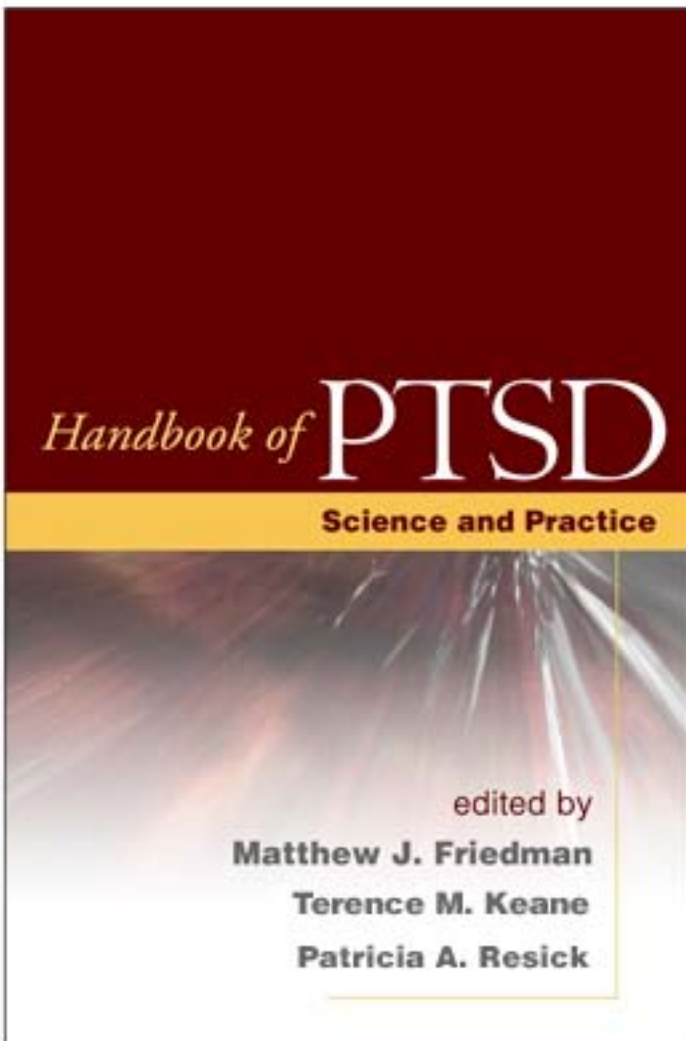




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Chapter 1

PTSD: Twenty-five years of progress and challenges

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Although symptoms reminiscent of posttraumatic stress disorder (PTSD) are illustrated in the world's great literature and have been noted following war or catastrophe over the centuries (Kilpatrick, Resnick, Freedy, et al., 1998; Saigh, 1992; Shay, 1994; See Chapter 2 by van der Kolk and Chapter 3 by Monson, Friedman & LaBash), the actual term posttraumatic stress disorder did not appear in our nosology until 1980. In the late 1800s, in the attempt to categorize psychological disorders, Kraepelin (1896, translated by Jablensky, 1985, p. 737) used the term "fright neurosis" (schreckneurose) to capture anxiety symptoms following accidents and injuries. After World War II and during the Korean War, the American Psychiatric Association produced the first Diagnostic and Statistical Manual of Mental Disorders (DSM-I, 1952), which included "gross stress reaction". This first DSM did not list the detailed criteria that we see today but did propose the diagnosis for people who were previously relatively normal, but who had symptoms resulting from their experiences with extreme stressors such as civilian catastrophe or combat. Strangely, at the height of the Vietnam War, the DSM II (American Psychiatric Association, 1968) was published and this category was eliminated. Some psychiatrists of that era assumed political motivations in the sudden disappearance of this diagnostic category (Bloom, 2000). According to Bloom, John Talbott, future president of the American Psychiatric Association, called for the return of the diagnostic category by the next year, 1969, because of his observations as a psychiatrist who had served in Vietnam, that there was no way to capture the symptoms he was observing with the current diagnostic system.

During the 1970s, a number of social movements in the US and around the world converged to bring attention to reactions following interpersonal violence as well as combat. The women's movement focused attention on sexual and physical assault of women from speak-outs and consciousness raising groups by the National Organization for Women. Laws were changed to reflect the understanding that abuse incidents within the family were crimes, of societal concern, and not merely family matters. Mandatory reporting of child abuse was enacted in all US states. Rape shield laws, marital rape laws and the legal recognition that rape could occur to boys and men as well as girls and women also changed attitudes and services provided. Landmark studies by Kemp and his colleagues (Gray, Cutler, Dean, & Kempe, 1977; Schmitt & Kempe, 1975) Burgess & Holmstrom (1973; 1974) and Walker (1979) resulted in descriptions of the child abuse syndrome, the rape trauma syndrome and the battered women's syndrome, respectively, and spawned a generation of research on those topics. The descriptions of responses to these forms of interpersonal traumas were much like those being described by the millions of Vietnam veterans who had returned from the war. As a result, when the revision of the DSM was considered, reactions to all traumatic events were pooled into one category.

In 1980, DSM-III (American Psychiatric Association, 1980) included posttraumatic stress disorder (PTSD) for the first time as an official diagnosis. PTSD, now classified as an anxiety disorder, had four criteria: 1) The existence of a recognizable stressor which would evoke distress in nearly anyone; 2) At least one of three types of reexperiencing symptoms (recurrent and intrusive recollections, recurrent dreams, or suddenly acting as if the traumatic event were recurring); 3) At least one indicator of numbing of responsiveness or reduced involvement in the world (diminished interest in activities, feeling of detachment and disinterest, or constricted affect); and 4) At least two of an array of other symptoms including hyperarousal or startle, sleep disturbance, survivor guilt, memory impairment or trouble concentrating, avoidance of activities reminiscent of the trauma, or intensification of symptoms when exposed to reminiscent events. Two subtypes were distinguished; acute, within the first six

months, and chronic or delayed with duration or onset occurring beyond six months. Interestingly, this earlier version of the DSM had separated numbing from effortful avoidance, a finding that has been established repeatedly, with factor analyses that have been conducted with the DSM-IV symptoms (American Psychiatric Association, 1994; King, Leskin, King, et al., 1998). Following the introduction of the diagnosis, there was a wave of prevalence studies to determine who develops the disorder and under what conditions, along with the development of valid and reliable assessment instruments for these criteria. Publications on treatment outcome studies began to appear by the mid-to late 1980s.

The introduction of PTSD into the DSM was not without controversy, which continues to this day. On one hand, clinicians, who had been seeking an appropriate nosological category for psychiatrically incapacitated holocaust survivors, rape victims, combat veterans, and other traumatized individuals, were delighted. They finally had a DSM-III diagnosis that validated the unique clinical phenomenology of their clientele. Recognition of the deleterious impact of traumatic stress provided a conceptual tool that transformed mental health practice and launched decades of research. For the first time, interest in the effects of trauma did not disappear with the end of a war.

On the other hand, critics of the diagnosis claimed and still claim: a] people have always had reactions to events and there is no need to pathologize it; b] that it is not a legitimate syndrome but a construct created by feminist and veteran special interest groups; c] that it serves a litigious rather than a clinical purpose because the explicit causal relationship between traumatic exposure and PTSD symptoms has opened the door to a multitude of frivolous lawsuits and disability claims in which the financial stakes are enormous; d] that verbal reports of both traumatic exposure and PTSD symptoms are unreliable; e] that traumatic memories are not valid; f] that the diagnosis is a Euro-American culture-bound syndrome that has no applicability to post-traumatic reactions within traditional cultures; and g] that it needlessly pathologizes the normal distress experienced by victims of abusive violence.

The next revision, the DSM-III-R (American Psychiatric Association, 1987) produced the criteria, which, for the most part, exist today. Five criteria were established; A) the stressor criterion; B) reexperiencing symptoms (at least one), C) avoidance symptoms (at least three), D) arousal symptoms (at least two), and E) duration criterion of one month. The acute designation was dropped from this iteration. The stressor criterion continued to define eligible stressors to be events “outside the range of usual human experience (i.e., outside the range of such common experiences as simple bereavement, chronic illness, business losses, and marital conflict)” and usually experienced with intense fear, terror, and helplessness (p. 247).

Avoidance symptoms included both efforts to avoid thoughts and reminders as well as numbing. However, it also included a sense of foreshortened future and amnesia for parts of the event. The arousal criterion included both direct (startle, hypervigilance and/or physiological reactivity upon stimulus exposure) or indirect (irritability/anger, sleep problems and/or difficulty concentrating,) indicators of physiological arousal. Once these reconfigured symptoms and clusters were established there was another wave of research that began to examine the individual symptoms, the clusters, and the configuration of the symptoms themselves. The committee assigned to conduct field trials for the DSM-IV were asked to focus on a few specific questions (Kilpatrick et al., 1998). One was whether Criterion A, the stressor criterion should be changed or dropped entirely. After the first wave of prevalence studies, it had become evident that “outside the range of normal experience”, was inaccurate, because most people experience at least one qualifying traumatic event in their lives and some events, while not frequent in one person’s life, are all too common across the population. Research asked whether people who experienced other stressful events such as divorce, the loss of a job, or the natural death of a loved one would also develop PTSD. They found that it made little difference whether there was a strict definition or nonrestrictive definition in the rates of PTSD; few people developed PTSD unless they had

experienced an extremely stressful life event. They did find more support for including a subjective distress component in Criterion A because of consistent findings that the level of panic, physiological arousal and dissociation present at the time of the event are predictors of later PTSD. Other questions posed in the field trial were with regard to placement of various symptoms and the threshold for Criterion C, the avoidance criterion (Kilpatrick, et al., 1998).

The DSM-IV was published in 1994 and slightly revised in 2000 (American Psychiatric Association, 1994; 2000). Several changes in PTSD diagnosis were formalized along with the introduction of a new disorder, Acute Stress Disorder. Despite some strong interest by the PTSD subcommittee to move the disorder out of the Anxiety Disorder group, the diagnosis remained where it was. Criterion A, now had two parts, 1) that the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others; and 2) the person's response involved intense fear helplessness, or horror. An item that had been listed under the arousal category (physiological reactivity on exposure to trauma cues) was moved to the reexperiencing criterion. The only other significant change was that the symptoms must cause significant distress or impairment in some realm of functioning (Criterion F).

The bigger development was the introduction of Acute Stress Disorder (ASD). This disorder emerged at the recommendation of the DSM-IV Dissociative Disorders Subcommittee with the observation that those people who had dissociative symptoms during or immediately after the traumatic event were most likely to develop PTSD. ASD was also introduced to bridge the diagnostic gap between the traumatic event and one month later when PTSD could be introduced. The criteria for ASD include the same stressor criterion as PTSD, and the presence of reexperiencing, avoidance, and arousal symptoms, although not in the 1, 3, 2 configuration required by PTSD. It differs, however, in that the person must experience at least three types of dissociative responses (amnesia, depersonalization, derealization, etc.). Like PTSD before it, ASD has proven to be controversial.

Criticisms of PTSD have not abated with the passage of time (Brewin, 2003; Rosen, 2004). Some have probably been exacerbated by concerns about the escalating number of PTSD disability claims recently filed by Canadian and American veterans. The cross-cultural argument currently rages within the context of Indonesian and Sri Lankan survivors of the 2005 Tsunami. These arguments also appear currently within the popular culture, due to increased attention from the mass media following the September 11th terrorist attacks, the South Asian Tsunami, Hurricane Katrina, and the wars in Iraq and Afghanistan. As a result, scientific debates about PTSD, previously restricted to professionals, have found their way into daily newspapers, popular magazines, radio talk shows and televised documentaries.

We believe that these criticisms demand a thoughtful and balanced response because they reflect concerns about PTSD that are shared both by the professional community and the public. Before we address these criticisms, however, it is necessary to review briefly the wealth of scientific information that has accrued since 1980 because of the new conceptual context provided by PTSD. Such research has not only transformed our understanding of how environmental events can alter psychological processes, brain function, and individual behavior but it has also generated new approaches to clinical treatment. Indeed, the translation of science into practice during the past twenty-five years is the major impact of the PTSD diagnosis.

Scientific Findings and Clinical Implications

Epidemiology

When PTSD was first operationalized in the DSM-III, exposure to traumatic stress was defined as “a catastrophic event beyond the range of normal human experience.” Epidemiological surveys, conducted since 1980 have shown otherwise (see Chapter 4 by Norris & Slone). In the US more than half of all adults (50% female and 60% male) will have been exposed to traumatic stress during the course of their lifetimes (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). In nations at war or subject to internal conflict, such as Algeria, Cambodia, Palestine, or the former Yugoslavia, traumatic exposure is much higher, ranging from 70-90% (de Jong, Komproe, van Ommeren, Masri, Mesfin, et al., 2001). Surveys of American military veterans have shown, as might be expected, high rates of exposure to warzone stress although prevalence estimates have varied in magnitude depending on the specific nature of each war and the war-specific demands of each deployment (Hoge, Castro, Messer, McGurk, Cotting, et al., 2004; Kang, Natanson, Mahan, Lee, & Murphy, 2003; Schlenger, Kulka, Fairbank, Hough, Jordon, et al., 1992).

One of the most robust findings in epidemiological research on PTSD is a dose response relationship between the severity of exposure to trauma and the onset of PTSD. Therefore, in the US, where lifetime trauma exposure is 50-60%, PTSD prevalence is 7.8% whereas in Algeria where trauma exposure is 92%, PTSD prevalence is 37.4% (de Jong et al., 2001; Kessler et al., 1995). This dose-response association has held up whether the traumatic experience has been sexual assault, war-zone exposure, a natural disaster or terrorist attack (Galea, Ahern, Resnick, Kilpatrick, Bucuvalas, et al., 2002; Kessler et al., 1995; Schlenger et al., 1992; Norris, Friedman, Watson, Byrne, Diaz, et al., 2002a; Norris, Friedman, & Watson, 2002b). Within this context, however, in the United States, the toxicity of interpersonal violence, such as in rape, is much higher than that of accidents; whereas wherein 45.9% female rape victims are likely to develop PTSD only 8.8% female accident survivors will develop the disorder (Kessler, et al, 1995; Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993). In developing nations, however, natural disasters are much more likely to produce PTSD because of the magnitude of resource loss associated with such exposure (Norris et al., 2002 a, b; see Chapter 4 by Norris & Slone).

It is also important to recognize that PTSD is not the only clinically significant consequence of traumatic exposure. Other psychiatric consequences include depression, other anxiety disorders and alcohol or drug abuse/dependency (Galea, et al., 2002; Shalev, Freedman, Peri, Brandes, Sahar, et al., 1998). Finally, there is accumulating evidence that when traumatized individuals develop PTSD, they are at greater risk to develop medical illnesses (Schnurr and Green, 2004; see Chapter 20 by Schnurr, Green & Kaltman).

The clinical implications of these data are clear. Given the fact that exposure to traumatic experiences occur in at least half of the adult American population, (and much more frequently within nations in conflict), mental health and medical clinicians should always take a trauma history as part of their routine intake. If there is a positive history of such exposure, the next step is to assess for the presence or absence of PTSD (see Chapter 15 by Keane, Brief, Pratt & Miller).

Risk Factors

Most people exposed to traumatic stress do not develop persistent PTSD. Even among female victims of rape, the most toxic traumatic experience, 54.1% will not exhibit full PTSD after three months whereas 91.2% female accident survivors never develop PTSD (Riggs, Rothbaum, & Foa, 1995; Rothbaum, Foa, Riggs, & Walsh, 1992). This means that most people have sufficient resilience to protect them from developing the disorder. Research on risk factors generally divides them into pre-traumatic, peri-traumatic, and post-traumatic factors (see Chapter 5 by Vogt, King, & King). Pre-traumatic factors include age, gender, previous trauma history, personal or family psychiatric history, educational level and the like. Although there has been a great deal of research identifying such factors,

they all have relatively low power to predict the likelihood of PTSD onset following traumatic exposure (Brewin, Andrews, & Valentine, 2000).

In addition to limited predictive power, it is not clear why certain pre-traumatic risk factors are associated with PTSD prevalence. For example, female rather than male gender predicts greater likelihood of developing PTSD following exposure to trauma. It is possible that this is just due to the greater likelihood of women to have experienced the events most likely to be associated with PTSD, such as child sexual abuse, rape or intimate partner violence (Kessler et al., 1995). However, such apparent gender differences may actually represent more complex phenomena such as gender differences in how trauma is conceptualized, potential gender-related differences in the PTSD construct itself, or how comorbid disorders contribute to this difference. Finally, there is evidence that whereas female gender predicts greater risk of PTSD, it may also predict more favorable responsiveness to PTSD treatment (see chapter 12 by Kimerling, Ouimette, & Weitlauf.).

With the recent characterization of the human genome, it won't be long before pre-traumatic factor research will include genotype assessment (see Chapter 11 by Segman, Shalev & Gelertner). Indeed, two studies on depression have shown a clear gene-environment interaction in which people exposed to three or more adverse events who also have two copies of the short form of the serotonin transporter gene are much likely to report depressive symptoms or suicidal behavior than those with two copies of the long form (Caspi, Sugden, Moffitt, et al., 2003; Kaufman, Yang, Douglas-Palumberi, et al., 2004). Given the nature of this gene-environment interaction, it would not be surprising if comparable results will be found with PTSD.

Peri-traumatic risk factors concern the nature of the traumatic experience, itself as well as one's reaction to it. The dose-response relationship between trauma exposure and PTSD onset, mentioned previously, applies here so that the severity of traumatic exposure predicts the likelihood of PTSD symptoms. Other peri-traumatic risk factors include exposure to atrocities, peri-traumatic dissociation, panic attacks, and other emotions. (Bernat, Ronfeldt, Calhoun, & Arias, 1998; Davis, Taylor, & Lurigio, 1996; Epstein, Saunders, & Kilpatrick, 1997; Galea et al., 2002; Ozer, Best, Lipsey, & Weiss, 2003).

The major *post*-traumatic factor is whether the traumatized person received social support followed by other posttraumatic stressors (Brewin et al., 2000). Indeed, social support appears to be the most important risk factor of all, receipt of which can protect trauma-exposed individuals from developing PTSD. Social support appears to be such a powerful factor that in one of the genetic depression studies mentioned above, social support significantly reduced the prevalence of depression among children with the greatest genetic vulnerability to adverse life events. (Kaufman et al., 2004).

Schnurr, Lunney, And Sengupta, (2004) have distinguished between risk factors for the onset of PTSD and those which predict maintenance of PTSD. Risk factors for persistence of PTSD emphasize current rather than past factors. They include current emotional sustenance, ongoing social support and recent adverse life events. The clinical significance of these findings is noteworthy. Assessment of risk factors, especially the strength and availability of social support, should be a routine part of any PTSD diagnostic interview. Furthermore, mobilization of social support, whenever possible, should be a part of any treatment plan. This applies whether the client has either chronic PTSD or an acute post-traumatic reaction and whether the clinician is providing treatment within a traditional clinical setting or an early intervention following a mass casualty within a public mental health context (see Chapter 25 by Watson).

Psychological Theory and Practice

PTSD invites explication in terms of classic experimental psychological theory to a far greater degree than any other psychiatric syndrome. It is one of the more interesting and unique disorders as

well, inasmuch as researchers, theorists, and clinicians have the rare opportunity to be present at the genesis of a disorder that began at a precise moment in time. Hence, there is a rich conceptual context within which to understand the disorder (see Chapter 3 by Monson, et al., and Chapter 6 by Cahill & Foa). Both conditioning and cognitive models have been proposed. Pavlovian fear conditioning, either as a unitary model (Kolb, 1989), or within the context of Mower's Two Factor Theory (Keane, Zimering, & Caddell, 1985; Keane & Barlow, 2002) has influenced research and treatment. Such models have inspired animal, psychophysiological and brain imagining research in addition to psychological investigations with clinical cohorts. Emotional Processing Theory (Foa and Kozak, 1985) has also been very influential. This theory proposes that pathological fear structures (Lang, 1977) activated by trauma exposure produce cognitive, behavioral and physiological anxiety. Finally, cognitive models derived from classical cognitive theory (Beck, 1979) postulate that it is the interpretation of the traumatic event, rather than the event itself that precipitates clinical symptoms.

A number of cognitive-behavioral therapies (CBT) have been derived from the aforementioned theories and tested with PTSD patients. What all CBT approaches have in common is that they elegantly translate theory into practice. As reviewed by Resick, Monson and Gutner (Chapter 17), the most successful treatments for PTSD are CBT approaches, most notably prolonged exposure, cognitive therapy, cognitive processing therapy and stress inoculation therapy. Indeed, all clinical practice guidelines for PTSD identify CBT as the treatment of choice (American Psychiatric Association, 2004; Foa, Keane, & Friedman, 2000; National Collaborating Centre for Mental Health, 2005; VA/DoD, 2004).

It is noteworthy that CBT has also been shown to be effective in treating acutely traumatized patients with Acute Stress Disorder within weeks of exposure to a traumatic event (see Chapter 16 by Litz & Maguen). This approach utilizes briefer versions of the prolonged exposure and cognitive restructuring protocols that have been so effective for chronic PTSD. Also CBT protocols have been modified so that they can be delivered through the internet or with the aid of virtual reality (See Chapter 23 by Welch and Rothbaum).

Although such progress is gratifying, it is important to recognize that there is still much work ahead. Almost all randomized clinical trials for PTSD have only tested components of CBT or single medications. Such studies have shown that approximately half of all CBT patients will achieve full remission of symptoms. That leaves another half who experience partial or no improvement after a course of CBT. Clearly there is room for new treatments, a better understanding of how to combine medications, combine medications with psychosocial treatments, and tests of whether these therapies work in real world settings. There are also questions about optimal strategies for specific phasing of treatments that may benefit those who typically drop-out of therapy early or do not benefit from a standard course of treatment. Indeed, future research will need to systematically investigate which treatment (or combination of treatments) is most effective for which PTSD patients under what conditions. Finally, it is imperative that we focus now on dissemination of evidence-based practices for the treatment of PTSD in clinical settings.

There has also been recent progress in developing clinical approaches for PTSD among children and adolescents (see Chapter 13 by Fairbank, Putnam & Harris and Chapter 18 by Saxe, McDonald & Ellis), thanks, in part, to establishment of the National Child Traumatic Stress Network in the United States. Progress with regard to older adults has lagged further behind (see Chapter 14 by Cook & Niederhe). In short, there is a real need for better understanding of the consequences of traumatic exposure and for developmentally-sensitive treatment approaches for people at either end of the life span.

Biological Theory and Practice

Thanks to advances in technology, biological research has progressed beyond animal models and neurohormonal assays to brain imaging and genetic research. It is noteworthy that a book on the neurobiology of PTSD published in 1995 had neither a chapter on brain imaging nor one on genetics, as in the present volume (Friedman, Charney, & Deutch 1995). The neurocircuitry that processes threatening stimuli centers on the amygdala with major reciprocal connections to the hypothalamus, hippocampus, locus coeruleus, raphe nuclei, mesolimbic, mesocortical and downstream autonomic systems. Major restraint on the amygdala is ordinarily exercised by the medial prefrontal cortex. In PTSD, amygdala activation is excessive while prefrontal cortical restraint is diminished (Charney, 2004; Davis & Whalen, 2001; see chapter 9 by Neumeister, Shannan, & Krystal and Chapter 10 by Southwick, Davis, Aikins, Rasmusson, Barron, et al.).

Many different neurohormones, neurotransmitters and neuropeptides play important roles in this stress-induced fear circuit. Thus, there are many potential opportunities to translate such basic knowledge into pharmacological practice. At present, two medications, both selective serotonin reuptake inhibitors (SSRIs) have received approval by the US Food and Drug Administration as indicated treatments for PTSD. There is growing research with other medications affecting different mechanisms, but few randomized clinical trials have been carried out so far. Given our growing knowledge in this area, and given the fact that only 30% patients receiving SSRIs achieve full remission, there is reason to expect that newer agents will prove more effective in the future (Friedman, 2002; see Chapter 19 by Friedman and Davidson).

Another significant translation of science into practice concerns the association between PTSD and physical illness (see Chapter 20 by Schnurr et al). Given the dysregulation of major neurohormonal and immunological systems among individuals with PTSD, it is perhaps not surprising that patients with PTSD are at greater risk for medical illness (Schnurr and Green, 2004) and for increased mortality due to cancer and cardiovascular illness (Boscorino, 2005). Again, as a mark of recent progress, such relationships were merely hypothesized in 1995 (Friedman & Schnurr, 1995). Now there is a compelling and rapidly growing database to verify these hypotheses.

Resilience, Prevention and Public Health

Two epidemiological findings have had a profound effect on our understanding about the risk of exposure to trauma and about the consequences of such exposure. First, as noted earlier (see Epidemiology) exposure to catastrophic stress is not unusual in the course of a lifetime. Second, most exposed individuals are resilient; they will not develop PTSD or some other disorder in the aftermath of traumatic events. Recent world events have thrust such scientific findings into the context of public policy and public health. They include terrorist attacks in New York, Madrid, Moscow, London and elsewhere, the Tsunami of 2004, Hurricane Katrina and many other man-made and natural disasters. The scientific question is: why are some individuals resilient while others develop PTSD following such catastrophic stressful experiences? The clinical question is: what can be done to fortify resilience among individuals who might otherwise be vulnerable to PTSD following traumatic exposure? And the public mental health question is: following mass casualties or large-scale disasters what can be done to prevent psychiatric morbidity in vulnerable populations?

From an historical perspective, these three questions are remarkable. It is only because of recent scientific progress that such questions could even be conceptualized. The new interest in resilience is emblematic both of maturity in the field and of technological advances. Resilience is a multidimensional construct including genetic, neurohormonal, cognitive, personality and social factors (see Chapter 24 by Layne, Warren, Watson, & Shalev). From the clinical and public health perspective,

the major question is: Can we teach vulnerable individuals to become more resilient? Our emerging understanding of the multidimensional mechanisms underlying resilience has given the term “stress inoculation,” a new meaning in the twenty-first century. This in turn has raised public policy and public mental health questions about the feasibility of preventing posttraumatic distress and PTSD in the population at large.

In the US, the September 11th terrorist attacks instigated a national initiative to understand the longitudinal course of psychological distress and psychiatric symptoms following exposure to mass casualties. In this regard civilian disaster mental health found much in common with military mental health. In both domains, it is recognized that most posttraumatic distress is a normal transient reaction from which complete recovery can be expected. A significant minority of both civilian and military traumatized individuals, however, will not recover but will go on to develop clinical problems that demand professional attention. Thus there are two trajectories following traumatic stress: normal transient distress or chronic clinical morbidity. The second trajectory requires treatment by traditional mental health professionals; indeed evidence-based early interventions have also been developed for acutely traumatized individuals (see Chapter 16 by Litz and Maguen). On the other hand, the first trajectory, affecting most of the population, demands a public mental health approach that fortifies resilience (see Ritchie, Watson, & Friedman, 2005; Chapter 25 by Watson, Gibson & Ruzek).

It is very exciting to consider the conceptual and clinical advances that have been made in this area during the last few years. Future research should produce a wide spectrum of scientific advances that will enhance our understanding of resilience (at the genetic, molecular, social, etc. levels) and thereby provide needed tools to foster prevention and facilitate recovery at both the individual and societal level.

Criticisms of the PTSD Construct

1. PTSD is not a legitimate diagnosis

We agree that men, women, and children have been exposed to traumatic events since pre-historic times. Indeed, there is a literary record of the adverse impact of such exposure recorded by Homer, Shakespeare, Dickens, Remarque, up to and including contemporary authors. A recent article using American Civil War archival data indicates that high rates of traumatic exposure were associated with high rates of physical and psychological morbidities (Pizarro, Silver & Prause, 2006). Attempts to record and understand such events and their consequences within a scientific or medical context are much more recent, dating back to the mid-nineteenth century. These latter observations have generated a number of somatic (e.g., soldier’s heart, effort syndrome, shell shock, neurocirculatory asthenia) and psychological (nostalgia, combat fatigue, traumatic neurosis) conceptual models (see Chapter 2 by van der Kolk and Chapter 3 by Monson, et al.). Reviewing some of the rich clinical (and literary) reports provided prior to 1980, it is clear that many authors were describing what would now be labeled PTSD. So what has been gained by this new conceptual and diagnostic construct?

It is evident that the explication and official adoption of PTSD as a DSM-III diagnosis ushered in a significant paradigm shift in mental health theory and practice. First, it highlighted the etiological importance of traumatic exposure as the precipitant of stress-induced alterations in cognition, emotion, brain function, and behavior. Dissemination of this model has provided a coherent context within which practitioners have been able to understand the pathway from traumatic exposure to clinical abnormalities. Second, the PTSD model has stimulated basic research (both human and animal) in which it has been possible to investigate the causal impact of extreme stress on molecular, hormonal, behavioral, and social expression. Recently, investigators have begun to explore gene-environment

interactions within this paradigm. Third, as noted earlier, the traumatic stress model has invited the elaboration of therapeutic strategies which have successfully ameliorated PTSD symptoms. Finally, PTSD was a unifying principle at a time when investigators were describing symptoms across a range of traumatic events such as child abuse syndrome, battered women's syndrome, rape trauma syndrome and Vietnam veterans syndrome. The important inductive leap of the DSM-III PTSD diagnosis was recognition that the reactions to these different types of events had more commonalities than differences. Subsequent research has shown that the same therapies can be used successfully across different types of traumas. These are all extraordinary advances that could not have occurred before post-traumatic distress and dysfunction was reconceptualized as PTSD.

Some objections to the PTSD diagnosis are historical. It is certainly possible that it would not have been included in DSM-III without strong support from veteran and feminist advocacy groups. Unlike depression, schizophrenia and other anxiety disorders, PTSD emerged from converging social movements rather than academic, clinical, or scientific initiatives. As a result, PTSD received an ambivalent, if not hostile, reception in many prominent psychiatric quarters when it was first introduced in 1980.

The response to this negative reception was an outpouring of research to rigorously test the legitimacy of PTSD as a diagnosis. This entire volume documents that early research and its more recent elaborations. The bottom line is that people who meet PTSD diagnostic criteria exhibit significant differences from nonaffected individuals as well as from individuals with depression, other anxiety disorders or other psychiatric disorders. Such research spans the spectrum from brain imaging to cognitive processing to clinical phenomenology to interpersonal dynamics. Factor analysis of the PTSD symptom clusters has generally validated the DSM-III-IV construct although there are questions about whether a four factor solution that splits avoidant from numbing symptoms is a better construct than the current three factor model (Friedman & Karam, in press). There can no longer be any doubt about the legitimacy of PTSD as a diagnosis.

2. PTSD needlessly pathologizes normal reactions to abusive violence

This criticism asserts that normal reactions to the abnormal conditions of political repression and torture (or interpersonal violence such as domestic violence) should be understood as appropriate coping responses to extremely stressful events. The argument further states that a psychiatric label such as PTSD removes such reactions from their appropriate socio-political-historical, context and thrusts them into the inappropriate domain of individual psychopathology. We reject this argument because it fails to acknowledge that some people will cope successfully with such events and manifest normal distress while others will exhibit clinically significant symptoms. This is another arena in which both public health and individual psychopathology models are applicable to different segments of a population exposed to the same traumatic stressor (see above: Resilience, Prevention, and Public Health).

As we have learned during the post 9/11 era of post-traumatic public mental health, most people exposed to severe stress have sufficient resilience to achieve full recovery. A significant minority, however, will develop acute and/or chronic psychiatric disorders among which PTSD is most prominent. The purpose of any medical diagnosis is to inform treatment decisions, not to "pathologize". Therefore, we reiterate that it is beneficial to detect PTSD among people exposed to traumatic stress in order to provide a treatment that may ameliorate their suffering.

3. PTSD is a culture-bound Euro-American Syndrome

The PTSD construct has been criticized from a cross-cultural perspective as an idiosyncratic Euro-American construct that fails to characterize the psychological impact of traumatic exposure in

traditional societies (Summerfield, 2004). We acknowledge that there may be culture-specific idioms of distress around the world that may do a better job describing the expression of post-traumatic distress in one ethnocultural context or another (Marsella, Friedman, Gerrity, & Scurfield, 1996; Green, Friedman, de Jong, et al, 2004). On the other hand, PTSD has been documented throughout the world (Green et al., 2004). De Jong et al. (2001) have documented high prevalence of PTSD in non-Western nations subjected to war or internal conflict such as Algeria, Cambodia, Palestine and the former Yugoslavia. An important recent report has a unique bearing on this issue because it compared people from widely different cultures who were exposed to a similar traumatic event. North and co-workers (North, Pfefferbaum, Narayanan, Thielman, McCoy, et al., 2005) compared Kenyan survivors of the bombing of the American embassy in Nairobi with American survivors of the bombing of the Federal Building in Oklahoma City. Both events were remarkably similar with respect to death, injury, destruction and other consequences. Similar too was PTSD prevalence among Africans and Americans exposed to these different traumatic events.

We agree with Osterman and de Jong (Chapter 21) that the time has come for the fields of mental health and anthropology to end the debate about the validity of the PTSD diagnosis. What is needed is a “culturally competent model of traumatic stress” that addresses how culture may differentially influence explanatory models of traumatic stress, how it is implicated in the appraisal of risk/protective factors, and how such understanding might contribute to diagnosis and treatment.

4. PTSD primarily serves a litigious rather than a clinical purpose

One of the reasons that PTSD has played so prominently in disability and legal claims is that it has been assumed that the traumatic event is causally related to PTSD symptom expression and hence, functional impairment. Although traumatic exposure is a necessary condition for the development of PTSD, it is not a sufficient condition. For example, the event most likely to result in PTSD is rape, yet only a minority of rape victims will be diagnosable with PTSD after a few months. Other risk factors play a role in symptom onset and duration as described above in the section on risk factors and in Chapter 5 (Vogt et al.). Despite the etiological complexity of PTSD onset, the stressor criterion is fundamental in personal injury litigation and in compensation and pension disability claims. This is because traumatic exposure establishes liability or responsibility for psychiatric sequelae in a context that puts PTSD in a category by itself with respect to other psychiatric diagnoses.

As noted by Sparr and Pitman (see Chapter 22) the geometric increase in PTSD claims in civil litigation is due to society’s growing recognition that traumatic exposure can have significant and long-lasting consequences. There is also concern that the redefinition of the stressor criterion in DSM-IV has opened the door to frivolous litigations in which PTSD-related damages or disabilities are dubious, at best. Another important factor that is driving much of this criticism is the sheer magnitude of money that has been awarded for successful personal injury suits or compensation and pension disability claims.

There is a significant difference, however, between challenging the utility of PTSD as a clinical diagnosis, and questioning the quality of forensic or disability evaluations performed by mental health professionals. We believe that minimal standards for such evaluations must be developed and enforced so that people who have a legitimate claim for compensation because of their PTSD are not penalized because of misuse or abuse of this diagnosis in civil litigation or in the disability claims process.

5. Traumatic memories are not valid

An important scientific question concerns the validity of traumatic memories. A review of the literature (see Chapter 7 by Brewin on memory and 8 De Prince and Freyd on dissociation) indicates

that trauma-related alterations in physiological arousal and information processing may affect how such input is encoded as a memory. Furthermore, the retrieval of such information may be affected by both current emotional state as well as by the presence of PTSD. Such appropriate concerns, notwithstanding, when external verification has been possible, it appears that most traumatic memories are appropriate representations of the stressful event in question. A particularly newsworthy manifestation of questions about the accuracy of trauma-related memories was sensationalized in the popular media as “the false memory syndrome”. The issue concerned memories of childhood sexual abuse that, once inaccessible, later became “recovered”. Some individuals who recovered such memories went on to sue the alleged perpetrator thereby transforming a complex, controversial and relatively obscure scientific and clinical question into a very public debate argued in the courtroom and mass media. It is now being documented that accurate traumatic memories may be lost and later recovered, although it is also clear that some recovered memories are not accurate. The veracity of any specific recovered memory must be judged on a case-by-case basis (see Roth and Friedman, 1998; Chapter 7 by Brewin).

6. Verbal reports are unreliable

A major theme throughout modern psychiatry has been the search for pathophysiological indicators that do not rely on verbal report. This is not just a challenge to PTSD assessment but to assessment of all DSM-IV diagnoses. We recognize the importance of this concern in some circles but see no reason why it should be cited as a specific problem for PTSD and not for any other psychiatric diagnosis.

There are several laboratory findings that hold promise as potential non-self-report assessment protocols for refining diagnostic precision (see chapters 9 and 10 by Neumeister et al. and Southwick et al., respectively). These include psychophysiological assessment with script-driven imagery or the startle response or utilization of pharmacological probes such as yohimbine or dexamethasone. At the moment, however, none have sufficient sensitivity or specificity for routine utilization in clinical practice.

In the meantime, we should not overlook the remarkable progress we have made in diagnostic assessment through development of structured clinical interviews and self-report instruments with excellent psychometric properties. In addition to improving diagnostic precision, such instruments have been utilized as dimensional measures to quantitate symptom severity and to monitor the effectiveness of therapeutic interventions (Wilson & Keane, 2004; see Chapter 15 by Keane, Brief, Pratt & Miller).

A remarkable recent study by Dohrenwend and colleagues (Dohrenwend, Turner, Turse, Adams, Koenen, et al, in press) indicates the high reliability of retrospective self-report data among a representative sample of 260 Vietnam Theater veterans who participated in the National Vietnam Veterans Readjustment Study (NVVRS). They compared verbal reports of combat exposure recorded by NVVRS investigators with a military historical measure consisting of military personnel files, military archival sources and historical accounts. Results showed a strong positive relationship between the documented military historical measure of exposure and the dichotomous verbal report-based assessment of high versus low/moderate war-zone stress previously constructed by NVVRS investigators. In short, this meticulous study indicates that verbal reports are usually quite reliable.

Summary

PTSD has been at the center of a number of controversies. Close examination of these contentious issues indicates that the arguments are generally not about PTSD per se, but about the

appropriateness of invoking PTSD within a controversial or adversarial context. Because the issue of causality or etiology is so clearly specified in PTSD, as in few other diagnoses, it is likely that it will continue to be applied or misapplied in a number of clinical, forensic and disability scenarios. An important goal is to respect the scientific evidence to assure appropriate applications in the future. It is also useful to recognize that, as in the recovered memory controversy, such contentious issues have spawned important basic and clinical research that has resulted in better mental health assessment and treatment.

The purpose of this volume is to document how far we have come during the past twenty-five years, so that we can generate forward momentum in the right directions. Translating the science concerning traumatic stress into better clinical practice is the underlying process. The goal is to understand the disorder, to optimize assessment and treatment for people who suffer from PTSD and other post-traumatic problems, and to identify processes that facilitate recovery from exposure to traumatic events.

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