# Ethnocultural Aspects of Posttraumatic Stress Disorder

## Issues, Research, and Clinical Applications

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# 1

# POSTTRAUMATIC STRESS DISORDER: AN OVERVIEW OF THE CONCEPT

MATTHEW J. FRIEDMAN and ANTHONY J. MARSELLA

> He tried to make love to her. . . . until he was defeated by sadness, or shame, and pushed her away. . . . But darkness was not good; he needed the rectangle of light from the street, because without it he felt trapped again in the abyss of the timeless ninety centimeters of his cell, fermenting in his own excrement, delirious . . . he knew that he was coming apart, as he had so often before, and he gave up the struggle, releasing his last hold on the present, letting himself plunge down the endless precipice. He felt the crusted straps on his ankles and wrists, the brutal charge, the torn tendons, the insulting voices demanding names, the unforgettable screams of Ana, tortured beside him, and of the others, hanging by their arms in the courtyard.
>
> From "Our Secret," by Isabelle Allende

> When he came home [from Vietnam] though, Henry was very different, and I'll say this: the change was no good. You could hardly expect him to change for the better, I know. But he was quiet, so quiet, and never comfortable sitting still anywhere but always up and moving around. . . . now you couldn't get him to laugh, or when he did it was more the sound of a man choking, a sound that stopped up the throats of other people around him. They got to leaving him alone most of the time, and I didn't blame them. It was a fact: Henry was jumpy and mean.
>
> From "The Red Convertible," by Louise Erdrich

## THE PTSD CONCEPT

Exposure to trauma has been a risk of the human experience throughout human history. Emotional reactions to extreme stress have been noted by historians and literary authors for 4,000 years (e.g., Figley, 1993; Veith, 1965). Homer's Ulysses (and probably Achilles) and Shakespeare's Henry

IV experienced profound posttraumatic stress reactions after exposure to war. Charles Dickens also experienced a traumatic stress reaction as a result of a major train wreck (Figley, 1993; Shay, 1991; Trimble, 1981).

However, it has only been since 1980, when the American Psychiatric Association's *DSM–III* (American Psychiatric Association; [APA] 1980) included the category of posttraumatic stress disorder (PTSD) in its official diagnostic and statistical manual that mental health clinicians had a suitable diagnostic option for the symptoms characteristic of survivors of traumatic and catastrophic stress. PTSD has provided a very new and useful conceptual and therapeutic approach to the psychological impact of trauma. Studies on adult victims of crime, war veterans, natural disaster survivors, and traumatized children (see Davidson, 1993) have shown that the revised (*DSM–III–R*) PTSD symptom clusters show good internal cohesiveness as a group.

> On the basis of multivariate and frequency studies in four different United States populations, the *DSM–III–R* symptom criteria for PTSD appear to bear out the constructs, traditions, and clinical wisdom embodied in the description. (Davidson, 1993, p. 151)

The same can be said for *DSM–IV* diagnostic criteria for PTSD because these same symptom clusters have been essentially preserved.

## Ethnocultural Aspects of PTSD

Most research and clinical experience validating PTSD as a diagnostic category was carried out in Western industrialized nations. Although there are many evocative literary accounts depicting posttraumatic intrusion and avoidant/numbing and arousal symptoms in non-Western protagonists, such as political torture survivors from South America (Allende, 1991) or a Chippewa Vietnam veteran (Erdrich, 1984), there has been little methodologically sound cross-cultural research on PTSD among people from non-Western ethnocultural backgrounds (e.g., Marsella, Friedman, Gerrity, & Scurfield, this volume; Marsella, Friedman, & Spain, 1993).

Furthermore, Chakraborty (1991), has actually objected to applying the PTSD diagnosis to people from non-Western traditional societies. He argues that PTSD is a Western culture-bound syndrome that should be restricted to Euro-American individuals. We believe that Chakraborty's conclusion may be too extreme, and that there are many aspects of posttraumatic stress that are universal even in the face of what appear to be ethnocultural variations. Because most studies of non-Western cohorts lack the requisite cross-cultural methodological rigor (Marsella, Friedman, & Spain, 1993), it is unclear whether PTSD per se is the most appropriate diagnostic construct to apply universally. But this does not mean that the construct lacks applicability or meaning across cultures.

The authors have argued elsewhere (a) that we must remain open to the possibility that ethnocultural differences in the expression of traumatic stress may not strictly conform to *DSM–IV* diagnostic criteria; (b) that ethnocultural tendencies may alter clinical phenomenology to such an extent that highly traumatized cohorts will exhibit surprisingly low rates of PTSD; and (c) that following traumatic exposure, members of some ethnocultural groups may exhibit a traumatic stress syndrome that overlaps with PTSD but features symptoms idiosyncratic to individuals from specific ethnocultural traditions (Friedman & Jaranson, 1994; Marsella, Friedman, & Spain, 1993).

It is important to note that because ethnocultural differences among people are tied to variations in the social construction of reality, the perception of what constitutes a traumatic exprience, as well as the individual and social response to it, may vary considerably. For example, among followers of religions in which individual responsibility for acts and their consequences is limited, the perceptions, expressions, and treatment of exposure to trauma may differ from individuals of religions in which individual responsibility is emphasized.

Diagnostic precision and cross-cultural methodology notwithstanding, a number of recent studies have demonstrated profound posttraumatic psychiatric sequelae among non-Westernized individuals such as Southeast Asian refugees (Kinzie, 1993); Sri Lankans exposed to civil war (Somasundaram, 1993); civilian survivors of the war in Afghanistan (Wardak, 1993); cyclone survivors in Fiji and Sri Lanka (Fairley, 1984; Patrick & Patrick, 1981); earthquake victims in Mexico, Ecuador, Japan, and China (Conyer, Amor, Medina-Mara, Caraveo, & De la Fuentes, 1987; Lima, et. al., 1989; McFarlane & Hua, 1993; Odaira, Iwadate, & Raphael, 1993); volcano survivors in Colombia (Lima, Pai, Santacruz, Lozano, & Luna, 1987); and political torture detainees in South Africa (Simpson, 1993).

We will set the stage for the following chapters by reviewing PTSD with respect to the following factors: epidemiology, diagnostic criteria, theoretical issues, diagnostic issues, research issues, and clinical issues. We will discuss pretraumatic, traumatic, and posttraumatic risk factors for developing PTSD. Finally, we will mention a few cultural factors that might influence vulnerability to or resistance against the development of PTSD following exposure to extreme stress.

## OVERVIEW OF THE RESEARCH LITERATURE

### Epidemiology

In both the *DSM–III* (p. 236) and *DSM–III–R* (p. 247) formulations of PTSD, the risk of exposure to a traumatic event was characterized as

"outside the range of usual human experience." A number of epidemiologic surveys have shown that exposure to trauma with subsequent development of PTSD was not uncommon. Among civilian residents of a large American city (Breslau, Davis, Andreski, & Peterson, 1991), and among veterans of the Vietnam War (Kulka, et al., 1990), lifetime prevalence rates of PTSD were approximately 9% and 30% respectively. One attempt to determine PTSD in the general population (Helzer, Robins, & McEvoy, 1987) of the St. Louis portion of the Epidemiologic Catchment Area Survey estimated a prevalence rate of 1 to 2%. Although there are instrumental and methodological reasons for rejecting this projection as a serious underestimation (Keane & Penk, 1988), even if we accept the Helzer et al. study's 1–2% figure, it yields an estimate of 2.4 to 4.8 million cases of PTSD in the United States alone. As noted by Keane (1990), with high rates of rape, criminal victimization, natural disaster, technological disaster, and war contributing to the development of PTSD, it is likely that the true prevalence rate of PTSD may substantially exceed the Helzer, et al. (1987) estimate. As a result, the latest revision of PTSD diagnostic criteria, the *DSM–IV*, no longer describes trauma as an event that is "outside the range of usual human experience." A detailed review of the epidemiology of PTSD and related stress disorders is presented in the chapter by DeGirolamo and McFarlane in Chapter 2 of the current volume.

### Current Diagnostic Criteria

The appendix to this chapter summarizes the *DSM–IV* diagnostic criteria (American Psychiatric Association, 1994). The *stressor* criterion, or Criterion A, specifies that a person has been exposed to a catastrophic event such as war, torture, rape, a natural disaster, or an industrial accident, for example. During such exposure the person has been confronted with actual or threatened death or injury, or a threat to the physical integrity of himself or herself, or others. Green (1993, p. 138) has proposed eight "generic" dimensions of trauma:

1. Threat to life and limb;
2. Severe physical harm or injury;
3. Receipt of intentional injury/harm;
4. Exposure to the grotesque;
5. Violent/sudden loss of a loved one;
6. Witnessing or learning of violence to a loved one;
7. Learning of exposure to a noxious agent;
8. Causing death or severe harm to another.

In addition to exposure per se, many investigators now believe it is important to emphasize that during traumatic exposure, an individual's subjective response was marked by intense fear, helplessness, or horror. As is

shown in the appendix, there is an emphasis on the subjective response to extreme stress, which has resulted in an expansion of the stressor criterion in *DSM–IV*. Criterion A–1 states that an individual has "experienced, witnessed, or been confronted by a catastrophic event (as in *DSM–III–R*)," whereas criterion A–2 is a new addition that addresses the person's emotional response to such traumatic exposure. Furthermore, Foa, Zenberg, & Rothbaum, (1992) have recently proposed that to qualify as traumatic, a stressor must not only be perceived as a potential threat to survival but must also be experienced as uncontrollable or unpredictable. Expansion of the stressor criterion to include such subjective components opens the door for consideration of psychological, experiential, and ethnocultural factors that mediate emotional responses to stressful events. Such factors also include the individual's subjective response to the historical, social, and political context in which stressful events occur.

The presence of an etiological factor in a diagnostic category, as with traumatic stress in PTSD, is relatively uncommon in the *DSM–III–R* and *DSM–IV*, and there is some controversy about where to draw the line between traumatic events and painful stressors that constitute the common vicissitudes of life such as divorce, failure, rejection, serious illness, and so on. Furthermore, the subjective appraisal of an historical event may determine whether an individual perceives a stressful episode as traumatic or merely difficult. After all, the subjective experience of trauma is a process by which information about an historical event is filtered through cognitive and emotional psychological processes.

Because of individual differences in this appraisal process, people appear to have varying trauma thresholds; some are more protected and some more vulnerable to developing clinical symptoms after exposure to extremely stressful situations. In this regard, there are some data suggesting that among people exposed to a traumatic stressor, those with a personal or family history of psychiatric illness are more likely to develop PTSD (Breslau, et al., 1991; Kulka, et al., 1990).

Criterion B, the *intrusive recollection* criterion, includes symptoms that are perhaps the most distinctive and readily identifiable symptoms of PTSD. For individuals with PTSD, the traumatic event remains—sometimes for decades or a lifetime—a dominating psychological experience that retains its power to evoke panic, terror, dread, grief, or despair in daytime fantasies, nightmares, and psychotic reenactments known as *PTSD flashbacks*. The only B-criterion change in the *DSM–IV* is that Symptom D–6 in *DSM–III–R* has now been moved to B–5.

The opening passage by Allende evokes an intrusive recollection about incarceration that escalates rapidly into a flashback about torture. Symptoms B–4 and B–5 reflect well-known clinical observations that traumamimetic stimuli can trigger recollections of the original event and have the power to evoke mental images, emotional responses, and physiological

reactions associated with the trauma. Because of this, researchers can reproduce PTSD symptoms in the laboratory by exposing affected individuals to auditory or visual traumamimetic stimuli (Blanchard, Kolb, Pallmeyer, & Gerardi, 1982; Malloy, Fairbank, & Keaner, 1983; Pitman, Orr, Forgue, de Jong, & Clairborn, 1987). Such symptoms can also be provoked pharmacologically by the adrenergic alpha-2 receptor antagonist, yohimbine, that acts directly upon the locus coeruleus in the brain (Southwick, et al., 1992).

The *avoidant/numbing* criterion, Criterion C, consists of symptoms reflecting behavioral, cognitive, or emotional strategies by which PTSD patients attempt to reduce the likelihood that they either will expose themselves to traumamimetic stimuli or, if exposed, will minimize the intensity of their psychological response. Behavioral strategies include avoiding any situation in which patients perceive a risk of confronting such stimuli. In its most extreme manifestation, avoidant behavior may superficially resemble agoraphobia because the person suffering from PTSD is afraid to leave the house for fear of confronting reminders of the traumatic event(s). Dissociation and psychogenic amnesia are included among avoidant/numbing symptoms by which individuals cut off the conscious experience of trauma-based memories and feelings.

Finally, because individuals with PTSD cannot tolerate strong emotions, especially those associated with the traumatic experience, they separate the cognitive from the emotional aspects of psychological experience and perceive only the former. Robert Lifton, observing this phenomenon in Hiroshima atom bomb survivors and later in Vietnam combat veterans, labeled it *psychic numbing* (1976; 1979). Psychic numbing is an emotional anesthesia that makes it extremely difficult for people with PTSD to participate in meaningful interpersonal relationships. There has been no change between *DSM–III–R* and *DSM–IV* for C symptoms.

Symptoms included in the D or *hyperarousal* criterion most closely resemble those in panic and generalized anxiety disorder. Whereas symptoms such as insomnia and irritability are generic anxiety symptoms, hypervigilance and startle are more unique. The hypervigilance in PTSD may sometimes become so intense as to appear like frank paranoia. The startle response has a unique neurobiological substrate (Davis, 1990) and may actually be the most pathognomonic PTSD symptom (Ornitz & Pynoos, 1989). D symptoms have remained unchanged in *DSM–IV*, except for that *DSM–III–R* D–6 has become *DSM–IV* B–5.

The E or *duration* criterion specifies how long symptoms must persist in order to qualify for the chronic or delayed PTSD diagnosis. In *DSM–III* the mandatory duration was 6 months. In *DSM–III–R* the duration was shortened to 1 month. This has remained the criterion in *DSM–IV*. The "F" or *functional impairment* criterion is a new addition to the PTSD diagnosis in *DSM–IV*. Also new are definitions of *acute* (duration of symp-

toms for less than 3 months) and *chronic* (duration of symptoms for greater than 3 months) PTSD.

### Theoretical Issues

PTSD differs from most other psychiatric disorders because there is a wealth of experimental literature from both psychology and neurobiology that appears pertinent to the clinical syndrome. Indeed, unlike most other psychiatric disorders, a number of animal models have been proposed for PTSD including learned helplessness/inescapable stress (Seligman & Beagley, 1975; van der Kolk, Greenberg, Boyd, & Krystal, 1985); fear potentiated startle (Davis, 1990); kindling (Friedman, 1988; van der Kolk, 1987); and time dependent sensitization (Yehuda & Antelman, 1993).

Because the hallmark of PTSD is the response to a traumatic event, and because PTSD symptoms can be triggered by exposure to traumamimetic stimuli, no other psychiatric syndrome can be conceptualized in terms of classic learning theory as well as PTSD. Keane, Zimmering, and Caddell (1985) have proposed a two-factor model of PTSD that invokes the seminal work of Mowrer (1939). Foa, et al. (1992) proposed an unpredictable/uncontrollable stress model of PTSD that is a modern extrapolation from Masserman's (1943) paradigm used to produce experimental neurosis in animals.

Other significant theoretical issues in psychology that have spurred ongoing PTSD research are a reformulation of James' (1890) model of arousal-based behavior (Blaskovich, 1990) and a reinterpretation of Lang's (1977) information processing theory of fear and anxiety (Creamer, Burgess, & Pattison, 1992; Litz & Keane, 1989). Finally, research on the appraisal process by which individuals decide whether a stressful event presents a threat or a challenge (Lazarus & Folkman, 1984) is, in our opinion, extremely relevant to cognitive processing of posttraumatic reactions as shown by Fairbank, Hansen, and Fitterling (1991). A review of this literature, however, is beyond the scope of this chapter.

As with the psychological literature, there is a wealth of pertinent neurobiological literature starting with the classic works of Cannon (1929) and Selye (1956) on adrenergic and hypothalamic-pituitary-adrenocortical (HPA) responses to stress, which has been reinterpreted by Dienstbier (1989). More recent animal research has shown that exposure to stress can produce alterations in a least seven different neurobiologic systems: adrenergic, HPA, dopaminergic, GABA-benzodiazepine, opioid, serotonergic, and NMDA (see reviews by Bremner, Davis, Southwick, Krystal, & Charney, 1993; Charney, Deutch, Krystal, Southwick, & Davis, 1993). As noted earlier, Kardiner (1941) was the first to suggest that World War I veterans with war neurosis were suffering from a central nervous system disorder that he labeled a *physioneurosis*. It has also been suggested that the cardi-

ovascular and other medical abnormalities (e.g., neurocirculatory asthenia) seen in World War II veterans (Cohen, White, & Johnson, 1948) may have actually reflected the underlying pathophysiology of PTSD (Friedman, 1991).

Recent findings in patients with PTSD suggest that the disorder may be associated with stable neurobiological alterations in both the central and autonomic nervous systems. Patients with combat-related PTSD have been shown to exhibit physiologic hyperarousal manifested by cardiovascular hyperactivity (Blanchard, et al., 1982), increased sensitivity and augmentation of the acoustic-startle eyeblink reflex (Ornitz & Pynoos, 1989), a reducer pattern of auditory evoked cortical potentials (Paige, Reid, Allen, & Newton, 1990), and sleep abnormalities (Ross, Ball, Sullivan, & Caroff, 1989). Stable neurobiological abnormalities have also been detected in the central noradrenergic, HPA, and endogenous opioid systems. These studies are reviewed extensively elsewhere (Bremner, et al., 1993; Charney, et al., 1992; Friedman, 1991; Friedman, Charney, & Deutch, 1995).

A number of animal models have been invoked to account for the observed stability of PTSD symptoms over time (Bremner, Southwick, & Charney, 1991; Friedman, 1988; van der Kolk, 1987; Yehuda, & Antelman, 1993). Many of these models postulate that exposure to trauma produces significant alteration and sensitization of crucial brain mechanisms that are not only responsible for the persistence of PTSD symptoms but also for increased vulnerability to the impact of subsequent traumatic events.

## Diagnostic Issues

Since PTSD has been an official diagnosis, there has been much greater clinical sensitivity to the impact of trauma on other psychiatric disorders. For instance, recent research has shown that childhood sexual or physical abuse is the rule rather than the exception in borderline personality disorder, multiple personality disorder, and somatization disorder (Davidson, 1993; Herman, 1992). Other *DSM–IV* disorders in which prior exposure to extreme physical or psychological stress appears to be instrumental include brief reactive psychosis, dissociative fugue, dissociative amnesia, conversion disorder, depersonalization disorder, dream anxiety disorder, and antisocial personality disorder (Davidson, 1993). There is also emerging evidence to suggest that at least some cases of attention deficit hyperactivity disorder in children can be ascribed to exposure to trauma before the age of 3 (Perry, 1994). In other words, although PTSD may be the most unequivocal and most easily diagnosed consequence of traumatic exposure, there are a number of other psychiatric disorders that may be caused or exacerbated by extreme stress.

The most important diagnostic controversy in the trauma field revolves around the fact that the PTSD syndrome does not include those symptoms most often seen in victims of prolonged interpersonal violence such as childhood incest, repeated domestic violence, prolonged captivity, or protracted torture. A new syndrome, proposed by Herman (1992), has had several named: victimization disorder, complex PTSD, or disorder of extreme stress not otherwise specified (DESNOS). Herman has proposed an alternative diagnostic formulation that emphasizes excessive somatization, dissociation, changes in affect, pathological changes in relationships, pathological changes in identity, self-injurious or suicidal behavior, and revictimization. She has argued that diagnoses such as borderline personality disorder, multiple personality disorder, and somatization disorder can be more appropriately conceptualized and treated as manifestations of DESNOS. However, data from field trials on the prevalence of DESNOS conducted by the *DSM–IV* PTSD Task Force did not result in the inclusion of DESNOS in the *DSM–IV*. Clearly, additional studies are needed.

### Research Design Issues

According to King and King (1991), much of the PTSD research is questionable because of numerous flaws in research design and statistical analysis. In their detailed review of validity issues in research on Vietnam War veteran adjustment, King and King (1991) concluded that

> the full causal network of factors regarding Vietnam veteran status is yet to be understood; and the accumulated literature, literally hundreds of studies, is fraught with threats to validity. Although recognizing that the methodological sophistication of Vietnam veteran studies has certainly improved over the past two decades, we maintain that many discrepant findings and conclusions can be attributed to differences in methodological approaches and certain weaknesses in the logic on which causal inferences are based. (pp. 107–108)

King and King (1991) offer a careful and detailed analysis of issues related to Cook and Campbell's (1979) classic monograph on design and analysis issues in quasi-experimental research designs typical of field research strategies in which the rigors of laboratory experimentation cannot be met. King and King (1991) discuss statistical conclusion validity, internal validity, construct validity of cause and effect, and external validity issues. Furthermore, the problems noted by King and King (1991) are compounded when ethnocultural differences are the topic of research. This is because of the rigorous requirements of cross-cultural research designed to limit ethnocentric bias in such areas as sampling, clinical criteria, measurement, and data analysis (see Marsella, Friedman, & Spain, 1993).

## Clinical Issues

### *Comorbidity*

If an individual meets diagnostic criteria for PTSD, it is likely that he or she will meet *DSM–IV* criteria for one or more additional diagnoses (Davidson and Foa, 1993; Kulka, et al., 1990). Most often, these comorbid diagnoses include major affective disorders or personality disorders. There is a legitimate question whether the high rate of diagnostic comorbidity seen with PTSD diagnoses is an artifact of our current decision rules for making the PTSD diagnosis because there are no exclusionary criteria in the *DSM–IV*. For example, Yehuda et al., (1993) have shown that the major depressive disorder (MDD) that often accompanies PTSD is a very different biological abnormality than primary major depression. These results suggest that the MDD seen with PTSD is actually a part of the PTSD syndrome itself rather than a comorbid depressive disorder as it currently must be classified by *DSM–IV*. The same argument may apply to other comorbid diagnoses such as panic disorder, generalized anxiety disorder, and obsessive–compulsive disorder. At the present time, however, high rates of comorbidity complicate treatment decisions concerning patients with PTSD because the clinician must decide whether to treat the comorbid disorders concurrently or sequentially. This is especially true when PTSD is comorbid with alcoholism or substance abuse (Kofoed, Friedman, & Peck, 1993).

### *Longitudinal Course and Chronicity*

Longitudinal studies have shown that PTSD can persist for decades and sometimes for a lifetime. In their 20-year follow-up of World War II veterans, Archibald and Tuddenham (1965) found that veterans with PTSD ("war neurosis" [sic]) not only continued to exhibit such symptoms but, in many cases, exhibited a worsened clinical status over time. Studies of World War II American military veterans, prisoners of war, and Dutch resistance fighters have shown that PTSD was present and virulent after 40 years (see review by Schnurr, 1991).

Longitudinal outcomes are variable, however. Whereas some trauma survivors may become free of most or all PTSD symptoms previously exhibited, others may remain seriously affected (Blank, 1993). Patients with persistent PTSD may manifest a chronic mental disorder with a longitudinal course marked by remissions and relapses (Friedman & Rosenheck, in press). Unlike schizophrenia and affective disorders, however, the triggers for relapse in PTSD are more readily identifiable. Such relapses often appear to be precipitated by further exposure to traumatic stimuli and situations. For example, during the massive 1990–91 deployment of troops to the Persian Gulf, American print and broadcast media bombarded the

public with information about the war. It appears that such war-related stimuli may have precipitated or exacerbated PTSD symptoms in older veterans because there was a marked increase in the number of veterans seeking treatment for PTSD from VA facilities (Gelsomino, Hayman, & Walker, 1992; Wolfe, Brown, & Buscela, 1992).

Although PTSD is not inherently a persistent mental disorder, there are patients with PTSD who are severely and chronically incapacitated. Like people with schizophrenia, these patients are severely ill, their social functioning is markedly restricted, and they often rely heavily on public housing, community support, and public mental health services. For some patients, PTSD is severe enough to result in repeated hospitalizations over many years and to require ongoing outpatient treatment as a persistent mental disorder (Friedman & Rosenheck, in press).

## Treatment

Solomon, Gerrity, and Muff, (1992) reviewed recent treatment literature and showed that out of 255 published articles on treatment for PTSD only 11 can be considered satisfactory randomized therapeutic trials (see also chapter 3 by Gerrity & Solomon in this volume). These include five drug trials, four studies on behavioral treatment, one study on cognitive–behavioral therapy, and one study comparing psychodynamic therapy, behavioral therapy, and hypnotherapy. Results from drug trials were inconsistent, whereas findings with behavioral treatment were more encouraging.

The most dramatic clinical improvement was reported by Foa and colleagues (Foa, Rothbaum, Murdock, & Riggs, 1991), who demonstrated the efficacy of cognitive–behavioral treatment for rape victims with PTSD in a randomized clinical trial. In addition, there are numerous reports on the effectiveness of cognitive–behavioral, psychodynamic, pharmacotherapeutic, group, and other treatments for PTSD (Marmar, Foy, Kagan, & Pynoos, 1993; Williams & Sommer, in press). However, as noted previously (Solomon et al., 1992; Gerrity & Solomon, this volume) there are few published randomized clinical trials to back-up claims of therapeutic efficacy.

Herman (1992) asserts that most treatment approaches for PTSD consist of three stages. In Stage 1, the emphasis is on establishing a safe environment, stabilization, and building trust. In Stage 2, the patient tells the story of the trauma. The major therapeutic task at this stage is to explore, reexperience, and integrate the traumatic memories and feelings that continue to intrude on the present. Stage 3 is devoted to integration of the trauma, personality rehabilitation, development of coping skills, and reconnection to family and society.

There are particular problems in assessment and treatment of patients from non-Western backgrounds. Most of the writing on this subject covers treatment of refugees and is reviewed in detail elsewhere (Friedman & Jaranson, 1994; Kinzie, 1989; Marsella, Borneman, Ekblad, & Orley, 1994; Owan, 1985; Westermeyer, 1989). Some therapeutic issues that need to be considered with regard to refugees are that treatment is often avoided until the patient is severely ill, and has multiple somatic complaints, severe depression or dissociative and paranoid symptoms. Building trust and establishing a safe therapeutic environment may be especially difficult when the therapist is from a different cultural or racial background than the patient. Futhermore, language problems or the need to use interpreters may compound these difficulties. In addition, culturally-based willingness to accept different therapeutic formats (e.g., individual vs. group) may have a decisive impact on the therapeutic process. And of course, the cultural sensitivity and sophistication of the therapist is of paramount importance when discussing pertinent aspects of the trauma, such as those related to sexual matters or to death.

## Risk Factors

The original *DSM–III* formulation emphasized that exposure to the traumatic event itself was primarily responsible for the later development of PTSD. It rejected the psychodynamic concept of war neurosis that was predicated on pretraumatic vulnerability. It was asserted that whereas individual differences, with regard to vulnerability or resilience, might have a major impact in mediating the consequences of ordinary stress, they were of little value when it came to mediating the impact of traumatic stress. Support for this position came from numerous studies showing that the best predictor of developing PTSD was the intensity of the trauma itself rather than personal psychological factors. For example, higher levels of PTSD were repeatedly found among Vietnam veterans who had experienced the highest levels of exposure to extreme events in the war zone (Kulka, et al., 1990). A similar dose–response relationship between trauma intensity and PTSD prevalence rates has been found in physically injured sexual assault victims (Winfield, George, Swartz, & Blazer, 1990), children exposed to a school yard sniper attack (Pynoos, et al., 1987), survivors of the Mount St. Helens volcanic eruption (Shore, Tatum, & Vollmer, 1986), and Australian firefighters who battled a large bush fire (McFarlane, 1988).

Despite this strong relationship, exposure to trauma is not a sufficient condition for the later development of PTSD. Many people exposed to war, interpersonal violence, or other trauma never develop the full PTSD syndrome (Breslau et al., 1991; Kulka et al., 1990). This has prompted research on risk factors for PTSD. Such research has focused on pretraumatic, traumatic, and posttraumatic factors.

Summarizing a number of studies, Davidson (1993) lists the following as risk factors for PTSD: positive history of psychiatric illness among first-degree relatives, early sexual or other childhood trauma, parental poverty, behavior disorder in childhood or adolescence, separation or divorce of parents before 10 years of age, high neuroticism, introversion, prior psychiatric disorder, poor self-confidence before age 15, life stress before and after the trauma, and being female. Fontana and Rosenheck (1993), focusing on Vietnam veterans, add the following pretrauma variables to Davidson's list: a background of academic difficulty, an unstable or problematic family, a father who had been in combat, problems with authorities, illegal drug use, and ethnic minority status. Finally, researchers have shown that among Dartmouth College graduates who later served in Vietnam freshman MMPI scores were predictive of later development of lifetime PTSD (Schnurr, Friedman, & Rosenburg, 1993).

In addition to severity and pretraumatic factors, the nature of the traumatic experience may affect the likelihood of developing PTSD. For veterans, being wounded in combat, witnessing or participating in abusive violence or atrocities, or receiving a disciplinary action such as a court martial has been reported to be related to the severity of PTSD (Fontana & Rosenheck, 1993). For rape victims, important factors appear to be prior acquaintance with the perpetrator, or threat to life (Herman, 1992).

Finally posttraumatic factors may have an important influence on facilitating recovery or promoting the development of PTSD. For instance, because the first 24 to 72 hours following traumatic exposure are particularly decisive, rapid therapeutic intervention such as critical-incident stress debriefing within this crucial period can often prevent the later development of PTSD (Mitchell, 1983). The quality of the posttrauma recovery environment may also determine the long-term clinical outcome (Williams & Summer, 1994). There is also reason to believe that social support, availability of help, and availability of someone in whom to confide about the trauma can reduce the likelihood of developing PTSD (Fontana & Rosenheck, 1993).

## Ethnocultural Traditions and Vulnerability to PTSD

There are many possible interactions between ethnocultural traditions and vulnerability to PTSD. For instance, given evidence that a number of neurobiological systems are affected by exposure to trauma and altered in individuals who suffer from PTSD, it is possible that genetic differences may confer greater or less vulnerability to PTSD on people from different ethnocultural backgrounds (see chapter 20 in the present volume by Lin, Poland, Anderson, & Lesser).

Through historical events and accidents of geography, some ethnocultural groups have had more exposure to trauma than others. In some

cases cultures have developed institutions or rituals for addressing the impact of traumatic exposure For example, the Navajo Enemy Way ceremony represents that culture's ritualized attempt to cleanse returning warriors from the deleterious impact of war trauma and to help them readjust to a peace-time society (see Manson et al., this volume). The ceremony is predicated on Navajo society's understanding that warriors may experience profound psychological consequences because of their exposure to the trauma of war. Family and the tribe accept responsibility for the impact of trauma on their courageous young men. The healing ceremony, the Enemy Way, is a ritual in which the extended family, clan, and surrounding community members actively join the warrior. Such collective acceptance of ceremonial participation facilitates processing of war trauma and reintegration into peacetime Navajo society.

Religion and cultural beliefs may differentially influence the meaning and subjective experience of trauma. For example, a terrifying physical ordeal might be appraised as a rite of passage in one cultural setting and as a traumatic event in another. Eastern versus Western beliefs about individual responsibility and the capacity of individuals to influence events may have profoundly different effects on the appraisal, meaning, and psychological impact of traumatic events. Futhermore, different ethnocultural practices may have a direct bearing on PTSD symptoms themselves as defined by the *DSM–IV*. For example, the meaning, experience, and subjective context for stress-induced dissociation may be very different for a Zen Buddhist monk than for an adult incest survivor of the Western world.

Finally, current information regarding risk factors for PTSD (Fairbank, Schlenger, Saigh, & Davidson, 1995) suggests that individuals from cultures that provide family stability, environmental safety, community support, and low rates of drug use might be less vulnerable to PTSD than others. Likewise, we would predict better long-term outcomes for traumatized individuals who are enmeshed in a culture where open discussion of the trauma is encouraged, where survivorship is honored, where victimization is not stigmatized, and where posttraumatic problems are normalized. The remainder of this volume is devoted to exploring some of these issues through more detailed discussions.

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## APPENDIX
## *DSM–IV* CRITERIA FOR PTSD
(American Psychiatric Association, 1994)

**A. The person has been exposed to a traumatic event in which both of the following were present:**

(1) the person experienced, witnessed, or been confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others

(2) the person's response involved intense fear, helplessness, or horror. Note: In children, this may be expressed instead by disorganized or agitated behavior.

**B. The traumatic event is persistently reexperienced in one (or more) of the following ways:**

(1) recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. Note: In young children, repetitive play may occur in which themes or aspects of the trauma are expressed.

(2) recurrent distressing dreams of the event. Note: In children, there may be frightening dreams without recognizable content.

(3) acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated. Note: In young children, trauma-specific reenactment may occur.

(4) intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event

(5) physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event

**C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:**

(1) efforts to avoid thoughts, feelings, or conversations associated with the trauma

(2) efforts to avoid activities, places, or people that arouse recollections of the trauma

(3) inability to recall an important aspect of the trauma

(4) markedly diminished interest or participation in significant activities;

(5) feeling of detachment or estrangement from others

(6) restricted range of affect (e.g., unable to have loving feelings)

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(7) sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span)

**D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:**

(1) difficulty falling or staying asleep
(2) irritability or outbursts of anger
(3) difficulty concentrating
(4) hypervigilance
(5) exaggerated startle response

**E. Duration of the disturbance (Symptoms in B, C, and D) is more than one month.**

**F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.**

*Specify if:*

Acute: if duration of symptoms is less than three months
Chronic: if duration of symptoms is three months or more

*Specify if:*

With Delayed Onset: if onset of symptoms at least six months after the stressor