Role of Fear of Fear and Safety Information in Moderating the Effects of Voluntary Hyperventilation

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The present study investigated the singular and joint effects of fear of somatic sensations and perceived safety of hypocapnia-induced bodily cues on nonclinical subjects' subjective and psychophysiological response to a hyperventilation challenge. Fear of fear was assessed with the Body Sensations Questionnaire (BSQ; High versus Low), and subjects were randomly assigned to one of two informational conditions (Safety Information versus No Safety Information). When anticipating hyperventilation, High BSQ-Safety Information subjects reported higher subjective anxiety compared to Low BSQ-Safety Information subjects. Similarly, High BSQ-Safety Information subjects reported significantly more symptoms during the anticipatory phase compared to subjects in each of the other three conditions. During hyperventilation, fear of somatic cues and safety information exerted independent effects on subjective responding. High BSQ subjects reported higher levels of subjective fear and physical symptoms compared to Low BSQ subjects; subjects who received safety information reported lower levels of anxiety and physical symptoms compared to those who did not receive safety information. High BSQ subjects' heightened subjective fear response persisted through the hyperventilation recovery period. There were no group differences on the psychophysiological variables across any of the experimental phases. These findings provide further support both that fear of fear contributes to heightened emotional responding to biological challenges and that emotional responding is also affected by conditions that alter the perceived threat of the challenge-induced cues.

The past decade has seen a proliferation of research on the etiology of panic disorder (PD) from both biological and psychological perspectives. Biological investigators have posited various neurobiological bases of panic disorder (Klein, 1981; Sheehan, 1982). Some of the recently hypothesized biological mechanisms of action include adrenergic dysregulation (Charney, Heninger, & Breier, 1984; Nutt, 1989), carbon dioxide chemosensitivity (Lousberg, Griez, & van den Hout, 1988), pH sensitivity (Papp et al., 1989), serotonergic hypersensitivity (Targum & Marshall, 1989), and functional subsensitivity of the

gamma-aminobutyric acid-benzodiazepine submolecular complex (Roy-Byrne, Cowley, Greenblatt, Shader, & Hommer, 1990).

Biological challenges represent the most widely employed experimental paradigm for investigating the neurobiological basis of panic disorder. Presumably, specific agents (e.g., sodium lactate, CO_2, caffeine) precipitate panic by triggering the neurobiochemical dysregulatory mechanism, which in turn creates excessive autonomic arousal. Indeed, PD subjects show significant increases in anxiety following infusions of sodium lactate (Liebowitz et al., 1984), inhalation of carbon dioxide (Griez, Lousberg, van den Hout, & van der Molen, 1987), voluntary hyperventilation (Rapee, 1986), and administrations of caffeine (Charney, Heninger, & Jatlow, 1985). However, recent psychological models of panic (i.e., Beck, 1988; Clark, 1986) offer an alternative explanation to account for challenge-induced panic: namely, that panic results from the catastrophic misinterpretation of challenge-induced bodily cues.

Salkovskis and Clark (1990) argued that the wide range of biochemical panic provoking agents produce panic through their ability to create misinterpretable physical sensations rather than through specific biochemical pathways. The psychological model has received support from several investigations suggesting that cognitive factors play a critical role in moderating the PD subjects' panic response. Salkovskis and Clark (1990) manipulated subjects' tendency to interpret hyperventilation-induced sensations as either positive (good psychological adjustment) or negative (risk for fainting). Subjects in the positive interpretation group experienced hyperventilation as pleasant, whereas subjects in the negative interpretation group experienced the hyperventilation as unpleasant. Several studies have demonstrated that cognitive factors influence subjects' emotional response to CO_2 inhalation (Rapee, Mattick, & Murrell, 1986; Telch & Harrington, 1993; van den Hout & Griez, 1982). For example, van den Hout and Griez (1982) told normal subjects that CO_2/O_2 inhalation would produce either tension or relaxation. Subjects given tension instructions reported an increase in tension sensations, whereas subjects in the relaxation condition reported an increase in relaxation sensations. Perceived control appears to be another moderator of panic. Panic patients who falsely believed they could regulate CO_2/O_2 levels during a challenge task were less likely to panic compared to patients who believed they had no control over inhaled CO_2/O_2 levels (Sanderson, Rapee, & Barlow, 1989).

Anxiety sensitivity (i.e., the predisposition to respond anxiously to arousal sensations) has been identified as another important moderator of anxiety in physiological challenge tasks. Subjects with high anxiety sensitivity report higher sensations and higher subjective anxiety following voluntary hyperventilation compared to subjects with low anxiety sensitivity (Holloway & McNally, 1987; Rapee & Medoro, 1992), independent of a history of panic attacks (Donnell & McNally, 1989). In fact, Rapee, Brown, Antony, and Barlow (1992) found that fear of fear, as measured by the anxiety sensitivity inventory (ASI), was the best predictor of anxiety in response to hyperventilation.

The present study sought to extend current research by directly manipulating the perceived safety of hypocapnia-induced bodily cues through the provision of corrective information regarding the benign nature of these bodily sensa-

tions. Specifically, the authors investigated the singular and joint effects of fear of somatic sensations and perceived safety of hypocapnia-induced somatic cues on the subjective and psychophysiological response to a hyperventilation challenge. It was hypothesized that subjects with high fear of bodily sensations would display greater emotional responding to the hyperventilation challenge compared to subjects with low fear of somatic sensations. It was also hypothesized that subjects who received safety information would display lower emotional responding to the hyperventilation challenge compared to subjects who did not receive safety information. Finally, it was hypothesized that safety information would have a greater influence on challenge-induced fear among those subjects displaying high fear of fear.

Method

Subjects

Ninety undergraduates took part in the study. Subjects ranged in age from 17 to 23 with a mean of 18.0 years (SD = 1.5). Most subjects (92%) were white and female (86%). Subjects were recruited from a pool of undergraduates enrolled in Introductory Psychology classes at a large southwestern university. Subjects received experimental class credit for their participation.

Screening

Subjects were contacted by phone and were administered a medical screen. Subjects with a history of cardiovascular disease, respiratory disorder, epilepsy, or high blood pressure were excluded.

Design

The study employed a 2 × 2 factorial design. Fear of somatic sensations (high versus low) and safety information about hyperventilation (safety information versus no safety information) served as between-subject factors. Assessments of subjective and psychophysiological emotional responding were conducted across four separate phases (i.e., baseline, anticipation of hyperventilation, hyperventilation, and recovery).

Group Assignment and Experimental Conditions

Groups exhibiting high versus low fear of somatic sensations were determined using a median split of the Body Sensations Questions (BSQ; median = 2.2; Chambless, Caputo, Bright, & Gallagher, 1984) administered at baseline. Subjects within BSQ groups were randomly assigned to Safety Information or No Safety Information conditions.

Safety information condition. Subjects assigned to this condition were provided with information about the benign physical consequences of hyperventilating:

> "I will be taking you through a rapid breathing procedure.
> This procedure will involve having you breathe for a period
> of 2 minutes at a significantly accelerated pace, approxi-

mately three times the rate you normally breathe. This
rapid breathing may produce a temporary and harmless
drop in your carbon dioxide level similar to that produced
if you were to blow up several balloons one after the other.
This drop in carbon dioxide may produce a number of
physical sensations. These include an increase in your heart
rate, feelings of lightheadedness or dizziness, numbness
or tingling in parts of your body, and feelings of breath-
lessness. It is important that you understand that these
are *harmless, temporary* sensations that go away with-
in a couple of minutes after you resume your normal
breathing."

No safety information condition. Those assigned to the no safety informa-
tion condition were provided less complete information on the specific nature
of the physical sensations accompanying voluntary hyperventilation. Subjects
were not provided a clear message that these sensations were normal and non-
dangerous. The specific instructions were as follows:

"I will be taking you through a rapid breathing procedure.
This procedure will involve having you breathe for a period
of 2 minutes at a significantly accelerated pace, approxi-
mately three times the rate you normally breathe. During
this procedure, you may experience a number of physical
sensations similar to those experienced during an anxiety
attack."

Assessment Procedure

Following the phone screen, eligible subjects were scheduled to participate
in the experiment. Following informed consent, participants completed an as-
sessment battery consisting of the Body Sensations Questionnaire (BSQ) and
a demographic questionnaire, which included questions regarding a history
of panic attacks. A physiological measurement apparatus for heart rate (HR),
skin conductance (SC), and skin temperature (TMP) was explained and at-
tached (see below). Following a 5-minute resting baseline, subjects were read
the instructional set (Safety Information, No Safety Information). Participants
then waited another 5 minutes to create an anticipatory period prior to the
voluntary hyperventilation procedure in which they were instructed via cas-
sette tape to breathe at a rate of 30 breaths per minute for 2 minutes. Measures
of subjective fear, physical symptom severity, HR, SC, and TMP were ob-
tained at each of four phases: (1) resting baseline (5 minutes), (2) anticipation
of hyperventilation (5 minutes), (3) hyperventilation (2 minutes), and (4) hyper-
ventilation recovery (5 minutes).

Psychophysiological Measures

Three measures of physiological arousal (HR, SC, TMP) were simultane-
ously monitored using a J&J I-330 Physiological Monitoring System. A pho-

toplethysmograph sensor placed against a fingertip was used to measure HR. Skin temperature was measured using a thermal sensor placed between thumb and index finger. Finger electrodes placed on the middle pads of the index and middle fingers were used to measure SC. Measurement was continuous throughout each phase of the experiment and averaged over 5-second samples.

Psychological Measures

Body Sensations Questionnaire (BSQ). The BSQ is a 17-item scale that assesses fear associated with common sensations of autonomic arousal (e.g., heart palpitations, dizziness). Each item is scored on a 5-point Likert scale ranging from 1 (Not Frightened) to 5 (Extremely Frightened). The scale has high internal consistency (Cronbach alpha = .87) and adequate test-retest reliability (r = .67, Chambless et al., 1984).

Subjective measures. (HVC, SUDS). The Hyperventilation Checklist (HVC) is a 16-item scale constructed by the authors, consisting of physical sensations (e.g., shortness of breath, palpitations) and fears (e.g., fear of dying, fear of going crazy) associated with hyperventilation. Two physical sensations rarely associated with hyperventilation were also included to assess for response bias (e.g., sweet taste in mouth, itchiness on bottom of feet). Symptoms are rated on a 5-point Likert scale ranging from 0 (Absent) to 4 (Very Severe). The HVC total score is the sum of the 14 symptoms. The two response bias items were analyzed separately. Subjective level of anxiety was measured using an 11-point Subjective Units of Discomfort Scale (SUDS). Ratings for SUDS were anchored along a panic-relevant continuum (0 = not at all anxious; 5 = moderately anxious; 10 = full-blown panic).

Results

Analytic Overview

Fifteen separate 2 × 2 analyses of covariance (ANCOVAs) were conducted on the subjective (SUDS, symptoms) and psychophysiological (HR, SC, TMP) dependent variables at each experimental phase following baseline (i.e., anticipation of hyperventilation, hyperventilation, and recovery).[1] Fear of Somatic Sensations (High, Low) and Safety Information (Yes, No) were entered as between-group factors, and the baseline measure of each dependent variable served as its own covariate. Simple means comparisons followed significant univariate effects.

Means and standard deviations for the psychological measures across the four phases of the experiment are presented in Table 1.

Group Differences at Baseline (Phase 1)

High BSQ subjects showed higher SUDS [$F(1, 78)$ = 11.3, $p < .01$] and symptoms [$F(1, 78)$ = 9.0, $p < .01$] compared to Low BSQ subjects. No other significant differences were found at baseline.

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[1] Bonferroni correction was used to correct within families of tests at each assessment period. The adjusted level of significance was .01 (.05/5).

SCHMIDT ET AL.

TABLE 1
Means and Standard Deviations of the Psychological Variables Across Experimental Phase

	Baseline				Anticipation				Hyperventilation				Recovery			
	BSQ-H		BSQ-L		BSQ-H		BSQ-L		BSQ-H		BSQ-L		BSQ-H		BSQ-L	
	SI	NSI	SI	NSI	SI	NSI	SI	NSI	SI	NSI	SI	NSI	SI	NSI	SI	NSI
Symptoms																
M	18.2	17.5	15.1	15.5	20.3_a	16.7_{ab}	14.8_b	15.7_{ab}	32.5	35.8	24.0	26.5	19.0	22.6	16.0	16.4
SD	5.7	4.4	1.7	1.8	7.7	3.7	1.2	2.3	12.2	11.4	6.6	7.3	5.3	10.0	1.6	2.4
SUDS																
M	2.2	1.5	0.6	1.0	2.6_a	1.5_b	0.5_b	1.1_b	4.8	5.3	2.2	3.0	2.0	2.7	0.6	1.0
SD	2.2	1.4	1.0	1.3	2.6	1.4	0.8	1.2	2.5	2.4	1.3	1.5	1.9	2.4	0.7	1.5

Note. BSQ-H = High Fear of Somatic Cues, BSQ-L = Low Fear of Somatic Cues, SI = Safety Information, NSI = No Safety Information.
Possible range of scores: Symptoms (0–56); SUDS (0–10).
Only the anticipatory phase produced significant interactions. Means with different subscripts are significantly different ($p < .05$).

Effects of Anticipatory Responding (Phase 2)

At phase 2, an unexpected BSQ × Safety Information interaction emerged for SUDS [$F(1, 77) = 4.7, p < .05$]. Simple means comparisons indicated that High BSQ subjects receiving Safety Information reported greater SUDS than Low BSQ subjects receiving Safety Information [$F(1, 43) = 4.3, p < .05$]. There were no other significant differences between groups.

There was a Safety Information main effect trend on symptom ratings among those subjects receiving Safety Information reporting significantly higher symptoms compared to those who did not receive safety information [$F(1, 77) = 5.4, p < .05$]. This effect was qualified by a significant BSQ × Safety Information interaction [$F(1, 77) = 11.9, p < .01$]. High BSQ-Safety Information subjects reported significantly more symptoms than subjects in each of the other three conditions (i.e., High BSQ-No Safety Information; Low BSQ-Safety Information; Low BSQ-No Safety Information). There were no significant differences between groups on any of the psychophysiological measures at phase 2.

Effects on Voluntary Hyperventilation (Phase 3)

Analysis of SUDS ratings for phase 3 indicated a significant main effect for BSQ [$F(1, 77) = 15.0, p < .01$] and a main effect trend for Safety Information [$F(1, 77) = 6.5, p < .02$]. High BSQ subjects reported higher SUDS compared to Low BSQ subjects. Subjects receiving safety information reported lower SUDS compared to those who did not receive information. A similar pattern of findings was observed for symptom ratings [$Fs(1, 77) = 8.9, 3.3, ps < .01, .07$, for BSQ and Safety Information, respectively]. High BSQ subjects reported significantly higher symptom scores compared to Low BSQ subjects, and subjects receiving safety information reported lower symptom scores compared to those who did not receive this information. There were no significant interactions during phase 3 and there were no significant differences on any of the psychophysiological measures.

Because fear of bodily sensations is elevated in panic disorder patients (Chambless et al., 1984), a history of panic, rather than a fear of somatic sensations, may account for the increased responding among the High BSQ subjects. In order to test this, we reanalyzed our data using history of panic rather than BSQ status as a between-subjects factor. Panic history was not a significant predictor of emotional responding to the hyperventilation challenge.

Effects on Posthyperventilation Responding (Phase 4)

Compared to Low BSQ subjects, High BSQ subjects continued to report significantly higher SUDS ($F(1, 77) = 8.0, p < .01$] and significantly greater symptoms [$F(1, 77) = 8.0, p < .01$] during the recovery period. There was also a trend toward lower SUDS and symptoms for subjects receiving safety information compared to those who did not receive safety information [$ps = .07, .09$ for SUDS, symptoms]. There were no other significant findings during the recovery period.

Response Bias

Separate 2 (High BSQ vs. Low BSQ) × 2 (Safety Information × No Safety Information) analyses of variance (ANOVAs) were conducted at each experimental phase with the bogus symptom items (i.e., sweet taste in mouth, itchiness on bottom of feet) as the dependent variables. These analyses revealed no significant group differences in the endorsement of bogus physical symptoms.

Response Patterns Across Phases

Within subject ANCOVAs across phases were used to assess change over time. Analyses were collapsed across BSQ and information groups. Separate analyses were conducted on SUDS ratings, symptom ratings, SC, HR, and TMP while adjusting for baseline differences on each dependent variable. Analyses of the SUDS and symptom ratings revealed a similar pattern of findings. There was an overall main effect for Time for SUDS ratings [$F(3, 356) = 33.48$, $p < .01$] and symptom ratings [$F(3, 356) = 72.60, p < .01$]. Subjects reported significantly higher SUDS and symptoms during hyperventilation compared to each of the other three phases. Analyses of the psychophysiological indices also revealed significant differences across phases. HR was significantly higher during hyperventilation than during the other three phases [$F(3, 353) = 19.74$, $p < .01$]. During phases 2–4, SC was significantly higher compared to phase 1 [$F(3, 355) = 10.20, p < .01$]. There was also a trend for higher SC during hyperventilation compared to phase 2 ($p = .08$). Temperature at recovery was significantly lower compared to phases 1 and 2 [$F(3, 355) = 9.91, p < .01$]. There was also a trend toward lower TMP during hyperventilation compared to phase 1 ($p = .06$). Overall means and standard deviations for the psychophysiological measures across the four phases of the experiment are presented in Table 2.

Discussion

The present study lends further support for the role of cognitive factors in determining subjects' emotional responding to biological challenge. Consistent with prediction, the authors found that both fear of somatic sensations and the provision of safety information regarding hypocapnia-induced physical sensations independently affected the subjective fear response to voluntary hyperventilation.

The study's findings support the fear-of-fear hypothesis, which predicts that subjects who are fearful of physical sensations should show heightened emotional responding when exposed to a challenge task. The data also indicate that the level of fear of fear, compared to panic history, is a better predictor of emotional responding to challenge. Consistent with earlier reports (Donnelly & McNally, 1989), panic history did not predict emotional responding to hyperventilation.

The present study extends earlier challenge work by using an alternative measure of fear of fear. Whereas most previous work has utilized the ASI, this study measured fear of fear with the BSQ. As would be predicted, the

TABLE 2
OVERALL MEANS AND STANDARD DEVIATIONS OF THE PSYCHOPHYSIOLOGICAL
VARIABLES ACROSS EXPERIMENTAL PHASE

	Baseline	Anticipation	Hyperventilation	Recovery
SC				
M	4.7_a	6.9_b	8.9_b	7.6_b
SD	4.2	6.7	4.7	4.7
HR				
M	78.1_a	77.8_a	90.0_b	76.4_a
SD	13.1	13.0	14.3	13.6
TMP				
M	91.8_a	91.3_a	90.0_{ab}	88.6_b
SD	3.8	4.0	4.2	5.0

Range of scores: SC (0.4–27.4), HR (35–119), TMP (73.8–96.7).
Means with different subscripts are significantly different ($p < .05$).

pattern of findings using the BSQ is comparable to those in studies using the ASI. These findings are also consistent with recent recommendations that highlight the need to utilize multiple measures of fear of fear (Lilienfeld, Turner, & Jacob, 1993).

Providing subjects with prechallenge safety information significantly dampened their subjective fear response during the challenge. These findings are consistent with other studies that have demonstrated that perceived safety (Rapee, Telfer, & Barlow, 1991) and safety information regarding the physical effects of lactate infusion (van der Molen, van den Hout, Vroemen, Lousberg, & Griez, 1986) reduce anxious responding to a challenge. The perception of safety appears to be just one of a growing list of cognitive factors that influence panic patients' and nonclinical subjects' emotional responding to biological challenge. One property that these factors (e.g., perceived control, perceived safety) share is alteration of the subjects' threat appraisal. The influence of cognitive factors runs contrary to the view that panic disorder subjects' heightened emotional responding is due solely to a biochemical defeat.

Although safety information decreased subjective responding during hyperventilation, High BSQ subjects who received safety information displayed somewhat higher subjective anxiety and significantly greater symptom severity during the anticipation phase of the experiment compared to High BSQ subjects who did not receive safety information. This unexpected finding may be due to an expectancy effect created by the safety information manipulation. A variety of studies have shown that expectancies can influence emotional responding during challenge tasks (Rapee et al., 1986; van den Hout & Griez, 1982). However, the effect of expectancies on the anticipation of a challenge has not been adequately assessed. In the present study, subjects who received safety information were given the expectancy that they would soon

experience a variety of physical sensations (e.g., increase in your heart rate, feelings of lightheadedness or dizziness, numbness or tingling in parts of your body, and feelings of breathlessness). The authors speculate that hearing a description of these specific physical sensations is likely to have created apprehension in those High BSQ subjects who are necessarily fearful of these sensations. Thus, although safety information may decrease anxiety during the challenge, safety information appears to increase apprehension during the anticipation of the challenge.

Although the safety information manipulation was intended to affect the subject's perception of safety during the challenge, the manipulation may have affected other cognitive appraisals relevant to anxious responding. Several appraisal domains are likely to contribute to anxious responding during the hyperventilation challenge, including the perception of safety (Rapee et al., 1991; Telch, Valentiner, & Bolte, 1993; van der Molen at al., 1986), anxiety expectancy (van den Hout & Griez, 1982), perceived control (Sanderson et al., 1989), and predictability (Mineka & Kihlstrom, 1978). It is conceivable that each of these cognitive appraisals was affected by the provision of safety information. Methodological refinements, which include the assessment of change in all relevant expectancy domains, are required to determine the specific effects of experimental manipulations on expectancies.

Based upon an analysis of SUDS scores, none of the subjects experienced a panic during the challenge task. Heightened levels of anxiety but a low frequency of panic is consistent with other studies using a hyperventilation challenge, even when patients with panic disorder are challenged (Donnell & McNally, 1989; Holloway & McNally, 1987; Rapee, 1986). Several factors about the hyperventilation task are likely to have contributed to the absence of panic. Specifically, the hyperventilation challenge task contains elements of predictability (i.e., known duration of 2 minutes) and controllability (i.e., the task is voluntary such that subjects could discontinue at any time), each of which can decrease emotional responding (Mineka & Kihlstrom, 1978). The absence of actual panic during the hyperventilation challenge limits the generalizability of these findings to an understanding of panic per se. An alternative methodology for the study of the pathogenesis of panic and panic disorder is the use of biological challenges that would produce higher levels of panic, such as CO_2 inhalation and sodium lactate infusion (Gorman et al., 1984; Telch & Harrington, 1993).

The study's failure to detect differences on the psychophysiological measures is consistent with other hyperventilation challenge studies that have measured autonomic arousal (Beck & Scott, 1988; Salkovskis & Clark, 1990). Those studies indicate that subjects experience virtually the same change in autonomic responding during hyperventilation. Despite between-group similarities in autonomic arousal during hyperventilation, High BSQ subjects reported higher levels of anxiety and greater physical symptom severity compared to Low BSQ subjects. What might account for the exaggeration of symptoms of physiological arousal among high fear of fear individuals? Information processing studies clearly demonstrate an attentional and memory bias for threat information in anxiety patients (Ehlers, Margraf, Davies, & Roth, 1988;

McNally, 1990). In addition, panic patients display greater acuity to internal bodily sensations (Ehlers & Breuer, 1992). Taken together, these findings indicate that high-fear-of-fear individuals are likely to more closely attend to, and consequently report, physical perturbations. Does this indicate that High BSQ subjects show a general response bias to reporting physiological sensations? This is unlikely, given that we found no effect of safety information or fear of fear status on the report of the bogus symptoms of sweet taste in mouth and itchiness on the bottom of feet. Thus, it appears that High BSQ subjects are reporting only symptoms that are consistent with arousal produced by the hyperventilation challenge.

In conclusion, the present study provides strong evidence that fear of bodily sensations and the perception of safety significantly influence fear responses to biologically induced somatic perturbations and suggest that these factors be considered in the design and interpretation of future panic provocation studies.

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