Post-Traumatic Stress Disorder: Ethical and Legal Relevance to the Criminal Justice System

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Background
New coverage of the recent wars in Afghanistan and Iraq, and the ensuing public education campaigns by the Department of Veterans Affairs and private veterans advocacy groups combine to call the public's attention to the many potential mental health problems associated with traumatic event exposure. Indeed, since 2001, Operation Iraqi Freedom (OIF) and Operation Enduring Freedom (OEF) combat and peacekeeping missions have been characterized by high levels of exposure to acts of extreme violence, with often gruesome effects. Less publically discussed is the fact that a surprisingly large number of United States civilians also report exposure to traumatic events, such as severe interpersonal violence, natural disasters, and serious automobile accidents. In fact, approximately 70% of randomly sampled respondents indicated that they have experienced an incident characterized by significant perceived life threat at some point during their lives.

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PTSD and its associated impairment do not always present in the same manner across those with the disorder. This wide variability in presentation can create some ethical and legal issues, of which criminal justice system members should be aware. Further, the continual evolution and, at times, unclear definition of the disorder, as well as variability in presentation, can lead to conflicting testimony in legal processes (see Mark Hamner in this issue) and civil lawsuits (see Bethany Wangelin and Peter Tuerk in this issue).

Description of the Disorder

Shell shock, battle fatigue, combat neurosis, and battered wives syndrome are well-known lay descriptors of negative emotional responses to trauma. The first edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-I) categorized such reactions as “Gross Stress Reaction” and, in the DSM-II, as “Transitory Situational Disturbance.” DSM-III was the first to refer to these symptoms as Posttraumatic Stress Disorder, and required that the traumatic event be “outside the range of usual human experience.” Subsequent research on frequency of traumatic event exposure and posttraumatic stress reactions resulted in several changes in the conceptualization of PTSD in the DSM-IV. First, the definition of a stressor thought to cause negative reactions was altered in light of the general population’s frequent exposure to traumatic events. Instead of requiring an unusual or rare experience, DSM-IV required that the initial stressor include: (1) confrontation with an event or events that involve actual or threatened death or serious injury, or a threat to the physical integrity of self or others; and (2) a response involving intense fear, helplessness, or horror. The definition also included a caveat for children that allows for reactions involving disorganized or agitated behavior. Such definitional changes had several important implications for the diagnosis. Specifically, the DSM-IV allowed for inclusion of non-rare experiences, such as severe reactions to minor traumas, as well as indirect experiences of trauma (a child witnessing parental violence). Further, this revised definition placed greater emphasis on an individual’s perception of threat at the time of the trauma.

The evolution of the PTSD diagnosis continues with the DSM-V. Major changes from the DSM-IV to the DSM-V include the elimination of Criterion A2, which states that individuals exposed to a traumatic event should experience an intense subjective emotional response such as fear, horror, or helplessness; the use of a four factor, or symptom category, model instead of the previous three factor model; and the addition of new symptoms to two of the newly renamed factors. Additionally, PTSD is no longer considered an anxiety disorder, but rather a trauma and stressor-related disorder. The four symptom categories of the DSM-V PTSD diagnosis are re-experiencing, avoidance, negative alterations in cognitions and mood, and alterations in arousal and reactivity.

In order to meet the re-experiencing criterion, one or more of the following re-experiencing symptoms is/are required: spontaneous or cued recurrent, involuntary, or distressing memories of the traumatic event(s); recurrent distressing dreams in which the content and/or affect of the dream is related to the event(s); dissociative reactions (e.g., flashbacks) in which the individual feels or acts as if the traumatic event(s) were recurring (such reactions may occur on a continuum, with the most extreme expression being a complete loss of awareness of present surroundings); intense or prolonged psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event(s); or a marked physiological reaction to reminders of the event(s). One of the following avoidance symptoms is required: efforts to avoid thoughts, feelings, or con-
versations related to the event, and efforts to avoid people, places, or activities that are reminders of the event. Three or more of the following negative alterations in cognitions and mood that are associated to the traumatic event(s) are required: inability to recall an important aspect of the event, persistent and exaggerated negative expectations about one's self, others, or the world, persistent distorted blame of self or others about the cause or consequences of the traumatic event (new symptom), pervasive negative emotional state (i.e., fear, horror, guilt, anger, or shame [new symptom]), decreased interest in previously enjoyed activities, feeling detached or estranged from others, or persistent inability to experience positive emotions (i.e., unable to have loving feelings, psychic numbing). Three or more of the following symptoms are required from the alterations in arousal and reactivity category: irritability, anger, or aggressive behavior, reckless or self-destructive behavior (new symptom), difficulty falling or staying asleep, difficulty concentrating, and an exaggerated startled response. Criteria for post-traumatic responses in children are somewhat different from criteria for adults.13

**Associated Features**

Other features associated with PTSD should be considered by criminal justice system personnel, as they may play a role in its presentation (i.e., in the behaviors of the person with the disorder). In addition to specific PTSD criteria, individuals with the disorder may also report difficulties in interpersonal relationships, problems with modulation of affect, "survivor guilt," self-destructive and impulsive behaviors, dissociative symptoms, somatic complaints, feelings of shame, despair, and hopelessness, and social withdrawal. Further, PTSD is associated with higher rates of panic disorder, agoraphobia, generalized anxiety disorder, obsessive-compulsive disorder, separation anxiety disorder, social phobia, specific phobia, alcohol abuse and dependence, and, most frequently, major depressive disorder.14 The temporal relationship of onset among these disorders is unclear, however. That is, traumatic events may increase the risk for multiple types of mental health problems; developing PTSD may create a vulnerability to other forms of psychological difficulties; and/or the presence of other psychopathology may create a vulnerability to PTSD.

**Epidemiology**

Overall, the population prevalence estimate of past-year PTSD is 3.5%, and estimates of lifetime PTSD range from 6.8% to 12.2%.15 PTSD prevalence estimates vary somewhat depending on the population sampled, methods of PTSD and trauma assessment used, current diagnostic definitions, and prevalence study methodology. Almost three decades ago, John Helzer and associates16 utilized data from the Epidemiologic Catchment Area Survey and the Diagnostic Interview Schedule17 and estimated the prevalence of lifetime PTSD to be 1% in the total population. This study, however, did not employ sensitive methodology for PTSD detection. Breslau, Davis, Andreski, and Peterson18 used a version of the DIS that was revised to be more sensitive to traumatic stress and found lifetime prevalence in the general population to be 9%. The lifetime prevalence rate among individuals exposed to a traumatic event was estimated to be 23%, which seems high compared to most other research. More recent research by Kessler's group, using state of the art methodology in the form of the National Comorbidity Studies, puts the lifetime rate between 6.8% and 7.8%.19

Women typically report more symptoms of PTSD than men,20 and the primary index events differ between men and women. Specifically, combat and witnessed violence are the most common precipitating events for men, while sexual and physical assaults are the most common precipitating events for women.21 The type of event experienced appears to influence risk of developing PTSD. Overall, sexual assault, physical assault, and motor vehicle accidents appear to yield the highest rates of PTSD.22 Other risk factors include being of non-Caucasian ethnicity,23 personal or familial history of psychopathology,24 and younger age.25

Given that the United States is currently at war, a focus on combat experience may be particularly justified. The National Vietnam Veterans Readjustment Study (NVVRS) revealed a PTSD prevalence of 9–15%, with an additional 8–11% of veterans in that sample suffering from sub-clinical PTSD.26 Further, veterans had significantly higher lifetime rates of major depression, dysthymia, obsessive-compulsive disorder, alcohol abuse or dependence, and antisocial personality disorder than the civilian control group. More recently, Karen Seal et al.27 conducted a population survey of 103,788 veterans active from 2001–2005 and found that over one-fourth received an MH diagnosis, with more than half of these receiving two or more diagnoses, underscoring the high comorbidity in this population. Additional studies demonstrate high rates of comorbid substance abuse, anxiety, mood, and personality disorders in combat veteran samples28 as well as severely impaired social and occupational functioning.29 Considering the current conflicts, Charles Hoge and colleagues demonstrated that OIF/OEF veterans suffer from PTSD, Major Depressive Disorder (MDD),
or Generalized Anxiety Disorder (GAD) at rates in excess of 15%.\textsuperscript{30}

**Etiology**

Theories proposed to explain reactions to exceptionally stressful or traumatic events draw from both psychological and biological realms. Learning theorists\textsuperscript{31} posit that through classical conditioning, salient stimuli or cues (conditioned stimuli) present during the traumatic event are associated with dangerous stimuli (unconditioned stimuli; e.g., rape, combat) and result in the conditioned response of fear, avoidance, and other negative affect and behavioral states. For example, the sound of gunshots, shrapnel wounds, and seeing friends killed by roadside improvised explosive devices are unconditioned stimuli that initially elicit feelings of fear and terror. Stimuli that resemble these things, such as fireworks, a fresh dirt mound by the side of the road, or even driving or the smell of diesel fuel may then elicit the PTSD fear response. Other contigous stimuli (e.g., people, places, time of day, smells, odors, tastes) also become able to elicit the fear response. In attempts to obviate the negative affect associated with the traumatic event, individuals often begin avoiding places, people, or situations that remind them of the trauma and that elicit the fear response. Thus, a veteran may not watch war movies, go near military bases, or be involved in any activities that include loud noises (e.g., fireworks); a rape victim may stop using public transportation or leaving her house after dark. Escaping, and subsequently avoiding, reminders of the trauma results in a reduction of anxiety, which reinforces avoidance behaviors.

In addition to learning theory, information processing theory highlights the role of cognitive appraisal and the meaning of the event for the individual who develops PTSD. Research findings support the notion that the perception of threat (i.e., worrying that a situation is dangerous when it probably is not) is a better predictor of subsequent PTSD symptoms than is the actual threat involved in the traumatic experience.\textsuperscript{32}

This theory proposes that individuals who experience trauma develop certain patterns of thinking and feeling about the world called fear structures that predispose one to perceive danger in situations that may pose only a low likelihood of danger. These fear structures consist of memory information related to stimuli associated with the traumatic event (e.g., the rapist was a tall man driving a green van), responses to the trauma (e.g., feelings, thoughts, behaviors), and the meaning of the trauma (e.g., how the individual perceives the trauma to have affected her life).\textsuperscript{33} Many stimuli that are not inherently dangerous (e.g., loud noises) are nonetheless incorporated into the fear structure.

When an individual is confronted with stimuli that are in some way associated with the trauma (dangerous or not), the fear network is activated. Due to avoidance behaviors, which include avoiding thoughts of the trauma, little opportunity is available for exposure to corrective information to modify the fear structure. Obviously, this theory shares several aspects of learning theory.

Another information processing theory holds that traumatized individuals sometimes develop problematic mental representations or belief systems, called schema. Schema are defined as ways of thinking about oneself, others, and the world.\textsuperscript{34} These schema influence the way people think and feel, as well as how they respond to stimuli in their environment. Lisa McCann et al.\textsuperscript{35} identified five schema that are vulnerable to distortion by traumatic events, including safety, trust, intimacy, power, and esteem. Patrice Resick and Monica Schnicke\textsuperscript{36} stated that the common responses following trauma are related to difficulties incorporating trauma experiences into existing belief systems. When individuals experience or encounter a schema-incongruent event, one that does not fit with their previous conceptualizations of the world, the experience and its accompanying emotions may be overwhelming.\textsuperscript{37} At this point, the individual must either alter the information to fit the schema (assimilation) or alter the schema to fit the information (accommodation). For example, if a woman's rape schema includes only the notion of a woman being raped by a stranger, and she is subsequently raped by an acquaintance, she may assimilate, or alter, the information by convincing herself that she was not raped. She might label the event as a miscommunication, or perhaps think that she did or said something that the offender misconstrued. By contrast, if the rape victim were to accommodate the information, she might change her schema to incorporate the possibility that someone known and trusted can be dangerous and can perpetrate an assault. Obviously, this may affect the nature and content of her testimony.

Resick and Schnicke\textsuperscript{38} noted another phenomenon in their work with rape victims: over-accommodation. This involves an extreme distortion in schema. For example, instead of changing one's schema to include the possibility that some trusted individuals can be dangerous, the victim of acquaintance rape may change her schema to suggest that all men are dangerous and cannot be trusted. Over-accommodation may result in dichotomized thought processes and restrict the cognitive flexibility with which individuals interpret and evaluate future information.

Biological etiological theories complement the aforementioned psychological theories. One area
of interest involves cortisol responses in those who develop PTSD. Rachel Yehuda and Joseph LeDoux\textsuperscript{29} reviewed biological parameters of PTSD in relation to clinical characteristics and found that increased release of adrenocorticotrophic hormone (ACTH) from the pituitary, cortisol from the adrenal cortex, and catecholamines from the adrenal medulla are all implicated in the disorder, in conjunction with the inability to return to physiological homeostasis after immediate exposure to a stressor. In general, increased release of these hormones following extreme stress events is a normal response; however, the problem occurs when

**Treatment**

Over 30 years of research with trauma-exposed individuals has led to effective treatments for PTSD.\textsuperscript{44} The Cochrane Reports and the VA/DoD Clinical Practice Guideline for Management of Posttraumatic Stress recognize trauma-focused cognitive behavioral treatments (CBTs), such as Prolonged Exposure (PE) or Cognitive Processing Therapy (CPT), as the gold-standard treatments for individuals with PTSD. These CBTs are based on the aforementioned learning and cognitive models of psychopathology. Common components of CBT for PTSD include cognitive restructuring, which targets negative core beliefs associated with PTSD (e.g., “The world is a dangerous place”; “I am incompetent.”); \textit{in vivo} exposure, which involves confrontation of feared situations that elicit distressing reminders of the trauma; and imaginal exposure, which involves repeated and prolonged “revisiting” of the trauma memory by providing a detailed, oral account. Research suggests that both cognitive restructuring and exposure techniques are effective at reducing PTSD symptoms.

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Additionally, previous studies examining chronic PTSD in combat veterans noticed “sustained physiological hyperarousal,” and increased catecholamine levels compared to control groups who experienced similar stressors.\textsuperscript{41} Less has been discussed regarding genetic predisposition for PTSD because many of the genetic risk factors have been identified retrospectively by comparing individuals with and without PTSD. However, while some studies have shown family history of psychopathology to be a notable risk factor for PTSD,\textsuperscript{42} others suggest that, compared to the victim’s perceived life threat and perceived support, a family history of psychopathology is a much weaker predictor for the development of PTSD symptoms.\textsuperscript{43} It is, again, important to note that the incongruence may be due to varying definitions of PTSD in the different versions of the DSM.

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ment. Further, even when CBT leads to symptom reductions, patients often fail to report significant improvements in psychosocial functioning. Thus, although these treatments alleviate emotional distress, they may not target the areas of most concern to patients (i.e., employment, relationship, and social functioning).

In addition to psychotherapy, several recent studies have examined pharmacotherapy as a potentially effective treatment option for PTSD. Dan Stein and colleagues conducted a meta-analysis of 30 pharmacotherapy studies comparing SSRIs (Selective Serotonin Reuptake Inhibitors) and SNRIs (Serotonin-Noradrenaline Reuptake Inhibitors) to MAOIs (Monoamine Oxidase Inhibitors), TCAs (Tricyclic Antidepressants), and placebo. They found that most studies examining SSRIs saw a significant reduction in symptom severity over the short term; however, the research suggests prescribers include the medication over a 12-month period to prevent relapse. Based on the robust findings in support of SSRIs, experts in this field agree that this class of medication be the first choice in pharmacotherapy for PTSD.

Benzodiazepines are also popular in the treatment of PTSD, despite very little empirical evidence to support their effectiveness. Experts suggest caution when prescribing this medication, especially because long-term use of benzodiazepines likely causes significant cognitive impairment. While research has shown that a combination of SSRIs and psychotherapy are effective in symptom reduction when one form of therapy is not enough, the use of benzodiazepines while undergoing psychotherapy can be detrimental to recovery.

**Conclusion**

Despite its evolving definition and diagnostic criteria, PTSD has been recognized in some form as a human response to severe stress events for centuries. It is a disorder affecting significant numbers of individuals, both military and civilian, across genders, socioeconomic status, and race. While the most significant predictor of the disorder is the subjective experience of a perception of threat to life, the actual percentage of the population exposed to traumatic events is rather high and most do not develop the full disorder. This is not to say that these exposed individuals are unaffected, however. Indeed, many do not meet full diagnostic threshold criteria, but nonetheless experience significant distress, albeit at a level less than others meeting full criteria. Thus, this “line” of diagnosis is ultimately a subjective decision by committee, rather than a determination of suffering and impairment vs. no suffering and impairment. The fifth edition of the Diagnostic and Statistical Manual of Mental Health Disorders is attempting to narrow the definition of PTSD and enhance its reliability. While there are effective treatments for the disorder, even those successfully treated often experience lingering, albeit dramatically reduced levels of, symptomatic intensity.

**References**


12. See APA, supra note 11.


19. See Kessler et al., supra note 14.


21. See Breslau (2002), supra note 20; see Breslau (2009), supra note 2; Kessler et al., supra note 21; Norris (1992), supra note 20.


25. See APA, supra note 11.

26. See APA, supra note 11.

27. See APA, supra note 11.


30. See Hoge et al. (2004), supra note 1; C. W. Hoge, J. L. Auchterlonie, and C. S. Ellis, "Mental Health Problems, Use of Mental Health Services, and Attrition from Military Service after Returning from Deployment to Iraq or Afghanistan," Journal of the American Medical Association 295, no. 9 (2006): 1023-1032; Kang et al. (2003), supra note 3; Seal et al. (2007), supra note 27.


35. See McCann et al. (1988), supra note 34.
36. See Resick and Schnicke, supra note 34.

37. Id.

38. Id.


40. Id.


48. See Bradley et al. (2005), supra note 44; Cloitre (2009), supra note 44; Powers et al. (2010), supra note 44.


52. Id.

53. Id.


55. See Stein et al., supra note 51.


57. See Foa, Davidson, and Frances (1999), supra note 54; Ballenger et al., (2002 and 2004), supra notes 54 and 11.

58. See Stein et al., supra note 51.


61. See Stein et al., supra note 51.