

Running Head: THE NEUROBIOLOGY OF POSTTRAUMATIC STRESS
DISORDER

The Neurobiology of Posttraumatic Stress Disorder

Elizabeth G. Vermilyea

Abstract

Under normal circumstances, the human stress response is geared toward adapting to a perceived threat. When the stress response is over activated or chronically activated, the body and brain can become altered and sensitized to new stressful stimuli. By studying the neurobiology of posttraumatic stress disorder, researchers and psychologists can better understand the impact of traumatic stress and more effective ways of treating it.

Table of Contents

Table of Contents	3
Introduction	4
The Traumatized Brain	5
Effects on biological rhythms	7
Effects on emotional systems	8
Effects on learning, memory, cognition, perception	10
Trauma and the body	11
The Clinical Implications of PTSD	12
Alterations in sense of self and worldview	12
Substance Abuse	16
Somatization	18
Multimodal Treatment of PTSD	18
Conclusion	20
References	21

The Neurobiology of Posttraumatic Stress Disorder

Stress is endemic to human life. Individuals expect to experience stressful situations that activate the autonomic nervous system and produce a host of physical, physiological, and emotional responses (Sapolsky, 1994; Bremner, 1995; Brunello, 2001; Bremner, 2006). To a large extent, stress is an appraisal-based experience with interpretations of events leading to either mild or strong responses (Sapolsky, 1994). However, there are some stressful situations that by virtue of being prolonged or chronic and severe can create lasting impressions and changes in an individual's physiology and psyche. Combat, child abuse and neglect, and domestic violence are examples of such stressors and can lead to disorders of extreme stress. This paper will endeavor to delineate the nature of the stress response, the neurobiological impact of traumatic experiences, and some of the ways individuals can recover from stress disorders.

Under normal circumstances, the human stress response is geared toward adapting to a perceived threat. These days, such threats may include the potential loss of a job, a car accident, or more severe types of interpersonal stress like domestic violence or combat. In general, the body responds according to a dose-response relationship; the more stress one experiences, the more likely one is to show a reaction. The more severe the stressor, the more extreme the response will be (Wilson, Friedman & Lindy, 2004). A traumatic event or situation creates psychological trauma when it overwhelms the individual's perceived ability to cope, and leaves that person fearing pain, death, or insanity. The individual feels emotionally, mentally, and physically overwhelmed during the

event. The circumstances of the event commonly include (1) abuse of power and betrayal of trust by the abuser, and (2) feelings of being trapped, helplessness, pain, confusion, and/or loss on the part of the victim. The body responds to stress by activating the general adaptation syndrome (GAS). The GAS is marked by an alarm state where the adrenal glands secrete epinephrine, norepinephrine, and cortisol. Changes in glucose metabolism make energy more available to the system. The heart rate climbs and blood flow increases. Cortisol is secreted by the adrenal cortex. As a glucocorticoid, cortisol has a powerful effect on glucose metabolism. Since nearly every cell in the body contains glucocorticoid receptors, the effects of the stress response can be far reaching (Carlson, 2007). The next phase is the resistance stage where the body remains on the alert, and stress hormones continue to be released. The final phase is the exhaustion stage whereby the stress response has overtaxed body systems, and the body needs to recharge itself (Kassin, Fein, & Markus, 2008). In the short-term, the stress response is a good way to manage a threat, but in the long-term, it breaks down body systems and can compromise the immune system.

The Traumatized Brain

Animal studies show that both positive and negative events early in life can influence neurobiological development and subsequent gene expression (Bremner, Southwick, & Charney, 1999; Broderick & Blewitt, 2006). The organization and functional capacity of the brain depends on a sequence of neurogenesis and environmentally stimulated gene expression that is unfortunately highly vulnerable to extreme stress and associated metabolic

changes (Anda, Felitti, Bremner, Walker, Whitfield, & Perry, et al., 2006; Broderick & Blewitt, 2006). Early stressors related to maternal separation show lasting effects on stress response systems. Repeated exposure to stressors early in life lead to persistent effects in the hypothalamic-pituitary-adrenal (HPA) axis, locus coeruleus and norepinephrine, benzodiazepine, serotonin, dopaminergic, neuropeptide systems, as well as brain memory systems including the hippocampus, amygdala, and prefrontal cortex (Bremner & Vermetten, 2001; Anda et al., 2006).

The sympathetic nervous system (SNS) is a first responder in a stressful situation. The brain is also mobilized via the endocrine system. The SNS is involved in the release of epinephrine and cortisol, glucose and other chemicals that aid in mobilization of the stress response. The degree of the response of the SNS may begin locally with the release of norepinephrine and extend to a global response involving the secretion of epinephrine and cortisol and activation of the hypothalamic-pituitary-adrenal (HPA) axis (Sapolsky, 1994; Bremner, 1995; Brunello, 2001; Bremner, 2006). Epinephrine is the short acting hormone, and glucocorticoids are the longer acting hormones that can cause many problems if they are chronically activated (Falconer, Bryant, Felmingham, Kemp, Gordon & Peduto, et al., 2008).

Early and chronic stressors cause long-term increases in glucocorticoid responses to stress in addition to decreased genetic expression of cortisol receptors in the hippocampus. Further, there is increased corticotropin-releasing factor (CRF) in the hypothalamus (Anda et al., 2006). These responses can lead

to a general dysregulation of the HPA system. This is significant since abnormally high levels of cortisol can damage neurons in the hippocampus (Solomon & Heide, 2005, Bremner, 2006). Excessive concentrations of adrenal hormones also serve to depress the immune system and contribute to physiological arousal. The excessive physiological arousal associated with posttraumatic stress disorder (PTSD) interferes with the regulation of autonomic responses. In cases of chronic stress and PTSD, individuals show lower cortisol levels, presumably due to the constant activation of the stress response system and the inability of the body to replenish needed hormones (Solomon & Heide, 2005).

Effects on biological rhythms

Sleep deprivation, interruption, and nightmares are PTSD sequelae that can lead to severe personal distress (Mellman, Pigeon, Nowell, & Nolan, 2007). The individual avoids sleep to avoid nightmares and ultimately suffers either more nightmares later due to the rebound effects of REM sleep (Carlson, 2006) or an amplified limbic system response brought on by sleep deprivation. According to Yoo, Gujar, Hu, Ferenc, and Walker (2007), sleep deprivation over time can impair a number of functions including immune regulation and metabolic control. A more significant finding for those diagnosed with PTSD is that under conditions of sleep deprivation, the increased magnitude of limbic activity was associated with losses in prefrontal control. The hyper limbic response also amplified reactions to negative emotional stimuli. Therefore, those with PTSD who are already sensitized to negative emotional stimuli are likely to experience

more negative emotional stimuli with greater amygdala reactivity. This connection further disrupts the individual's ability to replace traumatic associations with new learning and, at the very least, benign associations.

Effects on emotional systems

The rational brain with its intricate systems of association, learning, memory, and organization is poorly equipped to rid itself of emotions, thoughts, and impulses (Vasterling & Brewin, 2005; van der Kolk, 2006). Neuroimaging studies of individuals in highly emotional states show that intense emotions such as fear, sadness, and anger cause increased activity in subcortical brain regions and reduced blood flow in the frontal lobe. According to van der Kolk (2006), these data support the notion that when experiencing intense emotion, it is difficult for individuals to organize a modulated behavioral response.

The limbic system controls much of the human emotional response with the amygdala determining the emotional significance of incoming stimuli (Blumenfeld, 2002; Carlson, 2007). Corticolimbic dopamine and noradrenergic pathways modulate prefrontal cortical function under conditions of increasing cognitive or emotional demand, including persistent distress states, tasks involving high levels of cognitive challenge, and working memory tasks (Brady, 2005). With persistent distress states and the accompanying sleep deprivation that occurs with PTSD, the prefrontal cortex may not function properly in its role of affect modulation and cognitive controls (Falconer et al., 2008). A variety of early stressors, including maternal deprivation, result in increased glucocorticoid response to subsequent stressors (Bremner, 2006). This sensitization nearly

guarantees that those with PTSD will experience distress more often and over react to those experiences. The chronically overwhelming emotions often render individuals unable to use emotions as guides for effective action. Alexithymia describes this inability to identify the meaning of physical sensations and muscle activation. Without the ability to recognize physiological responses and activations to emotional content, individuals tend to be out of touch with their own needs and therefore unable to address them effectively. A corollary to this deficit is the tendency for those individuals to be unable to appreciate the meaning of emotional states in others as well. Such deficits lead to severe interpersonal difficulties and sometimes to relationship failures. According to van der Kolk (2006), since both neurochemistry and emotions are activated in order to bring about action dysregulation of these systems can bring about problematic or embarrassing actions in the service of managing a perceived threat. Thus emotional dysregulation begets behavioral dysfunction, which contributes to greater emotional dysregulation.

Additional consequences of PTSD include the prevalence of other co-occurring conditions such as depression. Hypercortisolemia is associated with stress-related hippocampal lesions and also with a subgroup of patients with depression and is more common in patients with trauma histories (De Bellis, Hooper, & Sapia, 2005). This depression is somewhat distinct from major clinical depression in that it is less responsive to traditional medications used to treat depression (Brunello, 2001).

Effects on learning, memory, cognition, perception

The hippocampus, a brain region involved in learning and memory, is particularly sensitive to the effects of stress. Although the hippocampus has the capacity to grow new neurons in adulthood, stress inhibits neurogenesis and memory function (Anda et al., 2006; Bremner, 2006). The hippocampus is a major target organ for glucocorticoids and is particularly sensitive to metabolic changes (Bremner & Vermetten, 2001; Carlson 2007). There is a well-established relationship between the presence of high levels of glucocorticoids released during stress and damage to the hippocampus and subsequent memory deficits (Bremner & Vermetten, 2001; Vasterling & Brewin, 2005; Bremner, 2006; van der Kolk, 2006). These high levels of glucocorticoids are also associated with deficits in new learning.

The extreme, repeated, and intermittent stressors that some traumatized persons have undergone have a very distinct impact on learning and memory. In some ways, the degree of disturbance in how one perceives and encodes experiences can be a measure of trauma (Allen, 1996; McEwan, 2000). Disturbances occur in the distortion and fragmentation of narrative memory, visual memory, sensory memory, emotional memory and behavioral memory (Bremner, Krystal, Southwick & Charney, 1995). Alterations may also be seen in sensory and social perception.

Traumatized persons may experience intrusive recollections of their traumatic experiences (flashbacks). These may appear similar to psychotic

hallucinations but are in fact different. Traumatized persons may also misread social cues, anticipating danger when there is none, and failing to recognize danger when it is near. In this way they may perceive helpers as malevolent, and may not be able to readily accept help (Allen, 1999; Sakvitne, Gamble, Pearlman & Lev, 2000; Vermilyea, 2000).

Contrary to what many believe, these reactions are not necessarily under volitional control. According to van der Kolk (2006), the traumatized brain reacts very specifically to traumatic reminders. When exposed, subjects show cerebral blood flow to subcortical structures such as the amygdala and anterior temporal pole accompanied by deactivation of the left anterior prefrontal cortex, especially Broca's area. What this means is that behaviorally, traumatized persons who are reminded of traumatic events experience an emotional surge and a lack of activation around verbal communication.

Trauma and the body

Individual's body (as well as his or her mind) remains in a constant state of hyperarousal and fear. For this reason, trauma also manifests in a startling array of physical complaints such as: gastrointestinal distress, headaches, migraines, muscle tension, chronic pain, gynecological complaints and sometimes in stress related disorders like chronic fatigue and fibromyalgia (Bremner, 2001; Anda et al., 2006).

The Clinical Implications of PTSD

The traumatized person may exhibit a number of significant personality characteristics as a result of his or her experiences. The personality formed in an environment of coercive control is not well adapted to adult life (Herman, 1992). Common adaptations to trauma include numbing, avoidance, detachment, dissociation, suicidality, self-harm, substance abuse, isolation. Each of these adaptations is an effort to equalize the difference between the traumatic experience and the survivor's ability to manage it (Vermilyea, 2000).

In the case of childhood trauma and particularly abuse and neglect, trauma that happens at the hands of caregivers is doubly destructive because it destroys the attachment relationship that the child would normally depend on to manage traumatic experiences (Perry et al., 1995; Schore, 2002; 2003). Many survivors of childhood trauma have experienced both the overwhelming emotional and physiological arousal of abuse and the absence of adequate soothing and comforting (De Bellis, & Thomas; 2003; De Bellis, 2005). This can derail the development of healthy coping skills and lead to dependence upon problematic adaptations that may affect the survivor for years to come.

Alterations in sense of self and worldview

Trauma deeply affects one's frame of reference as manifested by changes in identity, world-view, and spirituality. These alterations may lead traumatized persons to look at themselves and the world through a "trauma colored" lens. They may take in information only as it confirms their outlook and expectation of danger and victimization. They may be unable to recognize neutral or even

positive experiences if they fail to mesh with the trauma-focused viewpoint (Allen, 1999; Sakvitne et al., 2000; Vermilyea, 2000).

Trauma affects connections by isolating and alienating the victim/survivor. Trauma may lead to internal disconnection where the victim/survivor is so numb that he or she cannot even connect with his or her own thoughts or feelings. It may lead to disconnection in relationships because the victim/survivor feels different, changed, damaged, or similar feelings. The disconnecting power of traumatic stress is particularly insidious since relationships and secure connections are the best antidote for the experience and yet may become the most difficult to use (Allen, 1999).

Alterations in feelings and connections can be accompanied by changes in sense of self as deserving life, love and kindness. Many victim/survivors feel responsible for their traumatic experiences. Many are, in fact, blamed as well. The isolation and devastation of victimization can lead victim/survivors to conclude that they are now no longer valued by others, lovable, or able to recover from their experiences. Changes in sense of self worth may be seen as poor self-care, high risk behavior, self-denigrating speech, suicidality, self-harm, and/or an inability to feel confident about positive outcomes (Saakvitne et al., 2000).

Alterations in management of feelings may be seen in the individual's inability to identify, tolerate, modulate or integrate emotions. As a result, they may present extremes of emotional experience: either numb or overwhelmed with feelings. Such people are often discredited because numbness may be

interpreted as being unaffected by the victimization or being “cold.” Emotional volatility may be referred to as “hysteria” or being “out of control.” This too is discrediting. It is essential to understand that both numbness and emotional volatility are natural responses to trauma and victimization. The person who is unable to manage feelings may be irritable, display angry outbursts, spontaneous crying, flat emotions, and/or emotional displays that appear incongruous in a particular situation.

Trauma alters a person’s ability to rely on his or her own intelligence, insight, sense of perspective, and ability to foresee and weigh consequences. As such, many victim/survivors struggle with decision-making and judgment. The degree of desperation the individual feels may be directly related to his or her access to judgment: the greater the desperation, the harder it is to access judgment skills of intelligence, insight, and the ability to take into account potential consequences of one’s actions (Saakvitne et al., 2000).

Trauma deeply affects individuals’ beliefs and psychological needs, especially regarding the realms of safety, trust/dependence, esteem, intimacy/connection, and control. Each of these areas represents a core relational construct that determines how a person conducts him or herself in the world and in relationships. Alterations in these realms may manifest in terms of difficulties with safety. Some individuals are unable to manage personal safety, inability to recognize danger signs in relationships, lack of concern for personal safety, active self-harm (Allen, 1999; Sakvitne et al., 2000; Vermilyea, 2000). They may engage in a wide variety of highly risky behavior such as use of

substances, unprotected and/or promiscuous sexual activities (especially after sexual trauma), or reckless driving.

Traumatized persons often struggle with trust and dependence. They may demonstrate an inability to break away from dangerous relationships due to dependence, an inability to trust helpers, an inability to determine who and how much to trust, difficulty trusting one's self and one's instincts about relationships and the world at large, and an inability to connect with others in a mutually satisfying manner. Difficulties with trust and dependence naturally lead to struggles with esteem and intimacy. Many traumatized individuals profess an inability to know themselves or feel known by others. Some feel unworthy, dirty, sinful or toxic (Allen, 1999; Sakvitne et al., 2000; Vermilyea, 2000).

One way to manage the intensity of reactions to trauma is to take control. Thus over-controlled behavior is common, including obsessions or compulsions, checking, keeping one's personal environment highly regimented and predictable. The other side of the control dilemma yields under-controlled behavior including the inability to take reasonable responsibility in one's own life accompanied by a pervasive feeling of helplessness.

Common adaptations to trauma include numbing, avoidance, detachment, dissociation, suicidality, self-harm, substance abuse, isolation, all of which result in excessively risky behavior. Each of these adaptations is an effort to equalize the difference between the traumatic experience and the survivor's ability to handle it. Obviously, the younger the victim, the greater the gap between the impact of the event and the ability to handle it.

Substance Abuse

Traumatic events are very frequent in the life histories of addicted individuals and trauma issues affect progress at all stages of recovery. Traumatic events can precede substance abuse in childhood or adulthood. Traumatic events may also be precipitated by substance abuse: the drug-using lifestyle increases the likelihood of additional traumatic experiences (Brady, & Sinha, 2005). Substance use is thought to be one method that victim/survivors use to distance themselves from internal emotional, physical and spiritual pain. Traditional substance abuse treatment may be less effective or ineffective with trauma survivor clients, particularly in cases where trauma preceded the addiction. Effective treatment for this population will take into account the function of the substance abuse and offer alternative methods of meeting the needs that have historically been met through the use of substances.

When the anesthetic effects of substance are taken away, the survivor is left with overwhelming awareness of the painful emotional states, images and cognitions accompanying unresolved trauma. Typically, the survivor does not have coping skills adequate to the task of managing those states, hence the original onset of substance use and abuse. For such clients, a rigid insistence on immediate and complete abstinence is unrealistic and will likely lead to treatment failure, reinforcing the survivor's negative self-concept.

A reliable and valid protocol with good outcome data, the 25-session Seeking Safety program (Najavits, 2002) advocates five key principles in its overview. Firstly, safety is the priority. When a client has active substance abuse

and PTSD the most urgent clinical need is the establishment of safety. Safety is a *process* and may take time to establish. Discontinuing substance use, self-harm, risk-taking, suicide attempts and minimizing HIV risk are core aspects of safety that are intertwined with trauma issues. Letting go of dangerous relationships is another long-term safety issue for many victim/survivors.

Secondly, integrated treatment of PTSD and substance abuse means simultaneous treatment of both disorders for recovery. Present focused treatment is marked by extensive education about why substance abuse and PTSD frequently co-occur and exploration into the interactivity of the symptoms of both disorders (e.g., using cocaine to cope with flashbacks). It is important for clients to understand that healing from each disorder requires attention to both disorders. The third principle is a focus on ideals refers to sessions focus on positive ideals to combat beliefs acquired through trauma and/substance abuse. Examples include sessions on honesty, commitment, and self-care. The fourth principle is a focus on content areas including cognitive, behavioral, interpersonal, and case management. Relational issues are explored during all phases of the treatment. Cognitive/ behavioral techniques are employed, and case management addresses real life issues that can be stumbling blocks for victim/survivors with substance abuse histories.

Finally, attention to the therapist process involves emphasizing building an alliance, therapist coping and self-care, sharing control with the client, and working with countertransference. This is one example of a modified treatment for PTSD and substance abuse.

Somatization

Somatization is a condition marked by numerous physical complaints with no medical origin. Emotional conflicts and pain are manifested in the person's body. Kessler et. al. (1995), in the National Comorbidity Survey, found that somatization was 90 times more likely in those with PTSD than in those without PTSD. This shows an important but frequently overlooked connection between traumatic stress and physical complaints. Close attention should be paid to differentiate real medical illnesses from conversion symptoms.

Multimodal Treatment of PTSD

PTSD is varied, but the literature and outcome studies support several modalities. The National Center for PTSD (NCPTSD) advocates the use of cognitive behavioral therapy, exposure therapy, eye movement desensitization and reprocessing (EMDR) and the use of medication.

The goal of cognitive therapy consists of gaining understanding about traumatic stress symptoms and changing thought patterns associated with the traumatic events an individual may have experienced (Vermilyea, 2000). For example, one goal may be to reduce survivor-guilt feelings in an airline flight attendant who was not on one of the downed planes on 9/11 because she called out sick. Her guilt stemmed from the fact that another attendant worked in her place and was killed. Therapists also employ exposure therapy. In exposure therapy, the goal is to talk about traumatic events and experience the physiological and psychological arousal that accompanies those memories (Wilson, Friedman, & Lindy, 2004). Over time, exposure desensitizes the

individual and disarms the fear response.

EMDR is a technique used along with exposure and eye movements to reduce the intensity of some of the most disturbing aspects of traumatic memories. Although researchers are still studying the mechanisms of EMDR, there are good data that support the technique with those who have had single incident trauma experiences such as rape or natural disasters (NCPTSD, 2009).

In addition to therapeutic techniques, researchers advocate the use of selective serotonin reuptake inhibitors (SSRIs) in the treatment of PTSD (Vermetten et al., 2003). The theory behind the use of these drugs is that the sluggish serotonin system accounts for some of the depression and the impulsivity associated with PTSD.

Other therapy techniques may include brief psychodynamic psychotherapy which focuses on identification of triggers, coping with intense emotions from the past, being more aware of thoughts and feelings so as to avoid becoming overwhelmed, and improving one's sense of self-worth (Wilson, Friedman, & Lindy, 2004). Family therapy is sometimes necessary in cases where the spouse or children are affected by the individual with PTSD. It can be very important for family members to understand the symptoms of PTSD and to learn how to help.

Conclusions

PTSD is one of the most serious public mental health issues facing Americans today. With the wars in Afghanistan and Iraq and the vast numbers of troops involved, history tells us to expect high volumes of returnees with PTSD

symptoms. Although combat is the quintessential cause of PTSD, in the United States, more individuals, especially women and children, develop PTSD from assault, abuse, and domestic violence than any other cause (Bremner & Vermetten, 2001). Given the prevalence of violence in the home and in the world at large, it has become more and more essential for those who provide treatment for PTSD to move beyond the therapeutic couch and into the laboratory and the study of the neurobiology of trauma. With advances in brain imaging, researchers are now able to track the activities of stress hormones and neurotransmitters. They can perceive alterations in neural volume in significant brain regions implicated in PTSD. Not only are these findings supporting the notion that PTSD is a physiological condition, they provide crucial direction for treatment strategies. With continued research and application of findings in clinical settings the state of the art of PTSD treatment is becoming less of a scattergun approach and more scientific. Applying that science with skill and compassion in therapeutic settings is the best hope for effective treatment of PTSD.

References

- Allen, J. (1996). Neurobiological basis of posttraumatic stress disorder: Implications for patient education and treatment. *Bulletin of the Menninger Clinic*, 60(3), 377. Retrieved September 3, 2009, from Academic Search

Premier database.

- Allen, J. (1999). *Coping with Trauma*. American Psychiatric Press, Inc., Washington D.C.
- Anda, R. F., Felitti, V. J., Bremner, J. D., Walker, J. D., Whitfield, C., Perry, B. D., et al. (2006). The enduring effects of abuse and related adverse experiences in childhood. *European Archives of Psychiatry & Clinical Neuroscience*, 256(3), 174-186. doi:10.1007/s00406-005-0624-4
- Brady, K. T., & Sinha, R. (2005). Co-occurring mental and substance use disorders: The neurobiological effects of chronic stress. *American Journal of Psychiatry*, 162(8), 1483-1493. Retrieved from <http://dx.doi.org.library.capella.edu/10.1176/appi.ajp.162.8.1483>
- Bremner, J. D., Krystal, J. H., Southwick, S. M., & Charney, D. S. (1995). Functional neuroanatomical correlates of the effects of stress on memory. *Journal of Traumatic Stress*, 8(4), 527-553. Retrieved from <http://search.ebscohost.com.library.capella.edu/login.aspx?direct=true&db=sih&AN=9511170502&site=ehost-live&scope=site>
- Bremner, J. D., Southwick, S. M., & Charney, D. S. (1999). *The neurobiology of posttraumatic stress disorder: An integration of animal and human research*. Boston: Allyn and Bacon.
- Bremner, J. D., & Vermetten, E. (2001). Stress and development: Behavioral and

biological consequences. *Development and Psychopathology*, 13(3), 473-489. Retrieved from

<http://dx.doi.org.library.capella.edu/10.1017/S0954579401003042>

Bremner, J. D. (2006). Stress and brain atrophy. *CNS and Neurological Disorders - Drug Targets*, 5(5), 503-512. Retrieved from

<http://dx.doi.org.library.capella.edu/10.2174/187152706778559309>

Broderick, P. & Blewitt, P. (2006) *The life span: Human development for helping professionals* (2nd ed.). Pearson.

Brunello, N., Davidson, J. R. T., Deahl, M., Kessler, R. C., Mendlewicz, J., Racagni, G., et al. (2001). Posttraumatic stress disorder: Diagnosis and epidemiology, comorbidity and social consequences, biology and treatment. *Neuropsychobiology*, 43(3), 150-162. doi:10.1159/000054884

Carlson, N. R. (2007). *Physiology of Behavior*. (9th Ed.) Amherst, MA: Pearson Education.

Centonze, D., Palmieri, M. C., Boffa, L., Pierantozzi, M., Stanzione, P., Brusa, L., et al. (2005). Cortical hyperexcitability in post-traumatic stress disorder secondary to minor accidental head trauma: A neurophysiologic study. *Journal of Psychiatry & Neuroscience*, 30(2), 127-132. Retrieved from <http://search.ebscohost.com.library.capella.edu/login.aspx?direct=true&db=aph&AN=16199476&site=ehost-live&scope=site>

De Bellis, M. D., Hooper, S. R., & Sapia, J. L. (2005). *Early trauma exposure and*

the brain New York: Guilford Press.

- De Bellis, M. D., & Thomas, L. A. (2003). Biologic findings of post-traumatic stress disorder and child maltreatment. *Current Psychiatry Reports*, 5(2), 108-117. Retrieved from <http://dx.doi.org.library.capella.edu/10.1007/s11920-003-0027-z>
- Falconer, E., Bryant, R., Felmingham, K. L., Kemp, A. H., Gordon, E., Peduto, A., et al. (2008). The neural networks of inhibitory control in posttraumatic stress disorder. *Journal of Psychiatry & Neuroscience*, 33(5), 413-422. Retrieved from <http://search.ebscohost.com.library.capella.edu/login.aspx?direct=true&db=aph&AN=34080671&site=ehost-live&scope=site>
- Herman, J. (1992). *Trauma and Recovery*. Basic Books, New York.
- Kassin, S., Fein, S. & Markus, H. R. (2008) *Social psychology*. Houghton Mifflin.
- Kessler, R., Sonnega, A., Bromet, E., Hughes, M. & Nelson, C. (1995). *Posttraumatic stress disorder in the National Comorbidity Survey*. *Archives of General Psychiatry*, 52(12), 1048-1060.
- McEwen, B. S. (2003). Mood disorders and allostatic load. *Biological Psychiatry*, 54(3). Retrieved from [http://dx.doi.org.library.capella.edu/10.1016/S0006-3223\(03\)00177-X](http://dx.doi.org.library.capella.edu/10.1016/S0006-3223(03)00177-X)
- Mellman, T. A., Pigeon, W. R., Nowell, P. D., & Nolan, B. (2007). Relationships between REM sleep findings and PTSD symptoms during the early aftermath

of trauma. *Journal of Traumatic Stress*, 20(5), 893-901. doi:10.1002/jts.20246

Najavits, L. (2002). *Seeking safety: A treatment manual for PTSD and substance abuse*. New York: Guilford.

National Center for PTSD (2009). *Treatment for PTSD fact sheet*. Retrieved from http://ncptsd.va.gov/ncmain/ncdocs/fact_shts/fs_treatmentforptsd.html on September 12, 2009.

Perry, B. D., Pollard, R. A., Blakley, T. L., Baker, W. L., & Vigilante, D. (1995). Childhood trauma, the neurobiology of adaptation, and "use-dependent" development of the brain: How "states" become "traits". *Infant Mental Health Journal*, 16(4), 271-291. Retrieved from [http://dx.doi.org.library.capella.edu/10.1002/1097-0355\(199524\)16:4<271::AID-IMHJ2280160404>3.0.CO;2-B](http://dx.doi.org.library.capella.edu/10.1002/1097-0355(199524)16:4<271::AID-IMHJ2280160404>3.0.CO;2-B)

Saakvitne, K., Gamble, S., Pearlman, L. & Lev, B. (2000) *Risking connection: A manual for working with adult survivors of childhood trauma*. Baltimore: Sidran Press.

Sapolsky, R. (1994). *Why zebras don't get ulcers: A guide to stress, stress-related diseases, and coping*. New York: Freeman and Company.

Schore, A. N. (2002). Dysregulation of the right brain: A fundamental mechanism of traumatic attachment and the psychopathogenesis of posttraumatic stress disorder. *Australian & New Zealand Journal of Psychiatry*, 36(1), 9-30. doi:10.1046/j.1440-1614.2002.00996.x

- Schore, A. N. (2003). *The effects of relational trauma on right brain development, affect regulation, and infant mental health* New York: Norton.
- Solomon, E. P., & Heide, K. M. (2005). The biology of trauma: Implications for treatment. *Journal of Interpersonal Violence*, 20(1), 51-60. Retrieved from <http://dx.doi.org.library.capella.edu/10.1177/0886260504268119>
- Van der Kolk, B. A. (2006). Clinical implications of neuroscience research in PTSD. *Annals of the New York Academy of Sciences*, 1071, 277-293. Retrieved from PILOTS Database
- Vasterling, J. J., & Brewin, C. R. (2005). *Neuropsychology of PTSD: Biological, cognitive, and clinical perspectives* New York: Guilford Press.
- Vermetten, E., Vythilingam, M., Southwick, S. M., Charney, D. S., & Bremner, J. D. (2003). Long-term treatment with paroxetine increases verbal declarative memory and hippocampal volume in posttraumatic stress disorder. *Biological Psychiatry*, 54(7), Retrieved from [http://dx.doi.org.library.capella.edu/10.1016/S0006-3223\(03\)00634-6](http://dx.doi.org.library.capella.edu/10.1016/S0006-3223(03)00634-6)
- Vermilyea, E. (2000). *Growing beyond survival: A self-help toolkit for managing symptoms of traumatic stress*. Baltimore: Sidran Press.
- Wilson, J., Friedman, M. & Lindy, J. (2004). *Treating psychological trauma & PTSD*. Guilford.
- Yoo, S., Gujar, N., Hu, P., Ferenc, A., & Walker, M. (2007). The human emotional brain without sleep – a prefrontal amygdala connection. *Current*

Biology, 17(20), 877-878.