

Symptomatology and Psychopathology of Mental Health Problems After Disaster

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A variety of reactions are observed after a major trauma. In the majority of cases these resolve without any long-term consequences. In a significant proportion of individuals, however, recovery may be impaired, leading to long-term pathological disturbances. The most common of these is post-traumatic stress disorder (PTSD), which is characterized by symptoms of reexperiencing the trauma, avoidance and numbing, and hyperarousal. A range of other disorders may also be seen after trauma, and there is considerable overlap between PTSD symptoms and several other psychiatric conditions. Risk factors for PTSD include severe exposure to the trauma, female sex, low socioeconomic status, and a history of psychiatric illness. Although PTSD may resolve in the majority of cases, in some cases risk factors outweigh protective factors, and symptoms may persist for many years. PTSD often coexists with other psychiatric disorders, such as depression, anxiety disorders, and substance abuse, and with physical (somatization) symptoms. There is growing evidence that PTSD does not merely represent a normal response to stress, but rather is mediated by specific neurobiological dysfunctions. (*J Clin Psychiatry* 2006;67[suppl 2]:15–25)

Acute stress reactions are a normal and expected response to a traumatic event, seen in the majority of cases. Nevertheless, pathologic persistence of symptoms, or posttraumatic stress disorder (PTSD), is seen in a minority of cases. The development of PTSD depends on complex interrelationships between the nature of the trauma itself, the characteristics of the victim, and the social circumstances and support networks available to the victim. In each case, however, a central feature is the formation of a traumatic memory of the event.^{1,2} The challenges therefore are to understand the defining features of the event that form the basis of this traumatic memory and the factors that influence how the traumatic memory is subsequently manifested as acute and chronic illnesses.

This article reviews the symptomatology and psychopathology of major trauma experienced by disaster victims.

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POSTTRAUMATIC PSYCHOPATHOLOGY

A variety of reactions may be observed after a major trauma. The precise combination of reactions that is observed depends on numerous factors, including the severity and intensity of the initial trauma, the duration of exposure, and the individual characteristics and social circumstances of the survivor. These predictors will be discussed in more detail (see The Sequential Etiological Process Leading to PTSD and Its Predictors).

In a review of 160 studies of disaster victims, Norris et al.³ identified 6 discrete groups of outcomes following major trauma: specific psychological disorders such as PTSD, depression, or anxiety; nonspecific distress; health problems; chronic problems in living; resource loss; and problems specific to youth (Table 1). Overall, 77% of the studies identified specific psychological disorders such as PTSD, major depressive disorder (MDD), or anxiety, including generalized anxiety disorder (GAD) and panic disorder. PTSD was the most commonly observed disorder, being identified in 68% of studies, followed by depression in 36% and anxiety in 20%. In addition, health-related problems such as somatic complaints, sleep disturbances, and substance abuse were reported in 23% of studies.

Initial psychological reactions to trauma may include feelings of fear, horror, or helplessness (symptoms essential for the diagnosis of PTSD; see Symptoms of PTSD). Sometimes, individuals struggle to find the language to express the overwhelming emotion that is experienced and will use words such as "shock," "unbelievable," or "im-

Table 1. Outcomes Following Major Trauma^a

Specific psychological problems
Posttraumatic stress
Depression
Anxiety (including generalized anxiety disorder and panic disorder)
Nonspecific distress
Health problems
Somatic complaints
Sleep disturbances
Alcohol or other substance abuse
Problems in living
Interpersonal relationships
Occupational or financial stress
Environmental concerns
Disruption during rebuilding
Resource loss
Disruption of family and other social networks
Decreased social participation
Problems related to youth
Clinginess, dependence, aggression, etc, in young children
Delinquency in adolescents

^aBased on Norris et al.³

possible to take in.” For some, the emotion that is implicit in these words will only surface in the aftermath of survival, when the individual begins to reflect on the full reality of what has occurred.

Associated symptoms include feelings of guilt, shame or despair, increased hostility, domestic violence, withdrawal, social isolation, and loss of belief structures. Importantly, these symptoms may occur irrespective of whether specific psychological disorders such as PTSD are present. Somatic symptoms include gastrointestinal, cardiovascular, neurologic, musculoskeletal, respiratory, dermatologic, or urological problems.

Notwithstanding this broad range of psychological and somatic problems associated with trauma, trauma victims are generally highly resilient; most develop appropriate coping strategies and use social support networks to reach an understanding and acceptance of their experience. As a result, the majority recover with time, becoming able to resume normal activities and face reminders of their trauma, despite the associated distress. For example, in a study of 95 female rape victims,⁴ 94% met symptomatic criteria for PTSD when interviewed within 30 days of the assault, whereas only 65% did so at a mean of 5 weeks after the assault, and 47% showed PTSD symptoms after an average of 94 days. Similarly, in a study of survivors of the September 11 terrorist attacks in New York,⁵ PTSD symptoms had resolved within 1 year after the attacks in 57% of participants.

In disaster situations where people’s homes and livelihoods have been destroyed, the longitudinal course of recovery is likely to be quite different. Adversity in the aftermath of the disaster and the continued struggle for survival can escalate the individual’s distress in the wake of a sense of euphoria from having survived the initial onslaught of the disaster. The battle for survival in economi-

cally disadvantaged communities has the ability to wear people down.

SYMPTOMS OF PTSD

The Fourth Edition of the American Psychiatric Association *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) recognizes 3 distinct symptom clusters associated with PTSD: reexperiencing the event, avoidance and numbing, and hyperarousal (Table 2).⁶ To qualify for a diagnosis of PTSD, individuals must have been exposed to a stressor that triggers feelings of intense fear, helplessness, or horror, and the symptoms must produce clinically significant distress or functional impairment for a minimum of 4 weeks.

Negative thoughts about the self, other people, and the future are a common feature of PTSD. These can lead to a perception that the world is an extremely dangerous place, and that other people cannot be trusted, or that the victim is incompetent, that other people could have prevented the trauma, and that PTSD symptoms are a sign of weakness. Such negative thoughts are influenced by a variety of factors, including a history of trauma, prior personal or family psychopathology, and a lack of positive social support. Avoidance of situations that recall the trauma can strengthen negative perceptions; conversely, talking and thinking about the trauma can promote an organized, coherent narrative of the victim’s experience, enabling the victim to recognize that trauma is an uncommon event.

Patients with PTSD are often unable to structure their recollections of their trauma. In a positron emission tomography (PET) study,⁷ patients with PTSD did not show the bilateral activation of the dorsolateral prefrontal cortex that normally occurs during updating of working memory in response to trauma-neutral verbal information. By contrast, the PTSD patients showed increased bilateral activation of the superior parietal lobe, compared with control subjects. This might suggest increased reliance on visuospatial coding of information in working memory function, rather than verbal cues, among patients with PTSD. As a result, PTSD patients may have difficulty expressing their feelings of distress after trauma. The neurobiological basis of PTSD is discussed further (see The Neurobiology of PTSD).

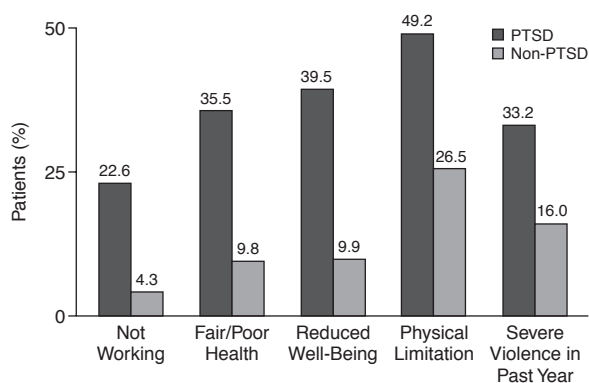
Physical Symptoms of PTSD

Although the DSM-IV criteria emphasize psychological symptoms in the diagnosis of PTSD, it should be noted that many patients with PTSD (particularly in primary care) present with predominantly physical, rather than psychological, symptoms. Such symptoms may include physical pain and lower gastrointestinal, dermatologic, or skeletomuscular disorders.⁸ Sleep disturbances, such as violent or injurious behavior during sleep, sleep paralysis, and sleep talking, are also common in patients with PTSD.

Table 2. Symptoms of Posttraumatic Stress Disorder According to DSM-IV^a

Reexperiencing	Avoidance and Numbing	Hyperarousal
Recurrent, intrusive, distressing memories	Avoiding thoughts, feeling, or conversations connected to the event	Difficulty falling or staying asleep
Recurrent distressing dreams		Irritability or outbursts of anger
Illusions, hallucinations, dissociative flashback episodes	Avoiding activities, places, or people connected to the event	Problems concentrating
Intense psychological distress when exposed to reminiscent cues	Amnesia about certain important aspects of the event	Hypervigilance
Physiological reactivity on exposure to external or internal cues	Decreased interest in once-enjoyed activities	Exaggerated startle response
	Feeling detached from others	
	Emotional numbing/restricted range of affect	
	A sense of foreshortened future	

^aAdapted from the American Psychiatric Association.⁶

Figure 1. Impaired Function and Quality of Life in Vietnam War Veterans With PTSD, Compared With Veterans Without PTSD^a

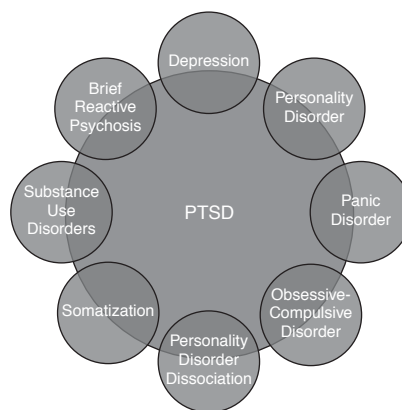
^aData from Zatzick et al.¹¹

Abbreviation: PTSD = posttraumatic stress disorder.

In one study of a general urban population,⁹ for example, sleep disturbances were present in approximately 70% of patients with PTSD. Fatigue and sense of ill health can contribute significantly to the disability that an individual develops.

Impact of PTSD Symptoms on Functioning and Quality of Life

PTSD can have a devastating impact on the victim. Data from the National Comorbidity Survey (NCS) in the United States show that individuals with PTSD are 6 times more likely than those without PTSD to attempt suicide and that, overall, 19% of PTSD patients will attempt suicide.¹⁰ This reflects a substantial impairment of quality of life and normal functioning in patients with PTSD. In a study of 1200 Vietnam War veterans,¹¹ for example, participants with PTSD were significantly more likely than those without PTSD to be not working, to report impaired health and physical functioning, and to have committed a violent act within the previous year (Figure 1). Similarly, in a longitudinal study of patients with anxiety disorders,¹² patients with PTSD showed significantly higher incidences of suicide attempts, alcohol abuse or dependence, and hospitalization for psychiatric

Figure 2. Symptom Overlap Between Posttraumatic Stress Disorder (PTSD) and Other Psychiatric Conditions^a

^aBased on Kessler et al.¹⁴

illness, compared with patients with a history of trauma but no PTSD.

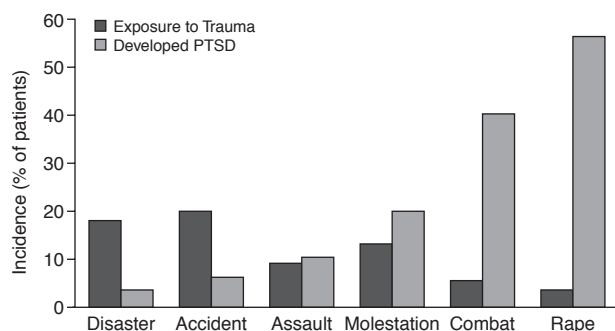
Overlap Between PTSD Symptoms and Those of Other Psychiatric Conditions

The diagnosis of PTSD is a useful organizing construct to categorize symptoms following trauma. However, it is important to recognize that there is considerable overlap between the symptoms of PTSD and those of a number of other psychiatric conditions, including MDD, GAD, panic disorder, obsessive-compulsive disorder, and reactive psychosis (Figure 2).^{13,14} Indeed, PTSD may not necessarily be the most common disorder after trauma; rather, it is the one whose onset is most easily defined.

THE SEQUENTIAL ETIOLOGICAL PROCESS LEADING TO PTSD AND ITS PREDICTORS

PTSD represents a failure of natural recovery following an acute stress, and as such is not a normal event. It is the fifth most common psychiatric disorder in the United States, with a lifetime prevalence of 7.8% in the NCS.¹⁴ However, trauma is a much more common occurrence, affecting 61% of men and 51% of women in the NCS.¹⁴

Figure 3. Incidence of Posttraumatic Stress Disorder (PTSD) in Relation to Type of Trauma in the National Comorbidity Survey^a



^aData from Kessler et al.¹⁴

Clearly, as noted above, not all traumatized individuals develop PTSD. The incidence is higher in specific high-risk groups, and certain types of trauma are more likely to result in PTSD than others.

Risk Factors for Acute PTSD

Data from the NCS show that the prevalence of generalized PTSD varies markedly according to the nature of the trauma involved. Although rape was a relatively uncommon trauma, it was associated with the highest prevalence of PTSD in both men and women (Figure 3).¹⁴ By contrast, natural disasters and accidents affected a higher proportion of the population, but accounted for relatively low prevalences of PTSD.

The same study showed that the lifetime incidence of PTSD was approximately twice as high in women as in men (10.4% vs. 5.0%, respectively, $p < .05$).¹⁴ The lifetime prevalence was higher among the previously married than among the currently married, but this was significant only in women (18.9% vs. 9.6%, $p = .004$). The prevalence was also higher among the married than among the never married, but this was significant only in men (6.1% vs. 1.9%, $p = .001$).

It is worth noting that the increased rate of PTSD in women may not be universal and may be influenced by cultural factors. For example, in the Australian replication of the NCS, the rates of PTSD were similar in men and women.¹⁵

Individual factors render some people more susceptible to PTSD than others. The review of 160 studies of disaster victims cited previously identified a number of risk factors for adverse outcomes such as PTSD, including severity of exposure to trauma, secondary stressors such as financial difficulties, prior psychiatric illness, and deteriorating psychosocial resources (Table 3).³

A meta-analysis by Brewin et al.¹⁶ examined the impact of 14 risk factors for generalized PTSD: gender, age at the

Table 3. Risk Factors for Posttraumatic Stress Disorder After a Major Disaster^a

Severe exposure to the trauma
Living in a highly disrupted community
Female gender
Belonging to an ethnic minority group
Middle age
Poverty or low socioeconomic status
Presence of children in the home
Presence of a distressed spouse
Psychiatric history
Impoverished support system

^aBased on Norris et al.³

time of trauma, socioeconomic status, education, intelligence, race, previous psychiatric history, reported abuse in childhood, reports of previous traumatization, reports of other adverse childhood factors, family history of psychiatric disorder, trauma severity, posttrauma life stress, and posttrauma social support. Each of these was found to be highly significant statistically, but the size of the effects varied markedly. The largest effects were seen with factors operating during or after the trauma: trauma severity, lack of social support, and posttrauma stress. In general, factors that were present before the trauma had relatively little effect on the risk of PTSD.

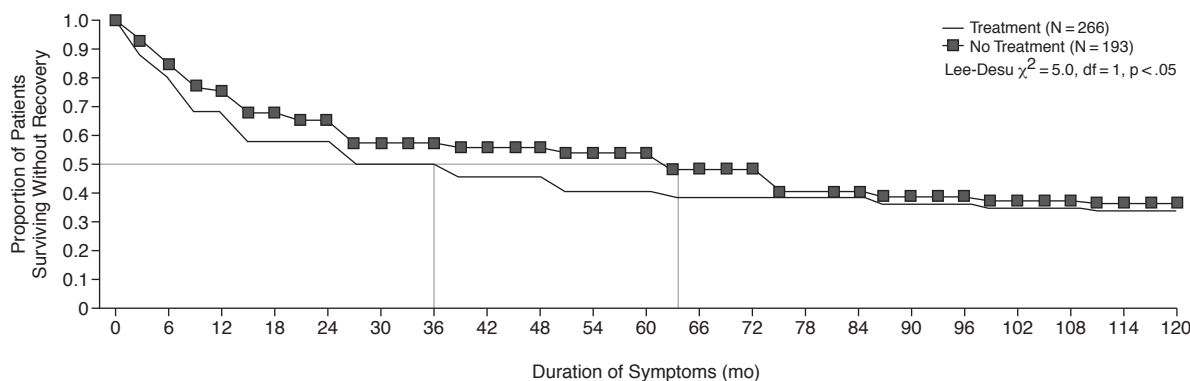
Time Course of PTSD

PTSD should be considered as occurring in a series of stages.¹ Symptoms developing within 4 weeks after the trauma are considered to represent an acute stress disorder that constitutes a normal response to stress. The majority of patients with such symptoms do not develop any pathological sequelae. Thus, PTSD does not begin in the immediate aftermath of the trauma, but may represent a lack of resolution of the acute stress response.¹ Although PTSD resolves in approximately 60% of cases (Figure 4),¹⁴ some individuals go on to develop chronic, unremitting PTSD.

According to the DSM-IV criteria, PTSD is considered acute if symptoms resolve within 3 months or chronic if symptoms persist for 3 months or longer.⁶ Acute PTSD is attributed to time-dependent sensitization following trauma. By contrast, chronic PTSD results from prolonged exposure to the normal adaptive responses to changing circumstances.¹⁷ Secondary stressors, such as loss of home or livelihood after a disaster, are likely to play an important role in the development of chronic PTSD.

Delayed-onset PTSD, which develops at least 6 months after the trauma, is recognized as a discrete subcategory of PTSD, but relatively little is known about this condition. The available evidence suggests that delayed-onset PTSD is uncommon following disasters.^{1,18} For example, in a study of 469 Australian firefighters exposed to a major bushfire,¹⁸ a delayed onset of PTSD was rare, and some patients who reported such a course could not recall their acute posttraumatic symptoms. Other studies have re-

Figure 4. Kaplan-Meier Curves Showing the Duration of Symptoms in Patients With Posttraumatic Stress Disorder in the National Comorbidity Survey^{a,b}



^aReprinted with permission from Kessler et al.¹⁴

^bMean duration of symptoms was 64 months in people who never received treatment, compared with 36 months in people who received treatment.

ported marked fluctuations in PTSD symptoms during long-term follow-up of survivors of natural disasters, which may explain the occurrence of at least some cases of apparently delayed onset of PTSD.^{1,19}

PTSD symptoms can persist for many years after the trauma. The NCS data show that the mean duration of symptoms in people who never received treatment was 64 months, compared with 36 months in people who received treatment for their symptoms.¹⁴ Moreover, in more than one third of affected individuals, symptoms persisted even after many years, and even when treatment was given (Figure 4). This is consistent with data from survivors of 2 disasters in the United Kingdom.^{20,21} In a study of survivors of the 1988 Piper Alpha oil platform fire,²⁰ 21% of participants met the most stringent criteria for PTSD more than 10 years after the event. Approximately one third of participants reported persistent feelings of guilt at 10 years, and this, together with physical injury during the disaster, was significantly associated with high levels of posttraumatic symptoms. A second study²¹ investigated the long-term outcome in survivors of the 1966 Aberfan disaster, in which a coal slag heap collapsed onto a primary school, killing 116 children. After 33 years, 46% of survivors had experienced PTSD at some time, compared with 20% of matched controls (odds ratio [OR] = 3.38, 95% confidence interval [CI] = 1.40 to 8.47), and 29% met the diagnostic criteria for current PTSD.

Risk Factors for Chronic PTSD

What factors influence the development of chronic PTSD? In a study of young adults,²² individuals with chronic PTSD showed a higher total number of PTSD symptoms, and higher rates of numbing and hyperreactivity to stressors, anxiety or affective disorders, and other comorbid medical conditions, compared with individuals

with nonchronic PTSD. Female sex and a family history of PTSD were independent risk factors for chronic PTSD.

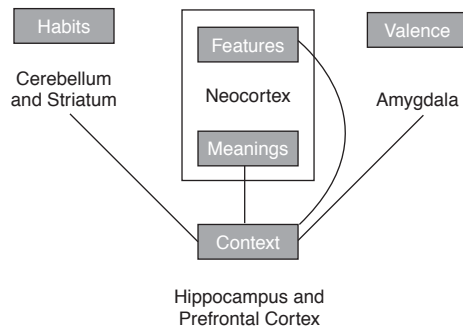
Avoidance behaviors in response to stressors also influence the development of chronic PTSD. Avoidance symptoms tend to increase with time, whereas intrusive symptoms decrease.^{1,23} Such symptoms may prevent recovery by limiting exposure to experiences that correct negative perceptions and beliefs and by preventing the organization of the memory and consignment of the trauma to the past. Furthermore, avoidance symptoms can maintain the individual's perception that the world is a dangerous place and that he or she cannot cope effectively with stress.

THE NEUROBIOLOGY OF PTSD

Stressful situations trigger adaptive responses aimed at maintaining a constant internal environment in the face of changing demands on the individual, a process known as allostasis.^{17,24} These responses, although beneficial in the short term, impose an allostatic load on the individual, which if maintained can result in PTSD and other disorders.

The hypothalamus-pituitary-adrenal (HPA) axis plays an important role in the acute response to stress. During the acute phase, glucocorticosteroid release has a number of effects that are beneficial for short-term survival, including suppression of immune function, activation of the autonomic nervous system, and replenishment of energy reserves by promoting the conversion of proteins and lipids into carbohydrates.^{24,25} However, prolonged stress results in sustained elevation of circulating glucocorticosteroids, which can produce structural and functional changes in areas of the brain involved in learning and memory processing (see Neuroanatomy of PTSD).²⁴

PTSD patients show a number of alterations in the HPA system. Concentrations of corticotropin-releasing factor

Figure 5. Anatomical Areas Involved in Memory^{a,b}

^aBased on Nadel and Moscovitch.²⁸

^bAccording to this model, semantic information (information about feelings and meanings of words) is stored in the neocortex, habitual information (that required for acquired skills such as playing a musical instrument) in the cerebellum and striatum, and information about feelings (valence) in the amygdala. The hippocampus and prefrontal cortex are necessary for retrieval of episodes and their contextual framework, and for extraction of semantic information to be stored in the neocortex. Posttraumatic stress disorder is associated with hippocampal damage, resulting in difficulties in forming structured contextual memories of the trauma.

(CRF) in the cerebrospinal fluid are elevated,²⁵ presumably reflecting hypersecretion of this peptide by the hypothalamus. Paradoxically, however, circulating cortisol concentrations are reduced, compared with individuals without PTSD.²⁶ This could be due to down-regulation of the adrenocorticotrophic hormone (ACTH) response to CRF,²⁷ which would result in decreased secretion of cortisol. In animal models, CRF release is associated with an increase in the number and sensitivity of glucocorticosteroid receptors in brain areas involved in memory and the control of fear and arousal responses, such as the hippocampus.²⁵ Such findings suggest that hypersecretion of CRF in PTSD patients would result in the promotion of anxiety and fear-related behaviors.

Neuroanatomy of PTSD

Data from animal and clinical studies are converging to indicate that multiple memory systems exist, with distinct anatomical localizations and organization.^{28,29} According to this view, different types of information are stored in the cerebellum, neocortex, and amygdala, and the hippocampus is necessary for “explicit” memory—the retrieval of episodes and their contextual framework (Figure 5).²⁸ The hippocampus is also responsible for suppressing the fear response in the amygdala under conditions of medium stress, but this pathway is blocked under conditions of extreme stress, resulting in an exaggerated fear response.^{30,31}

The hippocampus is a major target organ for glucocorticosteroids in the brain³² and is particularly vulnerable to neurotoxicity resulting from high levels of glucocorticosteroids following stress. A number of inter-

ventions, including prolonged stress, glucocorticosteroid treatment, and developmental lead exposure result in remodeling and atrophy of hippocampal pyramidal neurons.^{24,32,33} The effects of stress on the hippocampus can be blocked by an inhibitor of adrenal steroid synthesis, indicating that they are a result of prolonged exposure to glucocorticosteroids.²⁴ The effects of glucocorticosteroids on hippocampal cell volume may be due both to direct glucocorticoid effects on cell metabolism and an increased susceptibility to excitatory amino acids and other neurotoxic agents.³⁴

Studies using imaging techniques such as functional magnetic resonance imaging (fMRI) and PET have provided data on changes in hippocampal volume in patients with PTSD and healthy controls. For example, Bremner et al.,³⁵ in an MRI study of 26 Vietnam veterans with PTSD, showed that the volume of the right hippocampus was reduced by 8% ($p = .03$), compared with that in matched control subjects; the volume of the left hippocampus was also reduced, by 3.8%, but this difference was not statistically significant. In a second study,³⁶ quantitative volumetric MRI techniques were used to compare hippocampal volumes in 7 Vietnam veterans with PTSD, with those in 7 combat veterans without PTSD and 8 normal control subjects. Both left and right hippocampal volumes were significantly lower in veterans with PTSD than in the other 2 groups, and these differences remained after adjustment for age, whole brain volume, and lifetime alcohol consumption. A reduction in hippocampal volume, compared with control subjects, has also been reported in adult patients with PTSD related to childhood physical or sexual abuse.^{34,37} A meta-analysis of 9 studies³⁸ reported a significant reduction in volume of both the right and left hippocampi in 133 adults with PTSD compared with 148 healthy controls. However, other studies have failed to demonstrate such an association.³⁸

Several studies have reported correlations between decreases in hippocampal volume and PTSD symptoms. In a study of 21 women who reported being severely sexually abused during childhood,³⁷ the volume of the left hippocampus was decreased by 5%, compared with non-abused controls; there was a significant negative correlation between left hippocampal volume and the severity of dissociative symptoms ($r = -0.73$, $p < .0002$), although no correlation was seen between left hippocampal volume and measures of explicit memory functioning. Bremner et al.³⁴ reported a similar decrease in left hippocampal volume in adult survivors of childhood physical or sexual abuse. In this study, however, there was only a weak and nonsignificant correlation between left hippocampal volume and the number of PTSD symptoms present ($r = 0.29$, $p = .31$).

Such findings suggest that PTSD is associated with damage to the hippocampus, which might result in deficits in explicit memory functioning. It is important to recog-

nize, however, that the relationship between PTSD and hippocampal volume may not be causal. It is possible, for example, that hippocampal damage is present before the onset of PTSD and may in some way predispose the individual to develop the condition.³⁹

There are data to suggest that emotions associated with PTSD symptoms are mediated by the limbic and paralimbic systems in the right hemisphere.^{12,40-42} In one study, for example, activation of different brain areas was measured by PET in 8 PTSD patients following exposure to audiotaped trauma-related or neutral scripts.⁴⁰ Compared with control conditions, traumatic stimuli provoked marked activation of the right-sided limbic, paralimbic, and visual areas and decreased activation of the left inferior frontal and middle temporal cortex. Such activation of the visual cortex may be responsible for reexperiencing phenomena in PTSD, since it has been shown that visual imagery is mediated by topographically organized visual cortex.⁴³ However, reexperiencing phenomena are distinct from ordinary visual mental images—to the person experiencing a flashback, it feels as if the trauma is reoccurring⁶—and hence these may require activation or deactivation of other brain areas in addition to activation of visual cortex.⁴⁰ The finding that Broca's area (the left inferior frontal cortex and middle temporal cortex) was deactivated following exposure to trauma-related stimuli may indicate movement of resources from higher cognitive functions, such as language processing and verbalization. This would be consistent with the finding that PTSD patients have difficulty in cognitively structuring their traumatic memories and make less use of verbal memory in structuring their experiences.⁷

In summary, the available evidence suggests that PTSD is associated with an increased allostatic load, with prolonged activation of the HPA axis. A decrease in hippocampal volume might precede or follow, resulting in the impairment of explicit memory and perhaps also loss of restraint of fear responses mediated by the amygdala and other components of the limbic system. The damage to the hippocampus might mean that the individual is unable to form structured contextual memories of the trauma, while deactivation of Broca's area might prevent the individual from developing verbal representations of the trauma.

Neurochemical Systems Involved in the Psychopathology of PTSD

Exposure to traumatic stressors leads to activation of arousal responses mediated by the serotonergic and noradrenergic systems and to changes in numerous other neurotransmitter and neuroendocrine systems.^{44,45} In animal studies, serotonergic mechanisms have been shown to be involved in the conditioned fear responses, mediated by the amygdala, and involving CRF release, and in symptoms such as intrusions, depression, depersonalization, and avoidance behaviors.^{46,47}

Resilience and vulnerability in the face of extreme stress are mediated by multiple neurochemical and neuroendocrine mechanisms.⁴⁵ There is evidence that corticotropin-releasing hormone (CRH), dopaminergic and glutamatergic systems, and estrogens are among the factors involved in the mediation of vulnerability; conversely, factors mediating resilience include dehydroepiandrosterone (DHEA), neuropeptide Y, galanin, testosterone, serotonin acting via the 5-HT_{1A} receptor, and benzodiazepine receptor function.⁴⁵

Treatment of PTSD, whether by medication or psychotherapy, may reverse the functional and structural changes in the affected systems, leading to normalization of responses to stress. Evidence for this hypothesis comes from a study in which 11 patients with PTSD underwent single photon emission computed tomography (SPECT) scanning before and after treatment with a selective serotonin reuptake inhibitor (SSRI).⁴⁸ Significant deactivation of the left medial temporal cortex was observed following SSRI treatment, irrespective of antidepressant response. There was a significant correlation between reductions in PTSD symptoms and activation of the left paracingulate region (medial prefrontal cortex). Such findings suggest that SSRI treatment may eliminate learned fear responses by reversing the abnormal regulation of amygdala activity by the medial prefrontal cortex seen in PTSD.^{48,49} A second study,⁵⁰ involving 28 PTSD patients, has shown significant reductions in PTSD symptoms, which were associated with a 4.6% increase in mean hippocampal volume on MRI, following treatment with an SSRI for 9 to 12 months.

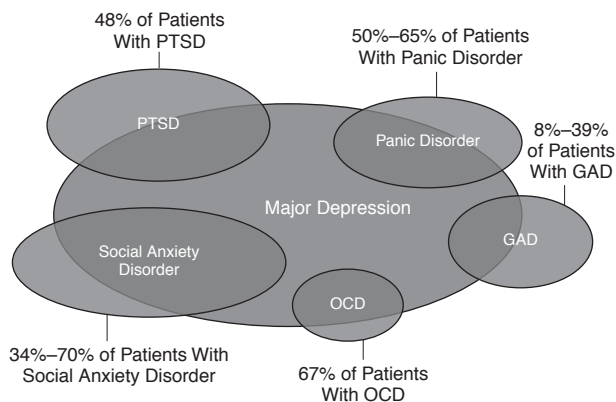
PSYCHIATRIC AND PHYSICAL COMORBIDITY FOLLOWING TRAUMA

Although PTSD may be the most common disorder following trauma, it represents only a part of the clinical picture. Comorbidity with other psychiatric or somatic disorders is common (Figure 6)^{6,14,51-54}; indeed, the epidemiologic evidence suggests that psychiatric comorbidity is the rule rather than the exception.¹³ In the NCS, a lifetime history of at least 1 other psychiatric disorder was present in 88.3% of men and 79% of women with a lifetime history of PTSD; 59% and 44%, respectively, had 3 or more concomitant disorders.¹⁴ Typically, however, PTSD precedes other disorders; in the NCS, PTSD was the primary diagnosis in 29% to 51% of men and 41% to 58% of women with comorbid disorders.¹⁴

Despite the high prevalence of comorbid disorders in patients with PTSD, little is known about risk factors for comorbidity. For example, a study of reactions to the September 11 terrorist attacks showed a higher prevalence of probable PTSD among residents of the New York City metropolitan area than among residents of other major U.S. urban centers, but noted that further research would

Figure 6. Comorbidity of PTSD and Other Psychiatric Disorders^a

Major depression is present in:



^aBased on references 6, 14, and 51–54. Abbreviations: GAD = generalized anxiety disorder, OCD = obsessive-compulsive disorder, PTSD = posttraumatic stress disorder.

be necessary to document the time course and outcome of psychiatric disorders in affected individuals.⁵⁵ This lack of data reflects the difficulties involved in designing epidemiologic studies of psychiatric illness after major disasters.⁵⁶ Such studies require careful attention to timing, sampling, measurement, and interpretation of data. Moreover, reactions to different types of trauma can vary considerably: for example, survivors of natural disasters may show different reactions to survivors of terrorist attacks, while even within the context of an individual disaster, differing reactions may be seen in subpopulations of survivors.⁵⁶

Traumatic Grief

Traumatic grief is common after a major disaster. The symptoms of traumatic grief are distinct from those of depression and anxiety, but show clinical correlations with those of depression. Moreover, traumatic grief symptoms are predictive of mental and physical health impairments, independent of the effect of depressive symptoms. For these reasons, traumatic grief is now considered to be a distinct clinical syndrome in its own right.⁵⁷

Traumatic grief can produce symptoms that overlap with those of PTSD, such as recurrent intrusive thoughts and images of death and avoidance of situations, activities, or people associated with the event. In addition, however, traumatic grief can produce symptoms such as intense yearning and longing for the dead person and extreme sadness rather than anxiety and arousal (Table 4).⁵⁷ As a result, the grieving process following a disaster may differ from that in the absence of trauma. In normal grief, the individual is able to retrieve positive memories of the deceased person, whereas following a disaster, traumatic memories may intrude and inhibit this process. By this

Table 4. Diagnostic Criteria for Traumatic Grief^a

Criterion A	Person has experienced the death of a significant other Response involves 3 of the 4 symptoms below experienced at least sometimes: Intrusive thoughts about the deceased Yearning for the deceased Searching for the deceased Loneliness as a result of the death
Criterion B	In response to the death, 4 of the following 8 symptoms are experienced as mostly true: Purposelessness or feelings of futility about the future Subjective sense of numbness, detachment, or absence of emotional responsiveness Difficulty acknowledging the death (eg, disbelief) Feeling that life is empty or meaningless Feeling that part of oneself has died Shattered worldview (eg, loss of sense of security, trust, or control) Assumes symptoms or harmful behaviors of, or related to, the deceased person Excessive irritability, bitterness, or anger related to the death
Criterion C	Duration of disturbance (symptoms listed above) is at least 2 months
Criterion D	The disturbance causes clinically significant impairment in social, occupational, or other important areas of functioning

^aAdapted with permission from Prigerson et al.⁵⁷

means, traumatic grief can itself give rise to and perpetuate PTSD symptoms.

Major Depressive Disorder

The core symptoms in MDD are a depressed mood and anhedonia (an inability to feel normal happiness or pleasure). These are usually accompanied by a range of both psychological symptoms, such as feelings of worthlessness, excessive guilt, and suicidality, and physical symptoms such as changes in appetite, sleep disturbances, and loss of energy.

MDD is the most common concomitant psychiatric disorder in patients with PTSD.¹³ In the NCS, for example, 48% of men and 49% of women with PTSD had a lifetime diagnosis of MDD.¹⁴ This is perhaps not surprising, as there is a well-established causal relationship between stressful events and depressive illness.⁵⁸ Conversely, however, a history of MDD is predictive of PTSD after exposure to major trauma.¹³ The frequent coexistence of PTSD and MDD reflects the shared neurobiology of the 2 conditions. In both cases, sensitization resulting from exposure to secondary stressors can lead to “kindling” effects that exacerbate symptoms and impair normal recovery.

The presence of MDD is associated with greater functional impairment in patients with PTSD. For example, in a study of trauma survivors recruited from an emergency room,⁵⁹ individuals with concomitant PTSD and MDD showed more severe symptoms and lower functioning than those with either disorder alone. Similarly, a study of motor accident survivors⁶⁰ showed that PTSD and MDD

were correlated but independent responses to the trauma; again, individuals with both disorders showed greater functional impairment and were less likely to show remission of symptoms over 6 months.

Generalized Anxiety Disorder

Generalized anxiety disorder is defined by excessive worry and apprehension about events or activities, occurring most days for at least 6 months, which is difficult to control and unrelated to other Axis I disorders.⁶ Psychological symptoms of GAD include persistent feelings of fearful anticipation, irritability, impaired concentration, and restlessness. Physical symptoms include muscle tension and symptoms of autonomic hyperarousal, such as palpitations and tightness or pain in the chest.

In the NCS, GAD occurred in 16.8% of men and 15% of women with PTSD.¹⁴ However, in contrast to comorbid depressive illness, in which PTSD is usually the primary disorder, PTSD is more likely to develop after anxiety disorders such as GAD.^{13,14} This suggests that symptoms of arousal and avoidance may develop as a coping mechanism following exposure to trauma.¹³

Panic Disorder

Panic disorder is characterized by recurrent, spontaneous episodes of intense anxiety with associated somatic and psychiatric symptoms (panic attacks). The incidence of panic disorder among persons with PTSD in the NCS was 7.3% in men and 12.6% in women.¹⁴ Since some studies have found that panic disorder tends to be more common in individuals exposed to traumas involving extreme autonomic arousal, hypervigilance, and unpredictability, it has been suggested that panic disorder and PTSD are interrelated, rather than comorbid, disorders.^{13,61} In panic disorder, however, fear and avoidance are related to the physical symptoms associated with panic attacks (anticipatory anxiety), whereas in PTSD they are specifically related to trauma-related memories and situations.¹³

Sometimes, the relationship between the panic symptoms and the subtle triggers of the traumatic experience goes undetected. For example, physical sensations such as pain and movement may have the same somatosensory quality as the traumatic experience, but the individual has little conscious awareness of this issue.

Substance Abuse

Abuse of nicotine, alcohol, or narcotic drugs is common among patients with PTSD.^{14,62-65} In the NCS, 52% of men and 28% of women with PTSD reported alcohol abuse or dependence, while 35% and 27%, respectively, reported abuse of or dependence on other substances.¹⁴ Increased substance use after trauma appears to be related to PTSD, rather than exposure to stress per se.^{63,64} The available evidence suggests that the association between PTSD and substance abuse is causal in nature.⁶⁶ For example, in a

longitudinal study of Australian firefighters exposed to a natural disaster, PTSD was associated with both increases and decreases in alcohol consumption, and these changes could be attributed to PTSD rather than exposure to stress.^{18,66,67} However, the association is not simple: for example, some people may use alcohol or narcotics as a form of self-medication to relieve the symptoms of PTSD, and this may obscure the association by modifying the underlying symptoms.⁶⁶

Although some studies have suggested that substance abuse is more common among combat veterans than among survivors of natural disasters,^{61,68} more recent evidence suggests that the intensity of the emotional reaction, rather than the nature of the trauma, is the predominant factor involved. For example, in a study of 84 individuals who sought support after the 1995 Oklahoma City bombing,⁶⁴ those who reported increased smoking or drinking after the attack showed more severe peritraumatic reactions, grief, posttraumatic stress, and worries about safety, and greater impairment of functioning, than those who did not. There were no significant differences in sensory or interpersonal exposure to the trauma between participants who increased their smoking or drinking and those who did not.

As with PTSD, substance abuse after trauma may have a long-term impact on health and well-being. In a study of New York City residents after the September 11 attacks,⁶⁵ the increase in substance use seen following the attacks persisted during the first 6 months after the attacks, whereas the incidence of PTSD and depression decreased by more than 50% during the same period.

Somatic Symptoms

Numerous physical (somatization) symptoms may coexist with PTSD, and these can have a significant impact on normal functioning and the course of PTSD.⁶⁹ In a study of young adults,⁷⁰ the incidence of somatization symptoms was 3 times higher in individuals with PTSD than in those without PTSD (24.7% vs. 8.2%, respectively). Moreover, a baseline history of PTSD was associated with an increased risk of pain (OR = 2.1) or conversion symptoms (OR = 2.3) during follow-up, compared with individuals without PTSD. Other studies have demonstrated increased rates of cardiopulmonary, neurologic, and musculoskeletal symptoms among combat veterans⁷¹ and firefighters⁷² with PTSD, compared with members of the same groups without PTSD.

Physical symptoms in patients with PTSD may result from a number of causes, including injuries sustained during the original trauma and its aftermath (for example, infectious diseases following a natural disaster), comorbid substance abuse, and physiological responses to secondary stressors, such as loss of home or livelihood. Physical symptoms may be present even when the issue of injury has been controlled for. The first explanation for this is

that the symptoms represent the awareness and reporting of psychophysiological concomitants of the dysregulation that is central to the PTSD. Alternatively, the symptoms may represent a somatic component of the traumatic memory.

The finding that physical symptoms are common in patients with PTSD has important diagnostic implications. There is a high probability that a person presenting with physical symptoms after a disaster may have both a physical and a psychological disorder. However, somatization is common; hence, the absence of a demonstrable medical disorder in a patient with physical symptoms does not mean that no disorder exists.

CONCLUSION

Acute stress reactions are common and expected after disaster and other trauma. While these can be distressing to the individual concerned, resilience is also common and most affected individuals recover with time. In some cases, however, recovery is incomplete, leading to a number of psychiatric conditions, of which PTSD is the most frequently encountered. PTSD often coexists with a variety of psychiatric and physical disorders, which further increase the burden of suffering experienced by the patient.

Neuroimaging and neurobiological studies are providing important new insights into the brain regions and pathways involved in the development of PTSD symptoms and the recovery from trauma. These insights are focusing attention on the potential benefits of the treatment of PTSD; studies with SSRIs suggest that these agents have positive effects in patients with PTSD,¹¹ and there is also evidence that they are able to normalize the characteristic psychobiology of PTSD.

Disclosure of off-label usage: The authors have determined that, to the best of their knowledge, no investigational information about pharmaceutical agents that is outside U.S. Food and Drug Administration–approved labeling has been presented in this article.

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