

# **Neurology and Trauma: Impact and Implications**

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As reported in Afifi, Asmundson, Taylor and Jang (2010), lifetime prevalence rates range from 64% to 90% for experiences of trauma and 1.4% to 11.2 % for posttraumatic stress disorder (PTSD). Although the majority of the population will be affected by trauma at some point in their life, the impact of these experiences will vary depending on the nature of the trauma, person and family characteristics, and available formal and informal resources accessed. Among the more severe effects is the development of PTSD.

PTSD involves a series of symptoms that develop from intensely fearful, horrific, and uncontrolled trauma where the person felt their life was in danger, they were injured or felt they would be injured, or they witnessed the death or injury of others. As outlined by the DSM-IV-TR (the Diagnostic and Statistical Manual from the American Psychological Association, 2000), the symptoms include: a) re-experiencing the traumatic event through intrusive memories, flashbacks, nightmares, and physiological responses similar to when the traumatic event was occurring (racing heart, dizziness, sweating, erratic breathing); b) avoidance and numbing such as avoiding situations and people that remind them of the trauma, amnesia for trauma related information, loss of interest in activities, social and emotional detachment, emotional numbing especially for feelings associated with intimacy, and feelings of a limited future; and c) increased arousal manifested by angry outbursts, problems sleeping, problems concentrating or completing tasks, exaggerated startle response, and hypervigilance. Many of these symptoms are typical following a trauma, but with PTSD they do not decrease or end. Symptoms tend to appear in the first three months of the trauma, but there may be delays of months or years before their onset.

Trauma that was severe in nature, of longer duration and was directly experienced by the individual is more likely to lead to PTSD. In terms of individual risk factors, personal and family mental health issues and previous trauma such as child abuse have been found to be the most consistent predictors of developing PTSD after a trauma. Further, PTSD is associated with the development of psychological disorders, suicidal behaviour, physical health problems and relationship and family problems.

Other trauma related diagnoses include adjustment disorder where the trauma is of any degree of severity and symptoms are not sufficient to warrant a diagnosis of PTSD. Events like a divorce or losing one's home due to bankruptcy can result in adjustment disorder. Acute stress disorder is experienced when PTSD symptoms occur at least four weeks after a traumatic event but end within a one-month period.

Interest in the impact of trauma and PTSD dates back to the "shell shock" of WWI and "combat fatigue" of WWII. In the 1980s studies on the neurological basis for these impacts began to proliferate, with a considerable amount of attention being given to the impact of childhood sexual abuse on cortisol levels and hippocampal size. Since this time the research has expanded

to examine the effects of trauma on other brain structures and processes. Knowing these effects can not only lead to greater understanding of traumatized individuals but to a more informed and compassionate approach to intervention. Because trauma can result from a variety of experiences, it has been suggested that a larger number of people than those identified as having been traumatized may be suffering from trauma effects (Afifi et al., 2010). It is therefore beneficial to all service providers to be aware of the effects of trauma in order to maximize the effectiveness of their intervention strategies.

This summary provides information about the current state of knowledge about the impact of trauma on neurological functioning and the subsequent behavioural effects. It is important to note that the field is always growing and therefore new information is always coming to light. There are still contradictory findings and unknown effects and interactions, however given the complexity of the area, a significant amount of knowledge has been gained. In order to make this summary and the corresponding graphics and annotated bibliography more manageable, the information focuses on post-traumatic stress disorder (PTSD). However, neurological responses are often similar for all traumas, with the difference being in the degree of neurological system dysregulation that occurs.

## **Neurological Impact of Trauma**

When a stressor occurs, a part of the brain called the amygdala makes a quick assessment of whether the situation requires a systemic response. If a response is judged to be necessary, it stimulates systems that prepare the body to effectively deal with the stressor and to take action. Responses to stressors can then be modified by the cortex, the part of the brain that produces more planned and well thought out behaviour and inhibits systemic over-reaction.

### **1. HPA-axis and Cortisol Levels**

In response to stressors and trauma, the amygdala stimulates the sympathetic nervous system which responds by preparing the body with a flight or fight response that includes an increased heart rate, as well as changes in muscle tone and breathing. The amygdala then stimulates the HPA-axis. The HPA-axis is a circuit of brain cells associated with the hypothalamus, pituitary gland, and the adrenal glands (HPA). The hypothalamus, which controls hormone functioning, secretes corticotropin-releasing hormone (CRH). This hormone promotes the release of two other hormones from the pituitary gland: beta-endorphins, which act to suppress pain during stressful situations and adrenocorticotrophic hormone (ACTH), which triggers the release of cortisol from the adrenal gland (Yehuda, 2002). Cortisol plays a role in the central nervous system by affecting learning, memory, and emotions. It is important to metabolism in that it regulates the storage and use of glucose. Cortisol also impacts the immune system by determining the length and strength of inflammatory response to injury and in part by contributing to the development of immune system cells (see Miller, Chen, & Zhou, 2007).

The system also releases hormones called catecholamines, and in general the more significant the stress the more catecholamines and cortisol are released. Catecholamines make more energy

available to the body in preparation for action. Cortisol makes the thalamus more sensitive to stimuli (van der Kolk, 2003). The thalamus is a brain structure that receives information from the senses and determines to what other brain areas this sensory information will be sent for further processing. By making the thalamus more responsive to stimuli, cortisol helps individuals focus on dangerous or threatening events in order to better cope and respond to these stressors. It helps individuals remember information relevant to the stressor and helps to stabilize stressful events in long-term memory, thus stressful situations are often remembered over other events. These responses may have survival value if the person is faced with the same or similar stressor in the future. Cortisol also works to modify or reduce the sympathetic system activity (systemic arousal) that prepares the body for action. It does so through a negative feedback loop, where increased production of cortisol in response to stress and trauma, overtime leads to inhibition of the HPA-axis response and thus to the suppression of cortisol production. Therefore, when the stressor is gone, the system goes back to a balanced state (Yehuda, 2002).

The research indicates that trauma and chronic stress can lead to dysregulation of the HPA-axis. For example, beta-endorphins help individuals better cope with pain while they are focusing their energy on dealing with a stressor. In cases of trauma and PTSD, beta-endorphins may keep being released as a result of re-experiencing the stressor through flashbacks and intrusive memories (van der Kolk, 2003). This continued release of beta-endorphins may then lead to symptoms such as avoidance of situations or thoughts that are reminiscent of the trauma, emotional numbing, loss of interest in life, and detachment from others. Re-traumatization would also contribute to the continued release of these hormones and further avoiding and numbing.

Dysregulation of the HPA-axis can lead to problems with the release of cortisol (Pfeffer, Altemus, Heo, & Jiang, 2009). The literature has revealed mixed results in terms of whether trauma is associated with increased or decreased levels of cortisol in the system. Although there was initial focus on identifying whether trauma resulted in increased or decreased levels of cortisol, it is becoming apparent that the pattern of cortisol production is more complex than simple an increase or decrease and is rather dependent on characteristics of the trauma experienced. In non-stressed individuals cortisol tends to be produced in higher amounts during the morning and then gradually decreases throughout the day. This pattern seems to be differentially disrupted with different types of trauma and stress. Miller, Chen and Zhou (2007) provide a comprehensive summary of the existing literature on cortisol production in relation to trauma related variables. Among the variables examined are: the timing since the onset of the stressor, characteristics related to the stress, and characteristics related to the individuals experiencing the stress.

#### **a) Timing Since the Onset of the Stressor**

The most consistent and prevalent research is on the time elapsed since the stressor. The research indicated that immediately after a stressor, activation of the HPA-axis occurs and thus there is a consequent increase in ACTH and cortisol levels (Delahanty, Nugent, Christopher, & Walsh, 2005). This initial increase in cortisol helps the person to focus on the stressor. However, the negative feedback loop that manages the activity of the HPA-axis means that high levels of cortisol in the system eventually lower the production of CRH and ACTH and because ACTH

promotes the release of cortisol, over time there is a consequent decrease in the production of cortisol (Miller et al., 2007).

### **b) Characteristics of the Stress**

Stress that presents a threat to physical wellbeing and that results from trauma is related to increased levels of cortisol. For stress that threatens a person's social self, like divorce or loss of employment, cortisol levels are elevated during the day, but are not higher over all. This may mean that while they are awake and dwelling on their situation, cortisol levels remain high, but are fairly low at night when they are sleeping (Miller et al., 2007). In stressful situations that are out of a person's control, cortisol levels are low in the morning (when they should be at their highest level) but high for the remaining of the day (when they should be decreasing), and thus are overall higher than in non-stressed individuals.

### **c) Characteristics of the Individuals Experiencing the Stress**

Feelings of shame in response to the stress have been associated with higher than normal levels of cortisol in the afternoon and evening. Feelings of loss also demonstrated a reversal of the typical pattern, with lower levels of cortisol being produced in the morning, but higher than normal levels being produced in the rest of the day. Because these results are based on relatively few studies and have not always been consistent and because other confounding variables such as the intensity of the emotion have not been investigated, they remain tentative and not interpretable. What is apparent, however, is that emotions associated with stress are linked to dysregulation of the HPA-axis.

Clearer results have been reported for individuals who develop depression or PTSD in response to trauma. People who have become depressed in response to trauma, show elevated levels of cortisol. On the other hand, individuals that develop PTSD show lower than typical levels of cortisol, with greater PTSD symptoms being associated with lower levels of this hormone (Delahanty, Raimonde, & Spoonster, 2000; Marshall, Blanco, Prinz, Liebowitz, Klein, & Coplan, 2002; Miller, et al., 2007; Olf, Guzelcan, de Vries, Assies, & Gersons, 2006; Shalev, Videlok, Peleg, Segman, Pitman, & Yehuda, 2007; Witteveen, Huizink, Slottje, Bramsen, Smid, & van der Ploeg, 2010; Yehuda, Morris, Lebinsky, Zelman, & Schmeidler, 2007). The literature also suggests that although cortisol levels are generally lower in those individuals suffering life threatening trauma and PTSD, these individuals show higher than normal levels in response to current stressors and challenges (de Kloet, Vermetten, Geuze, Kavelaars, Heijnen, & Westenberg, 2006; Handwerger, 2009; Marshall, et al., 2002). To explain this apparent paradox, it has been suggested that in individuals with PTSD, the HPA-axis negative feedback loop may come into effect because of the time lapse between the initial trauma and the development of PTSD.

The problem with research on psychological conditions such as depression and PTSD is the lack of ability to determine whether the pattern of cortisol production is the result of a person's psychological response to trauma, or if the person has a biological tendency to that makes them vulnerable to develop particular HPA-axis activity and cortisol production patterns that then result in certain psychological conditions. For example, does experiencing trauma and developing PTSD ultimately lead to lower cortisol levels or is the person born with a system that already produces less cortisol or an increase in the intensity of the negative feedback loop, and

this makes them more likely to develop PTSD in response to trauma? As Miller and colleagues (2007) state, more longitudinal research tracking HPA-axis activity and hormonal levels before and after trauma in individuals is required to clarify the direction of effect.

Whether cortisol patterns and levels are higher or lower than typical, these altered levels have been associated with certain behavioural effects. Low levels of cortisol may negatively affect an individuals' ability to process the event into long-term memory. Without this ability to process it as part of the past, the memory remains as part of the present, exerting its emotional impact through flashbacks, nightmares and fear. Some studies have associated low levels of cortisol with intrusive memories of the trauma (Delahanty et al., 2000).

High levels of cortisol may result in the nervous system becoming sensitized to psychologically threatening stimuli, a process known as kindling. Kindling leads the nervous system to react to even weak stimuli. Thus people who have experienced trauma may react to very weak stimuli that would not cause a reaction in non-traumatized people. Non-threatening events may be perceived as threatening because they bear some similarity to the traumatic event through certain sights, sounds, smells etc. Intrusive memories and negative thoughts can serve to fuel this association, as details and sensory association do not fade over time (Abercrombie, Kalin, Thurow, Rosenkrantz, & Davidson, 2003).

## **2. The Hippocampus**

The hippocampus is a brain structure located medial to the temporal lobe. It is part of the limbic system, which is a group of brain structures including the amygdala, hippocampus, and hypothalamus. The limbic system is involved in processing and regulating emotions, memories, and sexual arousal. In addition, the limbic system is activated during the body's stress response. When a person experiences a traumatic event, the thalamus sends information to the hippocampus. The hippocampus is involved in the formation of explicit knowledge based memories, working or short term memory, and memory for episodic events. The hippocampus is also involved in the regulation of stress and is strongly connected to the amygdala. Animal research has shown that chronic stress affects the hippocampus through the excessive release of glucocorticoids (hormones involved in glucose metabolism and immune system response), corticotropin-releasing hormone (see above), and glutamate (a major excitatory neurotransmitter that promotes internal or external behavioural responses); the inhibition of neurogenesis (the creation of new neurons); impaired long-term potentiation induction (a process vital to establishing new memories); inhibition of brain-derived neurotrophic factor (BDNF) which is a protein that encourages the growth of new neurons and synapses; and altering the function of serotonin receptors (Karl, Schaefer, Malta, Dorfel, Rohleder, & Werner, 2006). Serotonin is a neurotransmitter that promotes sleep, temperature regulation and mood. Neurotransmitters are the chemicals brain cells (neurons) use to communicate with each other; they relay the electrical messages between neurons. Because the hippocampus is involved in memory, learning, and stress regulation, all processes affected in PTSD, it has been the focus of much research on PTSD.

Bremner and colleagues (1995) found that patients with chronic PTSD perform significantly worse on hippocampal-based memory and learning tasks. For instance individuals with PTSD completing declarative memory (knowledge and facts) tasks demonstrated a failure or reduced activation in the hippocampus (Bremner, Vyhtilingam, Vermetten, Southwick, McGlashan, Nazeer, et al., 2003). Using positron emission tomography (PET) Shin and colleagues (2004) also found reduced hippocampal activation in people with PTSD during a word-stem completion task (testing recall of words by giving the first few letters as prompts).

Bremner et al., (1995) conducted the first neuroimaging study to find that the hippocampus was significantly smaller in patients with combat related PTSD. The majority of studies since then examining PTSD using magnetic resonance imaging (MRI) have shown that people with PTSD have a smaller hippocampus than matched controls (Freeman, Kimbrell, Booe, Myers, Cardwell, Lindquist, Hart, & Komoroski, 2006). In fact, this has been found in studies examining a number of different forms of trauma such as combat exposure (Gurvits, Shenton, Hokama, Ohta, Lasko, Gilbertson, et al., 1996), sexual and physical abuse (Bremner, Randal, Vermetten, Staib, Bronen, Mazure, et al., 1997; Stein, Koverola, Hanna, Torchia, & McClarty, 1997), childhood sexual abuse (Stein et al., 1997), cancer (Nakano, Wenner, Inagaki, Kugaya, Akechi, Matsuoka, et al., 2002), mixed trauma types (Villareal, Petropoulos, Hamilton, Rowland, Horan, Griego, Moreshead, Hart, & Brooks, 2002), and personal violence or accidents (Lindauer, Vlieger, Jalink, Olf, Carlier, Majoie, den Heeten, & Gersons, 2004). Studies using a technique known as proton magnetic resonance spectroscopy (H-MRS) have typically found abnormalities in hippocampal biochemistry in PTSD subjects, including lower levels of hippocampal *N*-acetylaspartate (NAA), an excitatory neurotransmitter associated with the health and stability of neurons, in individuals with PTSD relative to controls (Villareal et al., 2002).

The decreased size of the hippocampus is due to the degeneration of brain cells in that area. This deterioration can lead to individuals not being able to remember general and/or detailed facts about the trauma, having incomplete memories or no memory for the trauma, and dissociative episodes in response to trauma related stimuli (Vermetten & Bremner, 2002). These symptoms are all characteristics of PTSD.

The cause and timing of the hippocampal volume deficits and its relationship to PTSD remains unknown but several explanations have been forwarded. For instance, one hypothesis is that individuals with smaller hippocampal volumes or altered hippocampal biochemistry may be more vulnerable to developing PTSD if exposed to a traumatic event. A second hypothesis is that these hippocampal alterations follow the traumatic event as a consequence of neurobiological changes associated with chronic PTSD, such as dysregulation of cortisol levels. A causal relationship between trauma and hippocampal volume reduction is difficult to prove and most researchers agree that more longitudinal studies are needed to determine the timeline of these neurobiological changes. However, the findings suggest that trauma may lead to a degeneration of brain cells and a reduced ability to generate new brain cells and connections between brain cells, resulting to a lessened ability to form new memories.

It should be noted that a number of studies have failed to find differences in hippocampal size. For instance, studies examining children with PTSD (DeBellis, Hall, Boring, Frustaci, & Moritz, 2001; Carrion, Weems, Eliez, Patwardhan, Brown, Ray, et al., 2002; De Bellis, Keshavan,

Shifflett, Iyengar, Beers, Hall, et al., 2002), individuals with recent onset of PTSD (Bonne, 2001), women with a history of intimate partner violence (Pederson, Maurer, Kaminski, Zander, Peters, Stokes-Crowe, et al., 2004), burn patients (Winter & Ire, 2004), and combat veterans (Yehuda, et al., 2007) did not find hippocampal volume differences between PTSD subjects and both healthy and traumatized subjects without PTSD. Much debate exists within the literature regarding the meaning of these contradictory findings and discrepancies in results have been attributed to factors including: the selection and composition of the participants in the control groups, the age of the participants, participant's comorbidity with other psychiatric disorders such as depression and substance abuse, type of trauma exposure, the timing of the PTSD, and lastly, methodological concerns such as sample size.

### **3. Medial Prefrontal Cortex and Amygdala Functioning**

During a stressful event, the thalamus sends information to the amygdala, regarding the need to prepare for a threat. Specific details regarding the emotional content of the experience also come to the amygdala from the hippocampus. An emotional reaction to the experience is then generated by the amygdala that in turn sends information to the brainstem that then elicits appropriate reflexes and behavioural responses. In addition, the amygdala sends messages to the hypothalamus, where hormonal and systemic responses are triggered through the activity of the HPA-axis.

The prefrontal cortex, which lies directly behind the forehead, is the brain region where decisions about appropriate or required cognitive and/or emotional responses are made. It works to suppress or modify emotional responses generated by the amygdala. Thus an automatic reflexive fear reaction is reduced or stopped by messages from the prefrontal cortex indicating that the situation is safe and that there is no danger or threat. The cortex also generates a calmer, more strategic response to the situation (Zubieta, Chinitz, Lombardi, Fig, Cameron, & Liberzon, 1999). Another task of the prefrontal cortex is to filter out unimportant or unnecessary information, thereby allowing individuals to focus on information that is most relevant. Impairment of this filter process means that the person may be less able to discriminate between irrelevant and important stimuli.

The prefrontal cortex also interacts with the HPA-axis (Harvard Medical School, 2005). Reduced activity in the prefrontal cortex is associated with increased stress related HPA-axis activity and anxiety related behaviours, including emotional numbing and the subsequent inability to process positive emotions (Jatzko, Schmitt, Demirakca, Weimer, & Braus, 2006). On the other hand, increased prefrontal cortex activity is associated with less stress related HPA-axis activity and better management of emotions (Jaferi & Bhatnagar, 2007; King, Abelson, Britton, Phan, Taylor, & Liberzon, 2009; Weinberg, Johnson, Bhatt, & Spencer, 2010).

After a traumatic experience, many individuals have an overly responsive amygdala that generates fear responses across a wide variety of stimuli. Because the amygdala is especially responsive to fear and threat, traumatized individuals will likely be more focused on negative stimuli and events (Armony, Corbo, Clement, & Brunet, 2005; Brunetti, Sepede, Mingoia, Catani, Ferretti, Merla, Del Gratta, et al., 2010). Further, the prefrontal cortex may be more

focused on trying to modify the amygdala response to fear and threat and thus attending to more negative stimuli, leading in additional reduction in the person's ability to focus on positive stimuli (New, Fan, Murrough, Liu, Liebman, Guise, Tang et al., 2009). It has further been suggested that the emotional over-response in the amygdala may be due to an impairment of the prefrontal cortex's ability to inhibit or modify the amygdala's response to outside stimuli (Liberzon, Britton, & Phan, 2003; Shin, Orr, Carson, Rauch, Macklin, Lasko, Peters et al., 2004; Shin, Wright, Cannistraro, Wedig, McMillin, Martis, Macklin, et al., 2005; Weinberg et al., 2010). Without the more calming thoughts and judgments by the prefrontal cortex, automatic fear responses occur more frequently and to a broader range of events and situations. For example, although many individuals do not like being examined by a doctor, they are able to reduce their fear and anxiety (amygdala response) through self-talk (prefrontal cortex modification of anxiety response). If the part of the brain that generates self-talk and reasoning has been negatively affected by trauma, the person cannot calm themselves down and therefore they will feel fear and experience systemic responses associated with fear such as increased heart rate, muscle tension, and rapid breathing. Research has found that increases in amygdala reaction are linked to more severe PTSD symptoms while increases in prefrontal cortex response is associated with less severe PTSD symptoms (Shin, Rauch, & Pitman, 2006).

Thus, this over-reaction of the amygdala and impairment of the frontal cortex's ability to regulate emotions may be responsible for the symptoms of hyper-arousal in PTSD, including exaggerated startle responses, irritability, anger outbursts, hypervigilance, flashbacks, intrusive memories, and misinterpretation of harmless stimuli as potential threats. These create distracting thoughts and ideas that make it difficult for the person to concentrate, follow instructions, reason, and make sound judgments and decisions, symptoms commonly found in PTSD. It has been suggested that therapies that work to help individuals modify amygdala responses to memories of the trauma may be effective forms of intervention for PTSD. These therapies may include exposure therapy, intended to extinguish the fear response. Other therapeutic methods such as cognitive-behaviour therapy, mindfulness, and stress inoculation will help to strengthen the frontal cortex's moderating of the amygdala response and its capacity to filter out irrelevant information and focus on more positive emotions (see the section on treatment approaches).

### **Genetic Markers in the Development of PTSD**

The literature indicates that there is a moderate heritability component to PTSD and that it is most likely that several genes, rather than just one, contribute to a predisposition towards developing this condition (Nugent, Amstadter, & Koenen, 2008). Genetic predispositions interact with environmental factors to influence the development of PTSD and other psychological conditions. Family mental health is a risk factor that can have an impact in terms of genes as well as environment (see Afifi, et al., 2010). Children whose parents developed PTSD after a trauma were themselves more likely to develop this disorder following a trauma. Further, in studies comparing identical and fraternal twins raised together, identical twins had a greater likelihood of both developing PTSD than fraternal twins (see Afifi et al., 2010; Nugent, et al., 2008). Current studies have begun to explore which genes are involved in vulnerability to PTSD.



Genetic predispositions may come in the form of dysfunctions in the cortex (see above for the role the cortex plays in emotion regulation) or a smaller hippocampus (Pitman, Gilbertson, Gurvits, May, Lasko, Metzger, Shenton, et al., 2006). Another link has been found between individuals with an overly large cavum septum pellucidum (Pitman, et al., 2006), a cavity between the left and the right hemispheres of the brain that is present in all infants but closes by adulthood in the majority of people. Enlargements of this cavity have been associated with PTSD, schizophrenia, and chronic brain trauma. Predisposing genes may also be affecting some of the neurotransmitters, chemicals which allow communication among brain cells. Problems in both the serotonin system and to a lesser degree the dopamine system have been implicated in increased risk for PTSD. Serotonin is associated with regulation of mood, appetite, sleep patterns, temperature and the regeneration of neurons; lower levels have been linked to PTSD and other mental health issues such as depression. Dopamine is involved with arousal, voluntary movement and conditioned fear responses; higher levels have been associated with PTSD (Nugent, et al., 2008). It has also been suggested that maladaptive beliefs may have a genetic component, and therefore perceptions of threat and other beliefs characteristic of PTSD may, in part, be due to genetics.

The genetic evidence may have important implications for treatment. For example, it introduces the possibility of using drug therapy after a trauma to reduce the likelihood of developing PTSD. Certain drugs such as selective serotonin re-uptake inhibitors, that work to increase the amount of serotonin in the system, also help to regenerate neurons in the hippocampus (Harvard Medical School, 2005). In addition, elevated levels of corticotrophin releasing hormone (CRH) have been linked to problems with the HPA-axis (see summary above) and the sympathetic nervous system that prepares the body for some type of response (flight or fight) through increasing heart rate, perspiration, deep breathing, and blood pressure. Drugs that reduce CRH may curtail this dysfunction (Harvard Medical School, 2005).

## **Treatment Approaches for PTSD**

Yehuda (2002) states that the most frequent complaint made by trauma survivors is that they feel misunderstood by others. They are encouraged by service providers as well as others to leave their experience in the past and move on with their lives. What the research above has demonstrated is that the physiological response to trauma often means that they cannot put their experience in the past. It remains with them with as much intensity as when it first occurred, affecting every aspect of their lives and preventing them from moving on with life. This serves to make them feel even more alone and detached from others.

A variety of intervention methods may be helpful to individuals' recovery from trauma, however only a few have research evidence supporting their effectiveness. Cognitive-behaviour therapies (CBT), including exposure therapy are the most often applied form of trauma intervention. Generating a lot of research, they have demonstrated a great deal of effectiveness, particularly compared to supportive methods alone (Bisson, Ehlers, Matthews, Pilling, Richards, & Turner, 2007; Kornor, Winje, Ekeberg, Weisaeth, Kirkehei, Johansen, & Steiro, 2002; Mendes, Mello, Ventura, Passarela, & Mari, 2008). CBT focused on early trauma also proved more effective than supportive therapies in preventing initial trauma response from developing into PTSD

(Kornor et al., 2002). CBT addresses distortions in beliefs and emotions such as the environment or situation being threatening to personal safety and wellbeing and feelings of hopelessness and helplessness and replaces them with more accurate and rational perceptions. Memories of the trauma are relabelled and reorganized in ways that help the individual gain a sense of meaning and control (see Robertson, Humphries & Ray, 2004).

Exposure therapy has demonstrated effectiveness in recovery from trauma related distress (Jaeger, Echiverri, Zoeller, Post & Feeny, 2009). This therapeutic approach helps the individual gradually confront trauma related information through talking and journaling. Avoiding talking about and thinking about the traumatic event is often reinforcing because the person does not have to face the memories of the event, but it does nothing to resolve trauma related issues. Facing the fear provoking memories of the event that are often re-traumatizing, decreases symptoms such as avoidance and heightened arousal. When memories of the experience are confronted they lose their power to generate fear (see Robertson, et al., 2004). Combining exposure therapy and relaxation training is suggested. The relaxation exercises help individuals from being re-traumatized by the information and emotions that surface during exposure therapy.

A form of exposure therapy called imagery rescripting has individuals confront threatening images and change them to less threatening images or develop new positive images to replace the threatening ones (Holmes, Arntz, & Smucker, 2007). Rescripting can lead to more positive emotions and new perspectives. It has been associated with changes in anger, guilt, and shame (Arntz, Kindt, & Tiesema, 2007), and reducing PTSD symptoms in survivors of sexual assault (Krakow, Hollifield, Johnston, Koss, Schrader, et al., 2001) and childhood sexual abuse (Smucker, Dancu, Foa, & Niederee, 1995; Smucker & Niederee, 1995).

Narrative therapy combines exposure therapy with CBT to help individuals reconceptualise the event. It is based on the idea that a person's perceptions will influence their emotions and behaviours, and that the stories people tell about themselves and their lives reflect their these perceptions. Individuals are asked to tell their stories of the trauma to the therapist, thereby confronting their experiences. These stories may be narrow and focus on a dominant theme such as anger or helplessness. In therapy they are asked to tell their story from a different perspective, taking a different focus, entertaining different possible reactions, considering different contextual elements, or from a more desirable reality (Augusta-Scott & Dankwort, 2002). They are also encouraged to expand the detail and complexity of their story. Developing these different narratives can change their perceptions and help them realize their own power to effect more positive outcomes (White & Epston, 1990). There has been some evidence of the effectiveness of narrative therapy in survivors of mass violence, war, torture, rape, and childhood abuse (Anderson & Hiersteiner, 2008; McPherson, 2012; Mueller, 2009; Robjant & Fazel, 2010; Schauer, Neuner, & Elbert, 2011).

Despite evidence of its effectiveness alone or as part of other therapeutic approaches, there are studies (e.g. Protopopescu, Pan, Tuescher, Cloitre, Goldstein, Engeli, Epstein, et al., 2005) that indicate that exposure does not sensitize the person to traumatic content and that traumatic response diminishes gradually over time. If this is the case, talking, journaling, and other methods intended to help the person confront and thus extinguish the fear may not be helpful at all, and in fact, may be re-traumatizing. Despite these concerns, research has found that given a

choice of different types of therapy, individuals chose cognitive therapy followed by exposure therapy (Tarrier, Liversidge, & Gregg, 2006). In addition, there is considerable evidence that although distress may be experienced immediately after the disclosure, writing or talking about traumatic experiences has long term benefits for both physical and mental health (Cole, Kemeny, Taylor, & Visscher, 1996; Donnelly & Murray, 1991; Easterling, Antoni, Kumar, & Scheiderman, 1990; Easterling, L'Abate, Murray, & Pennebaker, 1999; Greenberg & Stone, 1992; Pennebaker, 1993; Pennebaker & Graybeal, 2001; Richman, 2006). Research has also found that individuals who have experienced trauma believe talking about their experiences is beneficial to the therapeutic process and their recovery (Angelo, Miller, Zoellner, & Feeny, 2008; Cochran, Pruitt, Fukuda, Zoellner, & Feeny, 2007).

Although more studies are needed to determine the nature of the effects of exposure therapy, it is generally believed that reducing or eliminating the fear arousal may be necessary before individuals are able to focus on learning and applying more cognitive strategies related to emotional regulation and logical thought, otherwise CBT may have little or no effect (Bryant, Felmingham, Kemp, Das, Hughes, Peduto, & Williams, 2007). This may be the reason that psychoeducational approaches by themselves have no evidence of effectiveness. It may also account for the lack of evidence for the effectiveness of critical incident stress debriefing, an intervention often done immediately after a trauma, in an attempt to prevent the development of PTSD or of reducing existing trauma symptoms. Research shows that traumatized individuals without PTSD had more prefrontal cortex activation after being presented with information on strategies for regulating their emotions than those with PTSD and that individuals responding positively to intervention had increased neuron activity in the cortex (Bryant, Felmingham, Whitford, Kemp, Hughes, Peduto, & Williams, 2008). It was believed that the lack of intense arousal, fear and avoidance, helped these individuals to face and manage their emotions and focus on learning and implementing healthier coping behaviours. Once symptoms of hyperarousal, avoidance, and distorted perceptions have been dealt with, the more social, relationship, employment and other life issues related to trauma response can be addressed (Herman, 1992; see Robertson et al., 2004). These studies indicate that CBT is potentially more effective for traumatized individuals who do not have PTSD or whose fear arousal has been reduced to the point that it does not interfere with attention to developing new skills and strategies.

Various meditative techniques have been found to reduce fear arousal and increase capacity to focus attention. Mindfulness based stress reduction (MBSR) has individuals focusing on sensations and thoughts as they occur without applying judgment or interpretation. The result is a greater awareness of their environments and of their own perceptions. Through this process individuals learn to attend to multiple sensations, thus helping to broaden their perspectives (Chatzisarantis & Haggard, 2007; Coffey & Hartman, 2008; Kabat-Zinn, 1990). Because attending to sensory experiences as they occur requires cognitive focus rather than reactive responses, systemic arousal is reduced. When systemic arousal is reduced and individuals slow down enough to attend to different sensations and cognitions, they develop new awareness of their environments, they become more open to new ideas, and they are increasingly able to apply new ways of solving problems (Follet, Palm, & Pearson, 2006).

MBSR has been associated with reduced stress arousal and more effective monitoring of internal states (Lutz, Stagter, Dunne, & Davidson, 2008). It has been used to facilitate emotion and

behaviour regulation (Gerbarg & Brown, 2011; Heim, Shugart, Craighead, & Nemeroff, 2010; Langer 2000; Wolfsdorf & Zlotnick, 2001) and has been effectively applied in interventions for depression (Finucan & Mercer, 2006; Germer, Siegel, & Fulton; Kimbrough, Magyari, Langenberg, Chesney, & Berman, 2010; Segal, Teasdale, & Williams, 2002; Owens, Walter, Chard, & Davis, 2011), anxiety (Finucan & Mercer, 2006; Kimbrough et al., 2010), PTSD (Batten, Orsillo, & Walser, 2005; Owens et al., 2011), chronic pain and stress (Kabat-Zinn, 1990). Lutz and colleagues (2008) suggest that it reduces traumatized individuals' tendency to ruminate on a particular thought or image.

Other forms of meditation that require the individual to focus on one stimulus while filtering out all other distracting stimuli have been helpful in increasing the ability to concentrate, to block out negative thoughts, and to reduce stress arousal. Further, with practice individuals become more proficient at developing this skill and thus less energy is required to achieve focused attention (Brefczynski-Lewis, Lutz, Schaefer, Levinson, & Davidson, 2007; Lutz, et al., 2008). These studies suggest that various forms of meditation have value in the treatment of trauma.

Stress inoculation therapy is a form of CBT that involves three phases: an education or conceptualization phase that provides information about trauma and trauma response and helps individuals establish short term, intermediate and long term goals; a rehearsal phase that has the individual practicing behavioural and cognitive coping strategies within a safe therapeutic environment; and the implementation phase that involves the person applying these strategies in their life, monitoring their effectiveness, and making necessary adjustments. Paired with exposure therapy, this particular form of CBT is believed to be effective in reducing trauma symptoms (Robertson et al., 2004). However, effectiveness studies are limited in number.

Similar to stress inoculation therapy, trauma management therapy combines exposure therapy, training in social and emotion management skills and an opportunity for practicing these skills. Although it has been applied solely in the treatment of combat veterans it has shown promising results (see Robertson et al., 2004). The link between these two forms of intervention with the successful CBT and exposure therapy likely account for some of their effectiveness.

In addition to CBT and exposure therapy, eye movement desensitization and reprocessing (EMDR) has been frequently used as an intervention method with trauma survivors, including those with PTSD (Ponnian & Hollon, 2009). EMDR involves having the person talk about their trauma while moving their eyes rhythmically from left to right. It is believed this will reset or activate connections between brain structures that were impaired by the experience of trauma (see Robertson et al., 2004). EMDR has been found to reduce PTSD symptoms better than relaxation training, biofeedback, and stress management (Bisson et al., 2007; Silver, Brooks, & Obenchain, 1995). Some have suggested that the exposure component of EMDR may account for its effectiveness, making it an alternate form of exposure therapy (see Robertson et al., 2004).

Emotion focused therapy has been applied in the treatment of complex trauma (Pavio & Pascual-Leone, 2010). It begins with a process of emotional awareness and acceptance, where individuals become more aware of their emotions and go through a process of describing and labelling these emotions. This is followed by a process of emotional transformation where adaptive and positive emotions are reinforced as guides to behaviour and maladaptive emotions

are the focus of change (Greenberg, 2004; Greenberg & Angus, 2004). One means of transforming negative emotions is to focus on and encourage positive emotions. Positive emotions are related to resilience and flexibility in thinking and they counteract negative emotions (Fredrickson, 2001; Davidson, 2000). Utilizing emotion focused coping strategies such as reappraisal and learning skills and strategies such as meditation, positive self talk, relaxation and self compassion helps individuals to manage overwhelming negative emotions (Greenberg, 2004). Because the transformation process often involves changing life narratives, this therapy works well with narrative therapy. Emotion focused therapy can enhance CBT by establishing effective emotion regulation skills before implementing exposure therapy and introducing relationship skills (Cohen, 2008; Greenberg & Goldman, 2008; Gurman & Jacobson, 2002; Johnson, 2002; 2004; Johnson, Bradley, Furrow, Lee, & Palmer, 2005).

Drug therapy in the form of antidepressants has been applied in the treatment of PTSD (Harvard Medical School, 2005). These drugs are also effective in dealing with comorbid depression and anxiety. Paroxetine, a selective serotonin re-uptake inhibitor, has been effective in treating re-experiencing, avoidance, numbing, and hyperarousal symptoms of PTSD (Hein, Jiang, Campbell, Hu, Miele, Cohen, Brigham, et al., 2009). The primary problem with the sole use of medication is that the underlying cause of PTSD is not addressed. In fact, more significant reductions in symptoms were found with exposure therapy, which does address the underlying cause of the symptoms. These effects were found for both PTSD and non-PTSD symptoms. Further, fewer individuals dropped out in the exposure therapy intervention (McFarlane & Yehuda, 2000). Drug and psychotherapies may be more effectively combined with CBT if the medication brings arousal levels down enough for the person to focus on more cognitive therapeutic interventions (see Robertson et al., 2004).

Given that the symptoms associated with trauma and PTSD vary considerably across individuals and time, it has been suggested that different approaches may be necessary (McFarlane & Yehuda, 2000). Perhaps the greatest predictor of therapeutic success is the client/therapist relationship, with trusting, comfortable relationships being conducive to progress and negative relationships having a detrimental effect (see Robertson, et al., 2004). Thus, a good therapeutic relationship, the application of exposure and cognitive-behavioural therapies, and flexibility in methods and approaches appear to be among the most effective components in recovery from experiences of trauma.

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