Earn CE credit.
Visit http://learning.counseling.org
to purchase and complete the test online.

Received 12/21/10
Revised 06/06/11
Accepted 06/20/11

Theory

Cognitive Behavioral Therapy and the Treatment of Posttraumatic Stress Disorder: Where Counseling and Neuroscience Meet

Ryan A. Makinson and J. Scott Young

There is increasing evidence to support the biological basis of mental disorders. Subsequently, understanding the neurobiological context from which mental distress arises can help counselors appropriately apply cognitive behavioral therapy and other well-researched cognitive interventions. The purpose of this article is to describe the neurobiological context underlying the formation and treatment of posttraumatic stress disorders, a mental disorder frequently encountered by counselors, from a cognitive therapy framework.

Keywords: neuroscience, neurobiology, posttraumatic stress disorder, cognitive behavioral therapy, neurobiological basis of behavior

Recent changes to the Council for Accreditation of Counseling and Related Educational Programs (2009) accreditation standards include the need for counselors-in-training to understand the neurobiological basis of behavior, which marks a new direction for the training of professional counselors who have historically reacted ambivalently toward medical models for understanding client concerns and treatments. Yet recent findings in neuroscience actually support the verbally based interventions that counselors typically use in treatment; therefore, there is much to be gained by counselors and counselor educators in understanding the basics of human neurobiology and how commonly used counseling interventions intervene on these biological systems. The National Institute of Mental Health (2010) stated in a recent strategic plan that "Important discoveries in areas such as genetics, neuroscience, and behavioral science largely account for the substantial gains in knowledge that have helped us to understand the complexities of mental illnesses and behavioral disorders over the past 15 years" ("Introduction," para. 4).

Given the increasingly biological focus of mental health research, the practicing counselor is faced with the task of understanding and using the emerging mental health treatments and explaining to clients, to reimbursing agencies, and to the broader public how counseling fits within the medically dominated mental health culture. Some counselors have long reacted ambivalently toward the pathologically oriented diagnostic categories of the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV;* American Psychiatric Association, 1994) system and the medication-dominated world of psychiatry. For example, the contrasting viewpoints on this issue were published in the *Journal of Counseling & Development* between Allen and Mary Ivey (1998, 1999) and Scott Hinkle (1999). Ivey and Ivey (1998) argued for a developmental interpretation to the *DSM-IV*, opposing what they called the "*pathological view*" (p. 334) of the manual. According to Ivey and Ivey, disorders could be viewed through a positive development tradition to lie not within the individual but within the contextual systems in which a person lives. Subsequently, disorders are viewed as a "logical response to a developmental history" (Ivey & Ivey, 1999, p. 484). By contrast, Hinkle (1999) argued that because anxiety and depressive disorders "are the most common clinical symptoms associated with presentation to counseling" (p. 475), the counseling profession is weakened if counselors shy away from direct participation in the *DSM* nomenclature and treatment parlance. As Hinkle indicated, "mental disorders according to the medical model describe disease processes, not people" (p. 475). Regardless of the reader's philosophical perspective, practicing counselors know participation in medical and psychiatric systems is necessary at times. Also, recent discoveries in the field of neuroscience are providing evidence that interventions often used by counselors have direct physiological impact on client neurobiology (Kennedy et al., 2007; Linden, 2006). For example, Felmingham et al. (2007) demonstrated significant differences in brain activity before and after 8 weeks of exposure therapy, which correlated

Ryan A. Makinson, Yerkes National Primate Research Center, Emory University; **J. Scott Young,** Department of Counseling and Educational Development, University of North Carolina at Greensboro. Correspondence concerning this article should be addressed to Ryan A. Makinson, Yerkes National Primate Research Center, Emory University, 954 Gatewood Road, Atlanta, GA 30329 (e-mail: makinson7888@gmail.com).

with a reduction in posttraumatic stress disorder (PTSD) symptom severity. Similarly, Paquette et al. (2003) found that cognitive behavioral therapy (CBT) alters the activation and metabolism of specific brain regions following successful treatment of spider phobia. These findings, along with others (for a detailed review, see Beauregard, 2007; Frewen, Dozois, & Lanius, 2008), are significant because they support the techniques, interventions, and approaches used by counselors and provide a mechanism by which counseling positively affects brain physiology. Within the emerging physiologically based treatment milieu, counselors should be prepared to articulate how cognitive counseling interventions make measurable changes to the client. Although cognitive-behavioral-based approaches are effective in the treatment of a number of psychiatric illnesses, adult PTSD is arguably one of the best understood mental disorders from a neurological perspective. It thus presents a valuable model for exploring not only the basic tenets of neurobiology but also the mechanisms behind its successful treatment. Furthermore, PTSD is a disorder that counselors will likely encounter in practice.

PTSD is a mental disorder characterized by a sudden onset of symptoms due to environmental exposure to a psychologically stressful event such as war, natural disaster, or sexual victimization. Thus, it provides a clear example of how, even in adulthood, neurological adaptation (in this case maladaptive changes) can functionally "rewire" the brain in a short period of time, resulting in a sustained array of clinical symptoms. The diagnostic criteria for PTSD are a history of exposure to a traumatic event meeting two criteria and symptoms from each of three symptom clusters: intrusive recollections, avoidant/numbing symptoms, and hyperarousal symptoms. A fifth criterion concerns duration of symptoms and a sixth assesses functioning (American Psychiatric Association, 2000).

The National Comorbidity Survey Replication, conducted between February 2001 and April 2003 (Kessler et al., 2005), determined that the estimated lifetime prevalence of PTSD among American adults is 6.8%, with women (9.7%) twice as likely as men (3.6%) to have the disorder at some point in their lives. These findings are very similar to those of the first National Comorbidity Survey conducted in the early 1990s (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995), which was composed of interviews of a representative national sample of 8,098 Americans ages 15 to 54 years. In this earlier sample, the estimated prevalence of lifetime PTSD was 7.8% in the general population. As in the more recent survey, women (10.4%) were more than twice as likely as men (5%) to have PTSD at some point in their lives (Kessler et al., 2005; Kessler et al., 1995).

The Neurobiological Basis of Behavior

Even though many therapeutic approaches are considered effective for the treatment of PTSD, this review will evaluate the use CBT and other cognitive-based practices as a top-down approach for the treatment of PTSD. In general, CBT treatment strategies incorporate the use of higher order thought processes by directing a client's attention in a deliberate manner, with the intent to correct for problematic thought patterns and behaviors (David & Szentagotai, 2006; Paquette et al., 2003; Ressler & Mayberg, 2007). However, as a way to better understand how directed forms of thinking are in turn manifested into changes in behavior, approaching the treatment of psychiatric illnesses, particularly PTSD, from a cognitive neurological perspective is illuminating. Moreover, such an alternative perspective can enhance counselors' understanding of when and what cognitive-based techniques are most appropriate and effective, as well as refine and improve current counseling practices (David & Szentagotai, 2006; De Raedt, 2006; Harvey, Bryant, & Tarrier, 2003; Linden, 2006).

To begin with, thoughts and emotions are the psychological manifestation of physical processes occurring in the brain, which is made up of roughly 100 billion neurons. How these neurons communicate with one another is paramount to one's psychobiological functioning. At a basic level, a neuron can send either an excitatory or inhibitory message to another neuron. The receiving neuron then integrates this information with input from other neurons. If this receiving neuron receives enough excitatory input, it will then relay a message to the next neuron. If the connecting neurons frequently induce the receiving neuron into relaying its message, the connectivity is strengthened over time and decreases the threshold for transmitting additional messages. Patterns of this neuron-to-neuron connectivity form complex neural networks that make up specialized brain regions. Variations in the physical characteristics of these networks give rise to specific properties that define how each section of the brain functions and communicates with other brain regions. Two such brain regions commonly involved in mental disorders (and particularly PTSD) are the prefrontal cortex (PFC) and the amygdala.

The PFC is a highly developed region of the brain that controls working memory, decision making, attention, and impulsivity. The PFC also elaborates thought, determines personality, and regulates emotionality. Furthermore, specific areas of the PFC play a multi-integrative role in emotional learning and expression. The PFC functions much like the gatekeeper for judgment and inhibition and is one of the primary areas of the brain actively recruited during CBT. Equally important, the PFC is responsible for counterbalancing the activity of limbic regions of the brain, which are involved in the generation of base emotions (fear, anxiety, rage, etc.) and implementing the body's stress response systems. Finally, the PFC is not responsible for the creation of but rather the expression and lasting retention of personal experiences (Banks, Eddy, Angstadt, Nathan, & Phan, 2007; Bishop, 2007; Shin et al., 2005).

Integral to emotional self-regulation is the neurological cross talk between the PFC and the amygdala. The amygdala, as part of the limbic system, is a brain region that gauges the

salience of potentially threatening stimuli by pairing negative emotions (often fear) with a particular event. The amygdala aids in the generation, expression, and experience of a negative emotional reaction to an external situation that informs the rest of the body of potential danger (Shin et al., 2005). The amygdala, via its connections with the PFC, also plays a key role in the retention of emotional memories. Like the PFC, the neurocircuitry of the amygdala is highly connected with various brain regions. These connections are vital in the event of a threatening situation by allowing the amygdala to direct life-saving responses, for example, fleeing from a hungry lion. However, a hypersensitive or dysregulated amygdala can provoke inappropriate responses to particular stimuli or overrepresent trauma-related memories. In these instances, the body's natural protective and survival mechanisms become maladaptive, and anxiety is created when no threat exists. A traumatic and emotionally distressing experience presumably initiates psychogenesis (i.e., the origin and development of a mental disorder) by enhancing amygdala activity. This can also disrupt PFC-dependent executive control, which normally provides inhibitory regulation over the amygdala and overrides fear-related messages through top-down processing (Banks et al., 2007; De Raedt, 2006; Quirk & Mueller, 2007). A mental disorder commonly encountered by counselors that involves inappropriate amygdala activation is PTSD (Bishop, 2007; Ressler & Mayberg, 2007; Shin et al., 2005).

The Neurobiology of Fear and PTSD

In general, there is considerable variation in the extent to which certain brain regions can be altered after birth. Although some structures are hardwired at birth and remain relatively stable, the PFC and amygdala, among others, display a remarkable degree of neuroplasticity into adulthood (Katz et al., 2009; McClung & Nestler, 2007). Broadly speaking, neuroplasticity refers to the modification of preexisting circuitry or changes to the structural interconnectedness between neurons or brain regions as a result of experience (De Raedt, 2006; Martin & Kandel, 1996). The ability to change patterns or the strength of connectivity between neurons can be an adaptive advantage. For example, in some cases of head trauma or stroke, neuroplasticity allows other neurons to take on the responsibilities of damaged neurons, thus mitigating or preventing a loss in functional ability. Synaptic plasticity, however, can sometimes be maladaptive (Sapolsky, 2003).

The processes of psychogenesis and successful treatment of PTSD exemplify how neurological alterations can either benefit or hinder psychological health and well-being. With PTSD, exposure to psychological trauma (e.g., war, natural disaster, or sexual victimization) induces neuroplastic changes that disrupt proper regulation of fear-related emotions leading to an overactive and unregulated amygdala (Shin et al., 2005). PTSD (and other fear-related illnesses) follows four critical steps in the processes of psychogenesis and expression (Ressler & Mayberg, 2007).

Step 1 involves a genetic and environmental predisposition, making some individuals more susceptible, or resilient, to environmentally induced stressors.

Step 2 is the traumatic experience itself. The frequency and severity of the traumatic event(s) appears to positively correlate with an increased prevalence and severity of PTSD symptoms (Watson & Shalev, 2005). Knowledge of a patient's psychological history (e.g., child abuse or military experiences) and/or of the traumatic event itself could help a counselor intuitively gauge the likelihood of an individual developing PTSD. For example, a soldier who has served multiple tours of duty may be at a higher risk for PTSD than a soldier with less combat exposure. Counselors should also look for signs of acute stress disorder, because these symptoms appear to be a predictor of later onset PTSD (Bryant, Harvey, Dang, Sackville, & Basten, 1998).

Step 3 is coupled with Step 2, whereby short-term fear-related memories are consolidated into long-term storage in the hours, days, and weeks following a traumatic event. After the consolidation process occurs, newly formed connections are more resistant to disruption (Maguschak & Ressler, 2008). Finally, Step 4 is the expression of abnormal symptoms that characterize the psychiatric illness.

Because PTSD is for the most part a manageable disorder, intervention along any of these steps can produce positive results; however, the efficacy of applied treatments is highly dependent on when and what methods are used (Ressler & Mayberg, 2007). Descriptions of well-researched treatments for PTSD, in the following section, show efficacy for treating PTSD symptoms by ameliorating the neurological changes the disorder entails from the CBT framework.

The Range of CBT Approaches

Prolonged Exposure (PE) Therapy

PE therapy is a specific form of exposure therapy developed to treat PTSD (Hembree, Rauch, & Foa, 2003). Exposure therapy was developed from behavioral theories of learning, specifically from Mowrer's (1960) two-factor learning theory (Foa, Steketee, & Rothbaum, 1989), which states that fear and avoidance are learned through both classical and instrumental conditioning. When this model is applied to PTSD, symptoms develop in two ways: (a) as traumatic experiences become associated with similar neutral stimuli that subsequently begin to cause the same anxiety and (b) then as the individual's avoidant behaviors become learned responses (negative reinforcement) to that anxiety.

PE is a manualized treatment that consists of nine to twelve 90-minute sessions. Initial sessions are used to gather information about the individual's stressors, as well as for psychoeducation about the treatment approach. Cognitive behavioral relaxation techniques are often taught in the initial sessions. Remaining sessions are devoted to imaginal exposure to the traumatic events (i.e., having the client visualize a traumatic

event and then using CBT to gain control of his or her reactions to this recall), and in vivo exposure homework (i.e., safe exposure to a feared stimuli using CBT strategies to overcome anxiety) is often assigned. Both of these techniques share the goal of habituating the individual to anxious responses to cues associated with their trauma (Foa et al., 1989; Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010).

PE is considered to have strong research support for its efficacy as a treatment of PTSD. In a meta-analysis of 16 randomized controlled trials of PE, Powers et al. (2010) found consistent support for the efficacy of the treatment over control conditions. The Institute of Medicine (2008) took on a systematic review of research on treatments of PTSD, at the request of the Department of Veterans Affairs, and found moderate to high evidence to support PE's efficacy as a treatment. The Department of Veterans Affairs has since developed a national program to train mental health staff in providing PE treatments (Karlin et al., 2010). Change is achieved through the habituation of anxiety response to cues associated with trauma.

Trauma-Focused CBT (TF-CBT)

TF-CBT draws from many theoretical models, including CBT, attachment, person-centered, and family therapy. According to the Child Sexual Abuse Task Force and Research and Practice Core, National Child Traumatic Stress Network (2004), TF-CBT was initially developed to treat trauma associated with child sexual abuse and has since been expanded to address a broad range of traumatic experiences. Alternatively, Ehlers and Clark (2008) described the development of TF-CBT as drawing from theory as well as a combination of phenomenological, experimental, and treatment development studies. TF-CBT uses the cognitive behavioral interventions of cognitive restructuring and exposure to help a client identify and shift cognitions and behaviors that maintain his or her PTSD symptoms.

Ehlers and Clark (2008) outlined the process of treatment in terms of three TF-CBT goals.

Goal 1: Identify and change negative personal meanings related to the trauma. This is achieved through *updating trauma memories*, a process that has three parts: (a) identifying peak experiences of distress during the trauma through imaginal reexperiencing, writing, and discussion; (b) updating the impression that the client had at the time with new information gained since the trauma and/or the use of cognitive restructuring; and (c) incorporating the new information into the image of the trauma through imaginal reliving or reading his or her narrative.

Goal 2: Reduce reexperiencing by using techniques that elaborate the trauma memory. These techniques are imaginal reliving, writing detailed description of the trauma (e.g., a trauma narrative), revisiting the site of the trauma, and discriminating triggers related to the trauma (i.e., identifying triggers to anxious thoughts and then using CBT techniques to break the link between the triggers and the traumatic memory).

Goal 3: Dropping dysfunctional behaviors and cognitive strategies. Identify, discuss, and create change in the behaviors and cognitions that underlie the client's PTSD symptoms.

Ponniah and Hollon (2009) discussed 24 studies that involved the combination of cognitive restructuring and exposure therapy in comparison to waiting lists and other treatments including repeated assessments, self-help, stress management, and supportive counseling. These studies evaluated TF-CBT varying from four to 20 sessions. TF-CBT was found to be more effective in reducing PTSD symptoms than were waiting lists, minimal attention, repeated assessments, and a self-help booklet. In studies that used diagnosis as an outcome measure, between 47% and 95% of clients treated with TF-CBT no longer met PTSD criteria after treatment. Ponniah and Hollon also investigated several studies that explored the effectiveness of TF-CBT compared with exposure alone or cognitive restructuring alone and concluded that further research is needed. Seidler and Wagner (2006) completed a meta-analysis of seven studies comparing the use of TF-CBT with eye-movement desensitization and reprocessing (EMDR) in the treatment of PTSD. They found that although both treatments are effective in the treatment of adults, PTSD research does not indicate one to be superior to the other.

EMDR

EMDR was developed by Francine Shapiro in 1990 to treat symptoms associated with distressing (traumatic) memories. EMDR has since been used to treat a broad range of symptoms and concerns (Shapiro & Maxfield, 2002). EMDR was developed as a comprehensive integrative psychotherapy approach using structured protocols that draw from psychodynamic, cognitive behavioral, interpersonal, experiential, and body-centered therapies (Shapiro & Maxfield, 2002). Shapiro developed the Adaptive Information Processing (AIP) model as a guide for EMDR clinical practice. The AIP model focuses on fully processing information related to traumatic and/or distressing experiences; Shapiro asserted that these unprocessed memories are responsible for the symptoms of PTSD.

In EMDR, the hypothesis is that eye movements (and other methods for dual-attention stimuli) decrease the vividness of and affect related to traumatic or distressing memories (Shapiro & Maxfield, 2002). EMDR involves eight phases, and Phases 3 through 8 are often repeated in sessions (Shapiro & Maxfield, 2002). Depending on the client's symptoms and needs, it may take a few sessions or even months to complete the eight phases of treatment described next.

Phase 1: Full client history, assessment of client readiness for EMDR, develop treatment plan, inform client of standard procedures and "targets" or goals for EMDR.

Phase 2: Preparation and stabilization phase involving establishment of rapport, setting reasonable client expectations, psychoeducation focusing on client symptoms. Assist client in enhancing and developing a sense of safety, affect management, and self-control. Self-calming techniques, such as "safe place visualizations," are introduced at this time.

Phase 3: Begin processing the traumatic incident. The client describes the most vivid visual memories associated with the incident and identifies current irrational beliefs brought forth by the image. Memories are rated on a scale ranging from 1 (*feels completely false*) to 7 (*feels completely true*). The client experiences the visual combined with the irrational belief, which frequently draws out strong feelings. The client identifies these emotions and rates level of distress using the Subjective Unit of Disturbance (SUD; Benjamin et al., 2010) scale. The client then identifies and locates feelings and sensations in his or her body that are related to this traumatic image/thought/feeling.

Phase 4: The client focuses on the image and associated negative belief and bodily sensations while simultaneously moving his or her eyes from side to side for 15 or more seconds by following the counselor's finger movements. This practice is known as dual-attention stimuli. Hand tapping, auditory stimulation, or other methods may be used in lieu of eye movement. After a set of dual-attention stimuli, the client is invited to go "blank" and take a deep breath. The client is then invited to share the new material (images, thoughts, sensations, etc.), which is the focus of the next set. This process is repeated many times, and often clients report shifts in affect, thoughts, and bodily sensations. The AIP conceptualization views these shifts as connections between the dysfunctionally stored traumatic memories and new healthy adaptive information. The fourth phase concludes when the client reports a distress level of 0 (using the SUD scale) related to the incident.

Phase 5: Focus includes expression, integration of client insights, and self-acceptance. This phase begins after the traumatic memory can be recalled without distress. The client pairs the memory with a new positive cognition while experiencing dual-attention stimuli in order to increase strength and confidence in the new positive beliefs.

Phase 6: Any remaining tension the client experiences in his or her body while focusing on the image and positive cognition is processed until the tension is relieved.

Phase 7: If a memory has not been fully processed, then the self-calming resources developed in Phase 2 are strengthened. Clients are educated about the processing that may continue in dreams, insights, memories, and so on, and are encouraged to journal and draw upon the skills learned in Phase 2.

Phase 8: The reevaluation phase. At the beginning of each session, the client and counselor determine treatment effectiveness, stability, behavioral change, integration, and areas for further attention.

EMDR is thought to work through the use of dual-attention stimuli to process the stored experience of trauma such that clients are able to access new adaptive information and/or memories and create new connections within their memory network, thus experiencing a reduction in PTSD symptoms (Shapiro & Maxfield, 2002).

EMDR is considered an established evidence-based treatment for PTSD for both children and adults (Cukor, Difede, Spitalnick, Rizzo, & Rothbaum, 2009; Davidson & Parker, 2001; Rodenburg, Benjamin, de Roos, Meijer, & Stams, 2009). In 2009, Ponniah and Hollon conducted a review of empirically supported treatments for adult acute stress disorder and PTSD. They found 14 studies that met their inclusion criteria in which EMDR was compared with treatments such as waiting list, standard care, placebo, fluoxetine, stress management, and TF-CBT. In most of these trials, EMDR treatment ranged from one to 12 sessions. At the 6-month follow-up, these studies indicated EMDR had superior outcomes to psychopharmacology. Studies reviewed by Ponniah and Hollon that used diagnostic criteria as an outcome measure indicated that between 77% and 90% of the study participants who completed treatment and analysis did not meet the diagnostic criteria for PTSD after completion of EMDR treatment.

Other researchers assert that EMDR is among the best short-term treatments for PTSD and point to a series of meta-analyses of treatment for PTSD showing that EMDR has positive effects and also compares favorably with pharmacotherapy (Högberg et al., 2008; Powers et al., 2010). Still, the practice and elements of EMDR continue to inspire controversy (Amstadter, McCart, & Ruggiero, 2007).

Mindfulness-Based Cognitive Therapy (MBCT)

Mindfulness-based practices have been used over the past 30 years to assist clients in responding more effectively to both physiological and psychological afflictions. Mindfulness-based stress reduction, developed by Jon Kabat-Zinn (1990), has demonstrated positive results among patients post–heart attack, individuals with chronic pain, and those with anxiety disorders. Similarly, MBCT for depression, a structured training approach developed by Segal, Williams, and Teasdale (2002) to address relapse prevention, has shown good clinical efficacy. These approaches share the core technique that Kabat-Zinn described as "paying attention in a particular way: on purpose, in the present moment, and nonjudgmentally" (p. 4).

When coupled with training in basic CBT ideas (learning to observe one's thinking), MBCT assists the individual in disengaging from self-perpetuating mental habits of ruminating on negative thought sequences that lead to anxiety or depressive relapse.

All MBCT approaches stress a state of moment-to-moment awareness and attention to one's current being with the goal of shifting away from a *doing stance* (e.g., "I need to change this") to a *being stance* (e.g., "I accept what is happening"). During mindfulness-based practice, deep breathing relaxes the body while thoughts are encountered without the intention of changing them. Among individuals with anxiety disorders (e.g., PTSD), the shift in mental function needed is from falling deeply into the content of negative thoughts toward a gentle observation of the way that one's thoughts form patterns, lead to feelings, and create body sensations, all of which are precursors to relapse-related mind states. By learning to recognize without deeply indulging in these thought patterns, the individual gains control of them. The training that clients receive in various approaches to MBCT share similar structured content. For example, Segal et al. (2002) used an eight-session model to teach clients to pay attention to their thoughts, feelings, and body sensations; to be in the moment; to decenter when negative thought patterns emerge; to learn acceptance and nonattachment; to let go of unhelpful cycles; and to practice being rather than doing.

Research on the effectiveness of MBCT is promising. Segal et al. (2002) conducted a randomized clinical trial with 145 individuals who were depressed and who were at least 3 months postdepression, placing the participants into either a treatment-as-usual group or into an MBCT group. Segal et al.'s findings indicated that in the 60 weeks following an eight-session MBCT treatment, individuals receiving the MBCT were half as likely to relapse as those receiving the usual treatments (seeking help from a family doctor).

Repeatedly practicing mindfulness has been evaluated empirically and found to have long-lasting positive effects. Siegel (2007) indicated that a key skill in mindfulness practice is developing the ability to observe one's own mind. He labeled this ability as *mindsight,* and it is associated with gaining deep insight and empathy. Furthermore, the process of *neural integration* (i.e., prefrontal neurons reaching out to differentiated areas of the brain and body) is an essential underlying mechanism for a sustained sense of well-being.

Scientific evidence of the physiological impact of mindfulness practice is growing. For example, a study of mindfulness meditation among Buddhist monks possessing extensive meditation experience found they were able to sustain very high rates of gamma wave activity compared with a control group (Lutz, Greischar, Rawlings, Ricard, & Davidson, 2004). Gamma waves are patterns of neural activity primarily associated with consciousness, critical thought, perception, and various higher cognitive functions (Vanderwolf, 2000). These Tibetan monks, according to the authors of the 2004 study, had the greatest degree of high-amplitude gamma activity yet reported in the literature. High gamma wave activity is an indication of fine-tuned and efficient neural networks, whereby populations of neurons between brains regions fire synchronously. Over time, mindfulness meditation presumably enhances and sharpens communication between distributed neural assemblies. Through continuous practice of this technique, self-directed focus promotes neuroplastic changes and can structurally alter the way the brain is wired. During this process, neural circuits that are used regularly are strengthened. As seen in the monks, decades of mindfulness meditation practice induced neuroplastic changes that enhanced cognitive control and strengthened communication among various brain regions, including connectivity between frontal and limbic brain structures.

Other Treatment Approaches

Studies have been conducted on the efficacy of a number of other psychotherapies in the treatment of PTSD, such as psychodynamic therapy (Brom, Kleber, & Defares, 1989; Van Etten & Taylor, 1998), hypnotherapy (Abramowitz, Barak, Ben-Avi, & Knobler, 2008; Brom et al., 1989; Van Etten & Taylor, 1998), family therapy (Glynn et al., 1999), couples therapy (Monson, Fredman, & Adair, 2008; Monson et al., 2011; Monson, Guthrie, & Stevens, 2003), and group therapy (Beck & Coffey, 2005; Beck, Coffey, Foy, Keane, & Blanchard, 2009; Dorrepaal et al., 2010; Giannopoulou, Dikaiakou, & Yule, 2006; Schnurr et al., 2003). Meta-analyses so far have indicated that although these treatments appear to improve symptoms compared with no treatment, they are not as substantially supported as TF-CBT, EMDR, and PE (Bisson et al., 2007; Institute of Medicine, 2008; Ponniah & Hollon, 2009).

The Role of CBT in Ameliorating Neurological Changes in PTSD

A traumatic event can induce abnormal structural plastic changes within the amygdala and elsewhere, but CBT can still help to functionally rewire the brain but with some notable differences. Once PTSD symptoms become apparent, neurons in the amygdala have formed strong interconnections in such a way that a neural network facilitating abnormally strong "flashbulb" memories are easily evoked by trigger stimuli (Sapolsky, 2003). These connections, once formed, are considered permanent and can last a lifetime. In such cases, the efficiency of CBT-based techniques, such as imaginal exposure therapy, does not rely on erasing these neuroanatomical configurations; rather, counseling seems to promote the formation of new inhibitory connections that counterbalance and diminish the excitatory activity of maladaptive PTSD circuitry (Maguschak & Ressler, 2008; Quirk & Mueller, 2007). In these circumstances, CBT is similar to placing a

kink in a hose to stop the flow of water. Inducing structural changes in the brain takes time, but it is possible through the use of CBT, which promotes a diminished expression of learned fear (Ressler & Mayberg, 2007). Moreover, exercising the PFC through CBT is important for successfully treating other disorders of affect and anxiety.

Support for the neurobiological effects of CBT stems from multiple lines of evidence (Lutz et al., 2004; Paquette et al., 2003); however, successful treatment of PTSD via CBT requires that counselors know when and how much to stimulate their clients during any treatment plan. As with any constructive challenge, stimulation is essential for developing resilience for future life stressors and promoting recovery from various psychiatric illnesses (Sapolsky, 2003). Although the methods used by counselors to stimulate their clients are numerous, Foa's (Foa & Kozak, 1986) emotional processing theory illustrates how the use of thoughts can stimulate a patient during imaginal exposure therapy, as well as the caveats of underengagement or overengagement. Exposure therapy treatment requires clients to actively and systematically confront stress-related memories and avoid trauma-related situations through thoughts and in vivo exposure (Foa & Kozak, 1986). In doing so, clients disconfirm pathological elements underlying PTSD symptomatology. This method, however, can be ineffective if optimal levels of stimulation are not achieved. Overengagement can retraumatize a client by surpassing his or her ability to effectively cope with distressing memories, whereas underengagement is also undesirable because a patient does not sufficiently confront anxiety-provoking memories (Rauch & Foa, 2006).

Counselors must recognize that these reactions, whether real or imagined, have corresponding physiological effects on the mind–body that can be approached from a neurological perspective. On one hand, overengagement may induce an overactivation of the body's stress response system and overwhelm top-down regulatory control of limbic brain regions, such as the amygdala. On the other hand, the client who is underengaged does not sufficiently activate particular cortical-limbic brain regions, and thus makes modifying the erroneous conditions underlying the disorder unlikely. As a way to see a reduction in PTSD symptomatology, controlled and repeated activation of both the emotional and cognitive centers of the brain is necessary. With optimal amounts of stimulation, repeated exposure to stressful memories in a safe environment appears to create a "neurological environment" wherein prefrontal-induced neuroadaptations (e.g., restructured thought patterns) involving neuroplasticity, and other molecular mechanisms, positively affect PTSD symptoms (Katz et al., 2009; McClung & Nestler, 2007; Shin et al., 2005).

For the majority of people who exhibit normal psychological functioning, the brain is in a delicate balance between the cognitive (the PFC) and the emotional (the limbic system) components. A disconnect between these regions can induce psychological disorders, such as PTSD or major depressive disorder. Other cognitive-based methods, however, can be used to promote strong neurological communication between brain regions and have therapeutic benefits (Lutz et al., 2004). Mindfulness meditation is one example that has received significant attention in treating depression, generalized anxiety, and PTSD (Chiesa & Serretti, 2011; Evans et al., 2008; Segal et al., 2002; Wells & Sembel, 2006).

Conclusion

Innovative research is beginning to shed light on the efficacy behind interventions used by counselors on brain functioning (Linden, 2006; Paquette et al., 2003), but identifying the underlying factors involved in the development and successful treatment of common psychiatric disorders is an ongoing endeavor for researchers. Although many of the methods used by counselors are supported by empirical evidence, additional scrutiny of the efficacy of different pharmacological and psychotherapy techniques, as well as the combination of the two, is warranted. These issues are addressed elsewhere and are the subject of ongoing debate as novel finds are emerging at a rapid pace (Cukor, Olden, Lee, & Difede, 2010; Kennedy et al., 2007).

What is clear, however, is that thoughts, emotions, and experiences can induce physiological changes that alter the way the mind works. Under ordinary circumstances, each component of one's psychobiological profile influences—and in turn is influenced by—the other to bring about a normal balance. Typically, positive psychological and learning experiences influence this balance in a beneficial manner that promotes specialization and adaption to both static and changing physical and social climates. When these modifications, however, manifest into a skewed version of reality that disrupts everyday functioning, active intervention is often necessary. In turn, one can approach the issue of abnormal functioning from many different angles. For example, psychiatric medications treat mental disorders from a bottom-up perspective and are typically very effective. The value of a top-down cognitive approach, however, lies in the ability to direct executive brain functions, which influence physiological and psychological states, in a manner that is safer and more personalized than what psychiatric drugs can offer. Whichever approach, or combination of approaches, is taken, logical deductions from findings in the lab can provide practicing counselors with invaluable knowledge. Better understanding leads to better treatment, and counselors have the unique potential to apply this knowledge in real time, giving clients the immediate care they require.

References

Abramowitz, E. G., Barak, Y., Ben-Avi, I., & Knobler, H. Y. (2008). Hypnotherapy in the treatment of chronic combat-related PTSD patients suffering from insomnia: A randomized, zolpidem-controlled clinical trial. *International Journal of Clinical and Experimental Hypnosis, 56,* 270–280. doi:10.1080/00207140802039672

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Amstadter, A. B., McCart, M. R., & Ruggiero, K. J. (2007). Psychosocial interventions for adults with crime-related PTSD. *Professional Psychology: Research and Practice, 38,* 640–651. doi:10.1037/0735-7028.38.6.640

Banks, S. J., Eddy, K. T., Angstadt, M., Nathan, P. J., & Phan, K. L. (2007). Amygdala-frontal connectivity during emotion regulation. *Social Cognitive and Affective Neuroscience, 2,* 303–312.

Beauregard, M. (2007). Mind does really matter: Evidence from neuroimaging studies of emotional self-regulation, psychotherapy, and placebo effect. *Progress in Neurobiology, 81,* 218–236.

Beck, J. G., & Coffey, S. F. (2005). Group cognitive behavioral treatment for PTSD: Treatment of motor vehicle accident survivors. *Cognitive and Behavioral Practice, 12,* 267–277.

Beck, J. G., Coffey, S. F., Foy, D. W., Keane, T. M., & Blanchard, E. B. (2009). Group cognitive behavior therapy for posttraumatic stress disorder: An initial randomized pilot study. *Behavior Therapy, 40,* 82–92.

Benjamin, C. L., O'Neil, K. A., Crawley, S. A., Beidas, R. S., Coles, M., & Kendall, P. C. (2010). Patterns and predictors of subjective units of distress in anxious youth. *Behavioral and Cognitive Psychotherapy, 38,* 497–504.

Bishop, S. J. (2007). Neurocognitive mechanisms of anxiety: An integrative account. *Trends in Cognitive Sciences, 11,* 307–316.

Bisson, J. I., Ehlers, A., Matthews, R., Pilling, S., Richards, D., & Turner, S. (2007). Psychological treatments for chronic posttraumatic stress disorder: Systematic review and meta-analysis. *British Journal of Psychiatry, 190,* 97–104.

Brom, D., Kleber, R. J., & Defares, P. B. (1989). Brief psychotherapy for posttraumatic stress disorders. *Journal of Consulting and Clinical Psychology, 57,* 607–612.

Bryant, R. A., Harvey, A. G., Dang, S. T., Sackville, T., & Basten, C. (1998). Treatment of acute stress disorder: A comparison of cognitive-behavioral therapy and supportive counseling. *Journal of Consulting and Clinical Psychology, 66,* 862–866.

Chiesa, A., & Serretti, A. (2011). Mindfulness based cognitive therapy for psychiatric disorders: A systematic review and meta-analysis. *Psychiatry Research, 187,* 441–453.

Child Sexual Abuse Task Force and Research and Practice Core, National Child Traumatic Stress Network. (2004). *How to implement trauma-focused cognitive behavioral therapy.* Retrieved from http://www.nctsn.org/nctsn_assets/pdfs/TF-CBT_Implementation_Manual.pdf

Council for Accreditation of Counseling and Related Educational Programs. (2009). *2009 standards*. Retrieved from http://www.cacrep.org/doc/2009%20Standards%20with%20cover.pdf

Cukor, J., Difede, J., Spitalnick, J., Rizzo, A., & Rothbaum, B. O. (2009). Emerging treatments for PTSD. *Clinical Psychology Review, 29,* 715–726.

Cukor, J., Olden, M., Lee, F., & Difede, J. (2010). Evidence-based treatments for PTSD, new directions, and special challenges. *Annals of the New York Academy of Sciences, 1208,* 82–89.

David, D., & Szentagotai, A. (2006). Cognitions in cognitive-behavioral psychotherapies: Toward an integrative model. *Clinical Psychology Review, 26,* 284–298.

Davidson, P. R., & Parker, K. C. H. (2001). Eye movement desensitization and reprocessing (EMDR): A meta-analysis. *Journal of Consulting and Clinical Psychology, 69,* 305–316. doi:10.1037/0022-006X.69.2.305

De Raedt, R. (2006). Does neuroscience hold promise for the further development of behavior therapy? The case of emotional change after exposure in anxiety and depression. *Scandinavian Journal of Psychology, 47,* 225–236.

Dorrepaal, E., Thomaes, K., Smit, J. H., van Balkom, A. J. L. M., van Dyck, R., Veltman, D. J., & Draijer, N. (2010). Stabilizing group treatment for complex posttraumatic stress disorder related to childhood abuse based on psycho-education and cognitive behavioral therapy: A pilot study. *Child Abuse & Neglect, 34,* 284–288.

Ehlers, A., & Clark, D. M. (2008). Post-traumatic stress disorder: The development of effective psychological treatments. *Nordic Journal of Psychiatry, 62,* 11–18.

Evans, S., Ferrando, S., Findler, M., Stowell, C., Smart, C., & Haglin, D. (2008). Mindfulness-based cognitive therapy for generalized anxiety disorder. *Journal of Anxiety Disorders, 22,* 716–721.

Felmingham, K., Kemp, A., Williams, L., Das, P., Hughes, G., & Peduto, A. (2007). Changes in anterior cingulate and amygdala after cognitive behavior therapy of posttraumatic stress disorder. *Psychological Science, 18,* 127–129.

Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin, 99,* 20–35.

Foa, E. B., Steketee, G., & Rothbaum, B. O. (1989). Behavioral/cognitive conceptualizations of post-traumatic stress disorder. *Behavior Therapy, 20,* 155–176.

Frewen, P. A., Dozois, D. J. A., & Lanius, R. A. (2008). Neuroimaging studies of psychological interventions for mood and anxiety disorders: Empirical and methodological review. *Clinical Psychology Review, 28,* 228–246.

Giannopoulou, I., Dikaiakou, A., & Yule, W. (2006). Cognitive-behavioural group intervention for PTSD symptoms in children following the Athens 1999 Earthquake: A pilot study. *Clinical Child Psychology and Psychiatry, 11,* 543–553. doi:10.1177/1359104506067876

Glynn, S. M., Eth, S., Randolph, E. T., Foy, D. W., Urbaitis, M., Boxer, L., & Crothers, J. (1999). A test of behavioral family therapy to augment exposure for combat-related posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology, 67,* 243–251.

Harvey, A. G., Bryant, R. A., & Tarrier, N. (2003). Cognitive behavior therapy for posttraumatic stress disorder. *Clinical Psychology Review, 23,* 501–522.

Hembree, E. A., Rauch, S. A. M., & Foa, E. B. (2003). Beyond the manual: The insider's guide to prolonged exposure therapy for PTSD. *Cognitive and Behavioral Practice, 10,* 22–30.

Hinkle, J. S. (1999). A voice from the trenches: A reaction to Ivey and Ivey (1998). *Journal of Counseling & Development, 77,* 474–483.

Högberg, G., Pagani, M., Sundin, O., Soares, J., Aberg-Wistedt, A., Tarnell, B., & Hallstrom, T. (2008). Treatment of post-traumatic stress disorder with eye movement desensitization and reprocessing: Outcome is stable in 35-month follow-up. *Psychiatry Research, 159,* 101–108.

Institute of Medicine. (2008). Treatment of posttraumatic stress disorder: An assessment of the evidence. Washington, DC: The National Academies Press.

Ivey, A. E., & Ivey, M. B. (1998). Reframing *DSM-IV:* Positive strategies from developmental counseling and therapy. *Journal of Counseling & Development, 76,* 334–350.

Ivey, A. E., & Ivey, M. B. (1999). Toward a developmental diagnostic and statistical manual: The vitality of a contextual framework. *Journal of Counseling & Development, 77,* 484–490.

Kabat-Zinn, J. (1990). *Full catastrophe living: Using the wisdom of your body and mind to face stress, pain, and illness*. New York, NY: Dell.

Karlin, B. E., Ruzek, J. I., Chard, K. M., Eftekhari, A., Monson, C. M., Hembree, E. A., & Foa, E. B. (2010). Dissemination of evidence-based psychological treatments for posttraumatic stress disorder in the Veterans Health Administration. *Journal of Traumatic Stress, 23,* 663–673.

Katz, M., Liu, C., Schaer, M., Parker, K. J., Ottet, M. C., & Epps, A. (2009). Prefrontal plasticity and stress inoculation-induced resilience. *Developmental Neuroscience, 31,* 293–299.

Kennedy, S. H., Konarski, J. Z., Segal, Z. V., Lau, M. A., Bieling, P. J., & McIntyre, R. S. (2007). Differences in brain glucose metabolism between responders to CBT and venlafaxine in a 16-week randomized controlled trial. *American Journal of Psychiatry, 164,* 778–788.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). *Archives of General Psychiatry, 62,* 593–602.

Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. (1995). *Archives of General Psychiatry, 52,* 1048–1060.

Linden, D. E. J. (2006). How psychotherapy changes the brain—The contribution of functional neuroimaging. *Molecular Psychiatry, 11,* 528–538.

Lutz, A., Greischar, L. L., Rawlings, N. B., Ricard, M., & Davidson, R. J. (2004). Long-term meditators self-induce high-amplitude gamma synchrony during mental practice. *Proceedings of the National Academy of Sciences of the United States of America, 101,* 16369–16373.

Maguschak, K. A., & Ressler, K. J. (2008). β-catenin is required for memory consolidation. *Nature Neuroscience, 11,* 1319–1326. doi:10.1038/nn.2198

Martin, K. C., & Kandel, E. R. (1996). Cell adhesion molecules, CREB, and the formation of new synaptic connections. *Neuron, 17,* 567–570.

McClung, C. A., & Nestler, E. J. (2007). Neuroplasticity mediated by altered gene expression. *Neuropsychopharmacology, 33,* 3–17.

Monson, C. M., Fredman, S. J., & Adair, K. C. (2008). Cognitive-behavioral conjoint therapy for posttraumatic stress disorder: Application to Operation Enduring and Iraqi Freedom veterans. *Journal of Clinical Psychology, 64,* 958–971.

Monson, C. M., Fredman, S. J., Adair, K. C., Stevens, S. P., Resick, P. A., Schnurr, P. P., & Macdonald, A. (2011). Cognitive-behavioral conjoint therapy for PTSD: Pilot results from a community sample. *Journal of Traumatic Stress, 24,* 97–101.

Monson, C. M., Guthrie, K. A., & Stevens, S. (2003). Cognitive-behavioral couple's treatment for posttraumatic stress disorder. *The Behavior Therapist, 26,* 393–402.

Mowrer, O. H. (1960). *Learning theory and behavior*. New York, NY: Wiley.

National Institute of Mental Health. (2010). *The National Institute of Mental Health strategic plan.* Retrieved from http://www.nimh.nih.gov/about/strategic-planning-reports/index.shtml#introduction

Paquette, V., Lévesque, J., Mensour, B., Leroux, J.-M., Beaudoin, G., & Bourgouin, P. (2003). "Change the mind and you change the brain": Effects of cognitive-behavioral therapy on the neural correlates of spider phobia. *NeuroImage, 18,* 401–409.

Ponniah, K., & Hollon, S. D. (2009). Empirically supported psychological treatments for adult acute stress disorder and posttraumatic stress disorder: A review. *Depression and Anxiety, 26,* 1086–1109.

Powers, M. B., Halpern, J. M., Ferenschak, M. P., Gillihan, S. J., & Foa, E. B. (2010). A meta-analytic review of prolonged exposure for posttraumatic stress disorder. *Clinical Psychology Review, 30,* 635–641.

Quirk, G. J., & Mueller, D. (2007). Neural mechanisms of extinction learning and retrieval. *Neuropsychopharmacology, 33,* 56–72.

Rauch, S., & Foa, E. B. (2006). Emotional processing theory (EPT) and exposure therapy for PTSD. *Journal of Contemporary Psychotherapy, 36,* 61–65.

Ressler, K. J., & Mayberg, H. S. (2007). Targeting abnormal neural circuits in mood and anxiety disorders: From the laboratory to the clinic. *Nature Neuroscience, 10,* 1116–1124. doi:10.1038/nn1944

Rodenburg, R., Benjamin, A., de Roos, C., Meijer, A. M., & Stams, G. J. (2009). Efficacy of EMDR in children: A meta-analysis. *Clinical Psychology Review, 29,* 599–606. doi:10.1016/j.cpr.2009.06.008

Sapolsky, R. M. (2003). Stress and plasticity in the limbic system. *Neurochemical Research, 28,* 1735–1742. doi:10.1023/A:1026021307833

Schnurr, P. P., Friedman, M. J., Foy, D. W., Shea, M., Hsieh, F. Y., Lavori, P. W., & Bernardy, N. C. (2003). Randomized trial of trauma-focused group therapy for posttraumatic stress disorder: Results from a Department of Veterans Affairs cooperative study. *Archives of General Psychiatry, 60,* 481–489.

Segal, Z. V., Williams, J. M. G., & Teasdale, J. D. (2002). *Mindfulness-based cognitive therapy for depression: A new approach to preventing relapse*. New York, NY: Guilford Press.

Seidler, G. H., & Wagner, F. E. (2006). Comparing the efficacy of EMDR and trauma-focused cognitive-behavioral therapy in the treatment of PTSD: A meta-analytic study. *Psychological Medicine, 36,* 1515–1522.

Shapiro, F., & Maxfield, L. (2002). Eye movement desensitization and reprocessing (EMDR): Information processing in the treatment of trauma. *Journal of Clinical Psychology, 58,* 933–946. doi:10.1002/jclp.10068

Shin, L. M., Wright, C. I., Cannistraro, P. A., Wedig, M. M., McMullin, K., & Martis, B. (2005). A functional magnetic resonance imaging study of amygdala and medial prefrontal cortex responses to overtly presented fearful faces in posttraumatic stress disorder. *Archives of General Psychiatry, 62,* 273–281.

Siegel, D. J. (2007). *The mindful brain: Reflection and attunement in the cultivation of well-being*. New York, NY: Norton.

Vanderwolf, C. H. (2000). Are neocortical gamma waves related to consciousness? *Brain Research, 855,* 217–224.

Van Etten, M. L., & Taylor, S. (1998). Comparative efficacy of treatments for post-traumatic stress disorder: A meta-analysis. *Clinical Psychology & Psychotherapy, 5,* 126–144.

Watson, P. J., & Shalev, A. Y. (2005). Assessment and treatment of adult acute responses to traumatic stress following mass traumatic events. *CNS Spectrums, 10,* 123–131.

Wells, A., & Sembel, S. (2006). Metacognitive therapy for PTSD: A core treatment manual. *Cognitive and Behavioral Practice, 11,* 365–377.