# ANXIETY SENSITIVITY: AN EXAMINATION OF THEORETICAL AND METHODOLOGICAL ISSUES

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Abstract — The construct of anxiety sensitivity (AS) has occupied an increasingly important place in theorizing and research on anxiety and anxiety disorders. Although a number of recent studies have provided support for the construct validity of the principal operationalization of AS, the Anxiety Sensitivity Index (ASI), the relation of the AS construct and the ASI to trait anxiety continues to be a source of controversy. Key issues in the AS-trait anxiety debate include the assimilative nature of traits and the concept of incremental validity. Recent research on AS lends some support to the claim that trait anxiety cannot fully account for AS findings. Important areas for future AS research include (1) demonstrating that AS is a risk factor for panic disorder and related conditions, rather than simply a consequence of these conditions, (2) developing and utilizing multiple operationalizations of constructs, (3) minimizing the impact of potentially inapplicable items, (4) testing for interactions between AS and other variables, and (5) testing hierarchical factor models that allow trait anxiety and AS to coexist as higher- and lower-order factors, respectively. Researchers in this area will need to develop alternative measures of the AS construct, recognize the distinction between different levels of trait specificity, clarify a number of theoretical issues relevant to the AS construct, and continue to subject predictions to stringent theoretical risks.

### INTRODUCTION

Anxiety is an emotion that is aversive for essentially all individuals. Nevertheless, some individuals are more disturbed by their anxiety than others: many people recover quickly from an episode of anxiety, whereas others spiral uncontrollably into a state of panic. The explanation for these individual differences has occupied theoreticians and researchers at least since Freud (Peterson & Reiss, 1987; Reiss, 1987). Nevertheless, there remains considerable disagreement concerning the nature and etiology of the "fear of anxiety"; i.e., the fear of one's own anxiety and anxiety-related symptoms (e.g., Jacob & Rapport, 1984; Ley, 1987; Reiss, 1991).

One of the earliest attempts to explain the fear of anxiety was made by Eysenck (1968, 1979), who invoked the concept of "incubation" to explain

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how anxiety can increase over time, even with repeated presentations of the nonreinforced conditioned stimulus (CS). According to Eysenck, if the CS is sufficiently aversive (i.e., "nocive"), the conditioned response (CR) can itself reinforce the CS or strengthen the unconditioned stimulus (UCS), leading to a positive feedback cycle in which anxiety escalates over time even in the absence of CS reinforcement. Eysenck hypothesized that incubation effects should be most pronounced in individuals with elevated neuroticism, introversion, or both, because such individuals would presumably be most likely to exhibit strong unconditioned responses (and thus strong CRs) to aversive stimuli. Although Eysenck's incubation theory has been criticized because of its lack of convincing empirical support (e.g., Kimmel, 1979; Mineka, 1979; Wolpe, 1979), it provided one of the first links between the fear of anxiety and individual differences in personality variables.

Goldstein and Chambless (1978) argued that "fear of fear" is a consequence of interoceptive classical conditioning of internal physical sensations (e.g., rapid heart beat, dizziness), which can then become a CS for the CR of anxiety and, in some cases, panic attacks (see also Jacob & Rapport, 1984; Turner, Beidel, & Jacob, 1988). According to Goldstein and Chambless, the fear of fear is typically a consequence, not a cause, of panic attacks. These authors and their colleagues (Chambless, Caputo, Bright, & Gallagher, 1984) developed two measures, the Body Sensations Ouestionnaire (BSO) and Agoraphobic Cognitions Questionnaire (ACQ), to assess the fear of fear typically observed in panic disorder patients following their attacks. In accord with Goldstein and Chambless's predictions, both of these indices have been found to distinguish panic disorder patients from normals (e.g., Chambless et al., 1984), although the specificity of the ACQ to panic disorder is unclear (Craske, Rachman, & Tallman, 1986). Despite its substantial heuristic value for the treatment of anxiety disorders (see McNally, 1990), Goldstein and Chambless's theory has been criticized on the grounds that it lacks strong empirical support and that it is does not clearly distinguish between CS and CR, or between UCS and UCR (Reiss, 1988; McNally, 1990).

An alternative (although probably not incompatible; see Chambless & Goldstein, 1988) explanation for the fear of anxiety has been proposed by cognitive theorists (e.g., Clark, 1986; Beck & Emery, 1985), who have argued that catastrophic misinterpretation of certain unexpected physical sensations, particularly those that can be exacerbated by anxiety (e.g., rapid heart beat), can lead to panic attacks. Specifically, these misinterpretations can lead to anxiety, which can in turn worsen the very sensations that triggered the misinterpretations. In predisposed individuals, this positive feedback cycle can escalate, culminating in a panic attack. Nevertheless, the cognitive model has been criticized on the basis of

findings that panic attacks often occur during sleep (Barlow, 1988) and that many panic disorder patients do not report catastrophic cognitions prior to or during their attacks (Rachman, Lopatka, & Levitt, 1988). Clark and others (e.g., Clark, 1988) have responded to these criticisms by conjecturing that catastrophic misinterpretations may be nonconscious in some cases. Nevertheless, this conjecture poses difficult, although perhaps not insurmountable (Cloitre, Shear, Cancienne, & Zeitlin, 1992), problems for the falsifiability of the cognitive model of panic disorder (McNally, 1990).

More recently, the construct of anxiety sensitivity (AS; Reiss & McNally, 1985; Reiss, Peterson, Gursky, & McNally, 1986), as well as the expectancy theory of anxiety within which it is embedded (see Reiss & McNally, 1985; Reiss, 1991, for discussions of the expectancy model), has emerged as a major new potential explanation for the fear of anxiety. Because AS has been posited to provide a novel explanation for panic disorder and related clinical phenomena, it appears to warrant a careful examination. Indeed, although AS research has been accelerating at a very rapid pace, there has never been a critical review of the AS literature by individuals who were not directly involved in the development and elaboration of the AS construct (cf. Reiss, 1991). Before proceeding, however, it might first be useful to briefly review the conceptual foundations of the AS construct.

### THEORETICAL UNDERPINNINGS OF AS

According to the developers of the Anxiety Sensitivity Index (ASI; Reiss et al., 1986), a 16-item self-report measure designed to assess individual differences in AS, AS refers to the extent to which individuals believe that anxiety or anxiety symptoms have adverse consequences. In other words, highly anxiety sensitive individuals possess cognitions that anxiety or anxiety-related sensations (e.g., rapid heart beat, fainting, trembling) portend dangerous or harmful outcomes. Thus, the proponents of the AS construct maintain that cognitive appraisal of events is causally primary in many panic attacks and similar phenomena. In this respect, the AS construct is consistent with cognitive models of panic, except that AS is hypothesized to be a stable individual difference variable that influences the cognitive interpretation of anxiety and anxiety-related symptoms in general. The AS construct is also similar in a number of ways to Ellis's (1979) concept of "discomfort anxiety", which refers to anxiety concerning one's own negative emotions, including anxiety itself.

Some proponents of the AS construct (e.g., Reiss, 1991) have argued that AS is *only one component* of the fear of anxiety; the other, anxiety expectancy, refers to the person's expectations that he or she will experience anxiety in a given situation. According to Reiss's expectancy theory, anxiety is due to both anxiety expectancy and AS (see Reiss, 1991). Nevertheless, at other times, the proponents of the AS construct have conceptualized AS as *identical* to the fear of anxiety (e.g., Reiss, 1988; Reiss, Peterson, & Gursky, 1988). These authors thus need to clarify the boundaries of the fear of anxiety construct.\*

How do individual differences in AS arise? According to the proponents of the AS construct, these differences can stem from a variety of sources, including social learning, information, history of panic attacks, a need to avoid embarrassment or illness, and physiological overreactivity (Reiss et al., 1986). In contrast to many previous theorists, these authors argue that AS is not necessarily a consequence of panic attacks, and that it may play a critical role in the etiology of these attacks. Thus, AS is posited by these authors to have important clinical implications, because highly anxiety-sensitive individuals are hypothesized to be at elevated risk for the development of panic disorder and several other disorders, including simple phobia (but see McNally, Taylor, Koch, & Louro, 1991; Taylor, Koch, & McNally, 1992), post-traumatic stress disorder (Taylor et al., 1992), and at least some subtypes of substance abuse (see Reiss, 1991).

### EVIDENCE FOR THE CONSTRUCT VALIDITY OF THE ASI

As noted earlier, the principal measure used to assess, and to test predictions derived from, the AS construct has been the ASI (Reiss et al., 1986). Much of the early research on the construct validity of the ASI has been reviewed elsewhere (McNally, 1990; Peterson & Reiss, 1987; Reiss, 1991). As these authors pointed out, a number of these studies appear to provide support for the ASI's construct validity.

For example, the ASI correlates highly to moderately highly with Chambless et al.'s (1984) ACQ and BSQ, which is consistent with the claim that the ASI assesses the fear of anxiety. The ASI has been found to be elevated in patients with panic disorder (e.g., Peterson & Reiss, 1987; Rapee, Ancis, & Barlow, 1987; Taylor et al., 1992; Telch, Lucas, & Nelson, 1989), and several other anxiety disorders, including posttraumatic stress disorder, social phobia, and obsessive-compulsive disorder (McNally, Luedke, Besyner, Peterson, Bohm, & Lips, 1987; McNally et al., 1991; Taylor et al., 1992). Moreover, the ASI is correlated with a history of prior panic attacks among college students (Donnell & McNally,

<sup>&#</sup>x27;McNally (personal communication, March 1992), one of the most active and prominent researchers in the field of AS, has recently informed us that he views the fear of anxiety as equivalent to AS alone, not to AS plus anxiety expectancy. Nevertheless, as other AS researchers (e.g., Reiss, 1988) appear to hold a dissenting view, this issue still appears to be in need of clarification.

1990). The ASI has been found to correlate with diagnostic severity among panic disorder patients, particularly at post-treatment (Jones & Barlow, 1991). Patients with mitral valve prolapse syndrome (MVPS) with concurrent panic disorder have higher ASI scores than MVPS patients without panic disorder (Lyons, Talano, Gitter, Martin, & Singer, 1986). In addition, the ASI scores of patients with panic disorder with agoraphobia have been found to decrease following cognitive-behavioral treatment (McNally & Lorenz, 1987). The ASI has also been found to predict the remission rate and severity of distress among panic disorder patients over 6month and 1-year intervals (Otto, Pollack, Sachs, & Rosenbaum, 1991).

There is also evidence that the ASI predicts increases in self-reported anxiety, and perhaps self-reported physical sensations related to anxiety, following challenge (i.e., potentially anxiety-provoking) procedures, such as hyperventilation (Donnell & McNally, 1989; Holloway & McNally, 1987; Rapee, Brown, Antony, & Barlow, 1992), mental arithmetic (Shostak & Peterson, 1990), and speaking about one's anxiety experiences (Maller & Reiss, 1987). In addition, Veltum and Goetsch (1991) found that the ASI predicts physiological responses (heart rate increases) following a challenge procedure (mental arithmetic). The results of these challenge studies are consistent with the claim that AS acts as an "amplifier" of preexisting anxiety, thereby resulting in differential reactivity of subjects to threatening stimuli (Reiss, 1991; Reiss & McNally, 1985). Moreover, because panic disorder patients tend to become more anxious than normals following challenge procedures (e.g., Liebowitz et al., 1984), these findings are consistent with the hypothesis that the ASI is a risk factor for subsequent panic disorder (Donnell & McNally, 1989).

Several recent studies, however, appear to raise potential questions concerning the ASI's construct validity. In a study of 294 college students, Craske and Krueger (1990) found that the ASI did not significantly predict the intensity of self-reported somatic sensations during sudden surges of arousal, either during waking or sleeping hours. This result is problematic if one assumes that AS leads individuals to misperceive their physiological sensations (e.g., overestimate their heart beat; but see Veltum & Goetsch, 1991). Nevertheless, if one assumes that AS leads individuals to view their anxiety sensations as threatening, but not to misperceive (i.e., incorrectly estimate) them, this finding may be supportive of the construct validity of the ASI. Craske and Krueger found that the ASI did, however, significantly predict the degree of anxiety during unexpected arousal surges.

Shostak and Peterson (1990) found no differences between high and low ASI scorers (college students) in electromyographic activity or systolic blood pressure following a potentially stressful mental arithmetic task. It could be argued (Shostak & Peterson, 1990), however, that the ASI should only be expected to predict anxiety, but not necessarily actual physiological symptoms, in response to potentially anxiety-provoking situations. For example, as noted above, high AS may predispose individuals to misperceive their anxiety-related physical sensations, which might in turn produce heightened anxiety. Nevertheless, these misperceptions may not necessarily produce increases in actual physiological symptoms. Alternatively, Shostak and Peterson's manipulation may not have been powerful enough to produce strong physiological reactions, particularly in normal subjects. Thus, the implications of their findings for the construct validity of the ASI are unclear.

Cox, Endler, and Swinson (1991) found that panickers receiving outpatient treatment scored higher on the ASI than did nonclinical panickers (i.e., untreated college students who had recently had at least one panic attack, as assessed by self-report; see Norton, Cox, & Malan, 1992, for a review of nonclinical panickers). Nevertheless, the ASI was a negative predictor of clinical status (i.e., it was associated with nonclinical, rather than clinical, panic) when entered in a multiple regression equation with several other predictors, including frequency of panic attacks and the extent of lifestyle restriction as a result of these attacks. Because Cox, Endler, and Swinson (1991) entered these variables in stepwise fashion, however, this finding may be unlikely to replicate (e.g., Cohen & Cohen, 1983). In addition, the ASI may have acquired a negative beta weight because of high correlations with the predictor variables (i.e., a suppressor effect).

Stewart, Knize, and Pihl (1992) found that the ASI failed to distinguish significantly between college students with vs. without a prior history of self-reported panic attacks. This result poses some difficulties for the claim that AS is a risk factor for the development of panic attacks (e.g., Reiss, 1991). Nevertheless, as the number of subjects with a history of panic attacks was relatively small (N = 22), this finding may have been a result of low statistical power. Moreover, at least one other investigator has reported higher ASI scores among college students with vs. without a history of panic attacks (Dorward, 1990). Thus, the negative findings of Stewart et al. (1992) require replication.

In summary, there is evidence that the ASI correlates in theoretically expected directions with a variety of external criteria. Although several investigators have recently reported negative findings with the ASI, the implications of these results for the construct validity of the ASI are unclear. In particular, it remains unclear whether the proponents of the AS construct predict that individuals with elevated AS should exhibit heightened physiological arousal (either self-reported or actual) to potentially anxiety-provoking stimuli, or simply heightened self-reported anxiety to these stimuli. The answer to this question appears to depend upon whether one hypothesizes that AS leads individuals to (1) simply interpret their anxiety-related symptoms as threatening (which presumably would lead to heightened self-reported anxiety in most cases), or also to (2) misperceive these symptoms, or (3) respond to these symptoms with elevated physiological symptoms of anxiety (or both). If hypothesis (1) is correct, then the findings of Craske and Krueger (1990) and Shostak and Peterson (1990) pose no difficulties for the construct validity of the ASI. If hypothesis (2) is correct, then the findings of Craske and Krueger (1990) raise questions concerning the construct validity of the ASI. Finally, if hypothesis (3) is correct, then the findings of both Craske and Krueger (1990) and Shostak and Peterson (1990) raise questions concerning the construct validity of the ASI. Clearly, further theoretical elaboration of the AS construct will be necessary before these three hypotheses can be better evaluated.

#### AS AND TRAIT ANXIETY

Central to both the construct validity of the ASI and the scientific status of the AS construct is the claim that AS is conceptually distinct from trait anxiety - i.e., the proneness to react anxiously to potentially anxietyprovoking stimuli. In other words, Reiss et al. assert that AS is a new construct that predicts findings not derivable from a more straightforward trait anxiety formulation of panic attacks (e.g., Ehlers et al., 1986). In addition, Reiss (1991, p. 147) has argued that the ASI is a "unique scale" that predicts clinical phenomena that extant trait anxiety measures do not. These contentions are important because panic attacks are found not only in panic disorder but in all anxiety disorders (Barlow, 1988), suggesting the possibility that elevated trait anxiety is a general risk factor for panic attacks. In addition, as noted above, Eysenck's (1968) model predicts that individuals who are high in neuroticism, which overlaps substantially with trait anxiety (Watson & Clark, 1984), should be especially prone to the incubation of anxiety and therefore to panic attacks (but see Kimmel, 1979: Mineka, 1979; Wolpe, 1979, for criticisms of Eysenck's model).\*

Moreover, most, if not all, of the construct validation studies cited earlier (see the section entitled "The Evidence for the Construct Validity of the ASI") are potentially vulnerable to the criticism that their results are attributable to trait anxiety (Lilienfeld, Jacob, & Turner, 1989). For example, patients with panic disorder, as well as those with other anxiety

<sup>&#</sup>x27;Interestingly, Reiss (1991) has recently stated that "the concept (of AS) has similarities to Eysenck's concept of neuroticism" (p. 142). As neuroticism overlaps substantially with trait anxiety (Watson & Clark, 1984), Reiss appears to acknowledge the conceptual and empirical overlap between AS and trait anxiety.

disorders, have elevated trait anxiety (Barlow, 1988), and many trait anxiety measures are sensitive to the effects of treatment for agoraphobia (Michelson, 1987). Thus, findings that AS (as assessed by the ASI) tends to be elevated in panic patients (e.g., Peterson & Reiss, 1987) and exhibits decreases in agoraphobics following cognitive-behavioral treatment (e.g., McNally & Lorenz, 1987) are also consistent with the hypothesis that the ASI is heavily saturated with trait anxiety. In addition, measures of general anxiety, like the ASI, predict outcome among patients with panic disorder (Otto et al., 1991).

Moreover, the findings of AS studies using challenge procedures (e.g., Donnell & McNally, 1989; Holloway & McNally, 1987) can, at least in principle, be explained by trait anxiety. These studies have typically demonstrated interactions between ASI level and threatening manipulations; i.e., high ASI subjects tend to exhibit greater increases in state anxiety following challenge compared with low ASI subjects. Although these results are consistent with an AS explanation, they appear to be equally consistent with a trait anxiety explanation. Traits, including trait anxiety, are inherently interactive constructs (e.g., Tellegen, 1981; in press) because they denote a propensity to react in characteristic ways given certain classes of stimuli. This notion can be traced to Allport (1937), who argued that "traits are often aroused in one type of situation and not in another; not all stimuli are equivalent in effectiveness. Successful adaptation and mastery require a trait to remain loose-knit, so that its determinative influence may be modified or checked according to special demands of the moment" (pp. 331-332). Thus, from Allport's perspective (also see Zuroff, 1986), a person possessing a high level of trait anxiety would not be expected to manifest high levels of state anxiety across all situations, but only in situations perceived as threatening. Consequently, the results of challenge procedures are consistent with both a trait anxiety and an AS explanation, because both trait anxiety (Spielberger, Gorsuch, & Luchene, 1970) and AS (Reiss, 1991; Reiss & McNally, 1985) have been hypothesized to produce differential reactivity of individuals to situations that are differentially anxiety-provoking (i.e., person-by-situation interactions).

This point is nicely illustrated in a study by Rappaport and Katkin (1972), who found that, relative to individuals with low trait anxiety (as assessed by the Taylor Manifest Anxiety Scale), individuals with high trait anxiety were no different in their rate of spontaneous skin conductance responses (SSCRs) at rest, but exhibited marked increases in their rate of SSCRs following a stressful task (a bogus "lie-detector" test). The authors concluded that "scores on the Manifest Anxiety Scale reflect 'reactive' anxiety, the autonomic components of which are differentially elicited by ego-involving stress situations" (p. 219). Several other researchers have

similarly reported greater increases in self-reported state anxiety for high trait anxiety subjects compared with low trait anxiety subjects, particularly when the manipulation is ego-threatening (e.g., Hodges, 1968; see Endler & Magnusson, 1976, for a review). In addition, there is some evidence that interactions between trait anxiety and threatening manipulations are especially likely when the trait anxiety measure and the manipulation are similar in content (e.g., a measure of physical danger anxiety and threat of painful electric shock; Kendall, 1978). Although not all researchers have found that trait anxiety is reactive in nature (see Watson & Clark, 1984, pp. 475–476, for a review of this literature and a discussion of possible reasons for negative findings), both the theoretical and empirical literature suggest that the results of challenge studies using the ASI are not necessarily inconsistent with a trait anxiety explanation.

Consequently, it is imperative for AS researchers to administer measures of trait anxiety in order to rule out the possibility that trait anxiety accounts for the findings of their studies. Nevertheless, because most of the investigations cited earlier (see the section entitled "Evidence for the Construct Validity of the ASI") apparently did not assess trait anxiety, this competing hypothesis cannot be convincingly excluded. The conceptual and empirical relation of AS and the ASI to trait anxiety therefore merits close scrutiny (Lilienfeld et al., 1989). Two issues appear to be especially relevant for evaluating Reiss et al.'s claims concerning the independence of AS from trait anxiety: the assimilative nature of traits and incremental validity.

# KEY ISSUES IN THE AS-TRAIT ANXIETY DEBATE The Assimilative Nature of Traits

Some authors have argued that invoking trait anxiety as an explanation for AS findings is inherently tautological. For example, Donnell and McNally (1990) argued that "the trait anxiety explanation for panic appears circular. That is, it 'explains' the tendency for people to experience anxiety attacks by invoking the tendency to experience anxiety in general (i.e., high trait anxiety)" (p. 84). They further asserted that "Although both trait anxiety and anxiety sensitivity are dispositional constructs, only the latter is embedded in a theory that explains why someone might panic in response to symptoms that are not inherently threatening" (p. 84). Put somewhat differently, it could be argued that, although the construct of trait anxiety denotes a general tendency to react anxiously to a large number of potentially anxiety-provoking stimuli, this construct cannot explain why certain individuals react with anxiety *specifically* to their own anxiety and anxiety-related sensations. McNally (1989), for example, asserted that "Unless one smuggles in the concept of anxiety sensitivity under the rubric of trait anxiety, there is no theoretical basis for predicting that people who respond with excessive fear to threatening stimuli in general should also respond with excessive fear to symptoms that are not *inherently* stressful" (p. 193; italics ours).

Nevertheless, these statements appear to neglect the contentions by a number of theorists that traits are "assimilative" in nature (e.g., Tellegen, in press; Wachtel, 1977). By assimilative, these theorists mean that traits influence how individuals construe and interpret stimuli. Thus, when Allport (1961, p. 347) stated that a trait has "the capacity to render many stimuli functionally equivalent", he intended to emphasize the tendency of traits to influence the interpretation of these stimuli. From this perspective, trait anxiety leads individuals to perceive a wide variety of situations as potentially threatening. With the likely exception of stimuli for which individuals are biologically "prepared" (Seligman, 1971; but see McNally, 1987, for a critical review), fear-provoking stimuli are not "inherently" threatening (cf. Donnell & McNally, 1990; McNally, 1989). Instead, trait anxiety substantially influences the extent to which ambiguous stimuli are interpreted as signifying threat. Beck and Emery (1985) have similarly pointed out that trait anxious individuals tend to overestimate the probability of danger in many aspects of their environment.

As an example of the assimilative nature of traits, Watson and Clark (1984) have conceptualized negative emotionality, a higher-order dimension that largely subsumes trait anxiety, as a propensity to construe minor hassles and annoyances as disastrous occurrences. In other words, according to Watson and Clark, one of the major ways in which the dimension of negative emotionality leads to chronic anxiety is by coloring individuals' interpretations of life events. From this perspective, invoking trait anxiety (or negative emotionality) as an explanation for AS findings is not tautological, and instead provides a potential explanation for why some individuals perceive their own anxiety and anxiety experiences to be more threatening than others.

In further support of the argument that trait anxiety has an assimilative quality, there is some evidence that individuals with high levels of trait anxiety are more likely than individuals with low levels of trait anxiety to interpret ambiguous stimuli as threatening (see Eysenck & Mathews, 1987, for a review). Haney (1973), for example, showed 110 subjects a series of slides with sentences that were ambiguous in meaning (e.g., "The index finger was placed on the tray"). Following each ambiguous sentence, subjects were shown a slide with two options (one nonthreatening, the other threatening), each of which would disambiguate the meaning of the sentence (e.g., "Finger: Pointing, Amputation"), and were asked to select the option that best matched their interpretation of the word. Haney found that subjects who were sensitizers on Byrne's (1961) Repression-

Sensitization Scale, which correlates highly with trait anxiety (Watson & Clark, 1984), were significantly more likely (r = .31 and .26 across two trials) to select the options linked to threatening or negative interpretations of the ambiguous sentences.

Eysenck, MacLeod, and Mathews (1987) presented 16 subjects with a series of homonyms (e.g., dye, die) delivered auditorily, and asked them to write down the first spelling of each homonym that came into their minds. Each homonym had both a nonthreatening and a threatening spelling and meaning. Moreover, half of the homonyms were relevant to physical health, and half to social and interpersonal difficulties. Eysenck et al. (1987) found that the correlation between trait anxiety, as assessed by the trait form of the State-Trait Anxiety Inventory (STAI; Spielberger et al., 1970), and the number of threatening interpretations of the homonyms was quite high (r = .60) and statistically significant. In contrast, the correlation between state anxiety, as assessed by the STAI, and threatening interpretations was low (r = .18) and nonsignificant. In addition, there was no significant effect for physically vs. socially threatening homonyms, suggesting that the assimilative effects of trait anxiety may be fairly pervasive.

Thus, there is suggestive evidence that individuals with elevated trait anxiety are likely to interpret ambiguous stimuli as portending threat, although it is clear that such evidence is relatively sparse. In addition, it is possible that these findings are due to differential familiarity of threat words in high vs. low trait anxiety subjects (Eysenck et al., 1987). Nevertheless, if the results of Haney (1973) and Eysenck et al. (1987) can be constructively replicated (Lykken, 1968), they would imply that many of the findings of AS research are consistent with a trait anxiety explanation. Specifically, individuals with elevated trait anxiety may tend to interpret ambiguous anxiety-related sensations (e.g., shortness of breath, rapid heart beat) as threatening, thus resulting in elevated AS. If so, the findings of Haney and Eysenck et al. would provide a link between trait anxiety and AS, because they would suggest that subjects with elevated trait anxiety tend to interpret a wide variety of ambiguous stimuli, including those relevant to their own anxiety symptoms, as portending danger. It is clear, however, that much further work will be necessary before the laboratory findings of Haney and Eysenck et al. can be generalized to the reactions of trait-anxious individuals to real world stimuli.

### Incremental Validity

A second major issue in the AS literature that, until recently, has remained largely unresolved is whether the findings of studies using

the ASI can be more parsimoniously explained by trait anxiety (Jacob & Lilienfeld, 1991; Lilienfeld et al., 1989). As noted earlier, a number of findings cited as providing evidence for the construct validity of the ASI (e.g., Reiss, 1991) might be explained by positing that the ASI is contaminated by trait anxiety. It is important to emphasize that the critical question is not whether AS is *equivalent* to trait anxiety, a point that seems to have been a source of persistent confusion in the literature. Reiss (1991), for example, presented evidence that correlations between the ASI and trait anxiety measures are far below unity and concluded that "These numbers are nowhere near the levels needed to support Lilienfeld et al.'s hypothesis that anxiety sensitivity is trait anxiety" (p. 146). Similarly, McNally (1990) asserted that "one cannot claim that trait anxiety and anxiety sensitivity are indistinguishable — and defend a distinction between trait and state anxiety — when the correlation between measures of trait anxiety and anxiety sensitivity is lower than the correlation between trait anxiety and state anxiety" (p. 408).

Nevertheless, the distinguishability of AS and trait anxiety has never been in question. For example, Lilienfeld et al. (1989) noted that the correlations between the ASI and trait anxiety measures, although consistently positive, are generally low to moderate (rs ranged from .3 to .5 in most studies). Moreover, they pointed out that "the ASI contains reliable variance unrelated to conventional trait anxiety measures" (p. 101) and that the results of the study by Holloway and McNally (1987) "suggest that the ASI may be contaminated by trait anxiety" (p. 102).\* Instead, the key question is whether the variance shared by the ASI and trait anxiety measures is the same variance accounting for the findings of AS research; i.e., the ASI may not contribute additional (i.e., incremental, Meehl, 1959) information over and above trait anxiety measures, at least in the studies conducted by the proponents of AS. Thus, although the ASI possesses variance not shared with trait anxiety indices, it must be demonstrated that this unshared variance relates to the phenomena of interest to AS researchers.

AS researchers have made a number of efforts to deal with the issue of incremental validity relative to trait anxiety measures. Reiss et al. (1986), in the first of such attempts, reported that the ASI shares variance with Geer's (1965) Fear Survey Schedule-II, a measure of common fears, over and above a measure of trait anxiety (the Taylor Manifest Anxiety

<sup>&</sup>quot;Nevertheless, Lilienfeld et al. (1989) may have unwittingly produced some confusion in the literature by their statement that "the results of these studies (on the ASI) can be equally accounted for by positing that the ASI measures trait anxiety" (p. 101). A more judicious wording of this statement would have read "... by positing that the ASI is in part a measure of trait anxiety".

Scale; Taylor, 1953) and a measure of the frequency of the same anxiety symptoms assessed by the ASI. The finding that the ASI contributes to the prediction of fears over and above trait anxiety measures has since been replicated by several investigators (see Reiss, 1991). Nevertheless, as noted earlier, many early studies using the ASI failed to include or report data on measures of trait anxiety (Lilienfeld et al., 1989). More recently, a number of AS researchers have apparently anticipated, and in a number of cases responded to, the criticisms of Lilienfeld et al. and others by including indices of trait anxiety in their investigations.

The results of four studies utilizing challenge procedures provide some support for the incremental validity of the ASI relative to trait anxiety indices. McNally (1989) reported that, in the Holloway and McNally (1987) study, the ASI predicted posthyperventilation anxiety and hyperventilation sensations significantly better than did the trait form of the STAI. McNally's analyses effectively addressed some of the criticisms of Holloway and McNally's findings (e.g., Lilienfeld et al., 1989).

Prassas, Jones, and Barlow (1990) found that the correlation between the ASI and a measure of state anxiety (the Profile of Mood States; McNair, Lorr, & Droppelman, 1971) administered after various challenge procedures (e.g., hyperventilation, chair-spinning) increased after partialling out trait anxiety. This important finding raises the possibility that, contrary to the conjecture of Lilienfeld et al. (1989), the variance that the ASI shares with trait anxiety measures may not be the same variance accounting for AS findings. If this result can be replicated, it would suggest that the developers of the ASI could improve their instrument's construct validity by *reducing* its saturation with trait anxiety (or by employing a trait anxiety measure conjointly with the ASI as a suppressor variable; e.g., see Cohen & Cohen, 1983).

As noted earlier, Shostak and Peterson (1990) found that the ASI did not significantly predict the intensity of college students' physiological sensations following a mental arithmetic task. Nevertheless, the authors found that the ASI, in contrast to the trait form of the STAI, significantly predicted posttest levels of state anxiety. Their analyses, however, do not permit the reader to determine whether the ASI predicted posttest anxiety significantly better than did the STAI, because tests for the significance of the difference between dependent correlations (Cohen & Cohen, 1983, pp. 56–57; Steiger, 1980) were not performed, and several relevant correlations were not reported.

Finally, Rapee et al. (1992) reported that, in a combined sample of anxiety disorder patients (N = 198) and nonanxious comparison subjects (N = 25), the ASI was the best predictor of the degree of panic or fear in response to both hyperventilation and carbon dioxide inhalation. The ASI was administered along with three other measures: the Self Analysis

Questionnaire (Lovibond, 1983; cited in Rapee et al., 1992), a measure containing subscales for anxiety, tension, and depression, the Social Interaction Anxiety Scale (Mattick & Clarke, 1988; cited in Rapee et al., 1992), a measure of social anxiety, and the Hamilton Anxiety Scale (Hamilton, 1959). Rapee et al. conducted stepwise multiple regressions utilizing all four measures, and found that the ASI entered the equation first (and was the only significant predictor) when response to both challenge procedures were the dependent variables. Nevertheless, because Rapee et al. did not conduct tests of the significance of the difference between dependent correlations (Cohen & Cohen, 1983; Steiger, 1980), it cannot be ascertained whether the ASI was a significantly better predictor of response to challenge compared with the other three measures.

Several other studies are also relevant to the issue of incremental validity. Taylor, Koch, and Crockett (1991) administered the ASI and the trait form of the STAI to 93 psychiatric outpatients and 142 undergraduates with spider phobia. Oblique principal component analyses of these measures suggested a two-component solution in both samples. Although the two components were moderately correlated in both samples (r = .39), with only a few exceptions the items of the STAI and ASI loaded on different factors. As the authors pointed out, their findings suggest that the ASI and STAI measure overlapping but separable constructs. In addition, Taylor et al. reported that the ASI significantly differentiated panic disorder from other anxiety disorders, whereas the STAI did not. Finally, they found that the ASI items that were most discriminating were those assessing interpretations of physical sensations; more will be said about this finding later.

Brown and Cash (1990) reported that panic patients and non-panic patients did not differ in their ASI scores after their trait scores on the STAI were controlled for statistically. This finding cannot be readily attributed to low statistical power, because Brown and Cash's sample size (N = 188) was large. Thus, Brown and Cash's results suggest that further investigation of the incremental validity of the ASI relative to trait anxiety measures is warranted (but see Taylor et al., 1992, for evidence that the ASI correlates with panic disorder and several other anxiety disorders even after covarying out scores on the trait form of the STAI).

Cox, Endler, Norton, and Swinson (1991) found that 50% of subjects with high ASI scores reported a history of panic attacks within the previous year; this compared with 20% and 11.1% of medium and low AS subjects, respectively. In addition, they reported that 41.5% of subjects with high ASI scores but no history of recent panic attacks scored one standard deviation above the mean or more on the Physical Danger anxiety subscale of the Endler Multidimensional Anxiety Scales (Endler, Edwards, & Vitelli, 1991), which they utilized as a measure of trait anxiety.

Although the authors concluded that their findings were consistent with the possibility that AS findings can be accounted for by trait anxiety, they did not examine whether the Physical Danger anxiety subscale was a better predictor of panic attack history compared with the ASI. In addition, they did not present a justification for interpreting this subscale as a measure of trait anxiety. Self-report indices of physical harm avoidance tend to have low correlations with measures of trait anxiety (Watson & Clark, 1984), and to load on a factor of fearfulness or constraint (Tellegen, 1978/82), rather than negative emotionality (which largely subsumes trait anxiety; Watson & Clark, 1984).

Otto et al. (1992) examined the relation between the ASI and various measures of hypochondriacal concerns in a sample of 50 panic disorder patients. They reported that the ASI was significantly correlated with five of seven illness attitudes scales (the seven correlations ranged from .18 to .49), which is consistent with the observation that hypochondriacal individuals are frightened of their own physical symptoms. Moreover, the ASI contributed significantly to the prediction of four of these five scales after the effects of both trait anxiety and trait depression (as assessed by the Beck Anxiety and Depression scales; Beck, Epstein, Brown, & Steer, 1988; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) were controlled for statistically using hierarchical multiple regression techniques.

Silverman, Fleisig, Rabian, and Peterson (1991) administered a children's version of the ASI (the Childhood Anxiety Sensitivity Index; CASI) to 76 nonclinical schoolchildren, along with three other measures: the Fear Survey Schedule for Children-Revised (FSSC-R; Ollendick, 1983), the trait form of the State-Trait Anxiety Inventory for Children (STAIC; Spielberger, 1973), and a measure of anxiety frequency containing the same stimulus items as the ASI (see Reiss, 1986). By means of hierarchical multiple regression techniques, Silverman et al. found that the CASI contributed significantly to the prediction of scores on the FSSC-R over and above the anxiety frequency measure, suggesting that the CASI assesses more than anxiety symptoms per se. Nevertheless, they did not examine the incremental validity of the CASI relative to the trait form of the STAI-C for predicting FSSC-R scores, although they reported that the correlation between the CASI and STAI-C was high (r = .62 to .64, depending upon the time interval between administrations). In a second study, Silverman et al. administered the same set of measures to a sample of 33 children with a variety of psychiatric diagnoses. They again found that the CASI contributed significantly to the prediction of FSSC-R scores over and above the anxiety frequency measure. Nevertheless, because of small sample size (only 18 children completed the STAI-C), the incremental validity of the CASI relative to the STAI-C was not examined. Again, however, the correlation between the CASI and STAI-C was quite

high (r = .62 to .72). Thus, Silverman et al.'s study is inconclusive regarding the incremental validity of the CASI relative to trait anxiety indices.

Finally, in a study of college students, Maller and Reiss (1992) found that ASI scores were significantly better predictors of panic attacks during the preceding year (as assessed by the PAQ) than were either the trait or state form of the STAI. This finding, like that of Donnell and McNally (1990), provides evidence for the postdictive validity of the ASI, and extends the results of the Donnell and McNally study by demonstrating that the ASI possesses incremental validity in the postdiction of panic attacks relative to trait and state anxiety indices.

In summary, there appears to be support for the contention that at least some of the findings of research on the AS construct cannot be fully accounted for by trait anxiety (also see Chambless & Graceley, 1989, for evidence that the ACQ and BSQ contribute to the prediction of panic disorder patients' self-reported avoidance behavior over and above a measure of trait anxiety), although the results of several studies (e.g., Brown & Cash, 1990) suggest that further research on this issue is necessary. In addition, because many of the earlier studies using the ASI did not include measures of trait anxiety, it is not clear whether their results can be attributed to AS per se. Moreover, some researchers continue to administer the ASI without measures of trait anxiety, or neglect to examine the extent to which findings using the ASI are potentially attributable to trait anxiety. Thus, investigators in this area will need to further examine the ASI's incremental validity relative to trait anxiety indices for a variety of criteria relevant to panic disorder and other anxiety disorders.

# IMPORTANT AREAS FOR FUTURE AS RESEARCH

Based upon our review of the AS literature, and upon developments in areas such as psychometrics and personality assessment, we have identified a number of important areas for future AS research. We believe that close attention to these issues will be needed to provide stronger support for the AS construct and for the construct validity of the ASI. More broadly, we believe that research on these issues may hold important implications for the conceptualization of both anxiety and anxiety disorders.

# Demonstrating that AS is a Risk Factor for Panic Disorder and Related Conditions

One of the central assumptions made by the proponents of the AS construct is that AS is a *risk factor* for panic disorder and related conditions, and is not simply a consequence of these conditions. This

is a key point because it is well known that following panic attacks, individuals tend to become hypersensitive to a wide variety of physical sensations (Goldstein & Chambless, 1982; Jacob & Rapport, 1984; Turner et al., 1988). Thus, such individuals might obtain high scores on the ASI solely as a result of this postpanic hypersensitivity.

Goldstein and Chambless (1982), for example, noted that "Having suffered one or more panic attacks, these people (panic disorder patients) become hyperalert to their sensations and interpret feelings of mild to moderate anxiety as signs of oncoming panic attacks . . ." (p. 55). Consistent with this observation, and contrary to the hypothesis that AS leads to increased misperception of physical symptoms (see the section entitled "Evidence for the Construct Validity of the ASI"), Veltum and Goetsch (1991) found that college students who were high AS scorers were more accurate than low AS scorers at heart rate estimation during a mental arithmetic task, although they found no differences between high and low AS scorers on this variable during caffeine ingestion. Similarly, Sarason (1979) pointed out that stress frequently leads to increased self-focus and self-preoccupation. This self-focused attention could, in turn, augment negative affect and anxiety in some individuals (Gibbons et al., 1985), leading to further increases in self-focused attention (also see Ingram, 1990). Thus, it is imperative for AS researchers to demonstrate that AS (1) precedes the development of panic attacks, because it might be argued that AS is simply a consequence of these attacks (also see McNally, 1990, p. 408) and (2) is a risk factor for the development of panic attacks, because it might be argued that elevated ASI scores observed in individuals prior to their first panic attack are not predictive of future attacks.

Donnell and McNally (1990) have taken an important step in addressing the first issue above by demonstrating that a large number of college students with high scores on the ASI have never experienced a spontaneous (i.e., unexpected) panic attack. Specifically, two-thirds of their sample who scored above the mean on the ASI had no prior history of spontaneous panic attacks, as assessed by a self-report measure (the Panic Attack Questionnaire or PAQ; Norton, Dorward, & Cox, 1986). This finding suggests that high AS may, in at least some cases, occur in the absence of a history of spontaneous panic attacks or panic disorder. This conclusion is further strengthened by findings that the PAQ may overestimate the frequency of spontaneous panic attacks (Brown & Cash, 1989), suggesting that even more than two-thirds of the high ASI scorers in Donnell and McNally's sample may have had no history of spontaneous panic attacks. Furthermore, Donnell and McNally (1989) found that high scores on the ASI were predictive of anxiety responses to hyperventilation even among subjects with no prior history of spontaneous panic attacks. In addition, a history of spontaneous panic attacks was only associated with anxiety

responses to hyperventilation among subjects with elevated ASI scores. Again, these results are consistent with the possibility that AS is risk factor for panic disorder.

Nevertheless, because Donnell and McNally assessed only spontaneous panic attacks in both studies, the possibility that many students with high ASI scores had a prior history of *non*spontaneous panic attacks cannot be excluded. These nonspontaneous panic attacks could, in turn, have led to the high levels of somatic preoccupation typically found among individuals with high AS. Thus, it will be important for AS researchers to demonstrate that high scores on the ASI can be found among individuals who have experienced neither spontaneous nor nonspontaneous panic attacks. In addition, because Donnell and McNally did not assess "limited symptom attacks" (i.e., panic-like experiences that fall short of the threshold for panic attacks; American Psychiatric Association, 1987, p. 238) in either study, it is possible that the elevated ASI scores of some subjects were a consequence of such attacks.

Several other investigators have addressed the issue of whether AS is a risk factor for panic disorder. In a longitudinal investigation of 100 adult patients with panic disorder, Pollack et al. (1990) found that patients who reported a history of childhood anxiety disorders (as assessed by retrospective structured psychiatric interviews) had significantly higher scores on the ASI compared with patients without such a history. Although the authors asserted that this result suggests "that elevated sensitivity to anxiety may be a trait manifestation of an anxiety diathesis and not just secondary to the experience of panic" (p. 15), a number of other plausible explanations are available. For example, elevated ASI scores in adulthood may, in some cases, reflect residual symptoms of a childhood anxiety disorder. Alternatively, adult panic disorder patients with a history of childhood anxiety disorders may be more severely affected than adult panic disorder patients without such a history. This difference in severity could in turn be reflected in elevated ASI scores. Nevertheless, as Pollack et al. (1990) did not control statistically for panic disorder severity, this possibility cannot be evaluated. In addition, Pollack et al.'s study is not directly relevant to the possibility that individuals with high ASI scores, but with no history of anxiety disorders, are at elevated risk for subsequent panic attacks or panic disorder.

Taylor and Rachman (1992) utilized causal modeling techniques to examine the correlations between a measure of AS (composed of items from the ASI, ACQ, and BSQ) and self-report indices of agoraphobic fears and agoraphobic avoidance in a sample of 330 college students. The authors found a satisfactory fit for their proposed model, in which AS led to both agoraphobic fears and agoraphobic avoidance. They reported that "exploratory analyses of plausible alternative models . . . failed to improve the goodness-of-fit" (p. 23), but did not specify what alternative models were tested. Because Taylor and Rachman did not assess panic attacks, a model in which panic attacks lead to agoraphobic fears, agoraphobic avoidance, and elevated AS could not be tested.

Only one study is directly relevant to the possibility that the ASI is a risk factor for panic attacks among subjects with no panic attack history. Maller and Reiss (1992) reported the results of a three-year follow-up of 151 college students assessed on the ASI. Of four subjects who experienced panic attacks (as assessed by the Anxiety Disorders Interview Schedule—Revised; DiNardo et al., 1985) for the first time during the three-year interval, three had high ASI scores at initial testing, whereas only one had a low score on the ASI at initial testing. Maller and Reiss's findings are consistent with the hypothesis that the ASI is a risk factor for initial panic attacks, although the small number of subjects in each group precludes clear-cut conclusions.

Thus, further prospective longitudinal studies of individuals with no history of panic attacks or panic disorder will be necessary to substantiate the claim that elevated AS is a risk factor for these clinical phenomena. In addition, studies of identical twins discordant for panic attack history would be extremely informative vis-à-vis the assertion that elevated AS is not simply a consequence of panic attacks; if the nonaffected co-twins of probands with a history of panic attacks were consistently found to have elevated AS, this would exclude the possibility that elevated AS results only from these attacks.

### Multiple Operationalization of Constructs

A persisting shortcoming in the AS literature is the excessive reliance upon single measures of constructs such as AS and trait anxiety, which appear to be complex and multifaceted. AS, for example, is presumably manifested in cognitive and affective domains (e.g., see Reiss et al., 1986) and has been found by several investigators to be multifactorial, at least at the lower-order level (e.g., Telch, Shermis, & Lucas, 1989, but see Taylor, Koch, McNally, & Crockett, 1992). Carrying the theoretical weight of a construct as complex as AS seems too heavy a burden to place on a single measure such as the ASI.

The same is true for measures of trait anxiety: virtually all studies of AS have employed the STAI or, in few cases, a similar measure, as the sole indicator of this construct. Trait anxiety, however, appears to be a highly multidimensional construct (Cox, Endler, Norton, & Swinson, 1991; Endler, Parker, Bagby, & Cox, 1991; Schalling, 1978) that may not be assessed adequately with brief measures such as the STAI. Indeed, evidence suggests that the trait portion of the STAI is largely or entirely

unidimensional (Kendall, Finch, Auerbach, Hooke, & Mikulka, 1976). The STAI and similar measures, for example, may provide inadequate coverage of somatic anxiety and muscle tension (e.g., Schalling, 1978), as well as of physical anxiety (Kendall, 1978), all of which appear to be important facets of trait anxiety. Thus, studies of the incremental validity of the ASI would benefit from the inclusion of trait anxiety measures that assess components of anxiety not adequately tapped by the STAI.

More broadly, overreliance upon single measures is problematic for theory testing, because two measures of the same construct may exhibit differential correlations with a criterion simply because one of them possesses superior psychometric properties (e.g., higher reliability). For example, it is conceivable, at least in principle, that the ASI possesses incremental validity relative to certain trait anxiety measures because it is more heavily saturated with trait anxiety than these measures. Multiple operationalizations of constructs have generally been demonstrated to produce increases in construct validity (Cole, Howard, & Maxwell, 1981), and we see no reason why this principle should not apply to AS and trait anxiety. Thus, we believe that it is incumbent upon researchers to utilize multiple measures of trait anxiety, and to develop additional measures of AS.

For example, Lilienfeld and Jones (in preparation) have developed an index of AS that, unlike most of the items on the ASI, explicitly assesses the cognitive component of this construct. Specifically, this measure asks respondents to estimate the probability that a given experience (e.g., rapid heart beat) presages harmful or dangerous consequences for them. This measure has the advantages of eliminating potentially inapplicable items (see the following section — "Minimizing the Impact of Potentially Inapplicable Items") and of providing a more direct measure of the cognitions posited by Reiss et al. (1986) to be central to the AS construct (see "Discussion"). Development of alternative measures of AS should permit multiple independent tests of this construct, and therefore subject this construct to stronger theoretical risks (Meehl, 1978).

### Minimizing the Impact of Potentially Inapplicable Items

One concern we have with the ASI is that some of its items may be essentially inapplicable to certain respondents. For example, items such as "It scares me when I feel faint" or "Other people notice when I feel shaky" may be largely or entirely irrelevant to subjects who rarely if ever feel faint or shaky. Such "double-barreled" items are potentially problematic, because some subjects may respond "No" to them because they never or virtually never have had the experience in question. This could produce a spurious correlation between the ASI and panic disorder (as well as similar criteria), because panic disorder patients are more likely than other subjects to experience anxiety-related symptoms, such as faintness and shakiness. Indeed, because many of the items on the ASI refer explicitly to panic symptoms, it may be this shared content, rather than the AS construct per se, that is primarily responsible for the ASI's relation to panic disorder and related criteria.

Taylor et al.'s (1991) finding that the ASI items that best discriminated panic disorder from other anxiety disorders were those involving fears of bodily sensations (e.g., feeling shaky, having a rapid heart beat) is consistent with this possibility. The authors concluded that "Fears that are directly related to the catastrophic misinterpretation of bodily sensations appear to be particularly important in panic disorder" (p. 309). Nevertheless, because panic disorder is defined primarily by physical sensations (American Psychiatric Association, 1987), Taylor et al.'s results may be a largely tautological consequence of the tendency of panic disorder patients to endorse items assessing their own symptoms. Taylor et al.'s finding that the ASI items not assessing physical sensations generally showed poor discrimination between panic disorder and other anxiety disorders is also consistent with this hypothesis, because there seems to be no theoretical reason to expect these items to be less valid indicators of the AS construct.

Jones and Barlow (1991) similarly reported that the ASI items that best discriminated patients with panic disorder, generalized anxiety disorder (GAD), social phobia, and somatoform disorders from one another were those items characteristic of the major clinical features of each category. For example, ASI items tapping "fears of heart/lung failure", which is a major concern of panic disorder patients, were more often endorsed by panic disorder patients than other patients, whereas ASI items tapping "stomach failure", which is not typically a major concern of panic disorder patients, were not. Although the authors interpreted their findings as supportive of the construct validity of the ASI, we believe that these findings raise questions regarding the ASI's construct validity.

As Nichols, Licht, and Pearl (1982) pointed out, researchers attempting to construct validate a personality measure must take precautions to ensure that correlations of this test with external criteria are not simply a consequence of psychologically uninteresting content overlap between test and criteria. They noted that "If . . . the overlapping item content does not contribute to the correlations of interest, researchers face evidence invalidating either the scale or the construct. Alternatively, if this content does contribute to correlations, researchers will find themselves unable to use the scale to test their hypotheses" (p. 572). We believe that the latter may have occurred in the AS literature, especially with regard to research on panic disorder. Moreover, this criticism may have important implications for AS research, because inapplicable items have been shown to produce substantially distorted correlations in Monte Carlo simulations (Waller, 1989).

The developers of the ASI could address this criticism in at least two ways: (1) revise the ASI to eliminate or rewrite potentially inapplicable items, or (2) include an inapplicable item option on the ASI in which respondents would be instructed to omit an item if they have never or virtually never experienced the symptom in question. If the findings using this revised ASI were similar or identical to those using the current version of the ASI, this would effectively answer the inapplicable item response criticism; in contrast, if these two sets of findings were different, this would call into question much of the previous research on the AS construct. Moreover, this potential problem with the ASI underscores the need for researchers to develop alternative measures of AS (see the section entitled "Multiple Operationalization of Constructs").

In response to this criticism, it could be argued that three teams of investigators (see Reiss, 1991) have reported that the ASI contributes incremental information over and above a measure of anxiety frequency (which contains the same stimulus items as the ASI) and a measure of trait anxiety in the prediction of self-reported fears of common objects and situations (see the section entitled "Incremental Validity"). Superficially, this finding addresses the problem of content–criterion overlap (at least with regard to research on fears), because the use of the anxiety frequency measure essentially controls for the ASI's symptom content. Nevertheless, because the word "scare" or "scares" appears in eight of the ASI's 16 items, a more parsimonious explanation for the partial correlation between the ASI and the FSS-II is that one measure of fear is highly associated with another measure of fear.

In turn, Reiss et al. (1988) attempted to address this criticism by developing a Dissimilar Fear Survey (DFS), which comprises fears that bear no superficial relation to one another (e.g., heights, cemeteries, snakes), and covarying out not only measures of trait anxiety and anxiety frequency, but also a measure of injury sensitivity (see "Testing Hierarchical Factor Models"), the Injury Sensitivity Index (ISI). The authors' rationale for this analysis was that removing the variance shared by the ISI and the DFS would control for the tendency of one fear to covary with other fears. Nevertheless, this analysis does not solve the problem, because the DFS contains a number of items relevant to physical injury (e.g., sharp objects, being a car passenger); thus, statistically controlling for the ISI simply removes the variance shared with these items, leaving the ASI to covary with items tapping other fears. This analysis thus fails to convincingly exclude the possibility that the covariation of the ASI with measures of other fears is a consequence of content-criterion overlap. Finally, it could be argued that if content-criterion overlap accounts for the correlation between the ASI and panic-related criteria, one would expect an equally high correlation between the ASI and several other anxiety disorders, such as GAD, because all DSM-III-R anxiety disorders are characterized by anxiety-related physical and cognitive symptoms. In fact, the relation between the ASI and GAD appears to be positive, but weaker than that between the ASI and panic disorder (Jones & Barlow, 1991; McNally et al., 1991). Broadly similar findings to those on GAD have been reported for most anxiety disorders, including obsessivecompulsive disorder and social phobia (Jones & Barlow, 1991; McNally et al., 1991).

Nevertheless, we find this argument less than convincing, because most of the ASI items assess concerns that are central to patients with panic disorder, but that are less central to patients with other anxiety disorders. GAD, for example, which possesses the most extensive criterion overlap with panic disorder of all DSM-III-R anxiety disorders, is characterized by pervasive concerns that are generalized to a number of different life circumstances (American Psychiatric Association, 1987). The ASI, however, is much more narrowly focused upon concerns regarding somatic (e.g., rapid heart beat) and, in some cases, cognitive and emotional (e.g., attentional difficulties, fears of losing control) symptoms of anxiety. These concerns, although present in many GAD patients, are far more central to patients with panic disorder. For example, patients with panic disorder score significantly more highly than patients with GAD on measures of somatic worry (Hoehn-Saric, 1982). This same argument would hold a fortiori for other DSM-III-R anxiety disorders, which possess less criterion overlap with panic disorder than does GAD.

### Testing for Interactions between AS and Other Variables

In response to criticisms of the AS construct, McNally (1989) argued that trait anxiety is insufficient to account for AS findings, and posited that "anxiety symptoms should not evoke further fear in trait-anxious persons who do not have concurrent high anxiety sensitivity" (p. 193). One plausible interpretation of McNally's statement is that only individuals with both high trait anxiety and high AS should be prone to panic disorder and other anxiety disorders, and to elevated state anxiety following challenge procedures (McNally has acknowledged that he concurs with this interpretation; March 1992).

One virtue of this hypothesis is its testability. If McNally is correct, the interaction of trait anxiety and AS should account for more variance in criteria such as panic disorder and postchallenge anxiety than the sum of these variables. Thus, it should be possible to test this prediction

by examining the significance (and effect sizes) of the interaction terms derived from two-way analyses of variance. Alternatively, if the ASI is scored in a continuous fashion,\* this hypothesis could be tested by means of moderated multiple regression (e.g., Stone, 1988; Zedeck, 1981), in which a product term representing the interaction of trait anxiety with AS is entered hierarchically following the main effects of these variables. If interactions between trait anxiety and AS were consistently found, the AS construct would have survived a stringent theoretical hurdle.

The interaction between AS and trait anxiety has recently been examined by Orsillo, Lilienfeld, and Heimberg (1992) in a sample of social phobics exposed to two challenge procedures: an individualized Behavior Test requiring subjects to perform a simulated feared situation (e.g., giving a public speech), and a modified Stroop (1935) task requiring subjects to color-name physical threat words, social threat words, and control words. Orsillo et al. (1992) found that, in several cases, the statistical interaction of AS with trait anxiety accounted for more variance in postchallenge anxiety reactions than the combined main effects of these two variables. Although these findings are preliminary and require replication, they provide support for both AS theory and the construct validity of the ASI. In addition, these findings suggest that examination of AS-trait anxiety interactions may enhance the prediction of anxiety responses to potentially anxiety-provoking stimuli, and perhaps even shed light upon the etiology of panic attacks.

We should point out, however, that Reiss (1991, p. 143) has hypothesized that the interaction of AS and *anxiety expectancy* should produce the highest risk for fears and phobias. In other words, individuals who both expect to experience anxiety and who are frightened of this anxiety should be most likely to develop a variety of fears. Nevertheless, if the interaction between AS and anxiety expectancy is hypothesized to result in the highest risk for fears, it is not clear why investigators (e.g., Reiss et al., 1986, 1988) have examined only the main effect of the ASI, because Reiss's model does not postulate that AS per se leads to increased risk for fears. Thus, the findings (discussed earlier) that the ASI is associated with self-report measures of fear are actually *inconsistent* with Reiss's model. Moreover, Reiss's model, in contrast to that of McNally (1989), makes

<sup>&#</sup>x27;A practice that concerns us is the tendency of anxiety sensitivity researchers to routinely dichotomize (e.g., Holloway & McNally, 1987) or trichotomize (e.g., Shostak & Peterson, 1990) scores on the ASI by means of median splits and related procedures. Dichotomization and similar practices typically result in a marked decrease in statistical power (Cohen, 1983), especially when distributions are approximately bivariate normal. Thus, we believe it should be incumbent upon researchers who dichotomize or trichotomize scores on the ASI to also report their findings using the ASI scored in a continuous fashion.

no mention of the role of trait anxiety or its interaction with AS in the genesis of fears. Thus, AS researchers will need to subject predictions derived from the AS construct to more direct tests, and to clarify the role of trait anxiety in the etiology of fears.

### **Testing Hierarchical Factor Models**

The question of whether trait anxiety and AS are "different" constructs may prove to be overly simplistic. Instead, the key to resolving the trait anxiety-AS controversy may lie in distinguishing between different levels of trait specificity. One possibility that we believe is worth exploring is that AS is a lower-order trait that is nested hierarchically within a higher-order dimension of trait anxiety (which itself appears to covary sufficiently with other traits to form a higher-order dimension of negative emotionality; see Tellegen, 1978/82; Watson & Clark, 1984, 1992).

As pointed out by Watson and Clark (1992), a prerequisite for a hierarchical factor model is that both general and specific factors influence the traits in the hierarchy. According to the hierarchical model proposed here, trait anxiety can be conceptualized as a general tendency to react anxiously to potentially anxiety-provoking stimuli, whereas AS is a more specific tendency to react anxiously to one's own anxiety and anxietyrelated sensations. Thus, AS contains both variance from the higher-order trait anxiety factor and specific variance that is largely or entirely unrelated to trait anxiety. Coexisting with AS at the lower-order level may be a number of other specific forms of anxiety-proneness, including the "injury sensitivity" and "social evaluation sensitivity" hypothesized by Reiss (1991) to play an important role in the development of fears. Although these lower-order "sensitivities" are separable, they may covary sufficiently highly to form a higher-order trait anxiety factor. In turn, AS may itself be divisible into still lower-order factors, such as anxiety regarding mental incapacitation and anxiety regarding physical symptoms (Telch et al., 1989; Wardle, Ahmad, & Hayward, 1990). A possible hierarchical factor model encompassing the relation between AS and trait anxiety is shown in Figure 1 (also see Watson & Clark, 1992, for an application of a hierarchical model to the dimension of negative emotionality).

If this hierarchical model is correct, both the trait anxiety and AS positions may have merit: AS may be conceptually closely related to trait anxiety, but measures of AS may nevertheless provide information that more global measures of trait anxiety do not. With regard to the latter, Mellstrom, Cicala, and Zuckerman (1976) found that specific indices of fears of animals and situations (e.g., snakes, heights) were superior to global indices of fear in their ability to predict behavior in



FIG. 1. Possible hierarchical factor model encompassing the relation between AS and trait anxiety. *Note:* The number of factors at the first, second, and third levels, although based upon research, should be regarded as provisional and subject to revision.

the presence of these stimuli. Similarly, social psychologists have found that the attitude-behavior relation is strongest when the attitude measure is specifically tailored to the behavior of interest (Ajzen & Fishbein, 1977). Thus, even though AS may to some extent be subsumable within a higher-order dimension of trait anxiety, the ASI may possess greater utility in the prediction of certain clinical phenomena (e.g., panic attacks and panic disorder) because it contains specific variance that is especially relevant for these phenomena.

This hierarchical model could be tested with confirmatory factor analysis (Long, 1983) by positing that AS and other specific fears and sensitivities load on a single higher-order trait anxiety factor, but that these fears and

sensitivities also contain specific variance largely or entirely unrelated to this trait anxiety factor. For example, injury sensitivity might be hypothesized to load on both a trait anxiety factor and a harm-avoidance or fearfulness factor (e.g., Tellegen, 1978/82), whereas AS might be hypothesized to load on both a trait anxiety factor and a somatic anxiety factor (e.g., Schalling, 1978). Although these models are of course conjectural, they illustrate but a few of the hypotheses concerning the relation of AS (and other traits) to trait anxiety that could be tested via confirmatory factor analysis.

In addition, this hierarchical model may help to explain four reasonably consistent findings in the AS literature. First, the positive, although not extremely high, correlation between the ASI and trait anxiety measures is consistent with this model, because the ASI is saturated with variance from the higher-order trait anxiety factor, but also contains variance that is largely or entirely uncorrelated with trait anxiety. Second, findings that the ASI possesses incremental validity relative to trait anxiety measures for a number of criteria (e.g., McNally, 1989) can be explained by positing that the ASI contains specific variance not shared with trait anxiety measures. Third, the findings that some individuals possess high AS, yet low trait anxiety, and vice-versa (e.g., Cox, Endler, & Swinson, 1991), are easily accommodated within a hierarchical model, because only a certain portion of the variance of the ASI derives from trait anxiety. Thus, for example, an individual could possess high trait anxiety and yet possess little of the specific content relevant to AS. Such an individual would thus receive a high score on a measure of trait anxiety, but might receive a relatively low score on the ASI. Fourth and finally, the hierarchical model may help to explain why patients with panic disorder have essentially equivalent levels of trait anxiety as GAD patients (Hoehn-Saric, 1982; McNally et al., 1991), but have higher levels of AS (Jones & Barlow, 1991; McNally et al., 1991). Specifically, panic disorder patients may differ from GAD patients largely or entirely in their levels of certain specific factors, such as somatic anxiety (Hoehn-Saric, 1982).

More generally, the discussion of hierarchical models highlights the need for researchers to examine the relation of AS to higher-order personality dimensions such as negative emotionality or neuroticism (Watson & Clark, 1984), and constraint. For example, the relation of AS to constraint, which represents a fearfulness or response-inhibition dimension (Tellegen, 1978/82), has not been explicitly examined in the AS literature. A possibility that, to our knowledge, has not been previously considered is that AS is a composite of negative emotionality and constraint; i.e., high ASI scorers may be characterized by tendencies both to experience negative emotions such as anxiety, as well as to fear these affects (see Tellegen, 1978/82; and Tellegen & Waller, in press, on the distinction between

anxiety and fear; also see Reiss, 1991). If this were the case, it might suggest that the propensity to experience panic attacks is a function of the statistical interaction between negative emotionality and constraint (or, at a lower-order level, between trait anxiety and fearfulness, respectively). Thus, individuals who are prone to panic attacks might be those who become highly anxious in response to stressors and then become frightened of this anxiety, leading to still higher anxiety, and so on. Thus, the "incubation" of anxiety seen in panic attacks could be a result of a reciprocal positive feedback cycle between negative emotionality and constraint. According to this model, high negative emotionality (and thus trait anxiety) would be a major risk factor for panic disorder. but only in the presence of elevated constraint (and thus fearfulness). Clearly, this model can only be regarded as speculative at the present time. Nevertheless, we believe that a consideration of higher-order personality dimensions should both clarify constructs such as AS and provide important clues to the etiology of panic disorder and perhaps other anxiety disorders.

Finally, this discussion also has important implications for research on the factor structure of the ASI. A major question in the AS literature has been whether the ASI consists of one factor (Peterson & Heilbronner, 1987; Taylor, Koch, McNally, & Crockett, 1992) or several correlated lower-order factors (Jones & Barlow, 1991; Telch, Shermis, & Lucas, 1989; Wardle et al., 1990). Studies of the factor and component structure of the ASI have yielded inconsistent results, with some researchers finding a single factor or component (e.g., Reiss et al., 1986; Taylor, Koch, McNally, & Crockett, 1992), and others finding multiple correlated lowerorder factors or components (e.g., Peterson & Heilbronner, 1987; Telch, Shermis, & Lucas, 1989).

Several authors have suggested that if the ASI were found to be multifactorial, its construct validity would be in serious doubt. For example, Taylor, Koch, McNally, and Crockett (1992) asserted that "The demonstration that the ASI is multifactorial would suggest either that the scale is lacking in construct validity . . . or that the construct of anxiety sensitivity is in need of modification" (p. 245). Nevertheless, the hierarchical model outlined here suggests that this conclusion is mistaken, because a measure can be multifactorial at a lower-order level and vet unifactorial at a higherorder level. Moreover, because trait anxiety symptoms are themselves clearly multifactorial (e.g., Endler, Parker, Bagby, & Cox, 1991; Schalling, 1978), it would be surprising if the fear of these symptoms, if assessed in sufficient detail, were not also multifactorial. Thus, far from indicting the construct validity of the ASI, the detection of separable, although intercorrelated, lower-order factors may instead suggest that the ASI provides adequate content coverage of some of the major domains of anxiety.

#### DISCUSSION

In 1989, Lilienfeld et al. asserted that "Until more stringent tests of the ASI's construct validity are conducted, the scientific status of the construct of anxiety sensitivity will remain less than convincing" (Lilienfeld et al., 1989, p. 102). Several years later, it appears that the evidence for the ASI's construct validity, as well as for Reiss et al.'s (1986) conceptualization of the AS construct, is somewhat stronger than it was when this statement appeared. Specifically, there now appears to be support for the contention that a number of the findings of AS research cannot be entirely accounted for by trait anxiety, although several negative findings suggest that this issue is not entirely closed.

Nevertheless, a number of unexplored theoretical and methodological issues in AS research remain. First, we believe that it is essential that researchers demonstrate that AS is a risk factor for panic attacks, panic disorder, and perhaps other anxiety disorders, and is not simply a consequence of these phenomena. Although some preliminary progress appears to have been made in this direction (Donnell & McNally, 1989, 1990; Maller & Reiss, 1992; Otto et al., 1991), we believe that further research on this issue should become a major priority among AS investigators.

Second, we are concerned that the content of the ASI is inherently confounded with a number of relevant criteria, particularly the symptoms of panic disorder. As a consequence, utilizing the ASI as the sole indicator of AS may render much of the research on the relation of AS to panic disorder (and perhaps other anxiety disorders) ambiguous with respect to the corroboration of the AS construct. Thus, we strongly encourage continuing research on the *construct* of AS, but believe that this research must move beyond a single measure in the testing of predictions derived from the AS construct. Moreover, the potential problem of content-criterion overlap further underscores the importance of longitudinal studies of high AS individuals with no history of panic attacks or panic disorder (e.g., Maller & Reiss, 1992). Findings indicating that these individuals are at heightened risk for subsequent panic disorder could not be attributed to content-criterion overlap, and would thus greatly strengthen the evidence for the construct validity of the ASI.

In addition, we believe that researchers need to consider the possibility that measures of AS differ from measures of trait anxiety not primarily in the higher-order construct they assess, but in their level of specificity. This hypothesis has the dual advantages of being testable by means of confirmatory factor models, and of providing a potential resolution to the trait anxiety–AS controversy. Specifically, AS measures may provide incremental information relative to trait anxiety measures not because they are unrelated to trait anxiety, but because they assess content that is especially relevant to panic disorder and related phenomena. The classical psychometric tradeoff between bandwidth and fidelity in psychological tests (Cronbach, 1990) seems relevant here; some tests (e.g., trait anxiety indices) assess a relatively broad spectrum of content at the expense of specificity, whereas others (e.g., the ASI) assess a relatively narrow range of content at the expense of breadth. Nevertheless, as noted earlier, it remains to be convincingly demonstrated that the specific content possessed by the ASI is not simply a function of construct-irrelevant item overlap with panic disorder and related conditions. Otherwise, the hierarchical model of AS and trait anxiety outlined here may be of little theoretical significance.

Investigators must also, we believe, clarify a number of important theoretical issues relevant to the AS construct. For example, researchers in this area have variously argued that AS is equivalent to the fear of anxiety, and that AS (along with anxiety expectancy) is only one component of the fear of anxiety. In addition, although anxiety expectancy has been hypothesized (Reiss, 1991) to interact with AS in increasing the risk of fears, researchers have consistently examined only the main effects of AS. Moreover, as we have noted elsewhere (Lilienfeld et al., 1989), the ASI appears to lack adequate content validity as a measure of the cognitions associated with the AS construct. Because most of the items on the ASI assess fears of anxiety and anxiety sensations, the extent to which this measure can be employed to make inferences about the cognitions putatively associated with these fears is unclear, particularly because measures of cognition and affect are often only weakly intercorrelated (Zajonc, 1984). Consequently, positive findings using the ASI provide relatively weak corroboration for the cognitive model of anxiety disorders that furnishes much of the theoretical underpinning of the AS construct (see Telch, Shermis, & Lucas, 1989, for a similar argument).

Finally, let us make clear that we are not suggesting that the AS construct be "replaced" by trait anxiety. Instead, as should be clear from our discussion of hierarchical factor models of AS and trait anxiety, we are arguing that the AS construct and alternative models based upon trait anxiety may not be as incompatible as some authors (e.g., Lilienfeld et al., 1989; Reiss, 1991) appear to have implied. Moreover, in contrast to AS, which derives from an explicit theory of cognitive expectancies (e.g., Reiss & McNally, 1985), trait anxiety is a descriptive construct that is not firmly embedded in a single theory. Nevertheless, there exist several plausible theories of trait anxiety that appear to have direct implications for the development of AS.

For example, Gray's (1982) neuropsychological theory of anxiety posits that individuals with high levels of activity in the Behavioral Inhibition System (BIS), a brain system consisting primarily of the septum, hippocampus, and orbitofrontal cortex, are overly sensitive to signals of punishment and related contingencies. Such individuals, according to Gray, are likely to be characterized by high levels of trait anxiety (but see Depue & Spoont, 1987), because they tend to interpret a large number of stimuli as harbingers of danger and other unpleasant outcomes. In turn, individuals with elevated BIS activity who, for example, have been given information concerning the adverse consequences of certain anxiety experiences (Rachman, 1977; Wolpe, 1981), who observe others suffer harm after experiencing anxiety (Reiss et al., 1986), who experience panic attacks (Goldstein & Chambless, 1978), or who possess elevated levels of certain personality characteristics (e.g., somatic anxiety, Schalling, 1978), may come to perceive ambiguous physical sensations as signaling danger, leading to elevated AS. This model would be consistent with the assimilative nature of trait anxiety discussed earlier, because individuals with high levels of BIS activity would have a low threshold for construing many stimuli, including their own anxiety symptoms, as threatening. In addition, this model would be consistent with a hierarchical relation between AS and trait anxiety, as this model seeks to explain how a general trait anxiety factor could become channeled into AS given specific environmental experiences, specific personality traits, or both.

Certainly, there are other plausible models of trait anxiety that have potential implications for the development of AS (e.g., Eysenck, 1981; Staats & Eifert, 1990, see especially p. 560). It is not our intention here to advocate one model over others, and it should be emphasized that all of these models will require further research before they can be considered to be adequately corroborated. The major point we wish to make is that a number of falsifiable models of the etiology of trait anxiety are potentially consistent with the AS construct, and may shed light upon the etiology of AS.

The construct of AS has clearly been of substantial heuristic value in the study of both anxiety and anxiety disorders, and the accelerating pace of recent research on this construct (e.g., Reiss, 1991) suggests that the significance of AS in theorizing and research in this area is likely to continue in the near future. Nevertheless, we believe that a number of major theoretical hurdles remain to be cleared before the AS construct can be considered to be strongly corroborated. It is therefore encouraging to note that AS researchers have recently begun to subject the AS construct to increasingly stringent theoretical risks (e.g., Orsillo et al., 1992; Otto, Pollack, Sachs, & Roxenbaum, 1992; Shostak & Peterson, 1990), many of which have provided more convincing support for this construct. Moreover, we are hopeful that, by pursuing the lines of research we have outlined, researchers will both further clarify the AS construct and move closer to unlocking the etiology of anxiety and anxiety disorders.

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