Post-traumatic Stress Disorder

Edited by Dan J. Stein,
Matthew J. Friedman and Carlos Blanco

Post-traumatic Stress Disorder

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Post-traumatic Stress Disorder

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Contents

Preface

Post-traumatic stress disorder (PTSD) is arguably the most controversial of all the psychiatric diagnoses. There are disagreements about the qualifying events that count as sufficiently traumatic to precipitate PTSD, disagreements about the nature of the typical symptoms that follow exposure to trauma, disagreements about how best to prevent and treat PTSD, and disagreements about what kind of compensation is owed to people with PTSD by society.

At the same time, there have been major advances in our understanding of many aspects of PTSD. The diagnostic classifications of both the World Health Organization (WHO) and the American Psychiatric Association (APA) include the same broad symptom categories (e.g. re-experiencing, avoidance/numbing and arousal) and emphasise that exposure to extremely stressful events can produce profound alterations in cognitions, emotions and behaviour that may persist for decades or a lifetime.

There is also a growing appreciation of the public health burden of PTSD. Trauma continues to be a pervasive aspect of life in the 21st century, in high-, middle- and low-income countries [1]. Furthermore, PTSD and other trauma-related disorders are highly prevalent and disabling, are often associated with other psychiatric and medical disorders, and lead to significant costs for society [2, 3].

We are gradually advancing our scientific understanding of how exposure to traumatic events can produce neurobiological and psychological alterations which, if untreated, may persist indefinitely [4]. Furthermore, although there is not complete consensus across different clinical guidelines [5], there is general agreement that cognitive behaviour therapy and certain medications are the most effective clinical approaches for PTSD.

Many challenges remain. Fundamental information on the psychobiology of PTSD must be translated into effective, evidence-based clinical interventions. The development and testing of additional evidence-based treatments, especially treatments that are culturally sensitive and effective in more traditional ethnocultural settings, is required [6]. A further challenge is to move beyond the traditional clinic to the public health arena, where the focus must shift to resilience, prevention and selective interventions for populations at risk following disasters or mass violence [7].

The World Psychiatric Association (WPA) Evidence & Experience series provides a useful opportunity to work towards an evidence-based and integrative approach to different psychiatric conditions. In this volume, expert clinicians and researchers from around the world rigorously synthesise the data on PTSD, and provide balanced and judicious approaches to the controversies and challenges noted above. The chapters cover many aspects of PTSD, ranging from work on epidemiology and nosology, through research on psychobiology, to work on pharmacotherapy, psychotherapy and community approaches to intervention. Commentaries on each chapter, again from authors around the globe, provide additional depth.

Taken together, this work documents the many advances in empirical work on PTSD, negotiates a middle path through the theoretical controversies and provides clinicians and policy-makers with a practical approach to clinical and community interventions. Given that the field has learned much in recent decades about the kinds of trauma that are typically associated with PTSD, about the natural course of symptoms in response to such traumas, about optimal ways to evaluate and measure such symptoms, and about the best pharmacotherapeutic, psychotherapeutic and community approaches to the prevention and management of PTSD, we believe that this volume is timely. We hope that it will be useful to a broad range of readers.

We thank the many individuals who contributed to this volume, particularly the chapter authors. We also thank Joan Marsh of Wiley-Blackwell, Helen Herrman and Mario Maj of the WPA, and Marianne Kastrup, for their guidance and support; their vision and enthusiasm were pivotal in ensuring the initiation and progress of the volume. We wish to dedicate it to those individuals who have shared their symptoms and histories with us, teaching us the clinical aspects of PTSD and providing inspiring models of courage and resilience in the face of immense adversity.

Dan J. Stein, Carlos Blanco, Matthew J. Friedman

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PTSD and Related Disorders

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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 neuro-circuitry 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 empiri-cally derive A1 events by detecting which of these six dimensions reliably predict PTSD B–F symptoms. Events characterised by such dimensions would be desig-nated 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 devel-opment 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 some-times 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].