

Editorial

Revisiting the Institute of Medicine report on the validity of posttraumatic stress disorder

Recently, a committee from the prestigious Institute of Medicine issued a report (IOM, 2006) on the scientific status of posttraumatic stress disorder (PTSD) [1]. On the basis of a review of the 5 criteria for diagnostic validity proposed by Robins and Guze [2] in their classic article, the committee concluded optimistically, “The evidence is robust for the core clinical features of PTSD... [and] progress has been made in fulfilling the remaining criteria” (p. 55). Thus, does the IOM report imply that PTSD’s construct validity is well on the way to being firmly established. In this commentary, we go beyond extant reviews on problems with the PTSD diagnosis [3] and provide a full assessment of the IOM report’s sanguine conclusions. In our reading of the report, we were struck by its failure to acknowledge serious challenges to the PTSD diagnosis. For example, a key distinguishing feature of PTSD is that it is not agnostic to etiology. Unlike virtually all diagnoses in the *Diagnostic and Statistical Manual of Mental Disorders (DSM)*, PTSD rests on the assumption of a specific etiology, whereby a distinct set of events (criterion A) is assumed to be the uniformly most potent contributor to outcome [4]. Numerous publications, however, have documented that (a) most individuals do not develop PTSD after criterion A events [5,6]; (b) nonevent variables typically contribute more to PTSD signs and symptoms than do traumatic events [7,8]; and (c) the PTSD clinical syndrome can present after noncriterion A events [9–11]. Even reviewers who accept the specific etiology hypothesis have taken time to address the “criterion A problem” [12]. Yet the IOM report made no mention of these concerns. Instead, the report simply stated, “The necessary cause of PTSD is by definition a traumatic event” (p. 23).

In the absence of a specific etiology, the distinctiveness of PTSD falls upon its defining symptom criteria and the requirement that it can be differentiated from other disorders. The IOM committee observed that the “differentiation requirement” was difficult to apply to PTSD because many of the construct’s symptoms are shared with other psychiatric disorders, including specific phobia, major depression, and generalized anxiety disorder. In making this point, the committee overlooked how this very overlap raises questions concerning the syndromal independence of PTSD. In

actuality, the situation is even more serious because the requisite number of symptoms for diagnosing PTSD can be obtained completely through a combination of symptom criteria for specific phobia and major depression [13].

Without considering this fundamental problem, IOM committee members referenced a single study that assessed the ability of clinicians to allocate various symptom statements to competing diagnostic categories (PTSD, major depression, generalized anxiety disorder) [14]. Because of its design, this study examined only content validity, not criterion validity. Moreover, this study did not consider the diagnosis of actual patients, nor did it speak to the basic problem of substantial symptom overlap in *DSM’s* criteria sets. Furthermore, the IOM report made no mention of multiple studies that have documented the high comorbidity and phenomenological overlap of PTSD with other disorders [15–17].

Other aspects of the IOM report give us considerable pause. The committee cited a single study to suggest that reduced hippocampal volume “has been found to be associated with PTSD,” without referencing numerous failures to replicate this finding [18,19] and without observing that similar findings have been obtained for conditions such as depression that share high comorbidity with PTSD [20,21]. The committee noted that PTSD has been “documented worldwide in postconflict settings” (p. 50), without attending to a growing literature on the cultural and historical variability of PTSD symptoms [22–25].

Moreover, the IOM report neglected to note that the validity of a construct is interpretable only within a theoretical context [26]. For example, the IOM report pointed to the familial aggregation of PTSD with other anxiety disorders and offered this finding in support of one of the Robins and Guze criteria for validity. Yet substantial familial aggregation should be expected only if a disorder is presumed to be caused primarily by genetic or shared environmental influences. In the case of PTSD, the presumed primary etiologic agent—a traumatic stressor—is typically a nonshared environmental event, meaning that it is usually experienced by some family members but not others [27]. Hence, the familial aggregation of PTSD with other anxiety

disorders does not strongly support the construct validity of PTSD as presently construed. Instead, this finding suggests that the etiology of PTSD may be because of, as much if not more, a shared nonspecific diathesis toward anxiety rather than to a traumatic stressor.

In summary, the IOM report is remarkable for failing to provide any hint of the intense scientific controversy or significant unresolved questions that concern the validity of PTSD. In making this observation, we do not question the heuristic value PTSD has had in generating research nor do we conclude that PTSD has no clinical use. Instead, we argue that the evidence for the validity of PTSD remains preliminary and at best mixed [3,28–30]. The serious challenges that beset the validity of this diagnosis cannot merely be brushed aside by adopting a lenient threshold for scientific evidence. Issues and controversies must be acknowledged and explored openly if science is to advance our understanding of the causes and consequences of posttraumatic psychiatric morbidity.

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