). Each item is measured on a 1 (*though never occurs*) to 5 (*thought always occurs*) scale. Chambless et al. (1984) reported that the ACQ has adequate test-retest reliability (r = .86) and internal consistency (α = .80). Following Carter, Hollon, Carson, and Shelton (1995), the instructions for the ACQ were modified so that participants indicated how often they experienced each thought “during the previous exercise.” Modified ACQ total scores were computed by summing responses to each item.

Modified Body Vigilance Scale (BVS).

A modified version of the BVS (Schmidt, Lerew, & Trakowski, 1997) was used to measure self-reported attention to panic-related bodily sensations. In its original form, the measure consists of four items. Three items assess the degree of attentional focus, perceived sensitivity to changes in bodily sensations, and the average amount of time spent attending to bodily sensations. The fourth item measures the extent to which the respondent reports attending to 15 panic-related bodily sensations (e.g., heart palpitations) that include all the DSM-IV (American Psychiatric Association, 1994) physical symptoms of panic attacks. The BVS has demonstrated good internal consistency and adequate test–retest reliability (Olatunji, Deacon, Abramowitz, & Valentiner, 2007; Schmidt, Lerew, & Trakowski, 1997). In the present study, the participants were given a modified version of the fourth item of the BVS, which involved rating the extent to which they attended to the 15 panic-related body sensations on a 0 (*none*) to 10 (*extreme*) scale during the hyperventilation task. Modified BVS total scores were computed by averaging responses to each item.

Hyperventilation Questionnaire (HQ).

The HQ (Rapee & Medoro, 1994) is comprised of three subscales that measure somatic, affective, and cognitive responses to interoceptive symptom induction exercises. The HQ is a 33-item measure with 20 items assessing somatic responses (e.g., "breathlessness"), 7 items assessing affective responses (e.g., "fear"), and 6 items assessing cognitive responses (e.g., "feeling of losing control"). In the present study, participants were asked to rate the extent to which they experienced each of the 33 descriptors on a 4-point scale ranging from 0 (“not at all”) to 3 (“markedly”). HQ scores were computed by summing responses to each item. In this study, the HQ was administered at baseline, and immediately following the first and second phases of hyperventilation. Internal consistency was good to excellent at all time points (αs = .81, .95, and .94, respectively). Due to a photocopier error that was not discovered until late in data collection, only 19 items were included in the HQ administered following the second hyperventilation phase.

Peak Anxiety (SUDs).

Participants rated their peak anxiety following the first and second phases of hyperventilation on a 0-100 scale where 0 = *no anxiety* and 100 = *extreme anxiety or panic*.

Behavioral Approach Task (BAT).

To measure avoidance of arousal-related sensations, participants were asked to hyperventilate for as long as possible. Participants breathed along with a voice recording that instructed them when to breathe in and out at a rate of 45 breaths per minute. The length of time participants hyperventilated was measured in seconds; individuals still engaged in the task after 5 minutes were stopped by the experimenter.

Safety Behaviors.

Safety behaviors are actions intended to prevent a feared outcome. They are hypothesized to maintain anxiety disorders by preventing the disconfirmation of maladaptive threat beliefs (Salkovskis, 1991). During the second hyperventilation phase, participants were given the option of engaging in two safety behaviors if they felt like they must: (a) leaning on a chair while hyperventilating, and (b) sitting down or leaning on the chair during the rest

period. The experimenter recorded whether each behavior was used, and if so, during which trials. A total safety behavior utilization score was computed by summing the number of trials during which the participant utilized one of these safety behaviors.

Procedure

Eligible students were invited to participate in a project described as “exploring the biological and psychological effects of over breathing.” Participants signed up for an individual time slot with a graduate level experimenter, and were randomly assigned a priori to the experimental condition or the control condition using a computer-generated randomization list (www.random.org). Written informed consent was obtained, and participants completed the baseline measures (demographics, HQ, and ASI-3). The biofeedback equipment was set up, as previously described, and the experimenter demonstrated hyperventilation by breathing deeply and rapidly with the voice recording. Next, participants underwent the first phase of hyperventilation, which concluded with the false feedback from the experimenter. At this point, participants were given a five minute rest period wherein they completed the HQ and reported peak anxiety during the task. Then, the experimenter described the safety behavior options to the participant, and then the second phase of hyperventilation began. After the hyperventilation, participants completed the panic response measures (peak anxiety, HQ, ACQ, and BVS) and the BAT. Finally, all participants were given an extensive debriefing regarding the deception and the harmless nature of the body sensations elicited by hyperventilation.

Results

Baseline Equivalence

Participants in the experimental and control conditions did not differ significantly with respect to age, $t(61) = 1.16$, $p = .25$, or sex, $\chi^2(1) = 0.08$, $p = .79$. To confirm that the experimental groups were comparable regarding baseline levels of state anxiety and AS, a series of *t*-tests were conducted examining between-group differences on the HQ, ASI-R respiratory scale, and ASI-3. As shown in Table 1, none of these tests were significant (all *p*s ≥ .20), indicating that the randomization procedure was successful.

Table 1: Baseline Measures of State Anxiety and Anxiety Sensitivity

Measure	Condition					
	Control (*n* = 30)		Experimental (*n* = 33)			
	M	***SD***	***M***	***SD***	***t***	***p***
HQ	4.20	3.05	5.48	4.59	1.29	.20
ASI-3 Total	10.77	7.91	11.67	7.76	0.45	.65
ASI-3 Physical	2.80	2.77	3.72	3.34	1.19	.24
ASI-3 Social	6.76	4.09	6.42	3.82	0.34	.73
ASI-3 Cognitive	1.20	2.07	1.51	2.41	0.55	.58

Note. HQ = Hyperventilation Questionnaire; ASI-R Respiratory = Anxiety Sensitivity Index-Revised, Fear of Respiratory Symptoms Score; ASI-3 = Anxiety Sensitivity Index-3; Physical = Physical Concerns Subscale; Social = Social Concerns Subscale; Cognitive = Cognitive Concerns Subscale.

Manipulation Check

To verify that the false physiological feedback in the experimental condition successfully induced the fear of fainting, a *t*-test was conducted to compare between-group differences on HQ item #27 ("feel like passing out"). Importantly, this item was rated immediately following the first hyperventilation phase and feedback, and specifically assessed the concern that was being manipulated. As expected, the experimental condition evidenced

significantly greater endorsement of this item (M = 1.18, SD = 1.04) than the control condition (M = .26, SD= .45), $t(44) = 4.58$, $p < .001$, $d = 1.15$.

Effects of Manipulation on Panic Symptoms

A series of t-tests were conducted to evaluate the hypotheses that compared to the control condition, the experimental condition would evidence higher scores on measures of peak anxiety and anxiety symptoms, behavioral avoidance, safety behavior utilization, catastrophic cognitions, and hypervigilance to body sensations. The results are summarized in Table 2.

Table 2: Between-Group Differences on Outcome Measures

	Condition						
	Control (n = 30)		Experimental (n = 33)				
Measure	***M***	***SD***	***M***	***SD***	***t***	***p***	***d***
Peak anxiety	27.53	22.07	42.27	26.64	2.40	.01	.62
ACQ	4.97	6.64	6.12	5.11	.77	.44	.19
ACQ “Pass out”	.73	.94	1.42	1.17	2.56	.01	.65
BVS	2.08	1.65	2.83	2.03	1.62	.11	.41
BVS “Faintness”	2.87	2.70	5.15	3.39	2.97	.004	.75
BVS “Dizziness”	3.37	2.67	5.51	3.40	2.80	.007	.70
BAT Time	248.50	63.25	194.09	65.08	3.36	.001	.85
HQ	10.40	8.53	17.09	12.24	2.53	.01	.63

Note. ACQ = Modified Agoraphobic Cognitions Questionnaire; BVS = Modified Body Vigilance Scale; BAT Time = duration of Behavioral Avoidance Task measured in seconds; HQ = Hyperventilation Questionnaire.

As predicted, compared to participants in the control condition, those in the experimental condition evidenced significantly higher peak anxiety, $t(60) = 2.40$, $p = .01$, $d = .62$, and more anxiety symptoms on the HQ, $t(57) = 2.53$, $p = .01$, $d = .63$, immediately following the second hyperventilation phase. Behavioral avoidance during the BAT was also significantly higher in the experimental condition, $t(61) = 3.36$, $p = .001$, $d = .85$. Only 11 participants in the study utilized one or more safety behaviors during the second hyperventilation phase, 8 of whom were in the experimental condition. Safety behaviors were used by 10.0% of participants in the control condition and 24.2% in the experimental condition ($p = .19$, Fisher’s Exact Test).

Contrary to prediction, participants in the experimental condition did not score significantly higher than control participants on the BVS, $t(60) = 1.62$, $p = .11$, $d = .40$, or the ACQ, $t(61) = .77$, $p = .44$, $d = .19$. However, total scores on these measures involve the aggregation of items assessing a broad range of anxious concerns, and the false physiological feedback in the present study was intended to elicit highly specific concerns about fainting. Accordingly, we examined between-group differences on the individual ACQ and BVS items most pertinent to the manipulation. On the ACQ item “I am going to pass out,” scores were significantly higher in the experimental condition, $t(61) = 2.56$, $p = .01$, $d = .65$. Similarly, the experimental condition scored significantly higher than the control condition on the BVS items assessing vigilance to “faintness”, $t(60) = 2.97$, $p = .005$, $d = .75$, and “dizziness,” $t(60) = 2.80$, $p = .007$, $d = .70$.

Mediation of Cognitive Bias on Outcome Variables

To further evaluate the effect of the manipulation on the outcome measures, mediational analyses were conducted. The purpose of these analyses was to evaluate the hypothesis that between-groups differences in anxious responding would be mediated by the belief that the body sensations associated with hyperventilation might cause a loss of consciousness. SPSS macro and procedures for testing mediation (Preacher & Hayes, 2004; Preacher, Rucker & Hayes, 2007) were used to determine whether scores on the HQ item “feel like passing out,” assessed immediately following the false physiological feedback manipulation, mediated the observed between-group

differences on outcome variables (peak anxiety, HQ scores, BAT avoidance, and the BVS "faintness" and "dizziness" items) assessed following the second hyperventilation phase. The unique effect of condition was represented by a dummy code. The same regression model and mediational analyses were conducted for each outcome variable.

The difference between conditions accounted for significant variance in peak anxiety, β = 14.74, *SE* = 6.20, *p* = .02; however, after the HQ item related to fainting was entered into the model, this path became non-significant, β = 5.46, *SE* = 6.77, *p* = .42. The total indirect path from the difference between conditions to peak anxiety via the HQ item was significant, β = 9.27, *SE* = 3.89, *p* = .02. Thus, significant, full mediation was demonstrated. The overall regression accounted for 18.9% of the variance for peak anxiety, $F(2, 60) = 6.98$, *p* = .002.

A similar mediational analysis was conducted on HQ scores. Condition accounted for significant variance in HQ scores, β = 6.69, *SE* = 2.68, *p* = .02. However, after the HQ fainting item was entered into the model, this path was non-significant, $\beta = -1.82$, *SE* = 2.17, *p* = .41. The total indirect path from condition to HQ scores via the HQ item was significant, β = 8.51, *SE* = 2.18, *p* < .001. Significant, full mediation was demonstrated, and the overall regression accounted for 55.7% of the variance in HQ scores, $F(2, 60) = 37.63$, *p* < .001.

For the ACQ item "I am going to pass out," condition accounted for significant variance in item scores, β = 0.69, *SE* = 0.27, *p* = .01. However, after the HQ fainting item was entered into the model, this path was non-significant, β = 0.05, *SE* = 0.26, *p* = .85. The total indirect path from condition to the ACQ "I am going to pass out" item via the HQ item was significant, β = 0.64, *SE* = 0.19, *p* < .001. Significant, full mediation was demonstrated, and the overall regression accounted for 35.6% of the variance in ACQ item scores, $F(2, 60) = 16.56$, *p* < .001.

The next two mediational analyses concerned the observed between-group differences in body vigilance to symptoms associated with fainting. The difference between conditions accounted for significant variance in body vigilance to "faintness," β = 2.28, *SE* = .78, *p* = .005. After the HQ item was entered into the model, this path became non-significant, $\beta = -.12$, *SE* = 0.65, *p* = .85. The total indirect path from condition to BVS "faintness" via the HQ fainting item was significant, β = 2.40, *SE* = 0.62, *p* < .001. Hence, significant, full mediation was demonstrated. The overall regression accounted for 55.0% of the variance in BVS "faintness," $F(2, 60) = 36.60$, *p* < .001. The same mediational model was evaluated for BVS "dizziness." Here, condition accounted for significant variance in body vigilance to dizziness, β = 2.15, *SE* = .78, *p* = .007; however, after the HQ item was entered into the model, this path became non-significant, β = 0.22, *SE* = 0.75, *p* = .77. The total indirect path from condition to BVS "dizziness" via the HQ item was significant, β = 1.93, *SE* = 0.56, *p* < .001. Thus, significant, full mediation was demonstrated. The overall regression accounted for 39.1% of the variance in BVS "dizziness," $F(2, 60) = 19.23$, *p* < .001.

In the final mediational model, condition accounted for significant variance in BAT time (i.e., avoidance), $\beta = -54.41$, *SE* = 16.20, *p* = .001. However, the mediator did not significantly affect BAT time, $\beta = -7.68$, *SE* = 10.09, *p* = .45, and the direct path between condition and BAT avoidance remained significant after entering the HQ fainting item in the model, $\beta = -47.37$, *SE* = 18.70, *p* = .01. Thus, concerns about fainting following the manipulation did not mediate the observed between-group difference in BAT avoidance.

Evaluation of the Context-Sensitivity Panic Vulnerability Hypothesis

A series of hierarchical regressions was conducted to test the hypothesis that the interaction between baseline AS specific to physical sensations and experimental condition would significantly predict fearful responses to the manipulation. The ASI-3 physical concerns subscale was centered to reduce potential multicollinearity (Cohen, Cohen, West, & Aiken, 1983). In each analysis, the effects of experimental condition (represented by a dummy code) and centered ASI-3 physical concerns subscale scores were entered into Step 1. The interaction of these variables was entered in Step 2. Separate analyses were conducted for peak anxiety, BAT time, ACQ: "Pass Out", BVS: "Faintness" and "Dizziness," and HQ scores. Post-hoc probing analyses for significant moderational effects (Holmbeck, 2002) were conducted to examine the extent to which physical AS potentiated fearful responding in the experimental condition as predicted by the context-sensitivity panic vulnerability model.

In the regression predicting peak anxiety, experimental condition and the ASI-3 physical concerns subscale predicted 15.8% of the variance, $F(2, 60) = 5.63$, $p = .006$. Experimental condition and physical AS were both significant, unique predictors in the first step. In Step 2, the interaction of experimental condition and physical AS explained significant additional variance in peak anxiety, R^2 change = .06, $F(1, 59) = 5.39$, $p = .04$. Post-hoc probing revealed that physical AS significantly predicted peak anxiety in the control condition ($\beta = .58$, $t = 3.08$, $p = .003$), but not in the experimental condition ($\beta = .08$, $t = .56$, $p = .58$).

In Step 1 of the regression predicting BAT time, experimental condition and ASI-3 physical concerns subscale scores predicted 16.1% of the variance, $F(2, 60) = 5.77$, $p = .005$, with experimental condition emerging as the only significant, unique predictor. In Step 2, the interaction did not explain significant additional variance, R^2 change = .01, $F(1, 59) = 4.11$, $p = .37$. In the regression predicting the catastrophic cognition "I will pass out," experimental condition and ASI-3 physical concerns subscale scores accounted for 23.6% of the variance, $F(2, 60) = 9.28$, $p < .001$. Experimental condition and physical AS were both significant predictors in the first step. In Step 2, the interaction variable did not explain significant additional variance, R^2 change = .01, $F(1, 59) = 6.55$, $p = .31$. Next, in the regression predicting BVS "Faintness," experimental condition and ASI-3 physical concerns subscale scores predicted 20.6% of the variance, $F(2, 60) = 7.78$, $p = .001$; both variables were significant, unique predictors. In Step 2, the interaction variable explained an additional 5.1% of the variance, $F(1, 59) = 6.80$, $p = .05$. Post-hoc probing analyses revealed that physical AS significantly predicted increased scores on BVS "Faintness" in the control condition ($\beta = .58$, $t = 3.16$, $p = .002$), but not in the experimental condition ($\beta = .11$, $t = .76$, $p = .45$).

In the regression predicting BVS "Dizziness," experimental condition and ASI-3 physical concerns subscale scores predicted 12.9% of the variance, $F(2, 60) = 4.44$, $p = .02$, with condition emerging as the only significant, unique predictor in the first step. In Step 2, the interaction term did not explain significant additional variance, R^2 change = .02, $F(1, 59) = 3.34$, $p = .29$. Finally, in the regression predicting HQ scores, experimental condition and physical AS predicted 11.4% of the variance, $F(2, 60) = 3.86$, $p = .03$. Experimental condition was the only significant, unique predictor in the first step. In Step 2, the interaction term explained a non-significant amount of additional variance, R^2 change = .03, $F(1, 59) = 3.29$, $p = .16$.

Discussion

The present study was conducted to test the hypothesis that AS causes panic symptoms through the process described in Cox's interactional model of panic. False physiological feedback provided to participants in the experimental condition successfully induced the belief that the body sensations associated with hyperventilation might produce a loss of consciousness. During a subsequent hyperventilation task, participants in the experimental condition, compared to those in the control condition, evidenced more frequent catastrophic thoughts about passing out, significantly higher peak anxiety, more anxiety symptoms, and increased hypervigilance to fainting-relevant body sensations. In addition, experimental participants evidenced significantly greater behavioral avoidance during a hyperventilation BAT. The magnitude of the effect of the manipulation on behavioral avoidance was large, and effect sizes for the other outcome measures were medium-to-large according to the criteria suggested by Cohen (1988). Because of the experimental nature of our design, and use of a sample of medically healthy young adults without a history of panic attacks, third variables that might account for the relationship between AS and panic (e.g., learning history) can be ruled out. The present findings suggest that, consistent with Cox's interactional formulation of panic, beliefs about the dangerousness of anxiety-related body sensations can cause a range of affective, cognitive, attentional, and behavioral responses to aversive anxiety-related body sensations similar to those associated with the experience of panic.

Mediational analyses were conducted to test the hypothesis that concerns about fainting elicited by the false physiological feedback manipulation would account for the higher levels of panic-related symptoms observed in the experimental group in response to the second hyperventilation challenge. As predicted, full mediation was attained for measures of peak anxiety, anxiety symptoms, and body vigilance. Because the mediator variable was assessed prior to the outcome variables, these findings indicate that experimentally-induced beliefs about the dangerousness of anxiety-related body sensations caused subsequent increases in anxious responding, thereby demonstrating true temporal mediation and lending support to the hypothesized causal role of AS in panic symptoms. It should also be noted that concerns about fainting did not mediate the large between-group effects on behavioral

avoidance. It is possible that other factors, such as reduced self-efficacy in tolerating the body sensations produced by hyperventilation, affected the behavior of participants in the experimental condition. Unfortunately, this possibility could not be examined in the present study.

Previous research has demonstrated that sensitivity to threat predicts fearful responding in the context of heightened threat (Telch et al., 2010). However, findings from the present study failed to support the context-sensitivity panic vulnerability hypothesis. In contrast to experimental condition, baseline AS specific to physical sensations did not consistently predict fearful responses to the second hyperventilation challenge. Moreover, physical AS failed to predict cognitive, behavioral, and emotional responses to the false feedback manipulation. These results indicate that fearful responses in the present study were more strongly influenced by the manipulation than by pre-existing sensitivity to threat. Accordingly, the present findings provide a controlled test of the relationship between AS and the development of panic symptoms that is largely free from the influence of extraneous variables.

Although use of safety behaviors (leaning on a chair or sitting down) during the second hyperventilation phase was more frequent among participants in the experimental condition than the control condition, this difference was not statistically significant. There are several possible explanations for this finding. First, participants were encouraged not to use the safety behaviors unless they deemed it absolutely necessary. Although these instructions were intended to avoid a ceiling effect wherein all participants used safety behaviors, they may have had the opposite effect by discouraging participants from seeking safety. A second possibility is highlighted by the findings of Powers, Smits, and Telch (2004), who reported that the mere availability of safety aids can produce the same effect as their utilization. That is, the knowledge that safety is available may provide as much reassurance as the engagement in safety behavior. This observation might explain why individuals in the experimental condition did not engage in significantly elevated rates of safety behavior utilization, relative to those in the control condition, despite their increased anxiety.

The results of this study have several implications for the prevention and treatment of panic attacks and panic disorder. Specifically, this study demonstrated that the presence of a specific belief causes panic-like symptoms. Therefore, panic might be prevented, and successfully treated, by dispelling this belief. Previous research (Gardenswartz & Craske, 2001; Schmidt et al., 2007) has found that brief interventions for individuals with high AS reduce AS substantially and prevent the subsequent development of panic disorder. The observation that symptom improvement following cognitive-behavioral therapy for panic disorder is mediated by the reduction of AS (Smits et al., 2004) further underscores the therapeutic value of strategies designed to reduce catastrophic misappraisals of threat. Clinicians might consider incorporating techniques such as interoceptive exposure that provide corrective information about the physiology of anxiety into standard treatment of other disorders when a person presents with high AS. For instance, Wald and Taylor (2007) added four weeks of interoceptive exposure prior to standard exposure therapy for posttraumatic stress disorder. They found that the interoceptive exposure substantially reduced both AS and posttraumatic stress disorder symptoms, and these symptoms continued to decrease through the remainder of treatment. These improvements were maintained at 1- and 3-month follow-ups. Taken together, the present findings suggest that because beliefs about the dangerousness of anxiety symptoms may give rise to panic symptoms, interventions that directly target AS may be especially useful in the prevention and treatment of panic disorder and other anxiety-related problems characterized by the fear of anxiety.

The present findings provide evidence in support of the theory that maladaptive cognitions about the dangerousness of anxiety-related body sensations can elicit panic symptoms. The present study was not intended to address the multitude of additional factors which may contribute to panic, and our findings should not be construed as suggesting that all panic attacks are caused by catastrophic beliefs. Indeed, as Bouton, Mineka, and Barlow (2001) have suggested, cognitive models have difficulty explaining the mechanisms which underlie certain manifestations of panic, such as nocturnal panic attacks and non-fearful panic. Results of the current study simply highlight one cognitively-mediated pathway through which panic symptoms may arise.

Several additional limitations of the present study should be considered. First, because the study used an undergraduate sample of medically healthy individuals without a history of panic attacks, it is unclear how well the present findings generalize to the general population. Conversely, the use of a healthy sample of individuals may

also be construed as a strength of the study as it is indicative of increased internal validity. Second, this study manipulated a specific physical concern related to fainting. Therefore, the present findings only support the cognitive model with respect to a very specific set of AS beliefs, and it remains to be seen whether these findings apply to other AS-symptom types (e.g., social and cognitive concerns). Third, this study used a multi-trait method of assessing panic-like symptoms as opposed to categorical measurement of the occurrence of panic attacks. As a result, the percentage of participants in this study who experienced bona fide panic attacks is unknown, and the results cannot be unequivocally generalized to the experience of panic attacks among individuals with panic disorder. Finally, hyperventilation and body vigilance were assessed via self-report only. Because objective physiological measurement of hyperventilation such as pCO^2 was not conducted, the validity of the manipulation is dependent on participants having followed study instructions to hyperventilate. The significant increase in HQ scores elicited by false physiological feedback provides indirect evidence that the present study was successful in this regard. The use of a self-report measure to assess body vigilance was necessitated by the present unavailability of a valid, objective measure of on-line attentional bias toward anxiety-related bodily sensations.

Our discussion of the present findings, in the context of both anxiety sensitivity and catastrophic misinterpretation theories of panic, risks conceptual confusion. Hence, it is important to tease apart the contributions of these trait and state variables. In the current study, the manipulation elicited the belief that hyperventilation-related sensations are likely to cause fainting. Subsequently, participants in the experimental condition reported experiencing more state catastrophic cognitions about fainting during the second hyperventilation task, and this increase was caused (i.e., fully mediated) by pre-existing beliefs about the dangerousness of hyperventilation-related body sensations. Although this belief was experimentally induced and representative of a temporary state, this study nevertheless demonstrates the cognitive, behavioral, attentional, and emotional consequences of having such a belief. Further, it would be expected that a persistent, stable belief would more consistently elicit an intense response related to the perceived dangerousness of feared sensations. Thus, our findings are consistent with Cox's interactional model in which beliefs about the meaning of sensations (AS) give rise to catastrophic cognitions in the presence of a specific physiological trigger.

In summary, this study provided a stringent test of the causal relationship between AS and panic symptomatology posited by Cox's interactional model (1996). A great deal of research has documented a robust and specific association between elevated AS and panic-related psychopathology (Taylor, 1999), but experimental evidence of causation has been elusive. Cox's interactional model provides a valuable framework for the evaluation of the cognitive model of panic as it highlights the collaborative function of trait (AS) and state (catastrophic misinterpretation) factors in panic attacks. The present study builds on previous research by demonstrating that experimentally-induced AS causes the subsequent development of anxiety, catastrophic cognitions, hypervigilance to feared body sensations, and behavioral avoidance in response to a panic challenge task. The present findings support cognitive conceptualizations of panic in which the tendency to misinterpret anxiety-related body sensations as threatening is viewed as a proximal cause of panic attacks. Future studies should be conducted to replicate these findings in other samples, and evaluate whether this effect applies to other domains of AS and panic triggers.

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