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EVALUATING THE MODERATING ROLE OF ANXIETY SENSITIVITY ON  
SMOKING IN TERMS OF PANIC PSYCHOPATHOLOGY:  
A PROSPECTIVE TEST AMONG DAILY SMOKERS

A Dissertation Presented

by

Alison Christine McLeish

to

The Faculty of the Graduate College

of

The University of Vermont

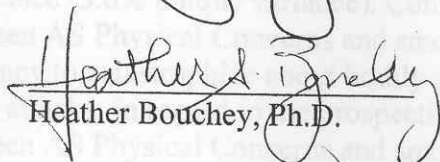
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Specializing in Psychology

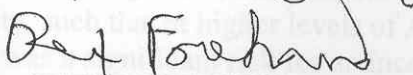
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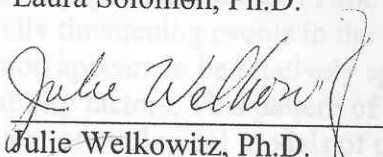
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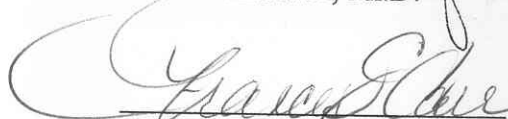
  
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## Abstract

The aim of the present investigation was to evaluate the moderating role of the physical concerns domain of anxiety sensitivity (AS) in the relation between smoking rate and panic vulnerability variables, both concurrently and prospectively, among a community-based sample of 125 daily smokers (60 females;  $M_{age} = 26.02$  years,  $SD = 10.98$ ). As hypothesized, there was a significant interaction between AS Physical Concerns and smoking rate in relation to agoraphobic avoidance, such that at higher levels of AS Physical Concerns and higher smoking rates, there was a risk for increased agoraphobic avoidance (3.6% unique variance). Contrary to prediction, however, the interaction between AS Physical Concerns and smoking rate did not significantly predict the tendency to catastrophize about bodily sensations, body vigilance, or lifetime history of panic attacks. In regard to the prospective analyses, there was a significant interaction between AS Physical Concerns and smoking rate in relation to Time 2 anticipatory anxiety, such that at higher levels of AS Physical Concerns and higher rates of smoking, there was a significant risk for an increase in anticipatory anxiety over the three-month follow-up period (5% unique variance). Contrary to prediction, the interaction between AS Physical Concerns and smoking rate did not significantly predict the occurrence of panic attacks during the three month follow-up period. The current findings suggest that daily smokers smoking at higher rates with high AS Physical Concerns may be more prone to engage in avoidance (Time 1 findings) and show increases in worry about potentially threatening events in the future (Time 2 anticipatory anxiety findings). This interaction appears to be relatively specific to only some aspects of panic-relevant vulnerability factors. This pattern of findings may be used to conceptually guide the refinement of etiological models of panic vulnerability that involve smoking behavior.

## Acknowledgements

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## Table of Contents

Acknowledgements.....	ii
List of Tables .....	v
List of Figures.....	vi
Introduction.....	1
Panic Disorder: Definition and Nature .....	2
Theoretical Models of Panic Disorder Development.....	4
Risk Factors: Conceptual Clarification.....	8
Smoking and Panic Disorder .....	10
Conceptual Model of Smoking and Panic .....	22
Present Study .....	27
Method .....	30
Participants.....	30
Measures .....	32
Time 1 Anxiety-related Measures.....	32
Time 1 Smoking-related Measures .....	35
Time 2 Measures.....	36
Procedure .....	37
Screening.....	37
Assessment.....	37
Follow-Up.....	38
Results.....	38

Statistical Analyses .....	38
Correlations for Theoretically-Relevant Variables.....	39
Time 1 Regression Equations .....	41
Time 2 Regression Equations .....	43
Graphical Representation of the Statistically Significant Interactions .....	44
Discussion.....	45
Findings from Time 1 Assessment .....	45
Findings from Time 2 Assessment .....	49
Clinical Implications.....	52
Study Limitations and Future Directions.....	53
Conclusions.....	55
References.....	57
Footnotes.....	76

## List of Tables

Table 1: Key Terms in Risk Factor Research .....	78
Table 2: Overview of Procedure .....	79
Table 3: Descriptive Data & Intercorrelations among Predictor & Criterion Variables ..	80
Table 4: Contribution of the Interaction between AS Physical Concerns and Smoking Frequency in Predicting Time 1 Panic-Relevant Variables .....	81
Table 5: Contribution of the Interaction between AS Physical Concerns and Smoking Frequency in Predicting Lifetime History of Panic Attacks .....	82
Table 6: Contribution of the Interaction between AS Physical Concerns and Smoking Frequency in Predicting Time 2 Panic Attacks.....	83
Table 7: Contribution of the Interaction between AS Physical Concerns and Smoking Frequency in Predicting Time 2 Anticipatory Anxiety.....	84



List of Figures

Figure 1: Conceptual Model depicting Anxiety Sensitivity Moderating the Effects of Smoking on Panic Vulnerability ..... 85

Figure 2: Time 1 Agoraphobic Avoidance as a function of AS Physical Concerns and number of cigarettes smoked per day among participants one-half of a standard deviation above and/or below the mean for each predictor. .... 86

Figure 3: Time 2 Anticipatory Anxiety as a function of AS Physical Concerns and number of cigarettes smoked per day among participants one-half of a standard deviation above and/or below the mean for each predictor. .... 87

## Introduction

Anxiety disorders are among the most common classes of psychopathology. In the National Comorbidity Survey and National Comorbidity Survey Replication (Kessler et al., 1994; Kessler, Chiu, Demler, & Walters, 2005; Kessler, Berglund, Demler, Jin, & Walters, 2005), anxiety disorders were more common than any other major group of diagnoses (with the exception of substance use disorders) with a lifetime prevalence rate of 25%. Aside from a high prevalence rate, anxiety disorders generally maintain a chronic, fluctuating course (Pine, Cohen, Gurley, Brook, & Ma, 1998), resulting in substantial impairment across the lifespan (Ferdinand & Verhulst, 1995). For instance, one international study (Ormel et al., 1994) found more than half of patients with Panic Disorder (PD) reported moderate to severe occupational dysfunction and physical disability; the disability was similar to that of major depressive disorder and greater than that for alcohol dependence. In addition to human suffering, anxiety disorders place a large burden on the financial and social resources of society. According to one recent estimate, the economic cost of anxiety disorders in the United States exceeds \$42 billion per year (Greenberg et al., 1999). Although there has been significant progress made in developing efficacious treatments for anxiety disorders among children and adults (e.g., Barlow, Gorman, Shear, & Woods, 2000), only a very small percentage of those in need of clinical services actually receive appropriate care [Institute of Medicine (IOM), 1989]. Interestingly, there have been very few attempts to develop and implement prevention programs for anxiety psychopathology, which has been suggested to be due, in part, to

the lack of understanding of what and how vulnerability processes impart risk for specific disorders (see Feldner, Zvolensky, & Schmidt, 2004, for an expanded discussion).

*Panic Disorder: Definition and Nature*

Panic Disorder is a debilitating disorder that affects approximately 1.7% of the adult U.S. population in any given year [National Institute of Mental Health (NIMH), 1999] and has a lifetime prevalence rate of 1.5%-3.5% worldwide [American Psychiatric Association (APA), 2000; Kessler, et al., 1994]. Panic disorder is characterized by recurrent, unexpected panic attacks and anxious apprehension about the possibility of experiencing future panic episodes (Bouton, Mineka, & Barlow, 2001). The disorder often is complicated by agoraphobia that can limit social involvement and/or personal mobility (APA, 2000), other psychological disorders, and physical health problems (Brown, Antony, & Barlow, 1995). Panic Disorder is regarded as a disorder of adulthood with a median age of onset of 24 (Burke, Burke, Regier, & Rae, 1990). However, the distribution of the age of onset for PD is bimodal with peaks at 15-24 and 45-54 years of age (APA, 2000). Thus, both younger adults and middle-aged adults are at risk for developing panic attacks and panic disorder, suggesting extending examinations of emotional vulnerability for this problem to relatively wide age ranges. In the NIMH Epidemiological Catchment Area study, Regier, Burke, and Burke (1990) found that PD occurs twice as often in women than in men (2.1% vs. .9%). Similar results were found in the National Comorbidity Survey, where the prevalence of PD was 2.5 times as likely in women as in men (Kessler et al., 1994).

Individuals with PD have one of the highest rates of service usage among the anxiety disorders (Greenberg et al., 1999). In fact, patients with PD may see as many as 10 or more physicians and undergo numerous expensive and unnecessary procedures before being diagnosed with the disorder (NIMH, 1995). PD is also associated with financial problems; in one investigation, 60% of those with PD were unemployed and 37% of men and 42% of women with PD received some sort of financial assistance (e.g., disability, welfare, unemployment; Leon, Portera, & Weissman, 1995). Despite the availability of efficacious treatments for PD, only 10-15% of individuals with the disorder receive empirically supported clinical services (Goisman et al., 1994). Moreover, there has been very little development in terms of preventive approaches for PD (APA, 2000; Kessler, et al., 1994).

It is noteworthy that a much larger percentage of the general population experience panic attacks without necessarily developing PD (Norton, Cox, & Malan, 1992). Typically, individuals who experience these nonclinical panic attacks do not experience these attacks as “spontaneous” or “uncued” as is generally the case in PD, but rather in certain threat-based contexts such as stressful daily experiences or traumatic life events (Bernstein et al., 2005). In fact, panic attacks are relatively common human experience and readily occur across various clinical conditions (Barlow, Brown, & Craske, 1994). From at least a heuristic level, some scholars have suggested panic attacks can be conveniently conceptualized as falling along a continuum of severity with nonclinical panickers scoring between clinical panickers and non-panickers on most measures of psychopathology (Cox, Endler, & Swinson, 1991). It is important to note,

however, that such a theoretical model (i.e., implicitly dimensional) has not been tested empirically. Nonetheless, clinical and nonclinical panickers tend to use different coping strategies to manage their panic (see Feldner, Zvolensky, & Leen-Feldner, 2004, for a review) and this aspect has been discussed, albeit not extensively tested, as one critical self-regulation difference that may help explain differential risk for developing panic disorder (cf. nonclinical panic attacks). For instance, descriptive research suggests nonclinical panickers employ positive coping strategies such as relaxation exercises, while clinical panickers typically use avoidance and distraction as coping strategies (Cox, Endler, Swinson, & Norton, 1992).

#### *Theoretical Models of Panic Disorder Development*

Several theories have been developed that attempt to explain PD. Although these theories are not necessarily mutually exclusive, they each offer a different “point of entry” in attempting to understand the etiology of PD. Biological theories point to an overly sensitive suffocation alarm as an explanation for PD (Klein, 1993). In normal individuals, when the suffocation alarm is activated, a “fight or flight” response occurs and the individual experiences symptoms of breathlessness and the urge to flee. From an evolutionary perspective, this system is purported to be adaptive as it might allow humans to survive natural disasters and attacks by predators. However, individuals with a hypersensitive suffocation alarm are believed to experience numerous “false alarms” where these same symptoms are experienced in the absence of any threat. Feelings of breathlessness are often followed by over-breathing and acute fear, which in these hypersensitive individuals can lead to a panic attack. Given that these panic attacks are

purported to be elicited by slight fluctuations in levels of carbon dioxide, this theory may account for patients' reports that their panic attacks seem to come "out of the blue." Empirical evidence, however, has not supported this theory. For example, Gorman et al. (2001) found that individuals with Premenstrual Dysphoric Disorder experienced panic attacks in response to 5% and 7% CO<sub>2</sub> inhalation at similar rates as individuals with PD. Thus, while individuals with PD experience greater levels of anxiety in response to CO<sub>2</sub> inhalation, it does not appear that this is necessarily due to specific physiological abnormalities. Moreover, a variety of other studies have found that suffocation-based fears (Eifert, Zvolensky, Sorrell, Hopko, & Lejuez, 1999; Taylor & Rachman, 1994) and changes in CO<sub>2</sub> are not associated with fear response to biological challenge (Schmidt, Telch, & Jaimez, 1996). These sources of data do not support major theoretical predictions derived from the suffocation-oriented theory of PD, and by extension, cast doubt on the validity of such a perspective.

Alternatively, Barlow (1991, 2002) and Bouton et al. (2001) posit that panic attacks are conditioning experiences that link anxiety and panic to both interoceptive and exteroceptive cues. This type of perspective represents a learning theory of PD development. Specifically, it is theorized that conditioned anxiety potentiates the next panic attack in a downward negative spiral that culminates in PD. In this context, these emotional events are not false alarms, but rather, learned alarms. The initial physical symptoms of a panic attack are associated with the later full blown panic attack. Thus, the individual becomes vigilant to slight fluctuations in physical functioning in order to "prepare" (automatically) for the next attack. Because of the similarity in the

unconditioned stimulus (US) and conditioned stimulus (CS), an index of “belongingness,” fairly robust conditioning is posited to occur. By avoiding situations that might elicit these interoceptive stimuli, the individual with PD prevents extinction of this conditioning. Although this learning theory perspective of PD development is a promising model for understanding PD, no empirical studies have been conducted to explicitly test this theory. There is, however, a variety of indirect evidence that would be consistent with this perspective, including that interoceptive-exteroceptive conditioning (e.g., fear response pairing with visually based internal stimuli such as heart rate) is quickly acquired and highly resistant to extinction (Forsyth & Eifert, 1996).

Clark’s (1986) cognitive theory of PD conceptualizes panic as stemming from catastrophic misinterpretations of bodily sensations. Individuals with PD perceive normal physical sensations in response to anxiety (e.g., heart palpitations) as a signal of impending doom (e.g., having a heart attack). This misinterpretation causes a further increase in anxiety, which then produces more physical sensations in a vicious cycle ending in a panic attack. This holds true especially for ambiguous autonomic stimuli. In fact, PD patients have been found to misinterpret ambiguous physical sensations more so than those with other anxiety disorders and non-clinical controls (Clark et al., 1997). Moreover, individuals who fear their own anxiety symptoms (i.e., high in anxiety sensitivity) report catastrophic interpretation of ambiguous situations even prior to the experience of panic attacks, suggesting the existence of a panic self-schema (Teachman, 2005). However, Rachman, Levitt, and Lopatka (1987) found that while panic episodes were associated with more threat-relevant and fearful cognitions as well as increased

physical symptoms, there were also a significant number of “non-cognitive” panic episodes (e.g., nocturnal panic). The cognitive theory of PD is unable to explain these non-cognitive panic episodes. Moreover, this cognitive theory of PD has been criticized theoretically because it is potentially unfalsifiable, particularly as it was originally conceived (McNally, 1999a).

Lastly, the Anxiety Sensitivity (AS) theory proposes that it is not the misinterpretation of bodily sensations per se, but fear of *actual* physical sensations that is the panicogenic process in PD development. AS is an individual difference variable that indexes a “fear of fear” or a fear of anxiety symptoms themselves. Individuals high in AS tend to believe that these physical sensations may cause serious physiological, psychological, or social consequences. For example, if a person perceives bodily sensations associated with autonomic arousal as a sign of imminent harm, this “high AS” individual will likely experience elevated levels of anxiety and be at an increased risk for panic. Moreover, this fear is posited to lead to avoidance of situations that would invoke these sensations (e.g., avoiding exercise). The AS theory has received a great deal of research attention in recent years with promising, independently replicated results. In fact, several prospective studies have shown that AS can predict later panic attacks and panic disorder (Schmidt, Lerew, & Jackson, 1997, 1999; Maller & Reiss, 1992) and that AS has exhibited greater specificity to panic than temperamental variables such as negative affectivity (Hayward, Killen, Kraemer, & Taylor, 2000). Extant research on AS is more fully discussed later in the paper.



Together, theoretical models of PD generally converge on the role of interoceptive distress and fears of such bodily perturbation in the generation of panic-related problems. Although naturally no one model can adequately account for all cases of PD development at the present time, there are some promising findings, particularly in regard to the AS theory. Nonetheless, it is noteworthy that none of the existing theories of PD development explicitly focus on the role of behavioral factors in panic etiology, and by extension, denote a specific role for behavioral factors in the disorder development. As will be discussed, this lack of focus on behavioral factors is striking given PD is highly associated with various types of addictive behaviors and that these behaviors typically precede the onset of panic attacks and PD (Zvolensky, Schmidt, & Stewart, 2003). In some respects, this lack of scientific attention to drug-related problems in PD is representative of a larger issue in anxiety disorder research: a failure to recognize and systematically examine the role of substance use problems as specific vulnerability factors for anxiety psychopathology (Zvolensky & Schmidt, 2004).

*Risk Factors: Conceptual Clarification*

Before proceeding further into a discussion of substance use problems in PD etiology, it is important to first clarify what is meant by the term “risk.” Most of the important theoretical work in defining risk concepts in contemporary clinical science has originated from cutting-edge work in developmental psychopathology by Kazdin, Kraemer, and their colleagues. They have defined a risk factor as a “characteristic, experience, or event that, if present, is associated with an increase in the probability (risk) of a particular outcome” (Kazdin, Kraemer, Kessler, Kupfer, & Offord, 1997, p.377).

Like psychological disorders, risk factors are not typically best understood as static entities. The association between risk factor and outcome depends on characteristics of the population (age, sex), characteristics of the risk factor (duration, point in development when exposed), other variables associated with the risk factor, and characteristics of the outcome (Kazdin et al., 1997). In brief, risk factors are probabilistic in that they affect the likelihood of a certain outcome but do not determine the outcome.

Risk factor research gives us an initial roadmap for identifying risk factors and a way to increase specificity in our theoretical models of the etiology of particular disorders. Kraemer et al. (1997) have conceptualized a hierarchy of influence of potential risk factors and outcomes (see Table 1). The first step in this progression is to determine whether or not there is an association between two characteristics. Next, temporal relations must be established in order to show that the characteristic is a risk factor for a particular outcome. Lastly, causality must be determined. A risk factor that is not causally involved in a disorder is deemed a “marker” and can be either variable or fixed – terms defined momentarily. If a risk factor is shown to influence the likelihood of the outcome when manipulated, then it can be considered a causal risk factor. For example, a great deal of recent research in the field of health psychology has attempted to elucidate the causes of obesity. One factor that has been highlighted as a potential cause of obesity is lack of exercise. In order to determine the nature of this association, studies would first have to show that lack of exercise and obesity are related, thus establishing them as correlates. The next step would be to determine whether or not lack of exercise preceded obesity. Research would then need to demonstrate that lack of exercise always preceded

obesity and that obesity never preceded lack of exercise. If this is shown to be the case, then lack of exercise would be considered a risk factor for obesity. It would then be necessary to determine what type of a risk factor lack of exercise may represent. If research did not show that lack of exercise caused obesity, yet still preceded obesity, then it would be considered a marker for obesity. Since lack of exercise is malleable, it would be a variable marker. Had this marker been gender, a variable that is not malleable (i.e., changeable), it would be a fixed marker. If lack of exercise were found to be a variable marker, future research would need to examine other correlates of lack of exercise to find alternative pathways to obesity. If, on the other hand, changes in amount of exercise changed one's risk for obesity, then exercise would be considered a causal risk factor for obesity. Although it is clear that exercise is not the only risk factor for obesity, it is one lifestyle variable that can be manipulated to decrease the likelihood of obesity. Collectively, this illustrative example highlights the importance and utility of explicating risk factor effects in terms of better understanding health-related problems.

#### *Smoking and Panic Disorder*

As briefly mentioned in the above section, there has been a general lack of attention paid to the role of addictive behaviors generally, and cigarette smoking specifically, in anxiety disorders research. This lack of attention is striking and potentially of great clinical concern, as there are likely bi-directional negative effects for addictive behaviors to increase the risk of panic-related problems as well as the converse (see e.g., Kushner, Abrams, & Borchardt, 2000; Kushner, Sher, & Beitman, 1990; Stewart, Samoluk, & MacDonald, 1999; Zvolensky & Bernstein, 2005; Zvolensky &

Schmidt, 2004; Zvolensky, Schmidt, & Stewart, 2003, for expanded discussions of this issue). Before discussing bi-directional influences, however, it is perhaps useful to point out one illustrative example of the degree of lack of knowledge of drug problems among treatment specialists using one of the most common of the addictive drugs – tobacco/cigarette smoking. One study recently examined knowledge and perceived competence regarding smoking cessation among mental health professionals who specialize in the treatment of anxiety disorders (Zvolensky, Baker, et al., 2005). Results indicated that therapists assess smoking behavior in only about 30% of clients, perceive themselves as “definitely unprepared” to deliver smoking cessation treatment, and only a minority (18%) have received formal training in empirically-based smoking cessation practices. When benchmarked against primary care physicians, anxiety specialists illustrated deficits on “basic” cessation counseling practices (e.g., assess for smoking behavior). Clearly, these data underscore that it is critically important to increase our scientific knowledge of smoking-related processes and clinical attention to their role in panic psychopathology.

*Co-occurrence of smoking and panic-related problems.* Smoking remains one of the leading preventable causes of death and disability in the U.S., accounting for 440,000, or 1 in 5, deaths each year [Centers for Disease Control and Prevention (CDC), 2004]. A number of efforts have been made to understand the associations between smoking and specific types of mental illness. The vast majority of this research has focused on schizophrenia and depressive disorders (e.g., Ginsberg, Hall, Reus, & Muñoz, 1995; Kinnunen, Doherty, Militello, & Garvey, 1996), whereas comparatively less attention has

been devoted to the link between smoking and anxiety disorders. This lack of attention is unfortunate, as anxiety disorders co-occur with smoking at rates that exceed those found in the general non-psychiatric population and many other psychiatric conditions (Amering et al., 1999; Beckham et al., 1997; Degenhardt, Hall, & Lynskey, 2001; McCabe et al., 2004; Orlando, Ellickson, & Jinnett, 2001; Pohl, Yeragani, Balon, Lycaki, & McBride, 1992). For instance, Lasser and colleagues (2000) recently found that among 4,000 respondents from the National Comorbidity Survey, current smoking rates for respondents with an anxiety disorder in the past month or lifetime were significantly greater than smoking rates among respondents with no mental illness. In this same study, reported rates of smoking among those with anxiety disorders were highest among individuals with panic-related problems (i.e., history of panic attacks and panic disorder) and other anxiety disorders where panic attacks are common (i.e., posttraumatic stress disorder and generalized anxiety disorder; Lasser et al., 2000). As a point of reference, the percentages of smoking among people with nonclinical panic attacks and panic disorder were greater than or equal to those found for major depressive disorder and dysthymia (Lasser et al., 2000). These studies also indicate that the observed associations between smoking and anxiety psychopathology are not due to sociodemographic characteristics (e.g., gender), other psychiatric conditions (e.g., major depressive disorder, alcohol dependence), or symptom overlap in diagnostic criteria for anxiety disorders and nicotine dependence (Zvolensky, Schmidt, & Stewart, 2003). In fact, a recent review found that, across all extant studies, the average rate of smoking among those with panic-related problems is approximately 40% and panic-related problems are

twice as prevalent among smokers compared to non-smokers (Zvolensky, Feldner, Leen-Feldner, & McLeish, 2005). Other research indicates that a greater proportion of individuals with PD report smoking at higher rates than individuals with social phobia and obsessive-compulsive disorder (McCabe et al., 2004). These data suggest that individuals with PD are likely to be heavier smokers than persons with other anxiety disorders, and perhaps are more dependent on nicotine.<sup>1</sup>

*Impact of Smoking on Panic-related Vulnerability.* There are, of course, a number of possible bi-directional influences between smoking and panic processes and outcomes (e.g., predisposition models, pathoplastic models, spectrum models, scar models; Clark, Watson, & Mineka, 1994). Recent research has begun to elucidate that panic vulnerability factors such as AS and a history of panic attacks are associated with (1) increases in the chance of early lapse and subsequent relapse to smoking (Brown, Kahler, Zvolensky, Lejuez, & Ramsey, 2001; Zvolensky, Feldner, Eifert, & Brown, 2001; Zvolensky, Lejuez, Kahler, & Brown, 2004); (2) heightened negative affect during quit attempts (Zvolensky, Baker et al., 2004; Zvolensky, Lejuez, et al., 2004; Zvolensky, Schmidt, et al., 2005); and (3) smoking principally aimed to reduce negative affect states (including withdrawal symptoms), but not for other motivational reasons (e.g., handling, taste; Comeau, Stewart, & Loba, 2001; Novak, Burgess, Clark, Zvolensky, & Brown, 2003; Zvolensky, Feldner, et al., 2004; Zvolensky, Kotov, Antipova, & Schmidt, 2005). These data highlight the potentially important role of panic vulnerability factors in relapse-related problems and the maintenance of smoking more generally (i.e.,

predisposition models; Clark et al., 1994). Yet, smoking also may be associated with increased risk of developing and/or exacerbating panic-related vulnerability.

Evidence has shown that smoking is associated with more severe panic problems (e.g., pathoplastic models; Clark et al., 1994). A number of studies have been completed in this domain. First, regular smokers with PD have been found to report more severe anxiety symptoms (especially anticipatory anxiety) and social impairment than non-smokers with PD (McCabe et al., 2004; Zvolensky, Schmidt, & McCreary, 2003). These findings appear to be specific to PD as compared to anxiety disorders in general. A recent study by Morissette, Brown, Kamholz, & Gulliver (in press) found that smokers with anxiety disorders, as compared to their non-smoking counterparts, reported higher levels of AS (overall, mental, and physical concerns), negative affect, anxiety, and agoraphobic avoidance. However, this association appears to be due to the influence of panic disorder with agoraphobia (PDA); when looking at smokers and nonsmokers with any anxiety disorder other than PDA, no differences were found. Similar to previous findings, among those with PDA, smokers reported greater interoceptive sensitivity, anxiety sensitivity (overall, mental, and physical concerns), and life interference than nonsmokers. Second, a biological challenge study indicated that smokers with PD reported greater levels of anxiety and bodily distress than smokers without PD and than nonsmokers with PD (matched on comorbidity criteria) at the post-challenge assessment and recovery period, but no differences in autonomic responding during the challenge or in recovery (Zvolensky, Leen-Feldner et al., 2004). Also, in terms of rate of recovery, the linear decrease in anxiety, but not bodily distress, was significantly steeper for nonsmokers with

PD than for smokers with PD. These data suggest smokers with PD are at risk for delayed anxiety-related recovery from panic-relevant bodily perturbation, and moreover, due to selection criteria, cannot be attributed to medical history or psychiatric comorbidity. Finally, another investigation explored panic-relevant cognitive processes in a sample of persons ( $n = 70$ ) who met criteria for either (1) a positive nonclinical panic attack history and regular smoking (smoking at least 10 cigarettes per day for  $\geq 12$  months; PASM), (2) a positive nonclinical panic attack history but no history of smoking (PA), or (3) regular smoking history alone (smoking at least 10 cigarettes per day for  $\geq 12$  months; SM) (Zvolensky, Forsyth, Fuse, Feldner, & Leen-Feldner, 2002). PASM participants demonstrated significantly greater body vigilance and anxiety sensitivity mental incapacitation concerns compared to persons in either the PA or SM groups. The observed effects, again, could not be attributed to self-reported physical health status or history of medical problems. Although thus far restricted to a small number of cross-sectional investigations, extant work indicates that there is indeed correlational evidence that smoking is associated with increased risk of panic-related problems among persons with a current history of PD and nonclinical panic attacks. These data help to establish an association between these factors, but do not elucidate the temporal nature of that association, and hence, cannot be used to infer etiological significance.

In addition to being associated with more severe panic problems, research suggests smoking actually increases the risk of panic problems (predisposition models; Clark et al., 1994). First, in regard to temporal nature, it is important to note that although the onset of daily smoking typically occurs between the ages of 15 and 20 and rarely after



age 25 (Breslau, Johnson, Hiripi, & Kessler, 2001), the median age of onset for PD is typically 24 (Burke et al., 1990). This sequence would indicate that smoking initiation generally precedes panic onset. As now will be discussed, outside of this general evidence, there are a number of studies that have more specifically examined the temporal relations between smoking onset and subsequent risk for panic vulnerability.

Using data from the Epidemiologic Study of Young Adults and the National Comorbidity Survey Tobacco Supplement, Breslau and Klein (1999) found that daily smoking increased the risk for the first occurrence of a panic attack or onset of PD, while there was no significant risk for panic attacks or PD increasing the risk for smoking. Individuals in the Epidemiological Study of Young Adults who were daily smokers and had no history of major depression, specifically, were 3.96 times more likely to experience a panic attacks than non-smokers (also without a history of major depression). There was also a significant difference between the likelihood of experiencing a first panic attack among those who continued to smoke and those who quit smoking (Hazard Ratios = 4.71 and 0.21, respectively). Results were even more striking among persons with PD. After controlling for a history of major depression, daily smokers were 13.13 times more likely to have PD than non-smokers. Again, the hazard ratio of smoking initiation after the onset of PD was not significant. Results from the National Comorbidity Study [reported in the same Breslau & Klein (1999) study], although somewhat lower, paralleled the results from the Epidemiologic Study of Young Adults. In a subsequent study by Breslau, Novak, and Kessler (2004) using the smoking supplement to the National Comorbidity Survey, results indicated that current but not

past smoking predicted later onset of PD and agoraphobia (Odds Ratios = 2.6 and 4.4, respectively, without controlling for comorbid disorders) and that the risk of developing these disorders decreased by half with each standard deviation unit of time since quitting. Interestingly, neither nicotine dependence nor early onset smoking predicted the first onset of psychiatric disorders. However, these studies were cross-sectional and employed a unimethod (self-report) assessment protocol. Additionally, the age of onset for both daily smoking and psychiatric disorders were assessed through retrospective recall and subsequently subject to memory biases. Thus, while these results suggest that smoking precedes the onset of panic, the reverse pathway cannot be definitively ruled out.

Addressing one of the limitations of the Breslau and Klein (1999) and Breslau et al. (2004) studies, smoking has been established as a risk factor for PD through several prospective studies. Johnson et al. (2000) conducted a longitudinal study examining the relation between heavy cigarette smoking (> 20 cigarettes per day) and anxiety disorders in adolescents and young adults. Results showed that anxiety disorders during adolescence were not significantly related to smoking in young adulthood, however, smoking in adolescence increased the risk for developing agoraphobia, GAD, and PD during early adulthood. These effects were above the variance accounted for by temperament, family history of psychopathology, drug/alcohol use and other theoretically-relevant factors. Adolescents who were heavy smokers, specifically, were 15.58 times more likely to develop PD in early adulthood than non-smokers. Interestingly, adolescents who smoked fewer than 20 cigarettes per day were not at elevated risk for the development of later anxiety disorders, potentially suggesting

heavier smoking levels impart greater panic-related risk. There are several interpretive caveats to take into consideration with this study. First, assessments were unimethod (interviews only) and therefore potentially subject to memory biases, recall distortion, and capitalization of method variance. Second, because of low numbers of subjects with certain diagnoses (< 10), sub-threshold levels of anxiety disorders were included in the analyses making it difficult to fully discern the parameters of the association between smoking and clinical levels of panic.

A second longitudinal study of smoking and panic was conducted in Germany over a 4 year period with over 2,500 participants (aged 14-24 years at baseline; Isensee, Wittchen, Stein, Höfler, & Lieb, 2003). Similar to Johnson et al. (2000), researchers found a unidirectional association between prior smoking and later onset of panic attacks/PD. Nicotine dependent smokers had an increased risk of later onset of panic attacks. The risk for onset of PD was significant only among those who were nicotine dependent smokers at baseline, although this association failed to reach statistical significance after controlling for comorbid disorders. However, there was also a significant association between panic and later onset of smoking dependence, making the temporal relation between smoking and panic unclear. This study is noteworthy for two reasons. First, it did not fully replicate the Johnson et al. (2000) study results in terms of directionality. Second, it was the first to measure nicotine dependence, rather than cigarettes consumed per day, as the primary smoking predictor variable. There are important differences between cigarette smoking exposure (cigarettes consumed per day) and nicotine dependence. In fact, these variables typically share only moderate

associations with one another (Piper et al., 2004), potentially owing to relatively poor conceptualizations of tobacco dependence. However, the Breslau et al. (2004) study would indicate that smoking rate rather than nicotine dependence may be the primary variable to examine. Future research will need to examine these two variables more systematically in order to explicate the role of smoking in regard to panic-related problems.

More recent research has attempted to identify other variables that influence the smoking panic association. Zvolensky, Sachs-Ericsson, Feldner, and Schmidt (in press) evaluated a moderational model of neuroticism on the association between smoking level and panic attacks and panic disorder using data from the National Comorbidity Survey (see below for a discussion of moderating variables). Participants ( $n = 924$ ) included current regular smokers, as defined by reporting smoking regularly during the past month. Findings indicated that a generalized tendency to experience negative affect (neuroticism) moderated the effects of maximum smoking frequency (i.e., number of cigarettes smoked per day during the period when smoking the most) on lifetime history of panic attacks and panic disorder even after controlling for drug dependence, alcohol dependence, major depression and dysthymia, and gender. These effects were remarkably specific to panic attacks and panic disorder, as no such moderational effects were apparent for other anxiety disorders. It also is noteworthy that the main effects of neuroticism and smoking frequency shared very little variance with one another ( $< 1\%$ ). This finding is important, as it indicates that these two panic risk factors are tapping different vulnerability processes.

Finally, McLeish, Zvolensky, Bonn-Miller, and Bernstein (in press) examined the moderating role of perceived health in the association between smoking and panic. In a sample of daily smokers ( $n = 220$ ), the interaction between health perceptions and smoking rate incrementally predicted anxiety variables, but not depressive symptoms even after controlling for alcohol consumption and gender. Furthermore, perceived health shared no variance with smoking rate, indicating that these two variables are unique factors. These results, in conjunction with Zvolensky, Sachs-Ericsson, et. al. (in press), suggest individual differences in certain affect variables that enhance emotional reactivity or perhaps learning may be relevant to understanding which smokers experience or go on to develop panic problems.

Together, a number of independently replicated investigations have found smoking is associated with increased risk for developing panic problems. However, most of the investigations are cross-sectional, focus on diagnostic status as a proxy for “panic problems,” rather than assess for theoretically-relevant panic processes (e.g., types of thinking patterns, behavioral styles), and do not confirm smoking status via biochemical verification. Perhaps most importantly, all but two studies have focused on documenting a main effect for smoking (see below for an additional example of a moderating effect). This research focus made sense given the overall level of knowledge development in the area. However, focusing solely on the main effect of smoking, as illustrated by the Zvolensky, Sachs-Ericsson et al. (in press) and McLeish et al. (in press) results, may be problematic and potentially misleading. To build upon this area of work, we need to begin to identify moderators for the observed smoking effects.

As defined by Baron and Kenny (1986) moderating variables are those that affect the direction or strength of association between an independent and dependent variable. In the present study, a moderator can be usefully conceptualized as a variable that influences the association between smoking and panic vulnerability. In contrast, mediators (variables that influence the relations between two correlated factors) serve to qualify and explicate the nature of the observed co-occurrence. In general, it is useful to conceptualize moderators as a pre-existing individual or environmental characteristic that increases or decreases the risk of a certain outcome (e.g., promoting greater panic attacks or greater risk of relapse). In contrast, mediators can be usefully conceptualized as factors that are “triggered” by the presence of a variable (e.g., smoking) and thereafter serve to account for the relation between that variable and an outcome (e.g., panic attacks).

In moderation, although both the independent and moderator variables may significantly predict an outcome variable, it is the *interaction* between the two that is of interest. Thus, moderators help to specify for whom or under what conditions a given risk factor like smoking has negative (in this case, panic-related) effects. In short, they help psychopathologists identify subpopulations with *possibly* (but not definitely) different causal mechanisms or course of illness. As such, they provide critically important information that can be used for highly pragmatic reasons, including guiding intervention planning. For example, they might suggest that certain pockets or collections of individuals might be the most apt to develop panic-related problems and therefore it may make the most sense to focus clinical activities (e.g., implementation of prevention programs) on this particular sub-group in the population.

Considering the above mentioned challenges to previous work, we now turn to a discussion of potentially moderating variables for smoking in regard to panic vulnerability.

### *Conceptual Model of Smoking and Panic*

An emerging pressing question from extant work is: what variables may moderate the effects of smoking on panic-related vulnerability? Zvolensky, Schmidt, and Stewart (2003) and Zvolensky and Bernstein (2005) have offered an integrative theoretical model for better understanding smoking-related effects on panic psychopathology. This model specifies a number of different theoretically-relevant pathways for smoking to exert negative effects on panic outcomes [e.g., via its effects on physical health functioning, direct pharmacological effects, perceived health status, promotion of life stress (particularly health-related adverse events and potentially time pressures secondary to “integrating” smoking into one’s occupational and personal life), maladaptive coping, and withdrawal symptoms] and a number of individual difference and contextual factors that can help clarify (or place explanatory parameters on) these pathway specific effects.

The focus of the present study is on clarifying individual difference characteristics that might moderate the effects of smoking on theoretically-relevant panic-related processes. Toward this end, there needs to be an attempt to conceptually integrate important work on AS and smoking in one overarching model. As briefly discussed above, AS is a dispositional, trait-like cognitive characteristic that is unique from the temperamental variable of trait anxiety (McNally, 1999b) and is theorized to predispose individuals to the development of panic problems (Reiss & Havercamp, 1996). For

example, if a person perceives bodily sensations that are associated with autonomic arousal as a sign of imminent personal harm, this “high anxiety sensitive” individual is theorized to experience elevated levels of anxiety and be at an increased risk for a panic attack. At least three lines of research have strongly supported this line of theorizing. First, prospective studies with adolescents and adults indicate AS predicts the future occurrence of panic attacks and worry about the future occurrence of such attacks (Schmidt et al., 1997; Weems, Hayward, Killen, & Taylor, 2002). These same prospective studies and other cross-sectional investigations indicate AS is relatively specific to PD and does not covary with other phenomena distinct from the syndrome (e.g., depression; Schmidt, Lerew et al., 1999). Second, AS is a significant predictor of responses to panic provocation procedures in the laboratory even after controlling for negative affectivity (Zinbarg, Brown, Barlow, & Rapee, 2001); these effects are limited to AS Physical Concerns, rather than Mental or Social Concerns. Thus, fear of bodily sensations and interoceptive cues are particularly relevant to panic vulnerability. Finally, AS is elevated among persons with a history of PD compared to those without the disorder (Taylor, Koch, & McNally, 1992). Because AS also decreases with remission of panic psychopathology through intervention (Telch et al., 1993), unlike many other panic risk factors (e.g., family history of PD, personal history of panic attacks), it can easily be targeted for therapeutic change in future prevention work.

AS may be critically important to PD vulnerability by serving to moderate the effect of smoking on the development of panic-related processes (see Figure 1 for a schematic of this process). Smoking promotes interoceptive sensations in a number of



ways, including withdrawal symptoms, cardiopulmonary impairment, and respiratory irritations, as well as medical diseases (CDC, 2004). In fact, recent research suggests one reason for the link between smoking and panic is due to the effects of smoking on respiratory function (Caldirola, Bellodi, Cammino, & Perna, 2004). Among persons with high levels of AS (particularly Physical Concerns), such interoceptive sensations are likely to be experienced as anxiety-provoking. Indeed, AS Physical Concerns has been shown to be a significant predictor of fear responding to bodily sensations during biological challenge (Brown, Smits, Powers, & Telch, 2003; Carter, Suchday, & Gore, 2001; Zinbarg et al., 2001; Zvolensky et al., 2002). Specifically, AS may increase the likelihood that such interoceptive events will be interpreted as uncontrollable or personally threatening, thereby intensifying anxious responding. The high AS Physical Concerns individual experiencing bodily sensations related to smoking would therefore be exposed to more frequent and intense aversive interoceptive learning trials. In this manner, smoking-related cues concerning somatic arousal or other interoceptive experiences are more likely to become phobic stimuli (Barlow, 2002). In contrast, individuals low in AS Physical Concerns may be less susceptible to the panic-related effects of smoking because they are less fearful of bodily sensations.

In this context, it is important to remember that cues that trigger panic attacks are not always immediately obvious to individuals, thus generating the perception that panic attacks are “out of the blue.” Additionally, interoceptive cues are typically perceived as less predictable than exteroceptive cues (Craske, 1991; Lejuez, Eifert, Zvolensky, & Richards, 2000). These latter points are important, because some of smoking-related

interoceptive effects (e.g., withdrawal) may be “predictable” and “expected” for regular smokers. However, it is unlikely smokers will *always* (i.e., on every single occasion) be aware of these cues and experience them as “predictable.” Moreover, even expected aversive bodily experiences may be potentially panic-relevant events for certain individuals.

Although not the focus of the present study, it is important to note that this “affective bind” might place these high-risk persons at risk for smoking as an affect management tactic. In fact, numerous lines of evidence support the idea that people, including regular smokers, use tobacco as a means of regulating their mood and coping with stress, attributing their smoking to its alleged anxiolytic properties (Frith, 1971); reliably reporting that they smoke more when stressed or anxious (Shiffman, 1993); and holding the expectation that smoking helps reduce negative affect (Copeland, Brandon, & Quinn, 1995). Although no single mechanism can presently explain the association between smoking and negative affect, a key finding that has emerged from this literature is that smoking effects on anxiety-related states are highly dependent on other factors related to affective processing (Gilbert & Gilbert, 1998). Specifically, research suggests that smoking-related effects on emotional processing are largely indirect and variability in response to smoking is strongly affected by individual difference factors (Kassel, Stroud, & Paronis, 2003).

There have only been two tests of the AS-smoking moderator hypothesis in regard to panic vulnerability. In one study, the sample was drawn from the population of adult residents of Moscow using a geographic sampling method, producing an

epidemiologically-defined (i.e., representative) sample (Zvolensky, Kotov, Antipova, & Schmidt, 2003). Results of this cross-sectional study indicated that AS moderated the effects of smoking ( $n = 95$  daily smokers from a larger sample of about 400 persons), as indexed by cigarettes per day ( $m = 15$  cigarettes per day), in terms of level of agoraphobic avoidance; this significant interaction accounted for approximately 10% of unique variance after controlling for their respective main effects and the theoretically-relevant factors of problem alcohol use and negative affectivity. No interaction, however, was found for panic attacks, potentially due to the fact that assessment of this factor was restricted to the past (most recent) week to enhance the validity of panic reports (but probably truncating variability). It also is noteworthy that AS and cigarette smoking were not correlated with one another, suggesting that they represent different risk factors for panic problems. The second test of the AS-smoking moderator hypothesis was conducted with a community sample of adolescents ( $n = 206$ ; Leen-Feldner, et al., in press). Results indicated that after controlling for gender, negative affect, and the main effects of AS and smoking rate, AS moderated the effects of smoking status (yes/no) in terms of panic attack symptoms (3% variance) and somatic complaints (1% variance), but not depression. Specifically, those individuals who were both current smokers and high in AS reported the highest rate of panic-related symptoms. As with the Zvolensky, Kotov, et al. (2003) study, AS and smoking shared little variance with one another providing further support that these two variables represent distinct risk factor for panic problems.

Overall, these findings suggest smokers are not a homogeneous group in regard to their risk for panic problems and that individuals differences in AS (or other affect-

enhancing factors) are a key factor in accounting for such differences. However, the results are limited in a number of important ways. First, the studies were cross-sectional in nature. Second, they relied on self-report and interview methodologies. Third, as with earlier studies, smoking status was not confirmed with biochemical assessments. Fourth, they focused on the general AS factor, leaving unclear whether or not the observed effects were better accounted for by one or more of the specific sub-domains (i.e., AS Physical Concerns, but not Mental or Social Concerns). Finally, there was a limited range of theoretically-relevant dependent variables and none focused on panic-related information processing.

#### *Present Study*

The overarching purpose of the proposed investigation was to replicate and extend past work by testing the moderating role of AS in regard to smoking frequency effects on panic vulnerability among a sample of adult regular smokers. Within this context, there were two main conceptually related, albeit distinct, domains to investigate: one dealing with a cross-sectional test (to broaden past work by identifying associations with heretofore undocumented panic-related processes) and the other involving a prospective test (to document changes in panic attacks and associations with anticipatory anxiety across time). In both cases, the present investigation sought to test the interaction between the physical concerns sub-domain of AS and cigarettes per day in regard to panic-relevant processes implicated in the aforementioned biopsychosocial model of panic disorder etiology (Zvolensky, Schmidt, & Stewart, 2003; Zvolensky & Bernstein, 2005). Cigarettes per day, rather than nicotine dependence, may be particularly relevant

to panic vulnerability, as it is a more direct index of “exposure” or “experience” and not addiction (i.e., tobacco dependence). That is, it may be a more direct assessment of “smoke exposure” and thereby presumably be more likely than tobacco dependence to be associated with increased risk of bodily sensations (e.g., via lung impairment). Likewise, AS Physical Concerns is the most theoretically-relevant composite for panic vulnerability and therefore the focus of the present study.

In regard to the cross-sectional test, it was expected that after controlling for negative affectivity and weekly average alcohol consumption (frequency x quantity composite), the interaction between the Physical Concerns subfactor of AS and cigarettes per day would be uniquely and significantly predictive of: (1) the tendency to catastrophize about bodily sensations (using Agoraphobic Cognitions Questionnaire), (2) body vigilance (using Body Vigilance Questionnaire), (3) agoraphobic avoidance [using interview-rated pre-morbid (i.e., prior to panic disorder development) agoraphobic avoidance behavior via the Panic Disorder Severity Scale], and (4) lifetime history of panic attacks (assessed via the SCID-NP [non patient version] panic attack module). These hypotheses were premised on previous work documenting an interactive effect of AS on smoking outcomes in terms of self-reported agoraphobic avoidance (Zvolensky, Schmidt, & McCreary, 2003) and the previously discussed conceptual model which suggests through learning experiences with smoking-related interoceptive cues (e.g., withdrawal symptoms, lung impairment) smokers with high, but not low, levels of fear of bodily sensations will be more apt to focus attention on such events, misinterpret/catastrophize them as personally dangerous, and cope with them via

avoidance-oriented affect regulation strategies (including but not limited to smoking) because they are perceived as personally “harmful” or “personally threatening.”<sup>2</sup>

In terms of the prospective test, it was expected that after controlling for Time 1 levels of panic attacks (or anticipatory anxiety in the second analysis), the interaction between the Physical Concerns subfactor of AS and cigarettes per day would, within a 3-month prospective assessment, be uniquely and significantly predictive of: (1) total frequency of panic attacks (assessed via the SCID-NP [non patient version] panic attack module) and (2) anticipatory anxiety (assessed via the Panic Disorder Severity Scale). This model did not use the same covariates as the cross-sectional tests, as the total sample size was smaller due to (a) studying only those persons with panic attacks and (b) attrition. The second set of hypotheses were attempting to document theoretically-relevant associations between the AS Physical Concerns and cigarettes per day interaction and future-based panic-relevant emotional vulnerability. Overall, it was expected that the form of the significant interaction would indicate persons high in AS Physical Concerns and average number of cigarettes consumed per day, compared to persons scoring low on one or both of these measures, would be more likely to experience a panic attack during the three-month follow-up period and exhibit higher levels of Time 2 anticipatory anxiety. Although this prospective test naturally was not focused on developmental processes per se due to the relatively limited window of time allowed in the assessment, it did permit us to chart the degree of variability (with a directional focus) in one key panic-relevant process. In this sense, it represents an

important facet of study at this stage of research development in regard to panic-smoking vulnerability processes.

## Method

### *Participants*

The sample consisted of 125 daily smokers (60 females;  $M_{age} = 26.02$  years,  $SD = 10.98$ ). Participants were recruited through the University of Vermont using advertisements for daily smokers in the campus newspaper and flyers posted on bulletin boards located in buildings and commons areas around campus. Participants also were recruited through the general community using newspaper advertisements and flyers posted in a local, well-traveled marketplace as well as in local restaurants and bars; identical advertisements were used for both the university and community sectors. The racial composition of the studied sample generally reflected that of the local population (State of Vermont Department of Health, 2000): approximately 95% of the sample was Caucasian, 4% African-American, and 1% other. Approximately 10% of the sample had at least a 4-year college education, 74% had some college education, 10% had a high school degree or the equivalent, and the remaining 6% did not have a high school education.

Participants smoked on average 17.6 cigarettes per day ( $SD = 8.43$ ), had smoked cigarettes regularly for 7.61 ( $SD = 8.49$ ) years, began cigarette smoking at a mean age of 13.48 ( $SD = 2.91$ ) years, and considered themselves regular smokers by a mean age of 15.79 ( $SD = 2.8$ ) years. When smoking tobacco the heaviest, participants averaged 24.43 ( $SD = 12.43$ ) cigarettes per day. The average level of nicotine dependence, as indexed by

the Fagerstrom Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991), was 3.37 ( $SD = 2.02$ ); this reflects a low level of overall nicotine dependence. Expired carbon monoxide (CO) levels also were evaluated to verify smoking status. The average CO level for the current sample was 16.2 ppm ( $SD = 11.23$ ); scores above 8 ppm are considered indicative of regular smoking (please see Smoking Measures section for details). 69.6% ( $n = 87$ ) of the participants were regular alcohol users, drinking 5 to 6 alcoholic beverages approximately 2 to 3 times per week.

Participants reported the following lifetime history of medical problems: 37.6% had allergies, 16.8% had experienced some type of head injury, 9.6% had been diagnosed with hypertension, 8% had been diagnosed with heart problems, 16% had been diagnosed with asthma, 6.4% had some other form of respiratory disease, and 2.4% had epilepsy. Participants were administered the Structured Clinical Interview for DSM-IV Axis I Disorders- Non-Patient Edition (First, Spitzer, Gibbon, & Williams, 1995) and reported the following history of current or past psychiatric problems: 28% had major depressive disorder, 25.6% had experienced non-clinical panic attacks, 11.2% had post-traumatic stress disorder, 4.8% had generalized anxiety disorder, 4.8% had social phobia, and 2.4% had obsessive-compulsive disorder.

Participants were excluded from the study if they displayed limited mental competency or the inability to give informed, written consent. Participants were not excluded for medical or psychiatric illness other than panic disorder. Participants with panic disorder were excluded from the study, as the aim of the study was to explore vulnerability processes for panic disorder; therefore, if panic disorder was not



exclusionary criterion, it would not be possible to ascertain if the AS Physical Concerns by smoking rate effects were simply attributable to this condition rather than being a potential risk-conferring process.

### *Measures*

#### *Time 1 Anxiety-related Measures*

*Structured Clinical Interview for DSM-IV Axis I Disorders- Non-Patient Edition (SCID-NP)*. The SCID-NP (First et al., 1995) is a well-established diagnostic interview for psychiatric problems, including panic attacks. It assesses Axis I disorders and provides a sub-module for panic attacks (including limited symptom attacks). The interview was administered in full at Time 1 in order to determine participants' history of psychiatric problems. Reliability ratings by an independent rater (MJZ) were completed on a random selection of 20% of the protocols, with no cases of disagreement being noted.

*Anxiety Sensitivity Index (ASI)*. The ASI (Reiss, Peterson, Gursky, & McNally, 1986) is a 16-item measure that asks respondents to rate on a 5-point Likert scale (0 = *very little* to 4 = *very much*) the degree to which they fear negative consequences stemming from anxiety symptoms. Responses to each item are summed to provide a total score from 0-64. Previous research indicates that the ASI is made up of one higher-order factor (ASI Total Score) and three lower-order factors: Physical, Psychological, and Social Concerns (Zinbarg, Barlow, & Brown, 1997; Stewart, Taylor, & Baker, 1997; Rodriguez, Bruce, Pagano, Spencer, & Keller, 2004). The ASI shows adequate test-retest reliability ( $r = .75$  for two weeks), criterion validity (e.g., individuals with agoraphobia

score higher than those with other anxiety disorders and those with no disorder), and is distinct from trait anxiety (Reiss et al., 1986). The ASI also shows specificity in its relation to anxiety but not to depression (Schmidt, Lerew, & Joiner, 1998). Recent findings converge on the observation that the Physical Concerns dimension, specifically, is most relevant to panic attack vulnerability (Zinbarg et al., 2001; Rodriguez et al., 2004). Thus, the AS Physical Concerns dimension was employed as a predictor variable at Time 1 in the present study.

*Agoraphobic Cognitions Questionnaire (ACQ)*. The ACQ is a 14-item scale measuring thoughts around the negative consequences of experiencing anxiety (Chambless, Caputo, Bright, & Gallagher, 1984). Items are rated on a 5-point Likert scale from (1) *thought never occurs* to (5) *thought always occurs*. The ACQ is comprised of 2 factors: social/behavioral concerns and physical concerns. The ACQ has been shown to have high internal consistency (Cronbach alpha = .87), moderate test-retest reliability ( $r = .67$  for one month) and sensitivity to changes due to treatment (Chambless et al., 1984). The ACQ can also discriminate clinical from non-clinical groups, especially individuals with anxiety disorders (Chambless & Gracely, 1989). The ACQ total score was used to index anxiety related cognitions and was administered at Time 1 (a criterion variable).

*Body Vigilance Scale (BVS)*. The BVS was employed to assess attentional focus on somatic symptoms (Schmidt, Lerew, & Trakowski, 1997). The BVS is a 4-item instrument in which respondents indicate on an 11-point Likert-type scale (0 = *none* to 10 = *extreme*) the degree to which they agree with a particular statement regarding attentional focus on body sensations and related processes. Specifically, three of the items

measure attentional focus, perceived sensitivity to changes in body sensations, and the average duration of time spent attending to body sensations. A fourth item involves having participants rate their attention to 15 body sensations, as defined by the DSM-IV physical symptoms for panic attacks. Responses to the fourth item are averaged to yield a single score for that item. Summing the four items derives a total score for the BVS. Research suggests that the BVS has adequate internal consistency ( $\alpha = .75$ ) and can be used to assess changes in bodily attention during cognitive-behavioral treatment for panic disorder (Schmidt, Lerew, & Trakowski, 1997). The BVS was administered at Time 1 in the present study and served as a primary index of body vigilance.

*Panic Disorder Severity Scale (PDSS)*. The PDSS is a semi-structured interview rating scale for PD (Shear, et al., 1997) that includes ratings of panic frequency and intensity, anticipatory anxiety, and avoidance of sensations and situations, and impairment in work and social functioning. Each of these symptoms is rated on a 0 (*None*) to 4 (*Extreme*) scale. The PDSS has good psychometric properties (Shear et al., 1997; Shear et al., 2001). In our lab, two clinicians making PDSS ratings in a dual interview were found to have consistently high reliability (Zvolensky, Leen-Feldner et al., 2004). In the present investigation, we computed two ratings: (1) anticipatory anxiety using the anticipatory anxiety question (item 3) and (2) agoraphobic avoidance using a composite of avoidance of situations and avoidance of bodily sensations (items 4 and 5). These ratings were examined separately because past work had shown relations of AS by smoking in regard to avoidance and other anxiety constructs (Zvolensky, Kotov et al.,

2003). Thus, it was important to separate avoidance from other anxiety factors relevant to panic vulnerability.

*Positive Affect Negative Affect Schedule (PANAS).* The PANAS is a mood measure commonly used in psychopathology research (Watson, Clark, & Tellegen, 1988). It assesses two global dimensions of affect: negative and positive. Only the negative affectivity scale (PANAS-NA) was used in this study. A large body of literature supports validity of the PANAS (Watson, 2000). For example, the PANAS-NA possess good internal consistency (Cronbach's alpha = .84-.87) and reliability ( $r = .71$ ) (Watson et al., 1988). In the present investigation, the PANAS-NA subscale was employed as a covariate.

#### *Time 1 Smoking-related Measures*

*Smoking History Questionnaire (SHQ).* Smoking history and pattern was assessed with the SHQ, a measure that includes items pertaining to smoking rate, age of onset of initiation, years of being a regular smoker, etc. The SHQ has successfully been used in previous studies as a measure of smoking history (Brown, Lejuez, Kahler, & Strong, 2002; Zvolensky, Lejuez, et al., 2004). The SHQ was administered at Time 1 only and the average cigarettes per day variable was used as a primary predictor variable.

*Fagerstrom Test for Nicotine Dependence (FTND).* The FTND is a six-item scale designed to assess gradations in tobacco dependence (Heatherton et al., 1991). The FTND is a revision of the *Fagerstrom Tolerance Questionnaire* (FTQ; Fagerstrom, 1978). The FTND has shown good internal consistency, positive relations with key smoking variables (e.g., saliva cotinine; Heatherton et al, 1991; Payne, Smith, McCracken,

McSherry, & Antony, 1994), and high degrees of test-retest reliability (Pomerleau, Carton, Lutzke, Flessland, & Pomerleau, 1994).

*Expired Carbon Monoxide.* Biochemical verification of smoking status was completed by carbon monoxide (CO) analysis of breath samples assessed using a Bedfont Micro III Smokerlyzer CO Monitor (Model EC50; Bedfont Scientific USA, Medford, NJ). Research indicates that 8 ppm is an optimal cutoff score for reliably discriminating smoking status (Jarvis, Tunstall-Pedoe, Feyerabend, Vesey, & Saloojee, 1987). Obtained values above this cutoff were considered indicative of smoking at Time 1.

*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). Most items are rated on a 5-point Likert scale from (0) *never* to (4) *daily or almost daily*. Scores range from 0-40 with a score of 8 indicating a likelihood of alcohol use problems. Major areas of problematic drinking that are assessed include: alcohol consumption, drinking behavior (dependence), adverse psychological reactions, and alcohol-related problems. There is a large body of literature attesting to the validity of the AUDIT (Saunders, Aasland, Babor, de la Fuente, & Grant, 1993). In the present investigation, we used the frequency and quantity items of the AUDIT to index alcohol consumption at Time 1 and employed this index as a covariate.

### *Time 2 Measures*

The *SCID-NP Panic Attack Module* was administered at Time 2 in order to assess panic attacks during the past three months (i.e., criterion variable). The *Panic Disorder*

*Severity Scale* (PDSS) was re-administered at Time 2 to document change in anticipatory anxiety ratings over the 3-month assessment. *Only* the SCID-NP panic module and PDSS were re-administered, rather than all panic-relevant measures, as these were the only two factors theorized to possibly show systematic change in the 3-month time period.

### *Procedure*

#### *Screening*

An overview of the entire procedure can be seen in Table 2. Initial screening procedures were completed via telephone. Interested individuals were given a brief description of the study and asked about smoking status. Potentially eligible participants then scheduled an assessment appointment at the Anxiety and Health Research Laboratory (AHRL).

#### *Assessment*

Upon arrival to the assessment appointment, participants were administered the SCID-NP to more thoroughly assess whether or not subjects met inclusion criteria and document their psychiatric history as well as the Panic Disorder Severity Scale. After completing the interview, smoking status was biochemically verified via CO analysis. Subjects then completed the following self-report measures: Anxiety Sensitivity Index, Agoraphobic Cognitions Questionnaire, Body Vigilance Scale, Positive and Negative Affect Scale, Smoking History Questionnaire, and the Alcohol Use Disorders Identification Test (about 90-minutes in total time for session 1). Subjects were then given instructions regarding procedures for the follow-up period and given \$20 compensation.

### *Follow-Up*

During the 3-month prospective phase, reminder calls were placed at 1.5 months to keep in touch with participants; specifically, at 1.5 months, we contacted participants via telephone to ensure that their contact information had not changed and reminded them about the upcoming follow-up assessment. If their phone number had changed or was disconnected, we then attempted to contact them via letter. After three months, subjects were contacted to schedule their follow-up assessment. Subjects returned to the laboratory and were administered the following measures: SCID-NP panic attack module and the Panic Disorder Severity Scale. Subjects were then debriefed and given \$30 compensation for their efforts.

## Results

### *Statistical Analyses*

The main and interactive effects of smoking rate and AS Physical Concerns for the primary dependent variables were evaluated using a hierarchical multiple regression (or logistic regression for binary dependent variables) procedure (Cohen & Cohen, 1983). For the first set of analyses, separate models were constructed for predicting (1) the tendency to catastrophize about bodily sensations (ACQ), (2) body vigilance (BVS), (3) agoraphobic avoidance (composite avoidance score from the PDSS), and (4) a lifetime history of panic attacks (SCID; this analysis used a logistic regression). Negative affectivity and weekly alcohol consumption were entered as covariates at step one in the model. At the second step the main effects for smoking rate and AS Physical Concerns were simultaneously entered into the model in order to estimate the amount of variance

accounted for by these variables individually. At the third step, the interaction term (mean centered) between smoking rate and AS Physical Concerns was entered into the model (Baron & Kenny, 1986).

Hierarchical multiple regression also was employed for the prospective test to determine if the interaction between AS Physical Concerns and cigarettes per day at Time 1 accounted for unique variance in predicting the presence of panic attacks during the follow-up period (SCID; logistic regression was employed for this analysis due to the binary nature of the dependent variable) and change in anticipatory anxiety at Time 2. Separate models were, again, constructed for each of the dependent variables: panic attacks and anticipatory anxiety at Time 2. History of panic attacks was entered as a covariate at step 1 for the panic attack analysis, and level of anticipatory anxiety at Time 1 was entered as a covariate for the anticipatory anxiety analysis; this approach allowed for an *initial test* of “change” for each factor, which is consistent with the *a priori* hypotheses. AS Physical Concerns and cigarettes smoked per day at Time 1 were entered together at step 2 to evaluate the main effects of these variables. Lastly, the mean centered interaction term (AS Physical Concerns x cigarettes per day at time 1) was entered at step 3 to test whether AS Physical Concerns at Time 1 moderated the relation between smoking history at Time 1 and panic attacks and anticipatory anxiety at Time 2.

*Correlations for Theoretically-Relevant Variables*

The first step to understand the nature of the data was to compute a series of conceptually-relevant zero-order correlations for each of the different assessment time points (Time 1 and 2). Here, zero-order correlations were first computed to assess the



association between the primary predictor variables (smoking rate and AS Physical Concerns) and the covariates at Time 1. Then, correlations were computed to assess the association between the primary predictor variables (smoking rate and AS Physical Concerns) and the primary dependent variables at Time 1. Finally, zero-order correlations were then computed for primary predictor variables (smoking rate and AS Physical Concerns) and the Time 2 dependent measures.

*Associations among predictor variables and covariates at Time 1.* Negative affectivity was significantly correlated with AS Physical Concerns ( $r = .67, p < .01$ ), but not alcohol consumption ( $r = .03, p = .70$ ) or smoking rate ( $r = .14, p = .11$ ). Alcohol consumption was significantly correlated with smoking rate ( $r = -.30, p < .01$ ), but not with AS Physical Concerns ( $r = .02, p = .84$ ). The correlation between smoking rate and AS Physical Concerns was minimal ( $r = -.04, p = .70$ ).

*Associations among predictor and criterion variables at Time 1.* Smoking rate was significantly associated with lifetime history of panic attacks ( $r = .19, p < .05$ ), but not the tendency to catastrophize about bodily sensations ( $r = .01, p = .95$ ), body vigilance ( $r = -.08, p = .35$ ), or agoraphobic avoidance ( $r = .10, p = .28$ ). AS Physical Concerns was significantly correlated with the tendency to catastrophize about bodily sensations ( $r = .67, p < .01$ ), body vigilance ( $r = .62, p < .01$ ), agoraphobic avoidance ( $r = .38, p < .01$ ), and a lifetime history of panic attacks ( $r = .23, p < .01$ ). The associations between the criterion variables were all significant except for the correlation between body vigilance and lifetime history of panic attacks ( $r = .15, p = .09$ ).

*Retention of participants during the follow-up period.* 83.2% of the participants ( $n = 104$ ) returned for the Time 2 assessment and were thus included in the Time 2 analyses. 12% ( $n = 15$ ) of the participants returning for the second assessment had experienced a panic attack during the follow-up period; this is a sizeable percentage, underscoring the clinically significant nature of this population, an issue that is returned to in the Discussion Section.

*Associations among the covariates, predictor variables and criterion variables at Time 2.* Lifetime history of panic attacks was significantly correlated with smoking rate ( $r = .19, p < .05$ ) and AS Physical Concerns ( $r = .23, p < .01$ ). Time 1 anticipatory anxiety was significantly associated with AS Physical Concerns ( $r = .29, p < .01$ ), but not smoking rate ( $r = .09, p = .34$ ). Smoking rate was not significantly associated with the presence of panic attacks during the follow-up period ( $r = .06, p = .56$ ) or Time 2 anticipatory anxiety ( $r = .06, p = .56$ ). Similarly, AS Physical Concerns was not significantly associated with the presence of panic attacks during the follow-up period ( $r = .10, p = .33$ ) or Time 2 anticipatory anxiety ( $r = .14, p = .17$ ).

#### *Time 1 Regression Equations*

Data for the Time 1 linear regression analyses are presented in Table 4. In terms of the tendency to catastrophize about bodily sensations, the first step accounted for 50.9% of the variance. Negative affectivity was a significant predictor at step 1 of the model ( $t = 10.91, \beta = .71, p < .01$ ), but alcohol consumption was not ( $t = -.06, \beta = -.004, p = .95$ ). A significant main effect for AS Physical Concerns was found at step 2 of the model ( $t = 3.49, \beta = .30, p < .01$ ), but not smoking rate ( $t = -.61, \beta = -.04, p = .54$ ).

Contrary to prediction, the interaction between AS Physical Concerns and smoking rate did not significantly predict the tendency to catastrophize about bodily sensations ( $t = .70, \beta = .04, p = .48$ ).<sup>3</sup>

In terms of body vigilance, the first step accounted for 22.4% of the variance. Negative affectivity was a significant predictor at step 1 of the model ( $t = 5.75, \beta = .47, p < .01$ ), but alcohol consumption was not a significant predictor ( $t = -.42, \beta = -.03, p = .68$ ). A significant main effect for AS Physical Concerns was found at step 2 of the model ( $t = 4.98, \beta = .51, p < .01$ ), but no main effect was observed for smoking rate ( $t = -1.36, \beta = -.11, p = .17$ ). Contrary to prediction, the interaction between AS Physical Concerns and smoking rate did not significantly predict body vigilance ( $t = .47, \beta = .04, p = .64$ ).

In terms of agoraphobic avoidance, the first step accounted for 18.3% of the variance. Negative affectivity was a significant predictor at step 1 of the model ( $t = 5.01, \beta = .42, p < .01$ ), but alcohol consumption was not a significant predictor ( $t = -1.04, \beta = -.09, p = .30$ ). There were no main effects for either AS Physical Concerns or smoking rate at step 2 of the model ( $t = .96, \beta = .11, p = .34$  and  $t = .90, \beta = .08, p = .37$ , respectively). As hypothesized, the interaction between AS Physical Concerns and smoking rate did significantly predict agoraphobic avoidance; the interaction accounted for 3.6% of unique variance ( $t = 2.29, \beta = .19, p < .05$ ).

Data for the Time 1 logistic regression is presented in Table 5. In terms of lifetime history of panic attacks, negative affectivity (OR = 1.15,  $p < .01$ , 95% CI = 1.08-1.22), but not alcohol consumption (OR = 1.03,  $p = .53$ , 95% CI = .94-1.13), was associated with a unique change in the odds of having a lifetime history of panic attacks. Neither AS

Physical Concerns (OR = .96,  $p = .33$ , 95% CI = .87-1.05) nor smoking rate (OR = 1.05,  $p = .12$ , 95% CI = .99-1.11) were associated with a unique change in the odds of having a lifetime history of panic attacks above and beyond the covariates. Finally, contrary to prediction, the interaction between AS Physical Concerns and smoking rate was not related to lifetime history of panic attacks (OR = 1.00,  $p = .71$ , 95% CI = .99-1.00).

### *Time 2 Regression Equations*

Data for the Time 2 logistic regression analysis is presented in Table 6. In terms of the presence of panic attacks during the three-month follow-up period, lifetime history of panic attacks (OR = 8.42,  $p < .01$ , 95% CI = 2.56-27.68) was associated with a unique change in the odds of experiencing a panic attack during the follow-up period. Neither AS Physical Concerns (OR = .99,  $p = .78$ , 95% CI = .92-1.07) nor smoking rate (OR = 1.00,  $p = 1.00$ , 95% CI = .93-1.07) were associated with a unique change in the odds of having panic attacks during the follow-up period above and beyond the covariate. Finally, contrary to prediction, the interaction between AS Physical Concerns and smoking rate was not related to increased likelihood of experiencing panic attacks during the follow-up period (OR = 1.00,  $p = .86$ , 95% CI = .99-1.01).

Data for the Time 2 linear regression analysis is presented in Table 7. In terms of anticipatory anxiety at Time 2, Time 1 anticipatory anxiety was not a significant predictor at step 1 ( $t = .28$ ,  $\beta = .03$ ,  $p = .78$ ). There were no main effects for either AS Physical Concerns or smoking rate at step 2 of the model ( $t = 1.38$ ,  $\beta = .14$ ,  $p = .17$  and  $t = .73$ ,  $\beta = .07$ ,  $p = .47$ , respectively). As hypothesized, the interaction between AS Physical

Concerns and smoking rate significantly predicted anticipatory anxiety at Time 2 ( $t = 2.32, \beta = .23, p < .05$ ).

#### *Graphical Representation of the Statistically Significant Interactions*

Interactions were examined, in regard to hypothesized moderation, to determine direction and significance. Statistically significant interactions were examined, in regard to hypothesized moderation, both graphically (see Cohen & Cohen, 1983, for a review) and analytically (Holmbeck, 2002), to determine direction and conceptual consistency with the model. Based on recommendations of Cohen and Cohen (1983; pp. 323, 419), the form of significant interactions were examined by inserting specific values for each predictor variable into the regression equations associated with the described analysis. Specifically, values for each predictor variable at one half of a standard deviation above and below the mean for AS Physical Concerns and smoking rate, respectively, were inserted into the regression equation and plotted.

Forms of the interactions supported the hypotheses (please see Figures 2 and 3). Among individuals with higher AS Physical Concerns, smoking a greater number of cigarettes was associated with higher levels of agoraphobic avoidance at Time 1, whereas smoking rate had a relatively weaker association with agoraphobic avoidance across the other variable combinations. A similar finding was evident for anticipatory anxiety at Time 2. Again, higher levels of AS Physical Concerns and higher rates of daily smoking were associated with the greatest elevations in anticipatory anxiety compared to other variable combinations. These findings are in accord with the hypothesized effects. Furthermore, based on recommendations of Holmbeck (2002), post-hoc probing analyses

were conducted on the data to examine moderation. Results indicated that moderation occurred for agoraphobic avoidance but not T2 anticipatory anxiety [ $t(116) = 4.03, p < .01$  and  $t(95) = 1.33, p = .19$ , respectively]. The association between smoking rate and agoraphobic avoidance was moderated when AS Physical Concerns was high.

## Discussion

There has been a growing level of clinical interest in better understanding the role of smoking and other addictive behaviors in psychiatric conditions in recent years (Zvolensky & Schmidt, 2004). This work has grown, at least partially, out of the recognition that smoking often co-occurs with psychological problems such as anxiety disorders and may be systematically related to the course of such psychopathology. Despite the direct clinical and public health importance of addressing such matters, we are only at the beginning stages of explicating the nature of smoking-anxiety associations. The present study represents an effort to empirically evaluate a cognitive vulnerability by smoking model related to panic processes using a cross-sectional and prospective measurement protocol.

### *Findings from Time 1 Assessment*

In regard to the cross-sectional analyses, as hypothesized, there was a significant interaction between AS Physical Concerns and smoking rate in relation to agoraphobic avoidance. These effects were above and beyond the variance accounted for by the theoretically-relevant covariates and respective main effects. Inspection of the form of the significant interaction was supportive of the theorized AS Physical Concerns by smoking model of panic-vulnerability (see Figure 2). Specifically, at higher levels of AS Physical

Concerns and higher smoking rates, there was a risk for increased agoraphobic avoidance. It is noteworthy that the size of the interaction effect was meaningful at 3.6% of unique variance (after controlling for the variance accounted for by the covariates and main effects; Abelson, 1985). Moreover, such results replicate and extend past work on AS and smoking in a Russian epidemiological sample (Zvolensky, Kotov et al., 2003). Contrary to prediction, however, the interaction between AS Physical Concerns and smoking rate did not significantly predict the tendency to catastrophize about bodily sensations, body vigilance, or lifetime history of panic attacks. Thus, three out of the four predictions were not supported from an *a priori* basis.

Overall, these Time 1 findings suggest that the interaction between the cognitive vulnerability variable of AS and smoking rate is relatively specific to panic-relevant avoidance behavior rather than applicable to all aspects of panic vulnerability (e.g., body vigilance, catastrophic thinking, and panic attacks). Though cross-sectional in nature, this pattern of findings, considered with those of Zvolensky, Kotov et al. (2003), may be used to conceptually guide the refinement of etiological models of panic vulnerability that involve smoking behavior. For example, rather than smoking rate and AS “combining” to confer risk for all aspects of panic vulnerability, perhaps these two risk factors may interplay only for certain risk processes like avoidance behavior. Then, once this type of maladaptive behavior pattern “emerges,” other panic-relevant processes like anticipatory anxiety, vigilance to somatic stimuli, panic attacks, catastrophic thinking, and so on, may theoretically “follow.” These types of issues are broadly concerned with “timing” of etiological processes.

Theory and research on avoidance learning would be broadly consistent with the above described type of affect regulation model (cf. self-medication model), whereby escape and avoidance responding at Time 1 increased the risk for anxiety symptoms at a future time period (Mineka, 1985). The basic premise in such models is that the organism learns a contingency between an aversive environmental event and a response pattern that serves to avoid contact with it (Zvolensky, Lejuez, & Eifert, 2000). Here, it is interesting that these learning processes often can operate beyond conscious awareness. Thus, there is an expected *disconnect* between declarative knowledge about various relevant stimuli and the degree of emotional learning that has transpired, particularly during the early phases of a conditioning process; a finding supported by a basic research (Bechara et al., 1995). This type of work may partially explain why there was an observed disconnect between self-rated avoidance (presumably, more automatized behavior tied to emotional learning) and cognitive factors in the present investigation; that is, between the self-regulation factor of avoidance and the studied cognitive (presumably, declarative knowledge) variables. Of course, given self-rating scales were used to assess both avoidance and cognitive factors, these argument is more a theoretical prediction derived from extant models to help explain the observed results rather than a complete explanation for the observed results.

Building from such reasoning, as applied *conceptually* to the present research, it may be that a daily smoker learns that certain situations may be evoking uncomfortable bodily sensations or anxiety symptoms (theoretically produced, at least partially, by smoking behavior over an extended period of time), and then starts to avoid such



situations (“agoraphobic avoidance”). Here, it should be noted that the avoidance assessment in the present study was geared toward “classic” general agoraphobic situations (e.g., driving). According to the present perspective, however, avoidance could be applied to more specific situations/behaviors as well (e.g., smoking to escape/avoid escalating negative affect states). That is, there should be great variability in the compensatory behaviors to reduce anxiety. As applied to smokers, tests involving smoking motives may be a useful way to empirically evaluate such matters with more specificity (e.g., AS by smoking rate predicting negative affect reduction smoking motives). Additionally, it is important to bear in mind that the cross-sectional methodology of the current study cannot differentiate such risk processes from concomitants or consequences and thereby does not permit an empirical analysis of these precise issues. However, the current results do set the exciting stage for future research to follow from this study and empirically evaluate such a model; a perspective that we return to in the discussion of the Time 2 analyses (see below).

It also is useful to briefly comment on the pattern of zero-order associations among predictor and criterion variables at Time 1. Consistent with extant research (Breslau & Klein, 1999), smoking rate and history of panic attacks were significantly associated with one another. However, smoking rate was not significantly associated with any of the panic-relevant process variables. Also, consistent with prior research, AS Physical Concerns was significantly positively associated with all of the panic-related variables. Furthermore, the association between AS Physical Concerns and smoking rate was negligible, suggesting that these two variables are unique, or at least, largely

independent risk factors. Of note, alcohol consumption was significantly negatively correlated with smoking rate; these two variables are typically positively associated with one another (e.g., Strine et al., 2005). This finding is likely due to unique characteristics of this sample. Specifically, although this was a primarily young, college-student sample, approximately 25% of the sample was from the community (older, community dwelling individuals). The demographic characteristics of this subset of the sample were somewhat different than those of the primarily college student sample. In particular, the community participants reported drinking significantly less, smoking more, were older, and more likely to have had drug or alcohol treatment (approximately 50% had a history of substance abuse treatment). Thus, there may be unique patterns between community and college-based samples in regard to smoking and alcohol use patterns.

#### *Findings from Time 2 Assessment*

In regard to the prospective analyses, there was a significant interaction between AS Physical Concerns and smoking rate in relation to Time 2 anticipatory anxiety. These effects were above and beyond the variance accounted for by the theoretically-relevant covariate and respective main effects. Post hoc probing analysis of the form of the significant interaction, however, was not supportive of the theorized AS Physical Concerns by smoking model of panic-vulnerability. This finding may have been due to insufficient power due to the smaller sample size at Time 2. Nonetheless, the size of the interaction effect was clinically meaningful at 5% of unique variance (Abelson, 1985). Drawing from research in other areas of psychology, prospective studies that follow participants for shorter periods of time tend to generate smaller effects than those with

longer follow-up periods (e.g., Rogosa, Brandt, & Zimowski, 1982). Thus, future studies using longer follow-up assessments are necessary for sufficient numbers of participants to show change in the outcome of interest or that multiple waves of data allow one to model change more reliably. Also, contrary to prediction, the interaction between AS Physical Concerns and smoking rate did not significantly predict the occurrence of panic attacks during the three month follow-up period.

Considered with the Time 1 data, the Time 2 results shed further empirical light on the nature of the AS Physical Concerns by smoking rate interaction in terms of panic vulnerability. Specifically, not only does the interaction between AS Physical Concerns and smoking rate concurrently predict agoraphobic avoidance (Time 1 analyses/findings), but it also predicts future anticipatory anxiety in a relatively short window of time (Time 2 analyses/findings). These findings may portend the type of panic-relevant vulnerability processes developing among this clinically-relevant daily smoking population. Specifically, daily smokers smoking at higher rates with high AS Physical Concerns may be more prone to engage in avoidance (Time 1 findings) and show increases in worry about potentially threatening events in the future (Time 2 anticipatory anxiety findings). One could anticipate that, given any number of adverse events (e.g., exposure to high stress situations, unexpected panic attacks), that these individuals (i.e., heavier smokers with high AS Physical Concerns) may be more prone than their lower smoking and lower AS Physical Concerns counterparts to be more vulnerable to adverse emotional learning between interoceptive cues and anxiety states. With more frequent learning experiences, or even one salient (emotionally powerful) event, these factors may theoretically “work

together” to potentiate risk for the future development of panic psychopathology. Clearly, this type of account, although grounded in empirical observation and conceptual models of panic-smoking comorbidity (Zvolensky & Bernstein, 2005), is currently highly speculative. It therefore represents an exciting area for future research. The utilization of time-sampling or ecological momentary memory assessment methodologies would be one way to systematically move this line of inquiry further.

It also is useful to briefly comment on the pattern of zero-order associations among predictor and criterion variables at Time 2. Smoking rate was not significantly associated with panic related processes (anticipatory anxiety) or the occurrence of panic attacks during the follow up period. In terms of panic attacks, this finding is consistent with past research where the association between smoking and future panic attacks was only significant among adolescents who smoked greater than 20 cigarettes per day (Johnson et al., 2000). Thus, examining these same questions among heavy smokers may be useful in isolating the parameters of the smoking rate-panic attack association. The lack of association between anticipatory anxiety and smoking rate is consistent with Time 1 findings and those of Zvolensky, Kotov, et al. (2003) where there was no association between smoking rate and anxious arousal symptoms (i.e., the pure, non-overlapping symptoms of anxiety compared to depression). Furthermore, AS Physical Concerns also was not associated with the Time 2 panic variables, likely due to the limited variability in the Time 2 criterion variables. Indeed, case-level data inspection indicated that while 12% of the Time 2 sample experienced panic attacks during the follow-up period, only 5 individuals experienced any anticipatory anxiety and none to a “severe degree.”

### *Clinical Implications*

Results of the current study suggest the potential utility of addressing smoking within the context of intervening among individuals at risk for developing panic attacks and panic disorder. Specifically, smokers with panic risk factors like AS report using smoking as a primary emotion regulation strategy (Zvolensky, Bonn-Miller et al., 2006), creating an interconnection between smoking and panic. This interplay creates a situation wherein intervention for one problem will ultimately be impaired, or perhaps altogether unsuccessful, unless the other problem also is addressed simultaneously. Case reports are consistent with this perspective (Zvolensky, Lejuez, Kahler, & Brown, 2003). As one illustrative example, when smokers high in cognitive-based risk for panic (e.g., high AS) make a smoking cessation attempt, they are (1) at high risk for being emotionally reactive to internal cues including, but not limited to, nicotine withdrawal (Zvolensky, Feldner, et al., 2005) and (2) “biased” to reduce such distress (avoidant coping), particularly when alternative coping strategies are not available. Thus, AS and perhaps other panic-specific factors would presumably “prime” motivational processing very early in the quit process. In particular, an individual that is highly sensitive to negative affect and other internal cues (e.g., high AS) would be apt to smoke, and thereby demonstrate early relapse, to ameliorate aversive states elicited by smoking discontinuation. This type of example theoretically illustrates the clinically-relevant linkages between panic factors and smoking behavior in terms of self-regulation processes and cessation. Similar examples could be made in regard to other directional effects; that is, from smoking to panic vulnerability, an issue fully addressed in the Introduction Section of the present study and

thus not repeated here. In total, if accurate, this forward feeding type of cycle creates the need for an integrated intervention that reduces panic problems while considering the role of smoking in the larger “psychological context” of therapeutic care. One could imagine it would be applicable to not only people at risk for developing panic psychopathology, but also smokers with full-blown panic problems.

#### *Study Limitations and Future Directions*

Although the present study adds to the extant literature on smoking and panic-relevant variables in a unique manner, there are a number of interpretive caveats that deserve further comment. First, although we used community-based advertisements in the recruitment of participants for the present investigation, it is noteworthy that the sample was comprised of relatively young adult daily smokers. The sample may have been younger, on average, than would be expected from typical community-based recruitment due to the fact that advertisements for the study were largely posted in areas of the community frequently visited by young adults (e.g., shopping centers, restaurants, bars) and therefore may have attracted younger adults to a greater extent than older adult smokers. Second, the sample was comprised of regular (daily), but not heavy, smokers. As previous research indicates that the panic-smoking association often is most apparent among heavy smokers, it may prove fruitful for future research to examine panic-vulnerability associations in light, moderate, and heavy smokers. Along these same lines, it may be useful to compare panic-vulnerability associations with smoking rate vs. nicotine dependence. A comparison of smoking rate and dependence in the prediction of panic problems would serve to increase specificity of the current biopsychosocial model

of panic disorder etiology by specifying which factor(s) play more formative roles in promoting risk. Third, although the sample was representative of the ethnic composition of the state of Vermont, it was comprised of predominately Caucasian young adults. To improve generalizability of the observed effects, future research could sample from locations with more diverse demographic characteristics. Fourth, a three-month follow up period is a fairly short period of time. Although we opted to design the present investigation for 3-month follow-up to establish precedent for examining these matters and assess retention rates among this at-risk population, large changes in symptomatology during this specific window of time were not evident. Future studies employing longer follow up periods would better document symptom progression.

Fifth, and somewhat related to the prospective design comment, is that we utilized hierarchical linear regression tools for indexing the observed effects. Although a reasonable analytic option, these techniques are not as powerful as random regression growth curve models and hazard models for modeling change. We used linear regression in the present study due to the size of the recruited sample and rather limited focus on one interactive process. Building from the present study, it would be advisable to construct more powerful tests and use even more sensitive analytic tools for indexing change.

Sixth, in terms of effect size for the observed significant interactions, meaningful effect sizes are being found (e.g., 3% to 5% of variance). However, the clinical significance of effects of this size depends on the context in which they are examined. Here, while such effect sizes may be a “good start”, they simultaneously underscore that the vast majority of variance is not explained. Clearly, continuing to refine and evaluate theoretically-

driven models of risk are critical. Seventh, self-report measures were utilized as the primary assessment methodology for many of the key constructs. The utilization of self-report methods does not fully protect against reporting errors and may be influenced by shared method variance. Thus, future studies could build on the present work by utilizing laboratory based assessments to provide information about smoking behavior in “real time.” For example, evaluating the predictive power of AS by smoking rate in the prediction of emotional responsivity to biological challenge would help document response patterns across systems. Again, such data could then be used to refine theoretical models. Finally, in the broadest “causal model” of panic vulnerability, moderators influence the relation between a given risk factor and panic outcomes. The present study, grounded in such a heuristic model to guide theoretical predictions, addressed one such moderator (AS). However, future work needs to build on the present study and address other moderating factors with specified theoretical relevance to the overarching panic vulnerability model. Additionally, research should begin to address mediators, which theoretically explain the relation between a given risk factor and panic psychopathology. This type of work will, as a “system”, begin to lay the groundwork for even more advanced tests that attempt to explicate the direction of such processes and incorporate multi-variable modeling of effects.

### *Conclusions*

Together, the present findings suggest daily smokers who have higher smoking rates and higher levels of AS Physical Concerns report greater agoraphobic avoidance and increases in anticipatory anxiety over a short period of time. The primary implication



of the present findings is that there may be segments of the cigarette smoking population who are at relatively greater risk for panic symptoms by virtue of individual differences in AS. The identification of such moderating effects is clinically important, as it helps to refine our understanding of complex associations between drug behavior and panic vulnerability.

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## Footnotes

<sup>1</sup>Regular cigarette smoking does not necessarily indicate that the individual is nicotine dependent. Nicotine dependence implies that the individual meets DSM-IV criteria for substance dependence which include symptoms of tolerance, withdrawal, inability to cut down or stop use, stopping social, occupational, or recreational activities because of substance use, and continued use despite knowledge of having a persistent physical or psychological problem because of substance use (APA, 2000). Most studies on smoking and PD do not indicate whether or not subjects are nicotine dependent per se. In this same context, it is important to note that the term “addiction” will be used instead of drug dependence. This is done for several key reasons. Specifically, “dependence” does not offer any explanatory meaning over and above “addiction.” Both reflect patterns of drug use that impart some cost on the individual; are difficult to stop; typically recur after discontinuation; and are characterized, in part, by tolerance and withdrawal. Also, by employing the term dependence, confusion at a conceptual level can be created because of the more specific term of physical dependence. Physical dependence reflects a state in which reduced drug levels elicit withdrawal symptoms.

<sup>2</sup>Gender was not included as a covariate in the current study as the sample size did not allow for it. Furthermore, there have been no gender effects found in past work or theoretical models; thus, there was little reason to examine this factor here. However, future research that involves larger sample sizes should address this factor and incorporate it into contemporary models.

<sup>3</sup>Due to the overlap between the tendency to misinterpret bodily sensations [measured by the Brief Body Sensations Interpretation Questionnaire (BBSIQ)] and the tendency to catastrophize about bodily sensations (ACQ), only the results from the ACQ are presented here and not for the BBSIQ as originally proposed. The validity of the BBSIQ in the current study was questionable as participants consistently had difficulty understanding the instructions for completing the BBSIQ despite explanations from the research team. Regression analyses were run using the BBSIQ as a criterion variable and the interaction between AS Physical Concerns and smoking rate did not significantly predict the tendency to misinterpret bodily sensations ( $t = -.89, \beta = -.07, p = .37$ ).

Table 1: *Key Terms in Risk Factor Research (from Kraemer et al., 1997)*

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<i>Correlate:</i>	Two characteristics shown to be associated without any implication of a temporal or directional relation.
<i>Risk Factor:</i>	A characteristic that has been shown to precede the outcome and to be associated with an increase in the likelihood of that outcome over base rates in the general population. This requires unequivocally demonstrating the temporal sequence that entails evidence that the outcome was not evident prior to or at the time of the antecedent event.
<i>Marker:</i>	A risk factor that is not causally involved in the outcome. Fixed marker is used to refer to risk factors that are not considered malleable; variable marker is used for those risk factors that change or can be changed.
<i>Causal Risk Factor:</i>	A risk factor that, when altered, has impact on the likelihood of the outcome. A causal role of the risk factor in the outcome is bolstered by collateral evidence regarding the mechanisms involved in the risk-outcome relation. Demonstration that an event is a causal risk factor is not tantamount to saying that antecedent event is <i>the</i> causal risk factor. Inherent in the risk factor approach is the possibility that there may be multiple causal risk factors and multiple paths toward an outcome of interest.

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Table 2: *Overview of Procedure*

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*Screening*

- Telephone Screening
- Appointment for assessment set

*Assessment*

- Informed consent
- Structured Interviews and medical screening
  - SCID-NP
  - Panic Disorder Severity Scale (PDSS)
- CO analysis
- Self-report questionnaires
  - Anxiety Sensitivity Index (ASI)
  - Agoraphobic Cognitions Questionnaire (ACQ)
  - Body Vigilance Scale (BVS)
  - Positive and Negative Affect Scale (PANAS)
  - Fagerstrom Test for Nicotine Dependence (FTND)
  - Smoking History Questionnaire (SHQ)
  - Alcohol Use Disorders Identification Test (AUDIT)
- Compensation

*Follow-Up*

- Interview
    - SCID-NP (panic attack module)
    - Panic Disorder Severity Scale (PDSS)
  - De-briefing
  - Compensation
-



Table 3: Descriptive Data and Intercorrelations among Predictor and Criterion Variables

	1	2	3	4	5	6	7	8	9	10	11	<i>M</i>	<i>SD</i>
1. PANAS-NA	-	.03	.14	.67**	.68**	.46**	.36**	.39**	.25**	.27**	.17	21.3	8.46
2. Alcohol	-	-	-.30**	.02	.00	.00	-.07	.01	-.17	-.05	-.11	6.68	5.36
3. Cig/Day	-	-	-	-.04	.01	-.08	.10	.19*	.09	.06	.06	17.6	8.43
4. ASI-PC	-	-	-	-	.67**	.62**	.38**	.23**	.29**	.10	.14	11.6	8.28
5. ACQ	-	-	-	-	-	.45**	.46**	.35**	.36**	.25**	.07	1.53	0.49
6. BVS	-	-	-	-	-	-	.26**	.15	.20*	.04	.08	16.0	8.28
7. T1 Avoidance	-	-	-	-	-	-	-	.41**	.81**	.08	-.06	0.21	0.73
8. T1 Panic Attacks	-	-	-	-	-	-	-	-	.36**	.36	.22*	-	-
9. T1 AA	-	-	-	-	-	-	-	-	-	.03	.03	0.12	0.52
10. T2 Panic Attacks	-	-	-	-	-	-	-	-	-	-	.42**	-	-
11. T2 AA	-	-	-	-	-	-	-	-	-	-	-	0.06	0.27

*Note.* A single asterisk indicates correlation is significant at .05 level; A double asterisk indicates correlation is significant at .01 level; PANAS: Positive and Negative Affect Scale- Negative Affectivity subscale (Watson, Clark, & Tellegen, 1988); Alcohol: Weekly Alcohol Consumption; Cig/Day: Daily Cigarettes; ASI-PC: Anxiety Sensitivity Index- Physical Concerns subscale (Reiss et al., 1986); ACQ: Agoraphobic Cognitions Questionnaire (Chambless et al., 1984); BVS: Body Vigilance Scale (Schmidt, Lerew, & Trakowski, 1997); T1 Avoidance: Agoraphobic Avoidance at Time 1 assessed via PDSS (Shear et al., 1997); T1 Panic Attacks: History of panic attacks (yes/no) assessed via SCID-NP (First et al., 1995); T1 AA: Anticipatory Anxiety at Time 1 assessed via PDSS (Shear et al., 1997); T2 Panic Attacks: Panic attacks during 3-month follow up period (yes/no) assessed via SCID-NP (First et al., 1995); T2 AA: Anticipatory Anxiety at Time 2 assessed via PDSS (Shear et al., 1997).

Table 4: *Contribution of the Interaction between AS Physical Concerns and Smoking Frequency in Predicting Time 1 Panic-Relevant Variables*

	$\Delta R^2$	$t$ (each predictor)	$\beta$	$sr^2$	$p$
<b>Criterion Variable: Catastrophizing Bodily Sensations</b>					
Step 1	.51				<.01
Negative Affectivity		10.91	.71	.50	<.01
Alcohol consumption		-.06	.00	.00	ns
Step 2	.05				<.01
AS-PC		3.49	.30	.10	<.01
Smoking Rate		-.61	-.04	.00	ns
Step 3	.00				ns
AS-PC x Smoking Rate		.70	.04	.00	ns
<b>Criterion Variable: Body Vigilance</b>					
Step 1	.22				<.01
Negative Affectivity		5.75	.47	.22	<.01
Alcohol consumption		-.42	-.03	.00	ns
Step 2	.16				<.01
AS-PC		4.98	.51	.18	<.01
Smoking Rate		-1.36	-.11	.02	ns
Step 3	.00				ns
AS-PC x Smoking Rate		.47	.04	.00	ns
<b>Criterion Variable: Agoraphobic Avoidance</b>					
Step 1	.18				<.01
Negative Affectivity		5.01	.42	.18	<.01
Alcohol consumption		-1.04	-.09	.01	ns
Step 2	.01				ns
AS-PC		.96	.11	.01	ns
Smoking Rate		.90	.08	.01	ns
Step 3	.04				<.05
AS-PC x Smoking Rate		2.29	.19	.04	<.05

*Note.*  $n = 125$ ; AS-PC = AS Physical Concerns;  $\beta$  = standardized beta weight;  $sr^2$  = squared partial correlation

Table 5: *Contribution of the Interaction between AS Physical Concerns and Smoking Frequency in Predicting Lifetime History of Panic Attacks*

	Panic Attacks
<u>Step 1</u>	
Negative Affectivity	<b>1.14 (1.08-1.22)*</b>
Alcohol Consumption	1.03 (.94-1.13)
<u>Step 2</u>	
AS-PC	.96 (.87-1.05)
Smoking Rate	1.05 (.99-1.11)
<u>Step 3</u>	
AS-PC x Smoking Rate	1.00 (.99-1.00)

Note. Significant ORs are in bold type. \* $p < .01$

Table 6: *Contribution of the Interaction between AS Physical Concerns and Smoking Frequency in Predicting Time 2 Panic Attacks*

	Time 2 Panic Attacks
<u>Step 1</u> Time 1 Panic Attacks	<b>8.42 (2.56-27.68)*</b>
<u>Step 2</u> AS-PC	.99 (.92-1.07)
Smoking Rate	1.00 (.93-1.07)
<u>Step 3</u> AS-PC x Smoking Rate	1.00 (.99-1.01)

Note. Significant ORs are in bold type. \* $p < .01$

Table 7: Contribution of the Interaction between AS Physical Concerns and Smoking Frequency in Predicting Time 2 Anticipatory Anxiety

	$\Delta R^2$	$t$ (each predictor)	$\beta$	$sr^2$	$p$
<b>Criterion Variable: Time 2 Anticipatory Anxiety</b>					
Step 1	.00				ns
T1 Anticipatory Anxiety		.28	.03	.00	ns
Step 2	.02				ns
AS-PC		1.38	.14	.02	ns
Smoking Rate		.73	.07	.00	ns
Step 3	.05				<.05
AS-PC x Smoking Rate		2.32	.23	.05	<.05

Note.  $n = 104$ ; AS-PC = AS Physical Concerns;  $\beta$  = standardized beta weight;  $sr^2$  = squared partial correlation

*Figure 1: Conceptual Model depicting Anxiety Sensitivity Moderating the Effects of Smoking on Panic Vulnerability*

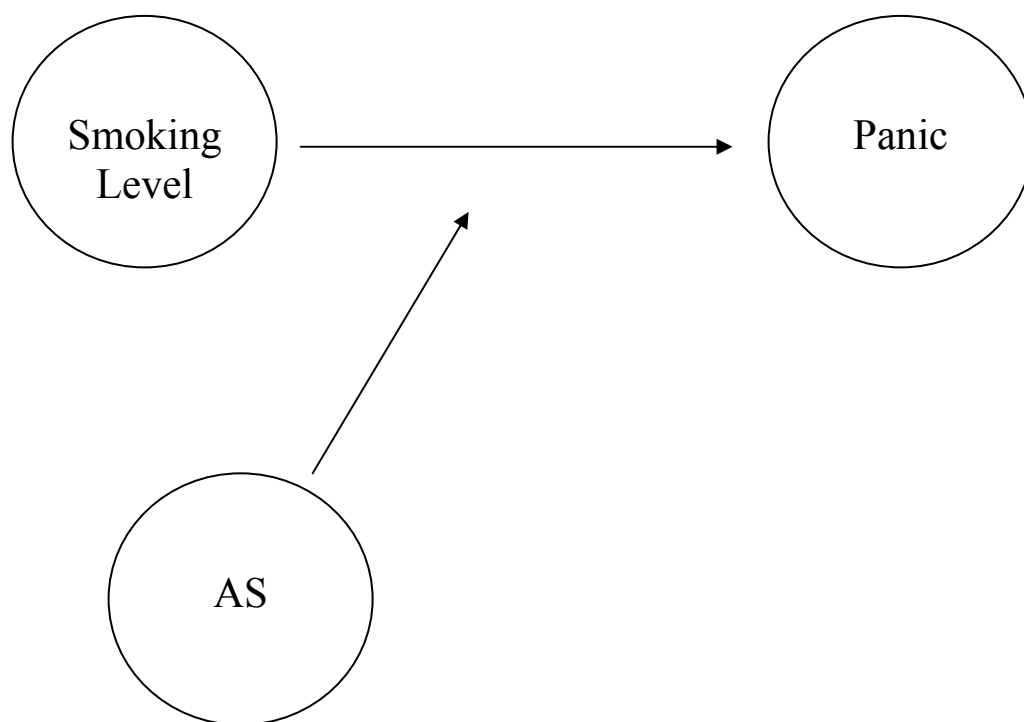


Figure 2: Time 1 Agoraphobic Avoidance, as indexed by the Panic Disorder Severity Scale (Shear et al., 1997), as a function of AS Physical Concerns and number of cigarettes smoked per day among participants one-half of a standard deviation above and/or below the mean for each predictor.

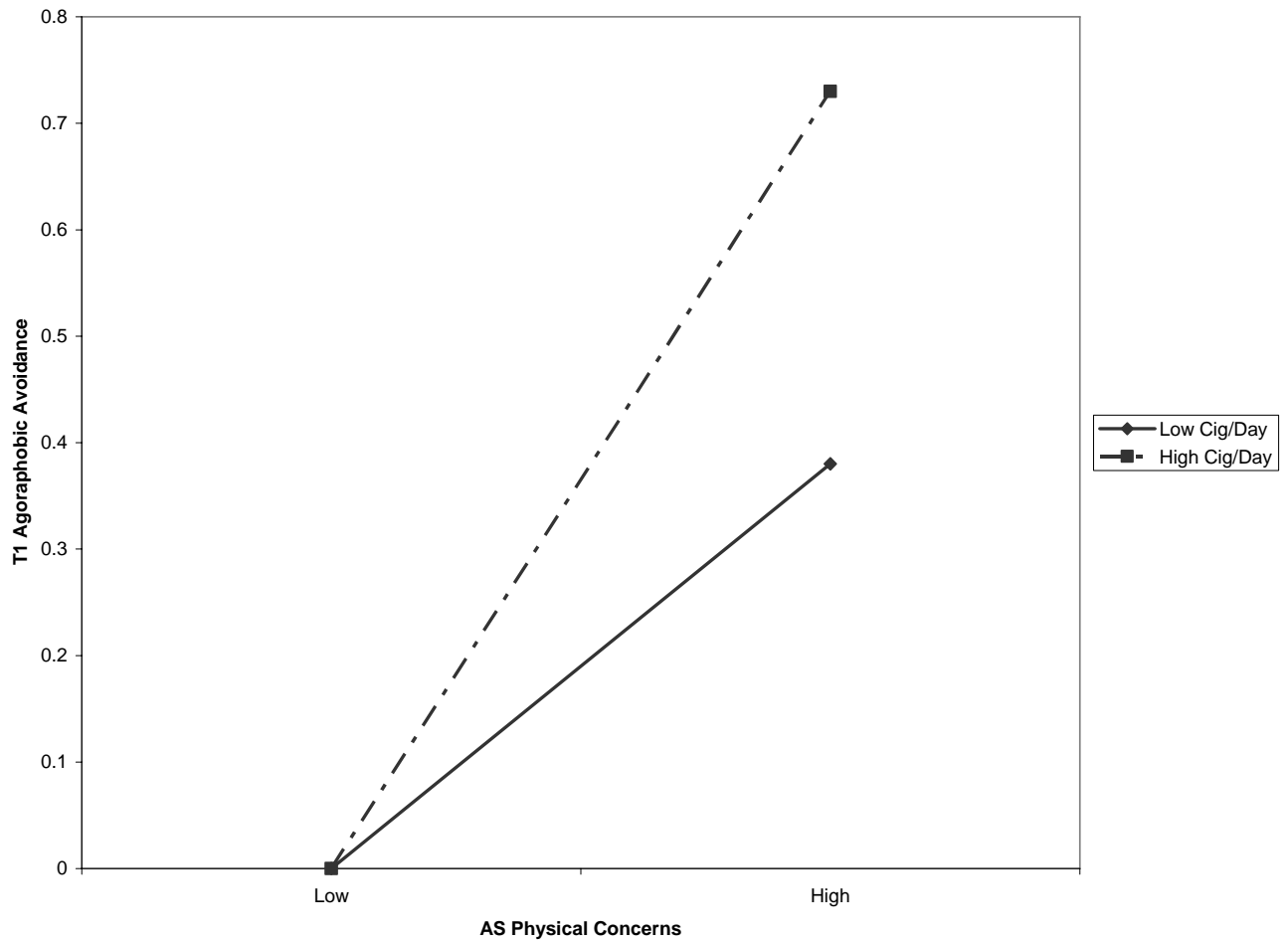


Figure 3: Time 2 Anticipatory Anxiety, as indexed by the Panic Disorder Severity Scale (Shear et al., 1997), as a function of AS Physical Concerns and number of cigarettes smoked per day among participants one-half of a standard deviation above and/or below the mean for each predictor.

