

Review

CONSIDERING PTSD FOR DSM-5

Matthew J. Friedman, M.D. Ph.D.,^{1,2} Patricia A. Resick, Ph.D.,^{1,3,4} Richard A. Bryant,⁵ and Chris R. Brewin⁶

This is a review of the relevant empirical literature concerning the DSM-IV-TR diagnostic criteria for PTSD. Most of this work has focused on Criteria A1 and A2, the two components of the A (Stressor) Criterion. With regard to A1, the review considers: (a) whether A1 is etiologically or temporally related to the PTSD symptoms; (b) whether it is possible to distinguish “traumatic” from “non-traumatic” stressors; and (c) whether A1 should be eliminated from DSM-5. Empirical literature regarding the utility of the A2 criterion indicates that there is little support for keeping the A2 criterion in DSM-5. The B (reexperiencing), C (avoidance/numbing) and D (hyperarousal) criteria are also reviewed. Confirmatory factor analyses suggest that the latent structure of PTSD appears to consist of four distinct symptom clusters rather than the three-cluster structure found in DSM-IV. It has also been shown that in addition to the fear-based symptoms emphasized in DSM-IV, traumatic exposure is also followed by dysphoric, anhedonic symptoms, aggressive/externalizing symptoms, guilt/shame symptoms, dissociative symptoms, and negative appraisals about oneself and the world. A new set of diagnostic criteria is proposed for DSM-5 that: (a) attempts to sharpen the A1 criterion; (b) eliminates the A2 criterion; (c) proposes four rather than three symptom clusters; and (d) expands the scope of the B–E criteria beyond a fear-based context. The final sections of this review consider: (a) partial/subsyndromal PTSD; (b) disorders of extreme stress not otherwise specified (DESNOS)/complex PTSD; (c) cross-cultural factors; (d) developmental factors; and (e) subtypes of PTSD. *Depression and Anxiety* 0:1–20, 2010. © 2010 Wiley-Liss, Inc.

Key words: PTSD; DSM-IV; DSM-V; diagnostic criteria; posttraumatic; syndromes

STATEMENT AND SIGNIFICANCE OF THE ISSUES

A number of key questions are being considered as we move forward with the development of The American Psychiatric Association’s (APA) fifth Diagnostic and Statistical Manual of Mental Disorders (DSM-5), regarding posttraumatic stress disorder (PTSD). These include: (1) Should the stressor criterion (Criterion A) be revised?; (2) Should other diagnostic criteria be revised and, if so, which ones?; and (3) Should other proposed posttraumatic syndromes also be included in DSM-5? We begin with a review of earlier conceptualizations of symptoms following exposure to traumatic events.

Poets, dramatists, and novelists, such as Homer, Shakespeare and Dickens, were the first to record the profound impact of traumatic stressors on cognitions,

¹National Center for PTSD, U.S. Department of Veterans Affairs, Vermont

²Dartmouth Medical School, Hanover, New Hampshire

³VA Boston Healthcare System, Massachusetts

⁴Boston University School of Medicine, Boston, Massachusetts

⁵School of Psychology, University of New South Wales, Sydney, New South Wales

⁶University College London, London, United Kingdom

¹Correspondence to: Matthew J. Friedman, National Center for PTSD, VA Medical Center, 215 North Main Street, White River Junction, VT 05009. E-mail: Matthew.Friedman@Dartmouth.edu

The authors report they have no financial relationships within the past 3 years to disclose.

Received for publication 19 March 2010; Revised 24 September 2010; Accepted 29 September 2010

DOI 10.1002/da.20767

Published online in Wiley Online Library (wileyonlinelibrary.com).

feelings and behavior. Clinical descriptions began to appear in the mid-nineteenth century as psychiatrists and other physicians on both sides of the Atlantic described syndromes among combat veterans (e.g. soldiers heart, Da Costa's syndrome, traumatic neurosis, shell shock, combat fatigue, neurocirculatory asthenia, etc.) and civilians (e.g. railway spine) that embody many, if not all, current PTSD symptoms.^[1] Different explanatory models proposing mechanisms through which traumatic stress might lead to (what is now called) PTSD were provided by psychoanalytic theory, Pavlovian fear conditioning models, Mowrer's two-factor theory, Selye's theories of stress and adaptation, and cognitive theories and neurobiology.^[1-3]

In DSM-I,^[4] "gross stress reaction" was an ill-defined diagnosis for classifying individuals who had been psychologically altered by exposure to military or civilian experiences. It was a useful diagnosis for initially classifying military veterans, ex-prisoners of war, rape victims, and Nazi Holocaust survivors. From a DSM-III perspective, however, the major problem was that gross stress reaction was considered a "temporary diagnosis," which would be changed to a "neurotic reaction" if the condition persisted.

DSM-II^[5] eliminated this diagnosis, leaving practitioners with no diagnostic option by which to classify clinically significant and persistent reactions to catastrophic experiences. "Situational Reaction" was the only diagnostic alternative. Because it included the full spectrum of adverse events from traumatic events to unpleasant experiences, it was seen as trivializing the impact of traumatic exposure. Furthermore, as with the DSM-I gross stress reaction, it was also considered a temporary and reversible clinical condition. By the mid- to-late 1970s, many mental health clinicians recognized the need for a new diagnosis for patients suffering from severe, chronic and sometimes irreversible syndromes following exposure to catastrophic events. Although not included in DSM-II, a number of syndromes had been described in the professional literature by that time, all named after the traumatic event itself such as: rape trauma syndrome, post-Vietnam syndrome, prisoner-of-war syndrome, concentration camp syndrome, war sailor syndrome, child abuse syndrome, battered women's syndrome, etc. The exciting new formulation that emerged during the DSM-III process^[6] was that all of these discrete syndromes could be adequately characterized by the specific symptoms proposed in the PTSD diagnostic criteria.

There have been some alterations of the original DSM-III PTSD criteria. The number of possible symptoms has increased from 12 to 17. The original three symptom clusters (e.g. reexperiencing, numbing, and miscellaneous) have been shuffled slightly to the present triad (e.g. reexperiencing, avoidance/numbing, and hyperarousal—see Table 1). But the fundamental construct, built into the diagnostic criteria, that exposure to overwhelming stress may precede the

TABLE 1. DSM-IV-TR diagnostic criteria for PTSD

- A1. The person experienced, witnessed, or was confronted with
an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others
- A2. The person's response involved intense fear, helplessness or horror
- B. Re-experiencing Symptoms (Requires one or more of):
- B1. Intrusive recollections
B2. Distressing nightmares
B3. Acting/feeling as though event were recurring (flashbacks)
B4. Psychological distress when exposed to traumatic reminders
B5. Physiological reactivity when exposed to traumatic reminders
- C. Avoidant/Numbing Symptoms (Requires three or more of):
- C1. Avoidance of thoughts, feelings or conversations associated with the stressor
C2. Avoidance of activities, places or people associated with the stressor
C3. Inability to recall important aspects of traumatic event
C4. Diminished interest in significant activities
C5. Detachment from others
C6. Restricted range of affect
C7. Sense of foreshortened future
- D. Hyperarousal Symptoms (Requires two or more of):
- D1. Sleep problems
D2. Irritability
D3. Concentration problems
D4. Hypervigilance
D5. Exaggerated startle response
- E. Duration of the disturbance is at least 1 month Acute—when the duration of symptoms is less than 3 months Chronic—when symptoms last 3 months or longer
With Delayed Onset—at least 6 months have elapsed between the traumatic event and onset of symptoms
- F. Requires significant distress or functional impairment

onset of clinically significant and persistent alterations in cognitions, feelings, and behavior has endured. Epidemiological studies have confirmed the DSM-III perspective and shown that exposure to extreme stress sometimes precedes severe and long-lasting psychopathology.^[7-11]

It has also become apparent that although specific PTSD symptoms (e.g. nightmares, avoidance behavior, hypervigilance, etc.) often are seen in the temporary distress exhibited by acutely traumatized individuals who recover normal function within days or weeks,^[12] it is the persistence of such symptoms that characterizes what is pathological about PTSD.^[13] In short, it appears that PTSD reflects a failure of adaptation, whereby normal acute reactions to extreme stress do not correct themselves over time.^[14,15]

METHOD OF LITERATURE REVIEW

We now review the DSM-IV-TR PTSD criteria (see Table 1) and propose how they might be improved in DSM-5. A search was conducted of the National Center for PTSD Published International Literature on Traumatic Stress database covering 1994–2010. Titles

and abstracts were searched using the key words: PTSD; Criteria A, B, C, D, E, F; Partial/Subsyndromal PTSD, DESNOS, Cross-Cultural Factors, Developmental Issues, Confirmatory Factor Analysis, and Dissociation. Relevant articles were supplemented by key reviews and analyses that preceded 1994.

RESULTS

THE A1 (STRESSOR) CRITERION

A distinctive feature of the diagnostic criteria for PTSD is the importance of traumatic exposure as stipulated in the A (stressor) Criterion. An excellent comprehensive review of this hotly debated matter can be found elsewhere.^[16] Indeed, in DSM-III, Criterion A was defined as “a recognizable stressor that would evoke significant symptoms of distress in almost anyone.” Drawing on general beliefs in the 1970s, it was thought that such experiences (e.g. rape, war, the Nazi Holocaust) were “generally outside the range of usual human experiences.” The DSM-III-R^[17] revision of DSM-III retained this language but stated in the clarifying text that traumatic exposure “is usually experienced with intense fear, horror, and helplessness.” DSM-III-R also widened traumatic exposure to include “learning about serious threat or harm to a friend or relative.” By the time DSM-IV^[18] was published, there was sufficient epidemiological research indicating that traumatic exposure was, unfortunately, not unusual but a relatively common occurrence among men, women, and children.

The DSM-IV Criterion A was divided into objective (A1) and subjective (A2) components. The A1 Criterion resembled the DSM-III-R Criterion A, except that a greater number of events were included as potential stressor events. These included: being diagnosed with a life threatening illness, child sexual abuse (without threatened or actual violence), learning about the sudden unexpected death of a family member or close friend, and learning that one’s child has a life threatening illness. In DSM-IV, however, in addition to exposure to an A1 event, it was necessary that exposed individuals experience an intense emotional reaction (Criterion A2) characterized as “fear, helplessness, or horror.” Although this had been foreshadowed in the DSM-III-R text description, the subjective response was now made an explicit (A2) Criterion. It is also worth noting that the timing of A2 was unclear and later subjected to different interpretations, with some saying it may happen after the event rather than being strictly peri-traumatic.

There were also distinctions between PTSD and adjustment disorder that are worth noting. First, it was stated in DSM-III that adjustment disorder results from a less severe (non-traumatic) stressor and does not include the PTSD reexperiencing (B), avoidance/numbing (C) and hyperarousal (D) symptoms. In contrast, DSM-IV states that individuals who develop B, C, and D symptoms following a low-magnitude (non-A1) stressor

should be diagnosed as having an adjustment disorder. This situation is incongruous because it allows for cases where the same symptoms can lead to two different diagnoses. Another distinction in DSM-III was that adjustment disorder was originally considered a time-limited disorder that resolves when the environmental precipitant has disappeared, whereas a chronic PTSD may persist for decades or a lifetime. DSM-IV, however, added a new category, “chronic adjustment disorder” that applies when the stressor persists for more than 6 months or when a stressor has “enduring consequences.” In other words, the duration of a post-stressor reaction can no longer distinguish PTSD from an adjustment disorder.

As we consider the DSM-IV A1 Criterion, there are several questions that must be addressed: (1) Should exposure to a potentially traumatic event be considered etiologically or temporally significant with regard to the later diagnosis of PTSD? (2) Can we really distinguish “traumatic” from “non-traumatic” stressors? and (3) Should the A1 Criterion be eliminated from DSM-5?

Etiological or temporal significance? The DSM-III and DSM-IV are unclear about the etiological significance of the traumatic event to the development of PTSD. In DSM-III, Criterion A refers to a “recognizable stressor that would evoke significant symptoms of distress in almost everyone” (p 238); in short, when B–D symptoms develop following A1 exposure, it presumes that the stressor has “caused” PTSD. DSM-IV-TR (p 463) also suggests that traumatic exposure is etiologically significant by referring to “characteristic symptoms resulting from exposure to trauma.” On the other hand, in the narrative section on PTSD (p 236) DSM-III states that “the essential feature of PTSD is the development of characteristic symptoms following a psychologically traumatic event”; in short, the stressor is an experience that temporally precedes the onset of PTSD. This statement is repeated in DSM-IV where it opens the discussion of PTSD (p 463).

Since 1980, we have learned that people differ with regard to risk of persistent PTSD symptoms following traumatic exposure and that most exposed individuals recover from traumas. As research on risk factors has been augmented by more recent findings regarding gene ! environment interactions, it has become clear that genetically based differences in resilience probably play a role in moderating the psychological impact of traumatic stress.^[19] Other risk factors, such as peri-traumatic dissociation, peri-traumatic negative emotions, or social support, also play a significant role in recovery.^[20–21] Considering individual differences regarding the likelihood of persistent PTSD symptoms following exposure to very stressful events, we must also recognize that events differ with regard to the conditional probability that PTSD will follow exposure. The conditional probability that PTSD will follow rape, for example, is much higher than that for exposure to natural disasters. In other words, there

is a complex interaction between individual susceptibility and the toxicity of a given stressful event. Therefore, while we acknowledge that no event, in and of itself, can cause PTSD, we must also recognize that some events are much more likely to do so than others.

Dohrenwend^[22] has developed a methodology for assessing the potential toxicity of different events. Reframing the stressor as a temporal antecedent with a different conditional probability of preceding the development of PTSD tempers the attribution of causality and makes it possible to retain a stress–diathesis (or vector–host) model of PTSD and to incorporate our growing understanding of how clinical outcomes are influenced by risk/protective factors and gene ! environment interactions. In short, exposure to an A1 event is a necessary but not sufficient condition for the subsequent development of PTSD.

Can we distinguish “traumatic” from “non-traumatic” events? As we consider temporal, vulnerability, and toxicity factors associated with exposure to A1 events, we must be careful to strike a proper balance so that the basic PTSD construct can remain a useful diagnosis. McNally^[23] has warned, “shifting the causal emphasis away from the stressor undermines the very rationale for having a diagnosis of PTSD in the first place” (p 598). Thus the question is, can we draw a line between “traumatic” stressors that characterize the A1 Criterion and “non-traumatic” stressors that might precede an adjustment reaction but, by definition, cannot precede PTSD?

As noted by Kilpatrick et al.^[24] when summarizing findings from the DSM-IV Field Trials, the argument over how to operationalize the A Criterion boiled down to a debate over how broad versus how narrow Criterion A should be. Proponents of the broad definition argued that Criterion A should include any event that can produce the PTSD symptoms. Advocates for a more restrictive definition feared that broadening the criterion would trivialize the PTSD diagnosis and defeat the purpose of the original DSM-III PTSD construct by permitting people exposed to less stressful events to meet the A Criterion. The DSM-IV Field Trials appeared to allay this concern because few people developed PTSD unless they experienced extremely stressful life events. Kilpatrick et al.^[25] have recently replicated the Field Trial findings in two independent cohorts, the Florida Hurricane Study (FHS) and the National Survey of Adolescents (NSA). They found that among the FHS study participants, 96.6% of those meeting PTSD B-F Criteria had previously been exposed to an A1 event. In the NSA study, 95.5% of those meeting B–F Criteria had been exposed to an A1 traumatic stressor. In other words, they found that very few people meet full PTSD diagnostic criteria without prior exposure to a recognizable traumatic event, as stipulated in DSM-IV.

On the other hand, concern has been expressed about the greater number of qualifying A1 events in DSM-IV

in comparison to DSM-III. It has been argued that expansion of qualifying A1 events has diluted the basic PTSD construct and permitted people to receive the PTSD diagnosis for less threatening events that should really be associated with an adjustment disorder or anxiety disorder NOS.^[25] This expansion has been called “bracket creep”^[23] or “criterion creep”^[26] and is presumed to have a particularly adverse impact in forensic settings or disability evaluations where it has been blamed for frivolous tort or compensation claims.

Breslau and Kessler^[27] tested the implications of the broad DSM-IV A1 Criterion that stipulated the events that are not included in DSM-III. Among a representative sample of 2,181 individuals, lifetime exposure to traumatic events was 68.1% when estimated by a narrow set of qualifying A1 events that included seven events of “assaultive violence” (e.g. combat, rape, assault, etc.) and seven “other injury events” (e.g. serious accident, natural disaster, witnessing death/serious injury, etc.). When the A1 Criterion was expanded to include five events from the expanded DSM-IV A1 definition, such as “learning about” traumatic events to close relatives (e.g. rape, assault, accident, etc.), lifetime prevalence of exposure to traumatic events increased to 89.6%. Thus, there was a 59.2% increase in lifetime exposure to a traumatic event due to the expanded A1 Criterion. More importantly, A1 events included within the expanded A1 Criterion contributed 38% of the total PTSD cases. Weathers and Keane^[16] have suggested that the wide discrepancy between the Kilpatrick et al.^[24] and Breslau and Kessler^[27] studies may have more to do with methodology than with the A1 Criterion, itself. Because the two studies used different methodological approaches, they cannot be directly compared.

Kilpatrick et al.^[25] have disputed the “bracket/criterion creep” arguments. They point both to the DSM-IV Field Trial as well as the aforementioned FHS and NSA data, all of which indicate that very few individuals meet B-F PTSD criteria without prior exposure to an A1 event. Brewin et al.^[14] make a similar argument. Non-A1 events most likely to precede the onset of PTSD B-F symptoms in both the DSM-IV Field Trial^[24] and the Breslau and Kessler^[27] study were sudden death, serious illness, or diagnosis of a child with a potentially terminal illness.^[25] This was also observed in the NSA where 80% of adolescents meeting B–F criteria in the absence of A1 reported a past year death or serious illness.^[25] So should these non-A1 events be designated A1 events? If so, will that dilute the PTSD construct?

Should the A1 criterion be eliminated? It has been proposed that it does not matter whether a broad or narrow definition is set for A1 because what really matters is whether people meet Criteria B, C, D, E, and F for PTSD and that PTSD caseness and prevalence would change very little if the A1 Criterion were completely eliminated. For example, Kilpatrick et al.^[25] found that of 1,543 adults exposed to one of

four Florida hurricanes, the PTSD prevalence was 11.6% (179 of 1,543) with no A1 (or A2) requirement and 11.2% (173 of 1,543) with the requirement for A1 (but not A2).

The DSM-IV PTSD Work Group also considered complete elimination of Criterion A. Although it acknowledged the possibility that someone might meet B, C, and D Criteria without meeting the A Criterion, this option was rejected because of concerns that “the loosening of Criterion A may lead to widespread and frivolous use of the concept” ([28]; p 347). Several authors have suggested that the full PTSD syndrome may be expressed following non-traumatic events and have thereby fortified the “bracket/criterion creep” arguments.^[23,29] Brewin et al.^[13] dismiss most of these reports as methodologically flawed because proper clinical interviews were not utilized and because the data showed an increase in the PTSD symptoms, but not the full diagnosis. They conclude that when assessed by a structured clinical interview, there are actually very few examples of individuals who do not meet the A Criterion who meet full PTSD diagnostic criteria. A related question is whether the non-A1 event actually served as a reminder or trigger for a previously traumatic event. Most of the epidemiology studies have not controlled for prior trauma and prior PTSD.

Brewin et al.^[14] have also proposed eliminating the A1 Criterion completely. First, they point out that there is not a unique relationship between the stressor criterion and PTSD because depression, other anxiety disorders, and substance use disorder may also follow exposure to an A1 event. Second, they cite evidence that non-A1 events may in some cases plausibly precede expression of B–F symptoms (e.g. repeated exposure to less intense but continuous threat). Finally, they discuss problems with the A2 Criterion (see below). Instead, they propose that Criterion A be abolished and that the PTSD diagnosis focus on a smaller cluster of symptoms (see B–D symptoms below). In this way, they argue that PTSD will be more comparable to affective and other anxiety disorders that focus exclusively on symptoms. According to Brewin et al., abolition of Criterion A will eliminate the need to draw a line between “traumatic” and “non-traumatic” stressors, will eliminate disagreements about the etiological versus temporal importance of Criterion A1, and will easily incorporate all that has been learned about gene ! environment interactions and individual differences regarding resilience.

Kilpatrick et al.^[25] disagree. They argue that shifting the focus from Criterion A to Criterion B (e.g. traumatic nightmares or flashbacks) or Criterion C (e.g. avoidance symptoms) still requires a judgment as to whether the focus of such symptoms is actually “traumatic.” In an unpublished survey of PTSD experts undertaken by APA as part of the DSM-5 process, there was a very strong support for retaining Criterion A1 but most experts proposed that it needed to be modified to address the issues discussed in this review.

Suggested modifications included: emphasizing the temporal, rather than presumed etiological relationship between A1 exposure and B–F symptoms and narrowing the criterion regarding second hand-exposure (e.g. the “learned about” criterion).

The major reason proposed for retaining Criterion A1 is that in the vast majority of cases PTSD does not develop unless an individual is exposed to an event or series of events that are intensely stressful. Such individuals are keenly aware of a significant discontinuity in their lives because of subsequent preoccupation with memories, feelings, and behaviors that are associated with that event. McNally^[23] has argued that the memory of the trauma is the “heart of the diagnosis” and the organizing core around which the B–F symptoms can be understood as a coherent syndrome. “One cannot have intrusive memories in the abstract. An intrusive memory must be a memory of something and that something is “the traumatic event” (p 599). Weathers and Keane^[16] emphasize that a qualifying A1 event must be one that entails “personal involvement with, if not direct exposure to catastrophic life events (p 115).”

After reviewing all of this evidence, the DSM-5 Work Group was persuaded that it was necessary to preserve Criterion A1 as an indispensable feature of PTSD. Intrusion and avoidance symptoms are incomprehensible without prior exposure to a traumatic event. The traumatic experience is usually a watershed event that marks a major discontinuity in the life trajectories of individuals affected with PTSD, unless the onset of the disorder is delayed. The language of A1 has been revised to emphasize that qualifying events must involve direct exposure to actual or threatened death, serious injury or a threat to the physical integrity of others.

A related question was whether A1 should be limited to direct exposure so that the “learning about” component of the A1 Criterion could be eliminated. Fortunately, there are data on indirect traumatic exposure to inform this decision. Specifically, a number of studies have assessed PTSD among family members whose spouse or child was murdered, assaulted sexually, killed in combat, killed in the 9/11 attack on the World Trade Center, or whose child died violently. Regarding indirect exposure to a traumatic event, the PTSD prevalence among 591 individuals who lost a family member due to homicide was 71.1%; it was 59.4% for physical assault and 55.2% for sexual assault of a family member.^[30] Forty-one percent of family survivors of homicide victims met PTSD criteria approximately 2.5 years after the trauma.^[31] Among 252 New Yorkers seeking primary care who had lost a close friend or family member in the 9/11 attacks, 17.1% met criteria for PTSD, compared to 7.7% of 677 primary care patients who had not experienced such losses.^[32] This same group of investigators also reported a PTSD prevalence of 21.5% among 843 adults who had lost a loved one during the 9/11

attacks.^[33] Two years after losing a child to a violent death, 21% of mothers and 14% of fathers continued to meet PTSD criteria.^[34] Among 37 mothers whose child survived the 1988 sinking of the cruise ship, “Jupiter,” 20 (54.1%) met criteria for PTSD.^[35]

Another aspect of indirect exposure concerns professionals who, though never in danger themselves, are exposed to the grotesque details of rape, genocide, or other abusive violence to others. Among military mortuary workers dealing with human remains after the USS Iowa gun turret explosion in 1989, PTSD prevalence was 11%.^[36] McCarroll et al. have documented elevations in PTSD prevalence among Gulf War military mortuary workers.^[37] Exposure to human remains by troops assigned to graves registration duties during the Gulf War was associated with 48 and 65% current and lifetime PTSD prevalence, respectively.^[38] Among Chinese rescue workers providing services after the 1999 Chi-Chi earthquake on Taiwan, PTSD prevalence was 21.4%.^[39] Dentists engaged in post-mortem dental identification following the 1993 fire at the Branch Davidian compound exhibited marked elevations in Impact of Event Scale scores compared to matched dentists who did not engage in that activity.^[40] Finally, two studies indicate that viewing television images of the 9/11 terrorist attacks^[41] or witnessing video footage of traumatic events in a newsroom^[42] are unlikely to lead to the PTSD symptoms.

An extensive review of this literature can be found elsewhere^[43] regarding elevated PTSD prevalence among civilian and military personnel exposed to traumatic death following combat, terrorism, and disasters. To summarize, “learning about” the death or traumatic exposure of a loved one has been shown to precede the onset of PTSD B–F Criteria symptoms in a significant number of family members and significant others, especially in the case of severe trauma such as homicide and violent death. Repeated exposure to human remains and other grotesque consequences of traumatic events among professionals who must endure such exposure in the course of their assigned duties may also lead to the onset of PTSD B–F symptoms. In contrast, exposure to such events through television or other electronic media is unlikely to provoke such symptoms.

As a result of this literature review, it was decided to retain the “learning about” component of Criterion A. The proposed DSM-5 revision limits such indirect exposure to learning about the traumatic exposure of a close friend or loved one or learning about aversive details of unnatural deaths, serious injury or serious assault to others. This includes learning about the homicide of a family member, learning about the gruesome death or grotesque details of rape, genocide, or other abusive violence to significant others. Learning about another person’s traumatic experience also applies to work-related exposure to gruesome and horrific evidence of traumatic events as with police

personnel, firefighters, graves registration workers, and emergency medical technicians. Finally, the revised A Criterion explicitly excludes witnessing traumatic events through electronic media, television, video games, movies, or pictures, unless this forms part of a person’s vocational role. Here is the A1 Criterion that has been proposed for DSM-5:

(A) The person was exposed to the following event(s): death or threatened death, actual or threatened serious injury, or actual or threatened sexual violation, in one or more of the following ways:

- (1) Experiencing the event(s) him/herself.
- (2) Witnessing the event(s) as they occurred to others.
- (3) Learning that the event(s) occurred to a close relative or close friend.
- (4) Experiencing repeated or extreme exposure to aversive details of the event(s) (e.g. first responders collecting body parts; police officers repeatedly exposed to details of child abuse).

Note: Witnessing or exposure to aversive details does not include events that are witnessed only in electronic media, television, movies or pictures, unless this is part of a person’s vocational role. Exposure to aversive details of death applies only to unnatural death.

THE A2 CRITERION

As noted above, the DSM-IV Work Group stipulated that exposure to an A1 event, per se, was not a sufficient condition for meeting the Stressor Criterion. Instead, individuals thus exposed must also experience an intense subjective reaction characterized as “fear, helplessness, or horror.” It was expected that the imposition of the A2 Criterion would insure that people would not be eligible for the PTSD diagnosis unless they had reacted strongly to a threatening event. It was also expected that imposition of the A2 Criterion would minimize any “frivolous” PTSD diagnoses due to broadening the A1 Criterion. Finally, based on data from the DSM-IV Field Trials,^[23] it was expected that few people exposed to low magnitude (non-traumatic) events would meet the A2 Criterion and therefore, would not be eligible for the PTSD diagnosis.

The utility of the A2 Criterion has been questioned. Brewin et al.^[44] found that intense levels of immediate post-exposure fear, helplessness, and horror were weakly predictive of PTSD 6 months later. They also found evidence that other posttraumatic emotional reactions (such as anger or shame) also predicted PTSD. There were, however, a small number of people who denied post-exposure A2 emotions who also met PTSD criteria at 6 months. Rizvi et al.^[45] in a prospective study of recent female rape or assault victims, reported similar findings. O’Donnell et al.^[46] reported that within a sample of A1 exposed individuals who went on to meet PTSD B–F criteria, a substantial minority, (23%), failed to receive a PTSD diagnosis because of the absence of A2. Furthermore, there were

no differences with regard to B–F symptom severity or impairment between the A2-positive and A2-negative cohorts. Creamer et al.^[47] examined a community sample of 6,104 adults with a history of trauma exposure and also found that a substantial minority of those who would otherwise have PTSD (24% males and 19% females) failed to meet the Criterion A2.

Three negative studies found no effect of A2 on PTSD prevalence: Breslau and Kessler^[27] in a community sample from Michigan; Schnurr et al.^[48] in a sample of older male military veterans; and Karam et al.^[49] in the World Health Organization’s World Mental Health Survey, which included almost 103,000 respondents.

An additional issue that needs to be recognized is that many people can develop PTSD B–F symptoms without having any emotional response to the event at the time. Trained military personnel may not experience fear, helplessness, or horror during or immediately following a trauma because of their training. They may only experience emotions after being removed from the war zone, which could be many months later. Many studies have shown that people can develop PTSD following mild traumatic brain injury (TBI), in which cases the person may be unaware of any peritraumatic emotional response because of the loss of consciousness.^[50] There is even evidence of PTSD following severe TBI, in which individuals were fully unaware of their responses for weeks or months after the event.^[51] These cases highlight the need to recognize that some people can develop PTSD in the absence of an A2 response. It also raises the question about the timeframe in which A2 is assessed. Most PTSD cases are evaluated months or years after a traumatic event, and the A2 requires a retrospective recall of how the person responded during or shortly after the event. There is evidence that recall of acute responses to trauma is unreliable and is influenced by mood biases associated with PTSD levels at the time of recall.^[52] This raises questions about the accuracy of retrospective A2 reports.

Based on such findings, a number of investigators have called for the elimination of the A2 Criterion because it does not enhance the identification of people who will develop PTSD and it has failed to counter the expansion of qualifying A1 events, discussed previously.^[46] Finally, McNally^[23] has argued that we should eliminate A2 because “in the language of behaviorism it confounds the response with the stimulus. In the language of medicine, it confounds the host with the pathogen” (p 598).

On the other hand, a consistent finding from five studies^[24,27,44,48,49] concerns the negative predictive value of A2. In other words, it is more useful for detecting people who will not develop PTSD than those who will. Schnurr et al.^[48] suggest that A2 can help to screen out individuals unlikely to develop PTSD. Although this may be extremely useful for some forms of screening, it does not appear to have a major

bearing on improving diagnostic accuracy. Based on these findings, it has been proposed that the A2 Criterion be eliminated in DSM-5.

THE B, C, AND D CRITERIA FACTOR STRUCTURE OF PTSD

The DSM-IV PTSD construct consists of three symptom clusters: B—reexperiencing, C—avoidance/numbing, and D—hyperarousal (see Table 1). Questions have been raised about how well this construct has held together in practice. In other words, what is the latent structure of PTSD? Are there three distinct symptom clusters? And are these three clusters subsumed by an over-arching construct, the PTSD diagnosis?

Many studies have utilized confirmatory factor analysis to test whether the three symptom clusters of DSM-IV provide the best model for the latent structure of PTSD. Different investigators have found two-, three- or four-factor models as the best fit for the data. As we review these findings, it is important to note that different PTSD assessment instruments were used by different investigators and that these instruments were administered to individuals whose PTSD developed from different types of traumatic experiences. It is also important to note that, contrary to good psychometric testing principles, almost all studies of the structure of PTSD have administered the PTSD items in the same order of the DSM criteria, such that covariation among symptoms could be affected by their position.

Only one study has supported the DSM-IV three-factor model,^[53] with three other studies supporting different three-factor models: reexperiencing/active avoidance, numbing/passive avoidance and arousal;^[54,55] as well as reexperiencing/avoidance, emotional numbing/hyperarousal, and hypervigilance/exaggerated startle.^[56]

Five studies support a two-factor solution although the specific factors have differed between studies. In four studies, the factors were characterized as reexperiencing/avoidance and numbing/hyperarousal.^[57–60] The fifth study found that a two-factor model in which intrusion/hyperarousal and an avoidance factor offered the best solution.^[61] It was noted in two of these studies that four-factor models also fit the data quite well and, perhaps, provided richer detail about symptoms.^[59,61]

Most studies have supported a four-factor model. Reexperiencing, avoidance, and arousal have emerged as distinct clusters in all of these studies. There has been some disagreement, however, about the fourth factor. In the majority of four-factor models, “numbing” emerged as a distinct cluster.^[62–70] In a few four-factor models, however, “dysphoria” (a combination of some numbing and hyperarousal symptoms also associated with depression) rather than “numbing” emerged as the fourth factor.^[71–73] A recent meta-analysis^[74] suggests that both are a good fit to the data but that the

four-factor solution with “dysphoria” enjoys a slight advantage over the four-factor solution with “numbing.” This general dysphoria factor might be considered to be related to the range of negative emotions frequently observed among individuals with PTSD.^[75] Brewin et al.^[14] have suggested that because of the overlap between the PTSD symptoms associated with this dysphoria factor and similar symptoms observed in depression and other anxiety disorders, they should be eliminated from the PTSD diagnostic criteria (see below). Finally, Andrews et al.^[70] reported that the model of best fit among a cohort of emergency personnel was a four-factor solution alongside a general PTSD factor.

Taken together, most confirmatory factor analyses support a four, rather than a three-factor DSM-IV model. Several studies indicate that serious consideration should be given to including a separate fourth, “numbing” symptom cluster in DSM-5. Furthermore, avoidance and numbing are consistently distinct from one another in both the four- and two-factor solutions. Other studies agree that emotional numbing is different than effortful avoidance but is better placed as one extreme along a range of negative emotions. Based on these findings, a four-factor model for PTSD has been proposed for DSM-5.

CAN THE B-D SYMPTOM CLUSTERS BE IMPROVED?¹

The empirical literature strongly suggests that, as noted earlier in this review, traumatic exposure may be followed by a variety of clinical presentations, including fear-based anxiety symptoms, dysphoric/anhedonic symptoms, aggressive/externalizing symptoms, guilt/shame symptoms, dissociative symptoms, and negative appraisals about oneself and the world. We have cited such information elsewhere,^[29,75,76] to argue that PTSD should be moved out of the Anxiety Disorders and classified within a separate category of event or trauma-related disorders. Here, we invoke such findings to suggest revisions to the DSM-IV PTSD diagnostic criteria (see Table 1), which, in our opinion, provide a better characterization of the spectrum of posttraumatic symptomatology encountered by clinicians on a regular basis.

CRITERION B

Brewin et al.^[13] propose that the two reexperiencing symptoms most characteristic of PTSD are flashbacks (B3) and traumatic nightmares (B2). Symptom B1

(intrusive recollections), they argue, is too similar to rumination seen in depression and other psychiatric disorders^[13] and therefore, is too nonspecific to be retained in PTSD. Item B1 in the DSM-IV is particularly problematic because it is worded “recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions.” In the DSM III, B1 required that the traumatic event is persistently reexperienced by “recurrent and intrusive distressing recollections of the event.” Not only does this wording include both stimuli and responses but also confuses imagery with thoughts. There is a growing body of evidence that intrusive imagery is quite different from thought processes such as rumination.^[75] Intrusive images in PTSD are sensory memories of short duration, have a here-and-now quality and lack context, while ruminative thoughts are evaluative and longer lasting.^[14,77–80] Furthermore, rumination appears to have a different function than intrusive symptoms. Ehrling et al.^[81–83] have conducted studies to examine rumination in PTSD. They found that rumination occurs both in response to intrusive imagery, but can also trigger intrusions. For the most part, rumination appears to function as a cognitive avoidance strategy. These findings should be explored further and may confirm that the reexperiencing symptoms should be limited to nightmares, flashbacks, and intrusive sensory experiences, (which could include auditory as well as visual, olfactory, tactile, etc. memories), but not abstract thoughts and appraisals about the traumatic event. Therefore, we believe that thoughts/ruminations should be eliminated from the B1 Criterion. Our proposal is to restrict this criterion to involuntary and intrusive distressing memories that usually include sensory, emotional, physiological, or behavioral (but not autobiographical memory) components. The emphasis is on spontaneous or triggered recurrent, involuntary, and intrusive distressing memories of the event that usually include sensory emotional, physiological, or behavioral components.

Criterion B2 (traumatic nightmares) is essentially unchanged, but has been loosened somewhat to include trauma-related material rather than requiring the dream to reproduce the traumatic event. The proposed Criterion B3 clarifies that the PTSD flashback is a dissociative reaction in which the individual experiences a sense of reliving the experience with sensory, emotional, physiological, or behavioral reactions and feels or acts as if the traumatic event were recurring.

Criteria B4 and B5 (emotional and physiological arousal) following exposure to traumatic reminders are, in the opinion of Brewin et al.^[14] too similar to symptoms found in specific and social phobic disorders and should also be eliminated because of overlap with these disorders. Emotional and physiological arousal are not actually intrusive symptoms, but are reactions to re-experienced imagery, so at most, they are indirect indicators that the traumatic memory is being re-experienced.

¹The proposed revision of B-D symptoms reviewed in this section is based on the work of the DSM-5 Trauma, PTSD, and Dissociative Disorders Sub-Work Group of the Anxiety Disorders Work Group. In addition to the authors, Dean Kilpatrick, Roberto Lewis-Fernandez, Katharine Phillips, David Spiegel, Robert Ursano, Robert Pynoos, Paula Schnurr, James Strain, Terry Keane, and Eric Vermetten participated in this process along with the authors.

A counter argument to this proposal is that B4/B5 elicitation of emotional and physiological reactivity to trauma-related stimuli is a key characteristic of PTSD. It is consistent with major fear conditioning models of the disorder. It is the rationale for critical laboratory paradigms in which distinctive alterations in psychological and neurobiological reactivity among PTSD participants can only be detected after exposure to trauma-related stimuli.^[84] And it is a principle that has been incorporated into our most effective exposure therapies where emotions and cognitions elicited by traumatic reminders are processed therapeutically.

In the proposed criteria for DSM-5, B4 and B5 are both retained and defined as triggered intrusive emotional and physiological experiences, respectively. B4 is an intense emotional distress that may be the only kind of recollection possible in individuals who sustained a TBI and have no conscious memories of the traumatic event. There is evidence that trauma survivors with severe TBI and with no memory of the event can still meet PTSD criteria because they satisfy B4 or B5 in response to traumatic reminders.^[51] In other words, these symptoms are conditioned responses in fear conditioning models. Here are the proposed Criterion B symptoms for DSM-5:

(B) Intrusion symptoms that are associated with the traumatic event(s) (that began after the traumatic event(s)), as evidenced by one or more of the following:

- (1) Spontaneous or cued recurrent, involuntary, and intrusive distressing memories of the traumatic event(s). Note: In children, repetitive play may occur in which themes or aspects of the traumatic event(s) are expressed (DSM-IV B₁).
- (2) Recurrent distressing dreams in which the content and/or affect of the dream is related to the event(s). Note: In children, there may be frightening dreams without recognizable content (DSM-IV B₂).
- (3) Dissociative reactions (e.g. flashbacks) in which the individual feels or acts as if the traumatic event(s) were recurring. (Such reactions may occur on a continuum, with the most extreme expression being a complete loss of awareness of present surroundings.) Note: In children, trauma-specific reenactment may occur in play (DSM-IV B₃).
- (4) Intense or prolonged psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event(s) (DSM-IV B₄).
- (5) Marked physiological reactions to reminders of the traumatic event(s) (DSM-IV B₅).

DSM-IV CRITERION C WILL BECOME DSM-5 CRITERIA C AND D

In keeping with previously reviewed confirmatory factor analyses, a four-factor model has been proposed. DSM-IV Criterion C will be divided into DSM-5 Criteria C and D.

Criterion C. Avoidance behavior (one symptom needed). As suggested by the literature, this cluster consists only of the two behavioral avoidance symptoms, which are completely unchanged from DSM-IV C1 and C2, as shown in Table 1.

Criterion D. Negative alterations in cognitions and mood. Brewin et al.^[14] propose that DSM-IV symptoms C3–7 should be eliminated because of overlap with depression and general dysphoric symptoms seen in other psychiatric disorders^[71] and with the anhedonic component of depression.^[85] There is evidence that removing items theorized to overlap with mood and other anxiety disorders has little effect on the prevalence, associated comorbidity, and functional impairment, or structural validity of PTSD.^[86] A counterargument is that the numbing/dysphoric component of the C Criterion is central to the PTSD syndrome because it encompasses a persistent negative mood state and negative cognitions associated with the traumatic event. Indeed, there are a number of negative appraisals and mood states associated with PTSD that constitute a separate cluster of symptoms distinct from reexperiencing, avoidance and arousal/reativity symptoms. They have been included in the proposed DSM-5 diagnostic criteria. Some current DSM-IV numbing (C3–7) symptoms have been retained, sometimes with clarifications or revisions in wording. In addition, two new symptoms have been proposed for this cluster.

Although catastrophic or maladaptive appraisals are very common across anxiety and mood disorders, it is important to include these in PTSD because there is overwhelming evidence that they are characteristic of traumatic stress responses that are associated with disorder or impairment.^[75] People with PTSD have erroneous cognitions about the causes or consequences of the traumatic event that leads them to blame themselves or others. This has been observed among survivors of childhood sexual abuse, rape/assault survivors, and military personnel. Addressing such self-blame or erroneous “other-blame” is a consistent component of cognitive behavioral therapy (CBT) with PTSD patients.^[87–89] A related erroneous appraisal is the common belief that one is inadequate, weak, or permanently changed for the worse since exposure to the traumatic event or that appraisals about the world and other people should be extremely altered because of the event (e.g. “nobody can be trusted, the world is entirely dangerous, people will always try to control me.”). This response has been reported in a range of populations, including emergency responders^[90] and adolescents,^[91] as well as victims of interpersonal violence.^[89,92] Such extreme negative appraisals about the self, others, or the world are so common in PTSD that a new symptom (DSM-5 D3) has been proposed for DSM-5.

Another maladaptive appraisal is DSM-IV symptom C7, a sense of foreshortened future, which we believe has been interpreted too narrowly in DSM-IV as the “belief that one’s life will be shorter or changed.”

As shown repeatedly in the empirical literature and consistently observed among people receiving CBT, patients with PTSD have persistent negative expectations about themselves, others, or their future (e.g. “I am a bad person”; “nothing good can happen to me”; “I can never trust again”). They do not expect to have a career, marriage, children, or a normal life span.^[90,92–96] In our opinion, this symptom should be retained (as DSM-5 D2) but understood to address persistent negative expectations regarding many important aspects of life rather than its current narrow restriction to negative expectations about one’s life span.

In addition to negative appraisals about past, present, and future, people with PTSD have a wide variety of negative emotional states besides fear, helplessness, and horror. As reviewed previously, one of the arguments for moving PTSD out of the Anxiety Disorders category is the presence of many other negative mood states. These include anger,^[92,97–100] guilt,^[101–103] and shame.^[97,104,105] An extensive review of this issue can be found in Miller and Resick.^[106] The strength of this evidence has convinced the DSM-5 Anxiety Work Group to propose that having a pervasive negative emotional state should be added as a new PTSD symptom (DSM-5 D4).

There is abundant evidence that other symptoms, currently included in the DSM-IV Numbing (C3–C7) cluster, should be retained in the proposed diagnostic criteria for PTSD. These include (DSM-IV C3, now DSM-5 D1) inability to remember at least one important aspect of the traumatic event. Such memory problems are often due to dissociative amnesia.^[107–108] Three other DSM-IV symptoms that should be retained are: diminished interest in significant activities (DSM-IV C4, now DSM-5 D5), feeling detached or estranged from others (DSM-IV C5, now DSM-5 D6) and psychic numbing, persistent inability to experience (mainly positive) emotions (DSM-IV C6, now DSM-5 D7). It should be noted that the DSM-5 definition has refined the psychic numbing definition to reflect difficulty in experiencing positive emotions on the basis of evidence that PTSD patients can strongly experience negative emotional states but can be nonresponsive to stimuli that would normally elicit a positive response.^[109] These are all consistently endorsed by individuals with PTSD as shown in the many confirmatory factor analyses reviewed previously.

To summarize, the weight of empirical evidence and confirmatory factor analyses suggest that the DSM-IV Avoidance/Numbing cluster be divided into two separate clusters: Persistent Avoidance of Stimuli associated with the trauma and Negative Alterations in Cognitions and Mood associated with the trauma. It appears that the dissociative aspects of amnesia should be emphasized for DSM-IV C3 and that the expectation of a reduced life span, foreshortened future (DSM-IV C7) be expanded to include negative expectations about one’s self, others, or one’s future. It has also been proposed that DSM-IV symptoms C4

(diminished interest in significant activities), C5 (feeling detached or estranged from others), and C6 (psychic numbing) should be retained unchanged, with the exception that C6 be more clearly focused on the inability to experience positive emotions. Finally, the empirical literature indicates that two new symptoms should be added: pervasive negative emotional state (e.g. fear, horror, anger, guilt, or shame) and persistent distorted blame of self or others about the cause or consequences of the traumatic event. Here are the proposed criteria for the new D Criterion in DSM-5:

(D) Negative alterations in cognitions and mood that are associated with the traumatic event(s) (that began or worsened after the traumatic event(s)), as evidenced by three or more of the following: (Note: In children, as evidenced by two or more of the following)[†]:

- (1) Inability to remember an important aspect of the traumatic event(s) (typically dissociative amnesia; not due to head injury, alcohol, or drugs) (DSM-IV C₃).
- (2) Persistent and exaggerated negative expectations about one’s self, others, or the world (e.g. “I am bad,” “no one can be trusted,” “I’ve lost my soul forever,” “my whole nervous system is permanently ruined,” “the world is completely dangerous”) (DSM-IV C₇).
- (3) Persistent distorted blame of self or others about the cause or consequences of the traumatic event(s) (new symptom).
- (4) Pervasive negative emotional state—for example: fear, horror, anger, guilt, or shame (new symptom).
- (5) Markedly diminished interest or participation in significant activities (DSM-IV C₄).
- (6) Feeling of detachment or estrangement from others (DSM-IV C₅).
- (7) Persistent inability to experience positive emotions (e.g. unable to have loving feelings, psychic numbing) (DSM-IV C₆).

[†]It should be noted that the proposed diagnostic thresholds (e.g. three D symptoms for adults and two for children) will be tested and possibly revised after the DSM-5 field trials.

DSM-IV CRITERION D WILL BECOME DSM-5 CRITERION E

Four of the five Criterion D symptoms are endorsed frequently by individuals with PTSD and will be retained, unchanged, in DSM-5. They are insomnia, problems in concentration, hypervigilance, and startle reactions. Our review of the literature suggests that this symptom cluster encompasses more than hyperarousal and would be better characterized as alterations in arousal and reactivity that are associated with the traumatic event. Such a reframing of this symptom cluster enables us to include behavioral, as well as emotional indicators of such posttraumatic alterations. In addition, it appears that the modification of DSM-IV D₂ (e.g. “irritability”) and addition of one new symptom (reckless behavior) belong in this cluster.

There is growing evidence, especially among military veterans, that PTSD is associated with more than an irritable mood state (DSM-IV C2). Indeed it appears that PTSD predicts aggressive behavior and violence among veteran cohorts following deployment to Vietnam, Iraq, and Afghanistan.^[110–113] Aggressive behavior has also been observed among female flood survivors with PTSD.^[114] In some cases, it is the aggressive behavior, rather than other PTSD symptoms that become the major clinical focus. Based on such findings, DSM-IV “irritability” has been replaced by new onset posttraumatic aggressive behavior in DSM-5. Finally, there is growing evidence that PTSD is associated with reckless and self-destructive behavior. Israeli adolescents, especially boys, exposed to recurrent terrorism exhibited marked increases in risk-taking behavior.^[115] Reckless driving has been observed among individuals with PTSD.^[116–118] Risky sexual behavior, sometimes associated with HIV risk has been reported among college women, female prisoners, and adult male survivors of childhood sexual abuse.^[119–120] Reckless behavior appears to be associated with PTSD to such an extent that it has been added to the diagnostic cluster assessing alterations in arousal and reactivity. It is of interest that such risky behaviors had previously been reported to have been associated with (predominantly female) traumatized individuals who met criteria for DESNOS/Complex PTSD (see below). Here are the proposed criteria for Criterion E in DSM-5:

(E) Alterations in arousal and reactivity that are associated with the traumatic event(s) (that began or worsened after the traumatic event(s)), as evidenced by three or more of the following: (Note: In children, as evidenced by two or more of the following)[†]:

- (1) Irritable, angry, or aggressive behavior (revised DSM-IV D₃).
- (2) Reckless or self-destructive behavior (new symptom).
- (3) Hypervigilance (DSM-IV D₄).
- (4) Exaggerated startle response (DSM-IV D₅).
- (5) Problems with concentration (DSM-IV D₃).
- (6) Sleep disturbance—for example, difficulty falling or staying asleep, or restless sleep. (DSM-IV D₁).

[†]It should be noted that the proposed diagnostic thresholds (e.g. three for adults and two for children) will be tested and possibly revised after the DSM-5 field trials.

THE DURATION (E) CRITERION

In DSM-IV, PTSD may be diagnosed at any time after a traumatic event, except during the first month. The DSM-IV rationale is that a 1-month window must be allowed before diagnosing PTSD in order to permit normal recovery to occur and to avoid pathologizing normal acute posttraumatic distress. Although the 1-month window has been useful in practice, and there is some supporting evidence in its favor,^[121] it has not yet been put on a firm empirical footing.

At present, the demarcation point between acute and chronic PTSD is 3 months. Longitudinal studies of non-sexual assault victims^[122] and motor vehicle accident survivors^[123] indicate that initially high PTSD rates tend to decline greatly and approach an asymptote at 3 months. Again, since there is little research on this question, the utility of such a distinction has not been established. In the absence of such evidence, we recommend that the distinction between acute and chronic PTSD be eliminated in DSM-5.

Delayed onset has been a unique aspect of PTSD, which has had a significant impact on compensation claims where the claimant may not have exhibited full PTSD symptoms for many years. A recent systematic review of 19 group studies indicated that delayed accounted for 38.2 and 15.3%, respectively of military and civilian cases of PTSD.^[124] PTSD in the absence of any prior symptoms was extremely rare; this analysis found that delayed usually involved subsyndromal PTSD symptoms that later escalated to the full syndrome (possibly because of breakdown of very effective avoidance that previously worked to suppress reexperiencing symptoms and emotions for some period of time).^[124] Whatever the trajectory, it is clear that delayed PTSD does occur.^[52,125,126] It is unclear whether it is more likely to occur following military than civilian trauma,^[124] but it appears to be very uncommon after natural disasters.^[127]

There are two issues that emerge from the current literature on delayed-onset PTSD. First, the cases may be better described as delayed expression of PTSD rather than delayed “onset,” which suggests that the disorder did not exist prior to meeting full PTSD criteria: this pattern is not supported by the data. Second, there is a question concerning the utility of having such a diagnosis. From a clinical perspective, one treats the condition according to the symptoms that a patient presents with at any particular time and the label of delayed onset does not alter treatment. Few other conditions specify whether there is a delayed onset, and establishing the exact time course may be affected by the unreliability of retrospective reports of one’s trajectory of symptoms over time. Recent research, however, supports the distinction between immediate and delayed “onset” PTSD because of evidence that they are the result of different etiological mechanisms.^[128]

Because of these considerations, the wording for “With Delayed Onset” (Criterion E in Table 1) will be modified as follows: “With Delayed Expression: if (the PTSD) diagnostic threshold is not exceeded at least 6 months after the event (although it is understood that onset and expression of some symptoms may be immediate).”

FUNCTIONAL IMPAIRMENT—THE F CRITERION

The DSM-IV added a “significant distress or functional impairment” (F) Criterion for PTSD and a number of other disorders. This means that a person who meets the requisite A–E criteria would not receive

a PTSD diagnosis unless he or she also exhibited clinically significant distress or functional impairment. But distress is already an integral part of several PTSD symptoms. Because the F Criterion is not unique to PTSD, DSM-5 is considering this in a much wider context. There are two distinct issues that must be addressed. First, should “significant distress” be linked to “functional impairment” or should it be assessed independently, and if so, how? Second, should functional impairment remain a diagnostic criterion? If so, how should it be assessed?

CONSIDERING THE PROPOSED PTSD DIAGNOSTIC CRITERIA FOR DSM-5

In DSM-IV, only B1–5 and C1–3 are specifically anchored to the traumatic event. In these proposed criteria, it is stipulated that all B–F symptoms “began or worsened after the traumatic event.” The proposed revision is really quite conservative. It now includes 20 rather than 17 symptoms. It has retained the basic PTSD template although the C Criterion (Avoidance/Numbing) has been divided into two clusters: Avoidance Behavior and Negative Alterations in Cognitions and Mood, as suggested by most confirmatory factor analyses of the DSM-IV symptoms. Fourteen DSM-IV symptoms (B2–5, C1–2, D1, 5–7, and E3–6) have been retained unchanged (or only slightly modified for clarification). Three DSM-IV symptoms have been significantly revised (B1, D2, and E1). Three new symptoms have been added to address negative appraisals and mood (D3, 4) and reckless, self-destructive behavior (E2). These new criteria address a number of issues that DSM-IV PTSD did not. By including aggressive behavior (E1) both “fight” and “flight” are now represented as stress symptoms. By including dissociative symptoms (B3 and D1) as well as reckless or self-destructive behavior (E2) along with the entire D cluster (negative alterations in cognitions and mood), a number of DESNOS symptoms are now included in the PTSD diagnostic criteria (see below). Symptoms specifying negative appraisals and cognitions (D2 and D3) address a major focus of CBT treatment that have research support as important components of PTSD.

Reckless, maladaptive behavior (E2) addresses an important posttraumatic symptom often seen in adolescents. The distinction between acute and chronic PTSD has been eliminated because there is little empirical support for such a distinction. Finally, it is clarified that “delayed expression” represents a greater than 6-month latency before the full PTSD diagnostic threshold is exceeded, although some symptoms may have been expressed immediately after traumatic exposure.

The proposed PTSD criteria for DSM-5 are, at this point, nothing more than a proposal. They will be tested in the DSM-5 field trials and other venues in

order to determine: (1) whether some symptoms are superfluous and can be eliminated; (2) whether B–E symptoms will cluster together, as proposed; (3) what thresholds within each symptom cluster work best; and (4) how PTSD caseness compares utilizing DSM-IV versus the proposed DSM-5 criteria. The proposed modifications in diagnostic criteria are based on collective clinical experience as well as on the literature review presented in this article. Subsequent research will tell us whether this is an improvement over the DSM-IV and whether the results from forthcoming field trials will lead to further revisions and refinements before finalizing the DSM-5 diagnostic criteria for PTSD.

SHOULD DSM-5 INCLUDE PARTIAL/ SUBSYNDROMAL PTSD?

Although there are subsyndromal diagnoses (dysthymia and cyclothymia) for major depression and bipolar affective disorder, respectively, there has not been a partial/subsyndromal PTSD diagnosis in either DSM-III or IV. The argument for adding such a diagnosis is that it would characterize people with clinically significant posttraumatic reactions who fail to exceed the PTSD diagnostic threshold (often for lack of one or two symptoms) for whom a diagnosis of adjustment disorder would be too nonspecific. The argument against the addition of a new subsyndromal category is (a) that adjustment disorder is the appropriate diagnosis for such individuals and (b) that it overpathologizes normative reactions. Approximately 60 publications have reported on the prevalence and morbidity of “partial” (or “subsyndromal”) PTSD among a wide assortment of traumatized individuals. A problem with all of this research is that partial PTSD has been defined differently by different investigators. For example, partial PTSD has variously been determined by an adjudication procedure^[129] or by hard and fast criteria that have differed from one investigator to the next.^[48,130,131]

If a partial PTSD diagnosis were added to DSM-5, it should have a similar relationship to full PTSD as dysthymia has to major depressive disorder or cyclothymia has to bipolar disorder. A number of reports do indicate that people with partial PTSD exhibit significantly less symptom severity and functional impairment than those with the full syndrome, but significantly more than no-PTSD cohorts.^[48,129–137] It is noteworthy that there have also been negative findings in which few differences were detected between partial and no-PTSD cohorts, whereas both differed significantly from full PTSD.^[131,138]

As stated previously, interpretation of those findings is complicated because partial PTSD was defined differently in many of these studies. Therefore, a standard set of diagnostic criteria must be developed so that research on this question can proceed and be analyzed properly. Despite suggestive results, it does not appear at this time that the evidence is strong

enough to warrant inclusion of partial PTSD as a distinct diagnosis in DSM-5.

Whatever clinical utility might have been provided by a subsyndromal PTSD diagnosis may be achieved with the newly proposed ASD/PTSD subtype of adjustment disorder. Adjustment disorder has been re-conceptualized as a stress-response syndrome and will be classified with PTSD in DSM-5.^[139] The ASD/PTSD subtype diagnosis will apply to either (a) individuals exposed to a Criterion A event who do not meet full PTSD diagnostic criteria (e.g. DSM-5 Criteria B–G) or (b) people who were not exposed to a Criterion A event but do fulfill some or all B–G PTSD criteria. Technically, this is not a change in diagnostic rules because adjustment disorder has always been the appropriate diagnosis for individuals in either category. The ASD/PTSD subtype, however, may provide a much more useful diagnostic niche for clinicians because it is much more specific than other adjustment disorder subtypes. It may also foster useful research, especially clinical trials, regarding effective treatments for people with the PTSD symptoms who do not exhibit the full disorder.

DISORDERS OF EXTREME STRESS NOT OTHERWISE SPECIFIED (DESNOS)/ COMPLEX PTSD

A number of investigators and clinicians have expressed dissatisfaction because, in their opinion, the PTSD symptom clusters fail to characterize clinically significant problems often exhibited by individuals exposed to severe and protracted traumatic exposure (most notably childhood sexual abuse victims, adult refugees, and adult torture survivors). They assert that although such individuals usually meet PTSD diagnostic criteria, their most significant clinical sequelae are not included within the PTSD construct. According to this argument, the most debilitating symptoms following protracted traumatic exposure include: behavioral difficulties (such as impulsivity, aggression, sexual acting out, alcohol/drug misuse, and self-destructive actions), emotional difficulties (such as affective lability, rage, depression, and panic), cognitive difficulties (such as dissociation and pathological changes in personal identity), interpersonal difficulties and somatization.^[140–142]

The concept of “Complex PTSD” was originally proposed by Judith Herman^[140] to encompass three non-PTSD posttraumatic disorders: Dissociative Identity Disorder, Borderline Personality Disorder, and Somatization Disorder. The DSM-IV Work Group suggested that this proposed syndrome be called DESNOS, rather than Complex PTSD. In addition to characterizing the psychiatric sequelae of protracted child abuse, DESNOS has been considered a very useful construct by mental health professionals who have worked with adult (often) non-Western patients exposed to forced migration or torture^[143,144] although there has been little research in this area.

During the DSM-IV process, a field trial was designed to determine whether there was empirical support for including DESNOS as a unique diagnosis in DSM-IV. Because only 8% of individuals with DESNOS did not also meet PTSD diagnostic criteria, it was considered too rare an occurrence to be classified as a separate diagnostic entity. During recent years, there has been some research, of uneven quality, designed to clarify the prevalence and construct validity of DESNOS.

The DESNOS construct raises a number of important questions about posttraumatic syndromes. Do we need another posttraumatic diagnosis to address clinically significant trauma-related symptoms not included in PTSD? Does protracted exposure to trauma, especially during developmentally sensitive periods, lead to a different pattern of symptoms than those included in PTSD? Is the DESNOS model useful in a cross-cultural context because it emphasizes both dissociation and somatization, two symptoms not included in the DSM-IV PTSD diagnostic criteria that are frequently observed in traumatized non-Western cohorts?^[145] Is it useful in clarifying the relationship between traumatic exposure and borderline personality disorder and dissociative disorders?^[140,146,147] A recent conceptualization recognizes that more complex cases of PTSD involve deficits in regulating emotional distress.^[148] This perspective recognizes that in addition to the core PTSD symptoms, complex presentations are more difficult to treat because they involve acting out, self-harm, and self-destructive relationships and behaviors.

This suggests that complex trauma presentations may not involve a distinct disorder, but rather a subtype of PTSD (possibly termed “complex PTSD”). It is noteworthy that patients with such self-destructive/emotional regulation problems benefit from traditional trauma-focused CBT if such treatment is preceded by emotion regulation training sessions^[149] or is perhaps a bit longer than the usual short CBT protocol.^[150] Another way to contextualize DESNOS, extrapolating from the growing body of factor analytic research on subtypes of PTSD might be to consider PTSD with or without internalizing or externalizing symptoms. Given that some people appear to have simple PTSD, some people have additional internalizing behaviors and symptoms, while other people exhibit externalizing behaviors along with their PTSD,^[106,151–154] these dimensions might provide a more parsimonious theoretical context for complex PTSD as well as the discussion below on subtypes. Somatization symptoms fall on the internalizing dimension along with depression and anxiety while anger/aggression, substance abuse and behaviors indicative of cluster B Axis II disorders fall on the externalizing dimension.

CROSS-CULTURAL FACTORS

The relevance of the PTSD diagnosis has been criticized from a cross-cultural perspective as a Euro-American

construct that has little relevance to posttraumatic syndromes encountered in traditional societies.^[155] Somatization and dissociation, two cardinal symptoms of posttraumatic reactions in traditional societies, according to Kirmayer,^[145] are missing from DSM-IV diagnostic criteria for PTSD (but not DESNOS). Although there may be culture-specific idioms of distress that provide a better characterization of posttraumatic distress syndromes found in one ethnocultural context or another,^[156] PTSD has been documented throughout the world.^[157–159] High prevalence rates have been reported in non-Western nations such as Algeria, Cambodia, Lebanon, Palestine, Nepal, and the former Yugoslavia.^[160–162] In addition, comparable PTSD prevalence has been found among Russian and American adolescents.^[163]

What is generally missing are studies in which similar traumatic events have affected people from widely different cultures. A notable exception is a study by North et al.^[164] who compared psychiatric morbidity among Kenyan survivors of the bombing of the American embassy in Nairobi with American survivors of the bombing of the Federal Building in Oklahoma City. Both events were remarkably similar with respect to death, injury, destruction, and other consequences. Similar too was PTSD prevalence among Africans and Americans exposed to each traumatic event, respectively. Another exception is a study by McCall and Resick,^[165] in which assault-related PTSD symptoms were assessed among Kalahari Bushmen of Africa, one of the few remaining primitive cultures. It was found that the concept of emotional numbing could not be translated, but reexperiencing, effortful avoidance, and arousal symptoms were very common.

In order to conduct such research, we must modify current instruments, develop and standardize in a Euro-American context, to ensure cultural equivalence and cultural sensitivity. Methodological approaches, such as back-translation, committee consensus and decentering, are needed if we are to make progress in this important area. Otherwise we risk under-diagnosis, over-diagnosis, or misdiagnosis.^[159,166,167]

As stated by Osterman and de Jong,^[159] we need a “culturally competent model of traumatic stress” that addresses how culture may differentially influence explanatory models of traumatic stress, how it is implicated in the appraisal of risk/protective factors, and how such understanding might contribute to diagnosis and treatment. A start has been made by Jobson,^[168] whose model proposes how differing cultural conceptualizations of the self affect vulnerability to PTSD. There is also a need to recognize that most traumatized populations across the world (i.e. outside traditional western setting settings) are subjected to ongoing threat, resettlement, and conflict. This scenario highlights a challenge in the PTSD assessment in which the trauma is “past” and assessment is occurring in a context of relative safety.

Depression and Anxiety

DEVELOPMENTAL ISSUES

Maturation, biological, and psychological changes affect the appraisal of and reaction to traumatic events as well as differences in expression of posttraumatic distress at either end of the life span.^[169,170] For children and adolescents who have experienced traumatic events, the developmental context must incorporate the dynamic and evolving relationship between experience, neurological processing, brain development, and affect regulation.^[171] Scheeringa et al.^[172] reported on the inadequacy of DSM-IV PTSD criteria to adequately characterize posttraumatic symptoms in infants and children less than 4 years old. They suggest that criteria anchored in observable behaviors should replace reports of subjective experiences or behaviors. A review of this crucial area is beyond the scope of this article but it has been an important focus of the DSM-5 process. Readers are referred to Scheeringa, Zeanah, and Cohen.^[173]

With few exceptions,^[174] the expression of posttraumatic distress has received very little attention at the other end of the life cycle. For elderly individuals, a developmental approach must address age-specific psychosocial, behavioral, and neurobiological factors that mediate and moderate trauma-related symptom expression and clinical course.^[170] PTSD among the elderly is often expressed within a context of negative health perceptions, primary care utilization, and suicidal ideation.^[175] Traumatic events may have occurred either many decades earlier or recently and may result in different responses because of developmental and physiological factors at the time of the trauma or at the present time. Potential differences based on remote or recent traumatic events have not been examined at this point in research.

To summarize, a comprehensive, longitudinal developmental approach is needed to explicate how posttraumatic memories are differentially encoded, stored, and retrieved by the immature and developing brain, on the one hand, and by the aging brain, on the other. Such an approach should help us to understand age-related differences in the clinical expression of PTSD and thereby suggest developmentally sensitive interventions for people needing treatment.

SUBTYPES OF PTSD

One potential limitation of the current PTSD definition is that it regards PTSD as a homogeneous condition insofar as there are no recognized subtypes of PTSD. It has been proposed that there may be two subtypes of PTSD that involve (a) ongoing “dissociative” rather than (b) “hyperarousal” reactions.^[176] In this context, dissociation has been conceptualized as an avoidance strategy to reduce the awareness of aversive emotions such as extreme anxiety.^[177] Griffin et al.^[177] found that recent rape victims reporting high peritraumatic dissociation showed a decrease in heart rate and skin conductance while talking about the trauma

compared to an increase in physiological responding among those with low peri-traumatic dissociation.

Neuroimaging studies suggest there may be distinctive prefrontal responses to trauma memories that distinguish individuals with dissociative from those with non-dissociative PTSD responses.^[178,179] For example, individuals displaying a “hyperarousal” PTSD response to traumatic narratives, with heightened autonomic and emotional reactivity to trauma scripts, have reduced bilateral medial frontal activity and left anterior cingulate activity relative to controls.^[180] In contrast, individuals displaying a “dissociative” PTSD response had significantly increased the right medial frontal, the right medial prefrontal, and the right anterior cingulate activity relative to controls.^[181] There was also a lack of amygdala response to trauma narratives in the dissociative group in contrast to excessive amygdala activity in the hyperarousal. It was hypothesized that the heightened prefrontal activity in dissociative PTSD may reflect greater emotional regulation and inhibition of limbic emotional networks, including amygdala.^[181] Subsequent studies have also shown that dissociative PTSD was associated with enhanced ventral prefrontal activity to conscious fear faces, and with increased activity in bilateral amygdala, insula, and left thalamus to non-conscious fear compared to non-dissociative PTSD. The dissociative group relative to non-dissociative PTSD also had reduced dorsomedial prefrontal activity to conscious fear faces.^[182] These initial findings suggest that further study is required to map the neural, symptom, and behavioral characteristics of potential dissociative and hyperarousal subtypes of PTSD.

In contrast to these findings, other studies have challenged the proposal that dissociation is associated with suppressed physiological responding. Several studies have found that dissociative patients do not display decreased autonomic responding in response to trauma cues.^[183–184] Another study assessed psychophysiological responses during the recounting of the trauma narrative, and found no difference in autonomic reactivity between high and low dissociators.^[185] These findings suggest that our current understanding of the role of dissociation in relation to arousal is very limited, and there is insufficient evidence at this point in time to confidently conclude that a dissociative subtype of PTSD is characterized by any particular form of emotional responding.

SUMMARY

Exposure to a traumatic event is a necessary condition that precedes the later development of PTSD. Such exposure is moderated on one hand by differences in individual vulnerability and on the other, by relative differences in the toxicity of stressful events. Onset of PTSD represents a clear discontinuity between pretraumatic and posttraumatic status because

dramatic trauma-related alterations in cognitions, emotions, and behavior are a hallmark of the disorder.

Based on current evidence, the proposed PTSD diagnostic criteria for DSM-5 have the following changes from DSM-IV:

The A1 Criterion has been retained and explicated with greater clarity. People who have been directly exposed or who witnessed a traumatic event will continue to meet the criterion. In addition, people who have learned that a close relative or friend was exposed to such events will qualify. Finally, professionals repeatedly exposed to the aversive details of traumatic events (such as military mortuary personnel, disaster workers, etc) will meet the criterion. The A2 Criterion has been eliminated.

The current three-factor model of PTSD will be replaced by a four-factor model consisting of the following Criteria: (B) Intrusion Symptoms, (C) Persistent Avoidance, (D) Alterations in Cognitions and Mood, and (E) Hyperarousal and Reactivity Symptoms

All 17 DSM-IV PTSD symptoms have been retained in DSM-5 although, in some cases, they have been clarified or revised. In addition, three new symptoms have been added: erroneous self- or other-blame regarding the trauma; negative mood states; and reckless and maladaptive behavior. DSM-IV irritability has become DSM-5 aggressive behavior.

The distinction between acute and chronic PTSD has been eliminated. Proposed diagnostic thresholds for both adults and children may be revised after field trials. A partial/subsyndromal PTSD diagnosis has not been proposed for DSM-5 due to insufficient evidence. It is likely that whatever clinical utility might have been achieved by such a diagnosis will be provided by the newly proposed ASD/PTSD subtype of adjustment disorder. There has been insufficient research on DESNOS since DSM-IV to support the inclusion of this separate diagnosis in DSM-5. It is also unlikely that a dissociative subtype of PTSD will be proposed for DSM-5 due to insufficient evidence.

REFERENCES

1. van der Kolk BA. The history of trauma in psychiatry. In: Friedman MJ, Keane TM, Resick PA, editors. *Handbook of PTSD: Science and Practice*. New York: Guilford Press; 2007.
2. Charney DS. Psychobiological mechanisms of resilience and vulnerability: implications for the successful adaptation to extreme stress. *Am J Psychol* 2004;161:195
3. Monson CM, Friedman MJ, La Bash H. A psychological history of posttraumatic stress disorder. In: Friedman MJ, Keane TM, Resick PA, editors. *PTSD, Science & Practice: A Comprehensive Handbook*. New York: Guilford Press; 2007:37–52.
4. American Psychiatric Association. Committee on Nomenclature and Statistics. *Diagnostic and Statistical Manual: Mental Disorders*. Washington, DC: American Psychiatric Association, Mental Hospital Service; 1952.
5. American Psychiatric Association. Committee on Nomenclature and Statistics. *Diagnostic and Statistical Manual of Mental*

- Disorders. 2nd ed. Washington, DC: American Psychiatric Association; 1968.
6. American Psychiatric Committee on Nomenclature and Statistics. *Diagnostic and Statistical Manual of Mental Disorders*. 3rd ed. Washington, DC: American Psychiatric Association; 1980.
 7. Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB. Posttraumatic stress disorder in the National Comorbidity Survey. *Arch Gen Psychiatry* 1995;52:1048–1060.
 8. Norris FH, Slone LB. The epidemiology of trauma and PTSD. In: Friedman MJ, Keane TM, Resick PA, editors. *Handbook of PTSD: Science and Practice*. New York: Guilford Press; 2007:78–98.
 9. Morgan L, Scourfield J, Williams D, Jasper A, Lewis G. The Aberfan disaster: 33-year follow-up of survivors. *Br J Psychiatry* 2003;182:532–536.
 10. Neria Y, Nandi A, Galea S. Post-traumatic stress disorder following disasters: a systematic review. *Psychol Med* 2008;38:467–480.
 11. Whalley MG, Brewin CR. Mental health following terrorist attacks. *Br J Psychiatry* 2007;190:94–96.
 12. Galea S, Ahern J, Resnick H, et al. Psychological sequelae of the September 11 terrorist attacks in New York City. *New Engl J Med* 2002;346:982–987.
 13. Brewin CR, Gregory JD, Lipton M, Burgess N. Intrusive images and memories in psychological disorders: characteristics, neural basis, and treatment implications. *Psychol Rev* 2010;117:210–232.
 14. Brewin CR, Lanius RA, Novac A, Schnyder U, Galea S. Reformulating PTSD for DSM-V: life after criterion A. *J Trauma Stress* 2009;22:366–373.
 15. Shalev AY. Psycho-biological perspectives on early reactions to traumatic events. In Orner R, Schnyder U, editors. *Reconstructing Early Intervention After Trauma*. Oxford: Oxford University Press; 2003:57–64.
 16. Weathers FW, Keane TM. The Criterion A problem revisited: controversies and challenges in defining and measuring psychological trauma. *J Trauma Stress* 2007;20:107–121.
 17. American Psychiatric Committee on Nomenclature and Statistics. *Diagnostic and Statistical Manual of Mental Disorders*. 3rd ed.-revised. Washington, DC: American Psychiatric Association; 1987.
 18. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 4th ed. Washington, DC: American Psychiatric Association; 1994.
 19. Koenen KC, Amsssstade AB, Nugent NR. Gene-environment interaction in posttraumatic stress disorder: an update. *J Trauma Stress* 2009;32:416–426.
 20. Brewin CR, Andrews B, Valentine JD. Meta-analysis of risk factors for posttraumatic stress disorder in trauma exposed adults. *J Consult Clin Psychol* 2000;68:748–766.
 21. Ozer EJ, Best SR, Lipsey TL, Weiss DL. Predictors of posttraumatic stress disorder and symptoms in adults: a meta-analysis. *Psychol Bull* 2003;129:52–73.
 22. Dohrenwend BP. Inventorying stressful life events as risk factors for psychopathology: toward resolution of the problem of intracategory variability. *Psychol Bull* 2006;132:477–495.
 23. McNally RJ. Can we fix PTSD in DSM-V? *Depress Anxiety* 2009;26:597–600.
 24. Kilpatrick DG, Resnick HS, Freedy JR, et al. Posttraumatic stress disorder field trial: evaluation of the PTSD construct—criteria A through E. In: Widiger TA, Frances AJ, Pincus HA, Ross R, First MB, Davis WW, editors. *DSM-IV Sourcebook*. Vol. 4. Washington, DC: American Psychiatric Association; 1998:803–838.
 25. Kilpatrick DG, Resnick HS, Acierno R. Should PTSD criterion A be retained? *J Trauma Stress* 2009;22:374–383.
 26. Rosen GM, Lilienfeld SO. Posttraumatic stress disorder: an empirical evaluation of core assumptions. *Clin Psychol Rev* 2008;28:837–868.
 27. Breslau N, Kessler RC. The stressor criterion in DSM-IV posttraumatic stress disorder: an empirical investigation. *Biol Psychiatry* 2001;50:699–704.
 28. Davidson JRT, Foa EB, editors. *Posttraumatic Stress Disorder: DSM-IV and Beyond*. 1st ed. Washington, DC: American Psychiatric Press; 1993.
 29. Friedman MJ. PTSD and other posttraumatic syndromes. In: McKay D, Abromowitz J, Taylor S, Asmundson G, editors. *Current Perspectives on the Anxiety Disorders: Implications for DSM-V and Beyond*. New York: Springer; 2009:377–410.
 30. Freedy JR, Resnick HS, Kilpatrick DG, Dansky BS, Tidwell RP. The psychological adjustment of recent crime victims in the criminal justice system. *J Interpers Violence* 1994;9:450–468.
 31. Amick-McMullan A, Kilpatrick DG, Veronen LJ. Family survivors of homicide victims: a behavioral analysis. *Behav Therapist* 1989;12:75–79.
 32. Neria Y, Olfson M, Gameroff MJ, et al. The mental health consequences of disaster-related loss: findings from primary care one year after the 9/11 terrorist attacks. *Psychiatry* 2008;71:339–348.
 33. Neria Y, Gross R, Litz B, et al. Prevalence and psychological correlates of complicated grief among bereaved adults 2.5–3.5 years after September 11th attacks. *J Trauma Stress* 2007;20:251–262.
 34. Murphy SA, Braun T, Tillery L, Cain KC, Johnson LC, Beaton RD. PTSD among bereaved parents following the violent deaths of their 12- to 28-year-old children: a longitudinal prospective analysis. *J Trauma Stress* 1999;12:273–291.
 35. Mirzamani SM. Mothers' psychological problems following disaster affecting their children. In: Corales TA, editor. *Focus on Posttraumatic Stress Disorder Research*, Hauppauge. New York: Nova Science Publishers; 2005:95–121.
 36. Ursano RJ, Fullerton CS, Kao T-C, Bhartiya VR. Longitudinal assessment of posttraumatic stress disorder and depression after exposure to traumatic death. *J Nerv Ment Dis* 1995;183:36–42.
 37. McCarroll JE, Ursano RJ, Fullerton CS, Liu X, Lundy A. Effect of exposure to death in a war mortuary on posttraumatic stress symptoms. *J Nerv Ment Dis* 2001;189:44–48.
 38. Sutker PB, Uddo M, Brailey K, Vasterling JJ, Errera P. Psychopathology in war-zone deployed and no deployed Operation Desert Storm troops assigned graves registration duties. *J Abnorm Psychol* 1994;103:383–390.
 39. Chang C-M, Lee L-C, Connor KM, Davidson JRT, Jeffries K, Lai T-J. Posttraumatic distress and coping strategies among rescue workers after an earthquake. *J Nerv Ment Dis* 2003;191:391–398.
 40. McCarroll JE, Fullerton CS, Ursano RJ, Hermsen JM. Posttraumatic stress symptoms following forensic dental identification Mt. Carmel, Waco, Texas. *Am J Psychiatry* 1996;153:778–782.
 41. Ahern J, Galea S, Resnick H, et al. Television images and psychological symptoms after the September 11 terrorist attack. *Psychiatry* 2002;65:289–300.
 42. Weidmann A, Papsdorf J. Witnessing trauma in the newsroom: posttraumatic symptoms in television journalists exposed to violent news clips. *J Nerv Ment Dis* 2010;198:264–271.
 43. Ursano RJ, Fullerton CS, Norwood AE, editors. *Terrorism and Disaster: Individual and Community Mental Health Interventions*. Cambridge: Cambridge University Press; 2003.

44. Brewin CR, Andrews B, Rose S. Fear, helplessness, and horror in posttraumatic stress disorder: investigating DSM-IV criterion A2 in victims of violent crime. *J Trauma Stress* 2000;13:499–509.
45. Rizvi SL, Kaysen DL, Gutner CA, Griffen MG, Resick PA. Beyond fear: the role of peritraumatic responses in posttraumatic and depressive symptoms among female crime victims. *J Interpers Violence* 2008;23:853–868.
46. O'Donnell ML, Creamer M, McFarlane AC, Silove D, Bryant RA. Should A2 be a diagnostic requirement for posttraumatic stress disorder in DSM-V? *Psychiatry Res* 2010;176:257–260.
47. Creamer M, McFarlane AC, Burgess P. Psychopathology following trauma: the role of subjective experience. *J Affect Disord* 2005;86:175–182.
48. Schnurr PP, Ford JD, Friedman MJ, Green BL, Dain BJ, Sengupta A. Predictors and outcomes of posttraumatic stress disorder in World War II veterans exposed to mustard gas. *J Consult Clin Psychol* 2000;68:258–268.
49. Karam EG, Andrews G, Bromet E, et al. The role of criterion A2 in the DSM-IV diagnosis of post-traumatic stress disorder. *Biol Psychiatry* 2010;68:465–473.
50. Bryant R. Posttraumatic stress disorder and mild brain injury: controversies, causes and consequences. *J Clin Exp Neuropsychol* 2001;23:718–728.
51. Bryant RA, Marosszeky JE, Crooks J, Gurka JA. Posttraumatic stress disorder following severe traumatic brain injury. *Am J Psychol* 2000;157:629–631.
52. Bryant RA, Harvey AG. Delayed-onset posttraumatic stress disorder: a prospective evaluation. *Aust N Z J Psychiatry* 2002;36:205–209.
53. Foy DW, Wood JL, King DW, King LA, Resnick HS. Lost Angeles Symptom Checklist: psychometric evidence with an adolescent sample. *Assessment* 1997;4:377–384.
54. Anthony JL, Lonigan C J, Hecht SA. Dimensionality of posttraumatic stress disorder symptoms in children exposed to disaster: results from confirmatory factor analysis. *J Abnorm Psychol* 1999;108:326–336.
55. Elhai JD, Gray MJ, Docherty AR, Kashdan TB, Kose S. Structural validity of the Posttraumatic Stress Disorder Checklist among college students with a trauma history. *J Interpers Violence* 2007;22:1471–1478.
56. Griesel D, Wessa M, Herta F. Psychometric qualities of the German version of the Posttraumatic Diagnostic Scale (PTDS). *Psychol Assess* 2006;18:262–268.
57. Buckley TC, Blanchard EB, Hickling EJ. A confirmatory factor analysis of posttraumatic stress symptoms. *Behav Res Ther* 1998;36:1091–1099.
58. Charney ME, Keane TM. Psychometric analyses of the clinician-administered PTSD Scale (CAPS)—Bosnian translation. *Cultur Divers Ethnic Minor Psychol* 2007;13:161–168.
59. Cuevas CA, Bollinger AR, Vielhauer MJ, et al. HIV/AIDS cost study: construct validity and factor structure of the PTSD Checklist in dually diagnosed HIV-seropositive adults. *J Trauma Practice* 2006;5:29–51.
60. Taylor S, Kuch K, Koch WJ, Crockett DJ, Passey G. The structure of posttraumatic stress symptoms. *J Abnorm Psychol* 1998;107:154–160.
61. Asmundson GJG, Wright KD, McCreary DR, Pedlar D. Posttraumatic stress disorder symptoms in United Nations peacekeepers: an examination of factor structure in peacekeepers with and without chronic pain. *Cogn Behav Therap* 2003;32:26–37.
62. Asmundson GJG, Frombach I, McQuaid JR, Pedrilli P, Lenox RJ, Stein MB. Dimensionality of posttraumatic stress symptoms: a confirmatory factor analysis of DSM-IV symptom clusters and other symptom models. *Behav Res Ther* 2000;38:203–214.
63. Duhamel KN, Ostroff J, Ashman T, et al. Construct validity of the Posttraumatic Stress Disorder Checklist in cancer survivors: analyses based on two samples. *Psychol Assess* 2004;16:255–266.
64. King DW, Leskin GA, King LA, Weathers FW. Confirmatory factor analysis of the Clinician-Administered PTSD Scale: evidence for the dimensionality of posttraumatic stress disorder. *Psychol Assess* 1998;10:90–96.
65. Marshall GN. Posttraumatic stress disorder symptom checklist: factor structure and English-Spanish measurement invariance. *J Trauma Stress* 2004;17:223–230.
66. McWilliams LA, Cox BJ, Asmundson GJG. Symptom structure of posttraumatic stress disorder in a nationally representative sample. *J Anxiety Disord* 2005;19:626–641.
67. Palmieri PA, Fitzgerald LF. Confirmatory factor analysis of posttraumatic stress symptoms in sexually harassed women. *J Trauma Stress* 2005;18:57–666.
68. Palmieri PA, Marshall GN, Schell TL. Confirmatory factor analysis of posttraumatic stress symptoms in Cambodian refugees. *J Trauma Stress* 2007;20:207–216.
69. Schinka JA, Brown LM, Borenstein AR, Mortimer JA. Confirmatory factor analysis of the PTSD Checklist in the elderly. *J Trauma Stress* 2007;20:281–289.
70. Andrews L, Joseph S, Shevlin M, Troop N. Confirmatory factor analysis of posttraumatic stress symptoms in emergency personnel: an examination of seven alternative models. *Pers Individ Differ* 2006;41:213–224.
71. Simms LJ, Watson D, Doebbeling BN. Confirmatory factor analyses of posttraumatic stress symptoms in deployed and nondeployed veterans of the Gulf war. *J Abnorm Psychol* 2002;111:637–647.
72. Elkit A, Shevlin M. The structure of PTSD symptoms: a test of alternative models using confirmatory factor analysis. *Br J Clin Psychol* 2007;46:299–313.
73. Palmieri PA, Watson DW, Difede J, King DW. Confirmatory factor analysis of the PTSD Checklist and the Clinician-Administered PTSD Scale in disaster workers exposed to the World Trade Center Ground Zero. *J Abnorm Psychol* 2007;116:329–341.
74. Yufik T, Simms LJ. A meta-analytic investigation of the structure of posttraumatic stress disorder symptoms. *J Abnorm Psychol* 2010 (in press).
75. Resick PA, Miller MW. Posttraumatic stress disorder: anxiety or traumatic stress disorder? *J Trauma Stress* 2009;22:391–398.
76. Spiegel DS, Friedman MJ, Resick PA, Brewin CR, Bryant RA, Strain JD. Is PTSD an anxiety disorder? *Depress Anxiety* 2010, submitted.
77. Ehlers A, Hackmann A, Michael T. Intrusive re-experiencing in post-traumatic stress disorder: phenomenology, theory, and therapy. *Memory* 2004;12:403–415.
78. Michael T, Ehlers A, Halligan SL, Clark DM. Unwanted memories of assault: what intrusion characteristics are associated with PTSD? *Behav Res Ther* 2005;43:613–628.
79. Michael T, Halligan S, Clark D, Ehlers A. Rumination in posttraumatic stress disorder. *Depress Anxiety* 2007;24:307–317.
80. Speckens A, Ehlers A, Hackmann A, Ruths F, Clark D. Intrusive memories and rumination in patients with post-traumatic stress disorder: a phenomenological comparison. *Memory* 2007;15:249–257.
81. Ehring T, Frank S, Ehlers A. The role of rumination and reduced concreteness in the maintenance of posttraumatic stress disorder and depression following trauma. *Cogn Ther Res* 2008;32:488–506.

82. Ehring T, Fuchs N, Klasener I. The effects of experimentally induced rumination versus distraction on analogue posttraumatic stress symptoms. *Behav Ther* 2009;40:403–413.
83. Ehring T, Szeimies A, Schaffrick C. An experimental analogue study into the role of abstract thinking in trauma-related rumination. *Behav Res Ther* 2009;47:285–293.
84. Craske MG, Rauch SL, Ursano R, Prenoveau J, Pine DS, Zinbarg RB. What is anxiety disorder? *Depress Anxiety* 2009;26:1066–1085.
85. Kashdon TB, Elha JD, Frueh, BC. Anhedonia and emotional numbing in combat veterans with PTSD. *Behav Res Ther* 2006;44:457–467.
86. Grubaugh AL, Long ME, Elhai JD, Frueh BC, Magruder KM. An examination of the construct validity of posttraumatic stress disorder with veterans. *Behav Res Ther* 2010;48:909–914.
87. Ehlers A, Clark DM, Dunmore E, Jaycox LH, Meadows E, Foa EB. Predicting response to exposure treatment in PTSD: the role of mental defeat and alienation. *J Trauma Stress* 1998;11:457–471.
88. Feiring C, Cleland C. Childhood sexual abuse and abuse-specific attributions of blame over 6 years following discovery. *Child Abuse Negl* 2007;31:1169–1186.
89. Resick PA, Nishith P, Weaver TL, Astin MC, Feuer CA. A comparison of cognitive processing therapy, prolonged exposure and a waiting condition for the treatment of posttraumatic stress disorder in female rape victims. *J Consult Clin Psychol* 2002;70:867–879.
90. Bryant RA, Guthrie RM. Maladaptive self-appraisals before trauma exposure predict posttraumatic stress disorder. *J Consult Clin Psychol* 2007;75:812–815.
91. Meiser-Stedman RA, Yule W, Smith PA, Glucksman E, Dalgleish T. Acute stress disorder and posttraumatic stress disorder in children and adolescents involved in assaults or motor vehicle accidents. *Am J Psychiatry* 2005;162:1381–1383.
92. Owens GP, Chard KM, Cox TA. The relationship between maladaptive cognitions, anger expression, and posttraumatic stress disorder among veterans in residential treatment. *J Aggress Maltreat Trauma* 2008;17:439.
93. Ehring T, Ehlers A, Glucksman E. Do cognitive models help in predicting the severity of posttraumatic stress disorder, phobia, and depression after motor vehicle accidents? A prospective longitudinal study. *J Consult Clin Psychol* 2008;76:219–230.
94. Karl A, Rabe S, Zollner T, Maercker A, Stopa L. Negative self-appraisals in treatment-seeking survivors of motor vehicle accidents. *J Anxiety Disord* 2009;23:775.
95. Moser JS, Hajcak G, Simons RF, Foa EB. Posttraumatic stress disorder symptoms in trauma-exposed college students: the role of trauma-related cognitions, gender, and negative affect. *J Anxiety Disord* 2007;21:1039–1049.
96. Resick PA, Galovski TE, Uhlmansiek MO, Scher CD, Clum G, Young-Xu Y. A randomized clinical trial to dismantle components of cognitive processing therapy for posttraumatic stress disorder in female victims of interpersonal violence. *J Consult Clin Psychol* 2008;76:243–258.
97. Andrews B, Brewin CR, Rose S, Kirk M. Predicting PTSD symptoms in victims of violent crime: the role of shame, anger, and childhood abuse. *J Abnorm Psychol* 2000;109:69–73.
98. Orth U, Cahill SP, Foa EB, Maercker A. Anger and posttraumatic stress disorder symptoms in crime victims: a longitudinal analysis. *J Consult Clin Psychol* 2008;76:208–218.
99. Riggs DS, Dancu CV, Gershuny BS, Greenberg D, Foa EB. Anger and post-traumatic stress disorder in female crime victims. *J Trauma Stress* 1992;5:613–625.
100. Taft CT, Street AE, Marshall AD, Dowdall DJ, Riggs DS. Posttraumatic stress disorder, anger, and partner abuse among Vietnam combat veterans. *J Fam Psychol* 2007;21:270–277.
101. Henning KR, Frueh BC. Combat guilt and its relationship to PTSD symptoms. *J Clin Psychol* 1997;53:801–808.
102. Kubany ES, Abueg FR, Owens JA, Brennan JM, Kaplan AS, Watson SB. Initial examination of a multidimensional model of trauma-related guilt: applications to combat veterans and battered women. *J Psychopathol Behav* 1995;17:353–376.
103. Nishith P, Nixon RDV, Resick PA. Resolution of trauma-related guilt following treatment of PTSD in female rape victims: a result of cognitive processing therapy targeting comorbid depression? *J Affect Disord* 2005;86:259–265.
104. Leskela J, Dieperink M, Thuras P. Shame and posttraumatic stress disorder. *J Trauma Stress* 2002;15:223–226.
105. Street AE, Arias I. Psychological abuse and posttraumatic stress disorder in battered women: examining the roles of shame and guilt. *Violence Vict* 2001;16:65–78.
106. Miller MW, Resick PA. Internalizing and externalizing subtypes in female sexual assault survivors: implications for the understanding of complex PTSD. *Behav Ther* 2007;38:58–71.
107. Lanius RA, Williamson PC, Bluhm R, et al. Functional connectivity of dissociative responses in posttraumatic stress disorder: a functional magnetic resonance imaging investigation. *Biol Psychiatry* 2005;57:873–884.
108. Sar V, Koyuncu A, Ozturk E, et al. Dissociative disorders in the psychiatric emergency ward. *Gen Hosp Psychiatry* 2007;29:45–50.
109. Litz BT, Gray MJ. Emotional numbing in posttraumatic stress disorder: current and future research directions. *Aust N Z J Psychiatry* 2002;36:198–294.
110. Jakupcak M, Conybeare D, Phelps L, et al. Anger, hostility, and aggression among Iraq and Afghanistan war veterans reporting PTSD and subthreshold PTSD. *J Trauma Stress* 2007;20:945–954.
111. Lasko NB, Gurvits TV, Kuhne AA, Orr SP, Pitman RK. Aggression and its correlates in Vietnam veterans with and without chronic posttraumatic stress disorder. *Compr Psychiatry* 1994;35:373–381.
112. Taft CT, Kaloupek DG, Schumm JA, et al. Posttraumatic stress disorder symptoms, physiological reactivity, alcohol problems, and aggression among military veterans. *J Abnorm Psychol* 2007;116:498–507.
113. Taft CT, Vogt DS, Marshall AD, Panuzio J, Niles BL. Aggression among combat veterans: relationships with combat exposure and symptoms of posttraumatic stress disorder, dysphoria, and anxiety. *J Trauma Stress* 2007;20:135–145.
114. Taft CT, Monson CM, Schumm JA, Watkins LE, Panuzio J, Resick PA. Posttraumatic stress disorder symptoms, relationship adjustment, and relationship aggression in a sample of female flood victims. *J Fam Violence* 2009;24:389–396.
115. Pat-Horenczyk R, Peled O, Miron T, Brom D, Villa Y, Chemtob C. Risk-taking behaviors among Israeli adolescents exposed to recurrent terrorism. *Am J Psychiatry* 2007;164:66–72.
116. Lapham SC, C’De Baca J, McMillan GP, Lapidus J. Psychiatric disorders in a sample of repeat impaired-driving offenders. *J Stud Alcohol* 2006;67:707–713.
117. Lowinger T, Solomon Z. PTSD, guilt, and shame among reckless drivers. *J Loss Trauma* 2004;9:327–344.
118. Fear NT, Iverson AC, Chatterjee A, et al. Risky driving among regular armed forces personnel from the United Kingdom. *Am J Prev Med* 2008;35:230–236.

119. Green BL, Krupnick JL, Stockton P, Goodman L, Corcoran CB, Petty R. Effects of adolescent trauma exposure on risky behavior in college women. *Psychiatry* 2005;68:363–378.
120. Hutton HE, Treisman GJ, Hunt WR, et al. *Psychiatr Serv* 2001;52:508–513.
121. Rothbaum BO, Foa EB, Riggs DS, Murdock T, Walsh W. A prospective examination of post-traumatic stress disorder in rape victims. *J Trauma Stress* 1992;5:455–475
122. Riggs DS, Rothbaum BO, Foa EB. A prospective examination of symptoms of posttraumatic stress disorder in victims of nonsexual assault. *J Interpers Violence* 1995;10:201–214.
123. Blanchard EB, Hickling EJ, Vollmer AJ, Loos WR, Buckley TC, Jaccard J. Short-term follow-up of post-traumatic stress symptoms in motor vehicle accident victims. *Behav Res Ther* 1995;33:369–377.
124. Andrews B, Brewin CR, Philpott R, Stewart L. Delayed-onset posttraumatic stress disorder: a systematic review of the evidence. *Am J Psychiatry* 2007;164:1319–1326.
125. Solomon Z, Mikulincer M. Trajectories of PTSD: a 20-year longitudinal study. *Am J Psychiatry* 2006;163:659–666.
126. Wolfe J, Erickson DJ, Sharkansky EJ, King DW, King LA. Course and predictors of posttraumatic stress disorder among Gulf War veterans: a prospective analysis. *J Consult Clin Psychol* 1999;67:520–528.
127. Norris FH, Murphy AD, Baker CK, et al. Severity, timing, and duration of reactions to trauma in the population: an example from Mexico. *Biol Psychiatry* 2003;53:769–778.
128. Andrews, B, Brewin CR, Stewart L, Philpott R, Hejdenberg J. Comparison of immediate and delayed-onset posttraumatic stress disorder in military veterans. *J Abnorm Psychol* 2009;118:767–777.
129. Weiss DS, Marmar CR, Schlenger WE, et al. The prevalence of lifetime and partial post-traumatic stress disorder in Vietnam theater veterans. *J Trauma Stress* 1992;5:365–376.
130. Breslau N, Lucia VC, Davis GC. Partial PTSD versus full PTSD: an empirical examination of associated impairment. *Psychol Med* 2004;34:1205–1214.
131. Favaro A, Tenconi E, Colombo G, Santonastaso P. Full and partial post-traumatic stress disorder among World War II prisoners of war. *Psychopathology* 2006;39:187–191.
132. Carlier IVE, Gersons BPR. Partial posttraumatic stress disorder (PTSD): the issue of psychological scars and the occurrence of PTSD symptoms. *J Nerv Ment Dis* 1995;183:107–109.
133. Lipschitz DS, Rasmussen AM, Anyan W, Cromwell P, Southwick SM. Clinical and functional correlates of posttraumatic stress disorder in urban adolescent girls at a primary care clinic. *J Am Acad Child Psychol* 2000;39:1104–1111.
134. Mylle J, Maes, M. Partial posttraumatic stress disorder revisited. *J Affect Disord* 2004;78:37–48.
135. Stein MB, Walker JR, Hazen AL, Forde DR. Full and partial posttraumatic stress disorder: findings from a community survey. *Am J Psychiatry* 1997;154:1114–1119.
136. Taylor S, Koch WJ. Anxiety disorders due to motor vehicle accidents: nature and treatment. *Clin Psychol Rev* 1995;15:721–738.
137. Zlotnick C, Rodriguez BF, Weisberg RB, et al. Chronicity in posttraumatic stress disorder and predictors of the course of posttraumatic stress disorder among primary care patients. *J Nerv Ment Dis* 2004;192:153–159.
138. Maia DB, Marmar CR, Metzler T, et al. Post-traumatic stress symptoms in an elite unit of Brazilian police officers: prevalence and impact on psychosocial functioning and on physical and mental health. *J Affect Disord* 2007;97:241–245.
139. Strain JJ, Friedman MJ. Adjustment disorders as stress response syndromes: considering the ASD/PTSD subtype proposed for DSM-5 (submitted).
140. Herman JL. Complex PTSD: a syndrome in survivors of prolonged and repeated trauma. *J Trauma Stress* 1992;5:377–391.
141. Linehan MM, Tutek DA, Heard HL, Armstrong HE. Interpersonal outcome of cognitive behavioral treatment for chronically suicidal borderline patients. *Am J Psychiatry* 1994;151:1771–1776.
142. van der Kolk BA, Roth S, Pelcovitz D, Sunday S, Spinazzola J. Disorders of extreme stress: the empirical foundation of a complex adaptation to trauma. *J Trauma Stress* 2005;18:389–399.
143. de Jong JTVM, Komproe IH, Spinazzola J, van der Kolk BA, Van Ommeren MH. DESNOS in three postconflict settings: assessing cross-cultural construct equivalence. *J Trauma Stress* 2005;18:13–21.
144. Sochting I, Corrado R, Cohen IM, Ley RG, Brasfield C. Traumatic pasts in Canadian aboriginal people: further support for a complex trauma conceptualization? *BC Med J* 2007;49:320–326.
145. Kirmayer LJ. Confusion of the senses: implications of ethnocultural variations in somatoform and dissociative disorders for PTSD. In: Marsella AJ, Friedman MJ, Gerrity ET, Scurfield RM, editors. *Ethnocultural Aspects of Posttraumatic Stress Disorder: Issues, Research, and Clinical Applications*. Washington, DC: American Psychological Association; 1996; 131–163.
146. Classen CC, Pain C, Field NP, Woods P. Posttraumatic personality disorder: a reformulation of complex posttraumatic stress disorder and borderline personality disorder. *Psychiatr Clin North Am* 2006;29:87–112.
147. Sansone RA, Songer DA, Miller KA. Childhood abuse, mental healthcare utilization, self-harm behavior, and multiple psychiatric diagnoses among inpatients with and without a borderline diagnosis. *Compr Psychiatry* 2005;26:117–120.
148. Cloitre M, Miranda R, Stovall-McClogh KC, Han H. Beyond PTSD: emotion regulation and interpersonal problems as predictors of functional impairment in survivors of childhood abuse. *Behav Ther* 2005;36:119–124.
149. Cloitre M, Koenen KC, Cohen LR, Han H. Skills training in affective and interpersonal regulation followed by exposure: a phase-based treatment for PTSD related to childhood abuse. *J Consult Clin Psychol* 2002;70:1067–1074.
150. Chard KM. An evaluation of cognitive processing therapy for the treatment of posttraumatic stress disorder related to childhood sexual abuse. *J Consult Clin Psychol* 2005;73:965–971.
151. Miller MW, Fogler JM, Wolf EJ, Kaloupek DG, Keane TM. The internalizing and externalizing structure of psychiatric comorbidity in Combat Veterans. *J Trauma Stress* 2008;21:58–65.
152. Forbes D, Elhai JD, Miller MW, Creamer M. Internalizing and externalizing classes in posttraumatic stress disorder: a latent class analysis. *J Trauma Stress* 2010;23:340–349.
153. Rielage JK, Hoyt T, Renshaw K. Internalizing and externalizing personality styles and psychopathology in OEF–OIF Veterans. *J Trauma Stress* 2010;23:350–357.
154. Sellbom M, Bagby RM. Identifying PTSD personality subtypes in a workplace trauma sample. *J Trauma Stress* 2009;22:471–475.
155. Summerfield DA. Cross-cultural perspectives on the medicalization of human suffering. In: Rosen GM, editor. *Posttraumatic*

- Stress Disorder: Issues and Controversies. England: Wiley; 2004;233–245.
156. Hinton DE, Lewis-Fernandez R. A critical review of DSM-IV-TR's diagnosis of PTSD from a cross-cultural perspective. *Depress Anxiety* 2010 (in press).
 157. Green BL, Friedman MJ, de Jong JTVM, et al. *Trauma Interventions in War and Peace: Prevention, Practice and Policy*. New York: Kluwer Academic/Plenum; 2003.
 158. Marsella AJ, Friedman MJ, Gerrity ET, Scurfield RM, editors. *Ethnocultural Aspects of Post-Traumatic Stress Disorder: Issues, Research and Clinical Applications*. Washington, DC: American Psychological Association; 1996:576.
 159. Osterman JE, deJong JTVM. Cultural issues and trauma. In: Friedman MJ, Resick PA, Keane TM, editors. *Handbook of PTSD: Science and Practice*. New York: Guilford Press; 2007:425–446.
 160. de Jong JTVM, Komproe IH, Van Ommeren MH, et al. Lifetime events and posttraumatic stress disorder in 4 post-conflict settings. *J Am Med Assoc* 2001;286:555–562.
 161. Hinton DE, Chhean D, Pich V, Pollack MH, Orr SP, Pitman RK. Assessment of posttraumatic stress disorder in Cambodian refugees using the Clinician-Administered PTSD Scale: psychometric properties and symptom severity. *J Trauma Stress* 2006;19:405–409.
 162. Thapa SB, Hauff E. Psychological distress among displaced persons during an armed conflict in Nepal. *Soc Psychiatr Psychiatr Epidemiol* 2005;40:672–679.
 163. Ruchkin V, Schwab-Stone M, Jones S, Cichetti DV, Kiposov R, Vermeiren R. Is posttraumatic stress in youth a culture-bound phenomenon? A comparison of symptom trends in selected U.S. and Russian communities. *Am J Psychiatry* 2005;162:538–544.
 164. North CS, Pfefferbaum B, Narayanan P, et al. Comparison of post-disaster psychiatric disorders after terrorist bombings in Nairobi and Oklahoma City. *Br J Psychiatry* 2005;186:487–493.
 165. McCall GJ, Resick PA. A pilot study of PTSD among the Kalahari Bushmen. *J Trauma Stress* 2003;16:445–450.
 166. Joyce PA, Berger R. Which language does PTSD speak? The “Westernization” of Mr. Sanchez. *J Trauma Pract* 2006;5:53–67.
 167. Smit J, van den Berg CE, Bekker LG, Seedat S, Stein DJ. Translation and cross-cultural adaptation of a mental health battery in an African setting. *Afr Health Sci* 2006;6:215–222.
 168. Jobson L. Drawing current posttraumatic stress disorder models into the cultural sphere: the development of the “threat to the conceptual self” model. *Clin Psychol Rev* 2009;29:368–381.
 169. Fairbank JA, Putnam FW, Harris WW. The prevalence and impact of child traumatic stress. In: Friedman MJ, Resick PA, Keane TM, editors. *Handbook of PTSD: Science and Practice*. New York: Guilford Press; 2007:229–251.
 170. Cook JM, Niederehe G. Trauma in older adults. In: Friedman MJ, Resick PA, Keane TM, editors. *Handbook of PTSD: Science and Practice*. New York: Guilford Press; 2007:252–276.
 171. Saxe GN, MacDonald HZ, Ellis H. 2007. Psychosocial approaches for children with posttraumatic stress disorder. In: Friedman MJ, Keane TM, Resick PA, editors. *PTSD, Science & Practice: A Comprehensive Handbook*. New York: Guilford Publications; 2007:359–375.
 172. Scheeringa MS, Zeanah CH, Drell MJ, Larieu JA. Two approaches to the diagnosis of posttraumatic stress disorder in infancy and early childhood. *J Am Acad Child Psychiatry* 1995; 34:191–200.
 173. Scheeringa MS, Zeanah CH, Cohen JA. PTSD in children and adolescents: towards an empirically based algorithm. *Depress Anxiety* 2010 (in press).
 174. Hiskey S, Luckie M, Davies S, Brewin CR. The emergence of posttraumatic distress in later life: a review. *J Geriatr Psychiatry Neurol* 2008;21:232–241.
 175. Rauch SA, Morales KH, Zubritsky C, Knott K, Oslin D. Posttraumatic stress, depression, and health among adults in primary care. *Am J Geriatr Psychiatry* 2006;14:316–324.
 176. Bremner JD. Acute and chronic responses to psychological trauma: where do we go from here? *Am J Psychiatry* 1999;156: 349–351.
 177. Griffin MG, Resick PA, Mechanic MB. Objective assessment of peritraumatic dissociation: psychophysiological indicators. *Am J Psychiatry* 1997;154:1081–1088.
 178. Lanius R, Bluhm R, Lanius U, Pain C. A review of neuroimaging studies in PTSD: heterogeneity of response to symptom provocation. *J Psychiatr Res* 2006;40:709–729.
 179. Lanius R, Vermetten E, Loewenstein RJ, et al. Emotion modulation in PTSD: clinical and neurobiological evidence for a dissociative subtype. *Am J Psychiatry* 2010;167:640–647.
 180. Lanius R, Williamson P, Densmore M, et al. Neural correlates of traumatic memories in posttraumatic stress disorder: a functional MRI investigation. *Am J Psychiatry* 2001;158: 1920–1922.
 181. Lanius R, Williamson P, Boksman K, et al. Brain activation during script-driven imagery induced dissociative responses in PTSD: a functional magnetic resonance imaging investigation. *Biol Psychiatry* 2002;52:305–311.
 182. Felmingham K, Kemp A, Williams L, et al. Dissociative responses to conscious and non-conscious fear impact underlying brain function in post-traumatic stress disorder. *Psychol Med* 2008;38:1771–1780.
 183. Griffin MG, Nishith P, Resick PA. Peritraumatic dissociation in domestic violence victims. Paper Presented at the 18th Annual Meeting of the International Society for Traumatic Stress Studies, Baltimore, MD; 2002.
 184. Kaufman ML, Kimble MO, Kaloupek DG, et al. Peritraumatic dissociation and physiological responses to trauma-relevant stimuli in Vietnam combat veterans with posttraumatic stress disorder. *J Nerv Ment Dis* 2002;190:167–194.
 185. Nixon RDV, Bryant RA. Physiological arousal and dissociation in acute trauma victims during trauma narratives. *J Trauma Stress* 2005;18:107–114.