Neurobiological Models in Posttraumatic Stress Disorder: Effects on Public Perception and Patient Care

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ABSTRACT. Concurrent with the many controversies that have surrounded the diagnosis of posttraumatic stress disorder (PTSD), there has been an increasing understanding of the neurobiology of trauma and PTSD. The result of this work has provided considerable evidence that trauma can change the brain and that individuals with PTSD are affected by a range of neurobiological alterations. These neurobiological findings are likely to have some impact on the controversies surrounding the field and significant implications for how the public views the trauma survivor as well as how survivors view themselves. After providing a brief primer on some of the most recent neurobiological findings, the clinical implications of such a "brain-based" model of PTSD are discussed.

KEYWORDS. Posttraumatic stress disorder, trauma, neurobiology, brain, controversy, vulnerability

Despite almost 25 five years as a formal American Psychological Association sanctioned diagnosis, posttraumatic stress disorder (PTSD) is

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still struggling for a level of credibility afforded to other psychiatric diagnoses (American Psychological Association, 1980). Although there are numerous reasons why this is the case, it is undeniable that it is partly because of PTSD being greatly affected by a number of unresolved issues that are quite controversial. Since its very inception, it has been a diagnosis that has fostered argument in the literature and in the press. When the American Psychiatric Association finally decided to include PTSD as a diagnosis in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–III*), there had already been years of controversy about whether its inclusion was justified, what symptoms should be included and in what format, and whether the diagnosis overall should be considered a dissociative disorder or an anxiety disorder (Horowitz, Weiss, & Marmar, 1987; Scott, 1990).

Despite the seriousness of the disorder and the long-term consequences it has on those affected, the unresolved issues surrounding PTSD have served to undermine the credibility of the disorder. Articles critiquing the validity of the PTSD diagnosis and its treatments abound in both the popular press and professional journals (for a review, see McNally, 2003). PTSD is widely criticized and even lampooned in popular media, but even peer-reviewed journal articles have carried titles such as "PTSD: A Problematic Diagnostic Category" (McHugh & Treisman, 2007) and "Saving PTSD from Itself in the *DSM–IV*" (Spitzer, First, & Wakefield, 2007). Just a brief search of the literature on "PTSD and controversy" reveals articles that are wide ranging and numerous. Of note are articles that discuss (a) the potential liabilities of critical incident stress debriefing, (b) the emergence of false "traumatic" memories in therapy, (c) the utilization of an acute stress disorder diagnosis that pathologizes early responses to trauma, (d) the over-inclusiveness of the word *trauma* to include everyday stressors, (e) the widespread use of therapies with questionable scientific support, (f) the faking of a PTSD diagnosis to receive compensation or disability, and (g) the use of a PTSD diagnosis as the basis for an insanity defense.

As a single example, the value of psychological debriefing was called into question after the 9/11 terrorist attacks on New York and Washington. This psychological debriefing debate was the catalyst for articles in Sunday editions of both the *New York Times* and the *London Times* (combined Sunday circulation of more than 3 million subscribers) urging disaster survivors to maintain a "stiff upper lip" (Baxter & Rogers, 2003) and to "repress yourself" (Slater, 2003). Both articles asserted that the best response to trauma was to not think about it. Neither article made reference to the vast evidence indicating that expressing oneself emotionally is the

key to recovery in cases in which the symptoms of PTSD linger beyond the first few months.

Not that a reader of either article would ever have noted such a distinction. When an article that reaches millions of individuals refers to the mental health professionals treating trauma survivors as the "trauma industry," such distinctions are likely to be lost. And when an article further describes the response of mental health professionals after 9/11 in terms reminiscent of a biblical plague (i.e., "swarms of therapists descended on New York City"; Slater, 2003, para. 3), credibility is clearly at issue.

There is really no way to account for the damage such controversy and such articles have on the public perception of the trauma survivor, the PTSD sufferer, and those who treat them. We can assume, however, that such controversy only adds to the pain of patients and makes needy individuals even less likely to access services in the future.

This might be an issue of little concern if PTSD were a disorder that was relatively rare or relatively innocuous. It is neither. PTSD is a disorder of significant concern because of both its frequency and its severity. The epidemiological evidence indicates that rates for PTSD vary depending on the sample studied; lifetime rates of 7.8% were found in a national sample, but victims of rape or combat showed lifetime rates greater than 30% (Breslau et al., 1998; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993). For those affected, the symptoms can potentially affect sleep, mood, concentration, dreams, states of consciousness, and interpersonal relationships. The social and occupational impact of the disorder is increasingly well recorded. There is evidence to indicate that individuals with PTSD work less, are more likely to be jailed, have poorer quality relationships, and have much higher rates of physical illness at a significant cost to the health care system (Kulka et al., 1990; Metzler, Hidalgo, Connor, & Davidson, 2000; Zatzick et al., 1997). Individuals with more chronic forms of the disorder function at levels comparable to individuals carrying other severe psychiatric diagnoses (Mazzeo, Beckham, Witvliet, Feldman, & Shivy, 2002; Warshaw, Fierman, Pratt, & Hunt, 1993).

The field appears to be at a crossroads. On one hand, it is struggling with a significant psychiatric disorder. On the other hand, its hands are tied by controversies that are likely to limit the number of individuals willing to seek treatment after trauma and discourages well-trained clinicians and researchers from working in the trauma field.

A broader sociocultural perspective (Baldwin, Williams, & Houts, 2005) provides a context in which these controversies can be seen as part of a larger, more gradual process of diagnostic evolution that is typical of many newer diagnoses. Although the controversies present considerable problems with respect to public perception, they are also critical to resolving seminal issues and laying down a strong theoretical and empirical basis for the diagnosis and treatment of PTSD. Eventually science will produce a much better understanding of even very complex issues such as "false memories," early intervention, and pseudoscientific treatments. In addition, research will eventually produce assessment strategies and decision trees that will help clinicians better separate false disability claims from the true.

In the meantime, part of the solution is to be found in the growing neurobiological models for trauma related disorders. Although there is no antidote for controversy that is superior to high-quality research directly addressing the controversies themselves, growing brain-based models have implications for how the public at large perceives the PTSD diagnosis as well as how survivors are perceived. This article discusses the growth of this model in the last decade, along with some of the more critical neurobiological systems associated with the presentation of PTSD, and the implications this model has for patient care and the public perception of PTSD.

Although it may appear speculative to tie the credibility of a psychological disorder to a brain-based model, this certainly would not be the first time in history that the brain has reclaimed a mental disorder from controversy and pseudoscience. The realization that an excess of porphoryns in the bloodstream affected the functioning of the brain rewrote the books on King George III of Great Britain. The realization that a tertiary form of syphilis was responsible for filling the asylums of the 19th and early 20th centuries dramatically changed public perception of the mentally ill at that time. In the 20th century, the efficacy of catecholamine antagonists in individuals with schizophrenia saved that disorder from prevailing theories of critical mothers, double-binds, insecure attachments, and poorly managed reinforcement schedules. There was no reason to believe that these perceptions of schizophrenia would have changed dramatically over the years, at least not until Henri Laborit made his critical discovery of chlorpromazine in 1952. In addition to providing great relief for many struggling with schizophrenia, it dramatically changed how individuals with mental illness were perceived. A more sympathetic and understanding stance was inevitable when it became

clear that the expression of schizophrenia had a significant biological component for which no one, parent and patient alike, could be held responsible.

The growth of a neurobiologically based model in the 1990s represented a significant change in the trauma field. Although neurobiological models were well established in the 1980s for disorders like bipolar disorder, schizophrenia, and depression, PTSD was still seen primarily as a normal reaction to an abnormal event. Embedded in the diagnosis itself was the assumption that the disorder was largely due to the toxic effects of the trauma rather than some constitutional weakness in the individual (APA, 1980, 1987). This was codified in the diagnosis in these early years by requiring that the person be exposed to an event outside the range of normal human experience (APA, 1980).

Many voiced two concerns with this model. First, it became clear that not everyone who experienced a traumatic event went on to develop PTSD. Second, it became apparent that trauma was not an experience "outside the range of normal human experience." In a nationwide epidemiological study carried out in the early 1990s, it was apparent that traumatic events, at least as they were currently defined in the *DSM*, were not at all uncommon (Kessler et al., 1995). Kessler and colleagues found that 61% of men and 51% of women had experienced some traumatic event during their lifetime. In urban areas where risks were higher, 90% of the individuals reported at least one traumatic event (Breslau et al., 1998). This research indicates that traumatic events, rather than being outside the range of normal human experience, were in fact part of life.

Concurrent with this recognition was the growing evidence that, despite the extent of trauma in the population, only a percentage of those individuals were developing long-term pathological responses to traumas (Breslau et al., 1998; Kessler et al., 1995; Kulka et al., 1990; Resnick et al., 1993). The high rates of trauma in combination with the lower rates of PTSD forced a reconsideration of the etiological and maintenance factors associated with the PTSD diagnosis. Neurobiological models have played a role in this rethinking and have challenged the field to think differently about how individuals develop PTSD, how those symptoms remit, and how to best treat individuals.

Reviewed next are biological findings that are clinically relevant to the practicing clinician. The review is not meant to be exhaustive (although further references are provided) but rather is intended to provide a series of examples of how trauma can change the brain, how treatments can change brains "back," and which critical neurobiological systems are

implicated in both symptom presentation as well as recovery. Such information can provide a heuristic for those not familiar with the neurobiology of PTSD on how to think about their patient's experience and what is involved in the process of recovery. Following is a discussion on how such information might be used in practice to allay patient concerns, gain confidence in the therapeutic process, and change the patient's perception of the both their trauma and themselves.

DOES TRAUMA CHANGE THE BRAIN?

Of considerable interest in the past decade has been the extent to which stress and trauma may change an individual's biology in a manner that results in temporary symptom presentation and, in some, the expression of PTSD. Although this work is in its earliest stages, it is already having an impact on the field; the very notion that trauma-related symptom presentations have biological correlates changes the view of trauma from a purely interpersonal, phenomenological experience to an experience that integrates biological reactions with one's personal reactions. If trauma changes one's neurobiology, then there is a possible causal explanation for one's posttrauma experiences (intrusive memories, insomnia, depersonalization) that may bring some order and understanding to the wide-ranging symptoms that follow in some who have been traumatized.

Discussed next are three levels in which the brain may be affected by trauma and PTSD: brain structure, endocrine functioning, and neurotransmitter levels. In each case, a specific example is provided regarding how alterations in such systems can result in a PTSD presentation. Citations are also provided for comprehensive review articles, chapters, and original research articles that are critical to our current understanding of the neurobiology of PTSD.

Brain Structure

Research with animals has convincingly established that long-term exposure to stress results in neural atrophy and cell death in a part of the brain called the hippocampus (Bremner, 2002; McEwen, 2002). For this reason, there have been numerous studies that have focused on this structure in humans. In fact, there have been numerous studies that have shown the hippocampus to be smaller in individuals with PTSD (Bremner, 2001; Bremner, Randall, Scott, & Bronen, 1995; Bremner et al., 2001; Gurvitz,

Shenton, Hokama, & Ohta, 1996; Lindauer et al., 2004; Wignall et al., 2004). Such findings have been present in some PTSD samples within a month of the trauma (Wignall et al., 2004) and in PTSD samples without comorbid alcohol abuse (Lindauer et al., 2004). However, there is also evidence that the hippocampus may be smaller before trauma exposure and may actually be a risk factor for PTSD (De Bellis et al., 2002; Gilberston et al., 2002; Woodward et al., 2006). Further work is necessary to clarify the relationship between the hippocampus in PTSD. However, work on other areas has been less mixed, and there has been evidence and models for brain structures functioning differently in individuals who develop PTSD (Carrion et al., 2001; Lanius et al., 2001; Shin et al., 2004).

One such structure is the thalamus, a medial structure in the brain that serves as a conduit for neural information to get from the senses to higher brain structures. It has been hypothesized that thalamic functioning plays a key role in dissociative responses often seen in trauma victims (Krystal, Bennet, Bremner, Southwick, & Charney, 1995; Krystal et al., 1999). Common dissociative symptoms include depersonalization (floating outside one's body), derealization (everything around seeming changed or unreal), alterations in time perception (time moving faster or slower than usual), and perceptual disturbances (distortions in sounds or images).

The thalamus also receives input from brain arousal structures, in addition to sensory input. The confluence of these inputs allows for the possibility of arousal systems to affect the quality and quantity of information that is passed along for further processing. In conditions of high stress (such as traumatic stress), arousal structures are highly activated and are likely to be disruptive to thalamic functioning. Under such circumstances, the thalamus cannot reliably relay sensory information—thus causing distortions. Such arousal-related distortions are likely to result in disturbed perceptual experiences that clinicians recognize as derealization, depersonalization, and alterations in time perception.

Hormones

In animals, it is clear that the brain and neuronal changes poststress are in part due to changes in the animals' hormone system (Henry, 2002). Under constant stress, animals secrete hormones that affect their brains and bodies (e.g., blood vessels, heart, immune system; Heim, Ehlert, & Hellhammer, 2000). In humans, it appears that similar hormonal release also results in damage to the brain and body. One stress hormone released from the adrenal glands, called cortisol, is shown to be processed very

differently in individuals with chronic negative stress responses (Anisman, Griffiths, Matheson, Ravindran, & Merali, 2001; Holsboer, 2000; Yehuda, Giller, Southwick, Lowy, & Mason, 1991). Typically, there are lower levels of this hormone in circulation in PTSD but higher levels of this hormone released in response to stress (Elzinga, Schmahl, Vermetten, van Dyck, & Bremner, 2003; Yehuda, 2002; Yehuda et al., 1995). It is thought that cortisol release causes some of the memory problems seen in PTSD as well as long-term health issues (Golier & Yehuda, 1998; Wong, 2002).

It appears that cortisol likely affects aspects of memory in PTSD by influencing the functional performance of the hippocampus. Recent evidence has indicated that acute cortisol elevations (like those that occur under conditions of stress or trauma) impair normal memory retrieval (Elzinga, Bakker, & Bremner, 2005; Het, Ramlow, & Wolf, 2005; Kuhlmann, Piel, & Wolf, 2005). Given that individuals with PTSD often secrete higher levels of cortisol in response to stress, it is possible that the difficulty remembering aspects of a trauma, as well as memory fragmentation (partial memories often with sensory components not integrated) associated with PTSD, are related to cortisol effects on the hippocampus.

Neurotransmitters

PTSD has been found to be associated with changes to serotonergic, GABA, glutamate, and the catecholamine systems. Perhaps best understood are changes to the noradrenergic (norepinephrine) system. In the brain stem, there is a structure called the locus coerulues (LC), which can be usefully referred to as the brain's "stress center." The LC sends signals all throughout the brain and is responsible for the brain's general level of activity and arousal and widespread release of norepinephrine (Aston-Jones, Ennis, Pieribone, Nickel, & Shipley, 1986). Stimulation of this part of the brain in animals results in anxious behavior (Berridge & Waterhouse, 2003). In individuals with PTSD, it is believed that the LC releases higher than average levels of norepinephrine throughout the brain (Aston-Jones, Valention, & Van Bockstaele, 1994; Zigmond, Finlay, & Sved, 1995). It is thought that this increased release results in the anxiety, unforgettable memories, poor sleep, and the hypervigilance often seen in PTSD patients (Adamec, 1997; Southwick et al., 1993).

For example, there is evidence indicating that increased norepinephrine (NE) release at the time of trauma is related to intrusive memories and flashbacks (Southwick et al., 1999). In the lab, norepinephrine has been

shown to improve recall of evocative stimuli (Cahill & McGaugh 1998). In individuals with PTSD, it is likely that traumatic memories are overconsolidated, partly due to increased NE release at the time of trauma and partly due to subsequent NE release because of posttrauma anxiety that further consolidates the memory. Such a model would explain intrusive memories and flashbacks that are primed by anxious situations in those with chronic PTSD.

One area of active debate in the field is the extent to which biological alterations seen in PTSD patients represent predisposing factors or consequences of the trauma. For example, the animal literature and the early work with humans seemed to convincingly state that stress and trauma caused damage to the hippocampus in PTSD patients. However, there has been recent evidence indicating that smaller hippocampi may be a preexisting condition in those who develop PTSD (De Bellis et al., 2002; Gilberston et al., 2002; Woodward et al., 2006; Woodward et al., 2007). It is possible that the smaller hippocampi may predispose someone to the development of PTSD. It is more likely, however, that the smaller hippocampi and the development of PTSD may have a common cause: ongoing childhood stress or maltreatment. This factor in possible combination with limited cognitive resources may have set some individuals on a neurodevelopmental path from childhood that made them more vulnerable to develop PTSD as an adult upon exposure to a trauma (De Bellis, 2001). This is consistent with a young but growing body of prospective and twin studies that show that childhood stress, neurological soft signs, and poor neurocognitive functioning (including low IQ) may be risk factors rather than consequences of PTSD (Breslau, Lucia, & Alvarado, 2006; Gilbertson et al., 2006; Gurvitz et al., 2006; Koenen, Moffitt, Poulton, Martin, & Kaspi 2007; Kremen et al., 2007; Pitman et al., 2006). Needless to say, sorting out which biological changes are risk factors and which are consequences has tremendous implications for whether the field uses prevention or treatment as an approach to the problems that patients face.

DO TREATMENTS CHANGE THE BRAIN?

Psychopharmacology

The public largely appreciates the value of psychopharmacology in the treatment of mental illness. The idea that some individuals with mental illness have a "chemical imbalance" that is remedied by replacing those

chemicals is an oversimplified but powerful explanation for sufferers. However, many continue to eschew the use of medications even in common disorders like depression and even when there may be clear benefits in mood for a percentage of those who take them. As clinicians are aware, it is the *idea* of taking medications that often is a hindrance. Many take psychiatric medication only with reluctance because doing so is an implicit acknowledgement of their illness. In addition, despite the improving side effect profiles of many prescriptions, many medications produce physical symptoms that patients consider intolerable.

Psychotherapy

Psychotherapy, to a large extent, faces the opposite problem. There may be no physical side effects, but potential patients struggle to envision how psychotherapy could possibly work. Although most individuals have an appreciation for how "getting it off your chest" might be of some help, the explanation often seems trite in light of the enormity of concerns and feelings that often coincide with trauma. A cathartic release might make most feel better if one is frustrated about one's job, but most individuals appreciate the fact that such an approach is insufficient in the face of PTSD.

The public does not yet appreciate that psychotherapy is a lot more than a cathartic exercise, and partly works by changing the chemistry of one's brain. There has been growing evidence to indicate that effective psychotherapy changes brain activity. For example, obsessive compulsive disorder is associated with hyperactivity in the frontal lobe as measures by increased blood flow in individuals who suffer from the disorder. Effective psychotherapy can reduce this hyperactivity, returning brain activity in this region to levels consistent with healthy individuals (Schwartz, Stoessel, Baxter, Martin, & Phelps, 1996). In a study of individuals with depression, both a psychotherapy approach and a psychopharmacological approach decreased prefrontal and left anterior cingulate activity and increased left temporal lobe metabolism—alterations that are consistent with recovery from the disorder (Brody et al., 2001). It is exactly these types of studies that have such promise in convincing the public that psychotherapists have treatments that work. Similarly designed studies are under way, investigating the efficacy of cognitive behavioural treatments for PTSD (Kimble, Rothbaum, Resick, Griffin, & Brewin, 2003), and initial work has demonstrated neuroanatomical changes with pharmacological treatments in PTSD (Bremner et al., 2005; Bremner & Vermetten, 2004).

MIDPOINT SUMMARY

In the past decade, there have been great advances in our understanding of the neurobiology of PTSD.

- There is considerable evidence showing that traumatic experiences affect the brain in a manner that may result in PTSD symptoms in some individuals.
- The functioning of brain structures like the thalamus are affected by extreme stress and likely result in symptoms such as dissociative responses.
- Hormonal changes, such as those in the cortisol system, may be implicated in traumatic amnesia and memory fragmentation.
- Neurotransmitter abnormalities such as those in the NE system are likely to result in intrusive memories and flashbacks.
- Treatments, whether pharmacological or psychotherapeutic, have biological correlates, that are informative regarding the process of recovery.
- Concurrent with this increasingly neurobiological evidence is the likelihood for change with respect to how patients perceive themselves and, in addition, are perceived by society.

RELEVANCE TO PERCEPTION AND PATIENT CARE

This type of work is important in one unanticipated fashion: A neurobiological understanding of PTSD is antithetical to the damage that the controversies have caused. The controversies have left two lasting impressions of the PTSD patient that have relevance to patient care: (a) Individuals with PTSD are mentally and morally weak, and/or (b) individuals with PTSD are faking or exaggerating their symptoms (Baxter & Rogers, 2003; Slater, 2003). However, if clinicians and patients have an appreciation for the fact that traumatic experiences can change the brain in the true PTSD patient, then the responsibility for the disorder shifts from the *person* to the *event,* and thus dispels the notions of mental inferiority, malingering, or a lack of effort.

Take, for example, the notion that PTSD patients are mentally weak. The roots of this idea come from impressions as well as evidence that most individuals who have been traumatized do not develop PTSD. Therefore, some erroneously conclude that the PTSD sufferer is mentally

inferior, cannot handle stress, or is not trying hard enough to move on with his or her life (Baxter & Rogers, 2003). However, if science can demonstrate that a traumatic event changes the neurobiology of the PTSD patient in a manner that is related to their subsequent difficulties, this would imply that there is nothing "wrong" with the patient that he or she can be responsible for. Before the trauma, the patient was PTSD free. After the trauma, the patient's neurobiology changed, and the PTSD symptoms developed. Thus the "blame" lies with the event and not the individual. As clinicians and as patients, such a perception is preferable to the alternatives.

Recent evidence for constitutional vulnerabilities (De Bellis, 2001) does run the risk of further stigmatizing PTSD patients as "mentally weak." However, there are two points to keep in mind. First, most preexisting vulnerability factors (e.g., an adverse childhood, neurological soft signs, or cognitive deficits) cannot be blamed on the patient. These factors are something that a patient is born with or experienced. Second, the vulnerability factors in no way alter the fact that the trauma changed their brain in some significant way that led to the development of PTSD. Hence, whether these factors would have led to PTSD in someone other than a particular patient is essentially irrelevant. Had it not been for the trauma, this patient would not have developed PTSD.

In addition, establishing that trauma changes the brain is equally important to dispelling the perception that trauma survivors are malingering or exaggerating their symptoms. If it can be demonstrated that trauma-related brain changes are directly related to the PTSD presentation, then the symptoms take on a new level of reality. If society can accept the trauma→brain change→PTSD pathway in the same way they accept the germ→infection→cold pathway, for example, it will help to establish the legitimacy of the PTSD presentation. Such a pathway cannot completely eradicate the fact that some individuals will fake PTSD or exaggerate their symptoms, in the same way the common cold pathway doesn't eradicate the possibility that someone might fake or exaggerate a cold. However, no one doubts that the common cold exists. Establishing the trauma→brain change→PTSD pathway will go a long way in reminding the public that most individuals who develop PTSD aren't expressing symptoms because they are faking it; they are expressing symptoms because they have been changed in the most fundamental of ways.

It is also important to keep in mind that controversies associated with the "false memory" syndrome and "pseudoscientific" treatments have also hurt the image of those treating trauma survivors. Attrition rates are

high early in therapy for the treatment of many disorders, and PTSD is no exception. Patients won't stay in treatment unless they believe that treatment may work, and negative media coverage of failure in "regression" treatments and families destroyed by recovered memories only further discourage potential patients (Slater, 2003). However, given that early neurobiological evidence indicates that treatments change the brain in a way that remediate symptoms, this can be very convincing information. It is a potent way to convince the public and patients that there are treatments that work.

It is important to realize that although psychotherapy is widely accepted in certain circles, it maintains a level of mysticism for the public at large (Jorm et al., 2006). How psychotherapy changes some for the better is still a mysterious process. In addition, trauma therapists are faced with the additional burden of trying to convince a survivor that talking about their trauma, a request that is counterintuitive to almost every instinct, is critical to the process of recovery. By highlighting studies demonstrating brain changes as a result of treatment, there is at least evidence that some treatments not only work, but *we know how and why they work*. Although forms of treatment without a strong neurobiological foundation are sure to continue, the fact that there are treatments available in which the mystique is removed will certainly improve the perception of PTSD treatment providers. Although it is possible that a patient might be aware that unorthodox treatments for trauma might still be available, this will go along way to establishing the credibility of trauma therapist to have neurobiologically validated treatments used by many in the field.

In the current climate of PTSD skepticism and the public's general acceptance of biological models for mental illness, the credibility of PTSD is partially linked to the extent to which it is seen as a neurobiological disorder (Kimble & Kaufman, 2004). Until that time, in the eyes of the public, PTSD will be a disorder someone is faking, has imagined, or is exaggerating. There are increasing numbers of studies demonstrating that trauma is prevalent in today's society, but it is the very fact that there are so many of these studies that weakens the argument for those *not* in the trauma field. Although such studies are designed to demonstrate the extent and severity of trauma and PTSD in today's society, they often serve to "water down" one's case in the eyes of the public.

It is for such reasons that understanding the neurobiology of the disorder is so critical. Neurobiological explanations for PTSD are still in their earliest stages, but they at least provide a tangible explanation for certain elements of the disorder (Bremner, Vermetten, Southwick, Krystal, &

Charney, 1998; Southwick et al., 1993; Yehuda, 2002). The facts that these theories are truly explanatory and give some order to a terribly complex phenomena, give them tremendous potency. For example, if there is any PTSD-related finding that has caught the imagination of the public and garnered considerable press, it is the concept that "stress changes the brain" (Bremner, 2002). Although clearly too general to be scientifically useful, this "sound bite" is very socially persuasive and draws power through its facile, explanatory quality. Accordingly, this phrase has founds its way to newspaper and magazine headlines throughout the world ("Baboon Key to Human Stress," 2001; "Stress Changes Adolescent Brain," 2003).

PERCEPTIONS OF THE SELF

The experience of a trauma involves elements of chance, personality, constitution, and interpersonal support that will never by completely understood through neurobiological findings alone. Although ultimately all these elements produce change at the neurobiological level, how the multiple neural systems interact to produce an individual's idiosyncratic response to the trauma requires knowledgeable professionals dedicated to the care of individuals deeply affected by a tragic experience.

Information about the neurobiology of a trauma, therefore, must be presented to the patient with great clinical care and with a thorough understanding of the patient. For example, discussing a brain-based model for PTSD is not information that would be valuable to share in the early stages after a trauma. Given that most individuals don't develop PTSD after a trauma, we can assume that pathological brain changes, if they occur at all in individuals who do not develop a chronic response, are changes that are not clinically relevant. Informing someone who presents with symptoms a few weeks after the trauma that their brain has been altered by the experience is unlikely to have any therapeutic benefit and may even lead to a self-fulfilling psychological response. Knowledge about the neurobiology of trauma and PTSD is only clinically valuable in cases of a chronic response in which the symptoms have not abated. For such individuals, knowing that their neurobiology has been changed in a way that can be unlearned provides not only an understanding of their current experiences but also a rationale for recovery and treatment.

This is not to say that viewing PTSD as a neurobiological disorder will not have its drawbacks. What will ultimately be a complex, brain-based

model of PTSD will hardly be straightforward for the public and the trauma survivor to grasp. This complexity is likely to result in some trite phrase that grossly misrepresents the realities of the disorder. In the same way that sufferers of bipolar disorder often see themselves as victims of a "salt imbalance," the trauma survivor may come to see themselves as having "stress-induced brain damage." The clinician will need to work closely with patients to help them understand that their brain has been changed, but not in a manner that treatment cannot help. What the brain has learned, as a function of the trauma, can be unlearned through the process of treatment.

In addition, a brain based model of PTSD presumes that there are some individuals who are biologically vulnerable. The fact that less than 30% of those exposed to even the most horrific traumas (e.g., combat, rape) develop PTSD suggests that trauma may interact with constitutional factors to cause PTSD in some cases but not others. As alluded to previously, research is beginning to indicate that those who go on to develop PTSD may have genetic, uterine, and childhood experiences that make them more vulnerable to the development of psychopathology after a trauma (Teicher, Anderson, Polcar, Anderson, & Navalta, 2002). Now, trauma survivors, in addition with coping with the idea that the world is a very dangerous place, have to cope with the idea that there may be something constitutionally wrong with them—that they are defective in some way. This is certainly a bitter medicine.

However, the fact that the vulnerability is neurobiologically based does have one critical implication: It minimizes the ability of PTSD patients to blame themselves. Guilt and shame are endemic in the PTSD patient and can be devastating to self-image, appropriate entitlement, and recovery. It is important for the PTSD patient to appreciate that one's constitution interacts with the idiosyncrasies of the trauma in a way that will either result in PTSD or it won't. It certainly isn't their fault. There are some individuals who *will not* have vulnerable interactions, and they *will not* develop PTSD. Those who *do not* develop PTSD after a trauma are not necessarily tougher, they are just more fortunate.

One of the facts about trauma survival is that almost all survivors have some PTSD-like symptoms in the early days and weeks after the trauma. In some individuals these symptoms recede; in others they either continue or worsen. Because the typical pattern of recovery often entails an early period of symptom presentation for all individuals, those who recover attribute their success to their efforts to "just put it out of my mind and move on" (Baxter & Rogers, 2003, p. 12). Often one will hear individuals

who have survived a trauma but do not develop PTSD state that "I had to just learn to move on." This language, often shared by psychotherapists, gives the impression that recovery is an entirely volitional issue.

Although a neurobiological perspective would acknowledge that effort is an important part of one's eventual *recovery* (Schwartz & Begley, 2003), the *development* of PTSD is fundamentally a different enterprise. There is little evidence indicating that there is anything a PTSD patient can do to change their level of vulnerability or the early course of the disorder although clinical evidence indicates that substance abuse and social isolation (avoidance) are likely to hinder the natural process of recovery (Koenen, Stellman, Stellman, & Sommer, 2003). This point of view, the idea that a patient has less control over whether they *develop* PTSD, when filtered down to the public at large will have tremendous benefits to our patients. This is because the single most important factor in the development of chronic PTSD is a lack of social support (Brewin, Andrews, & Valentine, 2000). A neurobiological perspective on PTSD will likely lead to increased social support in a manner similar to how neurobiological models of schizophrenia and depression led to softening of criticism of those struggling with those disorders. If the public begins to appreciate that *PTSD* development is not volitional, then the view toward the trauma survivor will be far more sympathetic. A fully educated public will stop telling trauma survivors to "just get over it" and start encouraging them toward treatments that we know can work.

If trauma survivors have historically been willing to attend "therapies" that can only be described as pseudoscientific, they will certainly come for empirically validated treatment that have a known biologically based rationale behind them. Fear of self-disclosure and shame are two of the primary reasons individuals do not seek treatment (Corrigan, 2004; Vogel & Wester, 2003). With a softening of public criticism of trauma survivors, more are certain to seek treatment. Although we can tell our patients that there is less we can do about the *development* of PTSD, we can also assure them that there is a lot that can be done about *recovery*. This infusion of hope in combination with increasingly sophisticated treatments is sure to lead to rates of recovery that will be resistant to any type of controversy.

REFERENCES

Adamec, R. (1997). Transmitter systems involved in neural plasticity underlying increased anxiety and defense—Implication for understanding anxiety following traumatic stress. *Neuroscience and Biobehavioral Reviews, 21*, 755–765.

American Psychiatric Association. (1980). *The diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: American Psychiatric Press.

American Psychiatric Association. (1987). *The diagnostic and statistical manual of mental disorders* (3rd rev. ed.). Washington, DC: American Psychiatric Press.

Anisman, H., Griffiths, J., Matheson, K., Ravindran, A. V., & Merali, Z. (2001). Posttraumatic stress symptoms and salivary cortisol levels. *American Journal of Psychiatry, 158*(9), 1509–1511.

Aston-Jones, G., Ennis, M., Pieribone, J., Nickel, W. T., & Shipley, M. T. (1986). The brain nucleus locus coeruleus: Restricted afferent control over a broad efferent network. *Science, 234*, 734–737.

Aston-Jones, G., Valention, R., & Van Bockstaele, E. J. (1994). Locus coeruleus, stress, and PTSD: Neurobiological and clinical parallels. In M. M. Murburg (Ed.), *Catecholamine function in posttraumatic stress disorder: Emerging concepts* (pp. 17–62). Washington, DC: APA Press.

Baboon key to human stress. (2001, February 18). *BBC News*. Retrieved on May 14, 2008, from http://news.bbc.co.uk/1/hi/in_depth/sci_tech/2001/san_francisco/1173924.stm

Baldwin, S. A., Williams, D. C., & Houts, A. C. (2005). The creation, expansion, embodiment of posttraumatic stress disorder. *The Scientific Review of Mental Health Practice, 3*, 33–52.

Baxter, S., & Rogers, L. (2003, March 2). Stiff upper lip beats stress counselling. *London Times*. Retrieved on May 14, 2008, from http://www.timesonline.co.uk/tol/news/uk/article1114963.ece

Berridge, C. W., & Waterhouse, B. D. (2003). The locus-coeruleus-noradrenergic system. *Brain Research Reviews, 42*, 33–84.

Bremner, J. D. (2001). Hypotheses and controversies related to effects of stress on the hippocampus: An argument for stress-induced damage to the hippocampus in patients with posttraumatic stress disorder. *Hippocampus, 11*(2), 75–81.

Bremner, J. D. (2002). Does stress damage the brain: *Understanding trauma-related disorders from a neurological perspective*. New York: W.W. Norton.

Bremner, J. D., Mletzko, T., Welter, S., Quinn, S., Williams, C., Brummer, M., et al. (2005). Effects of phenytoin on memory, cognition and brain structure in post-traumatic stress disorder: A pilot study. *Journal of Psychopharmacology, 192*, 159–165.

Bremner, J. D., Randall, P., Scott, T. M., & Bronen, R. A. (1995). MRI-based measurement of hippocampal volume in patients with combat-related posttraumatic stress disorder. *American Journal of Psychiatry, 152*, 973–981.

Bremner, J. D., & Vermetten, E. (2004). Neuroanatomical changes associated with pharmacotherapy in posttraumatic stress disorder. In R. Yehuda & B. McEwen (Eds.), *Biobehavioral stress response: Protective and damaging effects* (pp. 154–157). *Annals of the New York Academy of Sciences, 1032*, 154–157. New York, NY: New York Academy of Sciences.

Bremner, J. D., Vermetten, E., Southwick, S. M., Krystal, J. H., & Charney, D. S. (1998). Trauma, memory and dissociation: An integrative formulation. In J. D. Bremner & C. R. Marmar (Eds.), *Trauma, memory, and dissociation* (pp. 365–402). Washington, DC: American Psychiatric Press.

Bremner, J. D., Vythilingam, M., Vermetten, E., Southwick, S. M., McGlashan, T., Nazeer, A., et al. (2001). MRI and PET study of deficits in hippocampal structure and

function in women with childhood sexual abuse and posttraumatic stress disorder. *American Journal of Psychiatry, 158,* 1248–1251.

Breslau, N., Kessler, R. C., Chilcoat, H. D., Schultz, L. R., Davis, G. C., & Andreski, P. (1998). Trauma and posttraumatic stress disorder in the community: The 1996 Detroit area survey of trauma. *Archives of General Psychiatry, 55*(7), 626–632.

Breslau, N., Lucia, V. C., & Alvarado, G. F. (2006). Intelligence and other predisposing factors in exposure to trauma and posttraumatic stress disorder: A follow-up study at age 17 years. *Archives of General Psychiatry, 63,* 1238–1245.

Brewin, C. R., Andrews, B., & Valentine, J. D. (2000). Meta analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *Journal of Consulting and Clinical Psychology, 68,* 748–766.

Brody, A. L., Saxena, S., Stoessel, P., Gillies, L. A., Fairbanks, L. A., Alborzian, S., et al. (2001). Regional brain metabolic changes in patients with major depression treated with either paroxetine or interpersonal therapy: Preliminary findings. *Archives of General Psychiatry, 58,* 631–640.

Cahill, L., & McGaugh J. L. (1998). Mechanisms of emotional arousal and lasting declarative memory. *Trends in Neuroscience 21,* 294–299.

Carrion, V. G., Weems, C. F., Eliez, S., Patwardhan, A., Brown, W., Ray, R. D., et al. (2001). Attenuation of frontal asymmetry in pediatric posttraumatic stress disorder. *Biological Psychiatry, 50,* 943–951.

Corrigan, P. (2004). How stigma interferes with mental health care. *American Psychologist, 59,* 614–625.

De Bellis, M. D. (2001). Developmental traumatology: The psychobiological development of maltreated children and its implications for research, treatment, and policy. *Development and Psychopathology, 13,* 539–564.

De Bellis, M. D., Keshayan, M. S., Shifflett, H., Ivenger, S., Beers, S. R., Hall, J., et al. (2002). Brain structures in pediatric maltreatment-related posttraumatic stress disorder: A sociodemographically matched study. *Biological Psychiatry, 52,* 1066–1078.

Elzinga, B. M., Bakker, A., & Bremner, D. J. (2005). Stress induced cortisol elevations associated with impaired delayed but not immediate recall. *Psychiatry Research, 134*(3), 211–223.

Elzinga, B. M., Schmahl, C. S., Vermetten, E., van Dyck, R., & Bremner, J. D. (2003). Higher cortisol levels following exposure to traumatic reminders in abuse related PTSD. *Neuropsychopharmacology, 28,* 1656–1665.

Gilbertson, M. W., Paulus, L. A., Williston, S. K., Gurvits, T. V., Lasko, N. B., Pitman, R., et al. (2006). Neurocognitive function in monozygotic twins discordant for combat exposure: Relationship to posttraumatic stress disorder. *Journal of Abnormal Psychology, 115,* 484–495.

Gilbertson, M., Shenton, M. E., Ciszewski, A., Kasai, K., Lasko, N., Orr, S. P., et al. (2002). Smaller hippocampal volume predicts pathologic vulnerability to psychological trauma. *Nature Neuroscience, 5,* 1242–124.

Golier, J., & Yehuda, R. (1998). Neuroendocrine activity and memory-related impairments in posttraumatic stress disorder. *Development & Psychopathology, 10,* 857–869.

Gurvitz, T. V., Metzger, L. J., Lasko, N. B., Cannistraro, P. A., Tarhan, A. S., Gilbertson, M. W., et al. (2006). Subtle neurologic compromise as a vulnerability factor for combat-related posttraumatic stress disorder. *Archives of General Psychiatry, 63,* 571–576.

Gurvitz, T. V., Shenton, M. E., Hokama, H., & Ohta, H. (1996). Magnetic resonance imaging study of hippocampal volume in chronic, combat-related posttraumatic stress disorder. *Biological Psychiatry, 40,* 1091–1099.

Heim, C., Ehlert, U., & Hellhammer, D. H. (2000). The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. *Psychoneuroendocrinology, 25,* 1–35.

Henry, J. (2002). Biological basis of the stress response. *Integrative Physiological and Behavioral Science, 27,* 66–83.

Het, S., Ramlow, G., & Wolf, O. T. (2005). A meta-analytic review of the effects of acute cortisol administration on human memory. *Psychoneuroendocrinology, 30,* 771–784.

Holsboer, F. (2000). The corticosteroid receptor hypothesis of depression. *Neuropsychopharmacology, 23,* 477–501.

Horowitz, M. J., Weiss, D. S., & Marmar, C. (1987). Diagnosis of posttraumatic stress disorder. *Journal of Nervous and Mental Disease, 175*(5), 267–268.

Jorm, A. F., Barney, L. J., Christensen, H., Highet, N. J., Kelly, C. M., & Kitchener, B. A. (2006). Research on mental health literacy: What we know and what we still need to know. *Australian and New Zealand Journal of Psychiatry, 40,* 3–5.

Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. B. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry, 52,* 1048–1060.

Kimble, M. O., & Kaufman, M. L. (2004). Clinical correlates of neurological change in PTSD: An overview of critical systems. In L. A. Flashman (Ed.), *Psychiatric clinics of North America: Cognitive and neurological impairments in psychiatric disorders* (pp. 49–66). Philadelphia: W.B. Saunders.

Kimble, M. O., Rothbaum, B., Resick, P., Griffin, M., & Brewin, C. (2003, November). *Does psychotherapy change the brain?* Panel discussion at the Annual Meeting of the International Society of Traumatic Stress Studies, Chicago.

Koenen, K. C., Moffitt, T. E., Poulton, R., Martin, J., & Caspi, A. (2007). Early childhood factors associated with the development of post-traumatic stress disorder: Results from a longitudinal birth cohort. *Psychological Medicine, 37,* 181–192.

Koenen, K. C., Stellman, J. M., Stellman, S. D., & Sommer, J. F J. (2003). Risk factors for course of PTSD among Vietnam veterans: A 14 year follow-up of American Legionannaires. *Journal of Consulting and Clinical Psychology, 71,* 980–986.

Kremen, W. S., Koenen, K. C., Boake, C., Purcell, S., Eisen, S. A., Franz, C. E., et al. (2007). Pretrauma cognitive ability and risk for posttraumatic stress disorder: A twin study. *Archives of General Psychiatry, 64,* 361–368.

Krystal, J. H., Bennet, A., Bremner, J. B., Southwick, S., & Charney D. (1995). The cognitive neuroscience of dissociation and altered memory functions in PTSD. In M. J. Friedman, D. S. Charney, & A. Y. Deutsch (Eds.), *Neurobiological and clinical consequences of stress: From normal adaptation to PTSD* (pp. 239–269). New York: Raven.

Krystal, J. H., Bremner, J. D., D'Souza, D. C., Anand, A., Southwick, S. M., & Charney, D.S. (1999). The emerging biology of dissociative states. In A. Shalev, R. Yehuda, & A. McFarlane (Eds.), *The international handbook of human response to trauma* (pp. 307–320). New York: Plenum.

Kuhlmann, S., Piel, M., & Wolf, O.T. (2005). Impaired memory retrieval and psychosocial stress in healthy young men. *Journal of Neuroscience, 25,* 2977–2982.

Kulka, R. A., Schlenger, W. E., Fairbank, J. A., Hough, R. L., Jordan, B. K., Marmar, C. R., et al. (1990). *Trauma in the Vietnam war generation: Report of findings from the national Vietnam veterans readjustment study.* New York: Brunner/Mazel.

Lanius, R. A., Williamson, P. C., Densmore, M., Boksman, K., Gupta, M. A., Neufeld, R. W., et al. (2001). Neural correlates of traumatic memories in posttraumatic stress disorder: A functional MRI investigation. *American Journal of Psychiatry, 158,* 1920–1922.

Lindauer, R. J., Vlieger, E. J., Jalink, M., Olff, M., Carlier, I., Majoie, C.B., et al. (2004). Smaller hippocampal volume in Dutch police officers with posttraumatic stress disorder. *Biological Psychiatry, 56*(5), 356–363.

Mazzeo, S., Beckham, J. C., Witvliet, C., Feldman, M. E., & Shivy, V. A. (2002). A cluster analysis of symptom patterns and adjustment in Vietnam combat veterans with chronic PTSD. *Journal of Clinical Psychology, 58,* 1555–1571.

McEwen, B. S. (2002). The neurobiology and neuroendocrinology of stress: Implications for post-traumatic stress disorder from a basic science perspective. In R. Yehuda (Ed.), *Psychiatric clinics of North America: Recent advances in study of biological alterations in post-traumatic stress disorder* (pp. 469–494). Philadelphia: W.B. Saunders.

McHugh, P. R., & Treisman, G. (2007). PTSD: A problematic diagnostic category. *Journal of Anxiety Disorders, 21,* 211–222.

McNally, R. J. (2003). Progress and controversy in the study of posttraumatic stress disorder. *Annual Review of Psychology, 54,* 229–252.

Metzler, B. S., Hidalgo, R., Connor, K. M., & Davidson, J. R. T. (2000). Posttraumatic stress disorder: Prevalence, health care use and costs, and pharmacologic considerations. *Psychiatric Annals, 30*(12), 722–730.

Pitman, R. K., Gilbertson, M. W., Gurvits, T. V., May, F. S., Lasko, N. B., & Metzger, L. J. (2006). Clarifying the origin of biological abnormalities in PTSD through the study of identical twins discordant for combat exposure. *Annals of the New York Academy of Sciences, 1071,* 242–254.

Resnick, H. S., Kilpatrick, D. G., Dansky, B. S., Saunders, B. E., & Best, C. L. (1993). Prevalence of civilian trauma in post traumatic stress disorder in a representative national sample of women. *Journal of Consulting and Clinical Psychology, 61,* 984–991.

Schwartz, J., & Begley, S. (2003). The mind and the brain: Neuroplasticity and the power of mental force. *Journal of Nervous and Mental Disease, 191,* 765–766.

Schwartz, J. M., Stoessel, P. W., Baxter, L. W., Martin, K. M., & Phelps M. E. (1996). Systematic changes in cerebral glucose metabolic rate after successful behavior modification treatment of obsessive-compulsive disorder. *Archives of General Psychiatry, 53,* 109–113.

Scott, W. (1990). PTSD in *DSM-III*: A case in the politics of diagnosis and disease. *Social Problems, 37,* 294–310.

Shin, L. M., Orr, S. P., Carson, M. A., Rauch, S. L., Macklin, M. L., Lasko, N. B., et
(2004). Regional cerebral blood flow in the amygdala and medial prefrontal cort
during traumatic imagery in male and female Vietnam veterans with PTSD. *Archi
of General Psychiatry, 61,* 168–176.

Slater, L. (2003, February 23). Repress yourself. *New York Times.* Retrieved on May 14, 20(
from http://query. nytimes.com/gst/fullpage.html?res=9C0DE2D81F3AF930A15751C(
9659C8B63

Southwick, S. M., Bremner, J. D., Rasmusson, A., Morgan, C. A., Arnsten, A., & Cha
ney, D. S. (1999). Role of norepinephrine in the pathophyiology and treatment
PTSD. *Biological Psychiatry, 46,* 1192–1204.

Southwick. S. M., Krystal, J. H., Morgan, A. C., Johnson, D., Nagy, L. M., Nicolaou, A
et al. (1993). Abnormal neuroadrenergic functioning in PTSD. *Archives of Gener
Psychiatry, 50*(4), 266–274.

Spitzer, R. L., First, M. B., & Wakefield, J. C. (2007). Saving PTSD from itself in *DSM
IV. Journal of Anxiety Disorders, 21,* 233–240.

Stress changes adolescent brain. (2003, November 9). *The Age.* Retrieved on May 14, 20(
from http://www.theage.com.au/articles/2003/11/09/1068329409631.html? from=storyrl

Teicher, M. H., Anderson, S. L., Polcar, A., Anderson, C. M., & Navalta, C. P. (2002). Dev
opmental neurobiology of childhood stress and trauma. In R. Yehuda (Ed.), *Rece
advances in the study of biological alterations in PTSD* (pp. 397–426). Philadelphia: W.
Saunders.

Vogel, D. L., & Wester, S. R. (2003). To seek help or not to seek help: The risks of sel
disclosure. *Journal of Counseling Psychology, 50,* 351–361.

Warshaw, M. G., Fierman, E., Pratt, L., & Hunt, M. (1993). Quality of life and dissoci
tion in anxiety disorder patients with histories of trauma or PTSD. *American Journal
Psychiatry, 150,* 1512–1516.

Wignall, E. L., Dickson, J. M., Vaughan, P., Farrow, T., Wilkonson, I. D., Hunter, M. I
et al. (2004). Smaller hippocampal volume in patients with recent onset posttraumat
stress disorder. *Biological Psychiatry, 56,* 832–836.

Wong, S. (2002). PTSD: Advances in psychoimmunology. In R. Yehuda (Ed.), *Rece
advances in the biological alterations in PTSD* (pp. 369–384). Philadelphia: W.B. Saunde

Woodward, S. H., Kaloupek, D. G., Streeter, C. C., Kimble, M. O., Reiss, A. L., Eliez, S
et al. (2006). Hippocampal volume, posttraumatic stress disorder, and alcoholis
American Journal of Psychiatry, 163(4), 674–681.

Woodward, S. H., Kaloupek, D. G., Streeter, C. C., Kimble, M. O., Reiss, A. L., Eliez, S
et al. (2007). Brain, skull, and cerebrospinal fluid volumes in adult posttraumatic stre
disorder. *Journal of Traumatic Stress, 20,* 760–774.

Yehuda, R. (2002). Current status of cortisol findings in PTSD. *Psychiatric Clinics
North America, 25,* 341–368.

Yehuda, R., Giller, E. L., Southwick, S., Lowy, M., & Mason, J.W. (1991). Hypothalam
pituitary-adrenal dysfunction in posttraumatic stress disorder. *Biological Psychiatr
30,* 1031–1048.

Yehuda, R., Kahana, B., Binder-Byrnes, K., Southwick, S. M., Mason, J. W., & Giller, I
L. (1995). Low urinary cortisol excretion in Holocaust survivors with posttraumat
stress disorder. *American Journal of Psychiatry, 152,* 982–986.

Zatzick, D. F., Marmar, C. R., Weiss, D. S., Browner, W. S., Metzler, T. J., Golding, J. M., et al. (1997). PTSD and functioning and quality of life outcomes in a nationally representative sample of male Vietnam veterans. *American Journal of Psychiatry, 154,* 1690–1695.

Zigmond, M. J., Finlay, J. M., & Sved, A. F. (1995). Neurochemical studies of central neuroadrenergic responses to acute and chronic stress. In M. J. Friedman, D. S. Charney, & A. Y. Deutch (Eds.), *Neurobiological and clinical consequences of stress: From normal adaptation to PTSD* (pp. 45–60). Philadelphia: Lippincott-Raven.

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BOOK REVIEW

DISASTER MENTAL HEALTH SERVICES: A PRIMER FOR PRACTITIONERS (SERIES IN PSYCHOLOGICAL STRESS). Myers, D & Wee, D. F. (2004). *New York: Routledge. Hardcover: 312 pages. ISBN 1583910638. List Price: $59.95*

Disaster Mental Health Services: A Primer for Practitioners is a rich practice-oriented text that incorporates recent trends in this growing specialty. The numerous case examples reflect the authors' accumulated wealth of experience, clearly derived from time spent in the trenches of disaster's aftermath. This volume is divided into three sections: Disaster Concepts and Roles; Services, Program, and Workers; and New Issues and Challenges.

Major topics that every disaster mental health (DMH) practitioner must be familiar with are addressed. Myers and Wee review the characteristics and stages of disasters and subsequent intervention implications, consider the possible needs and attributes of specific populations, and underscore the importance of practitioner self-care. The authors describe their own model of DMH intervention (CODE-C DMHSM), advocate for the use of debriefing and Critical Incident Stress Management (CISM), and summarize findings from the groundbreaking 2002 National Institute of Mental Health (NIMH) report *Mental Health and Mass-Violence: Evidence-Based Early Psychological Interventions for Victims/Survivors of Mass Violence* in the context of new issues and challenges posed by terrorism and large-scale events.

Myers and Wee present essential DMH "take-home points." They also emphasize the intricate, collective nature of disasters, which increases the complexity of intervening during such trying times. In addition, the authors stress the importance of disaster preplanning, community needs

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