

PROGRESS AND CONTROVERSY IN THE STUDY OF POSTTRAUMATIC STRESS DISORDER

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■ **Abstract** Research on posttraumatic stress disorder (PTSD) has been notable for controversy as well as progress. This article concerns the evidence bearing on the most contentious issues in the field of traumatic stress: broadening of the definition of trauma, problems with the dose-response model of PTSD, distortion in the recollection of trauma, concerns about “phony combat vets,” psychologically toxic guilt as a traumatic stressor, risk factors for PTSD, possible brain-damaging effects of stress hormones, recovered memories of childhood sexual abuse, and the politics of trauma.

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INTRODUCTION

Research on anxiety disorders has increased dramatically since the early 1980s (Norton et al. 1995). The scientific literature is now vast, defying ready mastery, and even the finest, most ambitious works of scholarship are unavoidably synoptic

(e.g., Antony & Swinson 2000, Barlow 2002, Craske 1999). We have learned much about these syndromes, but with progress comes controversy, and the field has had no shortage of either. This has been especially true for posttraumatic stress disorder (PTSD). My purpose is to examine the evidence bearing on the most contentious issues in the field of traumatic stress studies.

THE EMERGENCE OF POSTTRAUMATIC STRESS DISORDER

Military psychiatrists have always recognized that horrific events could trigger acute stress symptoms in previously well-adjusted individuals (Shephard 2001) but most doctors believed these reactions subsided soon after the soldier left the battlefield (Wilson 1994). This conventional wisdom changed in the wake of the Vietnam War. Antiwar psychiatrists, such as Robert Lifton, argued that many veterans continued to suffer severe stress symptoms long after having returned home (Scott 1990). Other veterans, they said, appeared well adjusted upon return to civilian life, only to develop a delayed stress syndrome months or years later. Because there was no place in the existing diagnostic system for either a chronic stress syndrome or a delayed one, these psychiatrists lobbied for inclusion of “post-Vietnam syndrome” in the forthcoming third edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-III)* (APA 1980, Young 1995). They believed that certain features of this war—such as difficulty telling friend from foe, atrocities, and unclear military goals—made it especially likely that it would produce long-lasting psychiatric illness.

Members of the *DSM-III* task force were reluctant to endorse a diagnosis tied specifically to a historical event. Yet they eventually relented when veterans’ advocates persuaded them that the same stress syndrome occurred in survivors of other traumatic events, such as rape, natural disaster, or confinement in a concentration camp. Converging clinical evidence, pointing to a common syndromic consequence of trauma, clinched the inclusion of PTSD in *DSM-III*.

Ironically, historical scholarship has now confirmed that psychiatric casualties seldom occurred in the Vietnam War, relative to other wars: The rate of breakdown was only 12 cases per 1000 men. In contrast, the rate of psychiatric breakdown during the Korean War was 37 per 1000, and during World War II it ranged from 28 to 101 per 1000 (Dean 1997, p. 40). In yet another irony, one prominent sociologist, a former member of Vietnam Veterans Against the War, has argued that advocates for the PTSD diagnosis inappropriately medicalized political dissent when they conceptualized the problems of veterans as a form of mental illness (Lembcke 1998, pp. 101–26).

In any event, the *DSM-III* defined PTSD as a syndrome erupting in response to a “stressor that would evoke significant symptoms of distress in almost everyone” (APA 1980, p. 238). The diagnosis comprised three symptom clusters. The re-experiencing cluster included recurrent intrusive thoughts about the trauma,

traumatic nightmares, and “flashbacks.” The numbing cluster included feelings of detachment from others, loss of interest in activities, and constricted affect. The third cluster included miscellaneous symptoms such as exaggerated startle, sleep disturbance, and memory impairment or trouble concentrating.

The ratification of PTSD as a formal psychiatric disorder triggered an outpouring of research on trauma and motivated the founding of the International Society for Traumatic Stress Studies and the establishment of scholarly journals devoted to the topic (e.g., *Journal of Traumatic Stress*, launched in 1988). The field has been enriched by the efforts of clinical scientists specializing in trauma, and their findings have placed into sharp relief several contentious issues.

CONCEPTUAL BRACKET CREEP IN THE DEFINITION OF TRAUMA

PTSD is unusual among DSM syndromes in that the diagnostic criteria specify an etiologic event: exposure to a traumatic stressor. Regardless of whatever symptoms may be present, unless a person has been exposed to a qualifying stressor, the diagnosis cannot be made. The architects of *DSM-III* had in mind events such as combat, rape, and earthquakes as the kind of event capable of causing the disorder. However, *DSM-IV* defines traumatic exposure as “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 which evoked “intense fear, helplessness, or horror” (APA 1994, pp. 427–28, emphasis added). Despite references to life threat and injury, *DSM-IV* significantly broadens the definition of a traumatic stressor. For example, a person who merely learns about someone else being threatened with harm qualifies as having been exposed to trauma and is therefore eligible for a PTSD diagnosis (assuming fulfillment of symptomatic criteria).

By broadening the definition of traumatic stressor, *DSM-IV* codifies a kind of conceptual bracket creep. No longer must one be the direct (or even vicarious) recipient of trauma; merely being horrified by what has happened to others now counts as a PTSD-qualifying event. Such secondhand exposure seems qualitatively distinct from being subjected to artillery bombardment for days on end while huddled in a muddy trench. Yet prevailing nosologic practice brackets both kinds of event under the same stressor rubric. With such diverse events deemed causally relevant to PTSD, it will be difficult to identify common psychobiologic mechanisms underlying symptomatic expression.

Noting that a traumatic stressor need not be life-threatening, Avina & O’Donohue (2002) have recently argued that repeatedly overhearing jokes in the workplace may qualify as a stressor that triggers PTSD. PTSD induced by repeated exposure to sexual jokes and, of course, other more serious forms of sexual harassment in the workplace provides the justification for lawsuits to secure “appropriate monetary compensations,” argued Avina & O’Donohue (2002, p. 74).

Overhearing obnoxious sexual jokes in the workplace may provide a legal basis for litigation, but it seems unlikely to produce the same psychobiological state of PTSD as violent rape.

Conceptual bracket creep was strikingly evident in the recent national survey conducted by the RAND Corporation on the weekend following the September 11, 2001 terrorist attacks (Schuster et al. 2001). After interviewing a representative sample of 560 adults throughout the United States, Schuster et al. concluded that 44% of Americans “had substantial symptoms of stress” (p. 1507), ominously adding that the psychological effects of terrorism “are unlikely to disappear soon” (p. 1511) and that “clinicians should anticipate that even people far from the attacks will have trauma-related symptoms” (p. 1512). This research team asked respondents whether they had experienced any of five symptoms “since Tuesday” (i.e., September 11, 2001). Respondents were asked to rate each symptom on a five-point scale ranging from one (“not at all”) to five (“extremely”). A person qualified as “substantially stressed” if he or she assigned a rating of at least four (“quite a bit”) to one of the five symptoms. For example, someone who had “quite a bit” of anger at Osama bin Laden qualified as substantially stressed. As Wakefield & Spitzer (2002) have cogently argued, such surveys medicalize expectable human reactions by failing to discriminate between genuine symptoms of disorder and normal distress reactions.

PROBLEMS WITH THE DOSE-RESPONSE MODEL OF POSTTRAUMATIC STRESS DISORDER

The dose-response model holds that PTSD symptoms worsen as the severity of the stressor increases (March 1993). Many researchers interpret this model in terms of Pavlovian fear conditioning (e.g., Keane et al. 1985b). Hence, traumatic stressors function like unconditioned stimuli that elicit the unconditioned response of terror, establishing neutral cues as conditioned stimuli that elicit the conditioned response of fear. Accordingly, they believe that a laboratory rat’s reaction to inescapable electric shock parallels at least some aspects of the human response to overwhelming trauma (Foa et al. 1992, van der Kolk et al. 1985). Just as increasing severity of shock exacerbates a rat’s conditioned fear, so should increasing severity of trauma exacerbate a victim’s PTSD symptoms.

Some studies are consistent with this prediction. For example, a greater proportion of World War II combat veterans who had been tortured by the Japanese as prisoners of war (POWs) have current PTSD (70%) than do those who had never been captured and tortured (18%) (Sutker et al. 1993). Ex-servicemen wounded in Vietnam are two to three times more likely to have PTSD than are those who returned unharmed (Kulka et al. 1990, p. 54). Proximity to the epicenter of an earthquake predicted severity of PTSD symptoms (Pynoos et al. 1993), and the higher the rate of wounds and fatalities within a combat unit, the higher the rate of psychiatric casualties (Jones & Wessely 2001).

However, many studies fail to support the dose-response model (for reviews, see Bowman 1997, 1999). The relationship between dosage of trauma and resultant psychopathology is far from straightforward. For example, objective measures of accident severity are unrelated to PTSD symptoms among victims of motor vehicle (and other) accidents (Schnyder et al. 2001), and the number of torture episodes is unrelated to PTSD symptom severity among imprisoned Turkish political activists (Başoğlu et al. 1994). Of course, the relationship between dose and response might be nonlinear (Harvey & Yehuda 1999). That is, if PTSD symptoms reach near maximum severity after a certain dosage of exposure, further exposure might not add much to existing levels of psychiatric impairment. For example, a person who is tortured twice may have more symptoms than someone who had never been tortured. Yet a person who has been tortured a dozen times may be no more symptomatic than one who was only tortured twice. Unfortunately, by recasting the dose-response model in nonlinear terms, any pattern between dose and symptoms would be interpretable as confirming the model (except, of course, a linear one).

DISTORTION IN THE RECOLLECTION OF TRAUMA

Data are not the only source of trouble for the dose-response model of traumatic stress. It is plagued by serious measurement difficulties as well. The animal conditioning laboratory provides the conceptual basis for the model, but calibrating stressor magnitude in trauma studies is vastly more complicated than in Pavlovian conditioning experiments. Laboratory stressors are measurable in purely physical terms entirely independent of the animal's behavior (e.g., shock amperage, number of shocks). Yet in the trauma field, researchers usually rely on the retrospective self-reports of the survivors themselves as the sole basis for measuring stressor magnitude. This practice presupposes that psychiatrically distressed individuals can furnish reliable, objective accounts untarnished by clinical state. Scientists have recently shown just how unwarranted this presupposition is.

Several studies show that a survivor's current clinical state affects how he or she remembers the traumatic experience. Longitudinal studies on staff present at a fatal shooting at an elementary school (Schwarz et al. 1993), Gulf War veterans (Southwick et al. 1997), automobile accident survivors (Harvey & Bryant 2000), and former military peacekeepers who had served in Somalia (Roemer et al. 1998) all show that memory of trauma is affected by clinical state. In each of these studies researchers obtained self-reports of traumatic events on two occasions. The more PTSD symptoms a person had at time two, the more severe the person remembered the traumatic experience to have been. For example, Southwick et al. found that 88% of Gulf War veterans remembered their traumatic events differently at time two than how they had originally reported them two years earlier. Seventy-percent recalled a traumatic event at time two that they had not mentioned at time one (one month after the war), whereas 46% failed to mention a traumatic event at time two that they had mentioned at time one. The severity of PTSD symptoms at time two significantly predicted the number of traumatic events mentioned at time two that

were not mentioned at time one. Southwick et al. (1997) concluded that veterans with higher PTSD scores “tend to amplify their memory for traumatic events over time” (p. 176).

It is unlikely that the trauma survivors in these studies were lying. Rather, traumatic memories, like all autobiographical memories, are reconstructed from encoded elements distributed throughout the brain (Schacter 1996). The context of retrieval, including clinical state, affects how these recollections occur. Although scientists, who study fear conditioning in rats, once believed that emotional memories are indelible (LeDoux et al. 1989), they have recently discovered that even these memories are subject to alteration (Morrison et al. 2002). What is true for rats is even more true for people. Although people retain traumatic memories very well, even recollections of the most horrific events are not immune to alteration of time (McNally 2003).

THE SPECTER OF THE “PHONY COMBAT VET”

The aforementioned studies concern benign memory distortion, not dishonesty, but two other issues have come to the fore in the trauma field that pose greater problems than mere “normal” memory change. One problem concerns deliberate exaggeration of symptoms in Vietnam veterans seeking to obtain the PTSD diagnosis (Frueh et al. 2000). As many as 94% of veterans with PTSD apply for financial compensation for their illness (McGrath & Frueh 2002), and the incentive to do so is strong, especially for those with limited occupational opportunities (Mossman 1994). A veteran who obtains a service-connected disability rating of 100% for PTSD can earn more than \$36,000 per year, tax-free and indexed to inflation, for life (Burkett & Whitley 1998, p. 236). The financial loss is substantial should they ever recover from PTSD. This incentive structure does not mean that most PTSD veterans are malingerers. However, the ease of faking symptoms and the incentives for doing so should worry researchers, who need to ensure that subjects in their studies really do have the disorder. This problem is not confined to Vietnam veterans. The risk of malingered PTSD arises in civil suits following accidents in civilian life as well (e.g., Rosen 1995).

The second problem concerns men who claim to be suffering from combat-related PTSD but who either never saw combat, never served in Vietnam, or never served in the military at all. In their award-winning book, *Stolen Valor*, Vietnam veteran B.G. Burkett and investigative journalist Glenna Whitley thoroughly documented seemingly countless cases of “phony combat vets” (Burkett & Whitley 1998). By obtaining military records, via the Freedom of Information Act, of men whose names had appeared in public (e.g., leaders of veterans’ organizations, the actor Brian Dennehy), Burkett & Whitley discovered much fraud. Some alleged combat veterans never saw combat; others never served in Vietnam; and still others were never in the military at all. For example, by obtaining the records of members of the American Ex-POW Association, they discovered that nearly 30% of those

claiming to have been held captive by the North Vietnamese were never POWs (Burkett & Whitley 1998, pp. 502–3). The men investigated by Burkett & Whitley are not merely faking symptoms of PTSD; they are faking their history of exposure to trauma itself. Burkett & Whitley estimated that about 75% of those “receiving PTSD compensation are pretenders” (Burkett & Whitley 1998, p. 279).

Given that a vast amount of what we know about PTSD is based on the reports of Vietnam veterans, clinical researchers need to attend to the issues raised by Burkett & Whitley. They recommend obtaining military records directly from the National Personnel Records Center in St. Louis to verify self-reports of combat exposure rather than merely relying on a photocopy of the DD-214 furnished by the veteran himself. The DD-214 is a military transcript, issued to each veteran upon his discharge. It lists his military occupational specialty, receipt of awards (e.g., Combat Infantryman’s Badge, Purple Heart), and dates of service. At most, PTSD researchers have usually required only that the veteran furnish a photocopy of his DD-214 as proof of combat exposure, but there are two problems with this procedure. First, one can easily forge combat-related items on the DD-214 by, for example, typing “Purple Heart” on the form. Second, the clerk-typists responsible for completing these forms when a soldier was discharged sometimes failed to include relevant items on the DD-214. Hence, DD-214s may either overestimate or underestimate combat exposure. A veteran’s complete military file is much less vulnerable to these validity problems.

It is important to debunk two myths about these military archives. First, contrary to a persistent urban legend in the traumatic stress field, the fire that erupted in the National Personnel Records Center on July 12, 1973 did not destroy the files of Vietnam veterans (Stender & Walker 1974). Not only were the damaged records those of men who served before the Vietnam era, but government archivists were able to reconstruct 94% of them by relying on microfilm and archives stored elsewhere. Second, some veterans claim that the government erased all evidence of their top-secret, covert operations from their record. In reality, details of the covert mission will be redacted, leaving only the dates of the mission and the phrase “Classified Assignment.” However, the special training requisite for membership in elite units assigned to covert operations (e.g., the Green Berets) is not redacted (Burkett & Whitley 1998, pp. 285–86).

Burkett & Whitley believe that many studies of combat-related PTSD are contaminated by the inadvertent inclusion of subjects who have lied about their combat trauma. They are especially critical of the National Vietnam Veterans Readjustment Study (NVVRS), an epidemiologic survey that yielded a lifetime PTSD prevalence rate of 30.9% among men who had served in Vietnam (Kulka et al. 1990). This rate of PTSD is astonishingly high, given that only 15% of the men who served in Vietnam were assigned to combat units (Dean 1997, p. 209). To be sure, men with noncombat military occupational specialties (e.g., truck driver) sometimes got in harm’s way and developed PTSD. But even after we allow for the ambushed truck drivers and other trauma-exposed noncombatants, explaining the 30.9% PTSD prevalence rate is not easy.

According to Burkett & Whitley (1998), the NVVRS is fatally flawed because the research team failed to verify, via military records, the self-reported traumatic events of the subjects. The NVVRS researchers will likely have an opportunity to correct this oversight: A multimillion-dollar follow-up of the PTSD veterans is in the works. One can only hope that researchers will verify reports of combat exposure this time, thereby addressing Burkett & Whitley's critique empirically.

The most important psychophysiological study ever done on PTSD also required only that veterans furnish a copy of their DD-214 as evidence of trauma exposure (Keane et al. 1998). Recruiting Vietnam combat veterans from VA hospitals around the country, Keane et al. examined psychophysiological reactivity to personalized, audiotaped scripts describing actual combat events in 778 veterans with PTSD, 181 veterans with past PTSD, and 369 veterans with no history of PTSD. Relative to combat veterans who never had PTSD, those with the disorder exhibited greater heart rate, skin conductance, electromyographic activity (facial muscle), and diastolic blood pressure during autobiographical combat scripts. Similar findings occurred during a standardized audiovisual combat presentation. Those with past PTSD tended to fall midway between the other groups with regard to physiologic reactivity.

However, approximately one third of the PTSD subjects did not respond physiologically to combat-related stimuli. These data are open to multiple interpretations: (a) Psychophysiological assessment may fail to detect true cases of PTSD, perhaps because a subgroup of patients is biologically nonreactive to traumatic reminders, their self-reports notwithstanding. (b) Burkett & Whitley's critique implies that the large group of nonreactive subjects may have contained veterans who either exaggerated their symptoms or fabricated their histories of combat. Without consulting the veteran's military file, it is impossible to rule out any of these possibilities. If Burkett & Whitley are right, then removal of suspicious cases and reanalysis of only corroborated cases of combat-related PTSD would likely result in even stronger psychophysiological differences between PTSD and non-PTSD groups than Keane et al. had originally reported.

As someone who has done work in this area, I was concerned that phony combat vets might have slipped into my information-processing experiments on PTSD (McNally 1998). Following Burkett & Whitley's suggestion, I obtained military records from the National Personnel Records Center for 34 of my PTSD subjects. The archival data confirmed that all were genuine Vietnam veterans. No subject, for example, who claimed to have fought in Vietnam turned out to have been a cook stationed at Fort Riley, Kansas. Although the richness of the archival record varied across subjects, evidence of combat exposure was clear for most of them. The absence of phony vets in this small pilot study suggests that men who volunteer for research studies are from a different pool than those who have caught the attention of Burkett & Whitley. Indeed, Burkett & Whitley typically investigated men whose public statements and actions seemed suspicious. Nevertheless, the integrity of the PTSD database is at issue here, and researchers should attempt to verify combat experience by consulting military archives whenever possible.

GUILT, SHAME, AND TRAUMA

The conditioning model implies that traumatic stressors cause PTSD by producing toxic levels of fear in victims, but stressors can also traumatize by inciting guilt and shame, not just fear. Among Vietnam veterans, commission of atrocities predicts risk for PTSD beyond that attributable to combat exposure alone (Breslau & Davis 1987). Even among those qualifying for a PTSD diagnosis, commission of atrocities (or at least passive exposure to them) predicts severity of PTSD symptoms beyond that predicted by extent of combat exposure (Beckham et al. 1998, Yehuda et al. 1992).

The fact that guilt about having committed atrocities can produce PTSD not only underscores the moral complexity of trauma, but it exposes another limitation of the animal conditioning model of PTSD. Although some scholars have attempted to conceptualize atrocity involvement as a high-magnitude stressor for the perpetrator (March 1993), this gambit obscures important issues by reversing the roles of victim and victimizer. Unlike fear, which can be experienced by rats, guilt and shame are complex emotions emergent only in animals possessing a sense of self. As Kagan (1998) has observed, one cannot “model” guilt in the rodent conditioning laboratory without distorting the meaning of guilt beyond recognition. Only human beings capable of cognitive self-representation can experience complex self-referent emotions like shame and guilt. Pavlovian animal-conditioning models that reduce trauma to its biological basis cannot capture this uniquely human aspect of trauma.

Of course, committing atrocities can produce PTSD only if these actions violate the person’s sense of right and wrong. Brutal acts that are not interpreted as violating one’s moral code will not produce PTSD, regardless of how atrocious they appear to others. Osama bin Laden, for example, is unlikely to develop PTSD as a result of his orchestrating the attacks on the World Trade Center.

RISK FACTORS FOR POSTTRAUMATIC STRESS DISORDER

Epidemiologic surveys indicate that a large proportion of the American population has been exposed to traumatic stressors, but only a minority ever develops PTSD. The National Comorbidity Survey revealed that 60.7% of a random sample of American adults had been exposed to traumatic events, but only 8.2% of the men and 20.4% of the women had ever developed the disorder (Kessler et al. 1995). Among the survivors (nearly all injured) of the Oklahoma City terrorist bombing, only 34.3% developed PTSD (North et al. 1999).

Because traumatic stressors produce PTSD in a minority of victims, researchers have endeavored to identify risk factors that predict the emergence of the disorder among those exposed to trauma (Brewin et al. 2000, Yehuda 1999, Yehuda & McFarlane 1995). Risk factor research offends some people, who mistakenly

believe that it entails blaming the victim. Yet discovering risk factors is essential for understanding PTSD just as it is for heart disease and other conditions. The alternative to research is ignorance, and ignorance provides an unreliable basis for treatment and prevention of any illness, including PTSD.

Some putative risk factors have been identified among people who already have the disorder. For example, Vietnam veterans with PTSD report lower levels of social support than do those without the disorder (e.g., Keane et al. 1985a). It is difficult to interpret these results. Does a lack of social support slow recovery from the acute stress symptoms? Or do these symptoms alienate possible sources of social support? Or are both processes operative? However, some variables, identified cross-sectionally, are unlikely to be consequences of the illness, and therefore may constitute risk factors. Among these are lower intelligence (McNally & Shin 1995; Vasterling et al. 1997, 2002), neurological soft signs (nonspecific behavioral indicators of central nervous system impairment) (Gurvits et al. 2000), and neuroticism (Breslau et al. 1991, McFarlane 1989). Other apparent risk factors, albeit ascertained via retrospective self-reports, are unstable family during childhood (King et al. 1996), preexisting mood or anxiety disorder (Breslau et al. 1991, Smith et al. 1990), and a family history of anxiety or mood disorder (Breslau et al. 1991, Davidson et al. 1985). Individuals who developed PTSD in response to trauma during adulthood have reported having been sexually (Engel et al. 1993, Nishith et al. 2000) or physically (Bremner et al. 1993) abused during childhood.

Prospective studies designed to identify risk factors for PTSD are scarce. However, researchers have used archival data that predates exposure to trauma as a means of identifying variables that predict PTSD among the trauma-exposed. Schnurr et al. (1993) obtained collegiate Minnesota Multiphasic Personality Inventory scores of Dartmouth College graduates who later served in Vietnam. After controlling for amount of combat exposure, they found that elevations on several scales (i.e., Hypochondriasis, Masculinity-Femininity, Psychopathic Deviate, Paranoia) predicted PTSD symptoms. Using predeployment military testing data, Bramsen et al. (2000) found that negativistic personality traits predicted PTSD symptoms among Dutch peacekeepers who were stationed in the former Yugoslavia.

Macklin et al. (1998) obtained predeployment intelligence test scores for Vietnam combat veterans. The mean predeployment IQ for those who later developed PTSD fell within the normal range ($M = 106.3$), whereas the mean predeployment IQ for those who did not develop PTSD was well above average ($M = 119.0$). Lower intelligence predicted current severity of PTSD symptoms, even after Macklin et al. statistically controlled for extent of combat exposure. PTSD symptom severity was unrelated to differences between precombat and current intelligence, thereby indicating that lower intelligence increases risk for PTSD, rather than PTSD lowering current IQ scores. Stated differently, above-average cognitive ability may enhance a soldier's ability to cope with stressors, thereby buffering him against developing PTSD.

Silva et al. (2000) likewise found that IQ was the best predictor of resilience against PTSD among inner city trauma-exposed children and adolescents. These

children had been exposed to diverse traumatic events including witnessing robberies, being in fires, and experiencing physical or sexual abuse. Among those with above average intelligence, 67% had neither PTSD nor subthreshold PTSD, whereas among those with below average intelligence only 20% were free of PTSD or PTSD symptoms.

Researchers have also identified peritraumatic (i.e., during the trauma) variables that predict later PTSD. These studies are not truly prospective because measurement occurs after trauma exposure, but they are prospective in the sense that measurement occurs before the person has had time to develop the disorder. Some studies have shown that peritraumatic dissociation predicts PTSD. Time distortion and feelings of unreality did so among trauma-exposed Israeli citizens (Shalev et al. 1996), and time distortion and a sense of bodily distortion predicted PTSD among French citizens exposed to violent crimes (Birmes et al. 2001). Emotional numbing, depersonalization, motor restlessness, and a sense of reliving the trauma predicted PTSD among survivors of automobile accidents in Australia (Harvey & Bryant 1998). Dissociative symptoms—most commonly a sense of time slowing down or speeding up—increased the risk of PTSD by nearly a factor of five among American motor vehicle accident survivors (Ursano et al. 1999). Finally, elevated heart rate among civilian trauma survivors, assessed in the emergency room, predicted subsequent PTSD (Shalev et al. 1998).

DOES TRAUMATIC STRESS DAMAGE THE BRAIN?

Glucocorticoids—cortisol in primates—are released as part of the fight-flight response. Although these stress hormones adaptively facilitate defense in the short term, prolonged glucocorticoid exposure produces hippocampal atrophy, at least in rats and monkeys (for a review, see Sapolsky 2000). Accordingly, some psychiatrists have wondered whether extreme stress might have damaged the hippocampus of trauma survivors. The stress of having chronic PTSD might also wear away at the hippocampus (Bremner 2001).

Consistent with this hypothesis, several magnetic resonance imaging (MRI) studies have shown that the hippocampi of individuals with PTSD are smaller than those of matched control subjects. In one study Vietnam combat veterans with PTSD had significantly smaller (by 8%) right hippocampi than nonveteran control subjects (Bremner et al. 1995), and in another, those with PTSD had significantly smaller left (by 26%) and right (by 22%) hippocampi than a combined group of Vietnam combat veterans and healthy nonveteran control subjects (Gurvits et al. 1996).

Smaller hippocampi are not confined to veterans with combat-related PTSD. Adult PTSD sufferers with histories of childhood physical or sexual abuse have significantly smaller left (by 12%) hippocampi than do nonabused control subjects (Bremner et al. 1997). Women with childhood sexual abuse histories, most qualifying for PTSD, had significantly smaller (4.9%) left hippocampi than nonabused control subjects (Stein et al. 1997).

Although each of the aforementioned studies is consistent with the hypothesis that stress produces hippocampal atrophy in PTSD patients, other facts strongly argue against this interpretation. Rather than being elevated—as the atrophy hypothesis predicts—urinary cortisol levels in people with PTSD are often in the low-normal range (Mason et al. 1986; Yehuda et al. 1990, 1995). The normal range is 20–90 $\mu\text{g}/\text{day}$. In early studies PTSD patients usually had lower values (about 30–40 $\mu\text{g}/\text{day}$) than matched control subjects (about 50–60 $\mu\text{g}/\text{day}$) (Yehuda 1997). However, in a recent, large study on Vietnam veterans, Mason et al. (2001) failed to replicate their original finding of low cortisol in PTSD patients. Ironically, the mean value for these PTSD subjects (61.3 $\mu\text{g}/\text{day}$) was nearly identical to that of the nonpsychiatric control subjects (62.8 $\mu\text{g}/\text{day}$) in one of their original studies reporting low cortisol in PTSD (Yehuda et al. 1990). Thus, cortisol in PTSD is not maintained at high levels for a sufficiently long period of time to produce hippocampal atrophy. Short periods of very high levels of cortisol adaptively mobilize the person for fight or flight; only if such levels are maintained for months or years might damage occur.

For example, because of an adrenal tumor, patients with Cushing's syndrome experience extremely high levels of cortisol that results in explicit memory deficits and hippocampal atrophy (e.g., 508 $\mu\text{g}/\text{day}$) (Starkman et al. 1992). However, not only does surgical correction of the tumor normalize cortisol levels, but it eliminates memory deficits and enables the hippocampus to rebound to its normal size (Starkman et al. 1999).

Recent neuroimaging studies provide additional data against the hypothesis that stress shrinks the hippocampus of trauma survivors. Bonne et al. (2001) used MRI to scan 37 trauma survivors one week following their admission to a hospital emergency room. They rescanned them six months later. By this second assessment, 10 of the 37 subjects had developed PTSD. The PTSD and non-PTSD groups did not differ in hippocampal volume, and PTSD symptom severity was not correlated with hippocampal volume at either scan. Finally, hippocampal volume in the PTSD group remained stable over the course of the 6-month period.

In a landmark study, Gilbertson et al. (2002) may have decisively refuted the atrophy hypothesis. Using MRI, these researchers measured hippocampal volume in a series of monozygotic twin pairs. One cotwin fought in Vietnam and developed PTSD, whereas his brother did not serve in Vietnam and did not have PTSD. If trauma shrinks the hippocampus, then the hippocampi of the PTSD cotwins should be smaller than those of their nontraumatized twin brothers. Although Gilbertson et al. once again found smaller than average hippocampi in subjects with severe PTSD, they also found that the hippocampi of nontraumatized twins were just as small. Not only was PTSD symptom severity negatively correlated with total hippocampal volume in trauma-exposed subjects ($r = -0.64$), but PTSD symptom severity was just as negatively correlated with the total hippocampal volume of their twin brothers ($r = -0.70$). The concordance in hippocampal volume between pairs of identical twins—irrespective of the presence of PTSD or trauma history—strongly suggests genetic influence on hippocampal volume. Because the cotwins

had not been exposed to traumatic stress, Gilbertson et al. concluded that small hippocampi may constitute a preexisting vulnerability factor for PTSD among the trauma-exposed. These data are consistent with studies showing that PTSD patients report more neurodevelopmental abnormalities than do trauma-exposed people without the disorder (Gurvits et al. 2000). Compromised neurocognitive functioning may impede a person's ability to cope with traumatic stressors.

RECOVERED MEMORIES OF SEXUAL ABUSE

By far the most contentious issue in the field of traumatic stress concerns the accuracy of recovered memories of childhood sexual abuse (McNally 2003). In their award-winning book, Brown et al. (1998) asserted that "approximately a third of sexually abused victims report some period of their lives where they did not remember anything about the abuse and later recovered the memory of the abuse" (p. 196).

Accordingly, because of "massive repression" (Herman & Schatzow 1987, p. 12), Brown et al. (1998) believe that therapists must often apply special techniques to retrieve memories of trauma that presumably lie at the root of diverse symptoms. They assert, "Because some victims of sexual abuse will repress their memories by dissociating them from consciousness, hypnosis can be very valuable in retrieving these memories. Indeed, for some victims, hypnosis may provide the only avenue to the repressed memories" (p. 647).

This claim flies in the face of everything scientists have learned about hypnosis. Not only does hypnosis fail to enhance the accuracy of recollection, but it fosters the production of false memories that are mistakenly experienced as accurate (Kihlstrom 1997, Lynn et al. 1997, Steblay & Bothwell 1994).

The controversy concerning repressed and recovered memories of childhood sexual abuse has been deeply divisive in psychology and psychiatry. Some scholars argue that there is no convincing evidence that people can banish and then recover memories of horrific experiences (e.g., Pope et al. 1998), whereas others proclaim "overwhelming scientific support for the existence of repressed or dissociated memory" (Brown et al. 1998, pp. 538–39). What is most bizarre about this debate is that proponents on both sides appeal to the same scientific studies to support their diametrically opposed positions. How is this possible? Anyone who actually reads the contested studies, however, will immediately realize that the most influential advocates of the traumatic amnesia position misunderstand much of the science they cite. I can only provide illustrative examples of their misreadings here; for further analysis, see McNally (2003) and Piper et al. (2000).

For starters, consider Brown et al.'s (1999) claim that "the burden of proof is on them [skeptics of repressed memories] to show that repressed memories do not exist" (p. 125). They have it exactly backwards: The burden of proof lies on Brown et al. to provide convincing evidence that people can repress and later recover memories of trauma. For logical reasons, the skeptics cannot prove the null hypothesis that repression does not occur.

Many studies have shown that people complain about nonspecific memory impairment following exposure to traumatic events. This is the *DSM-III* symptom of “memory impairment or trouble concentrating” (APA 1980, p. 238). Brown et al. misunderstand what this symptom means, mistakenly believing that it “typically includes both hypermnesia and amnesia” (Brown et al. 1999, p. 27). In reality, it refers to neither; hypermnesia—vivid recollections of the trauma—is, of course, already covered by the diagnostic criterion of recurrent and intrusive recollections of the event. *DSM-III* memory impairment refers to forgetfulness in everyday life that emerges after exposure to a traumatic event; it does not refer to difficulty remembering the traumatic event itself (i.e., traumatic amnesia).

Brown et al.’s misconstrual is immediately apparent to anyone who reads the studies they cite in support of traumatic amnesia. For example, 88.2% of the witnesses to the catastrophic collapse of the Hyatt Regency Hotel skywalks in Kansas City experienced “repeated recollections” of the disaster, and 27.4% reported “memory difficulties” (Wilkinson 1983). Obviously, these individuals remembered the disaster all too well. Indeed, their preoccupation with vivid, intrusive recollections might have interfered with their ability to concentrate and remember ordinary things in everyday life.

While searching for evidence of traumatic amnesia, Brown et al. sometimes cite findings attributable to direct physical insult to the brain as relevant to psychic trauma. For example, they state that “Dollinger (1985) found that two of the 38 children studied after watching lightning strike and kill a playmate had no memory of the event” (Brown et al. 1998, pp. 609–10). Unfortunately, they fail to mention that both children had themselves been struck by side flashes from the main lightning bolt, knocked unconscious, and nearly killed (Dollinger 1985). Such cases of obvious organic amnesia are irrelevant to psychogenic, traumatic amnesia.

In yet another example, they cite the work of Wagenaar & Groeneweg (1990) as evidence that “amnesia for Nazi Holocaust camp experiences has also been reported” (Brown et al. 1998, p. 156). Wagenaar & Groeneweg studied concentration camp survivors 40 years after their liberation. These investigators compared the memory reports of former inmates with the depositions they had provided about their trauma four decades earlier. After comparing recent recollections with the original reports, Wagenaar & Groeneweg said that inmates exhibited “a remarkable degree of remembering” (p. 80) of their Holocaust experiences, all the more impressive in view of the starvation and head beatings many suffered. “There is no doubt,” emphasized Wagenaar & Groeneweg, “that almost all witnesses remember Camp Erika in great detail, even after 40 years” (p. 84). So, why did Brown et al. cite this study as indicating amnesia for Holocaust experiences? As it turns out, several former inmates had forgotten to mention several violent events, plus other minor details (e.g., the name of a sadistic guard), among all the horrific events they had vividly recalled. But with one exception, each camp survivor remembered these temporarily forgotten events after having examined their original depositions. Because autobiographical memory does not operate like a videorecorder, it is not surprising that some of these elderly individuals did not immediately recall every traumatic event that happened in the concentration camp.

Most of the studies adduced by Brown et al. in support of the concept of traumatic amnesia concern sexual abuse, and most suffer from the same fatal flaw. In the modal study (e.g., Briere & Conte 1993) sexual abuse survivors were asked whether there was ever a time when they could not remember their abuse. Nearly 60% said "yes." However, a reported inability to remember one's abuse implies unsuccessful retrieval attempts. If survivors were unaware of their abuse, on what basis would they attempt to recall it in the first place? Obviously, the best way to make sense out of affirmative replies to this question is to assume that survivors interpreted the question as asking, "Has there ever been a time when you did not think about your abuse?" Obviously, not thinking about one's abuse is not the same as being unable to recall it.

In one of the best studies on this topic Williams (1994) and her research team interviewed 129 women who had been medically evaluated for suspected sexual abuse about 17 years earlier when they were children. As part of this general health survey, the interviewers asked questions about childhood sexual experiences and recorded all events mentioned by the subjects. The researchers then judged whether any of the reported events matched the index event recorded in the hospital records. Although 38% ($n = 49$) of the women did not mention the index event for which they had been taken to the hospital many years earlier, most ($n = 33$) of them did describe other episodes of sexual abuse. However, 16 subjects denied ever having been sexually abused.

Although Brown et al. interpreted Williams's data as evidence for traumatic amnesia, other explanations are plausible. Some subjects may have been too young either to understand or remember what had happened to them. Several of the non-reporters were under 4 years of age when they were assessed for abuse at the hospital. Hence, ordinary childhood amnesia might have precluded their remembering (or even understanding) what had happened. Also, the 16 nonreporters may have remembered their abuse, but elected not to mention it to the interviewer. It would have been helpful if Williams had done a follow-up clarification interview with subjects who denied ever having been abused to determine whether they had actually forgotten the event or whether they were merely reluctant to report it.

Femina et al. (1990) did such a study with subjects who had documented histories of physical abuse. Those subjects who had mentioned this abuse in an earlier interview but had denied their abuse in a second interview were contacted a third time. During the third interview they were asked to explain the discrepancy between the first and second interviews. Each subject then acknowledged having remembered the abuse during the second interview and admitted to having denied it for various reasons (e.g., dislike of the interviewer). Without such clarification interviews to resolve discrepancies between official records and self-report denial of abuse, it is impossible to distinguish between failure to report abuse and failure to remember it.

The most intriguing evidence for recovered memories of abuse comes from case studies in which the events have been independently corroborated (e.g., Cheit 1998, 1999; Schooler et al. 1997). However, explaining even these cases does not require postulation of any special repression or dissociation mechanism. Consider,

for example, a girl who is fondled by a man and who does not think about this frightening (or perhaps confusing) episode for many years. As a young woman, she meets someone who reminds her of the perpetrator, and suddenly remembers the experience. Such a case would constitute a recovered memory of sexual abuse, but not repression or amnesia. That is, during the period when she had not thought about the episode, she might very well have remembered it if someone had asked her whether she had ever had any unwanted sexual experiences. Not only do these cases not involve any special dissociative mechanism, but they do not require therapeutic efforts to exhume presumptively repressed memories. Indeed, many people who report recalling memories of abuse after long periods of not thinking about them do not remember these experiences in psychotherapy (Herman & Harvey 1997, McNally et al. 2000a). One does not need a “recovered memory therapist” to be reminded of long forgotten episodes from one’s childhood.

Despite all the furor surrounding people who report recovering memories of sexual abuse, research on cognitive functioning in these individuals has scarcely begun. Our group has been conducting studies on four groups of individuals: adults who report remembering abuse after years of not thinking about it (“recovered memory” group); adults who believe they have been abused, but who have no conscious memories of trauma (“repressed memory” group); adults who have always remembered they had been abused (continuous memory group); and adults who deny an abuse history (control group) (McNally 2001). (The terms “recovered memory” and “repressed memory” are in quotes because we were not in a position to determine whether these individuals had, in fact, been abused. The terms, however, describe their phenomenologic experience.)

Some key findings emerging from this research are as follows: Repressed memory subjects report more symptoms of psychological distress (PTSD and depression symptoms) than do continuous memory subjects (who resemble controls), whereas recovered memory subjects fall midway between continuous memory and repressed memory subjects (McNally et al. 2000a). Recovered memory subjects tend to exhibit less imagination inflation than do control subjects following a guided imagery task designed to distort memory (Clancy et al. 1999). Unlike patients with PTSD (McNally 1998), however, including sexually abused children with the disorder (Dubner & Motta 1999), repressed and recovered memory subjects do not exhibit delayed color-naming of trauma words on the emotional Stroop task (McNally et al. 2000b). Repressed and recovered memory subjects do not exhibit a superior ability to forget trauma-related words on a directed forgetting task (McNally et al. 2001), contrary to the hypothesis that they ought to be superior at blocking out memory for material related to abuse. Moreover, childhood sexual abuse survivors with PTSD actually exhibit impaired ability to forget trauma-related material on this task (McNally et al. 1998). Recovered memory subjects are more prone to exhibit false memory effects on the list learning paradigm developed by Deese (1959) and Roediger & McDermott (1995) than are continuous memory subjects (Clancy et al. 2000). Subjects who report having been abducted by space aliens likewise exhibit this false memory effect in the laboratory (Clancy et al. 2002).

THE POLITICS OF TRAUMA

There is never a dull moment in the field of traumatic stress studies. Discoveries are continually intermixed with explosive social controversies. For example, on July 12, 1999, members of the United States Congress unanimously voted to condemn a scientific article on childhood sexual abuse for its alleged moral and methodological flaws. The article contained a meta-analysis of 59 studies that had addressed the long-term psychological correlates of childhood sexual abuse (Rind et al. 1998). Stunning many people, the meta-analysis revealed that subjects who had been sexually abused were nearly as well adjusted as their nonabused counterparts. Less than 1% of the variance in psychological adjustment was attributable to childhood sexual abuse. The meta-analysis indicated that childhood sexual abuse survivors were apparently more resilient than most mental health professionals (who see only the most traumatized cases) had ever suspected. Rind et al. took pains to emphasize that harmfulness must not be confused with wrongfulness: Merely because sexual abuse does not invariably produce long-term psychological damage does not make it morally permissible.

Despite this emphasis, the article incited an uproar among many mental health professionals, talk show hosts, and religious groups. Although scientists had rigorously reviewed the manuscript prior to publication, the critics assumed it must be flawed because it ran counter to prevailing opinion. Hence, they attempted to debunk it on methodological as well as on moral grounds. Politically conservative radio personality "Dr. Laura" railed against Rind and his colleagues and against the American Psychological Association for publishing the article in *Psychological Bulletin*. Condemning it as "junk science at its worst," (quoted in Lilienfeld 2002a, p. 178), she urged Congress to take formal action against the Association. Raymond D. Fowler, Chief Executive Officer of the American Psychological Association, initially defended the scientific quality of Rind et al.'s work and the integrity of peer review (Garrison & Kobor 2002) but when it became clear that Congress was about to condemn the APA itself, as well as Rind et al., Fowler (1999) abruptly reversed himself in a letter written to House Majority Whip, Tom DeLay. Fowler repudiated the article, apologizing to Congress that its policy implications had not been considered during the review process. Fowler also promised that the APA would "seek independent expert evaluation of the scientific quality" of Rind et al.'s article, and would encourage "refutations from researchers and practitioners with expertise in child sexual abuse in an upcoming issue of one of our premier journals."

Fowler's apology did not save Rind and his colleagues from the wrath of Congress. Passing unanimously, House Congressional Resolution 107 stated that "Congress condemns and denounces" Rind et al.'s "severely flawed" article (US House of Representatives 1999). The United States Senate unanimously approved the resolution on July 30, 1999. While political conservatives, religious fundamentalists, and some mental health professionals celebrated the Congressional condemnation, others saw it as a serious threat to scientific freedom. Many psychologists

were outraged that the APA had capitulated to political pressure (Lilienfeld 2002a,b).

Keeping his promise to Congress, Fowler asked the American Association for the Advancement of Science (AAAS) to evaluate the scientific merit of Rind et al.'s article. But after an initial evaluation uncovered "no clear evidence of improper application of methodology or other questionable practices on the part of the article's authors," the AAAS committee declined to conduct a full-scale review, noting that it saw no reason to second-guess an article that had already undergone expert peer review (Lerch 1999). In fact, the AAAS sharply rebuked the critics of Rind et al. for misrepresenting the article in the media and for failing to understand the meta-analytic methods they had attacked.

Reverting to the conventional methods of scholarship, some of the critics who had aided Congress in crafting the condemnation published critiques of Rind et al.'s work (Dallam et al. 2001, Ondersma et al. 2001). These were easily rebutted (Rind et al. 2001, Sher & Eisenberg 2002). Ironically, studies on nonclinical populations appearing since the publication of Rind et al.'s meta-analysis have uncovered adverse consequences of childhood sexual abuse (Kendler et al. 2000, Nelson et al. 2002).

In any event, the overriding lesson of this bizarre episode concerns the importance of maintaining a firewall between science and politics (Hunt 1999). This is especially true whenever the topic concerns trauma and its consequences.

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