Journal of Abnormal Psychology
1989, Vol. 98, No. 1, 100-102

0021-843X/89/$00.75

COMMENTS

Comment on Holloway and McNally's (1987) "Effects of Anxiety Sensitivity on the Response to Hyperventilation"

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Holloway and McNally (1987) found that normals with high scores on the Anxiety Sensitivity Index (ASI), an instrument developed to assess beliefs regarding the adverse consequences of anxiety, reported more anxiety and more frequent and intense somatic sensations following hyperventilation than did normals with low scores on the ASI. They concluded that this result provides support for the construct validity of the ASI and thus for the construct of anxiety sensitivity. Nevertheless, we argue that (a) the developers of the ASI have conflated beliefs regarding the adverse consequences of anxiety with fear of these consequences, (b) the accumulated evidence for the construct validity of the ASI is weak, and (c) Holloway and McNally's design and analyses do not permit them to exclude the more parsimonious explanation that trait anxiety accounts for their findings. Implications for research on anxiety sensitivity are discussed.

In a recent article in this journal, Holloway and McNally (1987) reported the results of an experiment in which normal subjects engaged in 5 min of voluntary hyperventilation. Subjects were subdivided according to their scores on Reiss, Peterson, Gursky, and McNally's (1986) Anxiety Sensitivity Index (ASI), a 16-item self-report instrument developed to assess beliefs regarding the adverse consequences of anxiety. Holloway and McNally found that high-anxiety-sensitivity (HAS) subjects reported higher subjective anxiety and more frequent and intense hyperventilatory sensations on a self-report checklist than did low-anxiety-sensitivity (LAS) subjects. The authors argued that these results provide support for the construct validity of the ASI and thus for the construct of anxiety sensitivity. We believe, however, that their conclusions warrant closer scrutiny.

Holloway and McNally stated that "anxiety sensitivity refers to beliefs that anxiety symptoms have harmful consequences apart from their immediate unpleasantness" (p. 330). Yet elsewhere they asserted that the ASI "was developed to measure the fear of becoming anxious" (p. 330). This same discrepancy can be found in Reiss et al.'s (1986) original article. Indeed, inspection of the item content of the ASI reveals that virtually all of the items appear to tap fear of anxiety and of anxiety symptoms (e.g., "It scares me when I am nervous," "When I notice that my heart is beating rapidly, I worry that I might have a heart attack"), rather than beliefs concerning the negative consequences of anxiety, as claimed by the test's developers. Reiss et al. and Holloway and McNally in effect make the unsubstantiated assumption that individuals who fear the consequences of anxiety necessarily possess cognitions that anxiety has harmful consequences. In doing so, they conflate two channels of Lang's (1968) tripartite system of anxiety, and leave unanswered the question of whether anxiety sensitivity is an affective construct, a cognitive construct, or both. This ambiguity is troublesome because cognition and affect appear to be partially independent systems that may become uncoupled in certain circumstances (Zajonc, 1980). Had Reiss et al. wished to measure *beliefs* that anxiety has harmful consequences, they might instead have asked respondents to rate the probabilities that specific anxiety-related symptoms (e.g., heart pounding) will lead to dangerous outcomes (e.g., heart attack). Instead, the imprecise specification of the construct of anxiety sensitivity may make it difficult to evaluate claims for the ASI's construct validity (Cronbach & Meehl, 1955).

A critical assertion made by Holloway and McNally is that "anxiety sensitivity is a dispositional construct distinct from trait anxiety" (p. 330). In addition, they claim that "abundant evidence supports the construct validity of the ASI" (p. 331). In our view, however, the evidence for the construct validity of the ASI is weak.

For example, Holloway and McNally cited studies showing that the ASI discriminates agoraphobics from other anxiety-disordered patients, and both of these groups from normals (Reiss et al., 1986), that the posttreatment ASI scores of agoraphobics normalize following cognitive-behavioral treatment (McNally & Lorenz, 1987),[1] and that the ASI moderates the relation between mitral valve prolapse syndrome and panic dis-

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[1] It is worth noting that many standard trait anxiety measures (e.g., the Taylor Manifest Anxiety Scale) are highly sensitive to treatment effects among agoraphobics (Michelson, 1987). This may be because many of these instruments assess a mixture of state and trait phenom-

order (Lyons, Talano, Gitter, Martin, & Singer, 1986; cited in Holloway & McNally, 1987). Impressive as these findings are, we do not concur that the accumulated evidence "supports the construct validity of the ASI." Rather, we argue that the results of these studies can be equally accounted for by positing that the ASI measures trait anxiety.[2] The construct validity of the ASI hinges not only upon its ability to satisfy predictions generated by the anxiety sensitivity hypothesis, but also upon its ability to satisfy predictions not derivable from a more parsimonious (e.g., trait anxiety) model.

Unfortunately, the evidence for the discriminant validity of the ASI from trait anxiety measures is inconsistent. Peterson and Heilbronner (1987) cited unpublished research by Seidenberg and Peterson showing a negligible (.07) correlation between the ASI and the Spielberger State-Trait Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1970) score in a sample of medical students. Because no descriptive data are reported, however, the potential role of sample-specific attenuating factors (e.g., range restriction) cannot be evaluated. In contrast, Reiss et al. (1986) reported that the ASI is moderately highly correlated with both the Taylor Manifest Anxiety Scale (TMAS; .43 and .50 in two samples) and a specially constructed Anxiety Frequency Checklist (AFC; .36 and .32 in the same two samples). Similarly, McNally and Lorenz (1987) found a .46 correlation between the ASI and the TMAS. Nevertheless, a positive correlation between the ASI and trait anxiety measures does not necessarily vitiate the anxiety sensitivity hypothesis, because individuals with high anxiety sensitivity might be expected to react more intensely and recover more slowly from anxiety-provoking experiences, perhaps leading them to have higher trait anxiety scores. The more critical question is whether the ASI possesses incremental validity relative to trait anxiety measures.

In this regard, Holloway and McNally cited the results of a hierarchical multiple regression analysis (Reiss et al., 1986) showing that the ASI predicts variance in a measure of fear of common objects and situations, Geer's (1965) Fear Survey Schedule-II (FSS-II), over and above the TMAS and the AFC. This result has recently been replicated by McNally and Lorenz (1987) using the TMAS alone. Both Holloway and McNally and Reiss et al. (1986) argued that this finding is consistent with the claim that the ASI measures anxiety sensitivity, rather than trait anxiety. This finding only demonstrates, however, that the ASI contains reliable variance unrelated to conventional trait anxiety measures. To make a strong claim for the ASI's construct validity, its proponents must demonstrate that this portion of the ASI's variance predicts results derivable from the anxiety sensitivity hypothesis. Neither Reiss et al. nor McNally and Lorenz furnished any convincing rationale regarding why anxiety sensitivity should relate to fears of common objects and situations. Moreover, the residual correlation of the ASI with an index of fear is compatible with such a diversity of explanations (e.g., that the ASI is a better measure of trait anxiety than conventional trait anxiety measures) that, in our view, it provides only minimal support for the ASI's construct validity.

These considerations raise questions concerning the interpretation of Holloway and McNally's results. Given the authors' interest in demonstrating that the ASI measures a disposition distinct from trait anxiety, it is surprising that they failed to include a measure of trait anxiety in their experiment. Holloway and McNally reported that, at the beginning of the experiment, HAS subjects had higher scores than did LAS subjects on a measure of state anxiety, the Tension-Anxiety subscale of the Profile of Mood States (POMS; McNair, Lorr, & Droppleman, 1971) as well as on a self-report hyperventilation checklist (HVC) consisting of somatic sensations frequently associated with hyperventilation. Why HAS subjects should report more anxiety and hyperventilation-related somatic sensations at pretest is unclear, particularly because at this point in the experiment subjects were not even informed that they would be asked to hyperventilate. The anxiety sensitivity hypothesis predicts that HAS subjects should report higher state anxiety than LAS subjects only in anxiety-provoking situations; these findings thus again raise the possibility that the ASI is contaminated by trait anxiety.

Holloway and McNally found that after performing an ANCOVA using pretest POMS anxiety scores as a covariate, the difference in posthyperventilation POMS anxiety scores between HAS and LAS subjects was no longer significant. This result clearly runs counter to prediction. According to the anxiety sensitivity hypothesis, HAS subjects should be more frightened by the somatic symptoms resulting from hyperventilation than LAS subjects, even if their baseline anxiety levels are equated with those of LAS subjects. As this represents the most straightforward test of the authors' hypotheses, the construct validity of the ASI can hardly be said to have received strong support.

In contrast to this negative finding, the authors reported that after covarying out pretest HVC scores, HAS subjects still obtained higher posthyperventilation HVC scores than did LAS subjects. Nevertheless, this analysis does not answer the more critical question of whether subjects who had higher posthyperventilation HVC scores were also those with higher trait anxiety. Two groups of subjects initially matched on anxiety- related somatic sensations might nevertheless differ in trait anxiety. Thus, differences in somatic sensations following hyperventilation could plausibly be attributed to preexisting differences in trait anxiety, rather than anxiety sensitivity. As noted earlier, however, Holloway and McNally did not include a trait anxiety

ena and/or because successful treatment may decrease anxiety-proneness.

[2] In reviewing the evidence for the construct validity of the ASI, Holloway and McNally also cited a study by Maller and Reiss (1987), who found an interesting interaction: HAS subjects, relative to LAS subjects, demonstrated significantly more anxiety-related speech disturbances when discussing their own anxiety experiences than when discussing neutral experiences. Maller and Reiss contended that these results cannot be accounted for by trait anxiety. We disagree. Although Maller and Reiss stated that "the concept of anxiety does not imply a tendency for anxiety stimuli to produce anxiety" (p. 271), it has been pointed out that trait constructs imply interactions (Tellegen, 1981); thus, individuals with high trait anxiety should exhibit a greater increase in anxiety in anxiety-provoking situations compared with individuals with low trait anxiety. Because discussing one's anxiety experiences is presumably more anxiety provoking for all subjects than is discussing neutral experiences (this is confirmed by inspection of Maller and Reiss's Table 1 on p. 269), Maller and Reiss's results can be explained by positing that the ASI assesses trait anxiety. Because Maller and Reiss failed to include a trait anxiety measure, however, this competing explanation cannot be investigated.

measure to attempt to rule out this possibility. Given the association of trait with state anxiety (Tellegen, 1985) the next most pertinent analysis would have been to use pretest state anxiety (i.e., POMS) scores as a covariate. Holloway and McNally reported no such analysis.

Thus, Holloway and McNally's design and analyses do not permit them to exclude the more parsimonious explanation that trait anxiety, rather than anxiety sensitivity, accounts for their findings. Their results suggest that the ASI may be contaminated by trait anxiety, and thus cast doubt upon the ASI's construct validity.

In summary, we contend that claims for the construct validity of the ASI are premature. The construct of anxiety sensitivity has been inconsistently defined, and the extant literature does not convincingly refute the rival hypothesis that the results of studies using the ASI can be explained by trait anxiety. If the problematic findings of Holloway and McNally are shown to be replicable, the proponents of the ASI will, in our view, need to either (a) revise their test to provide a purer measure of anxiety sensitivity uncontaminated by trait anxiety, or (b) broaden the construct of anxiety sensitivity to incorporate the results discussed here. We also urge that researchers include a measure of trait anxiety in all investigations of the ASI, so that the incremental validity of the latter relative to the former can be evaluated. 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.

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Received July 18, 1988
Accepted August 24, 1988 ■