THEORETICAL AND REVIEW ARTICLES


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Abstract — In a recent article in this journal, Taylor raised a number of important issues regarding our review of research on anxiety sensitivity (AS). Nonetheless, we contend that (a) Taylor's claim that the relationship between the ASI and self-report measures of fear-proneness is not attributable to method variance is unconvincing; (b) Taylor is incorrect that the expectancy model of anxiety predicts that only the main effect of AS, rather than the interaction between AS and anxiety expectancy, influences fear-proneness; (c) Taylor's analyses examining the interaction between AS and trait anxiety are questionable; (d) Taylor's assertion that the relationship between the ASI and panic disorder cannot be dismissed as tautological does not withstand close scrutiny; and (e) Taylor's claim that the ASI is unifactorial rather than multifactorial is not supported by available evidence. We discuss the implications of Taylor's analysis examining the hierarchical relation between AS and trait anxiety, and suggest research designs for elucidating the association between these two constructs.

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In a recent article in this journal, Taylor (1995b) provided a critical evaluation of our review of research on anxiety sensitivity (AS) (Lilienfeld, Turner, & Jacob, 1993). Although Taylor raised several important and useful issues in his critique, a number of his assertions warrant closer examination.

In particular, we contend that Taylor's commentary contains numerous conceptual and methodological errors and misunderstandings, and that some of his data and arguments actually raise further questions concerning the construct validity of the principal operationalization of AS, the Anxiety Sensitivity Index (ASI), and the expectancy theory of anxiety in which the AS construct is embedded (Reiss, 1991). In our reply, we address Taylor's (1995b) criticisms, discuss alternative interpretations of the findings he cited, and point to several new directions for AS research. To facilitate comparison with Taylor's commentary, we use the same subheadings as those he provided.

Prior to discussing Taylor's criticisms, however, it would be worthwhile reiterating our basic views concerning the status of the AS construct and the AS1 (Lilienfeld, Jacob, & Turner, 1989; Lilienfeld et al., 1993). First, although we do not dispute the contention that AS is predictively useful in the domain of anxiety and anxiety disorders, we question whether AS is independent of other constructs in the personality domain, such as trait anxiety. Many theoretical treatments of AS have either treated AS as unrelated to preexisting constructs, or have made little effort to address the relationship of AS to these constructs (e.g., McNally, 1990). In contrast, we argue that the construct of AS is best viewed as a lower order facet of trait anxiety, rather than as a unique construct (cf. Reiss, 1991).

Second, we contend that further progress in AS research will be facilitated by a better understanding of the etiological underpinnings of AS. Some proponents of the AS construct (e.g., McNally, 1990; Reiss, 1991) have not addressed in detail the question of the origins of AS (cf. McNally, 1994, pp. 117-118). Although AS has been posited to be a cognitive construct consisting of beliefs that anxiety symptoms lead to adverse consequences (Reiss, Peterson, Gurisky, & McNally, 1986, the genesis of these beliefs remains unclear. We argue that a better understanding of the relationship of AS to other personality constructs, particularly trait anxiety, may provide valuable insight into the etiology of the cognitions constituting the AS construct.

Third, we believe that the distinction between prediction and explanation (Pedhazur, 1982) has often been neglected in the AS literature. Specifically, although we concur with Taylor (1995b) that AS aids in the prediction of anxiety-related phenomena, we do not view these findings as persuasive evidence for the claim that AS necessarily helps to explain such phenomena. It is possible, for example, that AS is an output variable produced by the interaction of a higher order personality dimension (e.g., trait anxiety, negative affectivity) with classical conditioning, social learning, and other factors. If so, AS might not play a major causal role in the genesis of anxiety disorders (cf. Reiss, 1987, 1991), but could nonetheless be a useful predictive marker of certain anxiety-related phenomena.
Fourth, we argue that developers and users of the ASI must first demonstrate that this measure predicts anxiety-related phenomena in a nontautological fashion. Much of the evidence for the construct validity of the ASI derives from comparisons between panic disordered patients and other individuals (e.g., Taylor, Koch, & Crockett, 1991). Nonetheless, because many of the ASI's items assess symptoms that are already known to be prevalent among panic disordered patients, such comparisons are not especially informative vis-à-vis the ASI's construct validity and provide little or no new information regarding panic disorder. We discuss this issue further in the following section.

**METHOD VARIANCE**

In our review (Lilienfeld et al., 1993), we observed that the results of several studies (e.g., McNally & Lorenz, 1987; Reiss, Peterson, & Gursky, 1988) indicated that the ASI accounts for significant increments in variance in self-report measures of common fears, such as the Fear Survey Schedule-II (FSS-II; Geer, 1965) and the Fear Survey Schedule-III (FSS-III; Wolpe & Lang, 1964) relative to self-report measures of anxiety frequency.

Nonetheless, we also noted that "because the word 'scare' or 'scares' appears in 8 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" (p. 168). In his commentary, Taylor (1995b) argued that the relations between the ASI and self-report indices of common fears are unlikely to be attributable solely to method variance. He went on to assert that our "conclusion is tautological; to say that two measures are correlated because they are 'associated' with one another does not explain the basis of the correlation" (p. 165). Here Taylor overlooked an important point that was raised by Nicholls, Licht, and Pearl (1982), who described a dilemma that investigators commonly encounter when attempting to validate self-report measures:

This dilemma can occur when researchers overlook the item content of a personality scale and when equivalent content is found in other scales with which it is correlated. . . 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 scales to test their hypotheses (p. 572; emphasis added).

Thus, because of the extensive content overlap of ASI items with items assessing common fears, findings that the ASI correlates with measures of such fears do not provide strong evidence for the ASI's construct validity. Such findings, although consistent with the expectancy theory of anxiety (Reiss, 1991), also are consistent with the well-documented tendency for self-report measures of fear to covary across individuals (e.g., Trull & Hillerbrand,
Although Taylor (1995b) is certainly correct that this tendency is itself in need of explanation, the more important point is that this tendency renders the correlation of the ASI with measures of common fears largely uninformative with respect to the ASI's construct validity.

Thus, Taylor has not convinced us that the findings of McNally and Lorenz (1987) and others provide compelling evidence for either the construct validity of the ASI or the expectancy theory of anxiety. In contrast to Taylor, we believe that the burden of proof falls on the proponents of the ASI to demonstrate that this measure predicts novel clinical phenomena not simply derivable from the features of panic disorder.

Taylor (1995b) also attempted to address our criticism by reporting data demonstrating that a factor analytically derived measure of AS (consisting of a number of items containing the word 'scare') was not significantly correlated with either the animal or blood-injury fear scales ($r = .06$ in both cases) of the FSS-III. But these findings run counter to Taylor's assertion that the expectancy theory of anxiety "propose[s] that AS alone accounts for variance in fear-proneness" (p. 165). Consequently, the negligible correlation of the AS factor with the animal and blood-injury fear scales of the FSS-III calls into question the hypothesis that AS is associated with fear-proneness. Taylor cannot have it both ways: if he wishes to assert that the main effect of AS increases the probability of acquiring all common fears (see also following section entitled "Anxiety Sensitivity and Anxiety Expectancies"), then he cannot then use the finding that AS is unrelated to certain common fears as evidence for the ASI's construct validity. Rather than providing evidence for the ASI's discriminant validity, the finding that the ASI is uncorrelated with measures of certain common fears actually provides evidence against the ASI's convergent validity.

**ANXIETY SENSITIVITY AND ANXIETY EXPECTANCIES**

In our review (Lilienfeld et al., 1993), we argued that most investigators in the AS literature have examined only the main effect of AS, despite the fact that Reiss's (1991) model does not include the main effect of AS. In an article explicating the expectancy model of anxiety, Reiss (1991) proposed that AS and anxiety expectancy (the expectation that one will experience anxiety in a given situation) interact to influence fears of common objects and situations. The formula developed by Reiss (1991, p. 143) to operationalize his expectancy model contains six major variables, two of which are AS and anxiety expectancy. In this formula, only the interaction (i.e., multiplicative effect) of AS and anxiety expectancy are posited to influence fears; no main effect of AS is hypothesized.

In response, Taylor (1995b) distinguished between fear state and fear-proneness, with the former being fear elicited by a specific situation and the latter being a generalized propensity toward fear of many situations. He maintained that "The expectancy theory makes no stipulation that fear-proneness
must be due to such an interaction [between AS and anxiety expectancy]” (p. 165) and that this interaction is predicted to affect only fear states. Although Taylor’s clarification is potentially useful, it runs counter to Reiss’s (1991) elaboration of his expectancy model. Specifically, Reiss (1991) referred to the same formula as described above, which was derived from his expectancy theory, and asserted that this formula implies that people who are afraid of anxiety [i.e., people high in AS] should develop a fear of any situation in which there is even a small chance/expectation of becoming anxious; because there are many such situations, people who are extremely sensitive to anxiety should develop fears of many situations (p. 147; emphasis added).

Once again, because only the interaction of AS with anxiety expectancy, and not the main effect of AS, is included in Reiss’s (1991) formula, Taylor’s (1995b) claim that “the expectancy model offers no prediction concerning the relation between the interaction of AS and anxiety expectancy, on the one hand, and fear proneness, on the other, is in error.” The above quotation makes clear that Reiss’s formula is intended to account for individual differences in fear-proneness. If Taylor were correct that “AS alone accounts for variance in fear-proneness” (p. 165), then Reiss’s formula should include a term for the main effect of AS. As Valentiner, Telch, Ilai, and Hehmsoth (1993) noted, “Reiss and McNally imply that anxiety sensitivity . . . is important only in its relationship to expectations of anxiety. . . . In other words, in the absence of expectations of anxiety . . . anxiety sensitivity should have no effect on fear behavior” (p. 396).

Moreover, the phrase “in which there even is a small chance/expectation of becoming anxious” (Reiss, 1991, p. 147) appears to imply an interaction between AS and anxiety expectancy, because situations in which no such chance or expectation exists would not be predicted to elicit anxiety, even among high AS individuals.1

Since the appearance of our review, Valentiner et al. (1993) have subjected the portion of Reiss’s (1991) expectancy theory pertinent to AS to a direct test. Valentiner et al. exposed nonclinical subjects with claustrophobic fears to a behavioral approach test (exposure to a dark and narrow chamber) and found that the interaction between AS and anxiety expectancy accounted for unique variance in behavioral performance, but not in either self-reported fear or physiological responding. Further investigations along the

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1A potential source of confusion regarding the construct of anxiety expectancy is its situational specificity versus generality. Although Taylor (1995b) asserted that anxiety expectancies refer only to specific fear states, it may be that anxiety expectancy, like AS, has some degree of cross-situational generality. Thus, individuals who expect to experience anxiety in response to one potentially anxiety-provoking situation (e.g., a plane flight) may be somewhat more likely than other individuals to expect to experience anxiety in response to another potentially anxiety-provoking situation (e.g., a major speech).
lines of Valentiner et al. should help to clarify the relationship between AS and anxiety expectancies.

**INTERACTION BETWEEN ANXIETY SENSITIVITY AND TRAIT ANXIETY**

Taylor (1995b) mistakenly attributed to us (Lilienfeld et al., 1993) the hypothesis that AS and trait anxiety should interact (i.e., combine multiplicatively) to increase risk for anxiety and anxiety disorders, referring to it as “Lilienfeld et al.'s hypothesis about the importance of the AS × TA interaction” (p. 166). Before evaluating Taylor's arguments on this issue, we should note that this hypothesis is not ours. Instead, this hypothesis represents a fairly straightforward deduction from the theorizing of McNally (1989), who posited that “anxiety symptoms should not evoke further fear in trait-anxious persons who do not have concurrent anxiety sensitivity” (p. 193). Thus, a disconfirmation of this hypothesis should call into question certain of the theoretical assumptions underlying the AS construct and its relation to trait anxiety. In an effort to test this hypothesis, Taylor (1995b) reported the results of a multiple regression analysis on 100 subjects who were administered the ASI, the trait version of the STAI, and the FSS-III. Taylor found no significant interaction effects for any of the FSS-III subscales. Aside from the low power of moderated multiple regression to detect interactions (Jaccard, Turrisi, & Wan, 1990), there are several aspects of this analysis, and Taylor's interpretation of it, that are troublesome.

First, Taylor (1995b) subjected items from the ASI and the Trait version of the Spielberger State-Trait Anxiety Inventory (T-STAI) to a principal components analysis (PCA) with orthogonal rotation, and selected items with high loadings on either AS or trait anxiety components to represent these two constructs. Taylor did not explain, however, why he did not use the original ASI and T-STAI items to represent their respective constructs. It is unclear whether the items loading on these two components correspond to those on the original ASI and T-STAI, particularly because a sample size of 100 is unlikely to yield a stable component structure.

Second, it is unclear why Taylor (1995b) used PCA, rather than common factor analysis, to select AS and trait anxiety items. Because PCA analyzes the total variance of each item, and not simply the common variance among items, it can yield components containing shared error variance (Weiss, 1971).

Third, Taylor (1995b) incorrectly asserted that because of the orthogonal rotation, “a measure of AS was obtained that was uncorrelated with TA [trait anxiety]” (p. 166). Although orthogonal rotations will produce uncorrelated latent factors, the factor scores resulting from such rotations will almost always be correlated, in part because the assumption that the underlying factors are uncorrelated is frequently unwarranted (Gorsuch, 1983). This fact is relevant because the presence of sizeable correlations among predictor vari-
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ables lowers the multiple correlation between these predictors and the criterion, rendering it more difficult to detect significant interactions by means of multiple regression (Jaccard et al., 1990).

Fourth, because the multiple regression was not conducted hierarchically (i.e., with the interaction term entered after the main effects), as recommended by Cohen and Cohen (1983), one cannot determine the amount of unique variance accounted for by the interaction of AS and trait anxiety.

Fifth, Taylor (1995b) reported that the factor corresponding to AS was significantly related to both the animal and blood-injury subscales of the FSS-III, even with the effects of trait anxiety and the AS by trait anxiety interaction partialled out. This finding contradicts Taylor’s aforementioned assertion concerning the nonsignificant correlation of ASI items with these two FSS-III subscales, and calls into question his earlier claim that the relationship between the ASI and measures of common fears cannot be attributed to method covariance (see section entitled “Method Variance”).

In a recent study (Orsillo, Lilienfeld, & Heimberg, 1994), we reported preliminary evidence for AS by trait anxiety interactions in the prediction of response to challenge procedures (e.g., a behavior test designed to elicit speaking anxiety) among a sample of social phobics. Specifically, we found that in 2 of 5 cases, the interaction of AS and trait anxiety predicted significant amounts of unique variance in the response to challenge procedures; in a third case, this interaction was marginally significant. In all three cases, the interaction assumed the predicted form whereby AS served to amplify anxiety responding among subjects with elevated trait anxiety. Again, further studies will be needed to provide more conclusive tests of McNally’s (1989) hypothesis concerning the interaction between AS and trait anxiety.

WHAT DOES THE ASI MEASURE?

In our review (Lilienfeld et al., 1993), we argued that the findings of Taylor et al. (1991) are potentially tautological and do not provide strong corroboration for the construct validity of the ASI. These authors reported that the ASI items that best discriminated panic disorder from other anxiety disorders were those measuring fears of physical sensations (e.g., rapid heart beat, fainting). We pointed out, however, that “because panic disorder is defined largely by physical sensations . . . 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” (Lilienfeld et al., 1993, p. 167).

Taylor (1995b) responded to this criticism by suggesting that because panic disorder and panic attacks are defined only by the presence of physical sensations, and not by the fear of such sensations, their results “cannot be dismissed as tautological” (p. 167). This argument neglects the fact, however, that panic disordered patients are much more likely than other individuals to have experienced a number of the symptoms assessed by items on the ASI (e.g., faintness, heart palpitations). As we pointed out in our review (Lilienfeld et al., 1993),
such items may, therefore, be essentially inapplicable to many respondents. Monte Carlo simulations have shown that the presence of inapplicable items in questionnaires can lead to spurious validity coefficients (Waller, 1989).

Moreover, even if one accepts Taylor’s (1995b) premise that the symptoms assessed by the ASI have been experienced by essentially all individuals, it is well known that patients with panic disorder and panic attacks often develop fear of physiological symptoms that might trigger future attacks (Goldstein & Chambless, 1978). Thus, even if all individuals have experienced the symptoms assessed by the ASI, panic patients are much more likely than other individuals to fear such symptoms. Consequently, Taylor et al.’s (1991) finding that the ASI items most specific to panic disorder are those assessing physiological symptoms of panic attacks does not provide convincing support for the ASI’s construct validity.

In addition, Taylor (1995b) averred that “If content overlap is the primary (artifactual) basis for the strong relationship between the ASI and panic disorder, then one would expect an equally strong relation between the ASI and GAD [generalized anxiety disorder]” (p. 167). As Taylor noted, the correlation between the ASI and GAD, although positive, is weaker than the correlation between the ASI and panic disorder. Nonetheless, we addressed this very argument in our review (Lilienfeld et al., 1993). As we pointed out, the hypothesis that Taylor et al.’s (1991) results are tautological does not lead to the expectation that the ASI should relate as highly to GAD as to panic disorder, because the ASI focuses quite narrowly on catastrophic concerns regarding both somatic (e.g., rapid heart beat) and cognitive/emotional (e.g., fears of losing control) symptoms of anxiety, both of which are more typical of panic disorder than GAD. Moreover, panic disorder patients report significantly higher levels of somatic anxiety than GAD patients (Hoehn-Saric, 1982).

We concur with Taylor (1995b), however, that the findings of Telch and Harrington (1994) are consistent with the assertion that the ASI is not solely a measure of panic disorder symptoms. Telch and Harrington reported that even among nonclinical subjects with no history of panic attacks, high ASI scores were associated with CO₂-induced panic attacks. Assuming that their results can be replicated, Telch and Harrington’s findings provide support for the construct validity of the ASI and suggest that AS may be a useful construct for predicting the initiation of panic attacks and panic disorder.

REVISING THE ASI

In our review (Lilienfeld et al., 1993), we questioned Taylor et al.’s (1991) recommendation to delete those ASI items exhibiting poor discrimination between individuals with panic disorder and other individuals. We conjectured that these items exhibit poor discrimination simply because they do not refer to common symptoms of panic disorder (see also previous section entitled “What Does the ASI Measure?”) and noted that “there appears to be no theoretical reason to expect these items to be less valid indicators of the AS construct” (p. 167).
In response, Taylor (1995b) suggested that "On theoretical grounds there is good reason to delete these items from the ASI, because they confound AS with the fear of negative evaluation" (p. 168). This reasoning does not explain, however, why Taylor et al. (1991) did not recommend that such items as "When I notice that my heart is beating rapidly, I worry that I might have a heart attack" and "When my stomach is upset, I worry that I might be seriously ill" should also be deleted from the ASI, because these items confound AS with the fear of illness/injury, which is one of the other fundamental fears (along with AS and the fear of negative evaluation) of Reiss’s (1991) expectancy model.2 Thus, Taylor’s (1995b) arguments are inconsistent. We contend that the ASI items assessing fear of illness/injury, although as equally confounded with other fundamental fears as those Taylor et al. (1991) recommended deleting, may discriminate panic disorder patients from other individuals precisely because these items are confounded with the symptoms of panic disorder. If Taylor’s hypothesis regarding the confounding of the AS construct with Reiss’s other proposed fundamental fears were correct, then those ASI items that confound AS with illness/injury sensitivity should also fail to discriminate between panic disorder patients and other individuals. That this is not the case calls Taylor’s arguments into question.

FACTORIAL STRUCTURE OF THE ASI

Taylor (1995b) argued that the bulk of evidence currently suggests that the ASI is unifactorial rather than multifactorial. The evidence he provided to support this claim, however, is questionable. First, Taylor incorrectly asserted that the high internal consistency (Cronbach’s alpha) of the ASI “challenges [the] multifactorial conception [of this measure], and argue[s] instead for a single-factor solution” (p. 168). Cronbach’s alpha is substantially influenced by a number of variables other than scale homogeneity, including the number of items on the scale and the number of factors pertaining to each item. Consequently, Cronbach’s alpha cannot be used as an indicator of scale unidimensionality or homogeneity (Green, Lissitz, & Mulaik, 1977).

The more important point we raised (Lilienfeld et al., 1993), which Taylor (1995b) did not address, is that the question of the unifactorial versus multifactorial structure of the ASI is essentially irrelevant to its construct validity. Taylor, Koch, McNally, and Crockett (1992) had contended 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) (see also Taylor, 1995a, p. 245). This assertion

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2Although we discuss Reiss’s (1991) model of fundamental and common fears in the context of Taylor’s commentary, we believe that considerably more research will be needed before the distinction between these two classes of fears can be accepted. Reiss’s hypotheses concerning the number and nature of his proposed fundamental fears, as well as the emergence of common fears from these fears, remain largely conjectural.
neglects the fact that correlated lower order factors can coexist with a single higher order factor. It would be equally fallacious to argue, for example, that because most IQ tests (e.g., the WAIS-R) are multifactorial (Waller & Waldman, 1990), either such tests lack construct validity or the construct of “g” (general intelligence) requires either abandonment or modification. As Watson, Clark, and Harkness (1994) noted, “in the context of a hierarchical arrangement, superfactor and multidimensional models are in no way incompatible; rather, they represent different levels of generality and abstraction” (p. 25).

Taylor (1995b) also discussed the results of a confirmatory factor analysis of the ASI (Taylor et al., 1992) that indicated that although a model positing four orthogonal factors yielded the best fit, this fit was only marginally better than that of a single-factor solution. Taylor et al. also tested a model positing four intercorrelated factors and concluded that “The oblique factors . . . are intercorrelated to such an extent that they are more appropriately regarded as facets of a single construct” (p. 250). Nevertheless, this finding does not preclude the possibility that the ASI is multifactorial at a lower order level, particularly because the average factor intercorrelations were considerably below 1.0 (specifically, .5 to .6) in both clinical and student samples. Thus, Taylor et al.’s analyses do not demonstrate that the ASI is unifactorial, Taylor’s (1995b) assertions notwithstanding. A rigorous test of the possibility that the ASI is unifactorial would have been to constrain all of the factor intercorrelations to unity (i.e., 1.0), and to compare the fit of this model with an oblique factor model. If the former model were rejected, the hypothesis that the ASI is unifactorial could likewise be rejected.

**HIERARCHICAL MODEL OF FUNDAMENTAL FEARS**

Taylor (1995b) reported the results of a new analysis designed to test two levels of our proposed hierarchical structure of the relations among AS, trait anxiety, and negative emotionality (Lilienfeld et al., 1993, p. 172). Taylor subjected the T-STAI, ASI, and measures of the other two fundamental fears of Reiss’s (1991) expectancy model (viz., fear of negative evaluation and injury/illness sensitivity) to a confirmatory factor analysis. In the model tested, the three fundamental fears were posited as lower order factors, and trait anxiety was posited as a higher order (general) factor accounting for the common variance among the fundamental fears. Taylor reported satisfactory goodness-of-fit indices for this model, although in future analyses it will be important to compare the fit of this model against that of alternative models (e.g., a model positing correlated lower order factors, but no general factor).

The results of Taylor’s (1995b) analysis are consistent with our assertion (Lilienfeld, 1996; Lilienfeld, Jacob, & Turner, 1989; Lilienfeld et al., 1993) that AS is not distinct from trait anxiety, but can instead be viewed as a lower order facet of trait anxiety. If these results can be replicated, they would suggest that trait anxiety and AS may differ largely in their degree of generality
versus specificity: trait anxiety might be viewed as a general propensity to experience anxiety in response to potentially stress-provoking stimuli, whereas AS might be viewed as a more specific tendency to experience anxiety in response to physiological and cognitive/emotional symptoms associated with anxiety (see also McNally, 1989, 1996).

Moreover, a hierarchical relation between trait anxiety and AS suggests that measures of both constructs may be useful for different purposes. Measures of trait anxiety may possess incremental validity relative to measures of AS for predicting broad-band criteria relevant to anxiety, such as trait levels of negative affectivity (Watson & Clark, 1984) and diagnoses of GAD, although this prediction has not been subjected to an empirical test. Measures of AS appear to possess incremental validity relative to measures of trait anxiety (e.g., McNally, 1989; in press) in the prediction of certain narrow-band criteria relevant to anxiety, such as panic disorder, because measures of AS presumably contain unique variance relevant to fears of anxiety-related physical and cognitive/emotional symptoms. Additional work along the lines of Telch and Harrington (1994), however, will be required to demonstrate that this unique variance is not a consequence of the ASI’s content overlap with the symptoms of panic attacks and panic disorder.

DISCUSSION

Although Taylor’s (1995b) critique raises several important conceptual and methodological issues regarding the current status of research on AS, a number of his assertions appear to be unwarranted or unsubstantiated. In particular, Taylor has not provided convincing evidence that much of the data he cited in support of the construct validity of the ASI, such as the finding that the ASI distinguishes between panic disorder patients and other individuals (e.g., Taylor et al., 1991), cannot be regarded as a largely tautological consequence of the fact that the ASI assesses many of the symptoms of panic disorder. We agree with Taylor, however, that the results of challenge studies among subjects with no history of panic attacks (e.g., Donnell & McNally, 1989; Telch & Harrington, 1994) may help to provide more compelling support for the construct validity of the ASI and for the theoretical assumptions underlying the AS construct. Further studies such as those of Maller and Reiss (1992), which examine the course of nonclinical subjects with high levels of AS, will also be relevant for evaluating the construct validity of the ASI and the validity of Reiss’s (1991) expectancy model. Clearly, persuasive support for the ASI’s construct validity must derive from subjects without a prior history of panic attacks or panic disorder.

In addition, the empirical basis for Taylor’s assertion that the ASI is unifactorial is unconvincing. More important, this assertion has no necessary bearing on the construct validity of the ASI (cf. Taylor et al., 1991) because a hierarchical structure demands the presence of correlated lower order factors coexisting with a higher order factor.
The results of the confirmatory factor analysis reported by Taylor (1995b) provide preliminary support for one important component of Lilienfeld et al.’s (1993) hierarchical model of the relation between AS and trait anxiety and may point to fruitful etiological models of the relation between these two constructs.

In this respect, Taylor’s (1995b) assertion that “The theoretical status of TA [trait anxiety] is a major weakness of Lilienfeld et al.’s hierarchical model” (p. 171) is misconceived. Hierarchical models of personality variables are largely descriptive and are not intended to resolve definitively questions concerning the underlying causes of these variables. Nonetheless, such models can be helpful in suggesting plausible etiological models and in ruling out implausible etiological models. For example, a model indicating a hierarchical relation between trait anxiety and AS might suggest that trait anxiety is a source trait that gives rise to the surface traits of AS and Reiss’s other proposed fundamental fears (see Cattell, 1950, for a discussion of source and surface traits). Such a hierarchical model would be inconsistent, however, with the hypothesis that AS and trait anxiety arise from entirely different etiological influences. Moreover, Taylor’s claim that “Lilienfeld et al. are silent on (the issue)” of why trait anxiety leads to AS and other fundamental fears is mistaken, because in our review (Lilienfeld et al., 1993, pp. 176–177) we discussed how Gray’s (1982) neuropsychological model of anxiety might provide a framework for explaining how individual differences in trait anxiety become channeled into individual differences in AS, depending on direct experiences with anxiety and panic attacks, social learning experiences, information from others, and levels of other personality variables.

Multivariate behavior-genetic designs (e.g., twin designs) may be especially helpful in elucidating the relationship between trait anxiety and AS (as well as Reiss’s other hypothesized fundamental fears), as these designs can shed light on whether the covariation between trait anxiety and AS is attributable to genetic factors, shared environmental influences that are transmissible across individuals (e.g., modeling, information transmission), shared environmental factors that are not transmissible across individuals (e.g., idiosyncratic anxiety-provoking events), or some combination of all three factors. In addition, such designs can reveal the extent to which any genetic influences on AS are unique to this trait or are shared with trait anxiety. Finally, longitudinal studies of children and adolescents with high levels of trait anxiety or high scores on putative laboratory markers of trait anxiety (e.g., measures of behavioral inhibition; Kagan, Reznick, & Snidman, 1988) may provide valuable information concerning the development of AS. These studies may assist in the identification of risk factors for the emergence of elevated AS, as well as in the testing of causal models of the relation between trait anxiety and AS.

REFERENCES


