CHAPTER

6

Posttraumatic Stress Disorder (PTSD) in Veterans

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INTRODUCTION

Since the introduction of posttraumatic stress disorder (PTSD) to the psychiatric nomenclature, most research has focused on methods to define, assess, and treat it. However, defining the disorder has been difficult and remains the subject of debate despite the fact that there is a current definition used for diagnosis and classification by the *Diagnostic and Statistical Manual of Mental Disorders*, 4th Edition (*DSM-IV-TR*; American Psychiatric Association [APA], 2000) and by the International Statistical Classification of Diseases and Related Health Problems, 10th Revision (ICD-10; World Health Organization [WHO], 1992). The identification threshold of PTSD is hard to pinpoint and has created a gap in the literature. Research on proper and specific treatments has proved difficult, particularly in combat and in peacekeeping veterans afflicted with PTSD (Shalev, Bonne, & Eth, 1996). According to Forbes, Creamer, Hawthorne, Allen, and McHugh (2003), many studies have reported significant residual symptomatology after treatment and considerable heterogeneity of response to treatment, with the majority of trauma victims (i.e., veterans) gaining little from most models of intervention.

Recent studies by Zlotnick, Franklin, and Zimmerman (2002) and Marshall et al. (2001), suggest a similar problem for using universal diagnoses to conceptualize operational definitions of stressors, stress reactions, and stress-related disorders. The varying qualities of the criteria and the symptoms that make up those criteria expose a "labile, polymorphic" disorder (Solomon, 1993, p. 104). This polymorphic disorder is characterized by high variability when defined in empirical studies and demonstrates rapid changes in temporal manifestations from one period to the next. The heart of the threshold debate lies in the true difficulty of establishing a one-size-fits-all diagnostic yardstick. As with most clinical settings, clinical social workers and military mental health professionals in most armed

forces use the standard functional gauge established by the *DSM-IV-TR* (APA, 2000) for the diagnosis and treatment of PTSD.

In this chapter the current *DSM* criteria for PTSD is discussed, followed by a history of the taxonomic evolution of the disorder. This is followed by a discussion of the debate concerning thresholds for diagnosis. Finally, a case example is provided that illustrates a combat veteran suffering from PTSD.

POSTTRAUMATIC STRESS: *DSM* DIAGNOSTIC CRITERIA

The inclusion of PTSD in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-III*; APA, 1980) marked the formal recognition of a specific syndrome etiologically linked to traumatic events. The diagnostic criteria for PTSD provided a standardized means for assessing the effects of trauma and were initially validated with Vietnam veterans (Zimering, Caddell, Fairbank, & Keane, 1993). With the publication of the *DSM-III* (APA, 1980), the common symptoms experienced by a wide variety of persons were first grouped under a single diagnostic category (van der Kolk, 1996). The *DSM-III* has since undergone three revisions (*DSM-III-R*, *DSM-IV*, and *DSM-IV-TR*). The version of PTSD in the *DSM-IV-TR* (APA, 2000) came about from various sources; first, from a review of the available literature relative to clinical phenomenology, epidemiology, and in relationship to other disorders. That version of the diagnosis was also derived from a series of multisite clinical and community trials (Saigh & Bremner, 1999).

As defined in the *DSV-IV-TR*, PTSD typically develops following exposure to an occurrence that is threatening or is perceived to be threatening to the well-being of oneself or another person; where symptoms are grouped into the following three clusters: reexperiencing the event (e.g., intrusive thoughts), avoidance and emotional numbing (e.g., restricted affect), and hyperarousal (e.g., marked sleep disturbance and hypervigilance). Additionally, to satisfy criteria for diagnosis, a person must have the following symptoms or experiences:

- Exposure to an actual or perceived threat.
- Feelings of intense fear or helplessness.
- At least one reexperiencing symptom.
- At least three avoidance and numbing symptoms.
- At least two hyperarousal symptoms.
- Symptoms must be present for more than one month.
- There must be significant distress or impairment in social, occupational, or other functioning.

(Asmundson, Coons, Taylor, & Katz, 2002)

Recent findings indicate that lifetime PTSD prevalence rates for the general U.S. population are 9.2%; having met criteria for full PTSD and 2.2% having subthreshold PTSD (not meeting the full criteria and defined as having the reexperiencing cluster (B criterion) plus another symptom cluster (C or D symptom criteria) per *DSM IV* (Yarvis, Bordnick, Spivey, & Pedlar, 2005). Thus, the overall rate exceeds 11% (Manzer, 2003), and in

certain at-risk groups (e.g., survivors of sexual assault, motor vehicle accidents, and combat), the rates can be substantially higher (APA, 2000). At this writing, veteran PTSD rates from the conflicts in Iraq and Afghanistan are thought to be 12.5% and are somewhat consistent with previous conflicts such as Vietnam at 15% and Desert Storm at 9% to 24% (Hoge et al., 2004; Kulka et al., 1990; Stretch et al., 1996). However, some studies show that two or more deployments could increase rates of PTSD in veterans (Yarvis et al., 2005; Yarvis & Schiess, 2008).

PTSD is unique among psychiatric diagnoses because of the great importance placed upon the etiological agent, that is, the traumatic stressor. In fact, one cannot make a PTSD diagnosis unless the client has actually met the "stressor criterion," which means that he or she has been exposed to a historical event that is considered traumatic. Clinical experience with the PTSD diagnosis has shown, however, that there are individual differences regarding the capacity to cope with catastrophic stress. Therefore, although some people exposed to traumatic events do not develop PTSD, others go on to develop the full-blown syndrome. Such observations have prompted the recognition that trauma, like pain, is not an external phenomenon that can be completely objectified. Like pain, the traumatic experience is filtered through cognitive and emotional processes before it can be appraised as an extreme threat. Because of individual differences in this appraisal process, different people appear to have different trauma thresholds, some more protected from and some more vulnerable to developing clinical symptoms after exposure to extremely stressful situations. Although there is currently a renewed interest in subjective aspects of traumatic exposure, it must be emphasized that events such as rape, torture, genocide, and severe war zone stress can be experienced as traumatic events by nearly everyone. However there are some unique features of PTSD in warrior populations.

PTSD in Combat Veterans

There is growing evidence that physical injury during deployment is associated with a higher prevalence of mental health issues postdeployment. Military personnel are returning from combat with different levels and types of injuries than in previous wars. This is partially due to more rapid and sophisticated medical response on the battlefield and to improved protective equipment such as Kevlar vests. These protect soldiers from mortal internal injuries but not from extremity trauma or concussive brain injuries. Recent studies detailing the most common injuries have found that approximately one half involved the head or neck. The great majority of injuries were due to explosions, and many involve more than one area of the body (polytrauma). Several studies from the Defense and Veteran's Brain Injury Center (DVBIC) of soldiers returning from Afghanistan or Iraq document the occurrence of traumatic brain injury (TBI) in many service personnel. Chapter 10 of this handbook discusses traumatic brain injury in the military in further detail.

A history of experiencing physical injury during deployment is associated with a higher prevalence of PTSD postdeployment. Taber and Hurley (2009) note a recent survey of soldiers following their return from deployment with findings showing that 9% of military personnel who had not been injured while deployed screened positive for PTSD. The rate was almost doubled (16%) in those reporting bodily injury during deployment. This rate is similar to that of past veteran studies assessing the increased risk for PTSD due

to combat-related injury, and others report an increased incidence of PTSD per number of injury mechanisms: 14% for one injury, 29% for two, and 51% for three or more (Yarvis et al., 2005). These results are consistent with the incidence of PTSD symptoms following significant orthopedic trauma in civilians. However, what is unique about veterans experiencing TBI is that blast-related loss of consciousness and memory deficits can also contribute to combat-related PTSD (Taber & Hurley, 2009) The nosological boundary between PTSD and TBI is at times elusive; however, studies continue to support current definitions of PTSD.

PTSD now exists as a formalized and clearly defined psychiatric diagnosis. The evolution of the diagnosis highlights the historical, political, and social momentum to normalize an individual's symptoms following adversity. During the formative years of PTSD as a diagnosis many theorists created models to explain stress and embarked on empirical studies that would explore the neurobiology of stress. The following is a terse look at the history and conceptualization of PTSD.

History of PTSD

The diagnosis of PTSD was created to fill a gap in the prevailing psychiatric understanding by acknowledging that extremely traumatic events could produce chronic clinical disorders in normal individuals. The idea that stress could contribute to psychiatric symptoms has its roots in the early disaster literature, and the notion that stress stimulates psychiatric illness in normal individuals predates formal nosologic classifications systems (Kaplan & Sadock, 1998). A chronology of the history behind PTSD is summarized in Figure 6.1.

The Evolution of Contemporary Theories of PTSD

Modern theories of PTSD began with the 19th-century concept of traumatic neurosis. From the middle of the century, railway accidents resulted in increased litigation by injured persons suffering from chronic pain and paralysis. The development of the specialty of neurology initially attributed these apparent neurological deficits to spinal cord injury. However, clinical and autopsy evidence began to accumulate, revealing little correspondence between tissue destruction (usually absent) and degree of disability. By 1885 it was recognized that "railway spine" was a functional or accepted medical disorder (Jones, 1995).

Charcot, a French neurologist, who demonstrated the onset of paralysis and other symptoms in "hysterical" women, suggested to Freud in 1893 a psychological etiology of hysteria (Laughlin, 1967). Charcot retained his belief in the prevailing idea of a neurological cause of hysteria and its manifestations. In 1889, Charcot's student, Oppenheim, coined the term *traumatic neurosis* to describe what he believed was a "molecular derangement" of nerve tissue (Robitscher, 1971). Initially Freud accepted this notion, postulating with Breuer in their work, *Studies in Hysteria*, an organic hypnoid state that made a person vulnerable to hysterical symptoms when stimulated by a traumatic event (Strachey & Freud, 1957). Freud held that the traumatic event in hysteria was sexual trauma. Later, he postulated that fantasized sexual trauma could produce hysteria (Freud, 1955a). Additionally, Freud attributed war neurosis to conflicts in ego structures (i.e., id, ego, and superego) and instinctual drives (libido and destrudo; the urges to create and destroy, respectively) (Freud, 1955b).

1666	Diary of Samuel Pepys captures his posttraumatic reactions to the Great Fire of London.
1812	Combat stress reactions among Swiss soldiers documented by Napoleon's field surgeons.
1865	Military surgeons document combat trauma as Da Costa's syndrome, nostalgia, or soldier's irritable heart.
1885	Charcot recognizes that "railway spine" was a functional disorder characterized by paralysis and chronic pain after railway accidents.
1889	Oppenheim coins the term "traumatic neurosis" to describe a "molecular derangement of nerve tissue."
1893	Freud suggests that paralysis and other neurotic symptoms in women were antecedents to "hysteria."
1919	Frederick Mott and Ernest Southard document the neurological and psychological effects of war. "Shell shock" described by T.W. Salmon.
1943	Adler describes "post-traumatic mental complications" in the survivors of the Boston Cocoanut Grove Fire.
1945	Grinker and Spiegel enumerates symptoms of "combat neuroses" in "returnees" from World War II.
1952	*DSM-I* with diagnosis of Gross Stress Reaction addressed the "severe physical demands or extreme stress such as in combat or in civilian catastrophe" (p. 40) in response to problems observed in survivors of World War II.
1962	Buchenwald Syndrome documented in concentration camp survivors.
1968	The APA's Committee on Nomenclature and Statistics omits gross stress reaction and introduces "transient situational disturbance" in the *DSM-II*.
1974	Burgess and Holmstrom introduce the idea of Rape Trauma Syndrome.
1975	Delayed Stress Syndrome introduced by Horowitz and Solomon after study of veterans who had served in Southeast Asia.
1980	Posttraumatic stress disorder established in the *DSM-III*.
1987	*DSM-III-R*.
1992	ICD 10 establishes different criteria for posttraumatic reaction than *DSM-III-R*.
1994	Acute stress disorder established along with *DSM-IV*.
2000	DESNOS—Disorder of Extreme Stress Not Otherwise Specified established in *DSM-IV-TR*.

Figure 6.1 Chronology of the historical roots of posttraumatic stress disorder

The idea that psychological trauma could produce apparent physical disabilities became generally recognized, especially with the appearance of numerous "shell shock" casualties during World War I (Salmon, 1929). The pendulum swung from considering traumatic neuroses as neurologically based to considering them to be of purely psychological causation. Eventually traumatic neurosis was mostly subsumed under conversion or somatoform disorders, but many individuals whose symptoms took the form of mood and behavioral disturbances did not fit this categorization (Jones, 1995).

Investigators who studied psychiatric casualties in World War II combat veterans variously labeled the constellation of symptoms they saw as "traumatic war neurosis" (Kardiner & Spiegel, 1947), "combat exhaustion" (Swank, 1949), and "operational fatigue" (Grinker et al., 1946, I., II.). Whatever the label, it is clear that these investigators were seeing a condition much like what we now recognize as PTSD. For example, Kardiner and Spiegel described a chronic syndrome that included preoccupation with the traumatic stressor, nightmares, irritability, increased startle responsiveness, a tendency to angry outbursts, and general impairment of functioning. Many investigators attempted to determine the etiology of the syndrome by asking veterans about their premilitary home life and postwar adjustment as well as their experiences in combat. Grinker et al. (1946) found that air crew officers with "operational fatigue" were more likely than combat controls to report premilitary anxieties and neurotic trends and reported having experienced more symptoms while in combat. Similar findings were seen in enlisted flying personnel (Grinker et al., 1946, II.). In addition, parental discord, broken homes, and parental alcoholism (factors not assessed in the officers) were more likely in the enlisted men with operational fatigue. Swank (1949) found some evidence of premilitary personality disturbance in his study of more than 2,000 cases of "combat exhaustion," but also found that amount of combat exposure (e.g., unit casualty rate) predicted symptoms. He counseled against viewing cases of combat exhaustion as resulting entirely from either premilitary personality instability or combat stressors.

Follow-up studies of World War II veterans continued into the 1950s, when veterans of the Korean conflict were included as a comparison group in some investigations; they rarely have been studied on their own. A theme of chronicity began to emerge. Futterman and Pumpian-Mindlin (1951) reported a 10% prevalence of traumatic war neurosis in a series of 200 psychiatric patients in 1950. They noted as significant the fact that many of the men had not sought treatment even 5 years after the war. Brill and Beebe (1955) reported continuing impairment of functioning in a sample of almost 1,500 veterans who had been discharged for "psychoneurosis" in World War II, almost half of whom were combat casualties. Archibald et al. (1962) found World War II combat veterans with "gross stress syndrome" to have severe problems such as increased startle, sleep disturbance, and avoidance of activities reminiscent of combat. A follow-up of these men (Archibald & Tuddenham, 1965) that included Korean conflict veterans showed the same symptom profile and relatively more symptoms than in noncombat psychiatric patients or in combat controls. The Archibald studies are notable for their use of the Minnesota Multiphasic Personality Inventory, MMPI (www.ptsdsupport.net/ptsd_MMPI-2-test.html), which showed combat veterans to be elevated on the depression, hysteria, and hypochondriasis scales and to differ from noncombat psychiatric patients on 47 items.

Dobbs and Wilson (1960) reported an interesting experiment that foreshadowed current laboratory studies on the psychophysiological correlates of PTSD. They exposed World War II veterans with combat-related psychiatric symptoms, healthy combat controls from World War II and the Korean conflict, and nonveterans to audiotapes of combat sounds. Combat controls showed greater pulse, respiration, and EEG responsiveness to the stimuli than did nonveterans. The combat-symptoms group was so unable to tolerate the sounds that physiological recordings could not be made. Dobbs and Wilson commented on the chronicity of combat fatigue and discussed its similarity to animal models of neurosis.

Few studies of World War II or Korean conflict veterans were performed in the 1970s, when articles about Vietnam veterans began to emerge. The exceptions seemed to focus on prisoners of war (POW). For example, Klonoff et al. (1976) reported on the MMPI profiles of POWs and Beebe (1975) reported on POWs physical and mental health. Most studies indicated that prisoners interned by the Japanese or Koreans were more symptomatic than those interned by the Germans.

After the formalization of PTSD as a diagnosis, isolated case studies began calling attention to the fact that some veterans of wars prior to Vietnam had PTSD. Van Dyke, Zilberg, & Mckinnon (1985) reported on a World War II combat veteran who had been well functioning until a medically necessary retirement at age 53, when he began to experience combat nightmares for the first time. Subsequent to further medical problems, the man developed PTSD.

For veterans of World War II, PTSD would have been viewed as traumatic war neurosis. It was seen as occurring among combatants in a combat area with a relatively high degree of frequency. Focal points would be on guilt about killing or assailing defenseless enemy personnel, either military or civilian, as an important factor in the precipitation of a traumatic war neurosis. Traumatic war neurosis was also seen as occurring in conjunction with physical injury. An overidealization of the pretraumatic history was viewed as occurring in cases of traumatic war neurosis. The monotonous repetition of the traumatic war experiences and combat dreams in cases of traumatic war neurosis was seen as being caused by the transformation of the world into a threatening place.

Veterans of Vietnam were often pathologized for both psychological and sociological reasons. PTSD was coined during that war, and it was noticed that its veterans exhibited more severe PTSD symptoms and higher scores on measures of depression, hostility, and psychoticism than veterans of the past. They also had more survivor guilt, impairment of work and response, derealization, and suicidal tendencies. Vietnam veterans had a greater lifetime frequency of panic disorder and an earlier age of onset for alcoholism. Like World War II veterans, Vietnam veterans experienced an emergence of psychiatric diagnoses after the war. Today the signs and symptoms of PTSD are described in terms of an anxiety disorder (APA, 1980, 1987, 1994, 2000).

PSYCHOSOCIAL MODELS OF POSTTRAUMATIC STRESS

Change in the conceptualization of PTSD has created research barriers. One notable discrepancy pertains to the nature and sources of normal and traumatic stress; specifically, how the construct of stress is conceptualized and operationalized (Bride, 2001; Danieli, 1998; Danieli, Rodley, & Weisaeth, 1996; Lamerson & Kelloway, 1996; Lewis, 2003). The following section provides a brief, general overview of explanatory theories or psychosocial models that are frequently associated with posttraumatic stress.

Traditional psychodynamic models of posttraumatic stress have been used to explain the process of stress transmission and traumatization. Psychodynamic theory examines the extent to which a traumatic incident influences a person's perception of self or others. This model seeks to characterize environmental factors that influence subsequent stress response, such as the importance of early childhood trauma and early psychological

conflicts (Krystal, 1988). Affective responses result when conscious or unconscious representations of oneself or others are altered by trauma, resulting in conflict ridden representations of self and others. Traumatized individuals then mobilize their psychological defenses to cope with these discrepant meanings and emotions. With time, trauma responses continue, and the individual may regress to using primitive defenses, including splitting (e.g., either thinking people are good or bad) and dissociation (Marmar et al., 1994). The trauma victim will lose control of the sense of self and have affective instability. Unresolved early conflicts may evoke traumas from earlier developmental periods associated with attachment and protection issues, thereby reactivating the traumatic effects. Consequently, maladaptive coping pushes the trauma victim to repeat maladaptive relationship patterns, resulting in unstable interpersonal relationships (Resick, 2001).

A variety of psychodynamically oriented treatment approaches have been provided to clients with PTSD, including individual and group therapy in different institutional settings. The problem with this model and its associated therapies is that no obvious thread ties together the different psychodynamic interventions, and no systematic rationale connects the type of therapy to PTSD (Rothbaum & Foa, 1996).

In the 20th century one of the most important influences in the study of stress was the work of Hans Seyle, a physiologist and physician. Seyle introduced the General Adaptation Syndrome model in 1936 showing in three phases what the alleged effects of stress has on the body. The earliest conception of stress was defined as a response. Response-based theories of stress stem from Seyle's early medical research, which noted that stress was a "nonspecific response of the body to any demand placed on it" (Seyle, 1956, p. 1). Seyle observed that any adverse event could trigger a biological response. Seyle's definition was guided by his medical research in which he categorized common responses to a variety of events termed *stressors*. The stress response, which he described as a general adaptation syndrome, was characterized by many of the precursors to the vegetative symptoms of affective disorders, such as melancholy, loss of motivation, loss of appetite, and loss of energy. Underlying his presuppositions was the assumption that stress symptoms were somewhat universal and could therefore be generalized to the nature of the stressor (Lewis, 2003). Seyle described the general adaptation syndrome as being comprised of three stages: alarm and mobilization, resistance, and exhaustion. In the first stage, the autonomic nervous system (ANS) and the endocrine system are engaged as a sympathetic reaction to the "fight-or-flight" response (Cannon, 1914). This sympathetic response causes muscle groups and respiratory and circulatory systems to give the body additional energy to respond under acute stress situations. During the second stage, the body's physiological responses continue as long as necessary to combat the effects of the stressors. In the third stage, the body is fatigued from operating at this sustained high-energy level and comes down from this level as stressors subside. Seyle's conceptualization of stress was a foundation for the incorporation of the biological model, the transactional theory of stress, and learning theories associated with stress as applied to models of posttraumatic stress. The evolution of these models will be described in the following section.

The biological model is not a new concept in the study of trauma. In 1918, Meakins and Wilson studied the physiological responses of shell-shocked veterans. Kardiner (1941) labeled traumatic stress as *physioneurosis*, identifying a connection between psychological and physiological responses to stress. When researchers began studying traumatic stress

and PTSD, they made the assumption that the biological processes would reflect normal responses to stress as later conceptualized in Seyle's (1956) General Adaptation Syndrome. However, while the normal stress response is an acute reaction that rapidly reverts to homeostasis, the biological responses to PTSD reflect chronic or potentially permanent changes with increasing reactivity over time.

Studies utilizing the biological model of PTSD made such conclusions possible. Biological studies have allowed for independent tests of the distinctness of the disorder and its similarity to neurobiological changes observed following traumatic events. For example, one of the major symptom clusters of PTSD is associated with physiological reactivity (i.e., hypervigilance). Thus, a number of studies have been conducted using arousal as a marker for PTSD, with research targeting automatic nervous system (ANS) activity, heart rate, respiration, and blood pressure in traumatized groups (mostly Vietnam veterans) and nontraumatized (control) groups. In early studies, a number of investigations did find that PTSD subjects had higher heart rates and states of arousal than non-PTSD groups (Adler, 1943; Meakins & Wilson, 1918; Salmon, 1929). However, more recent studies have found this not to be the case (Pittman, Orr, Forgue, Altman, & deJong, 1990; Prins, Kaloupek, & Keane, 1995; Shalev, Freedman, & Peri, 1998).

In reviewing studies of baseline arousal, Prins et al. (1995), observed that there were a number of methodological differences that could account for these findings. Further comparisons to other anxiety disorders found that there were no differences (Pittman et al., 1990). Other shortcomings of the biological model were noted. Prins et al. (1995) suggested that rather than a biological difference, these investigations were picking up greater anticipatory anxiety in subjects with PTSD or other forms of anxiety than healthy comparison subjects. Shalev et al. (1998) looked at several variables associated with PTSD in a controlled study. They confirmed Seyle's idea about increased heart rate; however, they observed that other biological responses to acute stress might condition an individual to adapt to the alarm response and that coronary responses did not serve to maintain the PTSD symptoms over time in the majority of PTSD cases.

Numerous transactional theories challenge Seyle's assumptions. For example, Lazarus and Folkman (1984) assert that response-based conceptualizations do not provide a theoretical foundation to derive potential stressors or describe PTSD, observing a lack of systematic ways to prospectively identify stressors. Cooper, Dewe, and O'Driscoll (2001) argued that theories emphasizing the stress response ignore important contextual and environmental factors, such as intensity, frequency, and duration of the stimulus, often discussed in the PTSD literature (Lerner, 1976, 1991; Robinson & Lee, 2000). Lazarus and Folkman (1984) believed that there is no objective way to predict psychological stress without reference to properties of the individual in their context. To date, however, there is no empirical investigation that supports the transactional stress model. Despite the lack of empirical evidence, Lazarus and Folkman's response-based definitions of stress do have heuristic value in the overall traumatic stress literature by describing taxonomies of common physiological (van der Kolk, 1996), psychological (Solomon, 1993), and behavioral stress reactions (Rothbaum & Foa, 1996).

The diathesis-stress model is an amalgam of all the models discussed. In the strictest sense, it purports that individuals are genetically vulnerable (diathesis), or have had early childhood experiences or brain abnormalities that render them vulnerable to psychological

trauma (stress) (Deykin & Buka, 1997). The attraction to genetic investigations of PTSD is compelling. This research attempts to explain individual predispositions to traumatization and different patterns of stress response. Although there is increasing genetic evidence that parents that suffer from PTSD render children vulnerable to PTSD (Danieli, 1998), without evidentiary support for intergenerational transmission there should be concerns about overestimating genetic factors associated with PTSD.

Although there are numerous psychosocial theories advancing and explaining the etiology of PTSD, behavioral models have the strongest evidentiary support and are more widely accepted in the scientific community than the other models (Bisson, Jenkins, Alexander & Bannister, 1997; Devilly & Spence, 1999). The behavioral model of PTSD, as posited by Keane, Zimering, and Caddell (1985), puts forth that traumatized individuals acquire conditioned fears of a wide variety of trauma-related stimuli. Subsequently, they avoid these stimuli. Through the processes of higher-order conditioning and stimulus generalization, the number of feared stimuli continues to increase even after the trauma has occurred. For example, if a veteran witnessed and smelled burning bodies after a firefight, they might experience a trauma reaction to grilled chicken at a barbeque and begin to fear or avoid previously unconditioned stimuli such as barbeques.

The cognitive model of PTSD articulated by Foa, Steketee, and Rothbaum (1989) suggests that PTSD occurs when the traumatic incident reinforces negative beliefs concerning a client's safety and competence. Individuals with PTSD subscribe to the cognitive distortion that the world is a dangerous place and therefore live their lives in constant fear. These individuals may also perceive themselves as incompetent and are unlikely to confront challenging situations. According to this model, PTSD progresses and/or is maintained when actual trauma or threat-related material receives preferential information processing by an individual over less-threatening information. Maladaptive processing of this kind leads to distorted ways of perceiving and comprehending their environment (Lilienfeld, Lynn, & Lohr, 2003).

Scientific research has revealed a number of efficacious treatment procedures for the amelioration of PTSD symptoms. Most are based on behavioral models and involve procedures that have withstood rigorous scientific experimentation. By the same token, there are a number of models that possess terminology associated with science, but are not based on empirically supported scientific theory. These models failed to withstand empirical scrutiny and determine the results of their effects. As a result, poorly supported models pose a threat to the understanding of the classification of PTSD and cloud understanding of the extent of impairment caused by PTSD.

Case Vignette: SSG Brown

SSG Brown is a 27-year-old male who has been honorably discharged from the U.S. Army after serving in active combat in Iraq. SSG Brown is Latino, married, has two children, and is employed by a leading national retailer and he attends college classes. He joined the Army 2 days after 9/11. He was a soldier for nearly 9 years and served in the initial ground invasion of Baghdad in 2003, when the military still had much to learn about the signature injuries of the war, PTSD and TBI.

In Iraq, he sustained head and shoulder wounds in combat, ending his service as a highly decorated member of a scout platoon. SSG Brown has received outpatient treatment for the symptoms of PTSD. Like many returning warriors, SSG Brown faced an uncertain future regarding work and lifestyle after separating from the military. He said, "I had no idea of what I was going to do or how I was going to fit in or live in the general population." This uncertainty extended to core values, such as family, with SSG Brown stating "my kids didn't even know who I was." There was, however, clearly a sense of vocational duty in that his military training and experience were exemplary and had application in such disciplines as police work. Yet, this opportunity of interest was elusive, if not impossible, due to physical injuries incurred in combat. In fact, today's warriors experience much higher survival rates (more than 90% in OIF/OEF versus 80% in the Persian Gulf and 67% in Vietnam), but SSG Brown was left with the existential question of competency in the world postinjury and wondering if he should have survived.

SSG Brown desired to return to the familiar in Iraq. He missed the strong sense of meaning and purpose found in the war zone, the ability to protect his battle buddies, and the excitement associated with combat environments. The absence of the combat rush led SSG Brown to attempt to create a similar reaction by, for example, riding motorcycles with fellow veterans at speeds in excess of 165 mph on deserted stretches of highway, or by numbing the desire through excessive alcohol consumption. Regardless, with SSG Brown there always existed a sense of responsibility and propriety, where, for example, he and his group adhered to posted speed limits in school zones "[We] didn't want to hurt any kids." With Brown one can see the complex interplay of rules adapted for combat that do not readily work in U.S. society upon return.

SSG Brown was treated for PTSD. His symptoms included flashbacks, night sweats, insomnia, agitation, and hypervigilance. Other conditions stemming from PTSD include drinking or drug problems, feelings of hopelessness, employment problems, and relationship problems. SSG Brown was at risk for developing a drinking problem and was having difficulty adjusting to his home life in the "new normal." Recent returnees show increased risks of new-onset heavy drinking and other alcohol-related problems among personnel deployed with combat exposures as compared to those nondeployed. These behaviors are often explained as efforts to self-soothe or self-medicate to offset the hypervigilance symptoms such as anger, increased startle response, and marked sleep disturbance.

Additionally, Latinos may be greater risk of PTSD and readjustment compounded by traditional ethnic issues. As a consequence this population may subsequently have greater readjustment concerns.

Reintegration is a process, not a single event. Carter and McGoldrick (2005) discuss family systems moving through time rather than in cycles so as a member of the family's trajectory of experience changes, so does the family's life course. Returning warriors often have difficulty being around children as can be seen in SSG Brown's situation. Veterans with PTSD symptoms have greater interpersonal problems (e.g., difficulties expressing intimacy, lack of sociability), and poorer marital and family relationships as well. SSG Brown described engaging in other risk behaviors such as driving fast. It is important to note that hypervigilance causes adaptations that might be functional in the combat zone but not in one's civilian life. SSG Brown described missing the "rush" associated with combat. In addition to combat-driving on the highway at home he also was engaging in fast-driving cars and motorcycles that had more to do with missing the rush than the response generalization of combat adaptations. Soldiers returning to garrison life after

extended combat deployments may have difficulty adjusting, and may seek the adrenaline "rush" they have grown accustomed to in combat environments. Social workers must be cognizant of the need to help warriors like SSG Brown adjust back to duties in the "rear" on post or base as well as manage the symptoms of PTSD. In addition the provider must address the complex interplay of the warrior ethos and sense of duty, with the warrior's role in the family and sense of purpose.

Case Vignette Discussion Questions:

1. Discuss what you know about the case and possible resources for SSG Brown and his family.
2. What are the societal, cultural, and community contexts of the case?
3. What are elements of SSG Brown's world that could serve to maintain his problems?
4. How would you address the maintenance of problems?
5. What else would you want to know about the case and about SSG Brown's situation to develop a better understanding of him?

CONCLUSION

In the simplest terms, PTSD is discussed through each model as a stress response. When evaluating each model's conceptualization of how symptoms move from the abstract to the observable, consider that PTSD includes processes that: (a) attenuate and perpetuate maladaptive and prolonged psychological stress responses within the individual, observed as anxiety, tension, and levels of distress; and (b) appear to maintain maladaptive psychobiological processes. These processes include hyperarousal, hypervigilance, startle responses, sleep disturbance, cognitive distortions, and affective instability ranging on a continuum from anger to depression to varied forms of anxiety. For each of the models pertaining to PTSD, it is important that the processes can be both observed and measured.

Studies relying on unproven models of traumatic stress and dependence on the current diagnostic classification of posttraumatic stress disorder led to neglected study of posttraumatic sequelae that fall short of full criteria for PTSD. In a military population it is important to look at the full continuum of trauma-related care. Recent research demonstrates in military samples that PTSD may be best understood as a war-induced trauma spectrum disorder where all aspects of trauma to include so-called subthreshold posttraumatic disorders should be considered when educating, treating, and researching veterans (Yarvis et al., 2005; Yarvis & Schiess, 2008).

This chapter reviews the history of how PTSD has been defined, which models of PTSD best capture the construct, and the prevalence of PTSD. This review focuses on empirically supported studies of PTSD in various populations and empirically supported models that best describe it. As detailed in this chapter, numerous models of stress attempted to capture the process of PTSD and define the boundary between normal and pathological trauma responses, including, but not limited to, psychodynamic, general adaptation syndrome, biological, transactional, and behavioral, and various combinations of these models. Despite past and current research, empirical support for clear definitions of PTSD is still lacking

(Lilienfeld et al., 2003). Currently, there appears to be more pseudoscience than science with regard to conceptualizing PTSD and the diagnostic cutoffs separating normal stress responses from maladaptive stress responses (Lewis, 2003; Lilienfeld et al., 2003; Yarvis et al., 2005; Yarvis & Schiess, 2008; Yarvis & Spivey, 2003). Indeed, there is no consensus on a model, and many models exist to explain PTSD in only one particular population, such as veterans (Bartone & Asler, 1998; Goodwin, 1987; Lamerson & Kelloway, 1996; Weisaeth, 1994). Solomon (1993) called PTSD a labile-polymorphic disorder because the diagnostic yardstick and reliance on categorical models of PTSD do not fully explain the experience of the veteran. In an extensive review of the trauma literature, no one explanatory model or classification of PTSD has been scientifically proven to be predictive of clinical impairment in veterans.

This chapter also explores the history of views of the etiology of PTSD. It presents several models that served to explain how PTSD evolves and affects warriors.

CHAPTER DISCUSSION QUESTIONS

1. What beliefs and perceptions did you have about PTSD and about veterans before reading this chapter, and how did they change after reading this chapter?
2. How does the veteran experience compare and contrast to the civilian experience in general and with trauma?
3. In what ways is PTSD considered to be unique among psychiatric diagnoses?

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