

EDITORIAL

The uniqueness of the DSM definition of post-traumatic stress disorder: implications for research¹

The official definition of post-traumatic stress disorder (PTSD) in DSM-III and in subsequent DSM editions is based on a conceptual model that brackets traumatic or catastrophic events from less severe stressors and links them with a specific syndrome. The diagnosis of PTSD requires an identifiable stressor and the content of the defining symptoms refers to the stressor, for example, re-experiencing the stressor and avoidance of stimuli that symbolize the stressor. Temporal ordering is also required: when sleep problems and other symptoms of hyperarousal are part of the clinical picture, they must not have been present before the stressor occurred. The ICD-10 definition of PTSD follows the same model. The defining symptoms alone, without a connection to the stressor, are not regarded as PTSD (Green *et al.* 1995). Since the introduction of PTSD in DSM-III, the official definition has been adopted in most studies, although discussions about the validity of the definition have continued (Breslau & Davis, 1987; Davidson & Foa, 1993; Green *et al.* 1995). Although it is widely believed that other disorders (e.g. major depression) can be precipitated by external events, these disorders can occur independent of stressors and do not require a link with a traumatic event in their diagnostic criteria. Previous classifications that separated major depression into stress-related (reactive) or endogenous have been abandoned in newer versions of the DSM, because of lack of evidence of the validity of this distinction.

We discuss two methodological issues that have surfaced by the conceptualization of PTSD as a disorder in which an aetiological stressor is a necessary feature. The first concerns the diagnostic procedure for ascertaining the DSM-PTSD, and the second concerns co-morbid disorders that are defined in the DSM without reference to an aetiological stressor. The discussion applies to studies that adopt the DSM definition. The validity of the DSM definition of PTSD is not our concern here.

DIAGNOSTIC PROCEDURES: LINKING PTSD SYMPTOMS TO A SPECIFIC STRESSOR

In the standardized field assessments used in epidemiological research, the NIMH-DIS (Robins *et al.* 1989) and the WHO-CIDI (World Health Organization, 1990), PTSD is operationalized according to the DSM definition. These interview schedules pose questions about PTSD defining symptoms in reference to a specific traumatic event experienced by the respondent. For example, the DIS asks ‘Did you keep remembering the event when you didn’t want to?’ ‘Did you go out of your way to avoid activities or situations that might have reminded you of the event?’ ‘After the event, did you lose interest in doing things that used to be important to you?’ In the DIS or the CIDI, persons with a history of multiple traumas are asked to identify the worst or most distressing event, and PTSD symptoms are assessed in relation to that event. The interview permits an enquiry of PTSD symptoms in relation to several other traumatic events, and some persons might meet PTSD criteria in relation to more than one trauma. Recently, we have applied a method that involves the random selection of events from the list of each respondent’s qualifying events (one per

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respondent) and the assessment of PTSD symptoms in relation to that event. The approach was designed to provide an unbiased estimate of the conditional probability of PTSD given exposure, based on a representative sample of events from the entire pool of qualifying events (Breslau *et al.* 2000).

Several researchers have criticized the assessment of PTSD symptoms in reference to a specific event (Green, 1990; Solomon & Canino, 1990) and have suggested that the enquiry of PTSD symptoms be separated from the enquiry about traumatic events. Consistent with the criticism, Resnick *et al.* (1993) have applied a modified interview schedule, in which the enquiry focuses first on history of traumas, and then on PTSD symptoms, without reference to a specific event. The authors do not regard their approach as a change of the DSM definition of PTSD, but rather as an improved operationalization. They claim that the modified instrument has major advantages in that 'it does not require insight on the part of the respondent about symptom–event correspondence'. They also argue that their approach allows for a 'straightforward assessment of symptoms' presence in individuals who may have experienced multiple traumatic events'. There is some justification for the authors' first concern: one might doubt the respondents' ability to accurately attribute their mental disturbances to a specific traumatic experience. However, the approach amounts to assessing PTSD as if it were a configuration of defining symptoms, without the distinctive feature that requires that the individual symptoms be linked to a specific identifiable stressor. Symptoms of PTSD are not diagnostically specific most of them characterize other mental disorders and are used in the definition of those disorders, and some might result from abuse of psychoactive substances. It is their connection to a specific stressor that transforms the list of PTSD symptoms into a distinct DSM disorder.

Resnick *et al.*'s (1993) assumption that the attribution of symptoms to a stressor might be particularly hard for persons with multiple traumas also has some merit. However, disconnecting symptoms from a specific event might result in diagnosing PTSD in persons with a history of two or more events, by adding-up some symptoms connected with one event with other symptoms connected with another event, even if neither event, on its own, led to the full syndrome. Although these symptoms might have clinical utility, the ascertainment approach blurs and distorts the diagnosis as defined in the DSM.

TRAUMATIC EVENTS AND DISORDERS OTHER THAN PTSD: THE APPLICATION OF STANDARD RISK FACTOR ANALYSIS

Although the high co-occurrence of other psychiatric disorders with PTSD is well documented, the pathways that might explain this phenomenon remain unclear. Several alternative explanations have been suggested: (1) a pre-existing psychiatric disorder might increase a victim's susceptibility to the PTSD-inducing effects of the trauma; (2) a psychiatric disorder might increase the risk for exposure to traumatic events; (3) PTSD might lead to the onset of a secondary disorder; (4) stressors that lead to PTSD might also increase the risk for another disorder. Because in PTSD there is always an identified aetiological stressor, a logical question is 'Could not the same stressor also have caused the co-morbid disorder via a separate and distinct pathway?'. This last hypothesis has gained increased attention in recent research (Shalev & Yehuda, 1998; Yehuda *et al.* 1998).

To test whether exposure to PTSD-level stressor increases the risk for the onset of disorders, standard epidemiological methods are applicable. A risk factor for a disorder is evaluated by comparing the incidence rate of the disorder in persons with the risk factor (here, exposure to PTSD-level traumatic events) *versus* persons without the risk factor (here, no history of exposure). The hypothesis concerning the potential effect of traumatic events on a disorder other than PTSD, say, major depression, would be supported by evidence that the incidence of major depression is higher in persons exposed to traumatic events who did not succumb to PTSD, relative to an unexposed reference group. In contrast, evidence of an increased risk for major depression in exposed persons with PTSD (relative to unexposed) but not in exposed persons without PTSD would not support the hypothesis that the exposure to traumatic events is the cause. It would

suggest instead that PTSD might cause 'secondary' major depression or that PTSD and major depression share an underlying vulnerability (Chicoat & Breslau, 1998; Breslau *et al.* 2000).

The definitional distinction between PTSD and other disorders, in terms of the relationship to traumatic events, has not been universally attended to throughout the research literature. A recent example is Neria & Bromet (2000). They question the validity of inferences regarding the association between exposure to traumatic events and major depression, drawn from a comparison of exposed persons *versus* persons who were not exposed (Breslau *et al.* 2000). They note that standardized diagnostic interviews enquire about PTSD in relation to a traumatic event, whereas major depression (or other disorders) are ascertained without reference to traumatic events. They argue that this 'procedural difference will inevitably reduce the strength of the relationship found between extreme stress and these psychiatric disorders relative to PTSD'. Neria & Bromet's argument is based on a mistaken analogy between the measurement of the risk for PTSD and the measurement of the risk for major depression (or another disorder) associated with traumatic events. They fail to see the essential difference between the two. Because PTSD cannot occur without exposure, the risk for PTSD following exposure is measured by a conditional probability, which ranges from 0 to 1, and is the proportion of PTSD cases among persons exposed to traumatic events. In contrast, the definition of major depression does not require exposure to a traumatic event, and the exposure–depression association is measured by a relative risk, that is, by comparing the incidence of depression in exposed persons *versus* a reference group of unexposed persons. The conditional risk of PTSD and the relative risk of depression in victims of trauma cannot be directly compared. The critical point here is that the method of measuring the potential effects of PTSD-level traumatic events on the onset of disorders other than PTSD is different from the method used for measuring the traumatic events – PTSD relationship. In PTSD, there can be no measurement of relative risk with respect to the presence of a stressor *versus* the absence of a stressor, because PTSD, by definition, cannot occur without a stressor.

If, instead of using a reference group of unexposed persons, we estimated the risk for major depression following exposure based on respondents' attributions of depressive symptoms to a specific stressor, as we do for PTSD, we could not test whether exposed persons are at a higher risk for depression than they would have been if they were not exposed. And this is just what we need to know, because the possibility that the incidence of depression following exposure to stressors is not increased must be empirically refuted. In the case of PTSD, there is no such alternative, because there can be no PTSD without exposure. The difference between measuring PTSD and measuring major depression in the DIS or the CIDI is not a 'procedural difference', as Neria & Bromet call it, but a definitional difference. It would be a procedural difference if the same feature was a diagnostic criterion for both disorders, but the DSM directs us to detect the stressor criterion for PTSD only and not for major depression.

A fundamental premise of the DSM construction of PTSD is that, following exposure to traumatic events, the clinical features coalesce into an identifiable syndrome of PTSD. Neria & Bromet may be speculating beyond the DSM classification and may postulate that this syndrome coalescence occurs in the absence of traumatic events. Although they might be right, we are not aware of evidence along these lines. They also may be speculating that syndromes other than PTSD are coalescing as well in response to traumatic exposure (e.g. a post-traumatic depression syndrome). However, the existence of alternative syndromes does not preclude coalescence of PTSD, as described in the current DSM criteria.

RATES OF PTSD AND OTHER DISORDERS IN SAMPLES OF VICTIMS: WHAT DO THEY TELL?

Studies of survivors of civilian disasters and clinical studies of patients admitted to hospital following severe injury have generally reported markedly increased rates of co-morbid disorders among victims who succumbed to PTSD, compared to victims who did not succumb to PTSD (Kulka *et al.* 1990; Shalev *et al.* 1998; North *et al.* 1999). These findings do not support the notion

that exposure in stressors is the cause of the co-morbid disorders. If exposure were the cause, exposed persons without PTSD would be equal to those with PTSD with respect to the incidence rates of the other disorders. It should be noted that, a higher incidence of another disorder in persons with PTSD, compared to exposed persons without PTSD, does not in itself rule out the possibility that exposure might have played a part. A higher incidence of another disorder in persons with PTSD *versus* exposed persons with no PTSD might reflect the added effect of PTSD on the onset of a disorder, apart from the effect of exposure. To test the role of exposure *per se*, it is necessary to compare exposed persons with PTSD and without PTSD to unexposed persons; only this comparison can test whether exposure to traumatic events, independent of PTSD, is associated with an increased risk for the other disorder. Evidence of a higher incidence of another disorder in exposed persons with PTSD (relative to unexposed) but not in exposed persons without PTSD would not support the hypothesis that exposure plays a role.

CONCLUSIONS

The unique definitional feature of the DSM PTSD, as opposed to the disorders that are often diagnosed with it, chiefly major depression, anxiety disorders and substance use disorders, has important implications for research. These implications have not been systematically considered. As long as the DSM definition of PTSD requires an identifiable stressor and the linking of defining symptoms to the stressor, this essential feature cannot be ignored without the hazard of distorting the integrity of the diagnosis as explicitly defined. On the other hand, extending the required aetiological stressor in PTSD to other disorders that are not defined in relation to a stressor hinders efforts to address important questions concerning the causal relationships of trauma and PTSD with other disorders.

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