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Anxiety Sensitivity : Validity of the Anxiety Sensitivity Profile on a Clinical Sample

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ANXIETY SENSITIVITY: VALIDITY OF THE ANXIETY SENSITIVITY PROFILE ON A CLINICAL SAMPLE

By Sharon V. Elwell

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Dissertation Approval

This is to certify that the thesis presented to us by Sharon V. Elwell on the 13th day of May 2004, in partial fulfillment of the requirements for the degree of Doctor of Psychology, has been examined and is acceptable in both scholarship and literary quality.

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Abstract

The psychometric properties of the Anxiety Sensitivity Index (ASI) have been criticized. Although it has been the gold standard in research and assessment, its reliability (i.e., internal consistency) has been questioned. There are doubts about its ability to identify accurately the underlying factors for anxiety sensitivity. To provide a more accurate description of the anxiety sensitivity construct, Taylor and Cox (1998) developed the Anxiety Sensitivity Profile (ASP) and performed analyses based on a sample of 349 university students. The current study utilized a clinical sample to test the hypotheses that the ASP would have convergent validity with the ASI and discriminant validity with the trait version of the State-Trait Anxiety Inventory (STAI). This correlational study was conducted with a clinical sample of 105 adults, 19 to 65 years old, who have an anxiety disorder. Each subject completed the ASP, ASI, STAI, and the computerized version of the SCID-I/P. Results supported the hypotheses. Large correlations at the .01 level were found for the ASP and ASI total scores and ASP subscale scores. Modest correlations were found for the ASI and ASP total and subscale scores and the trait version of the STAI at the .05 level. Reliability (internal consistency) for the ASI total scores and ASP subscales was high. Therefore this study provides evidence for convergent validity with the ASI. It also provides necessary, although not sufficient evidence for construct validity for the ASP subscales. Internal consistency reliability cannot be determined for all of the underlying domains of the ASI, because one of the domains consists of only one item. This
evidence implies that the ASP is psychometrically superior to the ASI for research and treatment. Instead of the three underlying factors of the ASI, six factors are implied. Evidence is provided through convergent validity and internal consistency reliability found for the six ASP subscales. It is speculated that utilization of the ASP will support improved consistency in research through the use of congruence (i.e., matching the symptoms caused by a provocation task and the symptoms a person fears). Furthermore the ASP will support accurate identification of the domains underlying anxiety sensitivity that contribute to its association with all anxiety disorders. Finally, there are applied implications. These include the fact that those at risk can be identified and given brief cognitive-behavioral therapy as a preventive intervention. Also, treatment can target congruent cognitions, and elevated anxiety sensitivity at the end of treatment can be targeted for further interventions.
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Chapter One: Literature Review

Introduction

Attempts are being made to discover the factors involved in the etiology of anxiety pathology (Silverman & Weems, 1999). The goals of these attempts are the identification of individuals at risk and the application of appropriate preventive treatments to arrest the development of pathological anxiety. One risk factor is the construct referred to as anxiety sensitivity. It is currently viewed as an important, inherited predisposition for anxiety disorders in general and panic disorder in particular (Taylor & Cox, 1998). Initial studies have examined this construct in relation to prevention, treatment improvement, and maintenance of treatment gains with panic disorder (Otto & Reilly-Harrington, 1999).

The purpose of this paper will be to discuss some of the major themes regarding the anxiety sensitivity construct. Before entering into a description of the studies mentioned above, as well as the current study, the theoretical background for anxiety sensitivity is presented. This will include a discussion of the new conceptual developments that have been necessitated by challenges to the initial theory. Among these are the identification of a multifactorial structure and the number and identification of underlying domains. In addition, the part played by anxiety sensitivity in the etiology and maintenance of anxiety disorders will be discussed. This will include the role played by anxiety sensitivity, not only in panic disorder, but in other anxiety disorders as well.
From its initial conception, anxiety sensitivity has been defined as the fear of anxiety and anxiety-related symptoms (Reiss, 1987; Reiss & McNally, 1985; Reiss, Peterson, Gursky, & McNally, 1986). It has been hypothesized that those who fear the effects of anxiety on bodily sensations and cognitive processes believe there might be negative social (e.g., rejection due to visible signs such as shaking), physical (e.g., suffocation due to changes in respiration), or psychological results (e.g., losing their minds due to a decreased ability to concentrate; Zinbarg, Barlow, & Brown, 1997). It has been identified, in the negative effects of elevated anxiety sensitivity, as a factor related to panic disorder and to a lesser extent to the other anxiety disorders, with the possible exception of specific phobia (McNally & Lorenz, 1987; Reiss & McNally, 1985).

There have been challenges along the way in developing an understanding of the anxiety sensitivity construct. Meeting these challenges through continuing research is a necessary step for a well developed theoretical concept. The initial focus of this paper is on expectancy theory, which is the earliest attempt to provide a theoretical framework for the construct. According to this theory, anxiety sensitivity is perceived as one of three fundamental fears with a unifactorial structure that cannot be reduced to more specific fears. Anxiety sensitivity has also been viewed as a multifactorial concept that consists of at least four dimensions (i.e., anxiety sensitivity and at least three underlying factors; Lilienfeld, Turner, & Jacob, 1996; Taylor, 1996). Other aspects of expectancy theory have been questioned. For example, according to expectancy theory, anxiety sensitivity is a fear (Reiss, 1991). However, it has also been argued that it is not a fear, but possibly a variable that moderates fear, avoidance, and panic. Others have provided initial support
for the role of anxiety sensitivity as a partial mediator for panic (Stewart, Taylor, Jang, Cox, Watt, Fedoroff, & Borger, 2001).

Proceeding further with the multifactorial concept, investigators who subscribe to that view have not firmly established the number and identity of the underlying factors of anxiety sensitivity. Previously existing measures of this construct were not created to evaluate possible lower order factors (Cox, Borger, & Enns, 1999; Taylor & Cox, 1998; Taylor, Rabian, & Fedoroff, 1999). Although three factors (i.e., physical concerns, mental incapacitation concerns, and social concerns) have been identified with the Anxiety Sensitivity Index (ASI; Zinbarg et al., 1997), two studies have provided initial evidence for the existence of six factors (Taylor & Cox, 1998; Van der Does, Duijsens, Eurelings-Bontekoe, Verschuur, Spinhoven, 2003) based upon the six domains found in previous studies (Taylor, 1996). These results suggest that the three factor model may be an artifact caused by utilization of the ASI, which was not designed to explore the multifactorial structure of anxiety sensitivity (Taylor et al., 1999).

There are additional questions about the identification of the factors composing anxiety sensitivity, using the Anxiety Sensitivity Index (ASI). The factor labeled as the fear of observable reactions to anxiety has often been found in factor-analytic studies of the ASI. However, the fear of negative evaluation, a theoretical construct that is part of the expectancy theory framework, may be too heavily represented in some of the original ASI items (Cox et al. 1999). In some studies, experts advocate for the deletion of the items that assess the fear of negative evaluation (e.g., “It embarrasses me when my

In related research, evidence has been found for a hierarchical structure for the anxiety sensitivity construct using the ASI and factor analysis. Because of the choices made by investigators to focus on either the upper or lower level or to make no differentiation between higher and lower level factors, results have been contradictory. Because distinct factors may be linked to distinct mechanisms (Cattell, 1978), for instance, in the etiology of anxiety reactions, accurately establishing the validity of the AS construct, its measures, and the factors which make up the structure is important (Zinbarg, Mohlman, & Hong, 1999).

Turning next to the etiology of anxiety symptoms, there has been a growing body of evidence for the presence of elevated anxiety sensitivity across anxiety disorders. To understand the etiology of panic, it was initially, and continues to be important to explore the role that anxiety sensitivity plays in the development of panic attacks. With this exploration has come an agreement of theoretical and empirical efforts of the past 20 years that anxiety sensitivity occupies a principal position in the disposition and etiology of anxiety disorders in general and panic disorder in particular (Barlow, 1988, 1991; Clark, 1986; Goldstein & Chambless, 1987; McNally, 1990; Reiss, 1991; Reiss & McNally, 1985; Reiss et al. 1986; Zinbarg et al., 1997; Zinbarg et al., 1999;).

Related research has sought to examine evidence for predisposition and environmental learning for elevated anxiety sensitivity. A small part of these investigations has focused on the exploration of the roles played by interpretive,
attentional, memory, interoceptive acuity, and cognitive biases in the online processing of anxiety sensations. At the present time, results of these investigations have been quite inconsistent. With the goals of understanding etiology and the development of improved assessment and preventive treatment in mind, it is important to know whether or not individuals who are at risk for the development of panic and other anxiety disorders have the same biases as those who currently experience the symptoms of those disorders (McNally, 1999).

Other issues of etiology include the relationships of the anxiety sensitivity construct with the broader personality domains found in several theoretical models of personality. Recently there has been some focus on the relationships between anxiety sensitivity and the personality domains of neuroticism (NE) and trait anxiety (Eysenck, 1991; Reiss, 1997). A recent study that included the ASP (Van der Does et al., 2003) identified a significant relationship between AS and avoidance of harm, an underlying factor of the higher order dimension of Constraint (CN; Tellegen, 1978/1982; Tellegen & Waller, 1994).

The most widely used instrument to assess anxiety sensitivity in the previously mentioned investigations has been the 16-item Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992). The ASI is the gold standard. It has been shown to produce reliable scores, and to differentiate between panic disorder patients and patients with other psychiatric disorders, including anxiety disorders (Taylor, Koch, & McNally, 1992). Furthermore, ASI scores predict the occurrence of panic attacks in healthy individuals.
during a highly stressful period (Schmidt, Lerew, & Jackson, 1997; Schmidt, Lerew, & Jackson, 1999).

As previously mentioned, the question of whether or not anxiety sensitivity is uni- or multidimensional has been controversial (Lilienfeld, Turner, & Jacob, 1993; McNally, 1996). This question is important because if anxiety sensitivity is in fact multidimensional, relationships between a global measure of anxiety sensitivity and other variables may be obscured. For instance, the relationship between anxiety sensitivity and response to carbon dioxide challenge may be less strong than the relationship between an assumed but unproven anxiety sensitivity dimension (i.e., suffocation fear) and response to carbon dioxide challenge. Research findings in this area seemed to be inconsistent, with about half the number of studies supporting the unidimensional view and half supporting the multidimensional. However, it has been shown that a hierarchical model of anxiety sensitivity, as measured by the ASI, consisting of three partially distinct first-order factors and one general second-order factor, resolves these inconsistencies (Zinbarg et al. 1999). The three first-order factors are: (1) physical concerns, (2) mental incapacitation concerns, and (3) social concerns (Zinbarg et al. 1999). However, the results of factor analysis depend heavily on the comprehensiveness of the measurements. The ASI has only 16 items and may be too short to assess reliably the separate dimensions of anxiety sensitivity (Cox, Parker, & Swinson, 1996). In fact, the three factor model may be an artifact. It has also been argued that the ASI contains a number of problematic items (Blais, Otto, & Zucker, 2001). A re-analysis of previous studies
showed that an 11-item ASI with only two subscales might be a more precise index of anxiety sensitivity (Blais et al., 2001).

Due to the apparent limitations of the ASI, the development of more psychometrically sound measures of anxiety sensitivity is critical. As every clinician knows, common catastrophic cognitions of patients with panic disorder include more than two or three themes (e.g., fear of heart failure, suffocation fear, fear of losing control, fear of fainting, or fear of acting ridiculous). Furthermore, some patients believe that their symptoms cause future damage (Van der Does et al., 2003). Considering the importance of the concept of anxiety sensitivity for cognitive theory, the number and nature of first-order anxiety sensitivity dimensions is not trivial. It would be interesting, for instance, to determine whether or not different anxiety sensitivity profiles exist for patients who present at a cardiology department with atypical chest pain, for patients who are seen by neurologists for unexplainable dizziness, and for patients who consult pulmonologists because of attacks of breathlessness. Because only a small minority of these patients have DSM-IV panic disorder (Pollack, Kradin, Otto, Worthington, Gould, Sabatino, & Rosenbaum, 1996; Worthington, Pollack, Otto, Gould, Sabatino, Goldman, & Rosenbaum, 1997; Van Peski-Oosterbaan, Spinhoven, Van der Does, Willems, & Sterk; 1996; Van Peski-Oosterbaan, Spinhoven, Van der Does, Bruschke, & Rooijmans, 1999), these populations may constitute examples of single-dimension anxiety sensitivity elevations (Van der Does et al., 2003).

In an attempt to measure six dimensions of anxiety sensitivity identified across studies (Taylor, 1996), Taylor & Cox (1998) created a new measure, the 60-item Anxiety
Anxiety Sensitivity: Validity of the ASP

Sensitivity Profile (ASP). The ASP was designed to measure the following six dimensions: fear of (1) cardiovascular symptoms, (2) respiratory symptoms, (3) gastrointestinal symptoms, (4) neurological/dissociative symptoms, (5) publicly observable anxiety symptoms, and (6) cognitive dyscontrol. However, a factor analytic study with 349 college students revealed four dimensions and one second-order general dimension of anxiety sensitivity (Taylor & Cox, 1998). The four dimensions were fear of (1) respiratory symptoms, (2) cognitive dyscontrol, (3) gastrointestinal symptoms, and (4) cardiac symptoms. In comparison with the ASI factors (Zinbarg et al., 1999), physical concerns split into three dimensions and there is no dimension for social concerns. The appropriateness of this 4-dimensional model is questionable, however, since the eigenvalue plot indicated a one- or two-dimensional structure (Van der Does et al., 2003).

This measure may be psychometrically superior to the ASI because it has been designed to address the issues previously presented. All the items in the ASP appear to be specific to the anxiety sensitivity construct. In addition, the ASP has a sufficient number of items to establish internal consistency reliability and convergent validity for each scale. These are steps that are necessary, although not sufficient, to establish the construct validity of a measure. A sufficient number of items on each scale will also increase the possibility that the lower-order factors of anxiety sensitivity will be more accurately identified (Taylor & Cox, 1998). However, given that this measure has been examined in only one study with a non-clinical sample, generalizability needs to be established through the completion of additional studies with a clinical population (Kazdin, 1998). The college student sample used by Taylor and Cox (1998) has serious
limitations. It is a highly selective sample, particularly in regard to the restricted range of age and education and the expected range of ASP scores (Blais et al. 2001; Van der Does et al., 2003). In fact, the ASP item distributions were so skewed that they had to be log-transformed before further analysis could be performed (Taylor & Cox, 1998; Van der Does et al., 2003). A replication of this study in a more diverse population seems warranted before making any conclusions regarding the number of dimensions of the ASP (Van der Does et al., 2003).

This study addresses this issue through the use of a varied clinical sample consisting of individuals who have one or more anxiety disorders. This study seeks to establish further the reliability and validity of their instrument with a clinical population. It is important that these be established. By seeking also to provide evidence of convergent validity for the ASP with a clinical sample, this study may provide further support for the efforts of Taylor and Cox (1998), who are attempting to improve the understanding of the AS construct through their development of a measure (i.e., ASP) that may provide a more comprehensive identification of the underlying factors of anxiety sensitivity. This study will provide analyses that may establish convergent and divergent validity for the ASP. Convergent validity may be established through a high correlation with the ASI, the gold standard in measurement of the anxiety sensitivity construct.

In addition, this study may provide support for the concept that anxiety sensitivity is a multifactorial, rather than unifactorial construct. Additional support for the existence of six underlying factors, including the fear of observable reactions to anxiety, may be
Evidence that may be provided for reliability and validity of this construct might provide further evidence that anxiety sensitivity is not an artifact of another construct such as the fear of pain, or another trait. Consistent with previous investigations, the results of this investigation may provide further proof that individuals with other anxiety disorders also have elevated anxiety sensitivity. And finally, the current study may provide evidence that the ASP is a psychometrically superior instrument which can support accurate lower level factor identification and construct validity for the anxiety sensitivity general factor.

Theoretical Background and Rationale

Theoretical Background

*Expectancy theory.* Expectancy theory is the earliest attempt to provide a theoretical framework linking anxiety sensitivity as a causal factor to fear, anxiety, panic, and avoidance behavior (Reiss, 1980, 1991; Reiss & McNally, 1985; Taylor & Fedoroff, 1999). This theory was constructed to provide an understanding of the part that anxiety sensitivity plays in the impact of anxiety on people's functioning (Taylor & Fedoroff, 1999). Central to the framework provided by expectancy theory are three fundamental fears or sensitivities: (a) anxiety sensitivity, (b) the fear of illness, injury, and death, and, (c) the fear of negative evaluation (Reiss, 1991). This core has more recently been
modified and expanded (Reiss & Havercamp, 1996, 1997), but the main concept has been retained. According to current expectancy theory, anxiety sensitivity is still viewed as a fundamental fear, and with the other fundamental fears, increases fear, anxiety, and panic. It must be noted, however, that at the present time it has not been established that anxiety sensitivity is distinguishable from the fear of pain, or other fundamental fears (Taylor & Fedoroff, 1999). Neither has anxiety sensitivity been established as a cause of fear, anxiety, panic, and avoidance behavior.

In addition to fundamental fears, Reiss (1991) identified a second distinct category of fear, which he refers to as common fears. The criteria by which he differentiates these two categories are: (a) most people would describe exposure to stimuli that cause a fundamental fear as a negative experience, but the same cannot be said of common fears and (b) common fears such as fears of harmless animals, situations (e.g., heights, enclosed spaces) and social fears (e.g., public speaking, eating in public; Reiss, 1991), are based on fundamental fears. Thus, according to this theory, it appears that fundamental fears exist on a continuum that begins in the range of normal experience and increases to become problematic for some individuals. Common fears, on the other hand, are apparently linked to the continuum for the fundamental fears and affect the individual when their fundamental fears are in the problematic range.

In addition to these two criteria, fundamental fears cause the fear of a large number of stimuli while common fears do not. For example, an individual with a high level of anxiety sensitivity might have more than the normal fear of a number of situations, such as exposure to harmless snakes or to heights. These are situations that
the individual suspects will cause the experience of panic. They are feared because he or she has more than the normal fear of the possible occurrence of a panic attack during exposure to these situations (Reiss, 1991). To explain further, anxiety sensitivity is described as the fear of anxiety and the symptoms of anxiety, and therefore, the fear of having a panic attack would be perceived as the fundamental fear. The fear of specific situations (i.e., common fears) is the result of the interaction of fundamental fears and learning experiences (Taylor & Fedoroff, 1999).

When fundamental fears interact with learning experiences, individuals may acquire common fears, or fears that the individual had previously acquired may acquire additional strength (Taylor & Fedoroff, 1999). Central to this process are three forms of learning: (a) traumatic conditioning experiences, (b) observational learning, and (c) learning from verbal information regarding the threat of specific stimuli (Rachman, 1990). An illustration of this would be the acquisition of dog phobia by the individual who has been mauled by a dog. The fundamental fear of illness/injury/death sensitivity becomes connected with dogs (i.e., common fear) and anything that reminds the individual of dogs (i.e., learning). In addition, this fear may be increased by other fundamental fears, such as anxiety sensitivity. In this case the individual is also fearful of the anxiety that the dog evokes (Taylor & Fedoroff, 1999).

*Does anxiety sensitivity meet the criteria of a fundamental fear?* Anxiety sensitivity appears to meet the first of the criteria for fundamental fears (i.e., most people find exposure to certain stimuli to be a negative experience). Most people taking part in
studies using the Anxiety Sensitivity Index (Peterson & Reiss, 1987), currently the most frequently used measure of anxiety sensitivity, have indicated they have some fear of anxiety. Only a small number of people indicate an absence of this fear (Taylor & Fedoroff, 1999). The findings for a sample of 818 university students illustrated this point with only four students (0.5%) reporting a score of zero on the ASI (Stewart, Taylor, & Baker, 1997).

The second criterion is that common fears are based on, and can be logically reduced to, fundamental fears. The manner in which this is described has been illustrated in a number of published studies (Taylor & Fedoroff, 1999). For example, for individuals with high anxiety sensitivity, a fundamental fear, the fear of panic attacks is triggered by exposure to animals; this was found to be the main reason that some animal phobics were fearful of certain animals (McNally & Steketee, 1985). Those with the fundamental illness/injury/death sensitivity, which centered on the phobia of flying, were afraid that the plane would crash. Individuals with agoraphobia feared flying due to a fear of having a panic attack at some point during the flight due to anxiety sensitivity (McNally & Louro, 1992; Reiss, 1991).

Questions have arisen with the examination of two important components that are part of the second criterion for a fundamental fear (Reiss, 1991): (a) anxiety sensitivity must be distinguishable from other fears in order to be considered a fundamental fear, and (b) anxiety sensitivity cannot be further reduced to fears that are even more basic. These concepts have received mixed support in recent studies. Pertaining to the first concept, a factor analytic study using community volunteers as a sample found that
anxiety sensitivity was factorially distinct from illness/injury/death sensitivity and from the fear of negative evaluation (Taylor, 1993). Further evidence that anxiety sensitivity is factorially distinct from other fundamental fears was provided by factor analytic studies of mentally retarded samples in terms of Reiss' expanded list of sensitivities (Reiss & Havercamp, 1996, 1997). However, anxiety sensitivity overlaps with the fear of pain in other samples (Reiss & Havercamp, 1996, 1997), raising some doubt as to the ability of anxiety sensitivity to fulfill the requirements of the second criterion.

According to the second concept, as long as anxiety sensitivity cannot be reduced to more basic fears, it can be regarded as fundamental. There is growing evidence regarding this concept, which raises further doubt that anxiety sensitivity meets the requirements of the second criterion. In these studies using the ASI, anxiety sensitivity has been shown to consist of three correlated factors: (a) the fear of somatic sensations, (b) the fear of the loss of cognitive control, and (c) the fear of anxiety reactions that are observable by others. It has been proposed that other factors underlie even these (i.e., fear of death, insanity, and negative evaluation; Taylor & Fedoroff, 1999). If part of the purpose of expectancy theory is to provide a framework that explains the nature of anxiety sensitivity as a construct, it will need further development that takes into account these findings regarding factorial distinction and more basic underlying fears.

The opinion has been expressed that factorial distinction is not a criteria that would imply the identification of anxiety sensitivity as a fear, rather than as a variable moderating fear (e.g., anxiety and depression are distinct, but correlated, and one would not call depression a form of anxiety). S. Taylor (personal communication, November
Concerning the concept of anxiety sensitivity as a variable moderating or mediating fear rather than being a fundamental fear, S. Taylor (personal communication, November 26, 2001) reported that anxiety sensitivity may be both; it may be in some sense a fundamental fear and it also appears to influence other fears. Anxiety sensitivity is not fundamental in any absolute sense, because it can be decomposed into components (e.g., its factors, which, in turn, are influenced by more basic factors such as environmental and genetic determinants). The more important question is whether or not anxiety sensitivity predicts or causes important phenomena, and the evidence points
toward its mediational role. T. Brown (personal communication, November 25, 2001) reported that the conceptual thinking about anxiety sensitivity is that early experiences (e.g., parental modeling) result in anxiety sensitivity, which in turn may result in panic. Specifically, the assertion that childhood learning has a direct effect on anxiety sensitivity is not necessarily claimed by a moderated relationship, but is more likely to be that of a mediated relationship.

On one hand, as S. Taylor suggests (personal communication, November 26, 2001), it is possible to conceptualize anxiety sensitivity as a moderator variable of other fears. For example, anxiety sensitivity has been found to be present in social phobia and perhaps its presence and its level of intensity increase symptoms such as avoidance behavior that are related to the fear of negative evaluation. However, returning to a possible mediational role for anxiety sensitivity, one study has already claimed a partial mediational role for anxiety sensitivity for panic attacks. Stewart et al. (2001) found in a retrospective study that learning history for arousal-reactive somatic symptoms (i.e., nausea, racing heart, shortness of breath, and dizziness) directly influenced both anxiety sensitivity levels and panic frequency, and that anxiety sensitivity directly influenced panic frequency. Stewart et al. (2001) stated that learning history is an indirect cause of panic frequency, another requirement of the mediational relationship, as reported by T. Brown (personal communication, November 25, 2001). However, as T. Brown (2001) and Stewart et al. (2001) cautioned, this is a cross-sectional study, which cannot really address conditions of mediation because it evaluates a cross-sectional pattern of correlations, and cannot firmly establish causality.
Anxiety Sensitivity: Validity of the ASP

Expectancy theory and anxiety expectancies. Anxiety sensitivity and the larger theoretical framework of anxiety expectancy were also developed as an attempt to explain individual differences regarding the development of common fears (Reiss, 1980, 1991; Reiss & McNally, 1985). Through what appears to be a mediational role (i.e., through a certain relation or mechanism, A leads to B), expectancy theory attempts to provide a link between the individual’s fundamental fears and his or her assumptions of a negative outcome in specific situations. This approach emphasizes not only experiences of direct conditioning, but also focuses on the individual acquiring outcome expectancies, possibly through observational learning and verbal information regarding threat. The conditioned stimulus becomes linked with the expectation that the unconditioned stimulus will take place (Davey, 1992). This concept shares an emphasis on expectations with neo-conditioning theory (Rachman, 1991; Taylor & Fedoroff, 1999). In fact, expectancy theory was conceived through the extension of the neo-conditioning approach (Reiss, 1980, 1991; Reiss & McNally, 1985), which explains in detail the creation of fear-related responses, including anxiety, panic, avoidance, and escape behavior, through the combination of anxiety sensitivity and anxiety expectancies. Each individual’s expectations and sensitivities combine to produce his or her responses to fear (Taylor & Fedoroff, 1999).

Each individual’s expectations are his or her beliefs about what will take place when he or she is exposed to the feared object or situation. Examples of such expectations pertaining to each of the three fundamental fears or sensitivities are the belief that the plane will crash and all on board will either die or be severely disabled, the
expectation of having a panic attack during the flight, and the belief that other people will notice his or her fear of flying and negative evaluation by others will follow (Reiss, 1991). The underlying causes for fearing the foreseen event are the three fundamental sensitivities, also referred to as fundamental fears. Examples of these causes are the individual’s belief that he or she cannot stand the thought of being handicapped, that panic attacks cause heart attacks, and the belief that it would be horrible if people were to laugh or think he or she is stupid when they notice the individual’s fear of flying (Reiss, 1991). According to this theory there is a great variety of differences in sensitivities among individuals. Anxiety sensitivity is a person-specific factor and danger and anxiety expectancies are situation-specific factors (Reiss & McNally, 1985). The individual possesses anxiety sensitivity, which interacts with the expectation of danger that is specific to the situation.

*Fear, anxiety, and avoidance.* Expectancy theory seeks to identify and explain the interactions that cause pathological anxiety. To identify the possible interactions that result in fear, anxiety, and avoidance, six variables are perceived as having an influence on common fears (e.g., fear of heights). These are (a) the expectation of actual danger or disaster (e.g., the individual’s expectation that he or she might fall while looking over the cliff edge), (b) anxiety expectancy (e.g., the individual’s expectation that he or she will panic), (c) social disaster expectation (e.g., the expectation that people will laugh at the individual if they see that he or she is fearful of heights), (d) sensitivity to injury, illness, and death (e.g., the belief that it would be terrible to fall), (e) anxiety sensitivity (e.g. the
fear of anxiety and anxiety-related symptoms), and (f) negative evaluation sensitivity (e.g., the individual's belief that it would be catastrophic to experience people laughing at him or her; Taylor & Fedoroff, 1999).

When the person is exposed to his or her ordinary, but feared situation, the level of fear or anxiety experienced by the person, as suggested by the model, is partially decided by the interaction between anxiety sensitivity and the person's expectations of anxiety. The components of the individual's anxiety reaction that can be linked to a fear situation are not clear in Reiss' theory (e.g., shaking, rapid heart beating; Taylor & Fedoroff, 1999). It is possible that it refers to the individual's belief about the intensity of anxiety or fear that will be reached if exposure to the feared object or situation continues (Taylor & Fedoroff, 1999).

*The Expectancy Theory of Fear*

Proposed revisions for expectancy theory have been necessitated by closer examination of the experience of individuals with pathological anxiety and the evidence for the existence of lower-order factors for anxiety sensitivity. The following studies have given rise to additional interesting questions about the theory. The first of these involves the assumption that anxiety expectancies (i.e., the expectations of arousal) are empirically different from danger expectancies (i.e., the expectations of objective disaster). Scales were developed to assess both expectancies in order to test this view with three fears: (a) fear of flying, (b) fear of heights, and (c) fear of public speaking.
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(Gursky & Reiss, 1987). One hundred thirty-five college students completed the scales and the results were factor analyzed. The assumption was supported by findings of a two-factor solution for each fear in which danger and anxiety expectancies represent separate factors (Gursky & Reiss, 1987; Taylor & Fedoroff, 1999).

The fact that fear arises from the interaction between expectancies and sensitivities is another assumption for which three studies produced relevant results (Schoenberger, Kirsch, & Rosengard, 1991). Ninety-four college students, who feared snakes, were asked to approach and touch a live, harmless snake. Subjects rated danger expectancy, anxiety expectancy, and self-reported fear, completing measures of danger sensitivity (i.e., illness/injury/death sensitivity) and anxiety sensitivity. The prediction of self-reported fear was obtained through hierarchical regression analyses. As the regression coefficient for expected danger grew smaller, the danger sensitivity scores went higher, which suggests that these results are exactly opposite from those suggested by Reiss’ expectancy theory (Schoenberger et al., 1991).

In a related study, one hundred seventeen college students who were fearful of enclosed spaces were asked to walk down a long, narrow, darkened dead-end corridor (Valentiner, Telch, Ilai, & Hehmsoth, 1993). Significant predictors of avoidance behavior, as measured by the amount of time spent in the corridor, were danger expectancy, anxiety expectancy, and the interaction between anxiety sensitivity and anxiety expectancy. On the other hand, heart rate and self-reports of fear were not predicted by these variables. An older version of the expectancy theory was tested in this study (Reiss, 1980; Reiss & McNally, 1985), in which fear is determined by danger
expectancy and the anxiety sensitivity-by-anxiety expectancy interaction. The newer version (Reiss, 1991) proposes that fear is determined by three expectancy-by-sensitivity interactions. Nevertheless, the view that fear responding is a function of the interaction between anxiety sensitivity and anxiety expectancy receives only limited support from the results (i.e., only avoidance behavior was predicted; Taylor & Fedoroff, 1999; Valentiner et al., 1993).

In a study which explored the interaction between anxiety sensitivity and anxiety expectancy (Telch & Harrington, 1994), thirty-nine high anxiety sensitivity and 40 low anxiety sensitivity college students were asked to inhale a harmless mixture of 35% carbon dioxide and 65% oxygen. This compound is a strong panic inducer for people with high anxiety sensitivity because it causes bodily sensations that are related to anxiety arousal (McNally, 1996). Subjects were told to expect either a state of relaxation or a state of arousal. Inhalation-induced panic attacks were most frequent among high anxiety sensitivity subjects in the expect-relaxation condition (52%), compared with high anxiety sensitivity subjects in the expect-arousal condition (17%). Ratings for inhalation-induced fear were similar. Therefore, the findings were directly opposite to those predicted by the expectancy theory (i.e., a negative interaction between anxiety sensitivity and anxiety expectancy; Telch & Harrington, 1994). It is possible that in further revisions of this theory (Taylor and Fedoroff, 1999), the match or mismatch between what is expected and what actually occurs needs to be considered. It is also possible that other variables influenced the outcome. Perhaps high anxiety sensitivity subjects who expected arousal had a sense of safety or freedom from harm because the
anxiety sensitivity plays in the initiation and development of anxiety pathology. This section will promote an understanding of the fit of the present theoretical work with research efforts aimed at prevention and refinement of treatment.
The Possible Role of Anxiety Sensitivity in the Etiology and Maintenance of Anxiety Disorders

Sources of the fear of anxiety; This section will begin with a brief overview of the development of anxiety sensitivity. Anxiety sensitivity and relevant constructs, (e.g., the fear of fear), have been given, by agreement of theoretical and empirical efforts of the past 20 years, a principal position in the disposition and etiology of anxiety disorders in general, and panic disorder in particular (Barlow, 1988, 1991; Clark, 1986; Goldstein & Chambless, 1978; McNally, 1990; Reiss, 1991; Reiss & McNally, 1985; Reiss et al., 1986; Zinbarg et al., 1999;). Anxiety sensitivity is perceived to be a trait-like characteristic that exists before the development of pathological anxiety. Individual differences in anxiety are developed by the interaction of anxiety sensitivity with a number of varied experiences, leading to a collection of beliefs about the harmful results of anxiety arousal. Utilizing social learning theory, the individual is perceived as becoming fearful of the sensations of anxiety arousal through spoken and observational means (e.g., hearing others express fear of such sensations, receiving misinformation about the harmfulness of certain sensations, witnessing a catastrophic event such as the fatal heart attack of a loved one; Schmidt, Lerew, & Joiner, 1998); fears, however, can also develop through direct conditioning (i.e., the Pavlovian interoceptive formulation; Rachman, 1977). Therefore, from the anxiety sensitivity point of view, the individual can develop a fear of anxiety not only from direct experience with panic, but by other means as well (McNally, 1990). A high score on the ASI does not require the experience of
Anxiety sensitivity is a risk factor for anxiety disorders in general and panic disorder in particular (Reiss & McNally, 1985; McNally & Lorenz, 1987). This hypothesis has been confirmed in several studies. In an important longitudinal study, using a sample of cadets at the U.S. Air Force Academy during basic training (Schmidt et al., 1997), it was demonstrated that the occurrence of spontaneous panic attacks and anxiety symptoms were predicted by elevated ASI scores (Peterson & Reiss, 1987). Using a nonclinical sample of 151 college students assessed with the ASI, a three year follow-up study was conducted with a subsample of 23 high anxiety sensitivity subjects and 25 low anxiety sensitivity subjects (Maller & Reiss, 1992). The frequency and intensity of panic attacks during the follow-up period was predicted by scores on the ASI (Peterson & Reiss, 1987).

In addition, there is evidence that anxious responders to provocation challenges (e.g., voluntary hyperventilation; Rapee & Medoro, 1994) are predicted by elevated ASI scores (Taylor & Cox, 1998). In a one-year prospective study, ASI scores predicted the first spontaneous panic attack in normal controls as well as in patients with specific phobias (Ehlers, 1995). Maintenance/relapse was also predicted by ASI scores in panic disorder patients (Ehlers, 1995). In an investigation of alprazolam discontinuation in panic disordered patients, only one predictor, baseline-to-post taper change in ASI scores, predicted the proportion of medication-free patients at 6-month follow-up (Bruce, Spiegel, Gregg, & Nuzzarello, 1995).
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Challenges to expectancy theory. The development of these hypotheses (i.e., anxiety sensitivity is a trait-like characteristic that exists before the development of pathological anxiety; anxiety sensitivity is a risk factor since the occurrence of panic can be preceded by elevated anxiety sensitivity; McNally & Lorenz, 1987; Reiss & McNally, 1985; Schmidt et al., 1998), and their confirmation through the studies listed above has brought about a challenge to the original expectancy theory. Reiss (1991) stated that not only can panic attacks be preceded by anxiety sensitivity, but the experience of panic attacks and the severity of panic symptoms should be connected with high anxiety sensitivity (Peterson & Reiss, 1987). It was suggested by Reiss (1991) that expectancy theory could explain panic attacks, even spontaneous (unexpected) attacks, one of the criteria for panic disorder (American Psychiatric Association, 1994). Reiss (1991) proposed that the response of fear is the result of the individual’s belief about what will happen when he or she is faced with a feared object or situation (e.g., I expect to have a panic attack when I’m in the shopping mall). However, the manner in which anxiety sensitivity might trigger unexpected panic attacks is not explained by expectancy theory (Taylor & Fedoroff, 1999).

There is, however, a proposal that uses expectancy theory combined with Clark’s model (1986) to explain panic attacks (Taylor & Fedoroff, 1999). This proposal begins by stating that anxiety sensitivity is an anxiety amplification factor and is believed to be a diathesis factor in the etiology of anxiety disorders, especially panic disorder (Reiss, 1991; Reiss & Havercamp, 1996; Reiss & McNally, 1985). The series of actions through which these sensations could be increased to the level of panic due to the fear of the
bodily sensations of anxiety are depicted in Clark’s model (Clark, 1986). Therefore, it is possible to combine Clark’s cognitive model of panic with expectancy theory (Taylor, 1995a). To describe this further, the catastrophic misinterpretations of the bodily sensations of anxiety cause panic attacks (i.e., misinterpreting the sensations as signs of impending death, insanity, or loss of control), according to Clark. There is a lasting inclination to make such misinterpretations on the part of individuals who possess the tendency to have panic disorder. These individuals have elevated anxiety sensitivity, from the viewpoint of Reiss’ (1991) theory, because they expect to experience bodily sensations that are associated with anxiety (Taylor & Fedoroff, 1999).

This proposed model can explain panic attacks that are triggered by exposure to a feared object, such as a spider in the case of spider phobia. But how does it explain panic attacks that seem to come out of the blue? Because expectations of panic do not occur, it is possible that expectation theory does not explain the subjectively unexpected panic attacks. However, a response is proposed: the individual’s expectancy of anxiety, which is connected with a feared situation, is triggered by bodily sensations. These are connected with arousal occurring just prior to the panic attack (Taylor & Fedoroff, 1999). Also, the view that awareness of benign bodily sensations (e.g., faintness caused by rapidly changing posture, derealization caused by fatigue or bright fluorescent lights, and palpitations caused by mild exertion) is the trigger for unexpected panics is based on Clark’s (1986) model. The individual with elevated anxiety sensitivity tends to assume that these bodily sensations are linked with bodily harm after they are triggered and discovered. This process begins entry into the vicious cycle of panic (Taylor & Fedoroff,
On the other hand, these sensations would be considered temporary and disagreeable, but harmless, by an individual with low anxiety sensitivity (Watt, Stewart, & Cox, 1998).

The proposed role of anxiety sensitivity in the other anxiety disorders. The previous section contains information about the currently proposed manner in which the construct of anxiety sensitivity interacts with Clark's model (1986) and expectancy theory (Reiss, 1991) in the etiology of panic disorder. This section will discuss the role that anxiety sensitivity plays in the other anxiety disorders. Although research provides evidence that anxiety sensitivity is elevated in all anxiety disorders, with the exception of specific phobias, there is evidence that anxiety sensitivity is found to be most elevated in panic disorder and post traumatic stress disorder (PTSD; Taylor et al., 1992). With the readily perceived role which anxiety sensitivity plays in the vicious cycle of panic (Clark, 1986), the discovery that anxiety sensitivity is more elevated in panic disorder compared with most other anxiety disorders agrees with expectancy theory (Reiss, 1991). However, ASI scores are almost as high in PTSD as they are in panic disorder (Taylor et al., 1992; Taylor et al., 1999), while lower, but elevated, ASI scores are obtained from people with generalized anxiety disorder, obsessive-compulsive disorder, and social phobia (Taylor et al., 1992).

The cause for the level of elevation of ASI scores in PTSD may be found by examining the reason that anxiety sensitivity is elevated in panic disorder. As reported above, a reciprocal association between anxiety sensitivity and panic was suggested by
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Reiss (1991). Panic attacks may be caused by elevated anxiety sensitivity and anxiety sensitivity, in turn, may be increased by panic attacks because they are terrifying experiences (Taylor et al., 1999). The sufferer may come to fear the symptoms of anxiety in general when there are repeated attacks, because anxiety symptoms will be viewed as a warning that a panic attack might follow (Taylor et al., 1999). There might possibly be a similar reciprocal association for some symptoms of PTSD that increases anxiety sensitivity in the same manner in which panic attacks increase anxiety sensitivity (e.g., flashbacks), that is not present in OCD or GAD (Taylor et al., 1999).

The core criteria of PTSD are the re-experiencing of thoughts, images, and memories related to the trauma (American Psychiatric Association, 1994). A flashback, an experience that meets these criteria, appears to have a number of the same qualities possessed by panic attacks (Jones & Barlow, 1990). For instance, internal or external stimuli trigger both of these brief, intense experiences (Davidson & Foa, 1991), and both have many of the same symptoms in common (Burstein, 1985; Jones & Barlow, 1990; Mellman & Davis, 1985). Seventy-two percent of patients diagnosed with PTSD at one treatment site experienced flashbacks one or more times per week (Mellman & Davis, 1985). This provides evidence that flashbacks in PTSD may be as common as panic attacks in panic disorder. It has, in fact, been suggested that flashbacks may be a form of panic attack (Mellman & Davis, 1985). If panic attacks elevate anxiety sensitivity because they are rapid in onset, and are intense and terrifying experiences, the same causes in PTSD may elevate anxiety sensitivity (Taylor et al., 1992). In contrast, the symptoms which are criteria for OCD and GAD might elevate anxiety sensitivity less
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because they do not have the quality of a sudden outburst or crisis, which is part of panic attacks and flashbacks (Taylor et al., 1992). As reported by Taylor et al. (1999), this proposed explanation has not been explored.

Although the possibility that similar ASI scores for PTSD and panic disorder might be due in part to the similarity between panic attacks and flashbacks has not been tested, there is evidence in the Taylor et al. (1992) study that there is a different item response pattern across the disorders. For individuals who experience panic disorder, cardio-respiratory fears appear to be most salient, while psychological sensations (e.g., “When I cannot keep my mind on a task, I worry that I might be going crazy.”) are endorsed more frequently by PTSD patients (Cox et al., 1999). Through a process of associative learning (interoceptive conditioning; Bouton, Mineka, & Barlow, 2001), anxiety sensitivity may be inflated by trauma exposure. Anxiety sensitivity may then amplify PTSD symptoms. For example, the person may become alarmed by re-experiencing symptoms, believing them to be harbingers of insanity (Taylor, 2003).

Whether or not there is a distinct type of anxiety sensitivity which is more significantly related to PTSD might be determined by a multidimensional measure of anxiety sensitivity that is better than the ASI at evaluating the fear of cognitive dyscontrol (e.g., the ASP). It would be useful to test subjects who have not yet developed PTSD, but have been exposed to traumatic events, in order to evaluate such a measure’s ability to predict its onset in a prospective study. If successful, such a screen of people who are at risk for PTSD would identify individuals who might be helped by early intervention procedures such as critical incident stress debriefing (Cox et al., 1999). In addition, regardless of the
actual relationship between elevated anxiety sensitivity and PTSD, it is suggested that treatment of PTSD may be improved by incorporating treatments that reduce anxiety sensitivity, regardless of whether or not the person has comorbid panic disorder (Taylor, 2003).

In contrast to these studies, there are several other studies in which ASI scores have been found to be close to or even higher in social phobia, than those for panic disorder (Asmundson & Stein, 1994; Ball, Otto, Pollack, Uccello, & Rosenbaum, 1995; Hazen, Walker, & Stein, 1995; Maidenberg, Chen, Craske, Bohn, & Bystritsky, 1996; Marks, Lindsay, Marks, & Alkubaisy, 1988). A different pattern of item responding is also suggested for social phobia, in comparison with that of panic disorder, with significantly higher scores on three items: (a) “It is important to me not to appear nervous,” (b) “It embarrasses me when my stomach growls,” and (c) “Other people notice when I feel shaky,” (Cox et al., 1999).

The fear of negative evaluation, a proposed fundamental expectancy fear, which is separate from anxiety sensitivity theoretically (e.g., Reiss & McNally, 1985) and empirically (Taylor, 1993), appears to be assessed by the contents of these three items. It is also possible that in social phobia, the individuals who experience these symptoms may fear publicly observable anxiety symptoms (e.g., sweating, blushing, trembling), if they believe that these symptoms have harmful social consequences (Cox et al., 1999). Initial evidence has, in fact, been provided that both of these are separate factors within the ASI (McWilliams, Stewart, & MacPherson, 2000). However, the criticism remains that the ASI Social Concerns subscale is psychometrically weak because it contains so few items
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(e.g., Stewart et al., 1997). It is important, therefore, in different clinical situations, to seek further clarification of the nature of anxiety sensitivity through item-level or dimensional analyses in order to settle such issues (Cox et al., 1999) with a measure that adequately assesses this factor.

Significant contamination by the fear of negative evaluation of some of the original ASI items is a third possibility, which was empirically supported by the factor-analytic study involving the ASP (Taylor & Cox, 1998). In this study with a non-clinical sample, the fear of publicly observable symptoms did not have the weight required to be identified as a factor. It is also possible, that although the fear of negative evaluation does indeed saturate an expanded or revised measure of anxiety sensitivity, that it is spread throughout the items and thus is not observable as an obvious separate factor (Cox et al., 1999). Taylor and Cox (1998) also argue, as does the current study, that when there are sufficient items to reliably assess the fear of observable reactions to anxiety with a clinical sample, the range of scores is sufficient to provide evidence that this fear is an underlying factor of anxiety sensitivity.

The diagnoses of GAD and OCD are areas that have received less attention in systematic research (e.g., item level comparisons, factor analysis); however some studies have provided evidence for elevated ASI scores (Calamari, Wiegartz, Janeck, & Heffelfinger, 1996; Taylor et al., 1992; Zeitlin & McNally, 1993). It is possible, but not known, whether or not unique ASI patterns of responding would also be found for these disorders. Fear of cognitive dyscontrol, rather than the fear of physical sensations, would probably be more pertinent (Cox et al., 1999). Assessment of these clinical groups would
probably benefit from the addition of more cognitive dyscontrol items, as in the ASP, that are related to worry and intrusive thoughts and images (e.g., OCD: “It scares me when an unwanted image pops into my mind” and GAD: “It is frightening when I can’t stop myself from worrying.”). They would also assist with the prediction of onset, exacerbation, and relapse for OCD and GAD (Cox et al., 1999).

Elevation of anxiety sensitivity in specific phobia has not been supported by the available research (Taylor et al., 1992; Sandin, Chorot, & McNally, 1996) and it has been suggested that more relevance may be found in the other fundamental fears (e.g., fear of injury in blood/injury phobia). Nonetheless, anxiety sensitivity may have relevance for some types of specific phobias, or specific facets within it (e.g., fear of physical sensations in choking phobia). Whether or not specific fears and phobias can be amplified by anxiety sensitivity will have to be determined in future research (Cox et al., 1999).

**Information-Processing Biases for Threat**

An examination of information-processing biases provides a good follow-up discussion to the previously mentioned focus on attempts to identify the first-order factors of the anxiety sensitivity construct. Will individuals who have been identified as being at risk for panic disorder and other anxiety disorders on measures of anxiety sensitivity have the same biases as those who currently experience the symptoms of these disorders, including a high level of anxiety sensitivity? That is the main question to be
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answered in the following collections of studies. Relatively stable beliefs about the negative capacity of certain bodily sensations, which are covered in ASI items, are assumed to affect online processing of these sensations (McNally, 1999). Anxiety sensitivity and some of the information-processing biases co-occur with anxiety disorders. There seems to be an implied link, although a causal link has not yet been determined.

Interpretive bias. A tendency to interpret ambiguous events as sources of harm is assumed to be related to pathological anxiety. This factor is expected to increase the number of anxiety episodes, because it is quite common to experience events whose meaning is unclear in everyday life (McNally, 1999). There is solid evidence that biases for interpreting ambiguous information as threatening is a characteristic of people who have not only panic disorder, but who are also often agoraphobic (Harvey, Richards, Dziadosz, & Swindell, 1993; Kamieniecki, Wade, & Tsourtos, 1997; McNally & Foa, 1987; Stoler & McNally, 1991; Westling & Ost, 1995). Elevated anxiety sensitivity has been obtained uniformly in ASI scores, which were included in most of these studies. Reduction of anxiety sensitivity, and sometimes a termination of interpretive biases, has been related to recovery from panic disorder (McNally, 1999). On the other hand, whether or not the experience of panic attacks caused the interpretive bias, or if it was a pre-existing risk factor for the development of panic disorder, is not determined by these studies.
Attentional bias. Another assumption is that any bias for selective attention involving threat will cause anxiety episodes to occur more often due to the fact that people can pay attention only to a portion of existing stimuli at any given moment (Williams, Watts, MacLeod, & Mathews, 1988, 1997). Researchers testing this hypothesis in anxiety disordered patients, including those with panic disorder, have used dichotic listening, the Stroop color-naming test, and the dot-probe attention deployment paradigm (Asmundson, Sandler, Wilson, & Walker, 1992; Burgess, Jones, Robertson, Radcliffe, & Emerson, 1981; Ehlers, Margraf, Davies, & Roth, 1988; Hope, Rapee, Heimberg, & Dombeck, 1990; McNally, Amir, Louro, Lukach, Riemann, & Calamari, 1994; McNally, Riemann, & Kim, 1990; MacLeod, Mathews, & Tata, 1986; Williams, Mathews, & MacLeod, 1996). Results of these studies support this hypothesis. Selective processing of threat cues in most of these attentional tests is a characteristic of panic disordered individuals. On the other hand, some attempts at replication have failed and the response to panic concerns has not been consistent (Asmundson et al., 1992; McNally, 1999). Perhaps more attention needs to be given to individual differences and congruence. If threat cues were not congruent with lower-order anxiety sensitivity factors (i.e., somatic fears, fear of cognitive dyscontrol, fear of anxiety symptoms being observed) that have been endorsed by the individual, it is likely that no attentional bias would be observed.

Memory bias. Especially while experiencing conditions that cause arousal, people diagnosed with panic disorder report that thoughts of threat easily come to a level of
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awareness. This suggests that these thoughts of threat may be readily available. It also implies that information connected to their concerns is more available than other information and more easily recalled by panic disordered individuals than by people who do not have the same concerns (McNally, 1999). The evidence supports patient reports. Explicit (i.e., in the person’s awareness) memory biases for threat have been shown by panic disorder patients more than any other anxiety disorder group (Amir, McNally, Riemann, & Clements, 1996; Beck, Stanley, Averill, Baldwin, & Deagle, 1992; Becker, Rinck & Margraf, 1994; Cloitre, Shear, Cancienne, & Zeitlin, 1994; Ehlers et al. 1988; McNally, Foa, & Donnell, 1989; McNally, Otto, Yap, Pollack, & Hornig, in press; Otto, McNalley, Pollack, Chen, & Rosenbaum, 1994; Pauli, Dengler, Wiedemann, Montoya, Flor, Birbaumer, & Buchkremer, 1997; Rapee, 1994;). Nonetheless, researchers have begun exploration of other individual difference variables that may interact with the diagnostic criteria for panic to produce the symptoms, because memory biases for threat have at times been inconsistent (Otto et al., 1994; Rapee, 1994; McNally 1999). Again, this calls for an examination of the congruence of research variables with the individual’s endorsed lower-order anxiety sensitivity factors.

*Interoceptive acuity bias.* If panic patients possess the skill to detect certain dreaded bodily sensations better than other people, it is likely that their experience of panic will be increased. However, evidence supporting the existence of such a skill has not been consistent (Ehlers, Margraf, Roth, Taylor, & Birbaumer, 1988; Pauli, Marquardt, Hart, Nutzinger, Holzl, & Strain, 1991; Rapee, 1994). It appears that people
are not especially good at detecting dreaded bodily sensations just because they fear them (McNally, 1999). And once again, this calls for attention to congruence of research variables with the individual's endorsed lower-order anxiety sensitivity factors.

**Cognitive biases, high anxiety sensitivity, and people at risk for developing panic attacks.** Individuals with panic disorder often obtain higher scores on other measures, while also scoring higher than other anxiety-disordered people on the ASI (Taylor et al., 1992). This factor poses a problem when attempting to develop relationships between cognitive biases and anxiety sensitivity. In an effort to seek a remedy for this issue, research has begun with people who are assessed as being at risk for panic disorder due to elevated ASI scores (McNally, Hornig, Hoffman, & Han, 1997; Kim, 1992; Sturges & Goetsch, 1996; Stewart, Conrod, Gignac, & Pihl, 1998). Results thus far show that although risk for panic attacks and perhaps panic disorder is indicated by high scores on the ASI, it is not clear whether or not attention or acuity to bodily sensations, interpretation of their meaning, or ready access to memories about harm that influence online interpretation are affected by anxiety sensitivity beliefs. It therefore remains for future research to clarify further the cognitive biases that are possessed by individuals with high anxiety sensitivity, who have not yet had panic attacks, as well as determining exactly what biases are correlated with the disorder (McNally, 1999).
Anxiety Sensitivity and Personality

Until recently, little effort has been put into the exploration of the relationship of the broader personality domain and the anxiety sensitivity construct. This applies especially to any similarity which might exist between anxiety sensitivity and various traits (Lilienfeld, 1996), although quite a bit of work has been focused on the differences between anxiety sensitivity and neuroticism and trait anxiety (e.g., Reiss, 1997). Any work which has been, or will be completed in this area, will be of value in the areas of the etiology of anxiety sensitivity and the etiology of anxiety disorders (Lilienfeld, 1999).

The higher order personality dimension of Neuroticism in the three-factor model of Eysenck (1991), a trait which denotes a pervasive tendency to experience unpleasant or painful emotional states of all types, is moderately to highly related to the measures of anxiety sensitivity, according to studies reviewed by Lilienfeld (1999; Arrindell, 1993; Zinbarg & Barlow, 1996; Saviotti, Grandi, Savron, Ermentini, Bartolucci, Conti, & Fava, 1991). This result is in agreement with the view that anxiety sensitivity provides a basis for anxiety responding due to the fact that Neuroticism is thought to increase conditionability, which is reflected in the lability of the autonomic nervous system (Eysenck, 1991). Also, the fact that the measures of anxiety sensitivity have reliable variance not shared with Neuroticism is supported by studies of the incremental validity of anxiety sensitivity measures (Lilienfeld, 1997, 1999; McNally, 1996). This evidence supports the view that anxiety sensitivity is a separate construct.
Little relationship was found (Lilienfeld, 1996) between measures of anxiety sensitivity, other than the ASI, with the higher order dimension of Constraint, a fearfulness or behavioral inhibition dimension that is observed in the lower order traits of avoidance of harm, which measures sensitivity to danger, impulse control, and traditionalism in Telegen’s (1978/1982) conceptualization of the three-factor model (Telegen & Waller, 1994). This dimension is perceived as a tendency to have the experience of fear in the presence of cues for danger. Although individuals with high anxiety sensitivity might overreact to cues for danger when it is seen as unpreventable or unavoidable, these results provide evidence that people with high anxiety sensitivity might not be overly afraid of threat which is viewed as being in the future (Lilienfeld, 1999). On the other hand, it could also be stated that anxiety sensitivity appears to be involved with threat which is viewed as being in the future (e.g., I might suffocate; I might have a heart attack) as well as with avoidance behaviors. Perhaps the lack of relationship lies more in the connection of constraint with fear (i.e., in the present) and anxiety sensitivity with anxiety (i.e., in the future).

In a more recent study Van der Does et al., (2003) utilized the ASP to identify a moderately strong correlation of anxiety sensitivity with harm avoidance. This finding corroborates comparable findings with the ASI, which had been obtained in small samples and were therefore not statistically significant (Fava, Grandi, Belluardo, Savron, Raffi, Conti, & Saviotti, 1994; Savotti, Grandi, Savron, Ermentini, Bartolucci, Conti, & Fava, 1991). The fact that the previously reported correlations of harm avoidance with the ASI and the correlations with the ASP in Van der Does et al. (2003) were of the same
magnitude provides some indirect support for the position that the ASP is an alternative measure of anxiety sensitivity (Van der Does et al., 2003).

Further work is warranted on the relationship between the measures of anxiety sensitivity and the five-factor model of personality, which includes extraversion and conscientiousness dimensions and several lower order factors that are related to interpersonal functioning (Borger, Cox, Fuentes, & Ross, 1996). Interpersonal deficits (e.g., dependency), which appear to be associated with elevated anxiety sensitivity, may not only be causes, but also consequences or complications that are a result of elevated anxiety sensitivity (e.g., panic attacks, limited symptom attacks). Another possibility is that the relationship between anxiety sensitivity and interpersonal deficits is mediated by Neuroticism. Because Neuroticism is pervasive in its effect on self-concept and emotional adjustment (Watson & Clark, 1984), researchers need to take this factor under consideration when examining connections between anxiety sensitivity measures and interpersonal functioning (Lilienfeld, 1999).

A significant correlation has been found between anxiety sensitivity and Absorption, a tendency to become engrossed in sensory experiences, and thus possibly at risk for anxiety disorders (e.g., panic disorder) through hypersensitivity to interoceptive cues (Lilienfeld, 1996; Tellegen & Atkinson, 1974). Longitudinal studies should be used to investigate this possibility as well as the potential for a mediating role by Neuroticism in the relationship between anxiety sensitivity and Absorption, which is indicated by the moderate overlap between Absorption and Neuroticism (Lilienfeld, 1999; Tellegen, 1978/1982).
The data reviewed here (Lilienfeld, 1999) cannot provide information about the direction of the relationship between anxiety sensitivity and personality traits due to the fact that it is correlational (Lilienfeld, 1999). While the trait perspective (Cattell, 1950) supports the hypothesis that anxiety sensitivity and other lower order traits develop through the interaction of general higher order traits like Neuroticism with learning experiences, information, and other personality traits, it is also possible that the relationship goes in the opposite direction (Reiss, 1997). For example, anxiety sensitivity might cause Neuroticism and other higher order dimensions (Reiss, 1991; Reiss and Havercamp, 1996). New perspectives about the etiology of panic and the other anxiety disorders might be gained through efforts made to comprehend these progressions (Lilienfeld, 1999).

**Developmental trajectory of anxiety sensitivity.** No discussion about the etiology of anxiety sensitivity would be complete without discussing its development in children. It is also important, when examining the etiology of the fear of anxiety in children, to look at the work that has been completed regarding anxiety sensitivity and developmental issues. Currently, there are few existing studies that explore anxiety sensitivity in children. Fortunately, correction of this oversight has begun with a focus on whether or not a connection between anxiety sensitivity and negative emotional responses to stress-evoking tasks will be found with children, as is the case with adults (Silverman & Weems, 1999). For example, a measure was built of items concerning the fear of physical symptoms for the exploration of children’s reactions to exposure to internal and
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external stimuli (Unnewehr, Schneider, Margraf, Jenkins, & Forin, 1996). Results provided evidence that compared children having low levels of fear of the physical symptoms of anxiety, children with at least moderate levels of fear reported higher levels of anxiety in reaction to the hyperventilation task. On the State-Trait Anxiety Inventory for Children (STAIC), their scores were significantly higher immediately following completion of the task. They were also less likely to finish the task (Unnewehr et al., 1996). These findings suggest the inherent possibility that the construct of anxiety sensitivity is a predictor of the fear of anxiety in children. Elevated anxiety sensitivity may predispose children, as it does adults, to react in a negative manner to anxiety sensations (Unnewehr et al., 1996). This would also support the possibility that anxiety sensitivity exists, if not prior to, at least at the same time as the personality constructs discussed above.

Anxiety sensitivity and children: brain physiology and future areas of investigation. Recent work in developmental theory and the growing wealth of information regarding brain physiology will make it possible to enter new and exciting areas of investigation of the etiology of anxiety sensitivity. For instance it has been established that there is a greater likelihood of a higher level of physiological reactions connected to a lower threshold of reactivity in the amygdala and hypothalamus in children with a higher than normal level of behavioral inhibition (e.g., Kagan, Reznick, Gibbons, 1989; Kagan, Reznick, Snidman, 1987, 1988). Could this also develop into elevated anxiety sensitivity that is related to a lower than normal psychological tolerance
of their reactivity (Silverman & Weems, 1999)? In a similar examination of attachment theory (e.g., Bowlby, 1973), children may be vulnerable to anxiety and other problems when they are insecurely attached (Manassis & Bradley, 1994). It is conceivable that the experience of anxiety symptoms is effected by the insecure nature of these children's cognitive response styles and is played out in the form of elevated anxiety sensitivity (Silverman & Weems, 1999).

Heritable Components of Anxiety Sensitivity and Panic Disorder

Apparently there is a solidly heritable element in panic disorder, which has been supported decisively in twin studies (Kendler, Neale, Kessler, Heath, & Eaves, 1993; Perna, Caldirola, Aranncio & Bellodi, 1997; Skre, Onstad, Torgersen, Lygren, & Kringlen, 1993; Torgersen, 1983). Anxiety experienced in response to carbon dioxide inhalation is a tendency that runs in families (Perna, Bertani, Caldirola, & Bellodi, 1996; Perna, Cocci, Bertani, Arancio, & Bellodi, 1995). This supports the view that a modification in brainstem sensitivity to carbon dioxide, a hard-wired biological abnormality, is an inherited element in panic disorder. This view would label anxiety sensitivity as a physiological risk factor. On the other hand, the tendency to believe that the bodily sensations related to this challenge are physically threatening or dangerous (i.e., anxiety sensitivity) might be the factor that is inherited (Stein & Rapee, 1999).

As mentioned earlier, the relationship of anxiety sensitivity as a subcomponent of neuroticism or negative affectivity, the higher order factor, has been well-documented in research using measures of anxiety sensitivity (Lilienfeld, 1996; Rapee & Medoro, 1994).
In addition, a general tendency toward emotionality, sometimes referred to as the general neurotic syndrome (Andrews, 1996), has been well-documented as being inherited in anxiety disorders. The notion that a large part of the variance in anxiety sensitivity is probably genetically determined is therefore a logical assumption (Stein & Rapee, 1999).

It is likely that anxiety sensitivity, also a subcomponent of trait anxiety (Lilienfeld, 1996), is mediated by the same factors (i.e., genetic vulnerability, family factors, socialization experiences, and specific life events) that are being investigated regarding the origins of anxiety disorders (Hudson & Rapee, in press; Rapee, 1997). Possibly the element of anxiety sensitivity, which is linked to experience, is the trigger that decides which individuals who have inherited the disorder will actually experience the symptoms. The inheritable portion of anxiety sensitivity is the biological guide which is required for the development of panic disorder. This theoretical conceptualization could provide a framework for both biology and psychology in the etiology of panic disorder (Stein & Rapee, 1999).

Environmental Contributions to Elevated Anxiety Sensitivity

This is a fascinating area for current and future exploration. What triggers the onset of anxiety sensitivity, if it is established to be genetically based? In addition to preventive treatment interventions for the individual, will environmental factors be identified which can also be addressed as part of treatment? The following studies illustrate further the importance of thorough and accurate measures of the anxiety
sensitivity construct which have been developed through a thorough understanding of the underlying domains. Better measures will provide a more accurate presentation of the individual's beliefs which can be investigated for prevention and intervention.

**Parental reinforcement.** A sample of 551 university students participated in the extension of an earlier study on childhood learning experiences and panic attacks (Ehlers, 1993). The ASI was used for the assessment of anxiety sensitivity levels and the Panic Attack Questionnaire-Revised served as the measure for the assessment of panic history. The learning experiences of high-anxiety sensitivity students were found to be related to parental reinforcement of sick-role behavior involving somatic symptoms in general. Also, high-anxiety sensitivity students had received more special attention and/or directions from parents to take special care of themselves. This was in contrast to the expectation that the learning experiences of high-anxiety sensitivity students would be specific to anxiety symptoms (Watt et al., 1998). The notion that elevated levels of anxiety sensitivity may be the result of learning to catastrophize about bodily symptoms in general, not anxiety-related symptoms specifically, is suggested by this outcome (Watt et al., 1998). On the other hand, those who reported the experience of panic attacks reported more often that their learning experiences involved modeling and parental reinforcement that was specifically related to anxiety-related symptoms (Watt et al., 1998).

In results that are consistent with these and other retrospective studies (e.g., Watt & Stewart, 2000), initial empirical support for a partial mediation effect of anxiety
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sensitivity in the relationship between childhood learning and panic attacks in young adults has been provided (Stewart, Taylor, Jang, Cox, Watt, Fedoroff, & Borger, 2001). In the assessment of the learning experiences of 478 university students, structural equation modeling was used to find that: (1) learning history for arousal-reactive somatic symptoms (e.g., nausea, racing heart, shortness of breath, and dizziness) directly influenced both anxiety sensitivity levels and panic frequency, (2) anxiety sensitivity directly influenced panic frequency, and (3) learning history for arousal-non-reactive symptoms (e.g., colds, aches and pains, and rashes) directly influenced anxiety sensitivity, but did not directly influence panic frequency. This last finding lends further support to the concept that the source of elevated anxiety sensitivity may be found in learning to catastrophize about somatic symptoms in general rather than about anxiety-related symptoms in particular (Watt & Stewart, 2000; Watt et al., 1998).

Parental uncontrolled behavior. An additional finding is the prediction of ASI scores through the frequency of exposure to parental uncontrolled behavior due to drunkenness and/or anger, as well as the report by individuals with elevated anxiety sensitivity of significantly more episodes of exposure (Watt et al., 1998). Initial evidence has also been provided that anxiety sensitivity levels in young adulthood were related to retrospectively reported childhood exposure to parental problem-drinking behaviors (MacPherson, Stewart, & McWilliams, 2001). This finding adds to the growing literature suggesting that acquired anxiety sensitivity may play a modest mediating role in explaining relationships between childhood learning experiences and anxiety-related
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Symptoms in adulthood (e.g., MacPherson et al., 2001; Stewart et al., 2001; Watt & Stewart, 2000). In future research anxiety sensitivity levels and family history of alcohol use should be explored due to the fact that there is evidence for a connection between elevated anxiety sensitivity and alcohol use in young adults (Stewart, 1996). It is also possible that parental alcohol use was linked with the parental experience of anxiety symptoms and anxiety sensitivity. This has not been explored (MacPherson et al., 2001).

Chronic illness. And finally, in Watt et al. (1998), no difference was found between the panic groups and the anxiety sensitivity groups relative to the number of household members with chronic illness. Neither did this factor contribute significantly to the prediction of ASI scores. Given that previous findings indicate that chronically ill family members and the development of anxiety do have a positive relationship (Bianchi, 1971; Ehlers, 1993), this factor should be explored further in future research as to the cause of the difference (Watt et al., 1998).

Learning vs genetics. In Watt et al. (1998) only 11-12% of the variance in ASI scores was accounted for by learning experiences, which suggests that anxiety sensitivity is largely innate. On the other hand, both the genetic and environmental contributions to anxiety sensitivity were the focus in a twin study (Stein, Jang, and Livesley, 1999). In this study it was found that 45% of the variance in anxiety sensitivity levels was accounted for by a genetic component and over half of the variance in anxiety sensitivity levels was the result of environmental factors. Certain fears in children can be predicted
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at set ages such as separation anxiety in early childhood and fears of social evaluation in adolescence (Ollendick & King, 1991). A predictable onset for anxiety sensitivity would strongly support a genetic basis and future research could compare scores on measures of anxiety sensitivity of parents and children or twin pairs (Lilienfeld et al., 1996; Plomin, 1990; Watt et al., 1998).

Future research could also include an interview with high, medium and low anxiety sensitivity subjects, which could support the generation of hypotheses regarding issues in development such as: (a) various types of learning experiences (e.g., interoceptive conditioning, death of a significant other, trauma exposure), (b) predisposing personality factors (e.g., trait anxiety; Lilienfeld et al., 1996), (c) repeated benign exposures to anxiety-related bodily symptoms which might ‘inoculate’ a child against the development of anxiety sensitivity (i.e., ‘latent inhibition’), (d) a possible synergistic interaction of learning history factors, rather than a simple additive process, in the development of anxiety sensitivity (e.g., Ollendick & King, 1991), and (e) maintenance supported by habituation to anxiety-related sensations through repeated, non-traumatic exposure (Menzies & Clarke, 1995; Watt et al., 1998). Finally, the future exploration of more specific direct and indirect routes of learning, such as the separate influences of positive and negative reinforcement vs. punishment, would be valuable contributions to the study of the etiology of anxiety sensitivity. A child’s anxiety-related behaviors and complaints might be decreased by parental punishment, but it may also increase his or her anxiety due to learned concerns about the consequences of exhibiting anxiety symptoms (Stewart et al., 1997).
Prospective Evaluations of Anxiety Sensitivity

This type of investigation is quite valuable for the identification of the risk factors for anxiety sensitivity and for the maintenance of pathology over time, as well as for treatment maintenance and relapse. It also provides information regarding the level of anxiety sensitivity involvement and the involvement of other factors in the development of anxiety pathology. The four published longitudinal studies which have evaluated the role of anxiety sensitivity in the onset of anxiety symptoms in a nonclinical sample (Ehlers, 1995; Harrington, Schmidt, & Telch, 1996; Maller & Reiss, 1992; Schmidt et al., 1997) have provided consistent support for anxiety sensitivity as a risk factor for anxiety pathology and in particular for panic attacks. There is also evidence that anxiety sensitivity is a maintenance factor. This is stated in the two published longitudinal studies which evaluated subjects with clinical or subclinical anxiety symptoms (Bruce et al., 1995; Ehlers, 1995).

With the exception of the previously mentioned Schmidt et al. (1997) study involving more than 1,000 US Air Force Academy cadets, the low base rates for anxiety disorders in the general population, which according to the Epidemiological Catchment Area Study (Eaton & Keyl, 1990; Keyl & Eaton, 1990) are 2% for panic disorder, has meant that the studies are lacking in the power which is needed to detect low base rate phenomena (Schmidt, 1999). All of the other studies combined have only 15 cases that presented new panic attacks. Nonetheless, while noting the low rate of panic attacks, it is important to also note that a relationship between anxiety sensitivity and panic was found in the majority of these studies and a considerable number of first panic attacks were
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predicted by elevated anxiety sensitivity in the US Air Force Academy study (Schmidt, 1999).

On the other hand, these studies do not fully verify a relationship between anxiety sensitivity and the onset of anxiety at the clinical level. For example, it is difficult to decide whether or not high anxiety sensitivity existed prior to the development of anxiety disorders in the Maller and Reiss study (1992). Although they view this study as providing evidence for a solid relationship between anxiety sensitivity and anxiety disorders, there was no diagnostic evaluation at the start of the study (Schmidt, 1999). The Harrington et al. (1996) study did not find a relationship, but there was assessment for psychiatric history (Schmidt, 1999).

The Air Force data probably provides the best evidence that high anxiety sensitivity plays a part in the sequence of change that leads to anxiety syndromes. During five weeks of intense basic training a small number of cadets reported the experience of symptoms at clinical level, including multiple panic attacks, panic-related worry, high levels of anxiety symptoms, and anxiety-related impairment (Schmidt, 1999; Schmidt et al., 1997). During this period, in which individuals who were susceptible to anxiety symptoms were assessed while experiencing high levels of stress, ample evidence was provided for the diathesis-stress model (Schmidt, 1999).

Other variables are involved in the origin and development of panic. Gender, age, negative life events, and medical conditions are factors that make contributions to the development of anxiety, according to longitudinal epidemiological reports (Breslau & Davis, 1993; Keyl & Eaton, 1990; Eaton & Keyl, 1990; Schmidt & Telch, 1997).
Cognitive factors such as predictability, perceived control, and perceived safety, have been implicated in other studies due to their effect on the level of anxious responding in both clinical (Carter, Hollon, Carson, & Shelton, 1995; Rapee, Mattick, & Murrell, 1986; Sanderson, Rapee, & Barlow, 1989) and nonclinical populations (Schmidt, 1999; Schmidt & Lerew, 2002; Schmidt & Telch, 1994; Telch, Silverman, & Schmidt, 1996).

Because there is now evidence that anxiety sensitivity is involved in the origin and development of panic, there is also evidence that anxiety sensitivity is a risk factor, and not simply a byproduct of panic development. The next step is the use of cognitive behavioral therapy as a primary prevention intervention, since it has been shown to reduce anxiety sensitivity significantly (Bruce et al., 1995; Schmidt & Telch, 1997). Such an intervention can be used to act in advance, preventing anxiety and panic reactions in people who are at risk (Schmidt, 1999). The goals for prevention, treatment, and relapse prevention of the symptoms of anxiety, including an understanding of how the mechanics of anxiety sensitivity fit into the attainment of these goals, will be discussed in the next section. This understanding is necessary in order to ensure that all possible benefits to be obtained from this study will be utilized.
Possible Benefits to Be Derived from Promotion of the Understanding of the Mechanics of Anxiety Sensitivity for Prevention, Treatment, and Relapse Prevention of the Symptoms of Anxiety

This section provides an overview of the work that is focused on anxiety disorders and anxiety sensitivity. The current status of assessment, treatment, treatment maintenance, and research are briefly reviewed. Also reviewed are the gains to be made by current and future efforts in understanding the mechanics of anxiety sensitivity, its practical applications in these areas, as well as its prevention. It has been reported that anxiety disorders incur the largest direct and indirect cost to the national economy of all psychological disorders in the United States, totaling $46.6 billion, or 31.5% of the total cost of $147.8 billion for all psychological disorders in 1990. Particularly high were indirect economic costs such as productivity. Anxiety disorders claimed $35.4 billion, or 47.3% of the total of $74.9 billion for all indirect costs caused by mental illnesses (Dupont, 1993). If the etiology of anxiety sensitivity can be understood, the provision of interventions that will prevent the development of anxiety disorders can begin.

Prevention issues. In recent years significant growth has occurred in the treatment efficacy of anxiety disorders. Eighty percent of patients have been reported to be free of panic symptoms at the completion of treatment in clinics that specialize in anxiety disorder treatment in Europe and North America. This success has been maintained at follow-up evaluations of up to 2 years (Margraf, Barlow, Clark, & Telch,
1993). However, there is only limited research in progress, in which the purpose is the identification and control of onset factors, although prevention has often been part of the discussion of anxiety disorders (Norton, Asmundson, Cox, & Norton, 2000). Until most anxiety disorders are prevented, and the rest are effectively treated, the struggle will not have been completed. The most significant areas for research will be growth in understanding all of the anxiety disorders through clarification of the mechanisms which bring about the appearance of anxiety responses and anxiety disorders in individuals affected by them; this, of course, will include the protective factors which keep others who are exposed to the same experiences or environments from developing them (Heimberg, 2000).

New conceptual models of anxiety, depression, and related states, which are appearing with the use of sophisticated modeling techniques, far exceed the present perceptions of anxiety and depression. These new models ignore the present systems of categorization and bring about a much more accurate identification and understanding of the basic traits that cause the individual to be vulnerable to the development of these disorders (Barlow, 2000). From this basic research, a new group of interventions aimed more directly at underlying vulnerabilities and emphasizing the prevention of emotional disorders will be developed. The identification of children at risk for anxiety disorders is likely to be the first action. Following this, researchers will seek to understand more clearly the psychosocial factors that result in the onset of specific disorders, to rate the results of preventive interventions, and to perform projective studies of these children,
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which are based on new but strong findings on the chronicity of anxiety disorders (Barlow, 2000).

Recent research on behavioral inhibition (e.g., Kagan et al., 1989), attachment (e.g., Manassis and Bradley, 1994), family processes (Dadds, Barrett, Rapee, & Ryan, 1996), cognitive-behavioral treatments (e.g., Kendall, 1994; Silverman & Kurtines, 1996) and prevention programs (Dadds, Spence, Holland, Barrett, & Laurens, 1997) provides evidence that researchers have recently turned their attention toward anxiety in children. Their goals have been the development of an understanding of the etiology and maintenance, as well as the provision of prevention and treatment of anxiety disorders in this segment of the population (Silverman & Weems, 1999). Turning their attention not only toward anxiety sensitivity as a construct alone, but also as it is related to these areas of study, will bring about a greater increase in knowledge in these areas (Silverman & Weems, 1999). In fact, it has been discovered in a growing body of evidence that there of seems not only to be a number of similarities, but also a number differences in anxiety as it appears in children and adults (Silverman, 1993). Elevated anxiety sensitivity, as well as a great deal of what is apparent in anxious adults is also seen in anxious children. Possibly there are other similarities (e.g., anxiety sensitivity as a predictor of anxiety and panic disorder in youth) that will be of use, for example, in designing prevention programs (Silverman & Weems, 1999). Primary prevention would be greatly enhanced if it were found that high anxiety sensitivity is a risk factor for anxiety disorders. After screening children or adolescents, (e.g., in school settings) to find those with elevated anxiety sensitivity, anxiety sensitivity could be lowered by methods used in cognitive-
behavior therapy for panic disorder, which is designed specifically to treat the fear of the bodily sensations of anxiety. In theory, the risk for anxiety disorders, and possibly other disorders, would be decreased (Taylor, Rabian, & Fedoroff, 1999).

Findings very much like those found with the ASI have been found in recent studies using the Childhood Anxiety Sensitivity Index, an instrument which has strong initial support for reliability and validity (CASI; Silverman, Fleisig, Rabian, & Peterson, 1991). These findings have supported differential diagnosis and panic prediction, including children’s internal catastrophic attributions. They have also supported the appearance of a relationship between anxiety sensitivity, depression, and other negative cognitive states such as worry (Silverman & Weems, 1999). At the time of their report in Taylor (1999), Silverman & Weems wrote that these results had not been put into a useful theoretical framework. Replication and extension are also needed, due to the significance that these findings have for the prevention of anxiety disorders (Silverman & Weems, 1999).

In progress at the time of their report in Taylor (1999) was a study (Silverman & Weems, 1999) involving a follow-up on 273 children who completed the CASI in 1991. These children were being tracked when they were adolescents, 8 years after the initial assessment for the prediction of panic and other anxiety disorders. One of the next steps will be the design of prevention intervention programs if these results show that elevated CASI scores in children are indicators of a likely history of panic attacks. This is in contrast to children without elevated CASI scores who are not likely to have such a history (Silverman & Kurtines, 1996). Of course, without the means to provide accurate
assessment of those who are at risk for the development of anxiety disorders due to anxiety sensitivity, many individuals will not be identified and preventive interventions will not be provided.

*Research and assessment issues.* The perception of the causes of anxiety disorders needs to change so that accuracy of assessment and research is improved. In this section are proposals by several investigators suggesting that it is time to put unidimensional concepts aside and to adopt multidimensional concepts and measures for use in research and assessment. Panic disorder, which is related to the most elevated level of anxiety sensitivity, has received the most attention, as can be seen in the following material.

Much research has based design and assessment strategies on a unidimensional perception of panic (i.e., rather than a heterogeneous condition with identifiable subtypes, panic has been viewed as a single, unitary phenomenon). General improvement in results and increased predictive power could be brought about by the use of a multidimensional perspective that includes congruence (Cox, 1996). As an example in the area of research, the findings on memory bias in panic disorder continue to be inconsistent (Cox, 1996; Rapee, 1994).

A possible cause stems from basing research on a unidimensional perception rather than using a multidimensional perspective and identifying the lower-order factor for anxiety sensitivity that matches the individual's fears. Some panic disorder patients may connect words such as coronary and stroke with danger, producing a very strong effect. For a sample of patients more focused on dizziness or depersonalization, these
words would have little effect due to the lack of congruence. Studies could be improved by identifying trait subtypes and matching the stimulus (situation) with the trait (i.e., congruence), when performing panic provocation studies. Initial support has been obtained for this congruent interaction (Leliot & Bass, 1990). The prediction of an improvement in accuracy in panic disorder research can be tested if a multidimensional measure of anxiety sensitivity (e.g., ASP) that is psychometrically solid can be developed (e.g., when there is improved accuracy in matching the symptoms caused by a provocation task and the symptoms a person fears, precision in predicting panic attacks will improve; Cox, 1996; Cox et al., 1999).

Our comprehension of the reasons that certain bodily sensations trigger panic attacks in certain individuals will be increased by continued study of the anxiety sensitivity dimensions. Results of a recent study agree with this proposal (Cox et al., 1999). The fear of dyspnea (i.e., an aspect of anxiety sensitivity which means difficulty in breathing) caused an anxious response to a congruent trigger (i.e., carbon dioxide challenge), in a college student sample. This aspect of anxiety sensitivity was a better predictor of panic attacks than the ASI total score (McNally & Eke, 1996). In other research, it appears that ASI items that evaluate the individual’s beliefs about the catastrophic consequences of certain bodily sensations are more pertinent to the evaluation of panic disorder (Cox et al., 1999).

Finally, in assessment there are clinical implications for the hierarchical model of anxiety sensitivity, which suggests viewing the patient’s anxiety sensitivity profile on all of the first-order anxiety sensitivity factors (i.e., physical, social, and cognitive concerns;
Taylor et al., 1996; Zinbarg et al., 1997). For instance, there is evidence that patients who have social phobia as their principal diagnosis would tend to have a higher Anxiety Sensitivity-Social Concerns score than an Anxiety Sensitivity-Physical Concerns score on the ASI. (Zinbarg et al., 1997). Those who have a higher Anxiety Sensitivity-Physical Concerns score will probably have panic disorder as their first diagnosis (Taylor, 1996). Therefore, a more detailed evaluation and more confidence in a patient's principal diagnosis can be gained by checking the degree of match between his or her anxiety sensitivity profile, than by using only the total anxiety sensitivity score. Also, there is the possibility that themes in patients' concerns other than those related to their diagnoses can be identified with the use of such profiles (Zinbarg et al., 1997; Zinbarg & Schmidt, 2002). Such accuracy in assessment issues will lead to improved treatment interventions.

Treatment intervention research: children. Use of the CASI in treatment trials is a possibility, as is its potential use in prevention trials. The principal ways in which it could be used are as (a) an outcome variable, (b) a variable to prescribe specific treatment procedures, and (c) a predictor of treatment response. Recent research, which uses the CASI in each of these ways, has been promising (Silverman & Weems, 1999). In a multiple baseline study of cognitive-behavioral therapy for panic disorder with agoraphobia, the CASI was used as an outcome variable. Four adolescents, ages 13, 14, 16, and 17, were the subjects. It was found that the CASI is an accurate test for treatment-related change (Ollendick, 1995).
Some psychotherapy researchers are starting to see the significance of matching or prescribing treatment interventions to individual characteristics of patients, while they are aware that randomization of treatments across subjects is an excellent experimental method used to assess treatment efficacy (e.g., Beutler, 1991; Kazdin, 1993; Kearney & Silverman, 1990; Ost, Johansson, & Jerremalm, 1982). With the use of this alternative method, children with elevated anxiety sensitivity could be identified through scores on the CASI, perhaps in relationship with other measures. Prescriptive treatment could be given to these children, in which an intervention is assigned that targets anxiety sensitivity reduction in accordance with their scores. (Silverman & Weems, 1999). The use of the CASI in this way is supported by the results of two single case studies (Eisen & Silverman, 1993, 1998). These studies did not focus on anxiety sensitivity, but focused on certain types of response to anxiety. The children who were chosen for the prescribed treatment, which matched their symptom patterns of anxiety, experienced a better outcome. Three of the four subjects fell within normal limits and maintained this improvement at 6-month follow-up. None of the subjects who received nonprescriptive treatment experienced enough change to reduce scores to normal limits (Eisen & Silverman, 1998). The same multiple baseline design could be used for testing the usefulness of prescribing anxiety sensitivity reduction for children who experience elevated anxiety sensitivity (Silverman & Weems, 1999).

It is time now that researchers turn their attention to the identification of factors that predict the success or failure of treatment, as support mounts for the efficacy of treatments for children with anxiety disorders (e.g., Kendall, 1994). One of these factors
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might be elevated anxiety sensitivity. If research findings support this, in addition to giving the child the basic treatment package, it would suggest that anxiety sensitivity reduction be targeted also (Silverman & Weems, 1999). The possibility that the CASI can be used as a predictor and possible moderator of treatment success or failure has been explored in an introductory manner. Fifty-four children, ages 6-17, diagnosed with social phobia, overanxious disorder, or GAD were given exposure-based cognitive-behavioral group therapy in a controlled clinical trial. The results showed that children who tended to have elevated anxiety sensitivity prior to treatment did not improve, (i.e., still met DSM criteria for their presenting problem), compared with children who improved with treatment (i.e., no longer met diagnostic criteria; Silverman & Weems, 1999).

*Treatment intervention research: adults.* It has been suggested that focusing on a deeper understanding of the basic processes causing the anxiety disorders will support the most rapid growth in treatment, while it is also important to maintain the positive movement being made over the last decade in the direct study of treatment interventions. Among suggested areas that need attention are: (a) too many individuals do not respond or do not respond completely to treatments for which there is significant evidence of effectiveness and (b) too little is known about the predictors of treatment outcome (Heimberg, 2000).

At this time, new treatments have not directly resulted from the anxiety sensitivity interpretation of the fear of anxiety. However, there has been progress made in the prediction of treatment outcome. The ASI, the most widely used measure of anxiety
sensitivity, has been increasingly used as an outcome variable in treatment research on panic disorder (e.g., Telch et al., 1994), and it successfully predicts outcome (Clark, Salkovskis, Hackmann, Middleton, Anastasiades, & Gelder, 1994). For example, the only significant predictor of stable withdrawal from alprazolam among panic disorder patients was found to be the ASI in a study conducted by Bruce et al. (1995).

Scores on the ASI appear to be correlated with improvement following treatment. This is indicated by consistent results across studies that show reductions in scores following short-term therapist-directed cognitive-behavioral treatment for panic disorder (Otto and Reilly-Harrington, 1999). ASI scores for 160 treated subjects from seven studies showed a weighted average drop in scores of 14 points (Gould, Otto, and Pollack, 1995; Hazen, Walker, & Eldridge, 1996; Hegel, Ravaris, & Ahles, 1994; McNally & Lorenz, 1987; Penava, Otto, Maki, & Pollack (in press); Shear, Pilkonis, Cloitre, & Leon, 1994; Telch, Lucas, Schmidt, Hanna, Jaimez, & Lucas, 1993). In two longitudinal studies, remission during follow-up of panic attacks, panic-related anxiety, and phobic avoidance was predicted by a reduction of anxiety sensitivity to nonclinical range for anxiety-disordered individuals who received a complete cognitive-behavioral treatment protocol (Schmidt et al., 1997) and panic-free withdrawal of benzodiazepines (Bruce et al., 1995).

These results have given rise to speculation about the variables that may be linked to score reduction. In a few studies it does not appear to matter what treatment mode is used, or whether or not it focuses on fears of anxiety sensations, as does cognitive-behavioral treatment, which consists of cognitive restructuring and exposure
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interventions. When treatment is effective it appears to cause a reduction in ASI scores (Otto, Pollack, Sachs, & Rosenbaum 1991; Clark et al., 1994; Otto & Reilly-Harrington, 1999). Possibly the reduction of anxiety sensitivity through treatment is accomplished through a means which is not connected to the origin of these fears (Otto & Reilly-Harrington, 1999). Perhaps there is the need to consider other possible mediating variables such as the development of the individual’s belief that he or she has sense of control over outcome. There is some evidence that perceived control interacts with anxiety sensitivity to predict panic, and there is also evidence that perceived predictability interacts with anxiety sensitivity to predict anxiety (Schmidt & Lerew, 2002).

It is also possible that certain dimensions of anxiety sensitivity may be reduced by specific exposure exercises. General CBT procedures for panic disorder may be the most efficient means of treatment for those individuals with generally elevated anxiety sensitivity (i.e., elevated scores on multiple dimensions of anxiety sensitivity). Treatment that is matched to the symptoms of the individual may be best for others. This type of treatment involves the use of certain procedures to reduce elevations on specific dimensions of anxiety sensitivity (e.g., relying mainly on methods that induce cognitive anxiety symptoms in people who only have an extreme fear of cognitive dyscontrol; Taylor, Rabian, & Fedoroff, 1999).

There are also significant connections between relapse prevention and anxiety sensitivity theory and research. As effective as cognitive behavioral therapy and pharmacotherapy are in treating panic disorder, sometimes those who respond to
treatment relapse. Given the rate of success for measures of the fear of anxiety sensations in predicting long-term effectiveness of treatment, an important and necessary final stage appears to be the focus of attention on elevated scores in individuals who have finished acute treatment (e.g. Clark et al., 1994; Ehlers, 1995; Pollack, Otto, Sabatino, Majcher, Worthington, McArdele, and Rosenbaum, 1996; Otto & Reilly-Harrington, 1999). Assessment for anxiety sensitivity level at the end of treatment, with the provision of anxiety sensitivity-focused additional treatment for people with elevated anxiety sensitivity, has been suggested in these cases (Taylor, Rabian, & Fedoroff, 1999). Focus on anxiety sensitivity as a risk factor for panic disorder development also appears to be worthwhile. One study in which anxiety sensitivity was reduced in a nonclinical sample with a brief (three-session) cognitive-behavioral technique (Harrington, Telch, Abplanalp, & Hamilton, 1995) has provided the first evidence of effectiveness in this area (Otto & Reilly-Harrington, 1999).

Efforts to make comparisons between the effects of psychosocial treatment and pharmacotherapy on anxiety sensitivity have not been easy due to the fact that the ASI as a measure is not usually part of the studies which have evaluated the efficacy of pharmacotherapy in the treatment of panic disorder. Nonetheless, while there appear to be significant reductions in anxiety sensitivity with medications alone, patients with remaining symptoms of anxiety continue to have elevated ASI scores (Otto & Reilly-Harrington, 1999). There is evidence to support the use of cognitive behavioral therapy as an effective technique to reduce further the elevated ASI scores in these patients, and aid medication discontinuation (e.g., Bruce et al., 1995; Hegel et al., 1994).
The following section, while not inclusive, supports an understanding of the current status of research related to anxiety sensitivity. This includes development of an evolving conceptualization made possible by new and increasingly sophisticated research designs and statistical methods. Presented here is representative evidence for the dimensionality, hierarchical structure, and symptom specificity of anxiety sensitivity and its current measures.

Anxiety Sensitivity and Depression: Symptom Specificity and the Tripartite Model

It is of interest to discuss and understand the current status of the theoretical conceptualization and related research regarding anxiety and depression, and specifically anxiety sensitivity as a construct. While a number of existing studies provide evidence that anxiety sensitivity is a risk factor for anxiety, a relationship for anxiety sensitivity with depression has also been found (Otto, Pollack, Fava, Uccello, & Rosenbaum, 1995; Taylor, Koch, Woody, & McLean, 1996; Van der Does et al., 2003). Patients with comorbid major depression and panic disorder have been found to have higher ASI scores (M = 40.3, SD = 11.3) than panic disorder patients without depression (M = 31.4, SD = 9.6; Taylor et al., 1996). There is also increasing support for anxiety sensitivity, or some factor of anxiety sensitivity such as phrenophobia (i.e., the fear of the loss of
cognitive control), as a risk factor for depression (Schmidt, Lerew, & Jackson, 1997; Zinbarg, Brown, Barlow, & Rapee, 2001).

Whether or not a variable is a vulnerability factor can be decided, as much as possible, by three criteria which have been provided for non-experimental psychopathology research (Garber & Hollon, 1991): (a) covariance should exist between a vulnerability factor and an outcome, (b) the outcome should be preceded by the vulnerability factor in time, and (c) a third variable should not better explain the correlation between the outcome and the vulnerability factor (i.e., the relationship should be non-spurious).

There is evidence that the lower-order fear of the loss of cognitive control is the factor that may, in large part, provide the relationship (Taylor et al., 1996; Cox, Enns, Freeman, & Walker, 2001). Problems with concentration and decision-making are typically included in depression, and this may support the fear of the loss of cognitive control as a risk factor for anxiety. People who have specific fears of these symptoms (i.e., phrenophobics) would become more anxious when experiencing these conditions (Taylor et al. 1996; Cox et al., 2001). The fear of the loss of cognitive control is also conceptually consistent with anxiety sensitivity. This is due to the fact that symptoms such as poor concentration, and symptoms that are thought to be related, such as derealization and depersonalization, could lead to specific fears (e.g., fear of mental incompetence or irreversible depression; Cox et al., 2001). It is also plausible that the same attributional style associated with anxiety sensitivity is related to cognitive distortions, such as catastrophizing or likelihood overestimation, that lead to depression.
Anxiety sensitivity was not found to be predictive of depression. Possibly this is due to the well-established overlap of anxiety and depression ($r$ range: 0.25-0.60) and the stringent requirement of symptom specificity. It is possible to argue, nevertheless, that there are both unique and overlapping features in depression and anxiety, as has been put forth in the tripartite model examined by Clark and Watson (1991) and Brown, Barlow, and Chorpita (1998). This model, supported by a significant body of research, contains an anxiety-specific factor (i.e., physiological hyperarousal), as well as a factor that is unique to depression (i.e., anhedonia). This model provided the background for the covariance strategy used in the study conducted by Brown et al. (1998) that identified the
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common feature for anxiety and depression (i.e., negative affect) and supported a more accurate analysis of the features which are unique to each (Schmidt et al., 1998).

It is possible, when viewing the tripartite model, to see a link between anxiety sensitivity and hyperarousal, while anxiety sensitivity and anhedonia do not appear to have the specific relationship of risk factor and symptoms. This coincides with the view of elevated anxiety sensitivity as the predictive factor for panic attacks and panic disorder, which are perhaps the most typical examples of hyperarousal (Schmidt et al., 1998). This does not mean that some of the related symptoms of depression are not increased by anxiety sensitivity in the presence of anxiety symptoms. Again, when viewing the tripartite model, it might be possible to see that anxiety sensitivity increases negative affect at non-specific levels, while not directly having an influence on anhedonia (Schmidt et al., 1998). Also, as mentioned earlier in Taylor et al. (1996), an analysis of one of the first-order factors of anxiety sensitivity linked it with depression. The anxiety sensitivity factor, the fear of the loss of cognitive control, was found to be linked with both the unique features of anxiety and the unique features of depression (Schmidt et al. 1998; Zinbarg et al., 2001).

Anxiety Sensitivity: Dimensional or Categorical

Testing variables of interest for dimensionality is rarely done, but this factor can have great importance in the areas of theory and assessment. Work on this issue has begun (Taylor, Cox, Freeman, McNally, Stewart, & Swinson, 1999), and is using
recently formulated taxometric methods (Waller & Meehl, 1998). Whether a set of data serves as an example of a single dimensional variable or two potential, well-documented categories, can be determined with these statistical procedures. These procedures were used with a data set of ASI scores from 546 individuals with panic disorder (ASI mean = 35.3, SD = 12.0) and a sample of 546 university students used as the nonclinical controls (ASI mean = 19.3, SD = 9.8; Taylor et al., 1999). If anxiety sensitivity is categorical (taxonic), high anxiety sensitivity people will have a higher risk for psychopathology, especially panic attacks, and low anxiety sensitivity people will be at low risk. The taxon will be most of the people in the panic disorder group and the complement will be members of the control group (Taylor et al. 1999).

Factor analysis of the ASI has provided evidence that anxiety sensitivity is made up of at least three lower order factors that load on a single higher order factor in a hierarchical structure (Zinbarg et al., 1999). In order to conduct analyses on the lower order factors, more items are needed than are contained in the ASI. Therefore, this study had to be confined to analyses of the higher order factor (Taylor et al. 1999). All eight ASI items, which were used to form four miniscales in order to meet the criteria for this type of statistical procedure, had salient loadings on the higher order anxiety sensitivity factor in this study (Taylor et al. 1999). The same was found to be true in two other studies (Stewart et al., 1997; Zinbarg et al., 1997).

Three of six covariance analyses, which were based on the four miniscales, provide clear evidence that anxiety sensitivity is dimensional. The other three suggest that anxiety sensitivity is either dimensional, or that there is a category (taxon) present
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with a low base rate (i.e., \( \leq 10\% \)). If less than or equal to 10\% of the combined sample of panickers and students (i.e., 110 individuals) consists of a high anxiety sensitivity category (taxon), such a taxon could be present (Meehl & Yonce, 1996). If it could be established that such a low base rate taxon is present, then anxiety sensitivity would be categorical rather than dimensional.

However, it is suggested by the initial results of this study that anxiety sensitivity is dimensional and that it is not taxonic (categorical). First, three out of six covariance analyses clearly suggest that anxiety sensitivity is dimensional. Second, the majority of the sample of panic disordered individuals would not be included in the taxon, since a low base-rate taxon would be less than or equal to 10\% of the combined sample, while half of the sample was diagnosed with panic disorder. If there is a taxon, it is supposed to represent the group that is at high risk for psychopathology (Taylor et al. 1999).

If the base rate were 10\%, the taxon group (\( n = 110 \)) would have a mean ASI score of 51.8 (a range of 43-63). It would be made up of 106 individuals with panic disorder and 4 controls. This taxon would contain only 19\% of the people with panic disorder. The rest of the people who regularly experience panic attacks, along with almost all of the students, would be part of the complement group. Therefore, people who have a high risk factor for psychopathology are not identified, and little psychological sense is made by a low base-rate taxon (Taylor et al. 1999). It follows, then, that the tentative conclusion that can be drawn from the initial part of this study is that anxiety sensitivity, as a higher order construct, is dimensional (Taylor et al. 1999).
Evidence for the Hierarchical Structure and Factor Saturation of the ASI

Evaluating the evidence regarding the validity of the anxiety sensitivity construct and its most prominent measure, the ASI, is important due to the significant part played by anxiety sensitivity in the current conceptualizations of panic disorder and the other anxiety disorders (Zinbarg et al., 1999). Distinct factors may be linked to distinct mechanisms (Cattell, 1978) in, for instance, the etiology of anxiety reactions. One way in which to address these issues is factor analysis (Taylor & Cox, 1998). Currently, the factor structure of the ASI has been analyzed in 12 published studies and two or more unpublished factor-analytic studies with inconsistent results. As noted in earlier reviews of these studies, there has been support for a unidimensional structure and multidimensional structures that include up to four factors (Lilienfeld et al., 1993; McNally, 1996; Taylor, 1995a, 1995b).

How can a hierarchical structure hypothesis account for these contradictory results? Apparently differences in the number of factors extracted across studies are considered to be reasonable (a) when a hierarchical structure exists, and (b) when no differentiation between higher and lower level factors is directing the decision by investigators on the optimal number of factors (Goldberg & Dig man, 1994). This appears to have been the case due to the fact that the hypothesis was not stated until fairly recently (i.e., Lilienfeld et al. 1993). This hypothesis proposes a model that contains three correlated first-order factors, which are aspects of anxiety sensitivity that load on a single, higher order factor. This higher order factor is thought to be trait anxiety, which
in turn, is thought to be an aspect of negative affectivity (neuroticism; Cox, 1996; Zinbarg et al., 1997).

Therefore, many of the contradictory results in the currently existing body of research can be integrated if the hierarchical model is valid (Zinbarg et al., 1997). It also appears that those who argued for a multifactor solution were interested in the lower level of the hierarchy (e.g., Telch, Shermis, & Lucas, 1989; Wardle, Ahmad & Hayward, 1990), while those who argued for a single-factor solution were interested in the higher level (e.g., Reiss et al., 1986; Taylor et al., 1991; Taylor et al., 1992). Exploratory and confirmatory factor-analytic procedures have been used as strategies in research that has explored the hierarchical aspect of the factor structure of the ASI. The methods used by Peterson and Heilbronner (1987), and those used in several more recent studies, have been appropriate for this type of testing. An oblique rotation was used by Peterson and Heilbronner (1987), whose results showed moderate correlations among their first-order factors. It was also used by Taylor, Koch et al., (1996), whose results showed weak to moderate correlations for their three first-order factors (Zinbarg et al., 1999).

There is evidence for a possible second-order factor (i.e., anxiety sensitivity) in the first-order factor correlations in each of these studies. In addition to reporting a moderate correlation among their three first-order factors, Stewart et al., (1997) found evidence of a single second-order factor when they completed a higher order factor analysis of the correlations among their first-order factors. Clearly, this second order factor was a general factor because, in relating the items directly to the second order factor, all 16 items of the ASI were found to have salient loadings on this factor (Zinbarg
et al., 1999). Both panic-disorder patients and college students served as samples for several confirmatory factor analyses. In these analyses a unidimensional model and several four-factor models, which were based on the results of Peterson and Heilbronner (1987), Telch et al. (1989), and Wardel et al. (1990), were compared (Cox, Parker et al., 1996). After oblique and orthogonal solutions were examined with the four-factor models, it was discovered that neither the orthogonal four-factor models nor the unidimensional model fit patient or student data. On the other hand, the oblique four-factor models were found to provide acceptable levels of fit. They were also significantly better fits for both the patient and student sample data than the unidimensional models (Cox et al. 1996; Zinbarg et al., 1999).

These results provide evidence for a hierarchical factor structure for the ASI made up of several first-order factors, in addition to at least one second-order factor. This is due to the fact that oblique factors suggest the existence of higher order factors (Gorsuch, 1983; Zinbarg et al., 1999). A factor analysis of the correlations among the factors for the oblique four-factor model in the Cox et al. (1996) study suggests a second-order factor in both patient and student samples. The smallest of the four loadings of the first-order factors on the second-order factor for the patient sample was .60, and .52 for the student sample, indicating a very strong second-order factor (Zinbarg et al., 1999). Any estimate of general factor saturation above .50 is considered to be evidence that the total score is mainly a measure of a single construct (i.e., anxiety sensitivity; Zinbarg et al., 1997).
Zinbarg et al. (1997) examined a hierarchical model that was made of three orthogonal factors labeled as Anxiety Sensitivity-Physical Concerns, Anxiety Sensitivity-Mental Incapacitation Concerns, and Anxiety Sensitivity-Social Concerns in which each loaded on a single second-order factor rather than having the limitation of being orthogonal. Also examined were a unidimensional model and a model with the same three orthogonal factors. The hierarchical model afforded a fit that was significantly better than the others, when applied to the data of their anxiety disordered outpatient sample. Evidence was provided by a Schmid-Leiman transformation on the factor pattern matrix that the second order factor is a general factor. This consisted of the fact that 15 of the 16 ASI items had a loading of .30 or greater on this factor (Zinbarg et al., 1997). Very similar results were found in a study conducted by Stewart et al. (1997).

Based on these results, the following determinations have been made (Zinbarg et al., 1999): (a) three partially discrete first-order factors and one general, second-order factor make up the hierarchical factor structure of the ASI, (b) Anxiety Sensitivity-Physical Concerns, Anxiety Sensitivity-Mental Incapacitation Concerns, and Anxiety Sensitivity-Social Concerns are factor names, which are in agreement with items that tap for corresponding concerns on the ASI (Zinbarg et al., 1997), and (c) across populations used most often for evaluation of the ASI, which are anxiety-disordered outpatients and college students, results suggest that this hierarchical structure is quite similar (Zinbarg et al., 1999). Evidence for a higher order general factor does not justify, psychometrically, the use of a total score from a set of items such as the ASI. Before this is appropriate, the scale’s general factor saturation, or the proportion of variance in the total scores
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accounted for by the general factor, must be examined (Cronbach, 1951; Revelle, 1979; Zinbarg et al., 1997). Preliminary findings from this type of analysis of the ASI resulted in the estimation that the second-order, general factor explains approximately 60% of the variance in total scores (Zinbarg et al., 1997). Use of the ASI total score as a representative measure of the anxiety sensitivity construct, therefore, appears to be justified, but replication of these findings is needed (Zinbarg et al., 1999).

Regarding the methodological implications of the hierarchical model of anxiety sensitivity, it is important to be aware that most studies that have explored this area have used ASI total scores and not the subscales which are representative of the first-order anxiety sensitivity factors, with the exceptions of Stewart et al. (1997), Taylor et al. (1996), and Zinbarg et al. (1997). Whether the significant relationships between ASI total scores and several criteria that have been revealed in these studies are caused by the second-order, general anxiety sensitivity factor or one or more of the first-order factors is not clear (Zinbarg et al., 1999).

In addition, due to the fact that the subscales, which are linked to the lower order anxiety sensitivity factors, were not included in these studies, it is not possible to determine whether incremental validity of the second-order general factor, or one or more of the first-order anxiety sensitivity factors has been supported. It is impossible to tell what factor is predicting various criteria, even after controlling for trait anxiety (Lilienfeld, 1996; Lilienfeld et al., 1993; McNally, 1989, 1996; Reiss, 1991). Reanalyses of these studies using the ASI can be performed which calculate and analyze the subscales that are related to the first-order anxiety sensitivity factors. This process will
aid in deciding which level of the anxiety sensitivity hierarchy plays a part in producing pathology (Zinbarg et al., 1999).

In fact, this work has begun (Zinbarg et al., 2001) with the following findings based on a previous study (Rapee, Brown, Antony, & Barlow, 1992) that assessed fear responses to hyperventilation and 5.5% carbon dioxide challenges: (1) the strongest positive linear relation with panic-related phenomena among the three underlying factors of the ASI is shown by Anxiety Sensitivity-Physical Concerns and (2) the strongest positive linear relation with depressed mood is shown by Anxiety Sensitivity-Mental Incapacitation. Of course, it must be kept in mind that the challenges used were most congruent with the physical concerns factor of anxiety sensitivity and not with social threat or mental challenge. In addition, the results suggest a mediating role for some fairly general, non-specific anxiety components. Supportive of some specificity to panic disorder is the finding that subjects with panic disorder were more distressed by these challenges than subjects with other anxiety disorders. On the other hand, some degree of non-specificity is supported by the fact that the latter group of subjects showed greater distress in response to these challenges than did non-anxious controls (Rapee et al., 1992; Zinbarg et al., 2001).

Factor Structure of Other Measures of Anxiety Sensitivity

It is useful to examine other measures of anxiety sensitivity in order to determine whether or not there is agreement with the results of studies on the ASI factor structure.
Agreement will provide further support for the concept that anxiety sensitivity is a construct with a hierarchical structure. In fact, evidence supporting the multidimensionality of anxiety sensitivity has been found in all seven studies reported at this time. These include three studies of the Agoraphobia Cognitions Questionnaire (ACQ) and the Body Sensations Questionnaire (BSQ; Arrindell, 1993; Chambless, Caputo, Bright, & Gallagher, 1984; Marks, Basoglu, Alkubaisy, Sengun, & Marks, 1991), one of the Agoraphobic Cognitions Scale (ACS; Hoffart, Friis, & Martinsen, 1992), one of the Anxiety Sensitivity Beliefs Scale (ASBS; Kenardy, Evans, & Oei, 1992), and two of the Panic Appraisal Inventory (PAI; Feske & DeBeurs, 1996; Telch et al., 1989). Five of the studies contained tests of the possible hierarchical structure of anxiety sensitivity and four of them provided evidence that suggests a hierarchical model (Zinbarg et al., 1999).

The 60-item Anxiety Sensitivity Profile (ASP; Taylor & Cox, 1998) was developed in answer to concerns that the ASI may not have enough items to represent the type and number of lower-order factors of anxiety sensitivity (Zinbarg et al., 1997). At this point it has been used in a study that involved 349 university students. Evidence for four lower-order factors was provided by factor analysis: (1) fear of respiratory symptoms, (2) fear of cognitive dyscontrol, (3) fear of gastrointestinal symptoms, and (4) fear of cardiac symptoms. These factors loaded on and shared variance with a single higher-order factor, while also having unique variance. It is suggested by these results that anxiety sensitivity consists of independent contributions from four specific factors, while being the product of a general factor (Taylor & Cox, 1998).
While there is only a small amount of evidence that supports the construct validity of any of these measures, making it impossible to draw any firm conclusions from any of these studies when considered alone, the degree of convergence of the results across these studies is impressive. Also impressive is the amount of convergence between the results of these studies and the results from studies on the ASI factor structure. The fact that the convergence of results exists across different measures, rather than on one specific measure (e.g., ASI), provides evidence that anxiety sensitivity is part of a hierarchical structure (Zinbarg et al., 1999).

*Relationship of Trait Anxiety and Anxiety Sensitivity in the Hierarchical Structure*

The identification of individual difference variables plays a significant part in understanding the etiology of anxiety pathology. They are also important constructs that need to be considered in prevention, treatment, and symptom maintenance. Two such constructs are anxiety sensitivity and trait anxiety. There is a great deal of variation in people’s tendency to experience anxiety. A small provocation will result in the experience of anxiety symptoms by some, but others require the occurrence of extremely stressful circumstances before they report anxiety. These variations in anxiety proneness are indicated in the construct of trait anxiety (Reiss & McNally, 1985). The same individual differences among people are found in their fear of anxiety symptoms. While some people view them with anticipated anxiety, most view anxiety as no more than unpleasant. These individual differences are indicated in the construct of anxiety...
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sensitivity (Reiss & McNally, 1985). Beliefs that the symptoms of anxiety are harmful provide the basis for the fears of anxiety symptoms to which anxiety sensitivity refers. While a person with low anxiety sensitivity regards heart palpitations as no more than an unpleasant sensation, a person with high anxiety sensitivity may be certain that a heart attack is imminent. The leading measure of these variations in anxiety sensitivity has been the Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992; Reiss et al., 1986).

There were questions about whether or not trait anxiety and anxiety sensitivity are distinct entities subsequent to the initial studies of anxiety sensitivity. It was suggested that the ASI simply measures trait anxiety (Lilienfeld, 1996a, 1996b; Lilienfeld, Jacob, & Turner, 1989, 1993, 1996). Further empirical studies (e.g., Rapee & Medoro, 1994) and refinement of the concept of anxiety sensitivity (McNally, 1989, 1996a; 1996b; Taylor 1995, 1996) were the responses to these questions (McNally, 1999). There now appears to be agreement that anxiety sensitivity plainly indicates an empirically and conceptually distinct individual difference variable. Trait anxiety is now viewed as a higher order or third-order construct indicative of an individual who tends generally to experience fear in response to stressors (Lilienfeld et al., 1993). Anxiety sensitivity is indicative of an individual who tends specifically to experience fear in response to his or her own anxiety symptoms. It is now viewed as a lower-order or second-order construct. According to Reiss (1987, 1991; Reiss & McNally, 1985) factors of injury sensitivity and fear of negative evaluation covary with anxiety sensitivity as a result of their loadings on the third-order trait anxiety factor (Lilienfeld, 1996b; McNally, 1989). The notion that provides a basis for the modest correlation between measures of anxiety sensitivity and
those of trait anxiety is that anxiety sensitivity is an aspect of trait anxiety (Lilienfeld, 1996a).

Quite a lot has been accomplished to support the view that anxiety sensitivity is not merely a part of trait anxiety. Indeed, following a review of 11 studies that contained correlations between measures of trait anxiety and the ASI, it was determined that trait anxiety and anxiety sensitivity are related but separable constructs (McNally, 1996; Reiss, 1991). This agrees with Lilienfeld et al's (1993) proposed hierarchical structure. Use of the ACQ and BSQ also provided confirming evidence that anxiety sensitivity and trait anxiety are related but separable constructs (Chambless et al. 1984). The hypotheses that trait anxiety is a third-order factor and anxiety sensitivity is one of several second-order factors that can be broken down into first-order anxiety sensitivity factors agree with initial factor analytic evidence provided by Taylor (1995) and Zinbarg and Barlow (1996).

Limitations on the Dimensionality of Anxiety Sensitivity in the Current Literature

Children and older adults. Little has been done to examine the dimensionality of anxiety sensitivity in children and older adults. Confirmatory factor analysis has been initiated on the CASI to explore various models of its structure with preliminary evidence supporting multidimensionality in both child patients and nonpatients (Silverman, 1993). Data has recently been collected on the ASI from a group of community volunteers ages
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65 to 97, but there are no known reports in existence on this age group (Zinbarg et al., 1997).

Construct validity. This is an area of significance especially for assessment, as well as ultimately for prevention, treatment, and treatment maintenance. Current and future analyses of the construct validity of the measures of anxiety sensitivity can provide vital information about the extent of their usefulness. Preliminary evidence supports the construct validity of the three first-order anxiety sensitivity factors identified with analysis of the ASI. On the other hand, whether or not the subscales provide differential predictive validity with such clinically important variables as response to panic provocation procedures, treatment response, and course of disorder is not yet known. Therefore it cannot yet be concluded that distinguishing among these constructs is clinically important (Zinbarg et al., 1999). Reanalysis of data from earlier studies of panic provocation and panic control therapy is currently in process (Zinbarg et al., 1999; Barlow & colleagues, 1999) and one has been completed, as mentioned earlier (Zinbarg et al., 2001).

To Summarize

There are a number of theoretical pieces which have been addressed by the current study. The earliest attempt at providing a theoretical framework for anxiety sensitivity (i.e., expectancy theory; Reiss, 1980, 1991; Reiss & McNally, 1985; Taylor & Fedora, 1999) suggests that it has been perceived as having a unifactorial structure. It has
also been viewed as a multifactorial concept consisting of a general factor and three lower-order factors (Lilienfeld et al., 1996; Taylor, 1996). This study may provide support for the concept that anxiety sensitivity is a multifactorial construct consisting of anxiety sensitivity and six underlying factors. Evidence may be provided for reliability and validity that will add to the evidence provided by previous studies that anxiety sensitivity is not an artifact of another construct such as the fear of pain or another trait.

A handful of studies have suggested questions about expectancy theory’s efforts to link anxiety sensitivity and expectancy in order to explain fear, panic, and avoidance. It is argued that the theory needs to be expanded and modified to address all of these findings. Expectancy theory also claims that anxiety sensitivity is one of three fundamental fears. More recently the possibility has been suggested that it is not a fear, but a variable moderating fear. The results of one study suggest the role of partial mediator (Stewart et al., 2001). It is argued that it is quite possible that a mediating role will be found for anxiety sensitivity. However, there is not yet enough evidence to support that claim.

Returning to the issue of underlying factors, the factor labeled the fear of observable reactions to anxiety has often been found in factor-analytic studies of the ASI. There have been suggestions, however, that the fear of negative evaluation, one of the three fundamental fears identified in expectancy theory (Reiss, 1980, 1991; Reiss & McNally, 1985; Taylor & Fedora, 1999), may be too heavily represented in the ASI items (Cox et al. 1999). The current study provides evidence that if there are adequate items to assess this fear with a clinical sample, these items may provide the range of scores that
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are sufficient to provide both high reliability and convergent validity for this domain. In addition to evidence for the existence of this underlying factor, there is a growing body of evidence that supports the view that elevated anxiety sensitivity is present across anxiety disorders (Taylor et al., 1992). The nature of the clinical sample in the current study (i.e., representation of all anxiety disorders) provides support that is consistent with theoretical and empirical efforts of the past 20 years. That is to say, that anxiety sensitivity plays a role in the disposition and etiology of anxiety disorders in general, and panic disorder in particular (Zinbarg et al., 1999; Barlow, 1988, 1991; Clark, 1986; Goldstein & Chambless, 1987; McNally, 1990; Reiss, 1991; Reiss & McNally, 1985; Reiss et al., 1986).

Related to etiology is the issue of cognitive biases in the online processing of anxiety sensations. There is some agreement that the utilization of congruence between threat cues and beliefs will increase consistency in the results of the investigation of the role played by attentional, memory, and interoceptive acuity biases in the processing of anxiety sensations. It is important to know whether or not those who are at risk for the development of panic and other anxiety disorders have the same biases as those who currently experience the symptoms of those disorders. The current study may provide evidence that the ASP is a psychometrically superior measure that will provide a better assessment of beliefs about bodily sensations.

Etiology also includes the relationship of the personality domains and the anxiety sensitivity construct. Recently there has been some focus on the differences and similarities between the anxiety sensitivity construct and the personality domains of
neuroticism and trait anxiety (Reiss, 1993). Whether neuroticism precedes anxiety sensitivity, or anxiety sensitivity precedes neuroticism, is unknown at this time (Lilienfeld, 1999). A recent study that included the ASP (Van der Does et al., 2003) found a significant relationship for anxiety sensitivity and harm avoidance, an underlying factor for the higher order dimension of Constraint, a fearfulness or response inhibition dimension (Telegen, 1978, 1982; Telegen & Waller, 1994). Continued research in this area will further inform theory and all phases of intervention. The current study provides evidence that the ASP is the measure that will be the most accurate in the identification of the underlying factors of anxiety sensitivity, as well as the anxiety sensitivity construct when utilized in these investigations.

While utilizing the ASI and factor analysis in related research, evidence has been found for a hierarchical structure of the anxiety sensitivity construct. Results have been contradictory due to choices by investigators to focus on the upper or lower level, or to make no differentiation between higher and lower level factors. Accurately establishing the validity of the anxiety sensitivity construct, its measures, and the factors which make up the structure is important. This is due to the fact that distinct factors may be linked to distinct mechanisms (Cattell, 1978) in, for instance, the etiology of anxiety reactions (Zinbarg et al., 1999). Since the ASI subscales, which are linked to the lower order anxiety sensitivity factors, have not been included in most studies, support for incremental validity of either the general anxiety sensitivity factor or the lower order factors has not been determined (Lilienfeld, 1996; Lilienfeld et al. 1993; McNally, 1989, 1996; Reiss, 1991). Even with recent reanalyses that calculate and analyze the subscales
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(Zinbarg et al., 2001), there continue to be problems because the reliability of the ASI subscales has been questioned (Zinbarg et al., 1997).

As reported previously, the current study may provide evidence with a clinical sample that the ASP is psychometrically superior to the ASI because it has been designed to address the issues previously presented. All the items in the ASP appear to be specific to the anxiety sensitivity construct. In addition, the ASP has a sufficient number of items to establish internal consistency reliability and convergent validity for each scale. These are steps that are necessary, although not sufficient, to establish the construct validity of a measure. A sufficient number of items on each scale will also increase the possibility that the lower-order factors of anxiety sensitivity will be more accurately identified (Taylor & Cox, 1998).

Specific Hypotheses

To supply further support for the convergent validity of the ASP, the primary goal of this study was to correlate the subscale scores of the ASP with the full scores of the ASI. It was predicted that subscale scores for the ASP would tend to have large correlations with the ASI (i.e., rs ≥ 0.50; Cohen, 1988). Consideration of the relationship of the ASP subscales with trait anxiety (anxiety proneness) was the secondary aim. Trait anxiety was evaluated with the trait version of the State-Trait Anxiety Inventory (Spielberger, 1983). It was predicted that a modest relationship (correlation) would be found between the ASP subscales and trait anxiety. Such a relationship was found by
Taylor & Cox (1998) when using a non-clinical sample. Modest correlations ($r_s \approx 0.30$) have been found in earlier studies between trait anxiety and anxiety sensitivity, when it was assessed by the ASI.
Participants

One hundred five subjects were selected. A subset of the subjects was in treatment at health clinics, private practices, and an anxiety disorder treatment center. Other subjects were members of anxiety disorder support groups, students at the Philadelphia College of Osteopathic Medicine, or attendees of the 2003 Anxiety Disorders Association of America Conference. All were selected on the basis of the experience of anxiety symptoms. This was done in order to meet the goal of this study, which was to seek convergent validity for a new measure of anxiety sensitivity based on data derived from a clinical sample. Subjects were recruited with the use of flyers presented by mental health care providers and support group leaders. Flyers were also posted at the ADAA conference and in the e-mail of all the students at the Philadelphia College of Osteopathic Medicine. All participants were volunteers who were offered and paid $20 for an average of one hour of their time.

Participation criteria included: (1) being between 18 and 65 years of age, (2) a DSM-IV Axis I diagnosis of one or more anxiety disorders, (3) no diagnosis of current substance dependence, and (4) no diagnosis of current or past schizophrenia, current bipolar disorder, or organic mental disorder. Patients could be diagnosed with other DSM-IV disorders. The following anxiety disorders were represented: obsessive-compulsive disorder, generalized anxiety disorder, post traumatic stress disorder, panic
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disorder with and without agoraphobia, agoraphobia without panic, social phobia, specific phobia, acute stress disorder, anxiety disorder due to a general medical condition, and anxiety disorder not otherwise specified. Based on the inclusion and exclusion criteria, 105 subjects were selected for the study. Subjects were between the ages of 19 and 65 and consisted of 69 females and 36 males. All educational levels (i.e., grade 6 or less through completion of graduate or professional school) were represented. All categories of employment status were represented, including early retirement and disability. Retirement after age 65 was not represented due to the exclusionary requirement of age 66 and above. All categories of marital status were represented. Caucasian, Asian, Black, East Indian, and other ethnicities were represented.

Overview of the Research Design

The ASP was initially designed by Taylor & Cox (1998) for the purpose of performing a more extensive exploration of the factor structure of anxiety sensitivity with a nonclinical sample. A more inclusive evaluation of the factors, which are part of anxiety sensitivity, might be given by a measure like the ASP, in which items have been expanded to measure domains that have been found to be present in the ASI, but not always factorially demonstrated in this scale. Before the usefulness of the ASP can be evaluated, however, more factor-analytic studies with normative and clinical populations, as well as reliability and validity studies are needed (Peterson & Plehn, 1999).

This study was a correlational design. It was designed to evaluate convergent validity of the ASP through replication of the correlational aspect of their study (Taylor
Anxiety Sensitivity: Validity of the ASP & Cox, 1998), while using a clinical sample. Previous factor analytic studies of the ASI (Taylor, 1996) have suggested that anxiety sensitivity consists of six domains, which have been evaluated in the six 10-item scales of the ASP (Taylor & Cox, 1998). In this study patients were asked to report the degree of danger the symptoms presented from each of these domains: (1) fear of cardiovascular symptoms, (2) fear of respiratory symptoms, (3) fear of gastrointestinal symptoms, (4) fear of publicly observable anxiety reactions, (5) fear of dissociative and neurological symptoms, and (6) fear of cognitive dyscontrol (Taylor & Cox, 1998; Peterson & Plehn, 1999).

The SCID-I/P (First, Spitzer, Gibbon, & Williams, 1994) was used to support concurrent validity through comparison of its suggested diagnosis with the diagnosis assigned by clinicians employing interview techniques (Aiken, 1996). The ASI was included so that convergent validity of the ASP could be evaluated by correlating the subscale scores on the ASP with the total scores of the ASI. The State-Trait Anxiety Inventory (Spielberger, 1983) was included in order to provide information on the relationship between the ASP subscales and trait anxiety (i.e., discriminant validity; Aiken, 1996; Cox & Taylor, 1998).

Most subjects were assessed individually. Completion of the measures: (1) ASP, (2) ASI, and (3) Trait portion of the STAI was followed by completion of the computerized version of the SCID-I/P. However, when assessing 19 members of two of the support groups, two subjects were assessed simultaneously. In those situations, half of the subjects completed the paper and pencil measures after they completed the SCID-I/P.
Subjects were given the option of answering the questions of the SCID-I/P as they were read by the investigator, or using the computer to answer the questions themselves. All assessments were administered by the investigator.

**Measures**

The Anxiety Sensitivity Index (ASI) is a 16-item self-report questionnaire that measures the amount of concern that the individual has about the harmful consequences of anxiety and anxiety-related sensations that are the result of elevated autonomic arousal (Peterson & Reiss, 1992; Reiss et al., 1986; Zinbarg et al., 1999). Each item is rated on a 5-point Likert scale (range from very little to very much). The total score, which ranges from 0 to 64, is obtained by finding the sum of the item scores (Peterson & Plehn, 1999; Richards & Bertram, 2000; Watt et al., 1998). While the majority of the items focus on the fear of physical sensations, a few items touch on beliefs about mental control (e.g., inability to concentrate) and the observation by others of symptoms of anxiety (e.g., noticing shakiness; Peterson & Plehn, 1999).

Good psychometric properties of the Anxiety Sensitivity Index, including its incremental validity as a measure of the fear of anxiety, and factorial distinction from state or trait anxiety (McNally, 1994; Taylor et al., 1991), are supported by a significant body of evidence (Silverman & Weems, 1999; Watt et al., 1998). Good internal consistency (Peterson & Heilbronner, 1987; Taylor et al., 1991; Telch et al., 1989), test-retest reliability (Maller & Reiss, 1992; Reiss et al., 1986), and predictive validity (Peterson & Reiss, 1992; Reiss, 1991) have been well-documented in over 100 peer-
reviewed journal articles (Zinbarg et al., 1999). Differential concurrent validity for the three lower-order factors has been reported (Stewart et al., 1997; Taylor et al., 1996; Zinbarg & Barlow, 1996; Zinbarg et al., 1997).

The Anxiety Sensitivity Profile is a 60-item self-report questionnaire that measures the extent to which individuals agree with items on a 7-point Likert scale (range from likely to not likely). As reported earlier, the Anxiety Sensitivity Profile is made up of six scales. A single higher order factor with four lower order factors, each of which contains unique variance, was suggested by exploratory factor analysis on a sample of 349 college students. The lower order factors were named: (1) fear of respiratory distress, (2) fear of cognitive dyscontrol, (3) fear of gastrointestinal symptoms, and (4) fear of cardiac symptoms (Peterson & Plehn, 1999; Taylor & Cox, 1998; Van der Does et al., 2003).

Good levels of internal consistency were found for all six scales of the Anxiety Sensitivity Profile and the Anxiety Sensitivity Profile factors generally had large correlations (rs ≥ 0.50) with the Anxiety Sensitivity Index (Taylor & Cox, 1998). This relationship provides support for the convergent validity of the Anxiety Sensitivity Profile with a non-clinical sample. Also, modest correlations with the trait version of the State-Trait Anxiety Inventory, with overlapping variance ranging from 1 to 8%, are similar to findings for the Anxiety Sensitivity Index (Taylor, 1996; Taylor & Cox, 1998).

The State-Trait Anxiety Inventory (STAI; Spielberger, 1983) has served extensively to measure both state and trait anxiety levels (Richards & Bertram, 2000). Its 40 items on a 5-point Likert scale (range from almost never to almost always) reflect the
Anxiety Sensitivity: Validity of the ASP

individual’s anxiety-proneness. It is supported by substantial evidence as a psychometrically sound measure (Peterson & Reiss, 1987; Spielberger, 1983). It is reported to be a valid and reliable instrument that has been used extensively to measure the levels of both state and trait anxiety (Richards & Bertram, 1999).

Procedures

Subjects were recruited from the following sites: healthcare clinics, Anxiety and Agoraphobia Treatment Center, private practices, and a mental health clinic. Patients who presented with anxiety symptoms were given a flier about the study. The majority of subjects were given the flier by their treatment provider. Three subjects were given the flier by the intake clinician. Anxiety support group leaders informed group members about the study and gave fliers to potential subjects. At the Philadelphia College of Osteopathic Medicine, the flier about the study was e-mailed to all students. The flier told potential subjects where the assessment would take place, the setting, the available times, and the purpose. It contained the investigator’s name and phone number and space for their names and phone number. It also contained the possible risks and benefits of taking part in the study. Subjects’ signatures on the flier meant that the investigator had permission to get in touch with them about participation in the study. Those who signed the flier were contacted by phone. Any questions posed by potential subjects were answered. Those who chose to participate decided on an appointment date and time with the investigator. Subjects who participated at the Anxiety Disorders Association of
Anxiety Sensitivity: Validity of the ASP

America conference (ADAA) responded to a poster and the flier. They signed up for an assessment time during the conference with the investigator. All subjects whose response was connected to presentation of the flier by a treatment provider participated at their treatment site. Support group members participated at their meeting site or in their homes. ADAA conference attendees participated at the conference site.

Prior to the actual administration of the measures, all subjects at all sites were given a letter and an informed consent form. These explained the study in detail, including how long the testing would take, what would be done, and the fact that their confidentiality was protected by the storage of their test materials in either a locked file or locked room. They were informed that only the individuals involved in the research and members of the Institutional Review Board would have access to their records. And finally, they were given the option of having a copy of their test results sent to their treatment provider. Subjects who chose that option were asked to sign a release of information form. Subjects in treatment were also asked to sign a release of information that would accompany a form that requested the chart diagnoses from their treatment provider. Following this initial segment of the assessment, subjects were asked to complete a form that requested date of birth, gender, marital status, employment status, educational level, race, and current medications.

Most subjects then completed three paper and pencil self-report questionnaires, which consisted of the Anxiety Sensitivity Profile, Anxiety Sensitivity Index and the Trait portion of the State-Trait Anxiety Inventory. Each subject was asked to read a set of written instructions that was part of each questionnaire. These required approximately
30 minutes for completion. Most subjects were then tested individually with the computerized version of a structured clinical interview using the SCID-I/P (First et al., 1994), which required on average 30 minutes. As mentioned earlier, the battery and structured clinical interview were reversed in order when members of two support groups were tested two at a time. The investigator was available at all times to answer questions and provided assistance when appropriate during the test period. The investigator’s presence at all assessments ensured that the conditions were carried out as intended. The entire assessment consisted of one session and averaged one hour in length. Materials were reviewed for suicidal thoughts within a day of administration. If a subject was identified as actively suicidal, appropriate personnel at the treatment site, or the individual was contacted.
Chapter 3

Results

Based on the magnitude of factor loadings in the Taylor and Cox study (1998), which suggests that the solution is likely to be stable (i.e., replicable; Guadagnoli & Velicer, 1988; Taylor & Cox, 1998), the following were the expected results: (1) the subscale scores for the ASP will have large correlations with the total scores of the ASI (i.e., rs ≥ 0.50) and (2) there will be a modest, but significant correlation (rs ≈ 0.30) between the ASP subscale scores and trait anxiety (anxiety proneness), as assessed by the trait version of the State-Trait Anxiety Inventory.

Descriptive

To provide evidence for these hypotheses, one hundred five individuals who experience anxiety symptoms (69 females and 36 males) completed the ASP, ASI, STAI-Trait portion, and the SCID-I/P. Thirty subjects, or 29% of the sample, were diagnosed with a single anxiety disorder. Thirty-five subjects, or 33% of the sample, were diagnosed with two or more anxiety disorders. Forty subjects, or 38% of the sample, were diagnosed with at least one anxiety disorder, and were comorbid for other Axis I diagnoses. Thirty-four subjects or 32% of the sample were comorbid for depression. The anxiety disorders were represented as follows: (1) 69 subjects, or 66% were diagnosed with obsessive-compulsive disorder, (2) 38 subjects, or 36% were diagnosed with social
Anxiety Sensitivity: Validity of the ASP

phobia, (3) 35 subjects, or 33% were diagnosed with specific phobia, (4) 29 subjects, or 28% were diagnosed with agoraphobia without panic disorder, (5) 21 subjects, or 20% were diagnosed with post traumatic stress disorder, (6) 18 subjects, or 17% were diagnosed with panic disorder with agoraphobia, (8) 14 subjects, or 13% were diagnosed with generalized anxiety disorder, (9) 7 subjects, or 6% were diagnosed with panic disorder without agoraphobia, (10) 1 subject, or 1% was diagnosed with anxiety due to a general medical condition, (11) 1 subject, or 1% was diagnosed with anxiety disorder not otherwise specified, and (12) 1 subject, or 1% was diagnosed with acute stress disorder.

Scores of central tendency and variability for the ASP subscales and ASP, ASI, and STAI total scores are presented in Table 1. The mean total score on the ASI (i.e., 43.96) is high in relationship to the mean total score on the ASI in most studies that have used a clinical sample (e.g., Blais et al., 2001; Stewart, et al. 2001; Taylor, 1995; Zinbarg, et al., 1997). In most studies high scores range from 30 to 38. In the current study, the ASP mean total score was 210.97 (SD = 65.04). This is somewhat higher than the mean total score in the Van der Does et al. (2003) study, in which the ASP mean total score was 195.9 (SD = 74.4). Taylor and Cox (1998) did not provide mean scores for their college student sample.
Table 1

**Mean Subscale Scores and Standard Deviations for the Anxiety Sensitivity Profile (ASP)**

<table>
<thead>
<tr>
<th>Subscale</th>
<th>M</th>
<th>Std</th>
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</thead>
<tbody>
<tr>
<td>Cardio</td>
<td>3.70</td>
<td>1.40</td>
</tr>
<tr>
<td>Respira</td>
<td>4.02</td>
<td>1.49</td>
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<tr>
<td>Gastro</td>
<td>2.57</td>
<td>1.09</td>
</tr>
<tr>
<td>POAR</td>
<td>3.34</td>
<td>1.27</td>
</tr>
<tr>
<td>Disneuro</td>
<td>3.70</td>
<td>1.34</td>
</tr>
<tr>
<td>Cogdysc</td>
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<td>1.45</td>
</tr>
<tr>
<td>ASP</td>
<td>210.97</td>
<td>65.04</td>
</tr>
<tr>
<td>ASI</td>
<td>43.96</td>
<td>11.67</td>
</tr>
<tr>
<td>STAI</td>
<td>51.24</td>
<td>12.15</td>
</tr>
</tbody>
</table>

*Note.* Cardio = cardiovascular fears; Respira = respiratory fears; Gastro = gastrointestinal fears; POAR = fears of publicly observable anxiety reactions; disneuro = fears of dissociative and neurological symptoms; Cogdysc = fears of cognitive dyscontrol
**Internal Consistency**

Internal consistencies (i.e., Cronbach’s alpha; Cronbach, 1951) for the ASI total score and the ASP subscale scores are presented in Table 2. These were calculated based on the responses of the total clinical sample. Cronbach’s alpha, a coefficient of internal consistency, is a widely used reliability statistic. A recommended minimum is 0.80 (Loewenthal, 2001). The high internal reliability estimate for the ASI (i.e., .88), which is consistent with other studies (Zinbarg, et al., 1997), indicates high internal consistency. Internal reliability estimates for the subscales of the ASP ranged from .87 to .94, demonstrating that they are highly internally consistent. These are consistent with another study (i.e., all subscales above .88; Van der Does et al., 2003), that utilized the ASP with a clinical sample, as well as Cox and Taylor (i.e., all subscales above .87; Cox & Taylor, 1998).

Corrected item-subscale total score correlations were calculated for the ASP factors and are presented in Table 3. These were found by correlating the score on each item of a given factor with the corrected subscale total score. The corrected subscale total score was obtained by adding the scores of all the items on a given factor while excluding the targeted item. For example, for item 51 on Factor 1, the item subscale total score correlation was found by correlating the score on item 51 with the corrected total score (i.e., all items except 51 on Factor 1). All correlations are high, with the exception of five items, which are in the moderate range. They range from .37 to .86.
Table 2

Reliability Analysis of the Anxiety Sensitivity Index (ASI) Total Scores and the Anxiety Sensitivity Profile (ASP) Subscale Scores

<table>
<thead>
<tr>
<th>Reliability coefficients</th>
</tr>
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<tbody>
<tr>
<td>ASI</td>
</tr>
<tr>
<td>ASP subscales</td>
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<tr>
<td>Cogdysc</td>
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<tr>
<td>Disneuro</td>
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<tr>
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<tr>
<td>Gastro</td>
</tr>
<tr>
<td>Respira</td>
</tr>
<tr>
<td>Cardio</td>
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</table>

Note. Cardio = cardiovascular fears; Respira = respiratory fears; Gastro = gastrointestinal fears; POAR = fears of publicly observable anxiety reactions; Disneuro = fear of dissociative and neurological symptoms; Cogdysc = fear of cognitive dyscontrol
Table 3

Corrected Item-Subscale Total Score Correlations for ASP Factors

<table>
<thead>
<tr>
<th>Item</th>
<th>Factor 1 r</th>
<th>Item</th>
<th>Factor 2 r</th>
<th>Item</th>
<th>Factor 3 r</th>
<th>Item</th>
<th>Factor 4 r</th>
<th>Item</th>
<th>Factor 5 r</th>
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<th>Factor 6 r</th>
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<tr>
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<td>.73</td>
<td>57</td>
<td>.47</td>
<td>53</td>
<td>.63</td>
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<td>.70</td>
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<td>.62</td>
</tr>
<tr>
<td>42</td>
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<td>47</td>
<td>.82</td>
<td>28</td>
<td>.47</td>
<td>10</td>
<td>.62</td>
<td>43</td>
<td>.63</td>
<td>54</td>
<td>.76</td>
</tr>
</tbody>
</table>

**Note.** Factor 1 = Cardiovascular; Factor 2 = Respiratory; Factor 3 = Gastrointestinal; Factor 4 = Publicly Observable Anxiety Reactions; Factor 5 = Dissociative and Neurological Symptoms; Factor 6 = Cognitive Dyscontrol

p < .01, one-tailed
Hypothesis 1

The subscale scores for the ASP will have large correlations with the total scores of the ASI (i.e., $r_s \geq 0.50$). Table 4 presents the results of the correlations between the ASP subscales, the ASI total scores, and the trait version of the Stait-Trait Anxiety Inventory (STAI). To reach these results a Pearson product-moment Correlation was used, since it is one common technique for demonstrating the validity of new tests. It measures the degree and direction of linear relationship between two variables (Gravetter & Wallnau, 2000). In addition, parametric statistical analysis, including Pearson correlation, is suitable for interval data. Rating scales, such as those used in the ASP, ASI, and STAI, may be treated as interval scales (Loewenthal, 2001). Since directional predictions were made, one-tailed tests were used to establish statistical significance.

As predicted in hypothesis 1, all of the subscale scores for the ASP were found to have large correlations ($r_s \geq 0.50$) with the ASI. These correlations range from .50 to .66, are significant at the 0.01 level, and support the convergent validity of the six ASP subscales. The highest correlation was between the ASI and the POAR subscale (i.e., fears of publicly observable anxiety reactions). This subscale and the Disneuro subscale (i.e., fear of dissociative and neurological symptoms) were not identified as factors in factor analysis in the Taylor and Cox study (1998). However, they were identified as factors in a more recent study that utilized the ASP with a clinical sample in which a factor analysis was completed (Van der Does et al., 2003).

In Taylor and Cox (1998) a Pearson correlation was presented that includes the four ASP subscales that were identified as factors in factor analysis. In that study, three
of the four ASP subscales had somewhat lower correlations with the ASI (i.e., fear of respiratory symptoms = .41, fear of gastrointestinal symptoms = .50, and fear of cardiac symptoms = .50) in comparison with the current study. Only the fear of cognitive dyscontrol had a correlation (i.e., .57) that was consistent in both studies. On the other hand, the order of the four ASP subscale correlations with the ASI from lowest to highest in Taylor and Cox (1998) was consistent with the current study. From lowest to highest in order they are: (1) respiratory fears, (2) cardiovascular fears, (3) gastrointestinal fears, and (4) cognitive dyscontrol fears.

Intercorrelations between the ASP subscales in the current study ranged in size from modest to large (i.e., .34 to .80). These correlations are partially consistent with the other study that utilized the ASP with a clinical sample (i.e., .52 to .82; Van der Does et al., 2003). Eleven of fifteen intercorrelations in the current study were large (rs ≥ 0.50) relationships. The largest correlation was between the Cardio subscale (i.e., cardiovascular fears) and the Respira subscale (respiratory fears). The intercorrelations between the four ASP subscales identified as factors in the study using a nonclinical sample (Cox & Taylor, 1998) were in the modest range (i.e., from .36 to .48). With the exception of the correlation between cardiovascular and respiratory fears, the correlations for the same factors occupy the lower end (i.e., .34 to .54) of the range for the current study. The remaining larger correlations in the current study consist of relationships between the other subscales and the POAR (i.e., fears of publicly observable anxiety reactions) and Disneuro (i.e., fears of dissociative and neurological symptoms) subscales. As reported above, these domains were not identified as factors in factor analysis in the
Taylor and Cox (1998) study, but were identified as factors in the more recent study (Van der Does et al. 2003).

Hypothesis 2

There will be a modest, but significant correlation \((r_s \approx 0.30)\) between the ASP subscale scores and trait anxiety (anxiety proneness), as assessed by the trait version of the State-Trait Anxiety Inventory. Table 3 shows that the ASP subscales tended to have statistically significant, although modest correlations \((r_s \approx 0.30)\) with the trait version of the STAI. They range from .10 to .30 and were significant at the 0.05 level. These findings are consistent with those found for the ASI in other studies (Taylor, 1996), and support the view that AS and trait anxiety are correlated but distinct constructs (Taylor & Cox, 1998). They are also consistent with the findings of Taylor and Cox (1998), in which correlations of the ASP subscales and the STAI ranged from .11 to .29 with a nonclinical sample. Additional consistencies exist between highest and lowest subscale correlations of the ASP and STAI in the Taylor and Cox study (1998) and the current study. The highest correlation for both studies was between the fear of cognitive dyscontrol subscale and the STAI. The lowest correlations were between respiratory fears, gastrointestinal fears, cardiovascular fears and the STAI.

Table 5 presents the results of the correlations between the ASI, ASP, and the trait version of the STAI total scores. Because directional predictions were made, one-tailed tests were used to establish statistical significance. The ASP total score was found to
Table 4

Intercorrelations Between Subscales of the Anxiety Sensitivity Profile (ASP) and Total Scores of the Anxiety Sensitivity Index (ASI) and Stait-Trait Anxiety Inventory (STAI)

<table>
<thead>
<tr>
<th></th>
<th>STAI</th>
<th>ASI</th>
<th>Cardio</th>
<th>Respira</th>
<th>Gastro</th>
<th>POAR</th>
<th>Disneuro</th>
<th>Cogdysc</th>
</tr>
</thead>
<tbody>
<tr>
<td>STAI</td>
<td>----</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASI</td>
<td>.44**</td>
<td>----</td>
<td></td>
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</tr>
<tr>
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<td>.10</td>
<td>.55**</td>
<td>----</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
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<td>.80**</td>
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<td>.56**</td>
<td>.51**</td>
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<td>Disneuro</td>
<td>.23*</td>
<td>.52**</td>
<td>.62**</td>
<td>.64**</td>
<td>.46**</td>
<td>.69**</td>
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<tr>
<td>Cogdysc</td>
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<td>.57**</td>
<td>.40**</td>
<td>.34**</td>
<td>.54**</td>
<td>.68**</td>
<td>.66**</td>
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</tr>
</tbody>
</table>

Note. Cardio = cardiovascular fears; Respira = respiratory fears; Gastro = gastrointestinal fears; POAR = fears of publicly observable anxiety reactions; Disneuro = fears of dissociative and neurological symptoms; Cogdysc = fear of cognitive dyscontrol

**p < .01, one-tailed

* p < .05, one-tailed
have a large correlation ($rs \geq 0.50$) with the ASI (i.e., .689), was significant at the 0.01 level, and supports the convergent validity of the ASP.

A useful index of the accuracy of prediction can be derived by squaring the correlation coefficient. The resulting statistic is called the coefficient of determination because it measures the proportion of variability in one variable that can be determined from the relationship with the other variable (Aiken, 1996; Gravetter & Wallnau, 2000). In the current study, the coefficient of determination for the correlation between the ASP and ASI total scores is 0.475, so approximately 48% of the variance in the ASI total scores can be accounted for by variability in the ASP total scores. This indicates that 52% of the variability in the ASI total scores was unaccounted for by ASP total scores. This remainder is known as the coefficient of alienation (Aiken, 1996). The current study is consistent with Cox and Taylor (1998), whose study used a nonclinical sample. The coefficient of determination for the correlation between the ASP and the ASI total scores in their study was 0.44, so approximately 44% of the variance in the ASI total scores can be accounted for by variability in the ASP total scores. This indicates that 56% of the variability was unaccounted for by the ASP total scores.

Table 4 also shows that the ASP total score had a statistically significant, although modest correlation ($r \approx 0.30$) with the trait version of the STAI (i.e., .226), which was significant at the 0.05 level. This finding is consistent with those found for the ASI in other studies (Taylor, 1996), and supports the view that anxiety sensitivity and trait anxiety are correlated, but distinct constructs (Taylor & Cox, 1998). This is also consistent with the finding of Taylor and Cox (1998), in which the relationship between
the ASP and the STAI was .26 with a nonclinical sample. In the current study the
coefficient of determination for the correlation between the ASP total scores and the
STAI total scores is 0.051, so approximately 5% of the variance in the STAI total scores
can be accounted for by the variability in the ASP total scores. This indicates that 95% of
the variability in the STAI total scores was unaccounted for by the ASP total scores. The
current study is consistent with Taylor and Cox (1998). The coefficient of determination
for the correlation between the ASP total scores and the STAI total scores in their study is
0.06, so approximately 6% of the variance in the STAI total scores can be accounted for
by the variability in the ASP scores. This indicates that 94% of the variability was
unaccounted for by the ASP total scores.

The correlation of the trait version of the STAI and the ASI in the current study is
somewhat higher (i.e., .438) in comparison with other studies (Taylor, 1996). However, it
also is in the modest range and supports the view that anxiety sensitivity and trait anxiety
are correlated but distinct constructs. In the current study, the coefficient of
determination for the correlation between the ASI and STAI total scores is 0.192, so
approximately 19% of the variance in the STAI total scores can be accounted for by the
variability in the ASI scores. This indicates that 81% of the variability in the STAI total
scores was unaccounted for by the ASI total scores.
Table 5

Intercorrelations between the Anxiety Sensitivity Profile (ASP), Anxiety Sensitivity Index (ASI), and the Stait-Trait Anxiety Inventory (STAI) Total Scores

<table>
<thead>
<tr>
<th></th>
<th>ASP</th>
<th>ASI</th>
<th>STAI</th>
</tr>
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<tbody>
<tr>
<td>ASP</td>
<td>----</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASI</td>
<td>.689**</td>
<td>----</td>
<td></td>
</tr>
<tr>
<td>STAI</td>
<td>.226*</td>
<td>.438**</td>
<td>----</td>
</tr>
</tbody>
</table>

* * p < .01, one-tailed
* * p < .05, one-tailed
Summary and Explanation

This study has taken important steps in providing support for the construct validity of the ASP. Taylor and Cox (1998) conducted their investigation with a university sample. This study was the first investigation to utilize a clinical sample for the purpose of providing support for internal consistency reliability and convergent validity, necessary though not sufficient steps, for establishing the construct validity of an instrument. As expected, the findings provided further evidence of convergent validity for the ASP with the ASI, a psychometrically well-documented measure of anxiety sensitivity (Zinbarg et al., 1997). When assessing subjects who experienced the symptoms of anxiety, the ASP was found to have a large correlational relationship with the ASI (Taylor & Cox, 1998). The ASP was also found to have a modest correlational relationship with the Trait portion of the STAI, a measure of anxiety proneness. Both relationships (i.e., convergent and discriminant validity) contribute to construct validity. This is referred to as the multitrait-multimethod approach (Aiken, 1996; Campbell & Fiske, 1959).

Again, these were the expected results. This expectation was based on the magnitude of factor loadings in the Taylor and Cox study (1998), which suggested that the solution was likely to be stable (i.e., replicable; Guadagnoli & Velicer, 1988; Taylor
Results of studies of the ASI have provided evidence that factor structures do not vary greatly across clinical and non-clinical populations (Taylor, 1996; Zinbarg et al., 1997). In this study, the assumption was made that the same condition applies to correlational relationships, because they are a necessary part of factor analysis.

Convergent Findings

The internal consistency reliability results of the current study were generally quite similar to those of Taylor and Cox (1998) and Van der Does et al. (2003). These results demonstrate high internal consistency for the ASP subscales. This is important because adequate (i.e., internally consistent) measures of a given content domain (scale) are needed, although are not alone sufficient, to determine whether or not the domain corresponds to a distinct factor (Aiken, 1996; Comrey, 1978; Taylor & Cox, 1998). Alpha can also be used as a measure of the strength of a dimension once the existence of a single factor has been determined (Cortina, 1993; Taylor, 1996). In addition, the internal consistency of an instrument or subscale provides evidence that a single construct is being assessed (Switzer, Wisniewski, Belle, Dew, & Schultz, 1999). The current study also found generally high corrected item-subscale total score correlations. This is another, more refined measure of coefficient alpha reliability that indexes the cohesiveness of the scale.

The correlational results of this study were generally quite similar to those of the Taylor and Cox study (1998). In both studies, there were large correlations between the
Anxiety sensitivity: Validity of the ASP subscales and the ASI total score. This provides additional support for construct validity. The significant, but modest correlation found for the ASP with the trait version of the STAI, a measure for anxiety proneness, is consistent with the modest correlational relationship found for the ASI in this study and others (Taylor, 1996). This provides additional support for the view that anxiety sensitivity and trait anxiety are correlated but distinct constructs (Taylor & Cox, 1998).

In Taylor and Cox (1998), a Pearson product-moment correlation was presented that includes the four ASP subscales that were identified as factors in factor analysis. The order of the four ASP subscale correlations with the ASI total score from lowest to highest was consistent in their study and in the current study. From lowest to highest in order they are: (1) respiratory fears, (2) cardiovascular fears, (3) gastrointestinal fears, and (4) cognitive dyscontrol fears. Possibly this order (i.e., lower correlations for respiratory, cardiovascular, and gastrointestinal fears) is due to the fact that in factor analysis of the ASI, the fears of physiological sensations have been identified as one factor (i.e., Cox, Taylor, Borger, Fuentes, & Ross, 1996; Peterson & Heilbronner, 1987; Stewart et al., 1997; Taylor, 1996; Telch et al., 1989; Wardle et al., 1990; Zinbarg et al., 1997), rather than three. Unlike the ASP, which has an adequate number of items to identify the respiratory, cardiovascular, and gastrointestinal factors, the ASI has only 2 items related to cardiovascular fears, one item related to respiratory fears, and 2 items related to gastrointestinal fears. As stated earlier, internal consistency reliability is a necessary part of factor identification, and it is impossible to compute with a one-item scale. Due to the fact that score ranges are so limited, the possible value of correlations
with other variables is reduced. In turn, validity coefficients are reduced (Betz, 1995). It is of interest to note that when the ASI was expanded to include more items assessing cardiovascular and respiratory fears (ASI-R; Taylor & Cox, 1998), they both became distinct factors.

Another finding that is consistent with the findings of Taylor and Cox (1998), but differs from previously mentioned analyses of the ASI, is additional evidence suggesting that the fear of physiological sensations consists of three distinct factors: fears of cardiac, respiratory, and gastrointestinal symptoms. The evidence consists of the large correlations of the ASP subscales for these three fears with the full ASI score. This provides proof of convergent validity with a well-documented measure of anxiety sensitivity (i.e., the ASI). To support this evidence, as stated earlier, convergent validity provided by large correlations of these subscales with the ASI, as well as discriminant validity provided by a modest correlation of these subscales with the trait portion of the STAI, provide evidence for construct validity of these subscales (Kazdin, 1998).

Also consistent with the Taylor and Cox study (1998) are the highest and lowest subscale correlations of the ASP and STAI. The highest correlation for both studies was between the fear of cognitive dyscontrol subscale and the STAI. Possibly this is due to the fact that trait anxiety, which is assessed by the trait version of the STAI, is now viewed as a higher-order or third-order construct indicative of an individual who tends to experience fear in response to stressors (Lilienfeld et al., 1993). It is probable that the fear of cognitive dyscontrol would be more highly correlated with the tendency to experience fear in response to stressors. Problems with focus and concentration that
occur with elevated anxiety would also be stressors. Anxiety sensitivity, on the other hand, is indicative of an individual who tends specifically to experience fear in response to his or her own anxiety symptoms. Individuals who tend to experience fear in response to stressors do not necessarily fear a rapid heart beat, a change in breathing, or the gastrointestinal distress that can be experienced with anxiety. It follows that fears which are specific to anxiety sensitivity (physiological fears), a separate construct, would have lower correlations with a measure of trait anxiety, as they do in the current study and in Taylor and Cox (1998).

As reported earlier, the fear of cognitive dyscontrol has also been found to have a significant correlation with depression in earlier studies, while physiological fears had a significant relationship only with anxiety sensitivity (Schmidt et al., 1997; Zinbarg et al., 2001). Zinbarg et al. (2001) assessed fear responses to hyperventilation and 5.5% carbon dioxide challenges based on a previous study (Rapee et al., 1992). They found that the strongest positive linear relation with panic-related phenomena among the 3 underlying factors of the ASI was shown by Anxiety Sensitivity-Physical Concerns. They also found that the strongest positive linear relation with depressed mood was shown by Anxiety Sensitivity-Mental Incapacitation (i.e., fear of cognitive dyscontrol). It would be interesting in future research to assess the effect of improvements in focus and concentration on other symptoms of depression and anxiety.

As for the correlations between the ASP subscales, with the exception of the high correlation between cardiovascular and respiratory fears in the current study, correlations between the four identified factors (i.e., cardiovascular, respiratory, gastrointestinal, and
cognitive dyscontrol fears) in Cox and Taylor (1998) and the same subscales in the current study were somewhat matched. These factors had relationships in the modest range in Taylor and Cox (1998) and occupied the lower end in the current study (i.e., modest to large range). These relationships tend to be fears that are not a fear of the same result. For example, the fear of death due to a heart attack (i.e., cardiovascular fear) or suffocation (respiratory fear) is not the same result as the fear of illness linked to gastrointestinal distress or the fear of mental incapacitation due to problems with concentration or focus.

Finally, the coefficients of determination in both the current study and in Taylor and Cox (1998) for the ASP and ASI total scores were consistent. In both studies almost half of the variance in the ASI total scores can be accounted for by variability in the ASP total scores. It is likely that this common variance consists in part of the anxiety sensitivity factor and some of the underlying factors (i.e., the fear of cognitive dyscontrol and the fear of observable anxiety symptoms in the ASI and the ASP, the fear of physical sensations in the ASI, and cardiovascular, respiratory, and gastrointestinal fears in the ASP). It is also possible that common method variance plays a role. This is often the case when different subjects are used, and all the measures completed by the same informant show a similar level of correlation (Kazdin, 1995).

On the other hand, slightly over half of the variability in the ASI total scores was unaccounted for by ASP total scores in both studies. A small part of this was accounted for by the variability in the STAI total scores. It is likely that another portion is accounted for by the relationships with the higher order personality dimensions.
Neuroticism (Eysenck, 1991) is moderately to highly related to the measures of anxiety sensitivity, according to studies reviewed by Lilienfeld (1999; Arrindell, 1993; Zinbarg & Barlow, 1996; Saviotti et al., 1991). A significant relationship has been found for the ASI and the higher order dimension of Constraint (Telegen, 1978, 1982; Telegen & Waller, 1994). Significant relationships have been found for the ASP and avoidance of harm, a lower order trait of Constraint (Van der Does et al., 2003) and anxiety sensitivity and Absorption, the tendency to become engrossed in sensory experiences (Lilienfeld, 1996; Tellegen & Atkinson, 1974). It is also likely that the differences found in the existence of underlying factors of the ASP and the ASI account for another portion of the variability (i.e., the identification of three specific physiological concerns in the ASP vs. one inclusive factor for physical concerns in the ASI; the identification of the dissociation/neurological concerns factor in the ASP).

Another possible cause is the difference in approaches that are used in the test instructions. In the ASI the instructions suggest that individuals answer on the basis of the way each person thinks he or she might feel if he or she had such an experience (i.e., conditional wordings for instructions and questions). In the ASP it is suggested that individuals answer on the basis of the likelihood that if each person experienced a sensation that it would lead to something bad happening to him or her. Also, as pointed out earlier, a few of the items in the ASI refer to anxiety symptoms in a general manner (e.g., It scares me when I am nervous; Cox, 1996). All of the items in the ASP are specific to one of the six domains identified in earlier studies of anxiety sensitivity.
Anxiety Sensitivity: Validity of the ASP (Taylor, 1996). And finally, the number of items was increased drastically from 16 to 60 (Van der Does et al., 2003).

In addition, in the coefficient of determination, there is a small difference between the current study and the Taylor and Cox study (1998) in the variability which was unaccounted for by the correlation between ASI and ASP total scores. It is possible that this difference is accounted for by the difference in the range of scores which is connected to the identification of the two factors, fear of observable anxiety symptoms and dissociative and neurological fears that were not identified in Cox and Taylor (1998). These two factors were in fact identified by Van der Does et al. (2003) in factor analysis of the ASP.

Divergent Findings

Two results were higher than those found in most studies that have utilized the ASI. As reported earlier, the mean score on the ASI is high in relationship to the mean score on the ASI in most studies (e.g., Blais et al., 2001; Stewart, et al., 2001; Taylor, 1995; Zinbarg, et al., 1997). This score is important in validating the range of this study’s clinical subjects. It is also possible that it could be an artifact of the high rate of comorbidity in this sample. This could also explain the somewhat higher mean total scores for the ASP in the current study in comparison with the mean total scores for the ASP in Van der Does et al., (2003). The second result, the correlation of the trait version of the STAI and the ASI in the current study, is also somewhat higher in comparison with
other studies (Taylor, 1996). It remains in the modest range, supporting the view that anxiety sensitivity and trait anxiety are correlated, but distinct constructs. It is also possible that this result, as well, is an artifact of the high rate of comorbidity in this sample. In fact, Otto et al. (1995), Peterson and Reiss (1987), Schmidt et al. (1997) and Taylor et al. (1996) have reported increased ASI scores in patients with major depression.

Two additional findings are of note. The two ASP subscales, that measured the fear of publicly observable symptoms and dissociative/neurological fears, had correlations with the ASI total score which were as strong as the other subscales. This provides evidence for convergent validity. In addition, the high Cronbach’s alpha for the fear of publicly observable symptoms subscale (.87) in this study diverges from two earlier studies (Blais et al., 2001; Nunnaly & Bernstein, 1994). Analysis with the ASI has provided evidence that this factor has low reliability (.62; Zinbarg & Barlow, 1996). However, evidence for high reliability is provided when this fear is adequately assessed with a 10-item scale as it was in this study with the ASP. Alpha is influenced by the number of items in a scale and characteristically increases as the number of items increases. The incremental improvements made by adding items to the scale may be relatively large up to about 10 items (Shrout & Yager, 1989; Switzer et al., 1999). And again, Cronbach’s alpha, a coefficient of internal consistency, is a widely used reliability statistic. As stated earlier, the recommended minimum is 0.80 and all the subscales of the ASP easily met this requirement (Loewenthal, 2001).

Factor analysis performed by Taylor and Cox (1998) did not reveal that these two subscales are factors. However, it was speculated that these two factors would be
revealed when factor analysis is performed on a clinical sample assessed with the ASP. Furthermore, it was speculated that this will be explained by the fact that the non-clinical population used by Taylor and Cox (1998) had a limited range of scores in comparison with the scores of a clinical sample. The current study has provided evidence for two criteria that are necessary for the identification of factors (i.e., internal consistency and convergent validity). Additional evidence to support this speculation has been provided in which these two subscales were identified as factors in factor analysis when the ASP was utilized with a clinical sample (Van der Does et al., 2003).

In further exploration of divergent findings regarding these two additional factors, the results showed that not only all of the ASP subscales had large correlations with the ASI, but the highest correlation in the current study involved the fear of publicly observable anxiety reactions. It is possible that when utilizing a clinical sample, an increase in cardiovascular, respiratory, gastrointestinal, and cognitive dyscontrol fears is accompanied by an increase in the fear that these anxiety reactions will be observable. In addition, in Taylor and Cox (1998), the three somatic ASP subscales (i.e., fear of cardiovascular, gastrointestinal and respiratory symptoms), that were identified as factors in their study, had somewhat lower correlations with the ASI total score in comparison with the current study. It is possible that the higher correlations for these subscales in the current study are the result of an increased range of scores. This increased range of scores also supports a stronger linear relationship.

When examining the correlational relationships between the ASP subscales, it is notable that in the current study the relationship between cardiovascular and respiratory
fears was very high and, in fact, had the highest relationship. This probably is due to the fact that of all the subscales, these are the only two that have the same feared result (i.e., death). In contrast, Taylor and Cox (1998) found a modest relationship for these two factors, as they did for the others. This is probably due to the fact that a nonclinical sample will not identify the same catastrophic outcome as often for a rapidly beating heart or a change in respiration. The other correlational relationships in the current study between the ASP subscales that occupied the higher end of the range consisted of relationships between the fears of publicly observable anxiety reactions or dissociative and neurological symptoms and the other subscales. It is probable that individuals with anxiety sensitivity experience the fear that their anxiety symptoms will be observed as they experience the fear of the anxiety symptoms. Therefore, there is a strong linear relationship between these fears. The strong relationships between dissociative/neurological fears, cardiovascular fears, and respiratory fears is probably due to the likelihood that dissociation and light headedness are more likely to be experienced with a higher level of autonomic arousal when the fear is more extreme (i.e., death). The one correlation with dissociative/neurological fears that lies in the modest range is with gastrointestinal fears. That is probably due to the likelihood that dissociation and light headedness are less likely to be experienced when the expected result is less extreme (i.e., illness) and autonomic arousal is not as high.

Finally, all of the six subscale relationships in the Van der Does et al. (2003) study, that utilized a clinical sample, were large correlations. Their sample was assessed at intake. It is likely that their subjects experienced, on average, a higher level of anxiety
Anxiety sensitivity symptoms than those in the current study because none had the benefits of treatment. Therefore, it is speculated that there was an increased tendency for a stronger linear relationship between the subscales that occupied the modest to lower end of the large range in the current study (i.e., an increased tendency for one symptom to occur as another symptom occurred due to co-occurrence of symptoms).

**Contributions**

The ASP was constructed by Taylor and Cox (1998) on the basis of their previous research on the measurement of anxiety sensitivity (Cox et al., 1996; Taylor et al., 1992). Each of its six 10-item scales assesses a domain of anxiety sensitivity suggested by previous studies (Taylor, 1996). The results of the current investigation provide evidence with a clinical sample that all six scales have high levels of internal consistency (Nunnally, 1978). As stated earlier, adequate measures (e.g., internal consistency) of a given content domain (scale) are needed to determine whether or not the domain corresponds to a distinct factor (Comrey, 1978). Internal consistency cannot be established on one of the ASI scales (i.e., respiratory fears) since there is only one item (Betz, 1995).

On the other hand, the ASI is a psychometrically well-documented measure of anxiety sensitivity (Zinbarg et al., 1997). Based on this fact, convergent validity for the domains of the ASP was provided by each domain’s significant correlation with the ASI. Evidence for internal consistency for the six ASP subscales and a large correlational
relationship for all six scales with a well-documented measure of anxiety sensitivity provide further evidence that the ASI contains too few items on some of the domains to provide sufficient identification of all anxiety sensitivity factors.

Theoretical Implications

This study has provided evidence of convergent and discriminant validity with both ASP total and subscale scores. This provides additional support for the view that the general factor underlying the ASP total and subscale scores primarily reflects a unified anxiety sensitivity construct rather than an artifact, or some other substantive construct, such as trait anxiety (Loewenthal, 2001; Taylor & Cox, 1998; Zinberg et al., 1997). It is important that this was accomplished with the use of measures that use similar instructions and response scales to rule out such methodological differences as alternative explanations for discriminant validity (Zinbarg et al., 1997). Again, as reported previously, it is interesting to note that the factor analysis performed by Van der Does et al. (2003) suggested that the ASP is unidimensional. Nevertheless, they suggested that there may be circumstances in which it is useful to distinguish the six dimensions as originally proposed by Taylor and Cox (1998). The confirmatory factor analysis performed by Van der Does et al. (2003) showed that this solution is defendable despite high intercorrelations among factors.

Therefore, it can be argued that factor analysis with the ASP has begun to provide full evidence to support multiple dimensions and hierarchic structure (Van der Does et
Anxiety Sensitivity: Validity of the ASP

This study also takes necessary steps in that direction by providing evidence for convergent and discriminant validity and high internal consistency when the ASP is utilized with a clinical sample. In addition to providing additional evidence for the anxiety sensitivity construct, it has also provided evidence for the identification of factors that are not identified by the ASI. In fact, it has provided evidence for six factors. This in turn adds to the evidence that raises doubt that AS meets the requirements of the second criterion for expectancy theory. According to this criterion, as long as anxiety sensitivity cannot be reduced to more basic fears, it can be regarded as fundamental. The findings of the current study support the view that in order for expectancy theory to explain the nature of anxiety sensitivity as a construct, it will need further development that takes into account this and other findings regarding more basic underlying fears (Taylor & Fedoroff, 1999).

Research Implications

It is speculated that results of applied research will become more consistent through the use of congruence (Cox, 1996; Rapee, 1994). It has been suggested in the interactional model proposed by Cox (1996) that physiological concerns and the fear of cognitive dyscontrol are specific risk factors for different anxiety reactions (e.g., heart palpitations, rapid breathing, diarrhea, and decreased concentration). It is speculated that construct validity and content validity (i.e., an adequate identification of the underlying domains) for anxiety sensitivity provided with the ASP will support the application of
congruency. Congruency means that anxiety sensitivity interacts with congruent triggers, but not with incongruent triggers, to produce catastrophic thoughts and related panic. For example, the fear of cardiac symptoms interacts with the fear of heart palpitations, but not with incongruent triggers such as derealization, to produce anxiety and panic (Cox, 1996). The use of congruence will come about as design and assessment strategies abandon the unidimensional perception of panic and increasingly adopt the multidimensional perspective. The prediction of an improvement in accuracy in panic disorder research can be tested with a psychometrically solid multidimensional measure of anxiety sensitivity (e.g., ASP; i.e., when there is improved accuracy in matching the symptoms caused by a provocation task and the symptoms a person fears, precision in predicting panic attacks will improve; Cox, 1996; Cox et al., 1999).

Applied research will also benefit from improved identification of the role that anxiety sensitivity plays in the other anxiety disorders (i.e., post traumatic stress disorder, social phobia, generalized anxiety disorder, obsessive-compulsive disorder, and specific phobia). It is speculated that construct validity for the underlying domains of the ASP supports its utilization in the identification of the domains that are related to the other anxiety disorders. This suggests that the ASP can be utilized to identify the domains underlying anxiety sensitivity that contribute to the association with the other anxiety disorders. For example, as reported earlier, some studies show that ASI scores are almost as elevated in post traumatic stress disorder as they are in panic disorder (Taylor et al., 1992; Taylor et al., 1999). However, the possibility that the fear of cognitive dyscontrol
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is the domain that is most highly associated with post traumatic stress disorder cannot be reliably supported due to the psychometric limitations of the ASI.

There are several other studies in which ASI scores have been found to be close to, or even higher in social phobia, than those for panic disorder (Asmundson & Stein, 1994; Ball et al., 1995; Hazen et al. 1995; Maidenberg et al., 1996; Marks et al., 1988). A different pattern of item responding has been suggested for social phobia, in comparison with that of panic disorder, with significantly higher scores on three items: (a) “It is important to me not to appear nervous,” (b) “It embarrasses me when my stomach growls,” and (c) “Other people notice when I feel shaky,” (Cox et al., 1999). It is possible in social phobia, that the individuals who experience these symptoms may fear publicly observable anxiety symptoms (e.g., sweating, blushing, trembling), if they believe that these symptoms have harmful social consequences (Cox et al., 1999). As reported earlier, it is important, in different clinical situations, to seek further clarification about the nature of anxiety sensitivity through item-level or dimensional analyses in order to settle such issues (Cox et al., 1999). Initial evidence now exists that the ASP has the psychometric properties for these procedures.

The diagnoses of generalized anxiety disorder and obsessive compulsive disorder are two areas that have received less attention in systematic research (e.g., item level comparisons, factor analysis). However, some studies have provided evidence for elevated anxiety sensitivity scores (Calamari et al., 1996; Taylor et al., 1992; Zeitlin & McNally, 1993). While it is not known whether or not unique ASI patterns of responding would also be found for these disorders, the fear of cognitive dyscontrol would probably
be most associated with these diagnoses. Assessment of these groups will benefit from utilization of the ASP, which has the addition of more cognitive dyscontrol items that are related to worry and intrusive thoughts and images (Cox et al., 1999).

Applied Implications

Research findings are more likely to be externally valid when the samples of individuals are representative of the population to which the results are to be generalized. That is more often true of correlational studies, which are more generalizable to real-life situations, than of experiments which involve greater control over extraneous variables (Aiken, 1996). By necessity, the sample used in this investigation was highly representative of the population with which the ASP can be utilized. Slightly less than a third were diagnosed as having a single anxiety disorder, one third had comorbid anxiety disorders, and somewhat more than a third had anxiety disorders comorbid with other Axis I disorders. Given this wide distribution, it is probable that utilization of the ASP is generalizable to a clinical population. It is speculated that evidence of construct validity for the domains has positive ramifications for prevention, treatment, and maintenance of treatment affects. Those at risk can be identified and given brief cognitive-behavioral therapy as a preventive intervention. With adequate assessment using the ASP subscale scores (Zinbarg et al., 1997), treatment can target congruent cognitions. Those with elevated anxiety sensitivity at the end of treatment can receive further interventions. These will be aimed specifically at the identified risk factors for symptom relapse. It has
been suggested that all of these were possible with the ASI (Zinbarg et al., 1997). However, with a psychometrically adequate number of items on each domain in the ASP (i.e., internally consistent), there is a significant increase in the possibility that each individual’s fears will be adequately identified. For example, assessment of clinical groups who experience the symptoms of obsessive compulsive disorder and generalized anxiety disorder are likely to benefit from the addition of more cognitive dyscontrol items that are related to worry and intrusive thoughts and images (Cox et al., 1999). Some studies, although not all, have provided evidence that targeting congruent catastrophic concerns improves treatment (Taylor, 1999). It is speculated that prevention, treatment, and maintenance of treatment effects will be more effective due to an increase in accurate and more thorough identification of each individual’s concerns.

Another indicator that supports the future effectiveness of the ASP with a clinical population was provided by the previously reported larger correlations between some of the ASP subscales in the Van der Does et al. (2003) study in comparison with the current study. Since their sample was assessed at intake, it is likely that their subjects experienced a higher level of anxiety sensitivity symptoms than those in the current study because none had the benefits of treatment. Therefore, a larger range of scores produced a stronger linear relationship in comparison with the current study, which contained individuals who had not had the benefit of treatment, were in treatment, or had completed treatment. Therefore, there is initial evidence that the ASP will accurately assess levels of anxiety sensitivity not only prior to treatment, but during treatment, and at completion of treatment, as well.
Finally, there are additional issues for generalizability (i.e., age, educational level, marital status, ethnicity, and employment status). Recent studies have indicated that population characteristics such as the respondent’s age, gender, education level, and ethnicity can affect responses to items. These may lead to under-endorsement or over-endorsement of items, biases in recalling events, and/or respondent difficulty in interpreting questions (Switzer, 1999). The current investigation utilized a sample that had a good age distribution of individuals from 19 to 65 years of age and good distributions regarding educational levels, marital status, and employment status. While the majority of participants were Caucasian, several other cultural/ethnic groups were represented. The nature of the sample, evidence for convergent and discriminant validity provided by the current investigation, and the care taken in assessment procedures (i.e., investigator present to provide support at all times during assessment) provide further support for the use of the ASP with a varied clinical population.

Limitations

For this type of study a sample of 105 subjects meets the minimum requirement. This presents limitations for generalizability since it limits the number of participants on some of the disorders. The nature of the sample (i.e., assessment at intake, during treatment, post-treatment, and no treatment) causes the scores to be somewhat inaccurate. That is to say that they do not provide an accurate reflection of each individual’s level of symptoms without the effects of treatment. However, the effect on the investigation’s
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overall results is probably minimal. Another issue of generalizability, comorbidity with other disorders, presents as a benefit for applied implications, but brings into question the responses of the subjects and presents also as a limitation for generalizability.

The use of computers to aid in recording responses to both interviews and self-administered questionnaires has become more prevalent, and seems to provide a reliable, valid, and highly efficient means of assessing some attributes (e.g., Brugha, Kaul, Dignon, Teather, & Willis, 1996; Dignon 1996; Erdman, Klein, Greist, & Skare, 1992; Kobak, Reynolds, & Greist, 1993; Steer, Rissmiller, Ranieri, & Beck, 1994; Switzer et al., 1999; Thornicroft 1994). However, diagnosis could be questioned due to the fact that a computerized version of the SCID-I/P was used. During the period of time in which this software was utilized, psychometric properties had not been assessed. Therefore it is possible that the use of this version increases the possibility of false positives and negatives. In an effort to correct for this issue, when a subject was in treatment and consented to a request for the chart diagnosis from the treatment provider, such a request was made. Due to a lack of interest by subjects and treatment providers, only 29 chart diagnoses were obtained. These were not enough to make a valid comparison between chart diagnoses and diagnoses suggested by the computerized version of the SCID-IP.

Regarding discriminant validity, two measures may have no conceptual connection or relationship, but still show significant and moderate-to-high correlations because of common method variance. If method variance plays a significant role, as is often the case when different subjects are used, then all the measures completed by the same informant may show a similar level of correlation. In that case, it can be difficult to
demonstrate discriminant validity (Kazdin, 1995). However, this was not the case because the STAI scores in the current study were not higher. Discriminant validity raises another issue, which is social desirability. It has been recommended (Campbell, 1960) that in validating a new measure, it should be correlated with measures of social desirability, intelligence, and acquiescence. Such correlations would show that these other constructs are not part of the new measure. These constructs have been shown to have a pervasive influence across several domains. Also, their own construct validity is relatively well established. Therefore, it is quite likely that they contribute to and occasionally account for other new measures (Kazdin, 1995).

**Suggestion**

It is suggested that the ASP be shortened. Respondents may be reluctant to complete a lengthy questionnaire, both because of the time involved and perceptions that they will be asked to give confidential or sensitive types of information. Groups receiving medical or psychiatric treatment, for example, depending on the nature or severity of their illnesses, may have more difficulty in completing certain types of assessments such as self-administered questionnaires (Switzer et al., 1999). Van der Does et al. (2003) evaluated a considerably shortened version consisting of 24 items. They used the rationale that each of the subscales has some items that are almost identical. For instance, the following 3 items belong to the respiratory fears subscale: (1) you feel like you’re not getting enough air; (2) you feel like you can’t breathe properly,
and (3) you feel out of breath even though you haven’t been exerting yourself. They also suggested that a factor consisting of these items may be regarded as a semantic cluster with little psychological significance. Because all items of the current 60-item ASP were found to have high intercorrelations, Van der Does et al. (2003) simply included the first four items of each subscale. They found that each of the six shortened subscales had very high correlations with its full-length version. In addition, the internal consistencies (i.e., Cronbach’s alpha) of the 4-item scales were good. Their short ASP consists of items 1 through 19, 21 through 24, and 27 of the 60-item ASP. The results of the corrected item-subscale total score correlations in the current study, which were generally high, suggested that no items were indicated for removal due to low correlations.

Suggestions for Future Studies

Analysis of internal consistency and correlations of the measure under investigation with other instruments and variables with which the measure is expected to have certain relationships provide evidence for construct validity (Aiken, 1996). To provide additional evidence of construct validity for the ASP and verification of the hierarchical structure of the ASP, the design used by Cox and Taylor (1998) needs to be replicated with clinical samples and the inclusion of a confirmatory factor analysis (Switzer et al., 1999). The extent of the variance attributable to the group factors and to the general factor in relationships with criterion variables can then be estimated. This can be accomplished with the use of analyses of partial variance. In treatment studies, the
ASP subscale scores that correspond to treatment group factors, as well as total scores, can be analyzed for relationships with criterion variables (e.g., ASI, BSQ, ACQ). These analyses will be important for predictive validity of the ASP, including panic provocation responses, clinical course, and treatment response, and will provide evidence of the clinical usefulness of the ASP subscales and total scores (Zinbarg et al., 1997). Of course, the shortened ASP and the ASI should also be compared in a single study (Van der Does et al., 2003), because it is considered the gold standard. The usefulness of the shortened ASP may be further evaluated by investigating the predictive power of ASP scores in different challenge paradigms (Van der Does et al., 2003).

When launching an investigation, decisions about instrumentation often take into consideration the utility of making comparisons across studies or with normative samples. If it is desirable to make such normative comparisons, it will be critical to utilize a measure that has been used extensively in other populations (Switzer, 1999). Taylor and Cox (1998) and Van der Does et al. (2003) have provided initial comparisons with normative samples for the ASP. Such investigations should be continued, as well as initiated with the shortened ASP.

It is important and interesting to continue the assessment of structural relationships among the dimensions of the DSM-IV anxiety and mood disorders and the dimensions of negative affect, the personality domains of Neuroticism, Absorption, Constraint and its underlying factor, avoidance of harm, trait anxiety, anxiety sensitivity, and depression. Research models which connect depression, anxiety disorders, negative and positive affect, and autonomic arousal (Brown et al., 1998), and anxiety sensitivity,
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panic, and depressed mood, (Zinbarg et al., 2001) have been proposed and supported with the results of research that use new research designs and statistical modeling techniques. The current study provides evidence, utilizing the ASP, for the relationship between trait anxiety, anxiety sensitivity, and six underlying factors. Future studies, that continue assessment of structural relationships, need to consider use of the ASP, since there is now evidence that it appears to be an instrument that is superior to the ASI in assessing the anxiety sensitivity construct.

Another important issue to consider is the cultural appropriateness of the instrument for the study population. Most instruments used in social and behavioral research are based on middle-class, Western European/North American assumptions, values, and norms, and may not be entirely appropriate for other cultural groups (Switzer, 1999). For example, many of the classic symptoms of schizophrenia as defined by the Diagnostic and Statistical Manual IV (DSM; American Psychiatric Association 1994; e.g., delusions, hallucinations, disorganized speech) are part of the religious ceremonies or daily spiritual experiences of many cultural groups (Eaton, 1980). Culture-bound assumptions may pervade virtually all mental and physical health instruments. The current study included several cultural/ethnic groups and procedural efforts supported the successful use of the ASP with the individuals of those groups. Nonetheless, it is important that pilot studies target cultural/ethnic groups with whom the ASP will be utilized in a clinical setting in order to ensure its successful use with those populations. If there are differences in the characteristics of the ASP when it is applied to new populations, there might be serious implications for construct validity. Divergent factor
structures or internal consistency coefficients would imply that the ASP is not equivalent across cultural groups. Differences among groups should be interpreted with caution. These can be addressed by researchers in a variety of ways. For example, it may be possible to identify a different measure of the anxiety sensitivity construct that operates similarly across the population groups of interest. Alternatively, items that are biased may be eliminated or altered (Strommel, Given, Given, Kalaian, Schulz, & McCorkle, 1993; Switzer, 1999).

Other areas of future research include the relationship of anxiety sensitivity and locus of control orientation (Bakker, Spinhoven, Van der Does, Van Balkom, Van Dyck, 2002) and continued questions about the overlap of anxiety sensitivity and pain, which has been identified in some samples (Reiss & Havercamp, 1996, 1997). In the area of etiology, if the ASP has indeed identified more accurately and fully the mechanisms involved in anxiety sensitivity, future studies can use this information to decide whether or not anxiety sensitivity factors are associated with specific patterns of environmental and genetic influence (Taylor & Cox, 1998). Adults over the age of 65 have rarely been included in research (Taylor, 1999). They were excluded from the current study due to the possible effect of physical health status on responses to items (i.e., symptoms may reflect medical status rather than emotional distress; Switzer et al., 1999). The ASP needs to be included in pilot studies to investigate its effectiveness with this population.
Conclusion

This study has provided initial evidence with a clinical sample that the ASP and its six domains have construct validity. This implies that the items in the ASP identify anxiety sensitivity and its underlying fears. It has provided necessary, although not sufficient evidence that the ASP total score identifies a factor. In addition, it has provided the same evidence that the six domains or subscales of the ASP also identify factors. These are important steps that support the concepts of multiple dimensions and hierarchic structure. It is speculated that these theoretical concepts will prove to be meaningful and important in practical applications, as well as in research, given the existing evidence for common factors across depression and anxiety disorders.
References


Attention and memory for threat in panic disorder. *Behavior Research and Therapy, 30*, 619-629.


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*Psychometrika, 16*, 297-334.


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INSTRUCTIONS. It is very important that you read these instructions carefully so that you will be able to answer the questions that follow. The purpose of this questionnaire is to measure your level of fear of anxiety-related sensations. There are many anxiety-related sensations, including the following: palpitations (pounding heart or accelerated heart rate), sweating, trembling, shortness of breath, chest pain or discomfort, nausea, dizziness, feelings of unreality, chills, and hot flashes. People differ in their fears of these sensations; some people have little or no fear, others have mild or moderately severe fears, while others have very strong fears.

Anxiety sensations are feared if a person believes that these sensations have bad consequences. For example, people are frightened of palpitations if they believe these sensations could lead to a heart attack. People are frightened of dizziness if they believe that this sensation could mean that they are going crazy. People are frightened of publicly observable anxiety reactions (e.g., blushing or trembling) if they believe these reactions could cause others to ridicule or reject them.

We would like you to do two things for each of the items on the following pages:

1. Imagine that you are experiencing the sensation. Try to imagine this as vividly as possible.

2. Using the scale provided, rate the likelihood that if YOU experienced the sensation, it would lead to something bad happening to you, such as dying, going crazy, losing control, or being ridiculed or rejected by others. There are no right or wrong answers, and all responses will remain anonymous. Please note: We are not assessing whether or not you experience these sensations as a result of being anxious. We want to assess whether you believe that anxiety-related sensations would lead to something bad happening to you.

Practice item:

Imagine that you're experiencing the following sensation. What is the likelihood that this sensation would LEAD to something BAD happening to YOU? Circle the number that best indicates your choice:

<table>
<thead>
<tr>
<th>likely</th>
<th>Not likely</th>
<th>Somewhat Likely</th>
<th>Extremely</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Your legs feel unsteady.</td>
<td>1 2 3 4 5 6 7</td>
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</tbody>
</table>

Before you answer the following questions, please place a checkmark here if you fully understand the instructions you have read: ________.

If you don't understand the instructions, please ask for clarification.
<table>
<thead>
<tr>
<th>Sensation</th>
<th>Not at all likely</th>
<th>Somewhat likely</th>
<th>Extremely likely</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Your heart is pounding</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
<td></td>
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<tr>
<td>2. Your thoughts seem slower than usual</td>
<td>1 2 3 4 5 6 7</td>
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<td></td>
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<tr>
<td>3. You feel like you can't take a deep breath</td>
<td>1 2 3 4 5 6 7</td>
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<tr>
<td>4. Your stomach is making loud noises</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
<td></td>
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<tr>
<td>5. You have tingling sensations in your hands</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
<td></td>
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<tr>
<td>6. You have pain in your chest</td>
<td>1 2 3 4 5 6 7</td>
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<tr>
<td>7. Your thoughts seem jumbled</td>
<td>1 2 3 4 5 6 7</td>
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<tr>
<td>8. Your heart is beating so loud that you can hear it</td>
<td>1 2 3 4 5 6 7</td>
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<td>9. You feel like you're in a fog</td>
<td>1 2 3 4 5 6 7</td>
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<td>10. Hot flushes sweep over you</td>
<td>1 2 3 4 5 6 7</td>
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<tr>
<td>11. You have diarrhea</td>
<td>1 2 3 4 5 6 7</td>
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<tr>
<td>12. You are &quot;jumpy&quot; or easily startled</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
<td></td>
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<tr>
<td>13. You keep getting distracted by unwanted thoughts</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
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<tr>
<td>14. Your heart beats rapidly</td>
<td>1 2 3 4 5 6 7</td>
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<tr>
<td>15. You feel like you're suffocating</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
<td></td>
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<tr>
<td>16. You have a knot in your stomach</td>
<td>1 2 3 4 5 6 7</td>
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<tr>
<td>17. You feel numb all over</td>
<td>1 2 3 4 5 6 7</td>
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<tr>
<td>18. Thoughts seem to race through your mind</td>
<td>1 2 3 4 5 6 7</td>
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<tr>
<td>19. You feel out of breath even though you haven't been exerting yourself</td>
<td>1 2 3 4 5 6 7</td>
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<tr>
<td>20. Your heart pounds in your ears</td>
<td>1 2 3 4 5 6 7</td>
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<tr>
<td>21. You feel like something is stuck in your throat</td>
<td>1 2 3 4 5 6 7</td>
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<tr>
<td>22. Your body feels strange or different in some way</td>
<td>1 2 3 4 5 6 7</td>
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<tr>
<td>23. Your face sweats even though you're not hot</td>
<td>1 2 3 4 5 6 7</td>
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<tr>
<td>24. Your voice quavers (trembles or sounds shaky)</td>
<td>1 2 3 4 5 6 7</td>
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</tbody>
</table>
What is the likelihood that this sensation would LEAD to something BAD happening to YOU?

<table>
<thead>
<tr>
<th></th>
<th>Not at all likely</th>
<th>Somewhat likely</th>
<th>Extremely likely</th>
</tr>
</thead>
<tbody>
<tr>
<td>25. You can't keep your mind on a task</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>26. You have difficulty swallowing</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>27. Your stomach aches</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>28. You have burning sensations in your chest (heartburn)</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>29. Familiar surroundings seem strange or unreal to you</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>30. You feel like you're choking</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>31. You feel your heartbeat pulsing in your neck</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>32. You are constipated</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>33. You feel faint or lightheaded</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>34. Your heart starts beating slower</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>35. You shiver even though you're not cold</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>36. You have trouble thinking clearly</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>37. You feel that there's a lump in your throat</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>38. You feel like you're about to vomit</td>
<td>1</td>
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<tr>
<td>39. You're awake but feel like you're in a daze</td>
<td>1</td>
<td>2</td>
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<tr>
<td>40. Your stomach is upset</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>41. You have trouble remembering things</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>42. Your heart beats erratically</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>43. You have tingling sensations in your lips</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>44. Your mind goes blank</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>45. Your throat feels tight</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>46. You feel &quot;spacey&quot; or spaced out</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>47. You feel like you're not getting enough air</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>48. Your face blushes red</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
What is the likelihood that this sensation would LEAD to something BAD happening to YOU?

<table>
<thead>
<tr>
<th></th>
<th>Not at all likely</th>
<th>Somewhat likely</th>
<th>Extremely likely</th>
</tr>
</thead>
<tbody>
<tr>
<td>49. You feel bloated (gassy)</td>
<td>1 2 3 4 5 6 7</td>
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<tr>
<td>50. You feel sick in your stomach (nausea)</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
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<tr>
<td>51. Your heart skips a beat</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
<td></td>
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<tr>
<td>52. Your face feels numb</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
<td></td>
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<tr>
<td>53. The muscles in your face twitch</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
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<tr>
<td>54. You are easily distracted</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
<td></td>
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<tr>
<td>55. Your chest feels tight</td>
<td>1 2 3 4 5 6 7</td>
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<tr>
<td>56. You have difficulty concentrating</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
<td></td>
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<tr>
<td>57. You have to urinate more frequently than usual</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
<td></td>
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<tr>
<td>58. Your hands are trembling</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
<td></td>
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<tr>
<td>59. You feel like you can't breathe properly</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
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<tr>
<td>60. You feel like things are spinning around you (vertigo)</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
<td></td>
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</tbody>
</table>