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The absence of relation between anxiety sensitivity and fear conditioning using 20% versus 13% CO₂-enriched air as unconditioned stimuli[☆]

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Abstract

Anxiety sensitivity has been implicated as a risk factor in the development and maintenance of anxiety and fear-related disorders. Indeed, persons who score high on the anxiety sensitivity index (ASI) are generally more responsive to biological challenge procedures such as CO₂-inhalation that directly evoke the feared bodily events. One would expect, therefore, that persons high on anxiety sensitivity should be more conditionable and hence more likely to acquire fears, than persons low on anxiety sensitivity when CO₂-enriched air is used as an unconditioned stimulus (UCS). Undergraduates ($N = 96$), scoring high, medium and low on the ASI received 8 repeated 20-s inhalations of either 20 or 13% CO₂-enriched air (UCSs) paired with one of three CSs differing in fear-relevance (snake, heart and flowers). Several autonomic and self-report measures were assessed. Contrary to expectation, electrodermal and cardiac conditioned responses failed to discriminate between ASI groups. Yet, SUDS and severity and frequency of DSM-IV panic symptoms varied reliably as a function of anxiety sensitivity. Overall, the findings suggest that anxiety sensitivity is related to subjective fear-related complaints, but not autonomic responding and conditionability. We discuss clinical and theoretical implications for understanding the place of anxiety sensitivity in fear onset. © 1998 Elsevier Science Ltd. All rights reserved.

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1. Introduction

Anxiety sensitivity, or the fear of anxiety symptoms based on beliefs that such symptoms are harmful or dangerous, has been described as a psychological risk factor in the etiology and maintenance of fear and anxiety-related disorders (Reiss, 1991). As such, it has been suggested that anxiety sensitivity denotes a cognitive risk factor that mediates the tendency to respond to otherwise benign bodily sensations with fear (Taylor et al., 1992). Indeed, several studies have shown that persons scoring high on the Anxiety Sensitivity Index (ASI; Reiss et al., 1986) are also more likely to respond to biological challenge procedures that specifically evoke bodily sensations (e.g. hyperventilation and inhalations of CO₂-enriched air) and generally report more psychological distress and panic symptoms compared to persons low in anxiety sensitivity (Harrington et al., 1996; Schmidt et al., 1997). Yet, persons scoring high on the ASI are not necessarily more autonomically reactive or physiologically aroused in response to stress (Shostak and Peterson, 1990).

Findings from biological challenge procedures are generally consistent with the view that anxiety sensitivity serves to amplify fear responding (Reiss, 1991) and particularly subjective complaints. The tendency for behaviors related to anxiety sensitivity to amplify anxious behaviors should, in turn, result in a self-perpetuating positive spiral of increased anxious responding. For example, responding to a fast heart beat as a sign of cardiac arrest should, in turn, result in more subjective distress, arousal, heightened anxiety, greater heart beats, more negative appraisals and so on. Thus, anxiety sensitivity should produce additional anxiety and hence more autonomic and subjective arousal; however, evidence for differential autonomic arousal as a function of anxiety sensitivity is modest at best (e.g. Telch and Harrington, 1994).

Another psychological risk factor in the development of fear and anxiety-related problems is learning or conditioning. Numerous studies have demonstrated that fears can be acquired via pairings of a previously neutral verbal and nonverbal stimuli (NSs) with aversive unconditioned stimuli (UCSs) capable of evoking fear-related unconditioned responses (UCRs). Although the place of such conditioning is still much debated in explaining the etiology of clinical fears (see Menzies and Clarke, 1995; see also Forsyth and Chorpita, 1997), conditioning remains a robust fact. Much of the criticisms of conditioning models of phobic fear onset relates to confusion over what exactly is being conditioned and the critical process variables involved. For instance, is the UCS the critical variable?, the UCR? or some combination of both? Unfortunately, most of the criticisms of conditioning have emphasized the failure to find an environmental UCS to account for clinical fear onset, leading to conclusions such as no environmental UCS means no conditioning (cf. Menzies and Clarke, 1995). Yet, as Forsyth and Eifert (1996) suggest, the more critical process variable for conditioning is the bodily UCR and related dimensions such as amplitude, duration, and uncontrollability, not the UCS; an argument that has recently found increasing experimental support (see Forsyth et al., 1997; Forsyth and Eifert, 1998).

In an attempt to address the importance of the response in fear onset, our laboratory has used UCSs (e.g. high concentrations of carbon dioxide-enriched air [CO₂]) that evoke clinically relevant bodily sensations typical of persons with anxiety-related complaints, such as breathlessness, dizziness and chest pain (Forsyth et al., 1996; Forsyth and Eifert, 1998). Although preliminary, findings from several studies using CO₂-enriched air as a UCS have

demonstrated that the concomitant panic-like bodily sensations can function as conditioning events and that content-specific conditioning (fear-relevance effects) varies reliably as a function of the intensity of the UCS (Forsyth and Eifert, 1998) and particularly the UCR (Forsyth et al., 1996, 1997). Yet, no studies exist testing whether anxiety sensitivity mediates conditioning in cases involving the evocation of a wide range of bodily sensations as is typical of inhalations of high concentrations of CO₂-enriched air.

Given that high ASI participants typically respond with more fear to panic provocation procedures using CO₂-enriched air compared to low ASI participants, one would expect that high ASI participants should also be more likely to develop learned associations based on such responses. That is, anxiety sensitivity should mediate the strength and valence of bodily sensations produced by breathing CO₂-enriched air (i.e. the strength of the UCR) and hence the probability of strong emotional learning or conditioning. We did not address this issue in our previous research despite its potential theoretical and practical importance. Thus, in the present study we sought to address the role of anxiety sensitivity and its relation to conditioning by re-examining some of our previous data (see Forsyth and Eifert, 1998) that bear directly on this issue. The present analogue study examined the relation between anxiety sensitivity and conditioning in an undergraduate non-clinical population using 20% versus 13% CO₂-enriched air as UCSs. Given the importance of the response in accounting for conditioned fear onset and its place within the construct of anxiety sensitivity more generally, we chose to emphasize the relation between ASI and autonomic and self-reported responses regardless of the environmental CS viewed. Such an approach appeared justifiable for at least three reasons. First, our preliminary analyses showed that participants did not differ during habituation in terms of their electrodermal or cardiac orienting responses to the particular CSs used in this study (i.e. fear-relevant (e.g. snake, human heart) and fear-irrelevant (e.g. flowers)); a finding consistent with our previous study using similar CSs (see Forsyth et al., 1997; Forsyth and Eifert, 1998, for data related to the conditionability of the CSs used in the present study as a function of fear-relevance and UCS intensity). Second, as will be seen, our sample sizes precluded analyses with sufficient power to address the relation between ASI and content-specific fear-relevance using our particular CSs; however, we have reported the relation between fear-relevance and UCS intensity in relation to autonomic conditioning elsewhere (see Forsyth and Eifert, 1998). Third, and perhaps most importantly, for present purposes, the ASI construct appears more central to responding to one's own responses compared to responding to external events (Reiss and McNally, 1985; Reiss, 1991). Thus, it made more sense to emphasize subjective and physiological conditioned and unconditioned responses more globally in relation to ASI scores. Generally, we expected that high ASI participants would show stronger electrodermal and cardiac conditioning, give larger subjective distress ratings, and report more panic symptoms compared to low ASI participants.

2. Method

2.1. Participants

96 undergraduate volunteers (48 males and 48 females; $M = 19.8$ years of age, $S.D. = 3.13$) at West Virginia University served as participants and received course credit. All participants

were prescreened for medical and psychiatric conditions and those reporting past or present histories of any of the following were not allowed to participate: (a) cardiac or respiratory disease, (b) asthma, (c) epilepsy, (d) hypertension, (e) stroke, (f) psychiatric condition or (g) having witnessed someone panic, faint or have a heart attack. Participants were also excluded if they reported common specific fears (e.g. snakes) or if they had a history of panic attacks.

2.2. Materials and apparatus

2.2.1. Conditioned stimuli (CSS)

The CSs were three 25-s animated color video vignettes differing in fear-relevance: (a) exteroceptive fear-relevant (sidewinder snake); (b) an exteroceptive representation of an interoceptive fear-relevant stimulus (a human heart beating arrhythmically) and (c) neutral control (daisies swaying in the wind).

2.2.2. Unconditioned stimuli (UCSS)

The UCSs were 20-s inhalations of premixed 20% (20% CO₂, 21% O₂, 59% N₂) and 13% CO₂-enriched air (13% CO₂, 21% O₂, 66% N₂) administered through a continuous positive air pressure Downs C-Pap Mask with head strap. Attached to one free port of a manually controlled 3-way stop cock valve (Hans Rudolph) was a 30-l meteorological balloon which was inflated with the CO₂ mixture. Participants breathed the CO₂ gas directly from the balloon to minimize detection from pressurized CO₂ to nonpressurized room air. The C-Pap mask was connected to a free 22-mm port of the stop cock valve via 1.8-m of aerosol tubing and the remaining port was left unattached and fed room air. Manual control of the 3-way stop cock valve ensured uninterrupted breathing of the CO₂ gas, ease of switching between CO₂-enriched air and room air during acquisition trials and prevented the gases from being combined. The CO₂ apparatus was in a room adjacent to the subject chamber.

2.2.3. Physiological measures

A Coulbourn Modular Polygraph, interfaced with Coulbourn's Lablinc software (High Speed Videograph), was used for continuous digital monitoring of physiological responses. All physiological channels were calibrated on-line prior to sampling. Skin conductance was sampled using disposable 8-mm diameter Ag/AgCl electrodes which were coated with a 0.05-M concentration of NaCl and attached to the skin surface with concentric adhesive collars. Heart rate was sampled using Medi-Trace pre-gelled Ag/AgCl (#GC-11) disposable foam electrodes. Skin conductance levels (SCLs) and responses (SCRs) were directly recorded in microsiemens (mS) in an AC coupling mode with a Coulbourn S71-23 isolated skin conductance coupler which provides a constant 0.5 V across the electrodes. Electrodermal electrode placement followed a standard bipolar palmar configuration on the nondominant hand. Heart rate was sampled in beats/minute (bpm) using an analogue-to-digital Coulbourn S77-26 tachometer that was fed through a S75-01 bioamplifier. Heart rate was sampled using a standard bi-lateral electrode configuration (right and left of the sternum just below the clavicle).

2.2.4. Self-report measures

Prior to the experimental procedure, participants completed the following measures: (a) ASI (Reiss et al., 1986), a 16-item questionnaire designed to assess fear of anxiety-related symptoms and (b) the Body Sensations Questionnaire (BSQ; Chambless et al., 1984), a 17-item measure of fears associated with autonomic arousal (e.g. heart palpitations, dizziness).

During the procedure, participants rated their subjective units of distress (SUDS) following each CS presentation on a 100-min visual analogue scale anchored from *not at all distressed* to *very much distressed* (Wolpe, 1958).

The Diagnostic Symptoms Questionnaire (DSQ; Rapee et al., 1992), a 15-item measure of the presence and intensity of 12 somatic and three cognitive DSM-III-R (American Psychiatric Association, 1987) panic symptoms, was administered immediately following the procedure. Intensity ratings for each endorsed symptom are made on a 9-point Likert-type scale (0 = not at all to 8 = very strongly felt). The following composite measures can be derived from the DSQ: total number of physical symptoms and catastrophic and noncatastrophic thoughts, mean intensity of physical sensations, cognitive symptoms, and reported fear.

2.3. Procedure

Equal numbers of males and females were randomly assigned to one of three CS types and, within each CS type, to either 20 or 13% CO₂-enriched air (UCS). For ethical reasons, and to control for different expectancies regarding the consequences of breathing CO₂-enriched air, all participants were fully informed of several possible negative consequences of breathing CO₂-enriched air including breathlessness, dizziness, chest pain and tachycardia. A 10-min adaptation period was followed by three consecutive phases: habituation, acquisition and extinction. During habituation, participants breathed normal room air while watching four nonreinforced CS presentations. Acquisition consisted of eight reinforced CS+ presentations paired with CO₂ inhalations. During acquisition, UCS onset occurred 5-sec after CS+ onset and terminated at video offset. The UCS lasted for 20-s whereas all CSs were 25-s in length. The 5-s inter-stimulus interval (ISI) was not previously paired with the UCS and allowed for an assessment of conditioning across trials and phases. The inter-trial interval (ITI) varied between 35- and 180-s with a mean of 120-s (Forsyth et al., 1996). Extinction involved 10 nonreinforced CS presentations. SUDS ratings followed each CS presentation across all experimental phases. Following extinction, participants completed the DSQ to assess whether they had experienced any panic-like symptoms during the procedure.

2.4. Data reduction

Electrodermal responses (SCRs) were defined as the average onset to peak difference in skin conductance occurring within the 5-s interval prior to UCS onset across all phases. This score was averaged across trials within each phase, yielding three data points for the analysis which corresponded to each phase of the experiment. The cardiac acceleratory response (HR) was defined as the maximal onset to peak change in heart rate in the 5-s interval prior to the onset of the UCS across all phases. Cardiac response was averaged across trials within each phase, yielding three data points for the analysis. Subjective distress was defined as the average SUDS

rating across trials for each phase of the experiment. Lastly, bodily fears was operationalized as the total score on the BSQ and DSM-IV panic symptoms were derived from subscale scores on the DSQ assessing total number and severity of panic symptoms, physical symptoms and catastrophic thoughts.

3. Results

To assess the relation between anxiety sensitivity and conditioning, participants were divided into three equal groups ($n = 32$) based on their ASI total scores: high ASI ($ASI \geq 20.0$), moderate ASI ($ASI > 11.33$ or < 20.0) and low ASI ($ASI \leq 11.33$). Based on this grouping, a series of univariate ANOVAs were conducted on the following dependent measures: electrodermal (SCRs) and cardiac (HR) conditioned responses (CRs), SUDS ratings and BSQ and DSQ scores. All significant effects were followed by post-hoc comparisons using Scheffe's procedure, $p < 0.05$ ¹.

3.1. Primary analyses

3.1.1. Electrodermal conditioned response (SCRs)

As can be seen in Table 1, SCRs did not discriminate between ASI groups across all phases of the experiment.

3.1.2. Cardiac conditioned response (HR)

As with SCRs, cardiac conditioned responses did not discriminate between ASI groups across all phases of the experiment (see Table 1).

3.1.3. Subjective distress (SUDS)

Unlike SCRs and HR, SUDS ratings did discriminate between ASI groups for the acquisition phase of the experiment, with the high ASI group reporting approximately twice as much distress during acquisition compared to the moderate and low ASI groups (see Table 1).

3.1.4. Bodily fears (BSQ)

As with SUDS, report of bodily fears did discriminate between ASI groups in the expected direction such that greater anxiety sensitivity corresponded with greater self-report of bodily fears (see Table 2).

3.1.5. DSM-III-R panic symptoms (DSQ)

Report of panic symptoms experienced during the procedure did discriminate reliably between the high ASI group compared to the moderate and low ASI groups. As can be seen in

¹ Post-hoc comparisons were also performed with the more robust Tukey HSD method and results were equivocal to those described using the Scheffe procedure.

Table 1. Electrodermal, cardiac and SUDS responses as a function of ASI group

	ASI group (mean (S.D.))			<i>F</i>	<i>p</i>
	low	medium	high		
Electrodermal response					
Habituation	0.92 (0.80)	0.83 (0.83)	0.88 (0.93)	0.10	ns
Acquisition	0.90 (0.72)	0.92 (0.95)	1.06 (1.10)	0.28	ns
Extinction	0.53 (0.56)	0.56 (0.73)	0.84 (1.16)	1.27	ns
Cardiac response					
Habituation	1.31 (2.29)	2.64 (3.73)	2.11 (3.54)	1.36	ns
Acquisition	2.18 (1.72)	2.47 (1.96)	1.98 (1.81)	0.56	ns
Extinction	0.78 (1.31)	0.38 (0.97)	0.68 (1.08)	1.28	ns
SUDS ratings					
Habituation	10.10 (14.99)	10.44 (12.24)	19.93 (21.06)	3.57	0.05
Acquisition	34.10 ^a (24.85)	32.66 ^a (24.93)	60.17 ^b (25.15)	12.30	0.001
Extinction	9.06 (11.38)	8.86 (12.51)	16.51 (15.94)	3.38	0.05

Units for electrodermal responses (SCRs) are in microsiemens (mS); units for cardiac responses are in beats-per-minute (bpm); means with different superscripts differ, $p < 0.05$ (Scheffe); df for all univariate tests (2, 93).

Table 2, the high ASI group reported a greater number and severity of physical and cognitive panic symptoms compared to the other ASI groups.

3.2. Additional exploratory analyses

Additional analyses were conducted on the primary dependent measures with sex (male versus female) added as a between-group factor. Also, we examined electrodermal and cardiac

Table 2. Bodily fears (BSQ) and DSM-III-R panic symptoms (DSQ) as a function of ASI group

	ASI group (mean (S.D.))			<i>F</i>	<i>p</i>
	low	medium	high		
BSQ Total score	24.41 ^a (4.60)	31.31 ^b (5.93)	44.44 ^c (8.85)	73.87	0.01
DSQ Panic symptoms					
Number of symptoms	6.03 ^a (2.75)	6.06 ^a (2.50)	8.03 ^b (3.78)	4.48	0.01
Severity of symptoms	3.15 ^a (1.44)	2.94 ^a (1.12)	4.00 ^b (1.45)	5.54	0.01
Physical symptoms	1.52 ^a (1.06)	1.29 ^a (0.58)	2.26 ^b (1.44)	6.96	0.01
Distressing thoughts	4.25 ^a (2.40)	4.09 ^a (2.30)	5.94 ^b (3.23)	4.67	0.01

Means with different superscripts differ, $p < 0.05$ (Scheffe); df for all univariate tests (2, 93).

UCRs during acquisition to assess whether ASI or sex bears any relation to general psychophysiological reactivity to the CO₂ inhalations. Electrodermal and cardiac UCRs were operationalized as the average response during the last 10-s of the UCS minus the average response during the first 5-s of the UCS (CO₂ exposure) for each of the eight acquisition trials. Electrodermal and cardiac UCRs were further reduced into one average composite score representing each domain. Each composite score was intended to represent the magnitude of change in the UCS across the acquisition trials.

3.2.1. Effects of sex on autonomic CRS

Consistent with the previous analyses, conditioned electrodermal and cardiac responses did not discriminate between ASI groups or sex across all phases of the experiment.

3.2.2. Effects of sex of subjective indices

For all the subjective measures (i.e. BSQ, DSQ panic symptoms and SUDS ratings across phases), only three significant effects were found for sex. Generally, females reported more severe panic symptoms [$F(1, 90) = 4.37, p < 0.039$], a greater frequency of cognitive symptoms [$F(1, 90) = 25.84, p < 0.001$] and more subjective distress (SUDS) during acquisition [$F(1, 90) = 10.14, p < 0.002$] compared to males. As with the previous analyses, self-report domains reliably differed as a function of ASI group; however, habituation and extinction differences in SUDS ratings were no longer significant when sex was included as a between group factor. No other effects were significant.

3.2.3. Anxiety sensitivity and autonomic UCRS

There were no significant between-group differences for electrodermal or cardiac UCRs as a function of ASI group. There was, however, a significant effect for sex on HR, $F(1, 95) = 7.15, p < 0.01$, such that females ($M = 8.30, S.D. = 4.28$) exhibited greater increases in heart rate compared to males ($M = 5.78, S.D. = 4.46$). No other effects were significant.

4. Discussion

The central aim of the present study was to address whether anxiety sensitivity is a risk factor for conditioning when using UCSs capable of evoking subjective and bodily sensations which serve as the target of fear as captured by the ASI construct. Contrary to expectation, electrodermal and cardiac autonomic responses were unrelated to anxiety sensitivity and conditionability. That is, persons high in anxiety sensitivity did not differ in terms of the magnitude of autonomic conditioning compared to persons low or moderate in anxiety sensitivity. This negative finding is somewhat surprising given past research suggesting that persons high in anxiety sensitivity are more responsive to biological challenge procedures such as inhalations of high concentrations of CO₂-enriched air and arguments that anxiety sensitivity should potentiate fear responding in cases where there is even a small chance expectation of becoming anxious (Reiss, 1991). In the present study, a chance expectation was present in the form of instructions about the potential negative (not positive) consequences of breathing CO₂-enriched air. Further, we have demonstrated elsewhere that autonomic

conditioning effects vary reliably as a function of the CSs and UCSs used in the present study and that our UCSs were effective in establishing conditioning (Forsyth and Eifert, 1998).

Perhaps heightened responsiveness to CO₂-enriched air reflects more of a cognitive bias to respond to bodily sensations as signs of threat or danger than the actual sensations experienced. The consistent pattern of differences between ASI groups across the self-report response domains seems to support this conclusion. For instance, despite comparable levels of autonomic conditioned responses, high ASI participants consistently reported greater subjective distress and more overall panic symptoms than the moderate and low ASI groups. Although preliminary, this decoupling of autonomic and self-reported fear responding suggests that anxiety sensitivity may be a risk factor for subjective complaints produced by evoking salient bodily sensations, but that anxiety sensitivity is not necessarily a risk factor in terms of increasing the probability of autonomic fear conditioning or general autonomic arousal in response to CO₂ inhalation. In summary, the present findings cast doubt on the notion that anxiety sensitivity may be a risk factor in fear conditioning, especially in cases involving panic-like bodily sensations as a UCS. Indeed, anxiety sensitivity may be more predictive of whether persons report distress to bodily sensations, but not necessarily predictive of the actual magnitude of autonomic sensations experienced.

Although the present findings appear at odds with conceptual argument related to anxiety sensitivity as a risk factor in the development of fear and anxiety-related problems, they are not as discrepant as they may first appear. For instance, it is interesting to note that despite comparable levels of autonomic arousal to the UCSs, high ASI participants consistently over-estimated the same arousal as signs of threat or danger compared to participants scoring moderate-to-low on the ASI. This finding supports the view that ASI is related to a cognitive bias to interpret arousal in a threatening way, but not a tendency for persons high in anxiety sensitivity to experience more arousal or bodily sensations in response to inhalations of CO₂-enriched air. Further, this interpretive bias may operate independently of additional signs of arousal. That is, once benign bodily sensations are associated with a threatening interpretation, any signs of bodily arousal may be subsequently interpreted in a similar fashion regardless of their severity or intensity. However, the data presented here suggest that increased subjective distress and panic symptom complaints in participants scoring high on the ASI does not correspond to increased autonomic arousal as would be expected if a positive feedback loop was operative. In any event, additional work is clearly needed to address the role of anxiety sensitivity as a risk factor in the development of fear and anxiety-related disorders, and especially work that seeks to integrate cognitive risk factors and learning or conditioning processes. With the availability of more objective indices of fear derived from psychophysiological assessment and laboratory fear-induction procedures and new developments in ecologically valid laboratory fear conditioning procedures (e.g. Forsyth and Eifert, 1996, 1998; Forsyth et al., 1996), we should be well positioned to better understand the role of anxiety sensitivity as a risk factor in the etiology and maintenance of anxiety-related disorders.

Additional exploratory analyses addressed whether sex is a risk factor for fear conditioning when using CO₂ to induce panic-like symptoms. The effect of sex was significant for the UCR cardiac response, with females showing a greater change in heart rate across acquisition trials compared to males. In contrast, this effect was not seen for UCR electrodermal response.

Surprisingly, females did not differ from men on autonomic measures of conditioned cardiac arousal due to CO₂-enriched air panic induction. Females, however, did report greater overall subjective distress and panic symptoms compared to males. Yet, it is unclear why females did not show greater cardiac CRs compared to males, especially given that females showed significantly larger cardiac UCRs compared to males. Admittedly, we have no easy answer to explain this discrepancy. Yet, our past research suggests that the relation between the CR and UCR (and UCS intensity) is not perfectly coupled, and that the UCR is not necessarily a reliable predictor of a CR even within the same response system (i.e. the correlation between UCR and CR magnitude is less than one; see Forsyth et al., 1997). The general pattern of findings with regards to sex differences suggests that females are no more likely than males to show autonomic conditioning as a function of breathing CO₂-enriched air as a UCS. Yet, females are more likely to report cognitive symptoms of panic and to rate the symptoms they do experience as more severe compared to males. This suggests that the higher incidence of panic among females may be mediated by their cognitive interpretation of bodily sensations and their willingness to report such experiences, but not an increased tendency to experience more frequent or intense bodily sensations.

Before concluding, we wish to address several caveats about the present study. As indicated, this was an analogue non-clinical undergraduate population that was carefully selected to rule out other potential historical factors (medical and psychiatric conditions) that may mediate responsivity to breathing high concentrations of CO₂-enriched air. Such factors may also mediate the probability of conditioning. The results of the present study may, therefore, differ from those of previous studies because most of those studies did not employ such a rigorous screening process. Moreover, restricting subjects to those without related medical and psychiatric conditions may limit the generalizability of these findings to clinical populations. In the future, we plan to address these concerns by testing subjects who have with established DSM-IV diagnoses (e.g. panic disorder) as well as by looking more closely at other factors, such as family illness history, vicarious conditioning and suffocation fear, that may mediate responsivity to CO₂-enriched air and conditionability based on such responses. We also plan to include a noninvasive measure of end-tidal PCO₂ as a control to ensure an accurate index of amount of CO₂ inhaled, but also to address the relation between changes in ventilation and blood gas and fear responding.

An additional area for future research may be to further define and explore the parameters of cardiac and electrodermal responses to the CO₂. Although the present study indicates that gender, but not anxiety sensitivity, has an effect on an individual's response to CO₂, future studies might assess the effects of other variables, such as age, ethnicity or past experience with CO₂ exposure, as well as predictability, controllability and suffocation fear. Further, it would seem to be important to address whether anxiety sensitivity interacts with the content of particular stimuli (i.e. fear-relevance). We could not address this issue in the present study due to insufficient sample sizes; however, our previous analysis with these data suggest that fear-relevance is important and interacts with the intensity of the UCS (20% versus 13% CO₂) and UCR in increasing the probability of autonomic conditioning (see Forsyth et al., 1997; Forsyth and Eifert, 1998). Lastly, future studies may benefit from the use of a multi-factorial measure of anxiety sensitivity, rather than a singular unitary measure of ASI, one which decouples the physiological and subjective dimensions of anxiety sensitivity.

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