1 Post-Traumatic Stress Disorder: Definition, Prevalence, and Risk Factors

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Contents

Abstract

In this chapter, we provide the definition and diagnostic criteria for post-traumatic stress disorder (PTSD). Next, the prevalence data for this disorder are reviewed, with a particular focus on how prevalence rates vary with demographic characteristics (e.g., gender) and trauma type. The literature on risk and resilience factors for the development and maintenance of PTSD is then discussed. The chapter concludes with a discussion of contemporary statistical methods that may be used to advance our knowledge and understanding of PTSD.

Key Words: Demographic, epidemiology, meta-analyses, refugees, risk factor.

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INTRODUCTION

The global war on terrorism and the military actions in Iraq and Afghanistan (i.e., Operation Iraqi Freedom and Operation Enduring Freedom) represent significant risks to the mental health of the American forces assigned to these regions. Coupled with the impact of the terrorist attacks on the Pentagon and the World Trade Center (WTC), the diagnosis of post-traumatic stress disorder (PTSD) is currently receiving increased attention in both the scientific and popular literature. This chapter seeks to provide a current understanding of the nature of the diagnosis, its prevalence across a variety of different populations, and risk factors associated with developing it. In addition, we present an overview of an etiological model for PTSD previously proposed (*1,2*). We, finally, summarize the literature on structural equation modeling in PTSD, a statistical approach that has contributed greatly to our understanding of the associations and predictive value of many of the key variables underlying the development of PTSD.

Our ultimate understanding of this condition, to include its biological and psychological substrates, is premised on the use of a common definitional framework across scientific and clinical venues. Great progress in the field was stimulated by the inclusion of the diagnosis of PTSD in the psychiatric nomenclature in 1980 and by several attempts to strengthen the operational criteria employed to define cases and noncases of people who have PTSD (*3*). Our goal here is to contribute to continued investigation of scientific research on this topic that blends the best in behavioral science with the most contemporary models and methods of measuring and understanding the biological and physiological parameters associated with PTSD (*4*).

DEFINITION AND DIAGNOSTIC CRITERIA FOR PTSD

In its current conception, the Diagnostic and Statistical Manual of Mental Disorders (DSM) defines PTSD as stemming from an event in which one is exposed to serious threat of injury or death and then experiences extreme fear, helplessness, or horror. Three symptom clusters define the disorder. In addition to recurrent and intrusive recollections and dreams of the event, the reexperiencing cluster includes the experience of flashback episodes in which an individual experiences a recurrence of at least a portion of the trauma. Hyperarousal symptoms are characterized by an enhanced startle reaction and difficulty sleeping, concentrating, and controlling anger as well as hypervigilance for danger and a sense of a foreshortened future. Extreme distress and avoidance of cues or reminders of the trauma, as well as an inability to remember aspects of the event, also can accompany this disorder. Additional avoidance symptoms include emotional numbing, described as an inability to feel any positive emotions, such as love, contentment, satisfaction, or happiness.

The interpersonal, psychosocial, physical health, and societal consequences of PTSD contribute to the overall costs of developing this condition. People with PTSD are more likely to divorce, report trouble raising their children, engage in intimate partner aggression, experience depression and other psychological

problems, report poorer life satisfaction and physical health problems, become involved with the legal system, earn less, and change jobs frequently (*5–9*). These findings suggest that PTSD constitutes a major public health challenge for this nation and the world and highlight the importance of our complete understanding of the biological, psychological, and social factors associated with this condition. This review outlines our achievements to date in understanding the characteristics of PTSD and its prevalence, course, and treatment. Further, we provide a heuristic model for understanding the development of PTSD while specifying future directions for scientific work.

PREVALENCE OF PTSD

Initially, PTSD was considered a relatively rare condition, and traumatic events were considered extreme life stressors that were outside the range of normal human experience (*10*). Epidemiological studies, however, have since documented high prevalence rates of exposure to traumatic events in the general population and confirmed that PTSD occurs following a wide range of extreme life events (*11–13*). Most important, though, are the consistent findings indicating that, although exposure to potentially traumatic events is common, development of PTSD is relatively rare. Elucidation of the factors responsible for some people developing PTSD while others exposed to similar threatening events do not may inform our understanding of key variables in the etiology of this condition.

PTSD in the U.S. Population

Perhaps the most complete general U.S. population studies are those conducted by Kessler and colleagues. In the original National Comorbidity Survey (NCS) (*12*), 5,877 individuals aged 15 to 54 years in a nationally representative sample were interviewed using a structured diagnostic interview. An overall lifetime PTSD prevalence rate of 7.8% was found, with rates for women (10.4%) more than twice that for men (5.0%). Trauma exposure estimates indicated that about 60% of men and 51% of women were exposed to one or more traumatic events. In the National Comorbidity Survey Replication (NCS-R) (*13*), 5,692 individuals in a nationally representative sample were interviewed and had an overall lifetime PTSD prevalence rate of 6.8% detected.

Compared with the NCS and NCS-R, similar or slightly higher PTSD rates were obtained in selected, specialized samples. For instance, among 21- to 30-year-old members of a Detroit area health maintenance organization (HMO), 40% reported experiencing a trauma, and 9.5% met PTSD criteria (11.3% of women and 5.6% of men) (*11*). Similarly, among former Miami-Dade public school students aged 18–23 years, 11.5% met lifetime PTSD criteria (15.5% of women and 7.5% of men) (*14*). Further, among two American Indian tribes, lifetime PTSD rates were 14.2% and 16.1%, while past year rates were 4.8% and 5.8%, respectively, with higher rates consistently found among women compared with men (*15*).

Other studies focused on sexual assault and criminal victimization of women. For example, in a nationally representative sample of 4,008 women, Kilpatrick,

Edmunds, and Seymour (*16*) found that 13% reported a completed rape. Of those who were raped, lifetime and current PTSD rates were 32% and 12%, respectively. Similarly, using a national probability sample, Resnick, Kilpatrick, Dansky, Saunders, and Best (*17*) estimated that 36% of women had been criminally victimized, with 14.3% experiencing attempted rape or molestation and 12.7% experiencing a completed rape. They estimated lifetime and current PTSD rates to be 12% and 5%, respectively. Among those who were exposed to criminal victimization, rates of lifetime and current PTSD were 26% and 10%, respectively.

Clearly, the prevalence of exposure to traumatic events in the United States is far more common than anticipated in 1980 when the diagnosis of PTSD was incorporated into the diagnostic nomenclature. Even more surprising are findings indicating that the rate of current PTSD in the general population falls only behind major depression, attention deficit/hyperactivity disorder, specific phobia, and social anxiety disorder, making it the fifth most common psychiatric condition in the United States (*13*).

PTSD Among U.S. Combatants

Soldiers sent to fight wars and to keep peace are among those most at risk for trauma exposure and the development of PTSD. Despite the high frequency of military action and war worldwide, few countries have ever estimated the psychological toll of war. The major exception to this was the National Vietnam Veterans Readjustment Study (NVVRS) (*8*), which included a representative sample of 1,632 Vietnam theater veterans (VTVs), a matched sample of 716 Vietnam era veterans (VEVs), and 668 civilian comparison subjects. Of VTVs, 64% were exposed to trauma in their lives, compared with 48% of VEVs and 45% of civilians. More than 15% of male VTVs and 9% of female VTVs met criteria for current PTSD, and 30% of male VTVs and 27% of female VTVs met criteria for lifetime PTSD. Notably, the direction of this gender difference is opposite that of the civilian samples reviewed here, likely attributable to the different roles women had in the military at that time, the different types of stressors to which they were exposed, and the higher educational level of women in the study. In all cases, PTSD prevalence rates for VTVs were five to ten times higher than those found for the VEVs and civilians.

Litz, Orsillo, Friedman, Ehlich, and Batres (*18*) examined a sample of 3,461 active duty peacekeeping military troops who served in Somalia. Shortly after their return to the United States, 8% of these soldiers reported PTSD, a rate that did not differ for men and women. Eighteen months after their return, 6.5% of a subsample of 1,040 veterans met criteria for delayed-onset PTSD (*19*).

Several studies examined the impact of service in recent wars in the Persian Gulf. For instance, Wolfe, Brown, and Kelley (*20*) conducted a longitudinal study of 2,344 Gulf War I veterans and found PTSD prevalence rates of 4% for men and 9% for women. Studies using smaller convenience and reservist samples found PTSD rates in the range of 16–19% (*21,22*). Among soldiers

deployed during Operation Iraqi Freedom (OIF) and Operation Enduring Freedom (OEF), Hoge et al. (*23*) found that 6.2% of the Army soldiers met screening criteria for PTSD after deployment to Afghanistan, and 12.9% met criteria after deployment to Iraq. Among the Marine Corps soldiers deployed to Iraq, 12.2% met screening criteria for PTSD.

With the elevated prevalence rates of PTSD among combatants, as well as findings indicating that PTSD symptoms among Vietnam veterans typically do not remit (*24*), additional studies of combatants will be a national priority for an indefinite period of time. Further substantiating the course and nature of combat-related PTSD is the detailed study of the military's testing of mustard gas on soldiers during World War II. While this testing was not commonly known until the 1990s, fully 32% of those exposed continued to exhibit full PTSD symptomatology a half century later (*25*).

One study by Vasterling, Proctor, Amoroso, Kane, Heeren, and White (*26*) contributed to our understanding of the specific consequences of war zone exposure. Prior studies utilized cross-sectional designs to compare prevalence rates among exposed and nonexposed samples. The Neurocognition Deployment Health Study was able to examine 961 Army troops prior to and following their deployment to OIF. Measures of sustained attention, verbal learning, and visual spatial memory were impaired as a function of their service. Reaction time measures improved. These findings remained true even when depressive and PTSD symptoms were statistically controlled. These findings support the notion that the trauma exposure of war affects psychological functioning broadly, crossing the emotional and cognitive domains of functioning.

Refugees

Fazel, Wheeler, and Danesh (*27*) summarized the refugee data across multiple studies, including a total of 5,499 adult refugees resettled in Western countries. They found substantial variability in prevalence rates (ranging from 3% to 44%). When restricting analyses to more rigorously designed studies including at least 200 participants, the average PTSD prevalence rate was 9%. This conclusion is different from that of De Girolamo and McFarlane (*28*), who summarized 12 studies of refugees and reported that half of the studies had a PTSD rate equal to or higher than 50%. Several factors likely account for such variability in PTSD rates across studies, including the nature of the sample and the trauma experienced, the length of time since the trauma, and the lack of PTSD measures validated among refugee samples (*29–31*).

PTSD in Disaster Contexts

There is a burgeoning literature suggesting that a wide range of natural and human-made disasters can lead to the development of chronic PTSD. Green and colleagues found that 44% of survivors of the collapse of the Buffalo Creek

Dam in West Virginia in the late 1970s met criteria for PTSD, and 28% of the sample still met diagnostic criteria 14 years later (*32,33*). Similarly, McFarlane (*34*) studied the effects of Australian brush fires on a sample of firefighters. The PTSD prevalence rates at 4, 11, and 29 months postdisaster were estimated at 32%, 27%, and 30%, respectively.

In recent years, concern regarding the psychological consequences of terrorist attacks has increased substantially. In a review of studies reporting PTSD prevalence rates following terrorist attacks, Gidron (*35*) reported a mean rate of 28%. However, these studies varied greatly in subject sampling and in the timing of assessments.

Following the September 11, 2001, terrorist attacks on the WTC, telephone and Web-based methodologies were employed to examine levels of resultant PTSD symptoms throughout the United States. Rates of PTSD generally were higher based on regional proximity to the attacks. For example, Schlenger et al. (*36*) found the prevalence of probable PTSD to be 11.2% in the New York City metropolitan area but much lower in Washington, D.C. (2.7%), other metropolitan areas (3.6%), and the remainder of the country (4.0%). In a sample of adults residing in an area of Manhattan closest to the WTC, 7.5% reported symptoms consistent with a PTSD diagnosis, and 20.0% of a subsample residing closest to the WTC reported such symptoms (*37*).

These are just a few of the many different types of disasters examined to date. Overall, the epidemiological literature strongly suggests that various types of disasters contribute substantially to the development of PTSD.

AN ETIOLOGICAL MODEL OF PTSD

As with all psychiatric conditions, there is no single cause of PTSD. Yet, identification of the precipitating event or proximal cause is relatively straightforward. Keane and Barlow (*2*) proposed a triple vulnerability model of PTSD etiology based on theoretical descriptions of anxiety and fear. The three components of vulnerability are *(1)* preexisting psychological variables, *(2)* preexisting biological variables, and *(3)* the experience of a traumatic event. Unlike specific phobia, for which true alarms, false alarms, or (less often) simple transmission of information may develop into a specific phobic reaction, PTSD is hypothesized to emerge from one special chain of events. Intense basic emotions, such as true alarms (but also including rage or distress resulting from the overwhelming effects of traumatic events), lead to learned alarms. Learned alarms occur during exposure to situations that symbolize or resemble an aspect of the traumatic event, such as anniversaries of the trauma and thoughts, feelings, and memories of the event. As in any phobic reaction, the development of learned alarms can result in persistent avoidance of stimuli associated with the trauma. These are defining features of PTSD in the DSM-IV (*3*).

The pure experience of alarm or other intense emotions is not sufficient for the development of PTSD. Much as in other disorders, one must develop anxiety or

the sense that these events, including one's own emotional reactions to them, are proceeding in an unpredictable, uncontrollable manner. When negative affect emerges, anxious apprehension and preoccupation with the traumatic event begins, and collateral PTSD symptoms appear such as intrusive thoughts, emotional numbing and avoidance, and arousal.

The presence and severity of the various PTSD symptoms may be moderated to some extent by coping skills and social support. In PTSD, evidence already exists that these variables play a prominent role in determining whether the disorder develops.

RISK FACTORS FOR PTSD

Although many people experience traumatic events, the majority do not develop PTSD; individuals who experience the same or a similar stressor may or may not develop the disorder. The identification of the factors that might account for these differences can assist in our understanding of the etiology of the disorder and in the development of preventive approaches for people who are at highest risk for developing the disorder. Potential PTSD risk factors can be divided into three major categories: *(1)* preexisting factors inherent to the individual; *(2)* factors related to the traumatic event, including one's immediate response during the trauma; and *(3)* events that occur following the trauma. Although the last category may not be considered "causal" risk factors, such variables may help us better understand the commonly found severe, chronic cases of PTSD (*38*).

A Priori Factors

Discriminating the predisposition for developing PTSD from the predisposition for exposure to traumatic events represents a methodological and interpretive challenge. This issue is profoundly important for accurately identifying those variables that contribute to the development of PTSD. We begin to address this issue by examining preexisting factors that are related to trauma exposure and the development of PTSD.

Familial Psychopathology

Early fear conditioning research in animals suggests that there may be a genetic component associated with variability in sensitivity to environmental stress (*39*). Although studies of such a mechanism in humans do not exist, several twin and family studies have examined the heritability of PTSD.

Using the Vietnam Veteran Twin Registry, heritability factors were observed for exposure to combat (*40,41*) and PTSD symptoms (*41*). Despite an apparent genetic link for experiencing certain classes of traumatic events (*42,43*), genetic influences on trauma exposure do not appear to be shared with those influencing the development of PTSD when the trauma is combat (*44*). For noncombat interpersonal violence, there appears to be some association between genetic

influences on trauma exposure and genetic influences in the development of PTSD (*43*). Unfortunately, these studies used questionnaire data, thus deferring strong conclusions. In perhaps the most elegant study of genetic contributions to the development of PTSD, Orr and colleagues (*45*) used a wide variety of laboratory tasks (e.g., physiological reactivity, ERP) and standardized diagnostic tools in an attempt to elucidate the parameters that might underlie PTSD. They found little evidence of an inherited component for PTSD.

To help clarify conflicting results, Ozer, Best, Lipsey, and Weiss (*46*) recently applied meta-analysis to nine twin and family studies. They concluded that, overall, family history of psychopathology predicted a small but significant amount of variance of PTSD (average weighted effect size of $r = .17$). Interestingly, family history was more strongly related to PTSD when the traumatic experience involved noncombat, interpersonal violence than when it was combat or an accident. Similarly, in their earlier meta-analysis of 11 studies examining family history of psychopathology, Brewin, Andrews, and Valentine (*47*) found an average weighted effect size of $r = .13$, with the same effect found in military and civilian samples. These effect sizes are small, and it appears that unique environmental contributors to the development of PTSD are significantly stronger than any genetic influences measured to date (*48*).

The results of twin and family studies demonstrating genetic factors associated with PTSD suggest that genes influencing this risk may be identified. However, only a few genetic marker studies have been conducted, and there are, unfortunately, inconsistent results. Investigators examined allelic associations at the D2 dopamine receptor gene (DRD2), as it has previously been associated with other psychiatric disorders. Comings and colleagues (*49,50*) found associations between DRD2 and PTSD, but Gelernter et al. (*51*) did not. Thus, despite fairly consistent findings of a small association between familial psychopathology and the development of PTSD, the actual mechanism accounting for this association (either genetic or environmental) is far from clear.

Demographic Factors

Gender

Early epidemiological studies found relationships between demographic variables and exposure to trauma and between demographics and the development of PTSD. For example, we know that PTSD prevalence varies as a function of the type of trauma experienced; we also know that men are more likely to be exposed to a traumatic event during their lives; yet, we also know that women are more likely to develop PTSD. Some have hypothesized that this heightened rate of PTSD is a function of the types of events to which women are exposed (e.g., sexual assault). However, even when controlling for gender differences in types of traumas, women appear to remain at greater risk for developing PTSD than men (*12,52*). Similar gender differences in PTSD prevalence rates have been found in a variety of samples (e.g., Kosovar Albanians) (*53*), yet the average

weighted effect size across studies is small (r = .13) (*47*), and elucidating the mechanisms (i.e., psychological or biological) involved in these differences surely requires additional scientific study.

Age

Age at the time of a traumatic event is frequently seen as an important determinant of response, with the very young and the very old seen as carrying additional risk for disorder on exposure. Interestingly, among women in the NCS, no relationship existed between age and PTSD, and only a small positive relationship between age and exposure to traumatic events was found. In contrast, among men there was a strong positive correlation between age and PTSD, but this was due to increasing exposure to traumatic events over the life span (*12*). Across 29 studies, Brewin et al. (*46*) found that the average weighted effect of age on PTSD was minimal (r = .06).

Race

The data regarding race as a risk factor are complicated by great variability across data sets. For instance, in the NVVRS (*8*), PTSD prevalence rates were highest among Hispanic veterans and higher among African American veterans than the aggregate of Caucasian, Asian, and Native American veterans. These findings were largely, but not entirely, due to differences in rates of combat exposure. In the NCS, Kessler et al. (*12*) compared racial/ethnic groups and found that Caucasian and Hispanic participants reported higher rates of trauma exposure, whereas African American, Asian American, and Native American participants reported higher rates of PTSD following such exposures.

Using clinical interview data from the Hawaii Vietnam Veterans Project, Friedman, Schnurr, Sengupta, Holmes, and Ashcraft (*54*) reported that veterans of Japanese ancestry had lower odds of a current PTSD diagnosis compared with Caucasian veterans from the NVVRS data set, even after adjusting for age and combat exposure. Finally, in a sample of American Indian Vietnam veterans, Beals et al. (*55*) found that the American Indian sample had higher rates of current and lifetime PTSD than Caucasian participants of the NVVRS. Yet, when exposure to atrocities and violence was included in a multivariate model, ethnicity no longer predicted current or lifetime PTSD. Overall, racial/ethnic status does not appear to provide consistent differences in PTSD prevalence, and this variability across studies may be due to race/ethnicity not being a strong predictor of PTSD. Of note, Brewin et al.'s (*47*) meta-analysis found that the effect of racial/ethnic status as a predictor of PTSD was small (r = .05), with larger effect sizes found for socioeconomic status (SES; r = .14) and education (r = .10), variables often associated with racial/ethnic status in Western societies.

Marital Status

Few large-scale studies examined the relation between marital status and PTSD prevalence. In the NCS (*12*), marriage appeared to confer some level

of protection when one was exposed to a traumatic event, even when holding trauma exposure constant in the analyses. In contrast, in Breslau, Peterson, Poisson, Schultz, and Lucia's (*56*) Detroit trauma survey, marital status was not significantly associated with PTSD after controlling the type of trauma that the participants rated worst. As with other predictors of PTSD, the mechanisms for these associations are not yet known.

Prior Trauma and Life Adversity

Increasing evidence suggests that prior life trauma and cumulative adversity may increase risk of PTSD following a later trauma. Brewin et al. (*47*) found small effect sizes for childhood abuse ($r = .14$), other previous trauma ($r = .12$), and other adverse childhood factors ($r = .19$). Ozer et al. (*46*) also conducted a meta-analysis of prior trauma and found a small but significant effect size ($r = .17$). This effect did not vary based on the time elapsed since the trauma or if the trauma occurred in childhood or as an adult. However, the effect size did vary according to whether the prior trauma resulted from an accident, combat, or noncombat interpersonal violence ($r = .12$, $r = .18$, and $r = .27$, respectively). Interestingly, ethnic groups often differ in terms of distal and proximal life adversity, and variability in life adversity appears to decrease the relationship between ethnicity and PTSD, suggesting that race/ethnicity and other demographic differences may be markers for histories of unequal levels of life adversity (*14*).

Together, these studies suggest that prior trauma and life adversity may sensitize people to later traumas. With a fairly large degree of variability across study samples, it is possible that some individuals (e.g., nurses, firefighters) develop adaptive coping skills that may protect them from adverse responses to future traumas. Further, Bowman and Yehuda (*57*) suggested that if normal stress hormones (e.g., cortisol) are activated over prolonged periods of time, brain physiology and anatomy may be altered, leading to a depletion of hormones, resulting in an inadequate physiological response to later trauma exposure. Additional research on this as a possible biological mechanism is clearly needed.

Psychopathology Prior to the Trauma

Several studies have identified the prior existence of a psychiatric condition as a risk factor for the development of PTSD. For example, a recent prospective study of 2,949 Gulf War veterans indicated that PTSD symptoms more strongly predicted symptoms of depression over 2 years than vice versa (*58*). In both Brewin et al.'s (*47*) and Ozer et al.'s (*46*) meta-analyses, psychiatric history was found to confer only a small degree of risk for the development of PTSD ($r = .11$ and $.17$, respectively). However, when Ozer et al. examined a subset of studies that specifically examined prior depression, the degree of risk increased significantly ($r = .32$).

The presence of psychopathology such as an addictive disorder or conduct disorder may also lead to exposure to traumatic events themselves (*11,12*). This complex relationship is fundamental to our understanding of the effects of traumatic

events and PTSD. Careful assessment of the precipitating variables that contribute to a particular psychological condition can provide meaningful information about which condition to treat first when intervening with patients with multiple comorbid psychological disorders (*59*).

In an important prospective study of risk for developing PTSD, Bryant and Guthrie (*60*) measured maladaptive cognitions of student firefighters during their training and prior to deployment. Pretrauma catastrophic thinking strongly predicted (24% of the variance) the level of PTSD symptomatology 20 months after training was completed, indicating that a tendency to catastrophize about aversive events is a risk factor for the eventual development of PTSD.

The Traumatic Event Itself

Characteristics of the event itself, not surprisingly, predict the development and severity of PTSD. Most theoretical models suggest the presence of a dose-response model of PTSD that sees symptom severity as a function of traumatic event severity. Yet, operationally defining severity for various traumatic events is a complex task. Peritraumatic variables measured to date include a range of factors, such as physiology, affect, and cognitions that occur during the trauma, as well as particular aspects of the type of traumatic event.

Trauma Severity

Numerous studies defined the severity of a traumatic event in a variety of creative ways and found a significant association with PTSD severity. For example, across 49 studies Brewin et al. (*47*) found an average weighted effect size of $r = .23$ for the association between trauma severity and PTSD severity. In addition, Norris, Kaniasty, Conrad, Inman, and Murphy (*61*) suggested that severity is a strong predictor of PTSD cross-culturally.

Some studies examined proxies for traumatic event severity and then related them to severity of PTSD. For example, in survivors of the Oklahoma City bombing, suffering physical injuries was strongly related to PTSD symptoms 6 months later (*62*). In addition, among an Australian national sample, Rosenman (*63*) found that experiencing combat and rape or molestation were events that were especially likely to increase one's odds of developing PTSD. Finally, in their sample of Mexican adults, Norris et al. (*64*) found that exposure to violence in childhood was related to the chronicity of PTSD.

Perceived Life Threat and Peritraumatic Emotional Response

Across 12 studies, Ozer et al. (*46*) found a small-to-medium average weighted effect size of $r = .26$ for the strength of the relationship between perceived life threat and PTSD. Interestingly, the strength of the relationship was higher in studies with more time elapsed between the traumatic event and the assessment of PTSD; moreover, in a recent study perceived life threat was associated with maintenance of PTSD (*65*).

Ozer et al. (*46*) separately examined the relationship between peritraumatic emotional response (e.g., fear, helplessness, horror, guilt, shame) and PTSD and found a similar effect size across five studies ($r = .26$). Notably, Tucker, Pfefferbaum, Nixon, and Dickson (*62*) suggested that feeling nervous or afraid, expecting to die, and being upset by how others reacted during the Oklahoma City bombing explained 67% of the variance in PTSD symptoms 6 months later. Peritraumatic emotional distress also predicted the chronicity of PTSD symptoms (*64*). Interestingly, in their study of Somalia peacekeepers, Gray, Bolton, and Litz (*19*) found that negative perceptions of the mission were associated with immediate and chronic post-traumatic symptomatology.

Individual differences in peritraumatic emotional response may be due to differences in the meaning assigned to stressful events. Dunmore, Clark, and Ehlers (*66*) found that cognitions, such as negative beliefs about oneself and the world, measured shortly after the trauma were risk factors for PTSD severity 6 and 9 months later, even after controlling for the severity of the event. As reviewed by Bowman and Yehuda (*57*), several retrospective and cross-sectional studies found associations between cognitive beliefs and PTSD severity. Such cognitions included negative appraisals of trauma symptoms, low self-efficacy, and external locus of control.

Peritraumatic Dissociation

Peritraumatic dissociation includes an altered sense of time, "blanking out," and feeling disconnected from one's body (*67*). Across 16 studies, Ozer et al. (*46*) found a medium average weighted effect size ($r = .35$) for the strength of the relationship between peritraumatic dissociation and PTSD. Although the strength of this relationship did not differ according to the type of trauma experienced, it did vary as a function of the time elapsed between the trauma and symptom measurement, the type of sample, and the method of symptom assessment. Peritraumatic dissociation has also been found to play a role in the maintenance of PTSD (*65*).

To better understand this relationship, Gershuny, Cloitre, and Otto (*68*) examined the role that cognitions of panic (i.e., fears of death or losing control) might play in the association between peritraumatic dissociation and PTSD. They found that retrospectively reported fears of death and losing control during the trauma mediated the relationship between peritraumatic dissociation and PTSD. They suggest that peritraumatic dissociation might be part of the panic process, and the cognitive elements of panic may override the dissociative elements in their relative importance for PTSD development. These researchers noted that these cognitions may elicit dissociation.

Halligan, Michael, Clark, and Ehlers (*69*) studied survivors of a recent physical or sexual assault. They found that peritraumatic dissociation may lead to the disorganized trauma memories that are characteristic of PTSD. They also found that degree of disorganization in trauma memories and negative appraisals of that disorganization, as well as negative appraisals of intrusive memories, predicted PTSD symptoms over the course of 6 months; this was true even after controlling for actual

memory characteristics and depressive symptoms. Overall, cognitive variables such as cognitive processing during the trauma, trauma memory disorganization, persistent dissociation, and negative interpretations of trauma memories each predicted PTSD symptoms beyond objective and subjective measures of stressor severity.

Clearly, cognitive factors and their relationship to reports of peritraumatic dissociation are important areas of future inquiry. Yet, this work is centered on the accurate measurement of peritraumatic dissociation. For example, Marshall and Schell (*70*) found that individuals' recall of peritraumatic dissociation within days of the trauma differed markedly from their recall at 3- and 12-month follow-ups, and baseline dissociation did not predict subsequent PTSD symptom severity after controlling for initial symptom severity. Thus, it is possible that the cognitive factors inherent in Ehlers and Clark's (*71*) model and found in the Bryant and Guthrie (*60*) prospective study may ultimately prove most important to the prediction of PTSD.

Post-Trauma Factors

Very few studies actually include the assessment of post-trauma factors in terms of their contribution to the development and maintenance of PTSD. Social support is the one exception. Across 11 studies, Ozer et al. (*46*) found that perceived social support following the trauma event was associated with PTSD symptoms, with an average effect size falling in the small-to-medium range ($r = -.28$). This effect was strongest among studies in which more time elapsed between the trauma and the assessment. Similarly, Brewin et al. (*47*) found an average weighted effect size in the medium range ($r = -.40$) across 11 studies for the relationship between social support and PTSD symptoms. In both meta-analyses, this relationship was stronger among military/combat samples. Further, among Vietnam veterans, Schnurr, Lunney, and Sengupta (*65*) found that emotional sustenance and instrumental assistance, both at homecoming and currently, in addition to current levels of social support decreased the odds of lifetime PTSD; all variables except homecoming and current levels of assistance decreased the odds of chronic PTSD. Recently, King, Taft, King, Hammond, and Stone (*72*) examined the directionality of the relationship between PTSD and social support among a sample of Gulf War I veterans examined on multiple occasions. Using structural equation modeling and a cross-lagged panel design, they found that PTSD symptoms more strongly predicted subsequent social support than did social support predict subsequent PTSD symptoms. These findings support the idea that interpersonal problems associated with PTSD negatively influence one's support resources. Clearly, further work is needed to replicate these findings across samples and to examine the nature of this relationship.

MODELING THE PREDICTION OF PTSD

To begin to disentangle associations found in risk factor analyses, multivariate analytic methods such as structural equation modeling (SEM) are used to

simultaneously examine interrelationships among several predictors of PTSD. In a series of studies, Daniel and Lynda King and their colleagues applied SEM to a wide variety of theoretically driven variables in the NVVRS data set to understand the relationship of various factors to the prediction of PTSD following the Vietnam War. In the first of their series of studies, war zone stressor variables of atrocities/abusive violence, perceived threat, and malevolent war zone environment had direct effects on PTSD outcome, with malevolent environment exerting the largest effect. Traditional combat exposure had an indirect effect, influencing the development of PTSD primarily through the perceived threat that individuals reported (*73*).

Next, King, King, Foy, and Gudanowski (*74*) examined demographic variables, prewar factors (i.e., family environment, childhood antisocial behavior, maturity at entry to Vietnam, and prior trauma exposure) and war zone stressor variables as predictors of PTSD. In separate models for men and women, they found that war zone stressors were important contributors to PTSD, but additional variance was attributable to the prewar factors, particularly for men. Prior history of trauma and age at entry to Vietnam were important factors for men; for women, only prior trauma history contributed to the development of PTSD.

King, King, Fairbank, Keane, and Adams (*75*) then examined hardiness, structural social support, functional social support, and recent stressful life events as possible factors predicting PTSD. When examined together with the war zone stressor variables, all four variables had direct effects on PTSD development for men. For women, structural support did not predict PTSD development.

In the final report of their series of studies, King, King, Foy, Keane, and Fairbank (*76*) aggregated the pretrauma risk factors, the war zone stressors, and the post-trauma resilience-recovery variables to more comprehensively understand how these variables interrelate in the development of PTSD. Remarkably, these three categories of variables predicted 72% of the variability in PTSD among women and 70% among men. Prewar trauma exposure, exposure to abusive violence and life threat during the war, as well as postwar life stressors and functional social support were the strongest predictors of PTSD for women. Among the men, the key variables were the same as for women, plus younger age at entry to Vietnam, the malevolent war zone environment, and structural social support. While future studies should examine constitutional, physiological, and hereditary factors, the level of variance accounted for in these studies without the inclusion of biological variables is quite impressive.

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