INTRODUCTION

Of the many diagnoses in the Diagnostic and Statistical Manual IV-TR (DSM-IV-TR) [1], very few invoke an aetiology in their diagnostic criteria: (i) organic mental disorders (e.g. caused by a neurological abnormality); (ii) substance-use disorders (e.g. caused by psychoactive chemical agents); (iii) post-traumatic stress disorder (PTSD); (iv) acute stress disorder (ASD); and (v) adjustment disorders (ADs) [2] – the latter three are all caused by exposure to a stressful environmental event that exceeds the coping capacity of the affected individual. The presumed causal relationship between the stressor and PTSD, ASD and AD is complicated and controversial, as will be discussed below. Controversy notwithstanding, acceptance of this causal relationship, initially in the DSM-III [3], has equipped practitioners and scientists with a conceptual tool that has profoundly influenced clinical practice over the past 30 years.

PTSD is primarily a disorder of reactivity rather than of an altered baseline state as in major depressive disorder or general anxiety disorder. Its psychopathology is characteristically expressed during interactions with the interpersonal or physical environment. People with PTSD are consumed by concerns about personal safety. They persistently scan the environment for threatening stimuli. When in doubt, they are more likely to assume that danger is present and will react accordingly. The avoidance and hyperarousal symptoms described below can be understood within this context. The primacy of traumatic over other memories (e.g. the reexperiencing symptoms) can also be understood as a pathological exaggeration of an adaptive human response to remember as much as possible about dangerous encounters in order to avoid similar threats in the future.

The sustained anxiety about potential threats to life and limb, pervasive and uncontrollable sense of danger, and maladaptive preoccupation with concerns
about personal safety and the safety of one’s family can be explicated in terms of psychological models such as classic Pavlovian fear conditioning, two-factor theory or emotional processing theory [4–6]. The traumatic (unconditioned) stimulus (the rape, assault, disaster, etc.) automatically evokes the post-traumatic (unconditioned) emotional response (fear, helplessness and/or horror). The intensity of this emotional reaction provokes avoidance or protective behaviours that reduce the emotional impact of the stimulus. Conditioned stimuli, reminders of such traumatic events (e.g. seeing someone who resembles the original assailant, confronting war-zone reminders, exposure to high winds or torrential downpours reminiscent of a hurricane, etc.), evoke similar conditioned responses manifested as fear-induced avoidance and protective behaviours.

Such psychological models can also be explicated within the context of neurocircuitry that mediates the processing of threatening or fearful stimuli. In short, traumatic stimuli activate the amygdala, which in turn produces outputs to the hippocampus, medial prefrontal cortex, locus coeruleus, thalamus, hypothalamus, insula and dorsal/ventral striatum [7–9]. In PTSD, the normal restraint on the amygdala exerted by the medial prefrontal cortex – especially the anterior cingulate gyrus and orbitofrontal cortex – is severely disrupted. Such disinhibition of the amygdala creates an abnormal psychobiological state of hypervigilance in which innocuous or ambiguous stimuli are more likely to be misinterpreted as threatening. To be hypervigilant in a dangerous situation is adaptive. To remain so after the danger has passed is not.

Fear-conditioning models help to explain many PTSD symptoms such as intrusive recollections (e.g. nightmares and psychological/physiological reactions to traumatic reminders), avoidance behaviours and hyperarousal symptoms such as hypervigilence. Emotional numbing, another important manifestation of PTSD, has been explicated in terms of stress-induced analgesia [10]. Such emotional anaesthesia is potentially even more disruptive and disturbing to the affected individual and loved ones than other symptoms because it may produce an insurmountable emotional barrier between the PTSD patient and his or her family. Such individuals are unable to experience loving feelings or to reciprocate those of partners and children. As a result, they isolate themselves and become emotionally inaccessible to loved ones to whom they had previously been very close. They also cut themselves off from friends. Finally, there are PTSD symptoms that jeopardise the capacity to function effectively at work, such as diminished ability to concentrate, irritability and loss of interest in work or school. In short, there is a perceived discontinuity between the pre- and post-traumatic self. People with PTSD see themselves as altered by their traumatic experience. They feel as if they have been drastically and irrevocably changed by this encounter. Others have described this discontinuity as a ‘broken connection’ with the past [11]; or as ‘shattered assumptions’ about oneself and one’s world [12].
HISTORICAL ANTECEDENTS

Before the mid-nineteenth century, the psychological impact of exposure to traumatic stress was recorded by poets, dramatists and novelists. Trimble [13], Shay [14] and others have pointed out that Homer, Shakespeare and Dickens (to name only a few) had sophisticated understanding of the profound impact of traumatic stressors on cognitions, feelings and behaviour. Medicalisation of such invisible wounds, usually (but not always) received in combat, occurred on both sides of the Atlantic during the mid-nineteenth century. Explanatory models pointed to the heart (e.g. soldier’s heart, Da Costa’s syndrome and neurocirculatory asthenia), the nervous system (e.g. railway spine, shell shock) and the psyche (e.g. nostalgia, traumatic neurosis) as the (invisibly) affected system.

In the 1970s, spurred on by social movements in the USA and around the world, what had previously been contextualised primarily as a problem among military personnel and veterans was broadened to include victims of domestic violence, rape and child abuse. The women’s movement emphasised sexual and physical assault on women while child advocacy groups emphasised physical and sexual abuse in children. Thus, new clinical entities took their places alongside combat-related syndromes. These included: rape trauma syndrome, battered woman syndrome, child abuse syndrome and others [15–17].

In other words, by the late 1970s clinicians had a wide variety of post-traumatic diagnostic options from which to choose, although none were recognised in the DSM-II [18]. Indeed, from a PTSD perspective, DSM-II was a step backwards, since DSM-I [19] contained the ill-defined ‘gross stress reaction’, which provided a useful, but temporary, diagnostic niche for military veterans, ex-prisoners of war, rape victims and Nazi Holocaust survivors. (If ‘gross stress reaction’ persisted, the diagnosis had to be changed to ‘neurotic reaction’.) In DSM-II, however, even this diagnostic option was eliminated, so that ‘situational reaction’ was the only available diagnosis for people who exhibited clinically significant reactions to catastrophic experiences. Besides trivialising post-traumatic reactions (since this category included any unpleasant experience), ‘situational reactions’ were also considered temporary.

The DSM-III [3] process recognised that these differently labelled syndromes (e.g. rape trauma, post-Vietnam, war sailor, concentration camp syndromes, etc.) were all characterised by a very similar pattern of symptoms that became embodied within the PTSD diagnostic criteria. Hence, the emphasis shifted from the specific traumatic stressor to the relatively similar pattern of clinical expression that could be observed among survivors of a growing list of different severe stressful experiences. The various stressors were aggregated into Criterion A, while the clinical presentation was explicated by the PTSD symptoms themselves (Criteria B–D).
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 (reexperiencing, numbing and miscellaneous) have been rearranged into the present triad of reexperiencing, avoidance/numbing and hyperarousal. Criterion E (duration of symptoms must exceed one month) was included in the DSM-III-R in 1987 and Criterion F (that the symptoms must cause clinically significant distress or functional impairment) was added in the DSM-IV in 1994. Most importantly, the fundamental concept that exposure to overwhelming stress may precede the onset of clinically significant and persistent alterations in cognitions, feelings and behaviour has endured. Epidemiological studies have confirmed the DSM-III perspective and shown that exposure to extreme stress sometimes precedes severe and long-lasting psychopathology [20–24]. Such research has also shown, unfortunately, that exposure to traumatic stress is all too common across the population and that the prevalence of rape, domestic violence, child abuse and so on is unacceptably high. Thus, when it was time for the next revision of the diagnostic criteria for DSM-IV [25] it was clear that it was incorrect to characterise Criterion A, exposure to a traumatic event, as an event that ‘is generally outside the range of usual human experience’.

**PTSD: DSM-IV-TR DIAGNOSTIC CRITERIA**

**Criterion A1**

The DSM-IV Criterion A was divided into objective (A1) and subjective (A2) components. Criterion A1 resembled the DSM-III-R [26] Criterion A, except that a greater number of events were included as 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. The ‘learning about’ traumatic exposure (injury or death) of a loved one has proven to be one of the most controversial changes to Criterion A (see below). In DSM-IV, however, in addition to exposure to an A1 event, it was necessary that exposed individuals experience an intense (fear-conditioned) emotional reaction (Criterion A2) characterised as ‘fear, helplessness or horror’. Although this had been foreshadowed in DSM-III-R’s text description, the subjective response was now made an explicit (A2) criterion [27]. It is also worth noting that the timing of A2 was unclear and later subject to different interpretations, with some saying it might happen some time after the event rather than being strictly peritraumatic.

As we consider DSM-IV Criterion A1, there are several questions that must be addressed: (i) Should exposure to a potentially traumatic event be considered aetiologically or temporally significant with regard to the later development of PTSD? (ii) Can we really distinguish ‘traumatic’ from ‘nontraumatic’ stressors? (iii) Should Criterion A1 be eliminated from DSM-5?
Does traumatic exposure ‘cause’ PTSD?

DSM-III and DSM-IV are unclear about the aetiological significance of the Criterion A event [27, 28]. On the one hand, they both suggest that traumatic exposure ‘causes’ PTSD (e.g. ‘evokes’ the characteristic PTSD symptoms). On the other, they both suggest that the traumatic event constitutes a watershed experience that temporally precedes the expression of PTSD symptoms.

We have learned a number of things since 1980 that have a direct bearing on this question. First, we know that people differ with regard to resilience and vulnerability, so that most people exposed to traumatic events do not develop PTSD. Epidemiological research has identified a number of risk and protective factors that differentially affect the susceptibility of different individuals to develop PTSD following exposure. Resilience is a complicated attribute that includes genetic, psychobiological, cognitive, emotional, behavioural, cultural and social components [7, 29]. Second, we must also recognise that events differ with regard to the conditional probability that PTSD will follow exposure. For example, the conditional probability of PTSD following rape 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 recognise that some events are much more likely to precede PTSD onset than others. It is more appropriate to consider the stressor as a powerful temporal antecedent with a variable conditional probability of preceding the development of PTSD than as an event that ‘causes’ PTSD. Such a conceptualisation tempers the attribution of causality and makes it possible 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 a sufficient condition for the subsequent development of PTSD. With this understanding, however, it must be understood that exposure to the traumatic event is absolutely critical, genetic loading notwithstanding [30].

As noted by Kilpatrick et al. [31] when summarising findings from the DSM-IV Field Trials, the argument over how best to operationalise Criterion A boils down to a debate over how broad versus how narrow Criterion A should be. A broad definition of Criterion A would include any event that can produce PTSD symptoms. In contrast, advocates for a more restrictive definition fear that broadening the criterion would trivialise the PTSD diagnosis and defeat the purpose of the original DSM-III PTSD construct by permitting people exposed to less stressful events to meet Criterion A. The DSM-IV Field Trials appeared to allay this concern as few people developed PTSD unless they experienced extremely stressful life events. Kilpatrick et al. [32] have recently replicated this Field Trial finding in two independent cohorts, the Florida Hurricane Study (FHS) and the National Survey of Adolescents (NSA). They found that among FHS study participants, 96.6% of those meeting PTSD Criteria B–F had previously been
exposed to an A1 event. In the NSA study, 95.5% of those meeting Criteria B–F 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 recognisable traumatic event, as stipulated in DSM-IV.

Others, less comfortable with the greater number of qualifying A1 events in DSM-IV than in DSM-III, have objected that expansion of qualifying A1 events has diluted the basic PTSD construct. They have argued that under DSM-IV people who have received the PTSD diagnosis for less threatening events should really be diagnosed with an adjustment or anxiety disorder not otherwise specified (NOS) [32]. The major sticking point has been the DSM-IV addition of being ‘confronted with’ (or learning about) traumatic experiences of family members or close friends. This expansion has been called ‘bracket creep’ [30] or ‘criterion creep’ [33] 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 [34] tested the implications of the broad DSM-IV Criterion A1 verus DSM-III. Among a representative sample of over 2000 individuals, lifetime exposure to traumatic events defined by a narrow set of qualifying A1 events was compared to prevalence of exposure to a broad set of events. The narrow set 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.). The broad set further included five events from the category ‘learning about’ traumatic events affecting close relatives (e.g. rape, assault, accident, etc.). Narrow-set exposure was 68.1% compared to broad-set exposure of 89.6%. Thus, there was a 59.2% increase in lifetime exposure to a traumatic event due to the expanded Criterion A1. More importantly, A1 events included within the expanded Criterion A1 contributed 38% of total PTSD cases. Although the wide discrepancy between the Kilpatrick et al. [31] and Breslau and Kessler [34] studies may have more to do with methodology than with Criterion A1 itself, [27] this finding has fuelled the controversy about how best to operationalise Criterion A1.

Kilpatrick et al. [32] have disputed the ‘bracket/criterion creep’ arguments. They point out that the DSM-IV Field Trials, as well as the aforementioned FHS and NSA data, indicate that very few individuals meet PTSD Criteria B–F without prior exposure to an A1 event. Brewin et al. [35] make a similar argument (see below). The non-A1 events most likely to precede the onset of PTSD B–F symptoms were sudden death of close relatives, serious illness and having a child with a potentially terminal illness [31, 32, 34]. One might ask whether these current non-A1 events should be redesignated as A1 events and if so, whether that would dilute the PTSD construct.

Dohrenwend [36] has suggested a different and very thoughtful approach to this issue. He has proposed that prototypical major negative events be rated objectively along six dimensions: valence (negative), source (external, uncontrollable,
‘fateful’), unpredictable, central (life-threatening, deprivation of basic needs and goals), magnitude (likelihood of causing great negative changes) and likelihood to exhaust the individual. He further proposes that research be done to empirically derive A1 events by detecting which of these six dimensions reliably predict PTSD B–F symptoms. Events characterised by such dimensions would be designated A1 events while others would not. Dohrenwend has also argued that such a dimensional approach would obviate the need for a subjective Criterion A2 (see below). Research on this approach would be extremely useful. It would also be important to address the question of clinical feasibility by determining how well busy clinicians could utilise Dohrenwend’s approach in clinical practice.

It seems that major questions regarding Criterion A1 can only be addressed through more research. The basic investigative approach would require the development of a comprehensive menu of prototypical major negative events in order to find out which reliably precede the onset of PTSD B–F symptoms and which do not. In order to ensure generalisability, both clinical and population samples that included sufficient diversity to address related questions regarding trauma type (e.g. sexual, military, disaster), gender, ethnicity, age, cultural and other factors would be needed. Dohrenwend’s dimensional proposal could also be investigated in such a design. A longitudinal approach to this question would be best (ideally starting before traumatic exposure, but at the very least beginning immediately after such exposure).

Should Criterion A1 be eliminated?

It has been suggested that PTSD caseness and prevalence would change very little if Criterion A1 were completely eliminated. The DSM-IV PTSD Work Group also considered complete elimination of Criterion A but rejected this option because of concerns that ‘the loosening of Criterion A may lead to widespread and frivolous use of the concept’ [37]. Although several articles suggest that the full PTSD syndrome might be expressed following nontraumatic events (thereby fortifying ‘bracket/criterion creep’ arguments [30, 33]), most of these reports have been dismissed as methodologically flawed because proper clinical interviews are not utilised and because the data merely show an increase in PTSD symptoms, but not the full diagnosis. Indeed, when assessed by a structured clinical interview, there are actually very few examples of individuals who do not meet Criterion A who do meet full PTSD diagnostic criteria [35]. Furthermore, it is unclear in most of these reports whether the non-A1 event actually served as a reminder or trigger for a previously experienced traumatic event and therefore precipitated a PTSD relapse, rather than new-onset PTSD.

Arguments for eliminating Criterion A are: (i) traumatic exposure may sometimes precede onset of other diagnoses (e.g. depression, substance-use disorder) rather than PTSD; (ii) non-A1 events sometimes do appear to precede onset of PTSD B–F symptoms; (iii) it would bring PTSD more in line with other anxiety
and affective disorders which do not require that symptom onset be preceded by a specific event; and (iv) lack of utility of Criterion A2 [35]. Most PTSD experts, responding to an unpublished survey undertaken by APA as part of the DSM-5 process, strongly supported retaining Criterion A1 but generally agreed that it needed to be modified to address the issues discussed in this review. Suggested modifications included: emphasising the temporal rather than the aetiological relationship between A1 and B–F symptoms, narrowing the criterion to eliminate second-hand exposure (e.g. the ‘confronted by’ criteria) and incorporating Dohrenwend’s dimensional approach. All agreed that any final decisions should be informed by empirical evidence.

**Criterion A2**

As noted above, the DSM-IV Work Group stipulated that in addition to exposure to an A1 event, individuals thus exposed must also experience an intense subjective reaction characterised as ‘fear, helplessness or horror’. It was expected that imposition of Criterion A2 would ensure that the only people eligible for the PTSD diagnosis would be those who had reacted strongly to the threatening event. It was also expected that imposition of this new Criterion A2 would function as a ‘gatekeeper’ and keep out any ‘frivolous’ PTSD diagnoses due to broadening of Criterion A1. The expectation, based on data from the DSM-IV Field Trials [31], was that few people exposed to low-magnitude (nontraumatic) events would meet Criterion A2 and therefore that most would not be eligible for the PTSD diagnosis.

Research indicates that DSM-IV’s expectations regarding A2 have not been realised. As a result, the utility of Criterion A2 has been seriously questioned. Three negative studies found no effect of A2 on PTSD prevalence: in a community sample from Michigan; in a sample of older male military veterans; and in the World Health Organization’s World Mental Health Survey, which included almost 103,000 respondents [34, 38, 39].

People whose occupation requires frequent traumatic exposure, such as military, police and emergency medical personnel, may not experience fear, helplessness or horror during or immediately following a trauma exposure because of their training. Other studies show that a substantial minority of individuals within community samples (e.g. ~20%) may meet all PTSD A1, B–F Criteria without meeting A2. Except for the absence of A2, there were no differences with regard to severity or impairment between A2 positive and A2 negative cohorts [40, 41]. Similar results have been found with recent female rape or assault victims [42]. Furthermore, people can develop PTSD following mild traumatic brain injury (TBI), in which case they may be unaware of any peritraumatic emotional response because of a loss of consciousness [43, 44]. These examples all indicate that some people can develop PTSD without an A2 response.
Another problem with A2 concerns the timeframe in which it is assessed. Since most PTSD cases are evaluated months or years after a traumatic event, and since assessment of A2 requires a retrospective recall of how the person responded during or shortly after the event, there is concern that subsequent recall of acute responses to trauma is unreliable and is influenced by mood biases associated with PTSD levels (or other factors) at the time of recall [45]. Therefore, questions about the accuracy of retrospective A2 reports obtained at varying intervals between trauma exposure and assessment have raised additional concerns about the usefulness of A2.

Based on all of this information, a number of investigators have called for the elimination of Criterion A2. Not only has it failed to predict the likelihood of PTSD, but it has also failed to realise the expectations of DSM-IV that it would serve as a ‘gatekeeper’ to offset any increased prevalence of PTSD caused by the expansion of qualifying A1 events [40]. McNally [30] has argued that we should eliminate A2 because ‘in the language of behaviourism, it confounds the response with the stimulus. In the language of medicine, it confounds the host with the pathogen’ (page 598).

On the other hand, there is consistent evidence that the absence of A2 strongly predicts A1-exposed people who will not develop PTSD [31, 34, 38, 39, 46]. Schnurr et al. [38] suggest that A2 may be most useful during the immediate aftermath of a traumatic event, by identifying individuals unlikely to develop PTSD. While this may be extremely useful in a war zone or disaster triage site, it does not appear to have a major bearing on improving diagnostic accuracy.

Finally, A2’s ‘fear, helplessness and horror’ are all predicated on a fear-conditioning model of PTSD. This has been challenged as too narrow. There is now considerable data showing that other strong peritraumatic emotions are also associated with PTSD, such as: sadness, grief, anger, guilt, shame and disgust [31, 46–48].

**Summarising Criteria A1 and A2**

As DSM-5 moves forward, a major priority will be to address the aforementioned concerns regarding Criterion A. For A1, it will have to reduce the ambiguity about what is and what is not a traumatic event. For A2, it will have to consider the utility of this criterion in making the PTSD diagnosis and whether ‘fear, helplessness or horror’ should be expanded to include both peritraumatic dissociation and other intense peritraumatic emotions such as guilt, shame and anger. Given that peritraumatic emotions are likely to endure among those who do not recover from traumatic events and are eventually diagnosed with PTSD, it seems appropriate to include non-fear-based post-traumatic symptoms in DSM-5 [28].

Kilpatrick et al. [32] suggest that a key question about Criterion A is whether it should be designed to maximise sensitivity (thereby including all events that are
capable of producing PTSD) or whether it should maximise specificity (thereby limiting qualifying events to those most likely to precede PTSD). A broad, less restricted definition would ensure that all individuals meeting other PTSD criteria would be eligible for treatment or other services. A more restricted definition would resolve current ambiguities in tort or compensation cases. Kilpatrick et al. maintain that until consensus has been achieved regarding sensitivity versus specificity, it will be impossible to define Criterion A.

The proposed DSM-5 criteria for PTSD [28] have retained Criterion A. It is expected that in the narrative description its temporal rather than aetiological significance will be emphasised. The major reason proposed for retaining Criterion A is that PTSD does not develop unless an individual is exposed to an event that is intensely stressful. Such individuals are keenly aware of a significant discontinuity in their lives because of subsequent preoccupation with memories, feelings and behaviours that are associated with that event. This is consistent with recommendations from other investigators. For example, McNally [30] has argued that the memory of the trauma is the ‘heart of the diagnosis’ and the organising core around which the B–F symptoms can be understood as a coherent syndrome.

Proposed DSM-5 diagnostic criteria for PTSD [28] indicate that Criterion A1 will probably not change substantially because there is insufficient data to address the concerns outlined in this review. It has retained DSM-IV language emphasising that qualifying events must involve direct exposure to actual or threatened death, serious injury or a threat to the physical integrity of others. With regard to the most controversial aspect of DSM-IV Criterion A1, being ‘confronted by’ traumatic events, the proposal for DSM-5 limits such ‘confrontation’ to learning about the traumatic exposure of a close friend or loved one or learning about aversive details of unnatural deaths, serious injuries or serious assaults to others. This includes learning about the homicide of a family member, learning about a gruesome death or learning the grotesque details of rape, genocide or other abusive violence to others. It 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 Criterion A explicitly excludes witnessing traumatic events through electronic media, television, video games, movies or pictures.

Because of aforementioned concerns about differences in resilience and gene × environment interactions, there is legitimate concern that vulnerable individuals might develop bonafide B–F symptoms following events not generally considered ‘traumatic’. The proposed DSM-5 solution to this diagnostic issue is the addition of an ASD/PTSD subtype of AD. Such an approach would provide a diagnostic niche for vulnerable individuals who express PTSD B–F symptoms following exposure to a nontraumatic event [2].

As for Criterion A2, the current proposal is to eliminate it in DSM-5 for all the reasons cited above [28].
Factor structure of PTSD

The DSM-IV PTSD construct consists of three symptom clusters: B, reexperiencing; C, avoidance/numbing; and D, hyperarousal. Many studies have utilised confirmatory factor analysis to test whether the three symptom clusters of DSM-IV provide the best model for the latent structure of PTSD. A thorough review of this extensive literature can be found elsewhere [49]. In short, the vast majority of studies support a four-factor model. Five support a two-factor solution. Of the four studies supporting a three-factor model, only one mirrors the three factors found in the DSM-IV PTSD diagnostic criteria [50].

Among the four-factor models, reexperiencing, avoidance and arousal have emerged as distinct clusters in all studies. There has been disagreement, however, about the fourth factor. In many studies, ‘numbing’ has been identified, while a ‘dysphoria’ factor has emerged elsewhere [28]. What is most noteworthy, from a DSM-IV perspective, is that none of these studies support a single avoidance/numbing cluster. The general dysphoria factor might be considered to be related to the negative emotional state frequently observed among individuals with PTSD [47]. It also supports arguments that PTSD should be considered an internalising disorder within the dysthymic/anxious misery subcategory along with major depression, dysthymia and general anxiety disorder [51, 52].

In summary, most confirmatory factor analyses support a four-, rather than a three-factor DSM-IV model. The majority of studies indicate that serious consideration should be given to including a separate fourth, ‘numbing’, symptom cluster in DSM-5. Finally, there is virtually no evidence in support of DSM-IV’s Criterion C (avoidance/numbing) since avoidance and numbing are consistently distinct from one another in both the four- and two-factor solutions.

Based on these findings, the proposed DSM-5 criteria for PTSD [28] are nested within a four-factor model. The reexperiencing, avoidance and arousal (now renamed ‘arousal and reactivity’) clusters have remained. The fourth factor, ‘negative alterations in cognitions and mood’ replaces the DSM-IV numbing cluster. Most DSM-IV symptoms have been retained, although some have been redefined, while three new symptoms have been introduced (see below).

Can the B–D symptom clusters be improved?1

In addition to the fear-based anxiety symptoms, which provide the context for the current DSM-IV PTSD diagnostic criteria, the empirical literature strongly suggests that, as noted earlier in this review, traumatic exposure may be followed

1 The 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 author, Patricia Resick, Chris Brewin, Richard Bryant, Dean Kilpatrick, Roberto Lewis-Fernandez, Katherine Phillips, Terry Keane, David Spiegel, Robert Ursano, Robert Pynoos and Eric Vermetten participated in this process. The official review of that work can be found in Friedman et al. [28].
by a variety of non-fear-based anxiety symptoms such as dysphoric anhedonic 
symptoms, aggressive/externalising symptoms, guilt/shame symptoms, dissoci-
ative symptoms and negative appraisals about oneself and the world [28, 47]. Such 
findings suggest that the DSM-IV PTSD diagnostic criteria should be revised 
to incorporate such symptoms in order to provide a better characterisation of 
the spectrum of post-traumatic symptomatology encountered by clinicians on a 
regular basis.

Criterion B

Traumatic nightmares (B2) and dissociative flashbacks (B3) rank among the most 
recognisable and distinctive symptoms of PTSD. Indeed, some have suggested 
that only these two symptoms should be retained in the B cluster symptoms 
and that others be eliminated because they overlap with symptoms seen in other 
disorders [53]. Specifically, they argue that intrusive recollections (B1) be elimi-
nated because it is too similar to rumination seen in depression, while emotional 
and physiological arousal following exposure to traumatic reminders (B4 and 
B5) are too similar to symptoms found in specific and social phobia disorders. 
Despite such concerns, the current proposed DSM-5 criteria [28] have retained 
all three (B1, B4 and B5) controversial symptoms but have modified them to 
address these concerns.

The concern about B1 is that DSM-III/IV ‘recurrent intrusive recollections’ 
includes both intrusive images and thoughts, as discussed in more detail else-
where [28]. There is a growing body of evidence to show that intrusive imagery 
and recurrent thought processes such as ruminations are quite distinct [54], with 
the former occurring uniquely in PTSD and the latter also found in other disor-
ders such as depression. The intrusive images in PTSD are sensory memories of 
short duration, have a here-and-now quality and lack context, while ruminative 
thoughts in depression are evaluative and longer lasting. In addition, rumination 
appears to function as a cognitive avoidance strategy [35, 55–57]. Therefore, 
the proposed DSM-5 Criterion B1 was revised to eliminate thoughts/ruminations 
and to restrict this criterion to involuntary and intrusive distressing memories 
that usually include sensory, emotional, physiological or behavioural (but not 
cognitive) components.

B4 and B5 are triggered intrusive emotional and physiological experiences, 
respectively. The B4 and B5 criteria have been retained because it appears that 
elicitation of emotional and physiological reactivity to trauma-related stimuli is 
a key characteristic of PTSD. It is consistent with major fear-conditioning mod-
els of the disorder. It is the guiding rationale for critical laboratory paradigms 
in which distinctive alterations in psychological and neurobiological reactivity 
among PTSD participants can be reliably detected after exposure to trauma-
related stimuli [58]. Furthermore, it is a principle that has informed our most 
effective cognitive behaviour therapies (CBTs), where emotions and cognitions
elicited by traumatic reminders are processed therapeutically. B4 is intense emotional distress, which may be the only kind of recollection possible in individuals who sustained a TBI and have no conscious memories of the traumatic event. Indeed, it has been shown 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 [44]. In other words, these symptoms are conditioned responses in fear-conditioning models.

Criterion C

In accordance with the confirmatory factor analysis results reviewed above, DSM-IV’s avoidance/numbing criterion will be split into C (avoidance) and D (negative alterations in cognitions and mood clusters). As stated previously, the proposed DSM-5 criteria now reflect the true latent structure of PTSD, which consists of four, rather than three factors.

The avoidance (C1 and C2) symptoms are completely consistent with a fear-conditioning model of PTSD. Indeed, Brewin et al. [35] have proposed that these are the only C-cluster symptoms uniquely associated with PTSD. C1 consists of efforts to avoid internal reminders associated with the traumatic events (e.g. thoughts, feelings or physical sensations) while C2 consists of efforts to avoid external reminders of the traumatic event (e.g. people, places, conversations, activities, objects or situations). These avoidance symptoms have been preserved in the proposed criteria for DSM-5.

Perhaps the most noticeable change in the DSM-5 proposal is the new D cluster, which encompasses, redefines and recontextualises the DSM-IV C3–C7 ‘numbing’ symptoms. Some have proposed complete elimination of those symptoms because of their nonspecific overlap with general dysphoric symptoms present in other disorders [35, 59] and with the anhedonia found in depression [60], while others have argued strongly for expanding this cluster beyond a fear-conditioning context [28, 47]. As noted earlier, there are a number of negative appraisals and mood states associated with PTSD which have not been clearly explicated or included in DSM-III/IV. Some have been vaguely embedded within C3–C7 while others have not been included at all. According to the proposed criteria for DSM-5, they are now included in a unique cluster of symptoms which are distinct from reexperiencing, avoidance and arousal/reactivity symptoms.

There is very strong evidence that catastrophic or maladaptive appraisals are characteristic of traumatic stress responses that are associated with disorder or impairment [61]. Erroneous cognitions about the causes or consequences of the traumatic event, which lead individuals with PTSD to blame themselves or others, are major therapeutic targets in CBT. They are frequently found among survivors of childhood sexual abuse, rape/assault survivors and military personnel. Indeed, specific and effective strategies to address such self-blame is a consistent component of CBT for PTSD patients [61–63]. Sometimes, such self-blame is
due to perceived personal failings, inadequacies or weakness [64, 65]. Therefore, given the prominence of self-blame among individuals with PTSD, it has been proposed as a new symptom for DSM-5.

Another maladaptive appraisal is DSM-IV symptom C7, a sense of foreshortened future, which has often been interpreted too narrowly in DSM-IV as the ‘belief that one’s life will be shorter or changed’. The empirical literature supports the observations of CBT therapists that individuals 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 [64, 66–70]. This has been retained in DSM-5 with the explicit stipulation that it concerns persistent negative expectations regarding many important aspects of life and not just a negative expectation about one’s life span.

In addition to the negative appraisals about past, present and future included in the proposed DSM-5 Criterion D, people with PTSD have a wide variety of negative emotional states besides fear, helplessness and horror. Indeed, one of the arguments for moving PTSD out of the Anxiety Disorders category is the presence of many other negative mood states [71]. These include anger [69, 72–75], guilt [76–78] and shame [72, 79, 80]. This is the rationale for proposing a pervasive negative emotional state ‘as a new symptom for DSM-5’.

There is abundant evidence for retaining other symptoms currently included in the DSM-IV numbing (C3–C7) cluster. These include dissociative amnesia [81, 82], diminished interest in significant activities, feeling detached or estranged from others and psychic numbing: persistent inability to experience positive emotions. These are all consistently endorsed by individuals with PTSD, as shown in the many confirmatory factor analysis studies reviewed above.

To summarise, it is proposed that the DSM-IV avoidance/numbing cluster be divided into two separate clusters: Criterion C, ‘Persistent Avoidance of Stimuli Associated with the Trauma’ and Criterion D, ‘Negative Alterations in Cognitions and Mood Associated with the Trauma’. The dissociative aspects of amnesia are emphasised. The expectation of a foreshortened future has been explicitly expanded to include negative expectations about one’s self, others, or one’s future. Other DSM-5 symptoms that have been retained unchanged are: diminished interest in significant activities, feeling detached or estranged from others and psychic numbing. 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.

**Criterion D**

Four of the five DSM-IV Criterion D symptoms are endorsed frequently by individuals with PTSD and are retained, unchanged, in the proposed DSM-5
criteria. These are insomnia, problems in concentration, hypervigilence and startle reactions. A review of the literature suggests, however, that this symptom cluster encompasses more than hyperarousal and should also include alterations in reactivity that are associated with the traumatic event. Such a reframing of this symptom cluster makes it possible to include behavioural as well as emotional indicators of such post-traumatic alterations. Therefore, it has been proposed that two new symptoms be added to this cluster: aggressive behaviour and reckless behaviour [28].

There is growing evidence, especially among military veterans, that PTSD is associated with more than an irritable mood state (DSM-IV’s C2). Indeed, it appears that PTSD predicts aggressive behaviour and violence among military or veteran cohorts following deployment to a war zone [83–86]. Aggressive behaviour has also been observed among female flood survivors with PTSD [87]. Sometimes the aggressive behaviour, rather than other PTSD symptoms, becomes the major clinical focus. Such evidence prompted the DSM-5 proposal to include new-onset post-traumatic aggressive behaviour.

There is also growing evidence that PTSD is associated with reckless and self-destructive behaviour. This has been reported among Israeli adolescents, especially boys, exposed to recurrent terrorism, who exhibited marked increases in risk-taking behaviour [88]. Reckless driving has been observed among individuals with PTSD [89–91]. Finally, risky sexual behavior, sometimes associated with HIV risk, has been reported among college women, female prisoners and adult male survivors of childhood sexual abuse [92, 93]. Based on this evidence, it has been proposed that post-traumatic reckless behaviour be included as a new symptom in DSM-5.

**Duration – Criterion E**

A month must elapse between traumatic exposure and eligibility for a PTSD diagnosis. The DSM-III/IV rational for this stipulation is to set aside a window in which normal recovery can take place. Since most people exhibit distress during the acute aftermath of traumatic exposure, this one-month window provides an interval for normal post-traumatic recovery without pathologising normal distress exhibited during this period. Individuals who exhibit clinically significant distress or functional impairment during this initial month may qualify for either an ASD or an AD diagnosis (see below). The DSM-5 PTSD proposal preserves this one-month interval. It should be noted that because Criteria B–D in DSM-IV have been expanded to Criteria B–E in DSM-5, Criterion E in DSM-IV will become Criterion F in DSM-5.

‘Delayed-onset’ PTSD is also preserved in the DSM-5 proposal. This is an important consideration with regard to disability claims as well as forensic psychiatry. It appears that when delayed onset of the full PTSD syndrome occurs, it
is preceded by the presence of subsyndromal PTSD symptoms that have increased to exceed the diagnostic threshold. It is very rare for people to suddenly develop PTSD months or years after the trauma when they had been completely asymptomatic during this prodromal period. Indeed, delayed onsets of PTSD, which generally represent exacerbations or reactivations of prior symptoms, have been estimated to account for 38.2 and 15.3% of military and civilian cases of PTSD, respectively [94]. It is no longer controversial, however, that delayed-onset PTSD does actually occur [95–97], although it appears to be very uncommon after natural disasters [98]. Based on the current literature, it has been suggested that this delayed clinical trajectory might be better described as delayed-‘development’ PTSD rather than ‘onset’, since PTSD symptoms are usually present well before the traumatised individual meets full PTSD diagnostic criteria.

The final DSM-5 proposal concerning duration is to eliminate the current three-month demarcation point between acute and chronic PTSD. This DSM-IV distinction is based on longitudinal studies of rape victims [99] and motor-vehicle accident survivors [100], which indicate that initially high PTSD rates tend to decline steeply and approach an asymptote at three months. Given a paucity of research on this subject as well as questions about the utility of such a distinction, it has been proposed that the distinction between acute and chronic PTSD be eliminated in DSM-5.

**Functional impairment – Criterion F**

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 Criteria A–E would not receive a PTSD diagnosis unless he or she also exhibited clinically significant distress or functional impairment. Some regard this question as somewhat redundant since ‘significant distress’ is implicit in many PTSD symptoms. Because Criterion F 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 both be assessed independently, and if so, how? Second, should significant distress and/or functional impairment remain a diagnostic criterion? If not, how should such information be incorporated into a diagnostic assessment? However Criterion F is defined for DSM-5, it will become Criterion G, because Criteria B–D have expanded to Criteria B–E.

**Summary: proposed PTSD diagnostic criteria for DSM-5**

The proposed revisions incorporate the following evidence: (i) There is ambiguity in the wording of Criterion A1; (ii) Criterion A2 has no utility in predicting
onset of PTSD; and (iii) Criterion C appears to encompass two distinct clusters of symptoms, avoidance and numbing. The proposed diagnostic criteria represent a relatively conservative revision of DSM-IV with four clusters: reexperiencing symptoms, avoidance behaviour, negative alterations in cognitions and mood, and alterations in arousal and reactivity. Finally, in DSM-IV, only B1–5 and C1–2 are specifically anchored to the traumatic event. In these proposed criteria, it is stipulated that all B–G symptoms ‘began or worsened after the traumatic event’.

**ACUTE STRESS DISORDER**

ASD was introduced in DSM-IV to provide a diagnostic niche for acutely traumatised individuals with clinically significant post-traumatic symptoms within the first month following exposure to a traumatic event. Whereas PTSD cannot be diagnosed before one month has elapsed, ASD cannot be diagnosed after one month has elapsed. Since most people exhibit significant distress during the immediate aftermath of traumatic exposure, ASD cannot be diagnosed until at least two days have elapsed following the event. In military or post-disaster parlance, extreme distress during the first 48 hours is called an ‘acute stress reaction’ or ‘combat stress reaction’, a normal response to extreme stress from which normal recovery is expected. Finally, since most distressed acutely traumatised individuals will recover during the first month, most CBT therapists delay formal treatment until 10–14 days have passed since the traumatic event, to make sure that recovery is unlikely without psychotherapy.

Another important difference between ASD and PTSD is the greater emphasis on dissociative symptoms in the former diagnosis. In DSM-IV, an individual must exhibit at least three (out of five possible) dissociative symptoms to meet ASD diagnostic criteria, whereas none are required for PTSD. The five dissociative symptoms are: reduction in awareness, derealisation, depersonalisation, numbing and amnesia. Otherwise, an individual must only exhibit one symptom out of each of three PTSD symptom clusters: reexperiencing, avoidance and arousal.

As reviewed by Bryant *et al.* [101], there were two reasons for introducing ASD into DSM-IV: to describe clinically significant post-traumatic reactions during the first month and to predict survivors at high risk of developing subsequent PTSD [102, 103]. A systematic literature review assessed the prevalence and predictive capacity of ASD [104]. It also reviewed the predictive capacity of subsyndromal ASD (defined as meeting three of the four ASD symptom criteria, usually lacking the three-symptom dissociative requirement). The rates of full ASD range from 7 to 28% with a mean of 13%, whereas the prevalence of subsyndromal ASD has ranged from 10 to 32%, with a mean rate of 23%. The higher prevalence for subsyndromal PTSD is because a significant number of severely distressed people meet all ASD criteria except for dissociative symptoms during the first months after traumatic exposure.
Although people who meet ASD criteria are at higher risk for developing PTSD, the majority of people who develop PTSD have never met ASD criteria [105]. Predictability is considerably better for subsyndromal PTSD (when the requirement for three dissociative symptoms is eliminated). Bryant et al. [101] suggest that this may be at least partially explained by the fact that the reexperiencing, avoidance and arousal ASD symptom clusters are very similar to the same clusters in PTSD, while most ASD dissociative symptoms are not found in PTSD.

DSM-IV’s great emphasis on dissociation as a predictor of PTSD was based on evidence suggesting that peritraumatic dissociation was a risk factor for PTSD. Subsequent research has indicated that peritraumatic dissociation is not an independent predictor of PTSD [101]. Indeed, maintaining a requirement for these dissociative symptoms to meet ASD criteria has eliminated many high-risk, acutely traumatised individuals from consideration. Furthermore, persistent, rather than peritraumatic, dissociation appears to be more predictive of ASD and subsequent PTSD [106].

The proposed criteria for DSM-5 are predicated on the following: (i) clinically significant post-traumatic distress during the first month may take variable forms; (ii) dissociative symptoms may or may not be present during this period among people at high risk of developing PTSD; (iii) people who exhibit persistent distress during the first month following trauma exposure are at a higher risk of developing PTSD subsequently; and (iv) it is important to identify such individuals during this period so that they may receive effective CBT treatment in order to prevent the later development of PTSD [101]. As a result, the stipulation that acutely traumatised individuals meet ASD diagnostic criteria in each of four (dissociative, reexperiencing, avoidance and arousal) symptom categories has been eliminated in the proposed ASD criteria for DSM-5.

The proposed ASD Criterion A for DSM-5 is the same as that for PTSD, with clarification of Criterion A1 and elimination of A2 as discussed above. Individuals must exhibit eight out of the proposed fourteen symptoms, irrespective of the symptom category in which these symptoms reside. Proposed ASD symptoms for DSM-5 include: four reexperiencing symptoms (e.g. intrusive memories, traumatic nightmares, flashbacks and distress in response to traumatic reminders); three dissociative symptoms (e.g. emotional numbing, depersonalisation/derealisation and amnesia); two avoidance symptoms (e.g. avoidance of internal reminders and avoidance of external reminders); and five arousal symptoms (e.g. insomnia, hypervigilence, irritability, exaggerated startle response and agitation or restlessness). The proposed threshold of eight out of these fourteen symptoms is based on a recent analysis of three large datasets from Israel, the UK and Australia [101]. Future prospective longitudinal studies will determine how much the ASD diagnosis has been improved by these new criteria and whether the best threshold is eight rather than a different number of symptoms.
ADJUSTMENT DISORDERS

ADs are an important addition to the proposed DSM-5 category (or subcategory) of Anxiety Disorders, which encompass clinically meaningful syndromes preceded by a stressful or traumatic event (and which also include PTSD, ASD and dissociative disorders) [107]. ADs can be diagnosed at any time after an individual has been exposed to such events. Most precipitating events (e.g. rejections, failure, bankruptcy, etc.) are extremely distressing but do not exceed the threshold for a traumatic event, as in PTSD or ASD (see review by Strain [2]). If, however, an individual is exposed to a traumatic event but fails to meet other ASD or PTSD criteria, the proper diagnosis was AD in DSM-IV and will be the ASD/PTSD subtype of AD in DSM-5. Furthermore, if an individual exhibits the proposed DSM-5 ASD or PTSD symptoms without having been exposed to a traumatic event, he or she will also be diagnosed as the ASD/PTSD subtype of AD.

Because of its specificity, the ASD/PTSD subtype is an exception to all other AD subtypes in which the symptoms deliberately lack specificity. Strain [2] states that AD ‘constitutes a “lynchpin” between normality, problems of living and pathological psychiatric states’. The lack of specificity allows the tagging of early or temporary mental states when the clinical picture is vague and indistinct but the morbidity is greater than expected in a normal reaction. The proposed DSM-5 Criterion B is unchanged from DSM-IV and stipulates that AD symptoms must exhibit either functioned impairment or disproportioned distress after exposure to the stressor.

It is possible that AD is the greatest example of gene × environment interaction in the DSM since an event that constitutes a temporary but surmountable challenge for resilient individuals may precipitate a severe adjustment reaction in more vulnerable individuals. Furthermore, DSM-III’s six-month post-event window in which AD might be allowed (after which the diagnosis had to be dropped or changed to a more enduring psychiatric disorder) was changed in DSM-IV to a diagnosis of a chronic subtype if the stressor was still exerting its effects. It is proposed that this chronic subtype be retained in DSM-5.

A great advantage of AD is that its general nonspecificity has made it possible for clinicians to have a diagnosis that acknowledges the distressing and debilitating functional impairment of patients who require mental health treatment but do not meet criteria for any other psychiatric diagnosis. The problem is that because of such nonspecificity it has been very difficult to design epidemiological surveys, longitudinal studies or clinical treatment trials concerning people with this disorder. For the purposes of this review, addition of the uncharacteristically specific ASD/PTSD subtype may make it possible to conduct research regarding people who resemble ASD or PTSD patients but fail to meet full diagnostic criteria for either disorder. This is particularly useful since it is unlikely that a subsyndromal PTSD will be introduced into DSM-5.
PARTIAL/SUBSYNDROMAL PTSD

PTSD does not have a subsyndromal diagnosis comparable to dysthymia and cyclothymia in major depression and bipolar affective disorder, respectively. The argument for adding such a diagnosis is that considerable research indicates the likelihood of partial/subsyndromal PTSD and that it would have clinical utility by characterising people with clinically significant post-traumatic reactions who fail to exceed the PTSD diagnostic threshold (often for lack of one or two symptoms) and for whom a diagnosis of AD has previously been too nonspecific. The arguments against addition of a new subsyndromal category are: (i) that AD, especially with the proposed ASD/PTSD subtype, is the appropriate diagnosis for such individuals; and (ii) that it over-pathologises normative reactions. Approximately 60 publications have reported on the prevalence and morbidity of ‘partial/subsyndromal’ PTSD among a wide assortment of traumatised individuals. Unfortunately, throughout these studies partial PTSD has been defined differently by different investigators [28].

In some studies, there is evidence that partial PTSD has a similar relationship to full PTSD as dysthymia has to major depressive disorder or cyclothymia has to bipolar disorder. People with partial PTSD exhibit significantly less symptom severity and functional impairment that those with the full syndrome, but significantly more than no-PTSD cohorts. There are also negative reports, however, in which few differences are detected between partial- and no-PTSD cohorts, although both differed significantly from full PTSD [28].

As stated previously, it is difficult to compare and interpret many of these findings because partial PTSD was defined differently in different studies. Therefore, future research should be based on a standard set of diagnostic criteria so that questions about the uniqueness and utility of partial PTSD can be addressed appropriately. At this time, however, it appears unlikely that partial subsyndromal PTSD will appear as a distinct diagnosis in DSM-5. It is more likely that the ASD/PTSD subtype of AD will be expected to fill this diagnostic niche.

DESNOS/COMPLEX PTSD

Since the work of Judith Herman [108], there have been complaints that the PTSD symptom clusters fail to encompass clinically significant problems often exhibited by individuals exposed to severe and protracted traumatic events (most notably victims of childhood sexual abuse and adult refugees and torture survivors). It’s not that such individuals usually fail to meet PTSD diagnostic criteria, but, rather, that their most significant clinical symptoms are not included within the PTSD construct. These include: behavioural 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 – dissociative identity disorder), interpersonal difficulties and somatisation [108–110].

In characterising the psychiatric sequelae of protracted child abuse, DESNOS (disorders of extreme stress not otherwise specified) has been considered a very useful construct as well as an appropriate diagnosis for the clinical pattern seen in adult (often) non-Western patients exposed to forced migration or torture [111, 112], although there has been little research in this area.

DESNOS was carefully considered for inclusion in DSM-IV and assessed during the DSM-IV Field Trials. 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. Despite these findings, there is continued support for DESNOS in certain quarters and continued advocacy for its inclusion in DSM-5. During recent years, there has also been some research designed to clarify the prevalence and construct validity of DESNOS. Unfortunately, neither the quality nor quantity of such research has been persuasive [49].

Questions regarding the uniqueness and utility of DESNOS are far from settled. Given the strong belief among many seasoned clinicians that this is an important diagnosis that should be included in the DSM, we need rigorous research to determine whether exposure to trauma, especially during developmentally sensitive periods, may lead to a different pattern of symptoms than those included in PTSD. In a cross-cultural context, DESNOS may be especially useful because it emphasises both dissociation and somatisation, two symptoms not included in the DSM-IV PTSD diagnostic criteria that are frequently observed in traumatised non-Western cohorts [113]. It has also been reported that more complex cases of PTSD involve deficits in emotional regulation [114]. As a result, it has been proposed 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 behaviours. Future research could usefully assess the extent to which emotion-regulation symptoms load on to a separate factor and do in fact characterise individuals with PTSD who also exhibit such self-destructive/emotional regulation problems. Identification of such a factor would be consistent with evidence that these patients benefit from specific treatments that precede traditional trauma-focused CBT with emotion-regulation training sessions [115].

Friedman et al. [28] suggest that another way to contextualise DESNOS might be to consider a spectrum of PTSD subtypes in which some people appear to have simple PTSD, some have additional internalising behaviours and symptoms, and some exhibit externalising behaviours along with their PTSD [71, 116]. It’s possible that this approach might offer a more parsimonious construction of complex PTSD. Somatisation symptoms fall on the internalising dimension, along with depression and anxiety, while anger/aggression, substance abuse and behaviours
indicative of cluster B axis II disorders fall on the externalising dimension. Both models can be tested empirically.

**SUBTYPES OF PTSD**

It has been proposed that there may be two subtypes of PTSD that involve (i) ongoing ‘dissociative’ versus (ii) ‘hyperarousal’ reactions [117, 118]. In this context, dissociation has been conceptualised as an avoidance strategy to reduce awareness of aversive emotions such as extreme anxiety [119]. For example, it has been shown 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 peritraumatic dissociation [119]. Neuroimaging studies have provided additional evidence that there may be distinctive prefrontal responses to trauma memories in individuals with dissociative and nondissociative PTSD responses [120]. For example, the ‘hyperarousal’ PTSD response to traumatic narratives was associated with reduced bilateral medial frontal activity and left anterior cingulate activity relative to controls [121]. In contrast, individuals displaying a ‘dissociative’ PTSD response had reduced amygdala but significantly increased right medial frontal, right medial prefrontal and right anterior cingulate activity relative to controls [122]. Lanius *et al.* have hypothesised that the heightened prefrontal activity in dissociative PTSD may reflect greater emotional regulation and inhibition of limbic emotional networks, including amygdala [122]. Other neuroimaging findings that distinguish dissociative from nondissociative PTSD have also been reported [28].

These findings are controversial. Several studies have not found differences in autonomic arousal between dissociative and nondissociative individuals with PTSD [123–125]. It appears that our current understanding of the role of dissociation in relation to arousal is very limited, and it is unclear whether there is sufficient evidence to include a unique dissociative subtype of PTSD in DSM-5.

**CROSS-CULTURAL FACTORS**

PTSD has been criticised as a culture-bound Euro-American construct that has little relevance to the rest of the world, especially to traditional societies [126]. For example, somatisation and dissociation, two common expressions of post-traumatic distress in traditional societies [113], are missing from DSM-IV PTSD diagnostic criteria (but not DESNOS). Whereas there may be culture-specific idioms of distress that provide a better characterisation of PTSDs found in one ethnocultural context or another [127], PTSD has been documented throughout the world [128–130]. Specifically, high prevalence rates have been reported in
non-Western nations such as Algeria, Cambodia, Lebanon, Palestine, Nepal and the former Yugoslavia [131–133]. Furthermore, comparable PTSD prevalence has been found among Russian and American adolescents [134].

Unfortunately, direct comparisons in which similar traumatic events affected people from widely different cultures (Western versus non-Western) are rare. A notable exception is a study by North et al. [135], 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. Kenyans and Americans exposed to these events exhibited remarkably similar impacts with respect to death, injury, destruction and other consequences. In addition, PTSD prevalence among Africans and Americans exposed to the events was also very similar.

**DEVELOPMENTAL ISSUES**

Maturational biological and psychological changes affect the appraisal of and reaction to traumatic events and cause differences in the expression of post-traumatic distress at either end of the life span [136, 137]. 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 [138]. The proposed criteria for children and adolescents have incorporated developmental manifestations of PTSD criteria for adults [139]. Scheeringa et al. [140] have previously reported on the inadequacy of DSM-IV PTSD criteria to adequately characterise post-traumatic symptoms with regard to infants and children less than four years old. They suggest that criteria anchored in observable behaviours should replace reports of subjective experiences or behaviours. Based on these observations, and others, a developmental pre-school PTSD subtype has been proposed for DSM-5 [139].

In contrast to an expanding literature for children and adolescents, PTSD among the elderly has received very little attention. Here too a developmental approach must address age-specific psychosocial, behavioural and neurobiological factors that mediate and moderate trauma-related symptom expression and clinical course [136]. PTSD among the elderly is often expressed within a context of negative health perceptions, primary care utilisation and suicidal ideation [141]. Traumatic events may result in different responses if they occurred many decades ago rather than recently. Differences in developmental and physiological factors in effect either at the time of the trauma or at the present time may influence the clinical expression of PTSD and post-traumatic distress. This has not been examined in research carried out thus far.

To summarise, a comprehensive, longitudinal developmental approach is needed to explicate how traumatic memories are differentially encoded, stored
and retrieved by both immature and ageing individuals, how such differences affect the clinical expressions of PTSD, and what implications this has for treatment.

CONCLUSION

1. Empirical evidence regarding the PTSD DSM-IV diagnostic criteria suggest that: (i) Criterion A1 has proved ambiguous with respect to where/how to draw the line between traumatic and nontraumatic events; (ii) Criterion A2 has failed to demonstrate diagnostic utility; and (iii) confirmatory factor analysis strongly suggests that the latent structure of PTSD is best characterised by a four-factor model.

2. Proposed provisional criteria for DSM-5 (which may change after this review is published) are: (i) retain Criterion A1 but attempt to remove some of its ambiguity; (ii) eliminate Criterion A2; and (iii) propose a four-factor PTSD model, consisting of reexperiencing symptoms, avoidance behavior, negative alterations in cognitions and mood, and alterations in arousal and reactivity. In addition to DSM-IV PTSD symptoms consistent with a fear-conditioning model, the proposed new criteria are consistent with evidence that traumatic exposure may also be followed by a variety of non-fear-based anxiety symptoms such as dysphoric anhedonic symptoms, aggressive/externalising symptoms, guilt/shame symptoms, dissociative symptoms and negative appraisals about oneself and the future.

3. Proposed DSM-5 symptoms for ASD have eliminated the requirement that an individual must exhibit dissociative symptoms in order to meet diagnostic criteria. Although it is recognised that some acutely traumatised individuals may exhibit peritraumatic dissociation, it is proposed that ASD criteria may also be met by the presence of reexperiencing, avoidance and arousal symptoms without the presence of dissociative symptoms.

4. An ASD/PTSD subtype of AD has been proposed for DSM-5. This should provide a diagnostic niche for severely distressed individuals (i) who have been exposed to a traumatic event but do not meet full PTSD diagnostic criteria and (ii) who have been exposed to a very upsetting nontraumatic event but who exhibit the full range of PTSD symptoms.

5. It has been proposed for DSM-5 that PTSD, ASD, AD and dissociative disorders be clustered into a unique diagnostic category or subcategory (as part of Anxiety Disorders) for stress/trauma-induced disorders. Other candidate disorders currently under consideration are a dissociative subtype of PTSD, DESNOS/complex PTSD, prolonged/complicated grief and developmental trauma disorder. All were under consideration at the time this review was written.

6. There has been an effort in DSM-5 to make PTSD criteria developmentally and culturally sensitive.
After DSM-5 is finalised, we will all await empirical data and clinical experience to tell us whether the proposed new criteria are an improvement over DSM-IV. Whether they are or aren’t, they will certainly be grist for the mill of DSM-6.

REFERENCES


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