Men, women, and children have been exposed to traumatic events since prehistoric times. Indeed, a literary record of the adverse impact of such exposure can be found in the work of poets, dramatists, and novelists such as Homer, Shakespeare, Dickens, Remarque, up to and including contemporary authors (Kilpatrick et al., 1998; Saigh, 1992; Shay, 1994). 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-19th century. For example, archival compensation and pension data from the U.S. Civil War indicates that high rates of traumatic exposure were associated with high rates of physical and psychological morbidities (Pizarro, Silver, & Prause, 2006). 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 Weisaeth, Chapter 3, and Monson, Friedman, & La Bash, Chapter 4, this volume, on trauma in psychiatry and psychology, respectively). Reviewing some of the rich clinical (and literary) reports provided prior to 1980 (e.g., DSM-III; see below), it is clear that many authors were describing what would now be labeled posttraumatic stress disorder (PTSD). So what has been gained by this conceptual and diagnostic construct?

It is evident that the explication and adoption of PTSD as an official diagnosis in the American Psychiatric Association’s (1980) third edition of its Diagnostic and Statistical Manual of Mental Disorders (DSM-III) 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. More recently, investigators have begun to explore gene–environment interactions and epigenetic expression within this paradigm. Third, as noted earlier, the traumatic stress model has invited the elaboration of therapeutic strategies that 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. All of these extraordinary advances could not have occurred before posttraumatic distress and dysfunction were reconceptualized as PTSD.

It is certainly possible that PTSD 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 test rigorously the legitimacy of PTSD as a diagnosis. This entire volume documents the current state of the art of such research. The bottom line is that people who meet PTSD diagnostic criteria exhibit significant differences from nonaffected individuals, as well as from individuals with depression, anxiety disorders, or other psychiatric disorders. Such research spans the spectrum from brain imaging to cognitive processing, to clinical phenomenology, to interpersonal dynamics. Analyses of the PTSD symptom clusters have validated the PTSD construct from DSM-III through DSM-5 (American Psychiatric Association, 2013). There can no longer be any doubt about the reliability, validity, and heuristic value of PTSD as a diagnosis.

The actual term “posttraumatic stress disorder” did not appear in our nosology until 1980. In the late 1800s, as part of his effort to categorize psychiatric 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 Conflict, the American Psychiatric Association produced the first Diagnostic and Statistical Manual of Mental Disorders (DSM-I; American Psychiatric Association, 1952), which included “gross stress reaction.” This first DSM did not list the detailed criteria that we see today but it did propose a transient diagnosis for people who were previously relatively normal, but had symptoms resulting from their experiences with extreme stressors such as civilian catastrophe or combat. Strangely, at the height of the Vietnam War, 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 (2000), 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 United States 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 and of societal concern, not merely family matters. Mandatory reporting of child abuse was enacted in all U.S. states. Rape shield laws, marital rape laws, and the legal recognition that rape could happen to boys and men, as well as girls and women, also changed attitudes and services provided. Landmark studies by Burgess and Holmstrom (1973, 1974), Kempe and his colleagues (Gray, Cutler, Dean, & Kempe, 1977; Schmitt & Kempe, 1975), and Walker (1979) resulted in descriptions of the child abuse syndrome, the rape trauma syndrome, and the battered woman 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 (Figley, 1985; Friedman, 1981). 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 included PTSD for the first time as an official diagnosis. PTSD was classified as an anxiety disorder that had four criteria: (1) the existence of a recognizable stressor that 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 6 months, and chronic or delayed, with duration or onset occurring beyond 6 months. Interestingly, this earlier version of the DSM had separated numbing from effortful avoidance, a finding that has been established repeatedly, with factor analyses of DSM-IV symptoms (American Psychiatric Association, 1994; Friedman, Resick, Bryant, & Brewin, 2011; King, Leskin, King, & Weathers, 1998; see Friedman & Resick, Chapter 2, this volume, on DSM-5 diagnostic criteria for PTSD). 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.

On the 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, the new diagnosis also engendered criticisms, some of which continue to the present (see below).

The next revision, DSM-III-R (American Psychiatric Association, 1987), produced the criteria that, for the most part, exist today. Six 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); (E) a duration criterion of 1 month; and (F) significant distress or functional impairment. 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 efforts to avoid thoughts and reminders, and 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, another wave of research began to examine the individual symptoms, the clusters, and the configuration of the symptoms themselves. The committee assigned to conduct field trials for DSM-IV was 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, although infrequent in one person’s life, are all too common across the population. Researchers 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 the definition in the rates of PTSD was strict or nonrestrictive; 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 levels of panic, physiological arousal, and dissociation present at the time of the event were predictors of later PTSD. Other questions posed in the field trial concerned placement of various symptoms and the threshold for criterion C, the avoidance criterion (Kilpatrick et al., 1998). The subcommittee was not allowed to examine or change any of the symptoms or clusters.

DSM-IV was published by the American Psychiatric Association in 1994 and slightly revised in 2000. Several changes in the PTSD diagnosis were formalized, along with the introduction of a new disorder, acute stress disorder (ASD). Despite some strong interest by the PTSD subcommittee to move the disorder out of the anxiety disorders group, the diagnosis remained where it was. Criterion A now had two parts: (1) 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 ASD, which emerged at the recommendation of the DSM-IV Dissociative Disorders Subcommittee, with the observation that 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 occurrence of traumatic event and 1 month later, when PTSD could be introduced. 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. ASD differs, however, in that the person must
also experience at least three types of dissociative responses (amnesia, depersonalization, derealization, etc.). Like PTSD before it, ASD has proven to be controversial.

Most recently, PTSD (and ASD) diagnostic criteria have been revised in DSM-5. A detailed discussion of the DSM-5 process and revisions can be found in Friedman and Resick (Chapter 2, this volume). To briefly summarize:

1. PTSD is no longer categorized as an “anxiety disorder” but is now in a new category, “trauma and stressor-related disorders,” alongside acute stress disorder, adjustment disorders, and other related diagnoses.
2. The PTSD construct has been expanded to include other clinical phenotypes; in addition to the DSM-III/IV fear-based anxiety disorder, PTSD now includes anhedonic/dysphoric and externalizing phenotypes.
3. The latent structure of PTSD now comprises four (rather than DSM-IV’s three) symptom clusters (i.e., intrusion, avoidance, negative mood and cognitions, and arousal and reactivity).
4. DSM-IV’s criterion A2 (i.e., responding to the traumatic event with “fear, helplessness of horror”) has been eliminated.
5. DSM-IV’s 17 symptoms have been retained (although sometimes revised or clarified), and three new symptoms have been added.
6. Two new subtypes have been added, a dissociative subtype for people with derealization or depersonalization, along with the full PTSD syndrome and a preschool subtype for children 6 years and younger (see Friedman & Resick, Chapter 2, and Lanius et al., Chapter 13, this volume).

With regard to ASD, it is no longer necessary for traumatized individuals to exhibit any dissociative symptoms. Nine (out of 14) symptoms are needed for the diagnosis (Bryant, Friedman, Spiegel, Ursano, & Strain, 2011). Given recognition that acute post-traumatic reactions may comprise a variety of reactions, individuals who meet DSM-5 ASD diagnostic criteria may or may not exhibit dissociative symptoms. Research has shown that the presence or absence of dissociative symptoms does not affect the severity, morbidity, or longitudinal course of people with ASD (see Bryant, Chapter 22, this volume).

We begin by briefly reviewing 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 since DSM-III is the major impact of the PTSD diagnosis. Then we consider questions, controversies, and challenges regarding PTSD.

**Scientific Findings and Clinical Implications**

**Epidemiology**

When PTSD was first operationalized in 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. More than half of all U.S. adults (50% female and 60% male) are 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 to 90% (de Jong et al., 2001). Surveys of U.S. military veterans have shown, as might be expected, high rates of exposure to war-zone 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 et al., 2004; Kang, Natelson, Mahan, Lee, & Murphy, 2003; Schlenger 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 United States, 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 a terrorist attack (Galea et al., 2002; Kessler et al., 1995; Norris, Friedman, & Watson, 2002a; Norris et al., 2002b; Schlenger et al., 1992). Within this context, however, in the United States, the toxicity of interpersonal violence, such as that in rape, is much higher than that in accidents; whereas 45.9% of female rape victims are likely to develop PTSD, only 8.8% of female assault survivors 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., 2002a, 2002b; see Norris & Slone, Chapter 6, and Fairbank, Putnam, & Harris, Chapter 7, this volume, on the epidemiology of PTSD among adults and children, respectively).

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 et al., 1998). Finally, accumulating evidence indicates that when traumatized individuals develop PTSD, they are at greater risk to develop medical illnesses (Schnurr & Green, 2004; Schnurr, Wachen, Green, & Kaltman, Chapter 28, this volume). The clinical implications of these data are clear. Given that exposure to traumatic experiences occurs in at least half of the U.S. adult 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 Reardon, Brief, Miller, & Keane, Chapter 20, and Briggs, Nooner, & Amaya-Jackson, Chapter 21, this volume, on assessment of PTSD in adults and children).

**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% do not exhibit full PTSD after 3 months, and 78.8% of female assault survivors do not have PTSD after 3 months (Riggs, Rothbaum, & Foa, 1995; Rothbaum, Foa, Riggs, Murdock, & 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 pretraumatic, peritraumatic, and posttraumatic factors (see Vogt, King, & King, Chapter 8, this volume, on risk factors). Pretraumatic factors include age, gender, previous trauma history, personal or family psychiatric history, educational level, genotype, and the like.
Although a great deal of research has identified such factors, 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 pretraumatic 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 women's greater likelihood of having 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, the social context in which gender differences are expressed, or how comorbid disorders contribute to this difference (see Kimerling, Weitlauf, Iverson, Karpenko, & Jain, Chapter 17, this volume, on gender issues in PTSD). Finally, there is evidence that whereas female gender predicts greater risk of PTSD, it may also predict more favorable responsivity to treatment.

With the recent characterization of the human genome, it will not be long before pretraumatic factor research includes genotype assessment. Indeed, recent studies have identified a number of candidate genes that are being investigated regarding vulnerability versus resilience to PTSD following exposure to traumatic events. Given that genotype, epigenetic methylation, and gene expression differences likely accompany the development of psychopathologies such as PTSD, research incorporating all three forms of genetic information from the same traumatized individuals is needed. (See Koenen et al., Chapter 16, this volume, on gene x environment interactions.)

Peritraumatic 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 peritraumatic risk factors include exposure to atrocities, peritraumatic 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 posttraumatic factor is whether the traumatized person received social support, followed by other posttraumatic stressors (Brewin et al., 2000). Indeed, receipt of social support, which appears to be the most important protective factor of all, 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 earlier, social support significantly reduced the prevalence of depression among children with the greatest genetic vulnerability to adverse life events (Kaufman et al., 2004).

Schnittur, Lunney, and Sengupta (2004) have distinguished between risk factors for the onset of PTSD and those that predict maintenance of PTSD. Risk factors for persistence of PTSD emphasize current rather than past factors and 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 posttraumatic reaction, and whether the clinician is providing treatment within a traditional clinical setting or an early intervention following a
Historical Overview

mass casualty within a public mental health context (see Watson, Gibson, & Ruzek, Chapter 34, this volume, on prevention and public health).

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 Monson et al., Chapter 4, this volume, on the psychological history, and Gillihan, Cahill, & Foa, Chapter 9, this volume, on the psychological theories of PTSD). Both conditioning and cognitive models have been proposed. Pavlovian fear conditioning, either as a unitary model (Kolb, 1989) or within the context of Mowrer’s two-factor theory (Keane & Barlow, 2002; Keane, Zimering, & Caddell, 1985) has influenced research and treatment. Such models have inspired animal, psychophysiological, and brain imaging research, in addition to psychological investigations with clinical cohorts. Emotional processing theory (Foa & Kozak, 1986) 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, Rush, Shaw, & Emery, 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 (CBTs) have been derived from the aforementioned theories and tested with patients with PTSD. What all CBT approaches have in common is that they elegantly translate theory into practice. The most successful treatments for PTSD are CBT approaches, most notably prolonged exposure, cognitive therapy, cognitive processing therapy, and stress inoculation therapy. Several chapters in this volume review the empirical evidence supporting CBT approaches for adults (Resick, Monson, Gutner, & Maslej, Chapter 23), children and adolescents (Cohen & Mannarino, Chapter 24), couples and families (Monson, Macdonald, Fredman, Schumm, & Taft, Chapter 25) and in group formats (Beck & Sloan, Chapter 26). Indeed, all clinical practice guidelines for PTSD identify CBT as the treatment of choice (American Psychiatric Association, 2004; Australian Centre for Posttraumatic Mental Health, 2007; Foa, Keane, Friedman, & Cohen, 2009; Forbes et al., 2010; National Collaborating Centre for Mental Health, 2005; U.S. Department of Veterans Affairs/Department of Defense [VA/DoD], Clinical Practice Guideline Working Group, 2010).

It is noteworthy that CBT has also been shown to be effective in treating acutely traumatized patients with ASD within weeks of exposure to a traumatic event (see Bryant, Chapter 22, this volume). 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 (see Boasso, Kadesch, & Litz, Chapter 31, this volume), or remotely via telehealth or mobile phone applications (see Morland, Hoffman, Greene, & Rosen, Chapter 32, this volume).

In addition to CBT, eye movement desensitization and reprocessing (EMDR) has emerged as a first-line therapy for PTSD. Although there are strong disagreements about the mechanism of action of this approach, especially with regard to the importance of eye movements, the evidence regarding EMDR’s efficacy is strong enough for
it to be classified as a first-line treatment for PTSD in recent clinical practice guidelines (see Resick et al., Chapter 23, this volume, on psychosocial treatments).

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 achieve full remission of symptoms, leaving another half that 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, combined medication and psychosocial treatment, and tests of whether these therapies work in real-world settings. Also, questions about optimal strategies for specific phasing of treatments 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 investigate systematically which treatment (or combination of treatments) is most effective for which patients with PTSD under what conditions. Finally, it is imperative that we utilize the most advanced technologies for dissemination of evidence-based practices for the treatment of PTSD in clinical settings (see Ruzek & Landes, Chapter 35, this volume, on dissemination of treatments to implement the best clinical practices).

There has also been recent progress in developing clinical approaches for PTSD among children and adolescents (see Brown, Becker-Weidman, & Saxe, Chapter 18, this volume), thanks in part to establishment of the National Child Traumatic Stress Network in the United States. Progress with regard to older adults has lagged behind (see Cook, Spiro, & Kaloupek, Chapter 19, this volume). 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 lifespan.

**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 chapters on neither brain imaging nor genetics, as does this 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, and raphe nuclei; and 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, whereas prefrontal cortical restraint is diminished. Furthermore there have been great advances in our understanding of the neuroplasticity that mediates both posttraumatic psychopathology and recovery from PTSD (see Nash, Galatzer-Levy, Krystal, Duman, & Neumeister, Chapter 14, this volume, on neurocircuitry and neuroplasticity).

Many different neurohormones, neurotransmitters, and neuropeptides play important roles in this stress-induced fear circuit. (See Rasmusson & Shalev, Chapter 15, this volume, on neurobiological alterations associated with PTSD.) 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 U.S. Food and Drug Administration (FDA) approval as indicated treatments for PTSD. There is growing research with other medications affecting different mechanisms, but many more randomized clinical trials are needed. Given our growing knowledge in this area, and the fact that only 30% of patients receiving SSRIs achieve
full remission, there is reason to expect that newer agents will prove more effective in the future (see Friedman & Davidson, Chapter 27, this volume).

Another significant translation of science into practice concerns the association between PTSD and physical illness (see Schnurr et al., Chapter 28, this volume). Given the dysregulation of major neurohormonal and immunological systems in individuals with PTSD, it is perhaps not surprising that patients with PTSD are at greater risk for medical illness (Schnurr & Green, 2004) and for increased mortality due to cancer and cardiovascular illness (Boscarino, 2006). Again, as a mark of recent progress, in 1995 such relationships were merely hypothesized (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 do 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, including terrorist attacks in New York, Madrid, Moscow, London, Boston, and elsewhere; the tsunami of 2005; 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 a historical perspective, these three questions are remarkable. Only because of recent scientific progress can such questions even be conceptualized. The new interest in resilience is emblematic of both maturity in the field and technological advances. Resilience is a multidimensional construct that includes genetic, neurohormonal, cognitive, personality, and social factors (see Southwick, Douglas-Palumberi, & Pietrzak, Chapter 33, this volume, on resilience). From the clinical and public health perspective, the major question is: Can we teach vulnerable individuals to become more resilient? Our emergent understanding of the multidimensional mechanisms underlying resilience has given the term “stress inoculation” a new meaning in the 21st 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 (see Watson et al., Chapter 34, this volume, on public health and prevention).

In the United States, the September 11, 2001, 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, do not recover but go on to develop clinical problems that demand professional attention. Thus, there are several trajectories following traumatic stress: normal transient distress, early onset PTSD followed by recovery, or chronic clinical morbidity. On the one hand, the second and third trajectories
require treatment by traditional mental health professionals; indeed, evidence-based early interventions have also been developed for acutely traumatized individuals (see Bryant, Chapter 22, this volume). On the other hand, the first trajectory, affecting most of the population, demands a public mental health approach that fortifies resilience (see Southwick et al., Chapter 33, and Watson et al., Chapter 34, this volume, on resilience and prevention, respectively).

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 genetic, molecular, social, etc., levels), thereby providing needed tools to foster prevention and facilitate recovery at both individual and societal levels.

Criticisms of the PTSD Construct

Criticisms of PTSD as a diagnosis 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 veterans and civilians. The cross-cultural argument currently rages within the context of natural disasters (e.g., the 2005 Asian tsunami) or large-scale terrorist attacks (e.g., the bloodshed in Mumbai in 2011) that occurred in non-European American settings. These arguments also appear currently within the popular culture, due to increased attention from the mass media to ongoing terrorist attacks, natural disasters, wars, and industrial accidents around the world. 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. Critics of the diagnosis claim that (1) people have always had strong emotional reactions to stressful events, and there is no need to pathologize them; (2) PTSD serves a litigious rather than a clinical purpose; (3) the diagnosis is a European American culture-bound syndrome that has no applicability to posttraumatic reactions within traditional cultures; (4) verbal reports of both traumatic exposure and PTSD symptoms are unreliable; and (5) traumatic memories are not valid. We believe that these criticisms demand a thoughtful and balanced response because they reflect concerns about PTSD that are shared by both the professional community and the public.

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; e.g., 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 sociopolitical–historical context and thrusts them into the inappropriate domain of individual psychopathology. We reject this argument because it fails to acknowledge that some people cope successfully with such events and manifest normal distress, whereas others exhibit clinically significant symptoms. This is another area in which both public health and individual psychopathology models are applicable to different segments of a population exposed to the same traumatic stressor (see Southwick et al., Chapter 33, and Watson et al., Chapter 34, this volume, on resilience and prevention, and public health, respectively).
As we have learned during the post–9/11 era of posttraumatic public mental health, most people exposed to severe stress have sufficient resilience to achieve full recovery. A significant minority, however, develop acute and/or chronic psychiatric disorders, among which PTSD is most prominent. People who meet PTSD diagnostic criteria differ from nonaffected individuals with regard to symptom severity, chronicity, functional impairment, suicidal behavior, and (both psychiatric and medical) comorbidity. 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 to provide an effective treatment that may both ameliorate their suffering and prevent future adverse consequences.

**PTSD Is a Culture-Bound European American Syndrome**

The PTSD construct has been criticized from a cross-cultural perspective as an idiosyncratic European 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 posttraumatic distress in one ethnocultural context or another (Green et al., 2003; Marsella, Friedman, Gerrity, & Scurfield, 1996). On the other hand, PTSD has been documented throughout the world (Green et al., 2003) and the cross-cultural validity of PTSD has been demonstrated conclusively (Hinton & Lewis-Fernández, 2011; see Lewis-Fernández, Hinton, & Marques, Chapter 29, this volume, on culture and PTSD). de Jong and colleagues (2001) documented the 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 report, with a unique bearing on this issue, compared people from widely different cultures who were exposed to a similar traumatic event. North and colleagues (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. Finally, a very recent randomized clinical trial demonstrates the cross-cultural utility of the PTSD diagnosis, as well as the generalizability of evidence-based PTSD treatment in a non-Western arena. Female Congolese survivors of sexual violence who received group sessions of cognitive processing therapy exhibited marked reduction of PTSD symptoms and significant improvement in functional status compared to a comparison group that received supportive therapy. This improvement was sustained at the 6-month follow-up assessment (Bass et al., 2013).

**PTSD Primarily Serves a Litigious Rather Than a Clinical Purpose**

One of the reasons PTSD has played such a prominent role 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 are diagnosable with PTSD after a few months. Other risk factors play a role in symptom onset and duration, as described earlier in the section on risk factors (see
King, King, Kaiser, & Lee, Chapter 5, this volume). 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 Kilpatrick and McFarlane (Chapter 30, this volume, on forensic issues), 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. Another important factor driving much of this criticism is the sheer magnitude of money awarded for successful personal injury suits or compensation and pension disability claims.

There is also concern that the stressor (A) criterion has opened the door to frivolous litigations in which PTSD-related damages or disabilities are dubious at best. Although DSM-5 has tightened the definition of a “traumatic event” (see Friedman & Resick, Chapter 2, this volume), it cannot change the behavior of lawyers seeking to win monetary or other benefits for their clients.

There is a significant difference, however, between challenging the utility of PTSD as a clinical diagnosis and questioning how the diagnosis is applied or misapplied in litigation by attorneys or in disability evaluations by mental health professionals. We believe that minimal standards for such evaluations (e.g., utilizing evidence-based assessment instruments; see Reardon et al., Chapter 20, and Briggs et al., Chapter 21, this volume, on diagnostic assessment in adults and children, respectively) must be developed and enforced. This would ensure that people who have a legitimate claim for a favorable judgment or 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.

**Traumatic Memories Are Not Valid**

An important scientific question concerns the validity of traumatic memories. A review of the literature on PTSD-related alterations in cognition and memory (see Vasterling & Lippa, Chapter 10, Brewin, Chapter 11, and DePrince & Freyd, Chapter 12, this volume, on cognition, memory, and dissociation, respectively) 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 and 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 formerly inaccessible memories of childhood sexual abuse that were later “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 well 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 (Roth & Friedman, 1998; see Brewin, Chapter 11, this volume, on memory).
**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 a challenge to not only PTSD assessment but also assessment of all DSM-5 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.

Several laboratory findings hold promise as potential non-self-report assessment protocols for refining diagnostic precision (see Nash et al., Chapter 14, and Rasmusson & Shalev, Chapter 15, this volume, on neurobiology and neurocircuitry and neuroplasticity, 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 has 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 Reardon et al., Chapter 20, and Briggs et al., Chapter 21, this volume, on assessment in adults and children, respectively).

A remarkable study by Dohrenwend and colleagues (2006) 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 comprising 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 to 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 ensure 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.

Our purpose in this volume is to document how far we have come since DSM-III in 1980, so that we can generate forward momentum in the right directions. Improving our understanding of PTSD so that we can translate the science into better clinical practice is the overarching goal. This book is dedicated to advancing that understanding in
order to prevent PTSD in the first place and to optimize assessment and treatment for people who suffer from the disorder and related problems.

REFERENCES


