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ANXIETY SENSITIVITY AND PERCEIVED CONTROL OVER ANXIETY-RELATED
EVENTS: EVALUATING THE SINGULAR AND INTERACTIVE EFFECTS IN THE
PREDICTION OF ANXIOUS AND FEARFUL RESPONDING TO BODILY SENSATIONS

A Dissertation Presented

by

Kristin Lorraine Gregor

to

The Faculty of the Graduate College

of


The University of Vermont


In Partial Fulfillment of the Requirements
for the Degree of Doctor of Philosophy
Specializing in Psychology

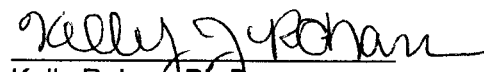
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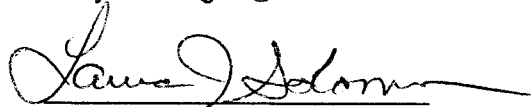
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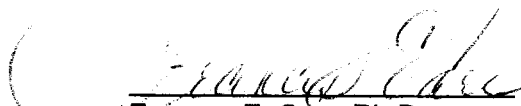
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Abstract

The current investigation examined the singular and interactive effects of anxiety sensitivity (AS) and perceived control over anxiety-related events in the prediction of panic symptoms using a biological challenge paradigm. Two hundred and twenty-nine participants ($m_{age} = 21.02$, $SD = 7.55$, 124 females) were recruited from the greater Burlington, Vermont community. Results indicated that pre-challenge AS, but not perceived control over anxiety-related events, significantly predicted post-challenge panic attack symptoms, anxiety focused on bodily sensations, and interest in returning for another challenge (behavioral avoidance). There were no interactive effects between AS and perceived control over anxiety-related events. For the physiological measures, pre-challenge AS was predictive of change in skin conductance level (pre-post challenge), and pre-challenge perceived control over anxiety-related events was predictive of change in respiration rate (breathes per minute). No significant effects were evident for heart rate and there were no significant interactive effects between AS and perceived control over anxiety-related events for any of the physiological variables. Findings of the investigation are discussed in relation to the role of AS and perceived control over anxiety-related events in terms of vulnerability for panic psychopathology.

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Introduction

The overarching goal of the present investigation was to examine the singular and interactive effects of two theoretically-relevant cognitive vulnerability factors for panic psychopathology – *anxiety sensitivity* (AS; McNally, 2002) and *perceived control over anxiety-related events* (Barlow, 1991) – in terms of their association with panic vulnerability in a laboratory setting.

Emotion States: Theoretical Perspectives

In the study of anxiety and its disorders, it is important for explanatory precision for scholarly work to accurately clarify the nature of emotional states. Historically, there has been much intellectual activity focused on the nature of emotional phenomena and the best way to conceptualize them. Extant theories, for example, have ranged from understanding emotion as a form of behavior (e.g., Ekman & Davidson, 1994; Izard, 1977), to a form of biological process (e.g., Cannon, 1929; Kagan, 1989), to a form of cognition (e.g., Beck, 1993; Lazarus, 1991). Such accounts have been critically important in helping studies of emotion clarify the boundaries and underlying processes involved with affective states. Yet, each of these theories has been met with challenges from both empirical and conceptual perspectives (see Barlow, 2002, for a discussion), leading to integrative theories that attempt to cull out the most meaningful core aspects of various perspectives in one overarching model. One of the most influential integrative theories of emotion in terms of anxiety and its disorders has been offered by Peter Lang (Lang, 1978, 1985, 1994).

Bioinformational theory of emotion. Lang (1994) has conceptualized emotion from an integrative perspective and utilized the analogy of a computer system to illustrate how it may function. Lang (1978, 1994), specifically, has theorized that emotion is a latent construct of “action tendencies” that are stored in memory (the “hard drive”)

and accessed through the processing of information. From Lang's (1994) perspective, emotion is best seen as "behavioral acts" (manifest indicators of the latent construct) that represent responses to stimuli in a particular context. These behavioral acts, or the software of a computer, include not only the information provided by the stimulus but also the response, such as avoidance or physiological responding. These responses provide "data," which is then stored in long term memory. To illustrate, when an individual experiences a potentially anxiety-provoking situation, he/she will theoretically "process" the information to determine if danger is present, and then, respond physiologically and behaviorally with an "appropriate" behavioral act (e.g., escape, in the case of an actual or perceived threat, and no avoidance, in the case of no immediate or perceived threat). Moreover, when an "anxiety event" occurs, this experience will presumably then be stored in long term memory, solidifying that situation as one to be feared or anticipated in future circumstances. This type of perspective attempts to integrate the various systems presumably involved with the experience of emotional states such as anxiety and fear. That is, there is not just one process applicable to one response system that is operative as past theories have hypothesized (e.g., Beck, 1993); rather, all three response systems – behavioral, physiological, and cognitive – act together to generate, execute, and ultimately, characterize an emotional event.

In total, scholars such as Lang have consistently conceptualized anxiety and fear states as biologically-driven, but not defined singularly (i.e., explained or accounted for by a single biological system), as reactions that help coordinate responding to environmental threats and challenges. Differences between emotion states can be apparent at numerous levels depending on the type of environmental threat or challenge encountered. For example, the triggering cues and nature of the emotion response (state) would be different for a state of fear compared to that of disgust, sadness, joy,

and so on. Though differences are expected between both positive and negative emotion states, there are general similarities, as well. For example, emotion states like anxiety and fear often are experienced with a sense of “priority” (demand attention), arise and discontinue abruptly (short time course), often operate without awareness early in the emotion generative sequence, and involve change across numerous systems (Ekman and Davidson, 1994). For these types of reasons, it is perhaps not surprising that individuals may experience (phenomenologically) emotional events – such as a fear episode – as personally powerful events that are beyond their control. Yet, at the same time, researchers addressing self and emotion regulation processes have observed that any given emotional event is not simply a pre-programmed sequence that unfolds without possible intervention (Thompson, 1991). Indeed, there are numerous points, theoretically, that an individual experiencing a given emotional state can intervene to shape the nature of the emotional response. This work is characterized by studies on self- and affect-regulation (Gross, 1999).

Three-system perspectives on anxiety and fear. Peter Lang’s multi-system view of emotional states has greatly influenced work on anxiety and its disorders. Indeed, this viewpoint has often been referred to as a “three-system perspective of anxiety and fear states.” The three systems reflected in this model are grossly characterized by physiology, cognition, and behavior (Lang, 1993). The three systems, which are characteristic of all anxiety and related states (e.g., fear, panic, worry, stress), differ in regard to their duration and magnitude of response. Additionally, the channels or response systems of “anxiety states” often are *independent* of one another (Rachman & Lopatka, 1986). As an example, a person who abruptly experiences heart palpitations and feelings of impending doom while in a classroom may not verbally report a panic attack. Yet, she may leave the immediate situation, if possible, and may be more likely to

avoid such situations in the future. In this case, physiological and overt behavioral responses are evident, even though verbal reports of anxiety are not present. Thus, there is *discordance* between response systems (i.e., at one point in time). Additionally, there often is response *desynchrony* (Rachman & Loptaka, 1986), whereby the relation between two response channels responds to treatment at dissimilar rates (i.e., over time). Often, one channel changes first, and the others change more slowly (Lang, 1994).

There are many distinct states that can be categorized under the label of “anxiety.” Although the scope of the present investigation will not permit a detailed description of all such states, these negative emotional experiences overlap considerably. That is, all anxiety states are characterized by the aforementioned three channels or response systems, yet differ in regard to the parameters of response (e.g., duration, magnitude, patterning of systems that are activated) and environmental features (e.g., type of environmental cues associated with the specific form of “anxiety” being studied). Due to these reasons, measurement of anxiety-related states is best achieved using a multimethod approach, wherein all three response channels can be measured and understood in relation to one another (Lang, 1994).

Anxiety responses. Anxiety is a primarily cognitive-affective state characterized by cognitive shifts that focuses attention on *approaching* threat and danger (Craske, 1999). It is thus best conceptualized as a state of “active mobilization and ongoing vigilance” and can be contrasted to that of worry, whereby the individual is in a state of “preparation and readiness.” The future-oriented nature of anxiety for approaching sources of threat typically means that individuals show less dramatic signs of change in physiological systems compared to fearful or panicked states, and greater levels of more elaborate cognitive-based responses (Lang, 1994). Anxiety also tends to be longer in

duration (e.g., lasting for hours at times at the extreme) compared to fear of panic states, which typically lasts on the order of 10 minutes or less (Barlow, 2002; Lang, 1994).

Fear responses. Historically, the term “panic” has roots dating back to Greek mythology, with the mythological character Pan eliciting “sudden fear” in travelers passing his home. Indeed, the term “panicking” as typically used in lay language reflects an understanding of *sudden* fear or distress (Barlow, 2002).

Though there has been debate about the distinctions between fear and panic states, most scholars of emotion currently theorize that these two states are far more similar than different (Craske, 1999; McNally, 1994). For example, both fear and panic states involve active fight-flight-freeze responses and are characterized by high degrees of physiological activation (e.g., rapid heart rate change), threat-oriented behavioral responses (e.g., escape), and low-level (i.e., not elaborative, higher-order) cognition (e.g., “I need to flee this situation now”). Thus, fear and panic states are oriented on current or *imminent* threat (cf. approaching or potential threat; Gray & McNaughton, 1996). Notwithstanding these similarities, one domain where fear and panic sometimes differ is in regard to the identification of the source of the threat (Craske, 1991). Here, research suggests that when an individual experiences a fear state as “out of the blue” (unidentified source threat), they typically refer to this experience as a “panic attack.” In contrast, when a source threat is identified, they are more apt to label the emotional state as “fear” (see Norton, Cox, & Malan, 1992, for a review). For the sake of clarity, from this point forward, the terms fear and panic will be used interchangeably.

Panic-Spectrum Psychopathology: A Brief Overview

Panic attacks are a subjective sense of extreme fear or impending doom accompanied by an autonomic nervous system surge and a strong flight-or-fight action tendency (Barlow, Brown, & Craske, 1994). Recent estimates of uncued (“out of the

blue”) panic attacks, as opposed to cued panic attacks (e.g., panic attacks with a situational trigger) in representative samples suggest that approximately 20% of individuals experience such attacks at one point in their lives and 11.2% in the past 12-months (Kessler, Chiu, Jin, Ruscio, Shear, & Walters, 2006), indicating that panic attacks are a relatively common psychological experience. These findings are generally consistent with earlier investigations using non-representative samples (e.g., Craske, Brown, Meadows, & Barlow, 1995). Many people experience panic attacks without necessarily developing panic disorder (i.e., nonclinical panic attacks; Norton et al., 1992). Typically, individuals who experience nonclinical panic attacks do not experience these attacks as spontaneous or uncued as is generally the case in panic disorder, but rather in stressful or threatening social situations (Norton, Harrison, Hauch, & Rhodes, 1985). Panic attacks can and do occur among those with and without other types of psychopathology (i.e., beyond panic disorder; Bryant & Panasetis, 2005). In fact, the prevalence of cued panic attacks is significantly higher than that of uncued attacks. For example, Craske and colleagues (1995) reported that approximately 60% of their participants reported a lifetime history of a cued panic attack and 40% of this sample reported at least one such attack in the past 3-months. Even when not accompanied by panic disorder, panic attacks, especially those that are uncued, can be associated with increased rates of disability (e.g., job, social, and familial functioning) and role impairment (Kessler et al., 2006). Some studies suggest panic attack onset tends to first occur between the ages of 12-13 years (Hayward et al., 1992; Macaulay & Kleinknecht, 1989; Warren & Zgourides, 1988). However, this age of onset literature should be viewed with caution, as estimates are drawn from non-representative samples of youth. As such, these investigations have captured presumably only a small segment of the overall panic cases, and hence, age of onset data is, by definition, circumspect.

A diagnosis of panic disorder involves both recurrent unexpected panic attacks *and* anxious apprehension about the possibility of experiencing future panic episodes (American Psychiatric Association [APA], 2000). Lifetime estimates of panic disorder without agoraphobia (see definition of agoraphobia below) are 3.7% and 1.1% for panic disorder with agoraphobia (Kessler et al., 2006). Twelve-month estimates for panic disorder (with or without agoraphobia) are approximately 2.8% (Kessler et al., 2006), making panic disorder a relatively common psychiatric disorder. This clinical condition is generally regarded as a disorder of adulthood with a median age of onset of 24 years (Burke, Burke, Regier, & Rae, 1990), although some emerging research has noted that another potential “peak onset period” may occur between 45-54 years (Burke et al., 1990). Panic disorder with and without agoraphobia is associated with a chronic, fluctuating course and high rates of both psychiatric comorbidity and substance use disorders (Zvolensky, Bernstein, Marshall, & Feldner, 2006).

Due to their anxiety about experiencing uncued, and perhaps cued, panic attacks, individuals with panic disorder often avoid potentially threatening situations (Feldner, Zvolensky, & Leen-Feldner, 2004), although not all persons with panic disorder will meet diagnostic criteria for agoraphobia. Agoraphobia often reflects a pattern of behavior characterized by consistent avoidance of threatening situations where a panic attack or high anxiety is perceived to be likely to occur (e.g., limited options to escape) or experiencing marked anxiety-related emotional distress when in such situations. Avoidance behavior can be multifaceted, ranging from certain physical environments to more specific stimuli (e.g., certain substances like caffeine; Rapee, Craske, & Barlow, 1995). Although agoraphobia does not necessarily need to be accompanied by the presence of panic attacks or panic disorder (Fava, Grandi, & Canestrari, 1988), many researchers conceptualize agoraphobia as a complication of (severe) panic disorder

(Barlow, 2002). Agoraphobia with or without panic disorder often is related to higher rates of clinically significant life impairment and severity of the illness (Kessler et al., 2006). The onset of agoraphobia with or without panic disorder is not as firmly established as that of panic attacks and panic disorder, although some research suggests it likely occurs later than typical onset for panic (Lindesay, 1991).

Vulnerability Terminology

Led by the work of Kraemer and colleagues, recent groundbreaking conceptualizations have led to a clearer understanding of various risk processes (Kazdin, Kraemer, Kessler, Kupfer, & Offord, 1997; Kraemer, et al., 1997; Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). Specifically, Kraemer and colleagues have standardized operational definitions for risk processes so that communication about such factors is more clearly and consistently presented across studies. For more comprehensive discussions of the issues involved in risk factor terminology, please see Kraemer, Lowe, and Kupfer, 2005. Please also see Table 1 for a listing of the key terms reviewed in this section of the document.

A *risk factor* is a variable that is related to, and temporally precedes, an unwanted outcome (Kraemer et al., 1997). Although it is perhaps most common for the outcome of interest to be a discrete diagnostic factor (e.g., panic attacks), risk factors also are fully applicable to continuously-defined process variables (e.g., change over time or growth in levels of bodily vigilance). *Causal risk factors* reflect variables that, when modified in some way (e.g., through an intervention), produce systematic change (increase or decrease) in the dependent variable of interest among persons who did not previously manifest such problems (Kraemer et al., 1997). Controlled research designs are necessary to document causal effects because they can serve to rule out other competing alternative explanations (e.g., “third variables”). *Proxy risk factors* are

variables that are related to an outcome of interest, but this association is due to the proxy risk factor's relationship with another causal risk factor (Kraemer et al., 2001). Thus, change in a proxy risk factor would not yield corresponding systematic change in an outcome variable; accordingly, a proxy risk factor may "mark" risk, but not explain or account for such risk.

Due to the theoretical and clinical importance of the ability to change a risk factor, both risk and proxy factors often are further categorized on the basis of whether or not they are *malleable* (i.e., can be changed or altered). When a risk factor cannot be changed, it can be classified as a *fixed marker* (e.g., gender), whereas when it can be changed, it can be classified as a *variable risk factor* (e.g., socioeconomic status; Kraemer et al., 2005). These terms clarify whether a variable that is related to an outcome over time can be changed; if it can be changed, it can be considered a "*risk factor*" and when it cannot, it is better characterized as a "*risk marker*." Both markers and causal risk factors may be important for identifying vulnerable individuals, but only variable causal risk factors will be the ultimate direct target of a clinical intervention.

The above terminology focuses on "main effect" oriented questions (i.e., the singular relation of one variable on another). That is, explicating the nature of an association between a variable and a specified outcome. This step represents only the first in a larger research process, whereby scientists work to identify the nature of the "complex causal chains" involved with any one risk process (Kraemer et al., 2001). Formative next steps in this area of study pertain to understanding mediating and moderating processes, and ultimately, multi-risk factor conceptualizations.

Theoretical Models of Panic Vulnerability

Barlow's (2002) model of the etiology of panic disorder is arguably the most comprehensive given its incorporation of learning processes as they relate to cognitive

and biological factors. The model was developed from observations and findings from clinical practice showing that not everyone who experiences sudden, unexpected physiological changes develops panic disorder, and not everyone who has a panic attack develops this clinical condition (McNally, 1994). These data indicate that there are likely additional vulnerability factors at work beyond the physiological factors and panic attacks, which are the explanatory crux of Barlow's model (see below for further explanation of these additional factors). Barlow (1988, 1991, 2002) postulated that cognitive-affective processing of these physiological factors as dangerous must occur in order to move the experience of harmless symptoms towards the development of panic disorder.

Barlow's (2002) model suggests that the distinguishing element between individuals who have panic attacks, but do and do not go on to develop panic disorder, rests largely on whether they develop *anxious apprehension* about the possibility of experiencing a future panic attack (Bouton, Mineka, & Barlow, 2001). This model begins with the recognition that panic attacks (referred to as a "false alarm" in his model) are a relatively common experience (Norton et al., 1992). Moreover, these attacks can occur in response to any number of aversive life events, including but not limited to acute (Verburg, Griez, Meijer, & Pols, 1995) and chronic (Craske, Poulton, Tsao, & Plotkin, 2001) physical illness, psychosocial stress (Zvolensky, Kotov, Antipova, & Schmidt, 2005), trauma (Bryant & Panasetis, 2001), and various aspects of drug use (Zvolensky, Bernstein, et. al, 2006). There are different generalized tendencies, such as genetic dispositions and temperament styles, to react to such stressors with excessive emotionality and perhaps a panic attack (Kendler et al., 1995; Martin, Jardine, Andrews, & Heath, 1988). Yet, such generalized tendencies do not appear to be, specifically or uniquely, associated with panic disorder unless an individual *also perceives somatic*

events as personally threatening and/or uncontrollable (Bouton et al., 2001). Under these circumstances, an association can develop between a “false alarm” and interoceptive sensations; that is, bodily and other internal cues become classically conditioned stimuli for anxiety and fear states (“learned alarms”). To the extent that bodily cues signal anxiety and fear and a person believes such sensations to be dangerous, avoidance of activities or situations that may trigger such cues can begin to emerge. Such avoidance is believed to occur in various forms (e.g., situational avoidance) *without* (necessarily) a full-blown diagnosis of agoraphobia (Feldner et al., 2004). From this perspective, a panic attack, particularly when unexpected or uncued, is necessary but not sufficient, for developing panic disorder. Thus, understanding the factors that increase the chance of learning that interoceptive cues are dangerous or uncontrollable is a central task. Please see Figure 1 for a schematic of this type of panic model.

Factors Empirically Related to Panic Disorder Vulnerability

A number of factors have been explored as risk factor candidates in the etiology of panic disorder (PD). For example, studies have examined the role of puberty (Hayward et al., 1992), parental modeling (Ehlers, 1993), autonomic inflexibility (Hoehn-Saric, McLeod, & Hipsley, 1995), physical illnesses (Craske et al., 2001), cigarette smoking (Breslau & Klein, 1999), marijuana use (Zvolensky, Bernstein et al., 2006), among others, as risk factors for panic disorder. Yet, of studied variables, anxiety sensitivity (AS) and perceived control over anxiety-related events have emerged as two theoretically-relevant factors with increasingly consistent empirical support. Moreover, unlike some of the other studied risk candidates (e.g., puberty), these two cognitive variables may be more specific to panic vulnerability. That is, these two risk factors do not simply covary with various sorts of psychopathology, but rather, show some

compelling conceptual and empirical specificity to panic problems. These two factors will now be introduced and the extant work related to them will be reviewed.

Anxiety Sensitivity

Background. Perhaps the most well-known cognitive factor related to panic vulnerability is *anxiety sensitivity* (AS; McNally, 2002). AS, defined as the fear of anxiety and anxiety-related sensations (Reiss & McNally, 1985), is a traitlike cognitive predisposition that can theoretically increase the risk of panic-spectrum psychopathology and other types of anxiety problems. The global AS construct encompasses multidimensional fears of anxiety-related physical symptoms, mental incapacitation, and social experiences (Zinbarg, Barlow, & Brown, 1997), all of which can theoretically amplify preexisting states of anxiety (McNally, 2002). For example, if a person believes bodily sensations are a sign of imminent personal harm or threat, this “high AS” individual would presumably experience escalating levels of anxiety when exposed to such internal cues. From this perspective, AS may increase the probability of anxious and fearful responding to internal cues (e.g., bodily sensations) and perhaps be associated with attention to, and avoidance of, threatening stimuli (Zvolensky & Forsyth, 2002).

Associations with panic-relevant processes. There is consistent evidence that AS is related to panic-spectrum psychopathology. One notable aspect of this scientific literature is that it is comprised of both cross-sectional and laboratory tests that have utilized a diverse array of methodological approaches and assessment modalities. In regard to cross-sectional tests, for example, there is consistent evidence that AS measured pre-exposure to biological challenge (panic provocation) is a significant predictor of post-challenge anxiety symptoms and panic attacks among nonclinical individuals (McNally & Eke, 1996). These effects are above and beyond indices of the

tendency to experience negative emotional symptoms (e.g., trait anxiety, negative affectivity), tend to be specific to self-reported distress (cf. psychophysiological responding), and are apparent from adolescence through adulthood (Leen-Feldner, Feldner, Bernstein, McCormick, & Zvolensky, 2005; Rabian, Embry, & McIntyre, 1999; Schmidt, 1999; Zvolensky, Feldner, Eifert, & Stewart, 2001). The size of the observed effects in such investigations have ranged from statistically small to large depending on the type of dependent measure employed using Cohen's (1988) metric (Zvolensky & Eifert, 2000). Other cross-sectional tests, although completed outside of the laboratory and reliant on self-report instruments, suggest that AS effects are similarly apparent in real-world settings and across a range of cultural groups (Zvolensky, Arrindell et al., 2003).

Prospective investigations similarly suggest that AS predicts panic attacks. In this domain, there have been studies that focus both on adolescents (Hayward, Killen, Kraemer, & Taylor, 2000; Weems, Hayward, Killen, & Taylor, 2002) and adults (Schmidt, Lerew, & Jackson, 1997, 1999; Schmidt & Lerew, 2002). To illustrate, Schmidt et al. (1997) examined approximately 1,100 air force cadets during basic training. The authors designed the study to examine prospectively whether AS is associated with the development of psychopathology when the cadets are under periods of stress. The authors hypothesized that AS would act as a cognitive diathesis in regard to increasing risk for the development of psychopathology, and panic in particular. Results indicated that AS predicted the development of panic attacks. Additionally, the Anxiety Sensitivity Index (ASI; Reiss, Peterson, Gursky, & McNally, 1986), a well-established measure of AS, could predict panic attacks above and beyond the effects accounted for by trait anxiety. The analyses concerning psychopathology, including panic disorder, were not possible because too few people showed a change in clinical status during the follow-up

assessment. This investigation by Schmidt and colleagues illustrates how AS can function as a cognitive diathesis for panic attacks within a diathesis-stress model for panic. Other investigations have replicated the above findings, testifying the utility of AS in predicting panic attacks (Hayward et al., 2000; Schmidt et al., 1999; Weems et al., 2002). However, these studies have only determined that AS is predictive of panic attacks, but not necessarily the *development* of panic-spectrum psychopathology. To date, only two investigations have established AS as a cognitive diathesis for the development panic attacks and panic disorder.

One such study by Maller and Reiss (1992) examined AS as a cognitive risk factor for panic attacks and panic disorder among a college student population ($n = 48$). Specifically, the authors hypothesized that participants with high AS (*mean* AS level = 32.6) would be at a greater risk of developing panic attacks and/or panic disorder over a period of three years following the initial investigation, in comparison to participants with low AS (*mean* AS level = 11.4). Consistent with hypotheses, Maller and Reiss found that participants with high AS were 5 times more likely to develop an anxiety disorder in the three year period, than those participants with low AS. The study was not able to evaluate explanatory specificity (i.e., AS predicting panic psychopathology compared to other anxiety disorders) due to the small sample size employed. An additional limitation of Maller and Reiss' study was that they did not assess the presence of past or current psychological disorders at the time of their initial investigation. Thus, it is not known if pre-morbid AS level was uniquely related to future psychopathology status.

More recently, Schmidt, Zvolensky, and Maner (2006) designed a prospective study to examine AS as a cognitive risk factor for the development of panic psychopathology among young adults ($n = 404$) over a 24 month period. However, Schmidt and colleagues took into consideration the limitations of the study by Maller and

Reiss, and thus, excluded individuals with a history of Axis-I psychopathology at the baseline assessment. Findings indicated that AS predicted the development of panic, anxiety disorders, and all Axis-I diagnoses (e.g., alcohol use and mood disorders) above and beyond baseline levels of trait anxiety. Specifically, those with high AS were at a 2.5 time greater risk of developing panic attacks and were at a 2 time greater risk of a diagnosis of an anxiety disorder or any Axis-I disorder. These findings by Schmidt and colleagues - paired with the results of Maller and Reiss - create a foundation of empirical evidence supporting the relation between AS and the development of clinically diagnosable anxiety conditions such as panic attacks and panic disorder. Though an important extension of the Maller and Reiss (1992) investigation, Schmidt, Zvolensky et al. (2006) did not provide compelling evidence of explanatory specificity of AS for panic disorder. These data should be therefore viewed with caution at the present time because the overall rates of disorder development across the three- year assessment were minimal, potentially washing out specificity effects due to a truncated range of upper-end variability in the dependent measures of interest.

Malleability. Given the above findings, a next critical step in understanding the relevance of AS to panic vulnerability is to determine to what extent this cognitive factor can change or be changed (malleability). Theoretically, models of AS predict that this construct can be altered via intervention (McNally, 2002). Researchers have theorized that learning to alter cognitive processes and beliefs about the perceived negative consequences of anxiety symptoms can be achieved through cognitive (e.g., thought restructuring) and behavioral tactics (e.g., interoceptive exposure; McNally, 1990). In support of this theoretical viewpoint, there is empirical evidence that AS is indeed malleable (see Otto & Reilly-Harrington, 1999, for a review). As one illustrative example, Telch and colleagues (1993) demonstrated that an 8-week group cognitive-behavioral

treatment for panic disorder, consisting of twelve 90-minute sessions, effectively reduced AS levels compared to a control condition. Specifically, the treatment group reported a decrease from a mean of 33.7 on the 16-item Anxiety Sensitivity Index (ASI; Reiss et al., 1986) at pre-treatment to 13.9 at the post-treatment assessment, and the reduced AS levels were maintained at a 6-month follow-up assessment; statistically and clinically significant change demarcating movement from a clinical level to a normative level. The control group reported no change in AS (ASI scores of 34.4 and 32.0 at the pre- and post-treatment assessments, respectively). Similar effects have been reported in panic interventions delivered via an individual format as well as in brief formats (i.e., four 60-minute sessions; Barlow, Craske, Cerny, & Klosko, 1989; Schmidt & Woolaway-Bickel, 2000; Westling & Ost, 1999). One study suggests AS may mediate treatment outcome for panic disorder (Smits, Powers, Cho, & Telch, 2004), but independent replication of such findings is an important next research task. Though there are very limited data on the ability to change AS among nonclinical individuals, extant treatment work on clinical populations suggests that this may be possible (Otto & Reilly-Harrington, 1999).

Summary of AS findings. In summary, research on AS suggests that it may currently be best characterized as a variable risk factor for panic-spectrum problems. Specifically, research provides evidence regarding temporal order and relations with panic-spectrum problems based on prospective, laboratory, and cross-sectional field research, as well as evidence regarding construct malleability based on intervention research among clinical samples. Yet, evidence that indicates changing this factor will alter the risk of panic-spectrum psychopathology from a preventative standpoint is lacking. Thus, it is not clear if AS represents a variable marker or variable causal risk factor for panic-spectrum psychopathology. To determine whether AS is a variable marker or variable causal risk factor it is important for future research to examine

changes in the construct prospectively following experimental (prevention-oriented) manipulation (e.g., intervention targeting AS compared to a control condition). Moreover, one notable limitation of past work in this domain is that studies have not expressly explored whether AS may work in a complimentary fashion with other cognitive (and perhaps related) risk factors. That is, studies have principally focused on “main effect” tests rather than interactive models. At some level, this aspect of such work is a natural reflection of the current developmental stage of scientific study in this domain. On the other hand, it is unlikely that AS (or any risk factor) works to confer risk for psychopathology. Accordingly, future research may benefit by exploring AS in relation to other putative risk factors for panic psychopathology in one overarching model.

Perceived Controllability Over Anxiety-Related Events

Background. A second cognitive factor postulated to be important to the pathogenesis of panic psychopathology is perceived control (defined as the degree to which one believes that they can control the onset, offset, or duration of an aversive, or perhaps, appetitive event; Zvolensky, Lejuez, & Eifert, 2000) over anxiety-related events. The study of control, defined in the most general sense, is highly complex, and even from some vantage points, controversial for a variety of methodological and theoretical reasons (see Zvolensky, Lejuez et al., 2000, for an expanded discussion). As one illustrative example, most scholars have suggested that control over some event exerts unique effects on emotional responding without recognizing that one (human or non-human animal) cannot have control without also having predictability over the same event. That is, there is a natural confounding of elements of prediction and control (Mineka, 1985; Zvolensky, Lejuez et al., 2000). Due to the lack of recognition of this and other complex factors related to the construct of control, various popular scientific literatures on this topic (e.g., health hardiness literature, perceived stress literature, locus

of control literature, hopelessness theories of depression literature) have been roundly criticized from a scientific point of view (Mineka, 1985; Zvolensky, Lejuez et al., 2000). At the same time, such critical attention has fostered a new appreciation for the study of control versus prediction, and this has led to new methodological advancements and insights into the nature of emotional (and behavioral) functioning. For example, specific laboratory procedures have been developed to equate for predictability when studying control and to remove the effects of control when studying predictability (Lejuez, Eifert, Zvolensky, & Richards, 2000).

In the study of anxiety and its disorders, there have been two principal approaches to studying ‘control processes.’ One can be loosely labeled as “experimental” in nature and involves random assignment and the manipulation of control (equating for predictability) over some internal or external event and tracking responses across systems as a function of that manipulation. This approach is by far the most powerful from an explanatory perspective: there is a license to engage in causal-oriented hypothesis testing in the immediate situation (Forsyth & Zvolensky, 2002). From a historical standpoint, this approach is typically used in studies of non-human animals, and to a large extent, has driven applied theorizing on the nature and effects of control on psychological functioning (see Mineka, 1985, for a review). While powerful, some argue that the challenge to this approach is that predictions are largely restricted to the immediate (laboratory) context, and hence, the ability to make generalizations beyond such a context can be questioned. Among human participants, there is the additional challenge that repeated sessions (or learning trials) rarely can be completed. The second approach is to assume – theoretically - that an individual can report on his/her ability to perceive certain specified events as within his/her control to varying degrees and evaluate relations with such responding to indices of psychological functioning. This

approach is, by definition, correlational in nature and therefore suffers from the well-known challenges inherent to such work (e.g., cannot explicate source of the effect in a fully unambiguous manner, as there is no random assignment, manipulation, or control over alternative third factors). The strength of this latter approach is that it allows researchers to evaluate the effects of a history of control, albeit confounding predictability, on dependent measures of interest, and from this perspective, bridges basic research on control to applied questions in an arguably more meaningful fashion.

Associations with panic-relevant processes: Experimental work. In one of the first tests of control processes as it relates to anxiety-related responding to bodily sensations, Sanderson, Rapee, and Barlow (1989) employed a biological challenge paradigm. Specifically, Sanderson and colleagues (1989) designed a CO₂-challenge study in which participants with panic disorder with agoraphobia ($n = 20$) were administered 5.5% CO₂-enriched air for fifteen minutes (following a five minute administration of compressed air). Participants were instructed that they would be able to adjust the CO₂ administration through the use of a dial, only when a light was illuminated (which was illuminated for the full fifteen minutes of gas administration for only 10 participants). However, they were encouraged to make adjustments only if absolutely necessary. Participants were not aware that their turning the dial would, in fact, not alter the gas administration. Results indicated that those individuals who could not control the CO₂ administration (e.g., the light did not illuminate) were more likely to report a greater number of panic symptoms, rated those symptoms as more intense, reported a greater number of catastrophic cognitions, and reported a greater number of panic attacks (80% of participants without control reported a panic attack during the challenge, whereas 20% of those participants with control reported an attack). The authors concluded that the illusion of control in *patients with panic disorder* may reduce the likelihood of experiencing a panic attack.

This work highlights the potential importance of perceived control among those with panic disorder, but does not permit conclusions about the possible etiological role of control.

To bridge work on perceived control and panic disorder etiology, Zvolensky, Lejuez, and Eifert (1998) examined whether a lack of control during repeated 20% CO₂ – enriched air administrations would influence self-reported anxiety, as well as measurements of physiological response to the gas administration. Nonclinical participants ($n = 30$) who were high in suffocation fear (a characteristic common in panic disorder) were randomized to a group in which offset control was permitted, or to a group in which control was not permitted. Results indicated that a lack of control over CO₂ administrations increased anxious responding. There were no physiological differences detected. These results conceptually replicate those of Sanderson and colleagues (1989) and suggest that perceived control may be applicable to nonclinical populations, and hence, theoretically relevant to the etiology of panic psychopathology (i.e., not solely attributable to panic disorder status).

In a subsequent investigation, Zvolensky, Eifert, Lejuez, and McNeil (1999) examined the effects of offset control (the ability to terminate an aversive event or stimulus) over 20% CO₂-enriched air on anxious responding. However, in addition to an examination of a lack of control, participants also experienced a loss of control (i.e., individuals with offset control in phase I no longer had control in phase II administrations of CO₂). Participants consisted of undergraduate students ($n = 30$) with moderate to high AS, but no history of psychopathology. Results indicated that those who lacked or lost control were more likely to experience anxious responding to the CO₂ administrations than those who had control or gained control. These results were not attributable to baseline anxiety. No effects were evident for physiological indices of autonomic arousal.

These findings add to the experimental literature suggesting that control over anxiety-related events (bodily cues) is meaningfully related to increased risk of anxious and fearful responding to interoceptive cues.

Associations with panic-relevant processes: Correlational work. One of the foremost obstacles to studying perceived control is specificity in relation to the “controlled stimulus.” That is, actual or perceived control needs to be examined in relation to a specific stimulus or set of stimuli, an issue, while somewhat intuitive, that has not actually been detailed or addressed in assessment measures for work translating experimental/laboratory research to non-laboratory settings. For example, sample items on perceived control instruments ask respondents to specify how much control they believe they have over their lives, but what aspect of their life is not explicated (e.g., sample item “How well I cope with difficult situations depends on whether I have outside help” does not specifically specify what situation within the person’s life is of relevance, and to a large extent, blurs the object of control.). This type of approach runs theoretically in contrast to our understanding of self-regulation processes, whereby behavioral responses are coordinated towards, or acted on, specific types of stimuli or events. With this interpretative background, we now turn to a discussion of perceived control over anxiety-related events, an area of study most central to the present investigation.

Correlational work relevant to anxiety and its disorders surrounding the construct of perceived control first began (from a contemporary perspective) with the studies aimed at developing the measure, the *Anxiety Control Questionnaire* (ACQ). Here, Rapee, Craske, Brown, and Barlow (1996) developed the ACQ to capture one’s perception of control over internal responses and external events. The first of five studies sought to develop the items within the measure. Fifty-three items were

administered to a group of outpatients ($n = 250$) from an anxiety disorders clinic. Following numerous exclusions of unnecessary items (as determined by inter-item correlations), the measure was reduced to 30 items (16 external event factors, and 14 internal event factors, to create one global factor) which exemplified strong internal consistency. The second study aimed to examine the internal consistency within a group of nonclinical participants ($n = 236$). Results were consistent with that of study one. Study three aimed to examine the stability and test-retest reliability of the measure. Sixty-nine undergraduate students completed the ACQ. Results indicated strong test-retest reliability for the ACQ as a whole and each of its subscales over a time period of one month. Study four examined the convergent and discriminant validity of the measure with 353 participants (with varying or no psychological diagnoses). The study revealed that that ACQ had good convergent (it was strongly correlated with other measures of anxiety and control) and discriminant validity. Finally, study five aimed to examine the malleability of the ACQ pre- and post-treatment within an anxiety clinic ($n = 19$ individuals with panic disorder with agoraphobia). Results indicated that total scores on the ACQ would increase (e.g., perception of control would improve) from pre- to post-treatment. In total, these studies determined that the ACQ exhibits good reliability and validity, and it is a malleable construct. Therefore, it is possible perceived control over anxiety-related events could be changed as a result of treatment.

There is very little empirical work on the ACQ. In one relevant investigation, Zvolensky and colleagues (2001) examined perceived control over anxiety-related events in terms of the prediction of panic-relevant interpretive biases for threat (automatic judgments about ambiguous information that vary in their threat relevance). Findings revealed that the less control one perceived to have over anxiety-related events, the greater the internal and external interpretative biases among nonclinical

participants. Such effects were not attributable to panic attacks, state anxiety, and did not vary by gender. These data suggest that there is merit in better understanding perceived control over anxiety-related events in relation to panic-relevant processes.

Subsequent factor analytic work on the ACQ suggests that the original factor solution attained by Rapee et al. (1996) may not be fully accurate. For example, Zebb and Moore (1998) found that the ACQ maintained a 3-factor solution. In a more rigorous study, Brown, White, Forsyth, and Barlow (2004) found a hierarchical model for perceived control over anxiety-related events using the ACQ. In this investigation, clinical participants ($n = 1500$) were recruited from a large anxiety and mood disorders clinic. Participants were randomly divided into three samples in order to replicate the factor structure of the ACQ. Results indicated that the latent structure of the ACQ differed from that observed in earlier work and that 15-items performed poorly and were removed from the “next iteration” of the instrument. On this revised 15-item ACQ instrument, there was a three-factor hierarchical solution: emotional control, threat control, and stress control [lower-order factors] that load on to a higher-order global perceived control over anxiety-related events construct. [Note. Please see the ACQ description in the Method Section for further information about the psychometric properties of this assessment tool.]

Malleability. Theoretically, perceived control over anxiety-related events is modifiable, and hence, could conceptually be studied as a risk factor. However, there are no independently replicable tests of this matter, and as a result, conclusions regarding this facet of risk factor nomenclature is premature.

Summary of perceived control findings. In summary, research on perceived control thus far suggests that it can be characterized as a variable risk factor or fixed marker for panic-spectrum problems. Specifically, there is evidence through both

experimental and correlational studies that perceived control is predictive of anxiety symptoms and may hold special theoretical relevance to panic-spectrum problems (Barlow, 2002). Although there is laboratory work suggesting this factor can be experimentally manipulated, there are currently no direct data that address temporal time course or malleability issues over time.

Anxiety Sensitivity and Perceived Control: Integrative Approaches

Background. Together, there is a clear theoretical basis and varying degrees of empirical support for the potential role of AS and perceived control over anxiety-related events as risk factor candidates for panic psychopathology. These cognitive factors can be considered “specific” in the sense that they do not uniformly covary with all types of psychopathology or show the same relations to even all anxiety disorders. Thus, unlike variables such as negative affectivity, neuroticism, behavioral inhibition, and behavioral inhibition sensitivity, scholars have begun to conceptualize these as “specific cognitive factors” relevant to panic psychopathology (Leen-Feldner, Zvolensky, & Feldner, 2004). As reviewed earlier, work on AS and control over anxiety-related events has almost exclusively been focused on explicating the main effects in relation to various panic-relevant processes. This is normative for this stage of research development, but represents only a relatively early facet of study from a larger perspective. It is important to extend such “main effect” work to more integrative types of tests. In fact, there have been some initial attempts to explore the interactive processes between AS and control over anxiety-related events. This work will now be reviewed in detail and concluded with a summary of knowledge thus far attained as well as its interpretative caveats. A conceptual model for the present investigation will then be offered.

Integrative empirical studies. In the earliest study in this domain, Telch, Silverman, and Schmidt (1996) examined the effects of AS and perceived control on

anxious responding to a biological challenge using caffeine as the panicogenic agent. The authors designed a study in which participants would either have control over the effects of caffeine through taking a caffeine antidote (control over the challenge), or not being offered an antidote (no control over the challenge). Telch and colleagues (1996) hypothesized that those individuals high in AS would be more likely to exhibit high levels of anxious responding to the challenge. However, the authors postulated that low perceived control would interact with AS, such that those individuals who have low control and high AS would experience more of an anxious response to the caffeine. Nonclinical participants ($n = 72$) high and low in AS were randomly assigned to either the perceived control condition or to the no control condition. Results were consistent with the hypothesis in that the effects of perceived control on anxious responding were found only in those individuals with high AS. That is, control over the caffeine challenge interacted with AS in terms of anxiety response to the provocation. This work provides an initial empirical basis to further examine the interactive processes between these two cognitive risk factor candidates for panic psychopathology.

In another investigation, offset control over eight 20% CO₂ administrations was experimentally manipulated in a large nonclinical population ($n = 96$) varying in AS (high or low) and gender (Zvolensky, Eifert, & Lejuez, 2001). High AS participants who lacked offset control reported significantly greater elevations in self-reported anxiety, emotional displeasure, arousal, and dyscontrol relative to their yoked counterparts with offset control. In contrast, low AS individuals responded with similar levels of cognitive and affective distress regardless of the offset control manipulation. Although the provocation procedure reliably produced bodily arousal relative to baseline, at a physiological level of analysis, no significant differences emerged across conditions. These results

conceptually replicate those of Telch et al. (1996) and extend such work to a novel biological challenge procedure.

Outside of laboratory studies, there have been two efforts to study AS and perceived control over anxiety-related events in relation to panic processes in one overarching model. In one investigation, Schmidt and Lerew (2002) examined the independent and interactive effects of perceived control, predictability, and AS while participants were going through military basic training, which is a highly stressful 5-week period of time. Participants were 1,139 Air Force Academy cadets enduring five weeks of basic training. The cadets were administered a battery of measures (including the Anxiety Sensitivity Index and a four item, non-empirically validated measure assessing predictability and perceived control over the basic training experience) during the first few days of training (Time 1), 2 weeks into the training (Time 2) and at the end of training (Time 3). Results indicated that AS interacted with perceived control for military training in the future prediction of anxiety symptoms. No such effects were apparent for perceived control or a triple interaction between all three cognitive factors (i.e., perceived control, predictability, and AS). These findings are conceptually in accord with past laboratory work, but are limited in the technologies employed (i.e., non-empirically grounded assessment tools).

In a final relevant investigation, White and colleagues (2006) examined perceived control over anxiety-related events, using the 15-item ACQ, and AS (as assessed by the 16-item ASI, Reiss et al, 1986) in the prediction of agoraphobia in patients diagnosed with panic disorder. Participants were outpatients at a large anxiety disorders clinic, diagnosed with panic disorder with or without agoraphobia ($n = 229$ panic disorder with agoraphobia, $n = 8$ panic disorder without agoraphobia). Results revealed that patients who had higher AS and lower perceptions of control, were more likely to exhibit

agoraphobic avoidance. However, when examining the interaction, results were consistent with hypotheses only when examining perceived control over external events, and not when examining perceived control over internal sensations. The authors suggested that these results may be due to specificity between external threats and agoraphobia, but also potentially due to the ACQ-Emotion Control scale not assessing the internal sensations that are fearful to individuals with panic disorder.

Conclusions, interpretative caveats, and theoretical synthesis regarding integrative tests of AS and control over-anxiety-related events. Extant work on the interactive effects of AS and perceived control over anxiety-related events is promising. All investigations conducted to date, which vary in approach and methodology, suggest that AS may interact with perceived control over anxiety-related events to predict anxiety symptoms or panic-relevant processes. At the same time, this work is limited in at least three notable respects. First, the two laboratory studies on this topic have arbitrarily divided participants in terms of AS, as a function of high versus low status. This approach can statistically inflate effect sizes as top and lower-end variability is only addressed (Cohen & Cohen, 1983), and therefore, somewhat biased conclusions may have been drawn from such work (i.e., variability between the extremes is not studied). Thus, future work should employ a continuous index of AS, which is more consistent with theoretical models of the construct (McNally, 2002). Second, only one of the two non-laboratory studies used empirically derived scales of perceived control over anxiety-related events and therefore conclusions about perceived control processes using the ACQ are highly limited. Moreover, of the relevant work, the White et al. (2006) investigation focused on a clinical population, leaving conclusions about the possible etiological role unclear (i.e., one cannot study etiologic processes by examining clinical participants due to a myriad confounding factors inherent to psychopathology; Forsyth &

Zvolensky, 2002). Thus, future work is needed to conceptually replicate and extend such work to panic-relevant processes among a nonclinical population. Third, the range of dependent measures employed to date has been limited and not fully in accord with panic-relevant processes. For example, studies have not consistently studied cornerstone features of panic vulnerability like anxiety focused on bodily sensations, intensity of panic symptoms, and avoidance. Accordingly, future study is needed to examine the interactive effects of AS and perceived control over anxiety-related events in a more comprehensive manner; one that tracks criterion variables along cognitive, physiological, and behavioral domains.

Theoretically, perceived control over anxiety-related events may impact or alter the panicogenic effects of AS in regard to panic-relevant processes. As reviewed above, there is a large theoretical and growing (human) empirical knowledge base that suggests lower levels of perceived control over anxiety-related events are central to panic vulnerability. Drawing from available work, perceived control over anxiety-related events may influence the strength of the AS-panic psychopathology relationship. Specifically, AS is likely to be most strongly related to panic-relevant processes when it is coupled with lower levels of perceived control over anxiety-related events (see Figure 2). The underlying rationale for this type of approach is that to the extent an individual who fears the negative consequences of anxiety-related sensations also perceives them to be relatively more uncontrollable, the more likely he/she would be to respond in a panicogenic fashion. For example, an individual may be more likely to experience greater levels of anxiety focused on bodily sensations and more intense panic attack symptoms. To the extent this experience is, in fact, more emotionally distressing, this same person would perhaps be more apt to want to avoid it in the future (proxy for panic-relevant avoidance). Alternatively, if a high AS person maintains a higher level of

perceived control over anxiety-related sensations, then, he/she may be at a diminished risk for panic-relevant responding. That is, perception of control may provide an adequate psychological resource to tolerate or cope with the stressor. Thus, this individual should theoretically be less likely to experience intense panic symptoms and so on. To a large extent, this type of perspective represents a dynamic model, whereby AS represents a psychological diathesis for panic that is affected by self-regulation resources (represented by perceived control over anxiety-related events).

Present Study

Together, the overarching aim of the present investigation was to explore the main and interactive relationship between AS and perceived control over anxiety-related events in the context of a biological challenge. The research design employed was a cross-sectional, group-based (correlational) design (Kazdin, 2003). There were three interrelated and convergent sets of research hypotheses. First, in regard to self-reported anxiety effects, it was hypothesized that AS and perceived control over anxiety-related events would demonstrate main and interactive effects in terms of predicting post-challenge anxiety focused on bodily sensations and intensity of panic attack symptoms (cognitive response indices of anxiety collected immediately following the biological challenge). Second, it was hypothesized that the AS and perceived control would demonstrate main and interactive effects for avoidance of future challenge participation (as measured by a face valid indicator of willingness to participate in future challenge protocols). Finally, although physiological effects across indices of autonomic arousal were assessed, no effects were expected for these factors as past work has not supported a cognitive vulnerability and physiological response effect (Zvolensky & Eifert, 2000). Overall, these significant interaction effects were expected to be apparent above

and beyond the respective main effects as well as pre-challenge anticipatory anxiety and gender.

Method

Participants

The sample consisted of 229 participants ($m_{age} = 21.02$, $SD = 7.55$, 124 females) who were recruited from the greater Burlington, Vermont community. Participants were recruited through the general community and university communities via newspaper ads and flyers advertising a laboratory study on ‘emotion.’ Overall, 92.6% of the sample was Caucasian, 1.7% was Hispanic, 1.3% was biracial, .9% was Asian, .9% identified themselves as “other,” and .4% was black. 2.2% of participants chose not to specify their race. In terms of highest level of education completed, .4% did not graduate from high school, 79.9% graduated from high school, 13.5% reported partial college education, 1.3% graduated from a 2-year college, 2.2% graduated from a 4-year college, .9% reported partial graduate education, .9% reported some graduate degree, and .9% chose not to specify their education level. Relevant demographics pertaining to substance use were collected. Within the sample, 41.5% of participants identified themselves as smokers; smoking an average 11.05 ($SD = 7.36$) cigarettes per day. Of those participants who identified themselves as alcohol drinkers, they stated they drank an average 5.29 drinks per occasion ($SD = 3.47$) and drank an average 1.90 days per week ($SD = 1.39$). 57.2% of participants reported having used marijuana in the past 30 days.

Exclusionary criteria for the investigation included: (1) current axis I psychopathology; (2) current use of psychotropic medication; (3) current suicidality or homicidality; (4) current or past chronic cardiopulmonary illness (e.g., chronic obstructive pulmonary disease; severe asthma), (5) current, acute respiratory illness (e.g.,

bronchitis), (6) seizure disorder, cardiac dysfunction, or other serious medical illness (e.g., history of seizures, emphysema); (7) pregnancy (specific to females); and (8) limited mental competency, inability to give informed, written consent. As in past work, these screening criteria were employed to increase the study's internal validity (i.e., panic-relevant responding related to AS or perceived control over anxiety-related events is not alternatively explained by co-occurring psychopathology also related to AS; Forsyth & Zvolensky, 2002); and to protect participants by decreasing the probability of unanticipated medical complications resulting from CO₂ inhalation (Zvolensky & Eifert, 2000). Psychiatric history and psychotropic medication usage were measured by the screening version of the Structured Clinical Interview-Non-Patient Version for DSM-IV (SCID-NP; First, Spitzer, Gibbon, & Williams, 1995). Medical exclusionary criteria were assessed within the context of the SCID interview using a supplemental set of (standardized) interview-based medical screening questions. This screening approach has been successfully used in past biological challenge work (e.g., Zvolensky, Leen-Feldner et al., 2004). Inter-rater reliability in our laboratory has been high for Axis I diagnoses (e.g., Zvolensky, Leen-Feldner et al., 2004). In the present study, each SCID was reviewed by a graduate-level doctoral student to ensure inter-rater agreement. No disagreements regarding inclusion/exclusion were observed.

Measures

Pre-Challenge Measures

Structured Clinical Interview for DSM-IV Axis I Disorders- Non-Patient Edition (SCID-NP). The SCID-NP screening interview (First, Spitzer, Gibbon, & Williams 1994) is a well-established diagnostic interview for psychiatric conditions as outlined in the *Diagnostic and Statistical Manual, Fourth Edition-Text Revision* (DSM-IV-TR; APA, 2000). The SCID-NP assesses Axis I disorders and panic attacks as well as substance

use behavior (e.g., smoking status and rate). The SCID-NP (non-patient) screener was used in this study given that participants are not identified as having a psychiatric disorder. The SCID-NP screener has been demonstrated to have adequate reliability and validity (Spitzer et al., 1994).

Smoking History Questionnaire (SHQ). Smoking history and pattern was assessed with the SHQ (Brown, Lejuez, Kahler, & Strong, 2002), a measure that includes items pertaining to smoking rate, age of onset of initiation, and years of being a daily smoker. The SHQ has successfully been used in previous studies as a measure of smoking history (Zvolensky, Lejuez, Kahler, & Brown, 2004).

Alcohol Use Disorders Identification Test (AUDIT). The AUDIT is a 10-item screening measure developed by the World Health Organization to identify individuals with alcohol problems (Babor, de la Fuente, Saunders, & Grant, 1992). There is a large body of literature attesting to the validity of the AUDIT (Saunders, Aasland, Babor, de la Fuente, & Grant, 1993). For this study, the AUDIT was used to measure alcohol consumption behavior.

Marijuana Smoking History Questionnaire (MSHQ). The MSHQ (Bonn-Miller & Zvolensky, 2005) assesses marijuana smoking use history and pattern. The MSHQ is a self-report instrument that includes items pertaining to marijuana smoking rate (frequency of use in lifetime and past 30 days), age of onset at initiation, years of being a regular marijuana smoker, and other descriptive information (e.g., number of attempts to discontinue using marijuana). The MSHQ has been employed successfully in past research (Bonn-Miller, Zvolensky, Leen-Feldner, Feldner, & Yartz, 2005).

Anxiety Sensitivity Index (ASI). The ASI (Reiss, Peterson, Gursky, & McNally, 1986) is a 16-item measure in which respondents indicate on a 5-point Likert-type scale (0 = "very little" to 4 = "very much") the degree to which they are concerned about

possible negative consequences of anxiety symptoms (e.g. “It scares me when I feel shaky”). Factor analyses of the scale indicate that it has a hierarchical structure, with three first-order factors labeled AS-Physical Concerns, AS-Mental Incapacitation Concerns, and AS-Social Concerns as well as a single, higher order general factor (Zinbarg, Barlow, & Brown, 1997). The ASI has high levels of internal consistency for the global score (range of alpha coefficients: 0.79 to 0.90) and good test-retest reliability ($r = .70$ for 3 years; Peterson & Reiss, 1992). The ASI is unique from, and demonstrates incremental validity relative to, trait anxiety (Rapee & Medoro, 1994) and negative affectivity (Zvolensky, Kotov, Antipova, & Schmidt, 2005). In the present investigation, the total ASI score was utilized, as it represents the global-order anxiety sensitivity factor and therefore takes into consideration different types of fears, including fears of panic-related somatic, cognitive, and social cues.

Anxiety Control Questionnaire (ACQ). The Anxiety Control Questionnaire (ACQ; Rapee, Craske, Brown, & Barlow, 1996) was used to measure perceptions of control for anxiety-related events. The ACQ was initially designed to index perceived control over internal and external events/situations that are relevant to anxiety-related problems. Participants indicate their level of agreement on a 6-point Likert-type scale (0 = *strongly disagree* to 5 = *strongly agree*) for control-oriented beliefs (e.g., “When I am put under stress, I am likely to lose control”). Although the original ACQ development study found the measure to be comprised of two factors (Rapee et al., 1996), subsequent work has not fully supported these earlier results for a variety of methodological reasons (Brown, White, Forsyth, & Barlow, 2004; Zebb & Moore, 1999). Brown and colleagues (2004) recently found a three-factor lower-order solution (Emotion Control, Threat Control, and Stress Control) that loaded on a single 15-item higher-order factor (global Perceived Control). This work, in turn, resulted in a 15-item revised version of the ACQ (i.e., certain

items were removed from the original version of the instrument due to problematic factor loadings). In the present investigation, the revised global ACQ score was utilized to index a generalized perception of control for anxiety-related events. This decision was based on two considerations. First, previous work has not fully substantiated the lower-order ACQ factors as clinically and theoretically meaningful (Brown et al., 2004), and second, the generalized factor is most consistent with contemporary theoretical models of panic vulnerability (Barlow, 2002).

Challenge Measures

Diagnostic Sensations Questionnaire (DSQ). The DSQ (Sanderson, Rapee, & Barlow, 1988, 1989) was used to assess DSM-IV panic attack symptoms immediately following the biological challenge. This measure is frequently employed in challenge work (Zvolensky, Lejuez, & Eifert, 1998). Ratings for the DSQ are made on a 9-point Likert type scale (0 = *not at all* to 8 = *very strongly felt*). The DSQ, specifically, lists DSM-IV panic symptoms and yields composite scores for a mean intensity level for cognitive (e.g., fear of going crazy) and physical (e.g., breathlessness or smothering sensations) symptoms.

Subjective Units of Distress Scale (SUDS). The SUDS (Wolpe, 1958) will be used to index self-reported anxiety. This Likert-type scale ranges from 0 (*no anxiety*) to 100 (*extreme anxiety*) in subjective ratings of anxiety. Participants completed these scales before the challenge procedure (as an index of anticipatory anxiety) and immediately after the challenge (as an index of maximal postchallenge anxiety).

Behavioral Avoidance. In order to index behavioral avoidance post-challenge, participants' willingness to participate in another CO₂ administration will be evaluated by a paper-and-pencil questionnaire at the end of the recovery period. This item asked participants to rate their *level* of willingness to participate in another CO₂ administration

study. Specifically, at the end of the recovery phase, participants were told that other CO₂ studies will be recruiting individuals for participation within the next 2 weeks via a written statement on the questionnaire. Then, participants were asked to indicate their willingness on a 100-point Likert-style questionnaire intended to assess participants' interest in returning for another CO₂ investigation (*0 = no desire to participate; 100 = definite desire to participate*). This type of index has been utilized successfully in the past with biological challenge paradigms and such work has shown that this measure is related to avoidance due to fear and is not correlated with boredom or other related emotional states such as frustration (Eifert & Heffner, 2003).

Materials and Apparatus

Laboratory sessions were conducted in a 3-meter X 3-meter experimental room in the Department of Psychology at the University of Vermont. Participants sat at a desk supporting a Dell Pentium computer with color monitor, which will be turned off during the entire duration of the procedure. After completing physiological hookup and providing experimental instructions (see *Procedure* for details), the experimenter ran and observed study participants from an adjacent control room containing an apparatus designed to provide participants with either room air or a mixture of 10% carbon dioxide-enriched air.

Carbon dioxide was stored in a 24-inch diameter hospital grade latex bag and delivered via 5-centimeter tubing to a positive-pressure C-pap mask worn by the participant. In addition to a one-way mirror, a video and audio monitoring system allowed the experimenter to observe all session events. It should be noted here that the risks for the CO₂ administration include temporary discomfort that may include racing heart sensations, increased breathing rate, shortness of breath, and dizziness. These effects are entirely harmless and painless; they disappear quickly when participants return to breathing normal room air. In one recent investigation, for example, a large sample of

participants ($n = 125$) underwent recurrent CO₂ administration or room air (Prenoveau, Forsyth, Kelly, & Barrios, 2006). These participants were then prospectively monitored for up to 1 year. Results of this controlled investigation indicated that the percentage of people who developed subsequent panic attacks did not differ by condition. Thus, these data indicate that CO₂ administration does not increase the risk of subsequent panic attacks in a nonclinical population, and hence, is a safe paradigm for use in research. The CO₂-inhalation was utilized as the PD-relevant challenge procedure because it can be safely employed, its parametric properties are well studied, and it can reliably produce bodily arousal and psychological symptoms relevant to panic states in nonclinical and clinical samples (Zvolensky & Eifert, 2000). Moreover, it has been safely and effectively used in previous work with adults across numerous research sites without incident for decades (e.g., Gorman et al., 2001).

A J&J Engineering I-330-C2 system was used to digitally record physiological data on-line at a sample rate of 1024 samples per second across all channels using J&J Engineering Physiolab Software. Three physiological variables were examined for the current study (Venables & Christie, 1980): respiration rate (a measure of breaths per minute), skin conductance levels (SCL; a measure of the basal level of sweat gland activity), and heart rate (a measure of beats per minute). Respiration rate was obtained using a Pneumograph sensor cable with PS-2 sensors as a manipulation check. The sensors were placed across the chest and secured with a Velcro strap, allowing a measure of chest excursion during respiration. SCL converted to microsiemens (μS) were obtained using an RV-5 skin resistance lead connected to SE-35 electrodes placed on the middle segment of the middle finger. Raw electrocardiogram data were collected with disposable Ag/AgCl electrodes placed in a standard bilateral configuration on the

palmar side of each wrist. Data were processed through a 1-100Hz bandpass filter designed to maximize R-wave frequency.

Design and Procedure

The present investigation implemented a correlational, group-based cross sectional design (Kazdin, 2003). See Table 2 for an overview of the study procedure. Community and university-based populations were focused on in the recruitment process. Specifically, participants were recruited through newspaper advertisements and flyers that describe a laboratory study on 'emotion.'

Interested persons responding to advertisements who contacted the research team were given a detailed description of the study over the phone. After providing verbal consent, the SCID-NP (screener) was administered by a trained research assistant via telephone. Those meeting inclusionary criteria were scheduled to attend a single laboratory session. Upon arrival, participants completed a written informed consent, which indicated that the procedure involved a single 4-min 10% CO₂-enriched air presentation. Participants then completed the pre-experimental measures. Each participant was then introduced to the laboratory setting for the challenge procedure. During the session, participants sat alone in the 8-ft x 12-ft sound attenuated experimental room, which contained a computer, chair, desk, and intercom that allowed participants to communicate freely with the experimenter in the adjacent room. Participants were seated in front of a table, on which a binder with the experimental, paper-pencil self-report measures was placed. Once the electrodes were attached standardized instructions were provided, including:

“Following the (10 minute) adaptation period, we will start the experimental portion of the study which will last approximately 4 minutes. During this period you will receive several inhalations of CO₂-enriched air that may produce physical and

mental sensations associated with bodily arousal. You may temporarily feel your heart racing, your palms might be sweaty, you might feel dizzy, and you might have some breathing problems.”

The study consisted of two phases. The first phase involved a 10-min baseline adaptation period during which participants sat quietly in the testing room breathing regular room air. Participants completed SUDS ratings at the beginning and end of the adaptation period. Phase two consisted of the automated delivery of one 4-min 10% CO₂-enriched air presentation. Participants completed a SUDs rating and the DSQ immediately after completing the 4-minute challenge exposure. Physiological data were gathered continuously across both phases. After the study, participants were debriefed and compensated \$20.

Data Analysis: General Approach

The main and interactive relations between AS and perceived control over anxiety-related events were evaluated in relation to responding to the CO₂ challenge using a hierarchical multiple regression procedure (Cohen & Cohen, 1983). Main effect variables were mean-centered prior to computing product/interaction terms (Aiken & West, 1991). Squared semi-partial correlations were used as indices of effect size in all models and were tested at a two-tailed alpha of .05.

Clarification of moderation versus interactive effects. Moderation refers to the examination of the statistical interaction between two independent variables (X and Y predicting Z; Baron & Kenny, 1986). As applied to the present study aims, this type of perspective suggests that the relationship between AS and panic-relevant responsivity to biological challenge may differ at different levels of perceived control over anxiety-related events. Under circumstances where X is a manipulated variable, in principal, there should be no relationship between X and Y (i.e., they should be independent;

Aiken & West, 1991). Yet, if X is not randomized or cannot be ethically manipulated, it may be correlated with Y (Baron & Kenny, 1986; Holmbeck, 1997). Some researchers suggest that under this circumstance of a correlation between X and Y an observed statistical interaction can still be considered moderation (Judd, Kenny, & McClelland, 2001), whereas others do not (Kraemer, Wilson, Fairburn, & Agras, 2002).

To a certain extent, these perspectives reflect work oriented *a priori* on different research questions (e.g., research focused on a direct manipulation, such as intervention studies wherein there is random assignment and an experimental manipulation, versus, that which the present study is not; Holmbeck, 1997). In the present model, some low-level association between AS and perceived control over anxiety-related events ($r = .10$) was expected, as has been found in past work (White et al., 2006). Accordingly, the present study was oriented on the aims using the label “moderation.” This approach is descriptively accurate and fully consistent with the theoretical basis of the present investigation. [It also should be noted here that the study aims do not reflect mediational processes. Such a test would be oriented on whether a variable accounts for a significant amount of the shared variance between a predictor and dependent variable (Holmbeck, 1997).] Thus, because neither theory or empirical evidence suggests that perceived control leads to AS and thereby panic problems, or that AS leads to perceived control and thereby panic problems (Barlow, 2002), mediation will not be tested.

Consistent with past research in this area (Zinbarg et al., 2001), separate models were constructed for predicting the criterion variables of anxiety focused on bodily sensations (postchallenge SUDS) and intensity of DSM-IV panic attack symptoms (mean panic attack symptom intensity score on DSQ) following the 10% CO₂ challenge. In the first step in the model, pre-challenge anticipatory anxiety (SUDS) and gender were

to be entered as covariates if they demonstrated significant relations with the dependent variables. In the second step, the main effects of perceived control over anxiety-related events (as indexed by the ACQ total score) and AS (as indexed by the ASI total score) were entered simultaneously. In the final step, the interaction term between ASI total score and the ACQ total score events were entered into the model (mean centered). It was expected that participants with low levels of perceived control and elevated AS, relative to all other variable combinations, will evidence the highest post-challenge DSQ and SUDS scores. Based on recommendations of Holmbeck (2002), if applicable, post-hoc probing analyses were conducted on the data to statistically document the nature of the interaction.

For avoidance, the same exact model was planned on being employed, except interest in returning for another CO₂ investigation (*0 = no desire to participate; 100 = definite desire to participate*) served as the dependent measure.

Hierarchical regression analyses were used to examine respiration rate, SCL, and heart rate during the challenge. Dependent measures were respiration rate (breathes per minute), SCL, and heart rate taken at the final 1-minute during CO₂ challenge. In these models, we controlled for the corresponding variable during baseline (last minute during adaptation period) as well as gender. The second and third steps in the model were identical to that in the just described hypotheses.

Results

Data Reduction Approach and Manipulation Check of Provocation Paradigm

After screening for outliers due to sampling error (e.g., participant movement), the integrity of the 10% CO₂– enriched air administered for 4-min to elicit anxiety and physiological responsiveness was examined. Standard data reduction strategies employed in past biological challenge work were employed for the physiological data

screening and reduction process (Zvolensky et al., 1998); specifically, any non-readable data (i.e., missing data due to human error such as an electrode falling off a participant) were eliminated. The data also were inspected for falling beyond an expected range per the recommendations of Venables and Christie (1980). If data were at an extreme (e.g., greater than 230 beats per minute), they were removed due to the likelihood of containing a sampling error of some type.

A paired-samples *t*-test indicated that the mean SUDS score post-challenge ($M = 53.93$, $SD = 27.75$) was significantly greater than the mean SUDS score pre-challenge ($M = 16.11$, $SD = 15.47$), $t(223) = -20.55$, $p < .001$). In addition, paired-samples *t*-tests indicated that the mean heart rate, SCL, and respiration rate scores post-challenge ($M = 91.87$, $SD = 14.47$; $M = 3.81$, $SD = 1.72$; $M = 19.75$, $SD = 4.09$ respectively) were significantly greater than at the final minute of the pre-challenge time period ($M = 81.55$, $SD = 10.35$; $M = 1.66$, $SD = 1.14$; $M = 16.08$, $SD = 3.30$ respectively), $t(179) = -9.71$, $p < .001$; $t(171) = -19.68$, $p < .001$; $t(166) = -10.11$, $p < .001$ respectively.

Descriptive Data and Zero-Order (or Bi-variate) Relations

Table 3 shows the inter-correlations, means, standard deviations, and the observed range (corrected for sampling error) for the predictor and criterion variables.

Correlations between the cognitive predictor variables and the dependent measures. Correlations between the ASI total score and ACQ total score and the dependent variables were then examined. As expected, ASI total score was significantly positively correlated with post-challenge SUDS (SUDS-PC; $r = .29$, $p < .001$), and postchallenge panic attack symptoms (DSQ total mean score; $r = .31$, $p < .001$), and negatively correlated with willingness to return for another challenge ($r = -.18$, $p < .01$). The ASI total score was not significantly associated with SCL ($r = .15$, $p = .06$; although a trend was evident), respiration rate ($r = -.01$, $p = .89$), or heart rate ($r = .11$, $p = .16$).

The ACQ total score was significantly negatively correlated with post-challenge SUDS ratings ($r = -.16, p < .05$), and total DSQ post-challenge scores ($r = -.14, p < .05$), but not with any other criterion variables.

Correlations between the covariates and the dependent variables. Correlations between the covariates and the dependent variables were then examined. Gender was significantly positively correlated with post-challenge SUDS ($r = .25, p < .001$), post-challenge panic attack symptoms (as indexed by the DSQ total score; $r = .21, p < .01$), and post-challenge heart rate ($r = .22, p < .01$), indicating that females are more likely to have higher post-challenge SUDS ratings, post-challenge panic attack symptoms, and post-challenge heart rate. Additionally, gender was significantly negatively correlated with willingness to return for another challenge ($r = -.32, p < .001$), indicating that females are less likely to express interest in returning than males. Gender was not significantly correlated with post-challenge SCL or respiration rate (breathes per minute). Pre-challenge SUDS ratings were significantly positively correlated with post-challenge SUDS ratings ($r = .39, p < .001$), post-challenge panic attack symptoms ($r = .28, p < .001$), and post-challenge skin conductance levels ($r = .16, p < .05$), but was not correlated with any other criterion variables.

Correlations between the cognitive predictor variables and the covariates. Finally, the relations between the predictor variables were examined. The ASI total score and ACQ total score were significantly negatively correlated ($r = -.42, p < .001$; 17.8% shared variance with one another [computed by squaring the zero-order correlation between the two variables]). The ASI total score was significantly positively correlated with both baseline SUDS rating ($r = .23, p < .001$) and gender ($r = .20, p < .01$; females reported higher AS levels than males). In addition, ACQ total score was significantly negatively correlated with both baseline SUDS rating ($r = -.24, p < .001$) and gender ($r = -.24, p <$

.001; females reported lower perceptions of control over anxiety-related events). The covariates were not significantly correlated with one another.

Hierarchical Regression Analyses

Tables 4 (self-report dependent measures) and 5 (physiological dependent measures) include a summary of the regression analyses.

*Self-reported panic attack symptoms, anxiety, and avoidance variables.*¹ For panic attack symptoms (DSQ), the predictor variables collectively explained 18% of the overall variance (adjusted $R^2 = .16$), $F(5, 212) = 9.29$, $p < .001$. At step one, the covariates of gender and baseline SUDS ratings accounted for 13% of unique variance (adjusted $R^2 = .12$), with both gender ($t(217) = 3.42$, $p < .01$) and baseline SUDS ($t(217) = 4.49$, $p < .001$) as significant predictors. At step two, the main effects of ASI and ACQ accounted for 5% of unique variance, with the model of step one and step two accounting for 18% (adjusted $R^2 = .16$). At step two, the ASI ($t(217) = 3.67$, $p < .001$) was a significant predictor. The ACQ was not a significant predictor. The interaction term (ASI x ACQ) at step three of the model also was not a significant predictor.

For post-challenge SUDS (SUDS-PC), the predictor variables explained 25% of the overall variance (adjusted $R^2 = .23$), $F(5, 213) = 14.19$, $p < .001$. Step one of the model accounted for 22% of the variance (adjusted $R^2 = .22$), with both gender ($t(218) = 4.32$, $p < .001$) and baseline SUDS ($t(218) = 6.62$, $p < .001$) as significant predictors. Step two of the model accounted for an additional unique 3% of the variance. The ASI ($t(218) = 2.82$, $p < .01$) was the only significant predictor. There was no significant interactive effect.

For interest in returning for a second challenge, the predictor variables collectively explained 16% of the overall variance (adjusted $R^2 = .14$), $F(5, 208) = 7.99$, $p < .001$. For the covariates, step one of the model accounted for 14% of the variance

(adjusted $R^2 = .13$). Only gender was a significant univariate predictor at this step of the model ($t(213) = -5.65, p < .001$). At step two of the model, the main effects accounted for a unique 2% of the variance. At this step of the model, the ASI ($t(213) = -2.22, p < .05$) was the only significant predictor. There was no significant interactive effect in the final step in the model.

Physiological variables. For respiration rate,^{2,3} the predictor variables collectively explained 9% of the overall variance (adjusted $R^2 = .06$), $F(5, 157) = 3.12, p < .05$. At step one, the covariates accounted for 7% of the variance (adjusted $R^2 = .05$), with only baseline respiration rate ($t(162) = 2.68, p < .01$) as a significant predictor. At step two, the main effects accounted for an additional 2% unique variance. At this step, only the ACQ ($t(162) = 2.03, p < .05$) was a significant predictor. There was no significant interactive effect at step three.

For SCL, the predictor variables collectively explained 35% of the overall variance (adjusted $R^2 = .33$), $F(5, 161) = 17.19, p < .001$. At step one of the model, the covariates accounted for 32% of the variance (adjusted $R^2 = .31$), with only baseline SCL ($t(166) = -.45, p < .001$) as a significant predictor. At step two, the model accounted for an additional 3% unique variance (adjusted $R^2 = .33$), with the ASI ($t(166) = 2.69, p < .01$) as the only significant predictor. There was no significant interactive effect at step three of the model.

Finally, for heart rate, the predictor variables collectively explained 21% of the overall variance (adjusted $R^2 = .18$), $F(5, 169) = 8.73, p < .001$. At step one of the model, the predictors accounted for 19% of the variance (adjusted $R^2 = .18$). For the covariates, both gender ($t(174) = 3.08, p < .01$), and baseline heart rate ($t(174) = 5.61, p < .001$) were significant univariate predictors. At step two of the model, the main effects accounted for a unique 2% of the variance, with neither the ASI nor ACQ showing

significance. Additionally, there was no significant interactive effect at step three of the model.

Discussion

The overarching aim of the present investigation was to examine the singular and interactive relationships between AS and perceived control over anxiety related events and panic-relevant responding in the context of a biological challenge paradigm. This investigation is important theoretically and clinically because it serves to help isolate the nature of putative cognitive vulnerability for anxious and fearful responding to bodily sensations, and by extension, the possible underpinnings involved with panic psychopathology.

Interactive Effects

Inconsistent with prediction, there was no evidence of a significant interactive effect between AS and perceived control over anxiety-related events for any of the studied dependent variables. There also was no significant effect evident for the interaction in regard to any of the studied physiological variables. Thus, these two cognitive factors did not demonstrate a synergistic relation to any aspect of panic vulnerability (self-report or physiological) among the present sample of young adults in this biological challenge paradigm. These null findings are not likely attributable to statistical power, as the overall sample size was comprised of 229 persons; a sample size that exceeded the planned power analysis based upon past work; no trends were evident. Additionally, post hoc analysis of the lower-order facets (subscales) in relation to the dependent measures did not yield any further evidence of a significant interactive effect. Accordingly, even with a large sample (greater than that originally proposed), in conjunction with *a priori* and post hoc analyses of facets of the predictor variables, no

significant effects were evident, lending no support for an interactive effect for AS and perceived control over anxiety-related events for the studied dependent variables.

Overall, such null findings are potentially noteworthy for at least two reasons. First, the current results are not in accord with conceptual models of panic vulnerability (Barlow, 1991; 2002) that suggest AS and perceived control over anxiety-related events, as individual difference factors, are interactively related to panicogenic responding. Based upon these findings, it is possible that conceptual models of panic psychopathology *etiology* that emphasize synergistic processes between AS and perceived control over anxiety-related events may need to be refined so that the interactive aspect of vulnerability is not specified or given a unique explanatory role. It is possible that these factors may each maintain unique relations to certain, but not all, panic-relevant process, but do not interplay with one another to confer risk above and beyond the main effects (see later discussion of main effects below). Due to the sampling tactics in the current study, the implications for the maintenance of panic psychopathology remains a fecund area in need of further empirical exploration (e.g., role of AS and perceived control over anxiety-related events among those with panic and other anxiety disorders).

Second, the present results also are inconsistent with past empirical work using experimental manipulations of offset control over bodily sensations (Sanderson et al., 1989; Zvolensky et al., 1998, 1999, 2001). The main difference between past empirical work and the current study is in the nature of the assessment and study of perceived control over anxiety-related events. Here, perceived control over anxiety-related events was measured continuously through a self-report device as an individual difference factor, whereas in previous work control over the offset of CO₂ administration in 'real time' was completed. Although these two aspects of control are conceptually related, the

two operationalizations of control variables, despite broad-based shared theoretical relevance to one another, may not be fully the same (i.e., equivalent constructs). One interpretation of the extant work, then, is perhaps a more robust degree of variability of control over anxiety-related events (indexed through an experimental manipulation), rather than a self-report device, is needed for an interactive effect for panic processes to be observed. Another (related) interpretation, as alluded to above, may be that perceived control over anxiety-related events, as measured via the ACQ, and experimental manipulations of offset control over a CO₂ administration are not isomorphic with one another despite sharing similarities in terms of being ‘control-oriented.’ That is, the ACQ is aimed at identifying individual differences in perceived control over anxiety-related events and experimental manipulations represent acute changes in the ability to control (terminate) an aversive interoceptive event. Thus, these two ‘control variables’ may, in fact, not be indexing the same latent construct.

Main Effects

Anxiety sensitivity. Consistent with expectation, there was evidence that AS was significantly and incrementally associated with greater post-challenge panic attack symptom ratings, positive reactivity in anxiety focused on bodily sensations (baseline to post-challenge changed in anxiety ratings), and less interest in returning for another challenge (behavioral avoidance; see Table 4). The size of the observed AS effects were small to medium in magnitude using Cohen and Cohen standards (1983), but evident above and beyond the variance attributed to gender and baseline anticipatory anxiety (accounting for a range of variance from 13% to 22% across the studied dependent variables) and shared variance with perceived control over anxiety-related events. The effect sizes observed in the current study should be considered in the context in which they were observed: after covarying for two other risk candidates (gender and baseline

anxiety) and among a sample that was screened for current psychopathology. These two elements provide an arguably 'conservative' test of the model in that there is (presumably) less upper-end variability (due to the screening criteria employed) yet still considered variance related to other factors (the covariates). As Abelson (1985) has persuasively argued, the relative degree of practical (clinical) significance of such findings rests in the context in which it was examined. Thus, despite relatively small effects for AS, the test itself was a strong one, and overall, lends further credibility to the explanatory value of this cognitive factor in terms of panic-relevant symptoms.

It also is noteworthy that the AS findings are fully consistent with past work, across a diverse range of samples, that has found this cognitive factor is related to self-reported anxiety and panic attack symptoms (McNally & Eke, 1996; Leen-Feldner, Feldner et al., 2005; Rabian et al., 1999; Schmidt, 1999; Zvolensky, Feldner et al., 2001). The current results uniquely extend past research in a novel manner by documenting that AS effects are not due to shared variance with perceived control over anxiety-related events, another putative cognitive vulnerability factor expressly highlighted in panic vulnerability models (Barlow, 2002). Additionally, the AS effects were found for multiple indices of panic vulnerability, including interest in returning for another biological challenge study in the near future (behavioral avoidance). Past work has typically focused only on one, and less commonly two, self-reported indices of post-challenge anxiety or panic symptoms, but rarely included multiple indices of 'fear reactivity' as was completed in the current work (Zvolensky & Eifert, 2000).

In terms of the physiological variables, AS was significantly and incrementally related to SCL, but not respiration rate or heart rate. Past work on AS using laboratory and non-laboratory (e.g., ambulatory monitoring) methodologies has typically not yielded significant relations to physiological indices (Zvolensky & Eifert, 2000). Of the

physiological variables that have occasionally shown a relation to AS, SCL has typically been the variable of interest (Stewart & Pihl, 1994). Most scholars have interpreted the lack of AS-physiological index findings to mean that this cognitive factor is primarily related to changes in cognitive-affective processes rather than objective physiologic change (Bernstein & Zvolensky, 2007; McNally, 2002). In the present study, only one of the three physiological variables showed a significant relation and the effect accounted for approximately 3% of unique variance (small effect). Thus, it is difficult to draw strong inferences regarding this association (due to the number of comparisons and small effect). However, the results could suggest that AS is related to change in the basal level of sweat gland activity following exposure to an aversive interoceptive event. Before confidence can be placed in this finding, it is central that the effect be replicated in an independent sample. With additional replication of the AS-SCL effect in a biological challenge paradigm in an independent sample, it is possible that this cognitive factor may be related to not only cognitive-affective aspects of anxious and fearful responding, but also sweat gland reactivity. There is limited study of AS-sweat gland activity, possible due, in part, to the many null physiologic findings reported at earlier time points (Zvolensky & Eifert, 2000). Thus, AS could promote, in theory, self-report and certain (specific) physiological aspects of responding (e.g., sweat gland activity) and serve as a generalized 'amplifier' of emotional responding to interoceptive cues.

Perceived control over anxiety-related events. In contrast to the observed AS effects, there was, again, uniformly no evidence that perceived control over anxiety-related events, as indexed by the ACQ, was related to post-challenge panic attack symptom ratings, anxiety focused on bodily sensations, or interest in returning for another challenge (behavior avoidance). These findings suggest that perceived control over anxiety-related events, as an individual difference variable, is not related to any

cornerstone aspect of panic vulnerability from the perspective of cognitive-affective indices. These findings are inconsistent with experimental studies of perceived control over anxiety-related events that have utilized a biological challenge paradigm (Sanderson et al., 1989; Zvolensky, Lejuez, et al., 1998; Zvolensky, Eifert, et al., 1999). There have not been studies focused on perceived control over anxiety-related events, as measured by the ACQ, in terms of the prediction of anxious and fearful responding to bodily sensations using laboratory methods. However, past work, although limited in overall scope, has indicated perceived control over anxiety-related events is related to interpretative bias for threat (Zvolensky, Heffner et al., 2001) and self-reported anxiety symptoms in non-laboratory tests (Rapee et al., 1996). There are numerous methodological differences between the present investigation and those reported earlier (e.g., different paradigms, different dependent measures; Rapee et al., 1996; Zvolensky, Heffner et al., 2001). Any number of differences between such studies could arguably be a source of differential findings. On the other hand, the current results - using an arguably highly rigorous methodology - may simply be accurate: relative to AS, there is not unique explanatory value of perceived control over anxiety-related events for self-reported and behavioral avoidance aspects of panic-relevant responding. If accurate, these findings would indicate that perceived control over anxiety-related events, as an individual difference factor, is less relevant than AS in terms of accounting for variability in anxious and fearful responding to bodily sensations.

In terms of perceived control over anxiety-related events and physiological responsiveness, there was a significant effect for respiration rate, but not SCL, or heart rate. The observed perceived control over anxiety-related events effect for respiration rate was small in magnitude (approximately 2% of unique variance). There have been no studies that have examined perceived control over anxiety-related events and

psychophysiological reactivity using a biological challenge paradigm and therefore no empirical literature that directly informs the observed findings. One study, in a different stream of work focused on acute pain-induction, did not find an association between the ACQ and heart rate, although respiration rate was not measured (Feldner & Hekmat, 2001).

Although caution should be employed in interpreting the ACQ-respiration effect in the current study due to the small effect size and number of physiological-oriented tests ($n = 3$) and attendant risk of family-wise error (an identical interpretative-based issue for the AS-SCL finding reported and discussed earlier), the results may point to the possibility that this cognitive factor is related to respiration rate. Specifically, to the extent an individual believes they have greater control over internal (aversive) events like bodily sensations, they may be more apt to regulate their breathing as a method for controlling their somatic response to such a stressor. This type of perspective is broadly consistent with the health hardiness literature in the field of health psychology. Here, perceptions of control over one's health status, among other concomitant related beliefs about personal commitment for change and a positivistic attitude about reactions to stressful life events, is related to more adaptive reactions to somatic perturbation and physical illness (Kobasa, 1993; Kobasa, Maddi, & Kahn, 1982). Future work is needed to better understand the association between perceived control over anxiety-related events and respiration rate. To the extent this cognitive factor is, in fact, related to respiratory-based self-regulation, it may be advisable to expressly target perceived control over anxiety-related events among persons with panic or related anxiety or even medical illnesses characterized by disturbances in breathing (e.g., asthma, chronic obstructive pulmonary disease). For example, by facilitating change in perceived control over anxiety-related events, it may be possible to prompt change in how an individual regulates their

breathing. Although naturally (highly) speculative at this stage in research development, this type of finding underscores the importance of considering cognitive factors in terms of understanding physiological processes at a broad-based level.

Other Noteworthy Observations

Although not a primary aim of the investigation, there are three other important observations from the present study that warrant brief comment.

Challenge paradigm. It should be noted that the challenge paradigm itself was successful in eliciting meaningful elevations in anxiety and panic symptoms as well as physiological responsiveness (heart rate, respiration rate, and skin conductance). Thus, a high degree of confidence can be placed in the robust nature of the present paradigm for eliciting panic-relevant periods of bodily perturbation and arousal. Nonetheless, the protocol employed did not elicit *panic attacks* per se. From a conservative vantage, the present paradigm involves elicitation of abrupt periods of anxiety status triggered by interoceptive cues. Similar to the type of approach utilized by Craske, Glover, and DeCola (1995), one way to evaluate whether a similar process would be evident for naturally occurring panic attacks would be to study panic episodes using ecological momentary recording devices among individuals with panic disorder. Aside from conceptually replicating laboratory findings to panic attacks, such work would usefully extend the present findings to a clinical population under naturalistic conditions and address linkages to anxiety and fear maintenance processes (cf. onset processes).

Overlap between AS and perceived control over anxiety-related events. Though not a focal point of the study per se, it is noteworthy that AS and perceived control over anxiety-related events were significantly negatively correlated, sharing 17.8% variance with one another. This finding is important because it indicates that these two panic-related risk factors are tapping different, albeit related, types of vulnerability processes.

Although there has been little study of the interrelationship between AS and perceived control over anxiety-related events, it appears that aside from being conceptually distinct, that they, in fact, also are empirically distinct. Such a finding is useful, in conjunction with their differential associations with the dependent measures, because it suggests that these factors are likely not one and the same and should be considered individually in the context of clinical preventative or treatment activities (see Synthesis of Clinical Implications Section for a further discussion of this issue).

Gender. In the present investigation, gender was significantly predictive of post-challenge panic attack symptom ratings and level of anxiety, interest in returning for a future challenge, and heart rate. Specifically, female participants were more likely to report higher ratings of panic attack symptoms and anxiety focused on bodily sensations, stated being less likely to want to return for another CO₂ challenge (behavioral avoidance), and had an increased heart rate. Gender is associated with differential emotional reactivity generally (Balswick & Avertt, 1977) and anxiety-related reactions specifically (Cameron & Hill, 1989). Research also indicates that women are more likely relative to men to meet diagnostic criteria for panic psychopathology (Clum & Knowles, 1991). Additionally, females typically report more and intense fears relative to males in nonclinical populations (Bekker, 1996). These data indicate that females relative to males are generally more apt to be emotionally responsive to bodily sensations. Although the source for heart rate-gender effects is less obvious, it is possible that females compared to males may be more cardiac reactive. Numerous factors affect heart rate, including fitness level, mood state, and related factors (e.g., consumption of caffeine in the recent time period). It is not possible to specify the source for such effects in the current study, but future work may be fruitfully focused on addressing such issues from an *a priori* perspective.

Synthesis of Clinical Implications

In addition to already described theoretical implications for refined models of panic vulnerability (in the interaction and main effect sub-sections, respectively), the present work serves to enhance our empirical understanding of clinically-relevant processes from a therapeutic perspective. For example, information about the nature of AS and perceived control over anxiety-related events can be used to help clients with panic psychopathology or those with pre-morbid panic vulnerabilities to understand tendencies to react to bodily events fearfully. With this knowledge, alternative, more adaptive strategies can perhaps be utilized to help such individuals to better cope with emotional stressors and thereby enhance psychological well-being. At a pragmatic level, AS could be specifically targeted through traditional cognitive-behavioral strategies of cognitive restructuring and interoceptive exposure. More specifically, a person's beliefs about anxiety as threatening would be targeted cognitively, but also through exposure to the sensations, so as to learn that these physical sensations are not harmful. Given the findings for perceived control over anxiety-related events, it does not seem that they need to be targeted in the context of AS. However, further research could determine if perceived control over anxiety-related events plays a role in panic psychopathology, and therefore, would require attention in treatment programs. Similarly, from a prevention standpoint for high AS individuals, psychoeducation about aversive sensations could be targeted at reducing fears about anxiety. Within this context, exposure focused on bodily sensations could be used to allow for practice in experiencing the symptoms and understanding the habituation process, thereby reducing beliefs that these symptoms are harmful, and thus preventing the development of panic psychopathology.

Interpretative Caveats

Beyond the already noted interpretative caveats of the present study, there are a number of other points for clarification. First, the present sample was limited in that it is comprised of a relatively homogenous (e.g., primarily Caucasian) group of young adults who volunteered to participate in the study for monetary reward. To rule out potential self-selection bias among persons with these characteristics and increase the generalizability of these findings, it will be important for researchers to draw from other populations and utilize recruitment tactics other than those used in the present study. Second, the behavioral avoidance findings should be viewed with caution. It is possible that the person's report of their desire to return for a future challenge may not be fully in line with their actual behavior, such that a participant may report that they would be willing to return, but may not actually follow through, and vice versa. It also is possible this measure of avoidance may tap boredom, frustration, or related factors rather than 'pure' fear-driven avoidance. Thus, future work should attempt to solidify this finding through a more rigorous methodology. For example, an experiment could be designed in which a second challenge is planned minutes following the first challenge, and then, determine objectively which participants are willing to stay for the second administration. Additionally, participants could be called a week following the challenge to determine if they wish to schedule another appointment. These approaches allow for a behavioral confirmation of the participants report to attend another CO₂ administration.

Third, menstrual and menopausal factors could be considered in future work. Sigmon and colleagues (2000) found that women high in AS also reported high levels of menstrual reactivity, which is defined as a perception that menstrual symptoms and bodily sensations are severe and distressing. Given these previous findings, future work should consider the effect of menstrual factors in examining cognitive vulnerability factors predicting anxious responding. It is possible that female participants who were

menstruating or menopausal in the present investigation may have been more likely to report higher levels of anxious responding following the challenge. Fourth, the present cross-sectional correlational design does not permit causal-oriented hypothesis testing. Although an attempt to strengthen confidence in the observed findings was achieved by controlling for theoretically-relevant factors, causal directions of the observed relations cannot be fully determined. Future work could build from the present study by evaluating the observed relations experimental methodologies. Finally, although the present investigation examined the two most well-established cognitive vulnerability factors for panic, there are other cognitive risk factors that may warrant examination (e.g., predictability over anxiety-related events, affect intensity, affect tolerance). By continuing to study cognitive factors in multi-risk factor models, clinically-relevant information concerning the interplay between such factors can be better understood and applied to understanding the enigmas of panic psychopathology.

Summary

Overall, the present investigation adds uniquely to the extant empirical literature on AS and perceived control over anxiety-related events and panic-relevant processes. Results suggest that although these two cognitive factors are moderately related to one another, AS demonstrates a more robust association than perceived control over anxiety-related events with intensity of panic attack symptoms, anxiety focused on bodily sensations, and willingness to participate in a future biological challenge study (behavioral avoidance) within the context of a CO₂ paradigm. Using this type of basic research to guide our understanding of clinically-relevant processes will continue to be an important task for translational research efforts focused on anxiety and its disorders.

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Footnotes

¹ Post-hoc analyses examining the role of the subscales of the constructs in predicting panic attack symptoms, anxiety, and interest in returning were conducted. Results indicated that the subscales were not significantly predictive of the criterion variables, nor was any combination of possible interaction.

² Analyses examining the physiological criterion variables (respiration rate, SCL, and heart rate) were conducted with baseline SUDS ratings as a covariate in addition to the analyses in the text. There was no difference in the results produced whether using baseline SUDS as a covariate or not. Thus, the original *a priori* model was retained and reported here.

³ Post-hoc analyses examined if the measure subscales (see also footnote #1) were predictive of the physiological variables (respiration rate, heart rate, and skin conductance level). No significant main or interactive effects were found.

Table 1: *Vulnerability Terminology*

Risk Factor

- a variable that is related to, and temporally precedes, an unwanted outcome

Causal Risk Factor

- reflect variables that, when modified in some way (e.g., through an intervention), produce systematic change (increase or decrease) in the dependent variable of interest among persons who did not previously manifest such problems

Proxy Risk Factor

- variables that are related to an outcome of interest, but this association is due to the proxy risk factor's relationship with another causal risk factor

Fixed Marker

- when a risk factor can not be changed

Variable Risk Factor

- when a risk factor can be changed

Table 2: *Overview of Procedure*

Recruitment

- Newspaper advertisement and flyers

Screening

- Appointment for assessment set

Laboratory appointment

- Informed consent
 - SCID-NP
 - Self-report questionnaires
 - Anxiety Sensitivity Index (ASI)
 - Positive and Negative Affect Scale (PANAS)
 - Anxiety Control Questionnaire (ACQ)
 - CO₂ Procedure
 - Diagnostic Sensations Questionnaire (DSQ)
 - Subjective Units of Distress
 - Physiological assessment
 - Debriefing
 - Compensation
-

Table 3: Descriptive Data and Zero-Order (or Bi-variate for Dichotomous Factors) Relations between Predictor and Criterion Variables

	Mean (SD)	Range	1	2	3	4	5	6	7	8	9	10
Predictor Variables												
1. Gender	M: 46%, F: 54%		-	-.02	.20**	-.24 [^]	.25 [^]	.21**	-.32 [^]	.18*	-.15	.22**
2. Baseline SUDS	16.06 (15.41)	0 - 100	-	-	.23 [^]	-.24 [^]	.39 [^]	.28 [^]	-.08	.12	.16*	.01
3. ASI	15.95 (7.89)	0 - 40	-	-	-	-.42 [^]	.29 [^]	.31 [^]	-.18**	-.01	.15	.11
4. ACQ	51.18 (10.73)	23 - 75	-	-	-	-	-.16*	-.14*	.03	.11	-.02	-.00
Criterion Variables												
5. SUDS-PC	53.92 (27.69)	0 -100	-	-	-	-	-	.68 [^]	-.25 [^]	.21**	.26**	.28 [^]
6. DSQ	3.06 (1.53)	.19 - 7.25	-	-	-	-	-	-	-.33 [^]	.31 [^]	.30 [^]	.38 [^]
7. Return	60.37 (31.89)	0 - 100	-	-	-	-	-	-	-	-.32 [^]	-.08	-.25**
8. BPM	19.58 (4.11)	10.32 - 29.11	-	-	-	-	-	-	-	-	.14	.38 [^]
9. SCL	3.81 (1.71)	1 - 8	-	-	-	-	-	-	-	-	-	.17*
10. HR	91.80 (14.47)	58.83 - 139.46	-	-	-	-	-	-	-	-	-	-

Note. $N = 229$. * $p < .05$, ** $p < .01$, [^] $p < .001$. M: Male, F: Female (dummy coded with females being 2 and males 1); Baseline SUDS: Subjective Units of Distress Scale one minute pre-challenge (Wolpe, 1958); ASI: Anxiety Sensitivity Index total score (Reiss et al., 1986); ACQ: Anxiety Control Questionnaire total score (Rapee et al., 1996); SUDS- PC: Subjective Units of Distress Scale Post-Challenge (Wolpe, 1958); DSQ: Diagnostic Sensations Questionnaire (Sanderson, Rapee, Barlow, 1988, 1989); Return: Willingness to return for another challenge; BPM: Breaths per minute/Respiration rate; SCL: skin conductance, HR: heart rate.

Table 4: Individual Variable Contributions Predicting the Self-report Criterion Variables

DV	Predictor	R ²	Adj.R ²	t (each predictor)	β	p
DSQ	Level 1	.13	.12			
	Gender			3.42	.22	<.01
	Baseline SUDS			4.49	.29	<.001
	Level 2	.18	.16			
	ASI			3.67	.25	<.001
	ACQ			1.08	.08	ns
	Level 3					
	ASI x ACQ	.18	.16			
				.23	.02	ns
SUDS-PC	Level 1	.22	.22			
	Gender			4.32	.26	<.001
	Baseline SUDS			6.62	.40	<.001
	Level 2	.25	.24			
	ASI			2.82	.19	<.01
	ACQ			1.07	.07	ns
	Level 3					
	ASI x ACQ	.25	.23			
				-.24	-.01	ns
Interest in Returning	Level 1	.14	.13			
	Gender			-5.65	-.36	<.001
	Baseline SUDS			-1.25	-.08	ns
	Level 2	.16	.14			
	ASI			-2.22	-.16	<.05
	ACQ			-1.65	-.12	ns
	Level 3					
	ASI x ACQ	.16	.14			
				-.59	-.04	ns

Note. N = 229. Baseline SUDS: Subjective Units of Distress Scale one minute pre-challenge (Wolpe, 1958); ASI: Anxiety Sensitivity Index total score (Reiss et al., 1986); ACQ: Anxiety Control Questionnaire total score (Rapee et al., 1996); DSQ: Diagnostic Sensations Questionnaire (Sanderson, Rapee, Barlow, 1988, 1989); SUDS- PC: Subjective Units of Distress Scale Post-Challenge (Wolpe, 1958)

Table 5: Individual Variable Contributions Predicting the Physiological Criterion Variables

DV	Predictor	R ²	Adj.R ²	t (each predictor)	β	p
Respiration Rate						
	Level 1	.07	.05			
	Gender			1.93	.15	ns
	Baseline BPM			2.68	.21	<.01
	Level 2	.09	.07			
	ASI			.32	.03	ns
	ACQ			2.03	.17	<.05
	Level 3					
	ASI x ACQ	.09	.06			
				-36	-.03	ns
SCL						
	Level 1	.32	.32			
	Gender			-.45	-.03	ns
	Baseline SCL			8.38	.56	<.001
	Level 2	.35	.33			
	ASI			2.69	.18	<.01
	ACQ			.82	.06	ns
	Level 3					
	ASI x ACQ	.35	.33			
				-.63	-.04	ns
Heart Rate						
	Level 1	.19	.18			
	Gender			3.08	.21	<.01
	Baseline HR			5.61	.38	<.001
	Level 2	.21	.19			
	ASI			1.37	.10	ns
	ACQ			1.27	.10	ns
	Level 3					
	ASI x ACQ	.21	.18			
				-.28	-.02	ns

Note. N = 229. Baseline SUDS: Subjective Units of Distress Scale one minute pre-challenge (Wolpe, 1958); ASI: Anxiety Sensitivity Index total score (Reiss et al., 1986); ACQ: Anxiety Control Questionnaire total score (Rapee et al., 1996). BPM: Breaths per minute/Respiration rate; SCL: skin conductance, HR: heart rate.

Figure 1: Model of panic vulnerability

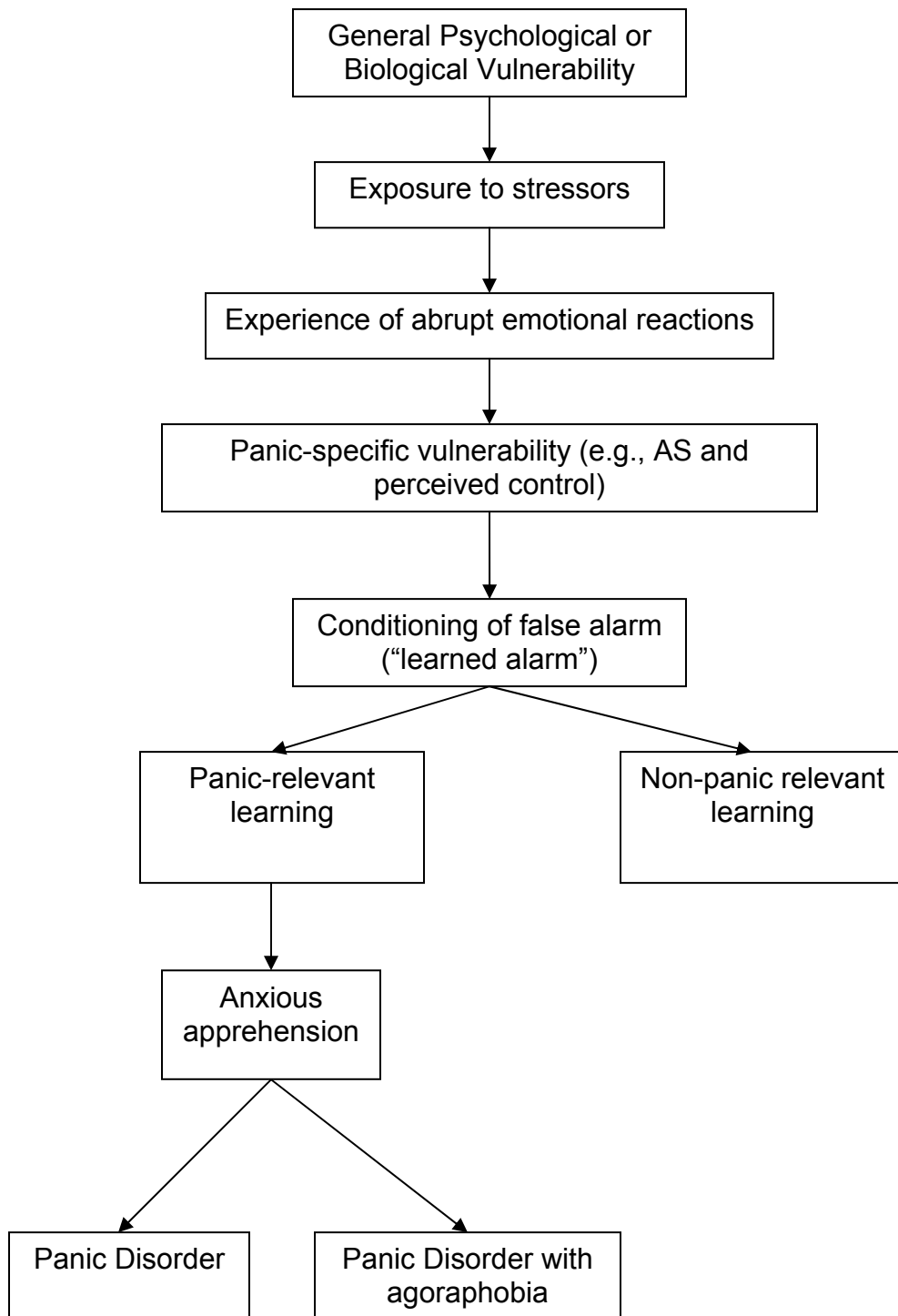


Figure 2: Conceptual model depicting perceived control moderating the effects of anxiety sensitivity predicting panic symptoms and avoidance

