PTSD Diagnostic Criteria: Understanding Etiology and Treatment

TO THE EDITOR: In their article, published in the January 2009 issue of the *Journal*, Carol S. North, M.D., M.P.E., et al. (1) offered recommendations concerning refinement and validation of the diagnosis of posttraumatic stress disorder (PTSD). The authors concluded that "avoidance and numbing symptoms represent the core of psychopathology as currently written in the DSM-IV-TR" (1, p. 39). These conclusions have important implications for DSM-V. However, there are other considerations about avoidance and numbing relevant to the criteria set for PTSD that the authors did not discuss. First, there is a growing body of factor analytic research showing that effortful avoidance (i.e., deliberate attempts to avoid trauma reminders) and numbing are distinct groups of PTSD symptoms (2). If factors identified by factor analysis correspond to distinct groups of causal mechanisms (3), then research into the etiology of PTSD would be hindered by blurring the distinction between effortful avoidance and numbing. Second, DSM-IV-TR criteria for PTSD include five numbing symptoms but only two effortful avoidance symptoms. In order to reliably assess various manifestations of effortful avoidance, more of these symptoms need to be included in DSM-V. Third, based on DSM-IV-TR criteria, PTSD can be diagnosed even in the absence of effortful avoidance. If effortful avoidance is one of the core features of PTSD, then such symptoms should be required to diagnose PTSD. Fourth, the existence of trauma-related, psychogenic amnesia is controversial (4), thereby undermining validity of the PTSD diagnosis. Even if such amnesia is a clinical reality, it is different than effortful avoidance of trauma memories and, more likely than not, distinct from numbing. This latter point is underscored by reanalysis of data from 60 PTSD patients in one of our recent studies (5) in which a 5-item numbing scale was created from scores on the Clinician-Administered PTSD Scale. Amnesia had a corrected item-total correlation that was no different from zero (r=–0.08) compared with a range of 0.16–0.54 for the other numbing items. In short, amnesia was unrelated to other numbing items. It seems prudent to omit this symptom from DSM-V. Finally, if avoidance and numbing are the core features, is PTSD simply a combination of specific phobia and depression (6)? An important step in validating PTSD is to demonstrate that it has incremental validity over specific phobia and depression—that is, to show that the diagnosis of PTSD conveys important information relevant to understanding the etiology and treatment of trauma-related psychopathology that is not conveyed by a combination of these other diagnoses.

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GORDON J.G. ASMUNDSON, PH.D.
Regina, Saskatchewan, Canada
STEVEN TAYLOR, PH.D.
Vancouver, British Columbia

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The Importance of Four-Factor Emotional Numbing and Dysphoria Models in PTSD

TO THE EDITOR: In their article, Dr. North et al. (1) raise many important points regarding the validation of the diagnosis of PTSD. However, the article overlooked a growing body of literature reporting four-factor emotional numbing and dysphoria models of PTSD (2). These studies used confirmatory factor analysis, which involves a priori specification of a factor structure and a rigorous method of testing hypothesized symptom structures, to examine the structure of PTSD symptoms in various trauma-exposed populations. Confirmatory factor analytic studies have generally failed to support the DSM-IV conceptualization of re-experiencing, avoidance, and hyperarousal symptom clusters in PTSD. Although a study of breast cancer patients found support for the DSM-IV three-cluster model and another study of motor vehicle accident victims found support for a two-factor model consisting of a re-experiencing/avoidance factor and an emotional numbing/hyperarousal factor, most confirmatory factor analytic studies have found support for one of two four-factor models (2). The emotional numbing model, which conceptualizes emotional numbing as distinct from avoidance and includes re-experiencing, avoidance, emotional numbing, and hyperarousal clusters, has provided the best representation of PTSD symptom structure in studies of community members with a lifetime history of PTSD, military combat veterans, military peacekeepers, Cambodian refugees, targets of workplace sexual harassment, young adult survivors of community violence, trauma-exposed medical patients, and cancer survivors. The dysphoria model includes distinct re-experiencing, avoidance, and hyperarousal factors as well as a general distress or dysphoria factor that combines emotional numbing and three DSM-IV hyperarousal symptoms (sleep disturbance, anger outbursts, difficulty concentrating). This factor structure has provided the best representation of PTSD symptom structure in military veterans of the Persian Gulf War and the Iraq/Afghanistan War, workers exposed to the September 11 attacks on the World Trade Center, college students indirectly exposed to the 9/11 World Trade Center attacks, sexual assault survivors, and bereaved individuals.

The four-factor PTSD models provide a theory driven and empirically supported framework for investigating the pathophysiology and informing the diagnosis and treatment of PTSD. For example, the emotional numbing cluster, but not any other symptom cluster, predicts poorer response to cognitive-behavioral treatment in PTSD (3). Taken together, these studies suggest that revisions of current diagnostic criteria for PTSD and efforts to validate the PTSD diagnosis should consider the presence of distinct re-experiencing, avoidance, emotional numbing/dysphoria, and hyperarousal symptom clusters in PTSD.

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ROBERT H. PIETRZAK, PH.D., M.P.H.
West Haven, Conn.
STEVEN M. SOUTHWICK, M.D.
West Haven, Conn.

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Dr. North Replies

TO THE EDITOR: Drs. Asmundson and Taylor and Drs. Pietrzak and Southwick suggest the importance of four-factor analysis models toward validation and revision of PTSD criteria. Although factor analysis may provide a worthwhile test of the natural co-occurrence of symptoms to define cohesive categories whose members tend to travel together, it is a starting point to establish alternative models of symptom clusters, which will then need to be subjected to validity testing. Establishment of a model through factor analysis is not a diagnostic validation procedure. Validity of a proposed set of diagnostic criteria is determined through established validation methods in the following five phases: examination of cohesive core characteristics, laboratory studies, exclusion criteria, follow-up studies, and family studies (1). Factor analysis methods may be contributory to the first of these validation phases. The models that perform best in validation studies would be potential candidates for consideration of revision of PTSD criteria in DSM-V. We would discourage revisions to DSM-V PTSD criteria based on factor analysis before the models that emerge from this research are subjected to validation procedures.

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CAROL S. NORTH, M.D., M.P.E.
Dallas, Tex.

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Odor Detection in Schizophrenia: Alternative Explanations

TO THE EDITOR: In their article published in the February 2009 issue of the *Journal*, Bruce I. Turetsky, M.D., and Paul J. Moberg, Ph.D., noted an odor-specific detection deficit in schizophrenia that may implicate abnormal intracellular cAMP signaling (1). The patients were able to normally detect citralva, a strong stimulator of adenyl cyclase, but not lyral, a weak stimulator. Drs. Turetsky and Moberg failed to note prior work that conflicts with the conclusions drawn from their results.

Impaired odor detection of other substances, including isoamyl acetate (2), dimethyl disulfide (3), and geraniol (4), has been demonstrated in schizophrenia. Individuals with schizophrenia have an impaired ability to detect geraniol, which is a strong adenyl cyclase stimulator similar to citralva. Citralva placed fourth and lyral placed 42nd in the ranking of adenyl cyclase response in tests of 44 odorants, and geraniol was the tenth most potent stimulator (5). In a study of odorant-stimulated electro-olfactogram response, citralva ranked fifth out of 36 odorants, lyral was ranked 21st, and geraniol was ranked eighth (6). Geraniol clearly resembles citralva in its ability to stimulate adenyl cyclase but differs in that geraniol cannot be detected normally by schizophrenia patients.

It seems that odor detection deficits in schizophrenia may have a more complex explanation than the specific signal transduction mechanism cited.

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MICHAEL J. SERBY, M.D.
New York, N.Y.