

## ABSTRACT

### Investigation of Different Trauma Types That May Result in PTSD

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Trauma is a common stressor that many people have experienced or will experience at some point during their lifetime. For some, this trauma will lead to persistent, chronic, stress and life-altering symptoms that are associated with posttraumatic stress disorder (PTSD). Several different types of trauma can lead to the development of PTSD. This review will focus on PTSD from military trauma, personal trauma (including sexual and physical abuse), exposure to natural disaster, and involvement in a motor vehicle accident. Individuals with PTSD struggle with neural abnormalities and the disorder also results in other pathology including depression and anxiety. Notably, PTSD does not appear to be trauma specific and various traumas, such as those discussed in this review, can all lead to the characteristic symptoms and pathology. Several efficacious treatments for PTSD have been identified including CBT, EMDR, PE, virtual reality, CPT, and IPT. Further research should continue to focus on efficacious treatments as well as providing resources to individuals who have experienced traumatic events. A special emphasis should be placed on making treatment available to victims that currently do not have the means to attain it.

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INVESTIGATION OF DIFFERENT TRAUMA TYPES THAT MAY RESULT  
IN PTSD

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## TABLE OF CONTENTS

Dedication.....	iii
Chapter One: Introduction to PTSD .....	1
Chapter Two: PTSD from War Trauma.....	8
Chapter Three: PTSD from Sexual or Physical Assault .....	17
Chapter Four: PTSD from Natural Disaster.....	25
Chapter Five: PTSD from Motor Vehicle Accident .....	33
Chapter Six: Discussion.....	41
Bibliography .....	43

## DEDICATION

To my parents, Jim and Shellie, for always believing in me and pushing me to set my sights high and encouraging me to never stop until I've met my goals. You are the best parents a person could ask for. Also, thank you to my husband, Jake, for all the love and support throughout this process. I love you all.

## CHAPTER ONE

### Introduction to PTSD

Posttraumatic Stress Disorder (PTSD) is a serious psychological disorder that negatively impacts those who suffer from its effects. Although the prevalence estimates vary, the suffering that an individual endures is undeniable and can include life interference, inability to work, and cognitive deficits related to neurological impairments. The causes of PTSD are heterogeneous and can include exposure to military trauma, personal trauma (physical and/or sexual abuse), exposure to natural disasters, and involvement in motor vehicle accidents, among others. The available clinical treatments are also varied.

#### *Diagnostic Criteria*

According to the DSM-5 (American Psychiatric Association, 2013) for adults, adolescents, and children six years and older, PTSD is defined by eight different criteria labeled A through H. The first criterion, A, is exposure to actual or threatened death, serious injury, or sexual violence in one or more ways including directly experiencing the traumatic event; witnessing the event as it occurred to others; learning that traumatic event occurred to a close friend or family member; or experiencing repeated or extreme exposure to aversive details of the traumatic event. The latter subset does not include traumatic exposure through electronic media, TV, movies, or pictures, unless the exposure is related to work.

The second criterion, B, is the presence of intrusion symptoms associated with the traumatic event, beginning after the traumatic event, such as recurrent, involuntary, and

intrusive distressing memories of the traumatic event; recurrent distressing dreams in which the content and/or the dream's affect are related to the traumatic event; dissociative reactions, such as flashbacks, in which the individual feels or acts as if the traumatic event was recurring; prolonged or intense psychological distress following exposure to internal or external cues that are symbolic or resemble an aspect of the traumatic event; and/or marked physiological reactions to external or internal cues that are symbolic or resemble an aspect of the traumatic event.

The third criterion, C, is the persistent avoidance of stimuli that are associated with the traumatic event, which begins after the traumatic event occurred, as is evident from either avoidance of or efforts to avoid distressing memories, thoughts, or feelings that are related or closely associated with the traumatic event and/or avoidance or efforts to avoid external reminders that cause distressing memories, thoughts, or feelings about or that are closely related to the traumatic event.

Criterion D is negative alterations in cognition and mood that are associated with the traumatic event, which begins or worsens after the traumatic event as evidenced by the existence of two or more symptoms including the inability to remember an important aspect of the traumatic event which is typically due to dissociative amnesia and not to other factors like head injury, alcohol, or drugs; persistent and exaggerated negative beliefs or expectations about oneself, others, or the world; persistent and distorted thoughts about the cause or consequences of the traumatic event that cause the individual to blame themselves or others; persistent negative emotional state; markedly diminished interest or participation in significant activities; feelings of detachment or estrangement from others; and/or persistent inability to experience positive emotions.

Criterion E, is defined by significant alterations in arousal and reactivity that are associated with the traumatic event, which begin or worsen after exposure to the traumatic event and must fulfil at least two of the following: irritable behavior and/or angry outbursts (with little/no provocation); reckless and/or self-destructive behavior; hypervigilance; exaggerated startle response; problems with concentration; and/or sleep disturbances such as difficulty falling asleep, staying asleep, or restless sleep.

Criterion F is the duration of the disturbances listed in B through E. The duration must be greater than one month.

Criterion G requires that the aforementioned disturbances cause clinically significant distress and/or impairment in social, occupational, or other functioning problems.

Finally, the last criterion, H, is the disturbance must not be attributable to the physiological effects of a substance, like medication or alcohol, and/or another medical condition.

### *Neural Correlates*

Cortisol is a glucocorticoid hormone that is responsible for many regulatory functions in the CNS, immune system, and metabolic system (Sapolsky, Romero, & Munck, 2000). Stress activates the hypothalamic-pituitary-adrenocortical (HPA) axis which results in the release of cortisol. With increased exposure to stress the HPA axis is activated for prolonged periods of time which leads to physiological wear and tear known as allostatic load (McEwen, 1998). Elevated cortisol levels inhibit the HPA by negative feedback mechanisms in the pituitary, hypothalamus, and hippocampus. PTSD has been

associated with lower basal cortisol levels, increased HPA feedback function, as well as ongoing sensitization of the HPA-axis (Morris, Compas, & Garber, 2012).

Surprisingly, in a meta-analysis of 37 articles including 828 people with PTSD and 800 controls, Meewisse, Reitsma, De Vries, Gersons, and Olf (2007) found no overall systematic difference in basal cortisol levels between individuals with PTSD and controls. These studies published between 1980 and 2005 focused on several different trauma-exposed population including combat veterans, victims of sexual or physical abuse (including childhood abuse), refugees, and other various trauma types. However, cortisol levels were assessed in several ways including plasma/serum, saliva, or 24h urinary free cortisol samples and this variation obscured true differences. Studies reported lower cortisol in subjects with PTSD when measuring plasma or serum cortisol. Differences in subgroups were also present with lower cortisol in subjects with PTSD in studies of physical and sexual abuse and when samples were collected in the afternoon. Based on these results the authors concluded that the presence of low cortisol levels may depend on the method of assessment, on gender and on the type of trauma exposure.

A meta-analysis of neuroimaging studies of individuals with PTSD engaged in script-driven symptom provocation, script-driven imagery, actual imagery, or sounds, found activation of several brain areas. These areas included the bilateral anterior and middle cingulate cortex, the right posterior cingulate cortex and also the bilateral medial frontal gyrus, especially at the level of the orbitofrontal cortex, middle frontal gyrus, and left insula (Boccia, D'Amico, Bianchini, Marano, Giannini, & Piccardi, 2016). They also found activation of the right hippocampus, parahippocampal gyrus, bilateral superior temporal gyrus, claustrum, and specific thalamic nuclei. But when examined in relation

to the specific type of trauma experienced, such as combat, sexual abuse or natural disasters, different brain “networks” were seen in different types of trauma, as will be discussed in the individual trauma sections of this paper. Woon, Sood, and Hedges (2010) also found reduced right hippocampal volume in subjects with PTSD. Patients who developed PTSD after a Tokyo subway sarin attack in 1995 displayed smaller bilateral amygdala volume and less gray matter density in the left anterior cingulate cortex than those without PTSD. Smaller left amygdala volume was further associated with greater severity of PTSD avoidance symptoms (Rogers et al, 2009).

Broekman, Olf, and Boer (2007) found the serotonin transporter gene might play a role in the degree of response to stressful events, especially in the sensitivity of individuals to the depressing sequelae of stressful events. Results for the dopamine system were inconsistent.

### *Prevalence*

According to the DSM-5, the 12-month prevalence of PTSD among U.S. adults is approximately 3.5% (APA, 2013).

In Sweden, 5.6% met the criteria for PTSD (Frans, Rimmo, Aberg, & Fredrikson, 2005). Increased migration over the twenty years prior to the study due to “war and other misfortunes” prompted the authors to compare immigrants to native citizens (p. 3). Subjects born abroad were three times more likely to develop PTSD. Notably, 7.4% women and 3.6% men reported the syndrome even though men reported experiencing greater trauma exposure.

Olf, Langeland, Draijer, and Gersons (2007) attempted to explain the higher rates of PTSD in women and found that women’s higher risk of PTSD might be due to several

factors: the type of trauma experienced, which tends to be interpersonal violence, specifically of a sexual nature; young age at the time of the trauma; stronger perceptions of threat as well as loss of control; higher levels of peritraumatic dissociation; insufficient social support; and greater use of alcohol in order to manage trauma-related symptoms.

Roberts, Gilman, Breslau, Breslau, and Koenen (2011) analyzed structured diagnostic interviews of 34,653 adults who responded to the 2004-2005 administration of the National Epidemiologic Survey on Alcohol and Related Conditions, comparing trauma exposure, risk for PTSD among those exposed to trauma, and treatment seeking behavior among Whites, Blacks, Hispanics and Asians in the US general population. The lifetime prevalence of PTSD was highest among Blacks, 8.7%, intermediate among Whites and Hispanics, 7.4% and 7.0% respectively, and lowest among Asians, 4.0%. Whites were more likely than other ethnicities to experience any trauma, to learn of a trauma to someone close, and/or to learn of an unexpected death. However, Blacks and Hispanics were at a higher risk of child maltreatment, especially witnessing domestic violence. Notably, Asians (of both genders), Black men, and Hispanic women had higher risk of war-related events than Whites. Also, minority groups were less likely to seek treatment for PTSD than Whites (Roberts et al, 2011).

### *Treatment*

Many people that face traumatic events in their life do not develop PTSD and are able to move beyond the aversive experience. For subjects with persistent PTSD, however, it is much more difficult to put the trauma into the past. According to Ehlers and Clark (2000) three things must change in order for those with PTSD to do this, and they are: 1) the trauma memory needs to be elaborated and integrated into the context of

the individual's preceding and subsequent experience in order to reduce intrusive re-experiencing; 2) problematic appraisals of the trauma and/or its results that maintain the sense of current threat need to be modified; and 3) dysfunctional behavioral and cognitive strategies that prevents memory elaboration, intensifies symptoms or hinders reassessment of problematic appraisals need to be eliminated.

Trauma-focused cognitive-behavioral therapy showed benefits over usual care, defined as waiting-list or other psychological interventions, on all measures of PTSD symptoms. The treatments with the most empirical support were trauma-focused psychological treatments that specifically address the patient's troubling memories of the traumatic event as well as the personal meanings of the event and its consequences. This suggested that psychological treatment focused on trauma, such as trauma-focused cognitive-behavioral therapy or eye movement desensitization and reprocessing, are most effective for chronic PTSD (Bisson et al, 2007).

## CHAPTER TWO

### PTSD from War Trauma

Due to extreme and constant exposure to stress of deployment, war and combat, military personnel are at increased risk of developing PTSD. And even though the risk is elevated, the majority of military personnel still don't develop the disorder. In combat and non-combat veterans alike, there is a correlation between the degree of PTSD symptoms and the severity of combat exposure (Myers, VanMeenen, & Servatius, 2012). Behavioral inhibition, withdrawing from or avoiding novel social or non-social situations, and anxiety also correlated to PTSD symptom severity. Specifically, behavioral inhibition was related to criteria C symptoms. This, however, is not to say that non-combat veterans can't develop PTSD. Non-combat veterans with little to no history of combat exposure are still at risk of experiencing and reporting high rates of PTSD symptoms, likely linked to stress from deployment or reintegration into civilian life and other stressors related to service. Booth-Kewley, Larson, Highfill-McRoy, Garland, and Gaskin (2010) found that deployment-related stressors, such as "lack of time", "concerns or problems back home", and "problems with supervisor(s) or chain of command", were more strongly associated than combat exposure with self-reported PTSD symptoms (p.71). Regardless of the exposure military personnel have to combat, PTSD causes considerable deficits and requires our attention and resources to provide the best help for these individuals.

Although PTSD has been recognized for over thirty years (APA, 1980), there is still controversy over the legitimacy of the disorder specifically among military personnel. Fisher (2014) identified two common beliefs held by public officials,

clinicians and stakeholders. First, PTSD is viewed by some as an overly generalized or invalid diagnostic category that is often induced or can be falsified by veterans or service members. These individuals believe that PTSD is over-diagnosed. Second, others believe that many service members and veterans do not seek treatment when experiencing PTSD symptoms and thus the disorder is under-diagnosed.

Many interviewees, especially public officials, claimed that PTSD is over-diagnosed among service members and veterans. Most credited the high number of diagnoses to a flawed diagnostic category or to diagnosis-seeking behavior of individual service members. Of those that blamed diagnostic category for the over-diagnosis of PTSD, one of two arguments was made. First, PTSD is a sociopolitical, not clinical, construct created by the psychiatric community as well as veterans' advocates, and therefore is not a valid clinical diagnosis. Second, PTSD is a "catch-all" diagnosis. There was also a less popular held opinion that the current PTSD construct is not completely invalid, but that it confuses other diagnoses like depression and anxiety (Fisher, 2014).

Participants that focused on the military personnel's behavior as an explanation, expressed three separate claims for the over-diagnosis of PTSD. First, veterans are part of an overly entitled group that leads to seeking disability compensation. Second, veterans are "incentivized" to either seek or maintain a diagnosis of PTSD. That is, the veterans' disability compensation encourages some service members and veterans to become and/or remain ill. Third, and finally, veterans are advised to seek a diagnosis and disability compensation by individuals within the VA or by veterans' advocates for every perceived physical or psychological ailment upon discharge in order to prove that any existing problems, or those that might arise, are service-connected (Fisher, 2014).

There were also individuals that claimed PTSD is under-diagnosed in military personnel. Underdiagnosis is due to stigmatization and/or discrimination, creating a barrier to seeking either a PTSD diagnosis or treatment (Fisher, 2014). Several interviewees also thought that service members might fear their outward display of PTSD symptoms or their seeking of treatment would threaten their military careers. Many individuals who reported a need to reduce stigma in the military also conveyed other messages, such as asserting that individuals had an invalid diagnosis or labeling them as malingerers, which could lead to the stigmatization of service members and veterans expressing symptoms or seeking treatment for PTSD, thus creating a paradox.

### *Prevalence*

Even though PTSD has been recognized since 1980, there is still not a definitive estimate for the prevalence of PTSD amongst military personnel. Estimates have ranged from 2 to 17% and beyond (Richardson, Frueh, & Acierno, 2010) and are affected by combat roles and cultural background as well as issues in the designs of the multiple studies including sampling strategies, varied measures, and inclusion/exclusion criteria (Creamer, Wade, Fletcher, & Forbes, 2011). Notably, a common result in several studies is that US military personnel report higher rates of PTSD than other western nations (Richardson et al., 2010). However, US military personnel also tend to be younger, experience longer deployments, and have a lower socioeconomic status than do other military personnel which might contribute to their increased reports of PTSD symptoms (Creamer et al., 2011).

### *Predictors*

Multiple factors aside from the actual trauma experience were associated with PTSD in military personnel, namely pre-trauma factors including being female, having lower education, being non-White, not being an officer, serving in the army, more deployments, a longer total length of deployments, experiencing general adverse life events, experiencing prior trauma, and having prior psychological problems (Xue, Ge, Tang, Liu, Kang, Wang, & Zhang, 2015). A large amount of variance was accounted for by race, rank, branch of service, occupation, number of deployments, length of deployments, experiencing adverse life events, and having prior psychological problems. There were inconsistencies in results for, marital status, smoking, low socioeconomic status, and prior trauma as related to PTSD. Several of these factors are also reflected in the DSM-5 (APA, 2013).

When considering peri-traumatic risk factors and their effect on the development of PTSD, combat specialists are more likely to suffer from PTSD, and the risk is increased with greater combat exposure, including experiencing the discharge of a weapon. Other risk factors identified were witnessing someone being wounded or killed, severe trauma, and deployment related stressors. Variance components included unit support, combat exposure, discharging a weapon, witnessing someone be wounded or killed, and trauma severity (Xue et al, 2015). Military personnel were more likely to develop PTSD after they returned from deployment if they suffered from ensuing adverse life events.

These results show that the list of risk factors that interact to determine whether or not military personnel will develop PTSD is lengthy and includes personal variables,

peri-combat factors, military related factors and life events after returning from combat. There are several identified risk factors, but as noted by Xue and colleagues (2015), there is also a vast amount of variance for each risk factor and not all studies have come to the same consensus for each. Since the results are inconclusive, identifying at least a hierarchy of risk factors is an area of PTSD research that needs further work yet, we will most likely never have a stable list of risk factors that result in the development of PTSD because the nature of the military and war changes over time.

### *Cognitive Deficits*

Cortical thickness is a result of the size, density, and arrangement of neurons, glial cells and nerve fibers. Cortical thinning usually reflects changes in the boundary between grey and white matter that is related to a loss of dendrites and dendritic spines or changes in myelination within specific brain systems (Geuze, Westenberg, Heinecke, de Kloet, Boebel, and Vermetten, 2008). Significantly lower cortical thickness values in the frontal and temporal lobes was observed. Specifically, abnormalities in the bilateral superior and middle frontal gyri, left inferior frontal gyrus, as well as the left superior temporal gyrus were seen in veterans with PTSD relative to controls (Geuze et al., 2008). This reduction in the frontal areas of the brain is likely associated with the decline in working memory present in subjects with PTSD. Deficits present in frontal and temporal lobes of PTSD subjects have been identified in other studies (e.g. Francati, Vermetten, & Bremner, 2007; Nemeroff, Bremner, Foa, Mayber, North, & Stein, 2006) as related to many of the cognitive deficits present in PTSD such as impairments in memory, increased fear response, reckless or destructive behavior, and problems concentrating. Geuze cites other work that indicates regions including the dorsolateral prefrontal cortex, the ventrolateral

prefrontal cortex, the medial prefrontal cortex, the anterior cingulate cortex, and orbital frontal cortex exhibit altered function in PTSD patients compared to controls (Geuze et al., 2008).

### *Treatment*

Once PTSD has developed, there are various approaches to treatment. Creamer and colleagues list some effective treatments for PTSD. One such effective treatment for PTSD is eye movement desensitization and reprocessing (EMDR). EMDR requires individuals to focus their attention on a traumatic memory whilst simultaneously tracking the therapist's finger as it moves across the subjects' visual field, and then continue to engage in a restructuring of the memory (Boccia, Piccardi, Cordellieri, Guariglia, & Giannini, 2015). Eye movement is the most common bilateral stimulation, however, auditory or sensory stimulation might also be used. Several theories provide possible explanations for the effectiveness of EMDR including the fact that stimulation acts on disintegrated information that is related to the traumatic event, stimulation boosts the processing of emotional memories, or stimulation increases emotional processing in general. EMDR has also been successful in mixed trauma samples, indicating that it is beneficial for more than just combat related trauma.

Another successful treatment is trauma-focused cognitive behavioral treatment (TFCBT) and TFCBT is commonly the first intervention when treating PTSD in military personnel. TFCBT helps the individual confront memories of their traumatic experience as well as the feared and avoided external situations that remind them of these events. It also addresses beliefs and maladaptive interpretations about the events that are interfering

with recovery. Two interventions within TFCBT that have been shown to be efficacious include prolonged exposure and cognitive processing therapy (Creamer et al., 2011).

Prolonged exposure therapy is the most utilized exposure treatment program for PTSD and it has been shown to be efficacious in reducing the symptoms of PTSD as well as related depression, anger, and guilt (Rauch et al., 2009; Smith et al., 2015). Three primary components comprise prolonged exposure therapy: psychoeducation, in vivo exposure, and imaginal exposure followed by processing. The Veterans Health Administration conducted a nationwide prolonged exposure training initiative in order to make evidence-based treatment more readily available to Veterans diagnosed with PTSD (Smith et al., 2015). Due to the vast number of Veterans in need of PTSD treatment, interest in group therapies has grown in order to keep up with the increasing demand of treatment. Group therapies may yield potential benefits compared to individual therapy such as the opportunity for positive social interaction and bonding. Unfortunately, in recent studies the effect size for group exposure treatment has been much smaller than the effect size seen for individual exposure treatment (Smith et al., 2015). Smith and colleagues designed and implemented a treatment plan that incorporated both group and individual prolonged exposure treatment and found the treatment to be effective (having a moderate to large effect size), more so than group treatment alone, which typically had a small to moderate effect size.

Another approach that appears to be promising for the treatment of PTSD in military personnel is virtual reality. Virtual reality treatment integrates computer graphics and visual displays that enable the users to feel immersed in the virtual environments. In order for a reduction in fear to occur, the fear relevant information that is coupled with

the patient's memory of the traumatic event must be accessed and activated through emotional engagement. The theory is that several exposures to the fear source in a safe environment would thus lead to a decrease in anxiety, through habituation and extinction. Virtual reality suggests that these obstacles can be addressed by delivering multiple sensory cues that might evoke the traumatic memory in a safe, controlled environment. One of the benefits of virtual reality treatment is that it represents a graded and systematic approach that can be adapted to each individual person which could combat the high rates of dropouts (Cukor et al., 2009).

There is a current virtual reality treatment titled, "Virtual Iraq," which allows the clinician to specialize the scene, sounds, vibrations, even the smells that the patient is experiencing in order to accurately match it to a similar traumatic event they had experienced in Iraq. The program has been shown to be efficacious and a 50% decrease in symptoms was reported in a clinical trial involving 20 participants (Cukor et al., 2009).

When virtual reality treatment was augmented with D-cycloserine (Seromycin), no overall differences on PTSD symptoms were observed at any time point. However, the D-cycloserine group reported the lowest scores both on cortisol reactivity and startle response during virtual reality scenes at post-treatment (Rothbaum et al., 2014). There are challenges with the virtual reality approach, namely the expense of the systems and the need to create several new environments in order to match each traumatic event and offer the best treatment.

Aside from clinical therapy, pharmacotherapy has also been effective in PTSD. Specifically, the administration of selective serotonin re-uptake inhibitors (SSRIs) has reduced the severity of PTSD symptoms as well as helped prevent relapse (Creamer et

al., 2011). Unfortunately, information regarding the treatment of PTSD in military personnel is less developed than one would hope for. This might be influenced by current military training that constructs styles of thought and behavior that are not conducive to help-seeking and response to treatment. Active military personnel also might avoid seeking help due to stigma and career-related concerns.

Although there are claims that PTSD is over or under-diagnosed in the military population, whatever the incidence, it is still recognized as a debilitating disorder that potentially alters the life of individuals for many years. Estimations of how many military personnel may suffer from PTSD are quite varied, ranging from 2-17% and beyond, but it goes without question that a significant number of people report severe PTSD symptoms and need treatment. Luckily, there are treatments for these individuals. The most common treatment is prolonged exposure but there are also other efficacious alternatives such as virtual reality that might help to reduce symptoms. There is still much research to be done in further developing treatments and tailoring them in order to offer the best care but the current efforts are headed in the right direction.

## CHAPTER THREE

### PTSD from Sexual or Physical Assault

One form of trauma that is all too common is sexual or physical abuse. The National Intimate Partner and Sexual Violence Survey included 18,049 people, 9,970 were women and 8,709 were men. The study found that 17.4% of women in the United States have been raped at some point in their lifetime (Walters, Chen, & Breiding, 2013). The survey also found that 43.3% of women reported experiencing some other form of sexual trauma including sexual coercion, unwanted sexual contact, and non-contact unwanted sexual experiences. The Australian National Mental Health and Well-being Survey suggested 27.4% of women have experienced gender based violence (Rees et al., 2011). Lifetime prevalence rates were highest for sexual assault (14.7%), followed by stalking (10.0%), rape (8.1%), and physical forms of intimate partner violence (7.8%). Also, the National Intimate Partner and Sexual Violence Survey reported that 0.7% of men reported having been raped and 20.8% reported experiencing other sexual trauma (Walters et al., 2013).

#### *Prevalence*

Suggested prevalence estimates vary for sexual or physical abuse. One specific form of sexual abuse that has been evaluated is military sexual abuse. A recent study reported that approximately 40% of female veterans reported experiencing sexual assault during their military experience and of those 35% met the clinical cutoff on the PTSD Checklist (Kintzle et al., 2015). A similar value of 40.8% was reported in another study that included men and women veterans (Voelkel, Pukay-Martin, Walter, & Chard, 2015).

Lower prevalence estimates have also been suggested. In a recent study, evaluating both men and women veterans, 15% (77.4% women and 5.8% men) had experienced military sexual assault (Tiet et al., 2015). Of these individuals, 68% of men and 69% of women had PTSD at the baseline assessment.

Prevalence estimates have also been suggested for non-military sexual assault. In one study, 481 veterans (273 men and 208 women) were recruited based on archived data of individuals who had been admitted to a residential PTSD treatment program from the Cincinnati Veterans Affairs Medical Center. Thus, not surprisingly 96.7% were diagnosed with current PTSD based on symptoms outlined in the DSM-IV (Voelkel, et al., 2015). Of the total veterans, 59.3% endorsed an index trauma different from military sexual assault including childhood sexual assault (8.9%) and physical assault (3.3%) amongst others. Another study indicated the prevalence of PTSD among women who have been exposed to one form of gender based violence to be about 15% (Rees et al., 2011). The PTSD prevalence estimates increased with an increase in the number of gender based violence events a women experienced (two: 30% and three-four: 56%). Gender based violence included physical violence, interpersonal violence, rape, other forms of sexual assault, and stalking.

As indicated by these studies, the percentage of victims affected by military sexual assault ranges from approximately 8% to 41%. Prevalence estimates have also been suggested for non-military sexual assault. These values range from approximately 3% to 56% depending on the number of traumatic events. It is also apparent from these studies that PTSD from sexual or physical abuse is a common occurrence and often

prevalence estimates are reported that are much greater than the value represented in the DSM-5 (APA, 2013).

### *Predictors*

Dunmore, Clark, and Ehlers (2001) aimed to define cognitive variables that significantly predicted PTSD severity. To do so, they assessed 57 victims of physical or sexual assault within four-months of assault, they also conducted six- and nine-month follow up assessments after the assault. Their results identified several cognitive variables that significantly predicted PTSD severity. The variables found were: cognitive processing style during assault, either mental defeat, mental confusion, or detachment; appraisal of assault sequelae; appraisal of symptoms; perceived negative responses of others and permanent change; negative beliefs about self and world; and maladaptive control strategies, such as avoidance and/or safety seeking (Dunmore et al., 2001). These predictors are reflected in the DSM-5 and they are important because they may provide crucial information in order to personalize PTSD treatment in order to target these factors and lead to more positive treatment outcomes.

### *Cognitive Deficits*

In an attempt to measure resting cerebral glucose metabolism and perfusion patterns in females with PTSD resulting from sexual assault, Kim and colleagues (2012) performed imaging tests (brain perfusion SPECT and <sup>18</sup>F-fluorodeoxyglucose PET) on 12 women with a current PTSD diagnosis. Compared to healthy controls, the individuals with PTSD had significantly reduced cerebral blood perfusion in the left hippocampus and the left basal ganglia (Kim et al., 2012). Also, the PTSD group had lower cerebral

glucose metabolic activity in the left hippocampus, left superior temporal gyri, and precentral gyri, relative to control subjects. Decreased blood flow in the left hippocampus and increased activation of the left superior temporal gyri to emotional stimuli have been suggested elsewhere in the literature (Bremner et al., 2003). However, in this study glucose metabolism in the cerebellum was also higher for individuals with PTSD. The authors proposed that the decreased perfusion and increased glucose metabolism might be related to memory deficiencies and facilitating fear extinction (Kim et al., 2012). They also suggested that the increased cerebellar activity may be linked to hyperarousal as well as increased heart rate variation, exaggerated startle response, and sleep abnormalities, which are all commonly associated with PTSD.

Deficits in the volume of the amygdala have also been suggested in the literature (Veer et al., 2015). Veer and colleagues (2015) examined a sample of 12 females diagnosed with PTSD who had experienced interpersonal trauma related to emotional abuse, emotional neglect, and/or sexual and/or physical abuse during childhood or adolescence. Results indicated that although there was no difference in the volumes of the bilateral hippocampus and left amygdala, the volume of the right amygdala was smaller than the healthy controls. Specifically, the differences were seen at the surface of the basolateral and superficial nuclei groups, which receive major input and feedback projections from sensory and prefrontal brain regions. The basolateral group is important for promoting emotional memory formation and fear conditioning. Results also showed that right amygdala volumes were associated with severity of sexual assault during childhood.

Another brain region that is commonly linked to PTSD is the insula. The insula is hypothesized to show exaggerated reactivity to the anticipation of aversive stimuli. To test this, 15 women with a history of intimate partner violence performed a task involving cued anticipation to images of both positive and negative events during fMRI. Their results were compared to healthy controls (Simmons et al., 2008). Although both groups exhibited activation in the bilateral anterior insula during anticipation of negative compared to positive stimuli, the PTSD subjects showed greater activation in the right anterior/middle insula during anticipation of negative compared to positive stimuli. Also, functional connectivity between activation in bilateral anterior insula and bilateral amygdala were significantly weaker in the women with PTSD than the control subjects. A positive correlation was also found between hyperarousal scores and activity in the left anterior insula for PTSD individuals. Results suggest that the anterior/middle insula activation might function as a warning associated with the anticipation of aversive symptoms.

Boccia and colleagues (2016) conducted a review of 16 papers that reported 55 individual experiments involving a total of 815 individuals to identify clusters of different neural changes associated with PTSD after different traumatic events. They performed an activation likelihood estimation analysis and found that for individuals with PTSD due to physical and/or sexual abuse, clusters of activation were observed in the bilateral anterior cingulate cortex and middle cingulate cortex, precuneus, superior occipital gyrus, and middle frontal gyrus. Activation of the middle frontal gyrus to emotional stimuli was identified in another study (Bremner et al., 2003). Similarly, decreased blood flow in the anterior cingulate cortex has also been suggested. The anterior cingulate, middle

cingulate, precuneus and middle frontal gyrus were brain regions that were commonly activated for all trauma types (Boccia et al., 2016). These areas are significant because the cingulate cortex is hypothesized to be involved in the facilitation and expression of conditioned fear.

As indicated by these studies, there are several inconsistencies that occur when investigating cognitive deficits and brain regions that are affected by PTSD from sexual assault. However, there are several brain regions that are commonly noted as being affected by PTSD. These regions are directly related to the many deficits that are characteristic of the disorder and the negative impact that they have on an individual's life.

### *Treatment*

One form of treatment for PTSD in sexual assault victims is cognitive processing therapy (CPT). Resick and Schnicke (1992) conducted a study in which 19 sexual assault survivors received CPT. Treatment consisted of 12 weekly sessions in a group format. Participants were assessed at pretreatment, posttreatment, as well as three- and six-month follow-ups. Results indicated that the sexual assault participants significantly improved from pretreatment to posttreatment on both PTSD and depression measures compared to controls. Participants also maintained their improvement for six months. Several other studies have examined CPT for sexual or physical abuse and have obtained results indicating that the treatment is successful in reducing PTSD and depression symptoms and improvement of symptoms is maintained one year after treatment (Chard, 2005; Walter, Buckley, Simpson, & Chard, 2014; Voelkel et al., 2015).

Cognitive-behavioral therapy (CBT) is another treatment that has been utilized to decrease PTSD and related symptoms. In one study, 74 women who had experienced childhood sexual assault and met the criteria for PTSD were randomized into three groups: CBT, problem-solving therapy (present-centered therapy; PCT), and wait-list (McDonagh et al., 2005). Both CBT and PCT were conducted in 14 individual sessions, approximately one session per week. After completion of treatment, both CBT and PCT resulted in significant improvements in PTSD symptom severity compared to participants that were wait-listed. Results also indicated that CBT and PCT were not significantly different in their ability to achieve sustained symptom reduction and improvement in cognitive distortions. However, CBT did show an advantage in achieving sustained change in PTSD diagnosis.

Cloitre, Koenen, Cohen, and Han (2002) examined the effects of CBT and prolonged exposure (PE) on a sample of 58 women that had a current diagnosis of PTSD related to childhood abuse. Individuals were randomly assigned to either a 16 session CBT or were waitlisted. The CBT employed in this study had two-phases. The first phase was eight weekly sessions of skills training in affect and interpersonal regulation (STAIR). The second phase was eight sessions of modified PE. Participants were evaluated pretreatment, and twice posttreatment at three- and nine-month follow up assessments. Results indicated that compared to the wait-list controls, women that received CBT showed significant improvement on affect regulation problems, interpersonal skills deficits, and PTSD symptoms. The improvements were maintained at both the three- and nine-month follow ups. STAIR/Exposure treatment was successful in another study in

attaining remission of PTSD and reducing problems with emotional regulation, anger expression, and anxiety, as well as maintaining these results (Cloitre et al., 2010).

Tiet and colleagues conducted a study involving 837 veterans (574 completed the four-month follow-up assessment) from seven VA PTSD specialty intensive treatment programs at five sites across the U.S. Of the 41 men that had experienced military sexual trauma, 68.36% reported PTSD symptoms at baseline. At the four-month follow-up assessment, 66.08% reported PTSD symptoms. In the same study there were 82 women that reported military sexual trauma. At baseline, 69.01% had PTSD symptoms and this number was reduced to 61.47% at the four-month follow-up assessment (Tiet et al., 2015). Treatment programs included individual and group CPT, prolonged exposure therapy, and eye-movement desensitization and reprocessing therapy. They found that patients who experienced military sexual trauma did not have worse treatment outcomes than those that did not experience military sexual trauma. However, results indicated that military sexual trauma predicted longer length of stay (as a mediator) which in turn predicted lower PTSD, lower depression, lower violence symptoms, and better quality of life.

With the high occurrence of sexual assault, it is important to be aware of the predictors to distinguish those individuals that are at the highest risk of developing PTSD to ensure that they get the help they need as quickly as possible. Also, although there are efficacious treatments, specifically CPT and CBT, it is ever important to perfect these treatments and ensure that the individual has access to the best treatment that fits their needs.

## CHAPTER FOUR

### PTSD from Natural Disaster

Hundreds of natural disasters occur each year, killing thousands of individuals and affecting many more. The Centre for Research on the Epidemiology of Disasters defines a disaster as “a situation or event which overwhelms local capacity, necessitating a request to a national or international level for external assistance or an unforeseen and often sudden event that causes great damage, destruction, and human suffering” (Guha-Sapir, Hoyois, & Below, 2015). Six natural disaster subgroup classifications exist according to EM-DAT, a worldwide database on disasters: 1) geophysical, including earthquakes, mass movement (dry), and volcanic activity 2) hydrological, including floods, landslides, and wave action 3) meteorological, including storms, extreme temperatures, and fog 4) climatological, including droughts, glacial lake outbursts, and wildfires 5) biological, including animal accidents, epidemics, and insect infestations and finally, 6) extra-terrestrial, which includes impact and space weather.

In 2014, 324 natural disasters in 99 countries were reported and caused the deaths of 7,823 people (Guha-Sapir et al., 2015). These natural disasters also left in their wake 140.7 million victims. Because of their nature, these disasters are often widespread and located all over the globe. The countries that had the most natural disasters were China, the United States, India, Japan, and the Philippines. These countries accounted for 31.1% of the total occurrence. According to the report, these countries are typically on the list of countries that experience the most disasters in a year.

The 10 countries that reported the most fatalities in 2014 represented 75.3% of the total disaster mortality (Guha-Sapir et al., 2015). These countries are Nepal, Afghanistan, Sri Lanka, Solomon Islands, Vanuatu, Tonga, Peru, Bolivia, Burundi, and Serbia. It is important to note that amongst the top 10 countries that had the highest mortality from natural disaster, seven countries are classified as low-income or lower-middle income economies. These seven countries accounted for 46.1% of the reported mortality.

The same trend of low-income and lower-middle income economies dominating the top 10 list is again seen for countries that rank highest in disaster victims—excluding fatalities. The top 10 countries in terms of victims represented 75.8% of the total 140.7 million victims reported. Asia dominated this category, accounting for 67.2% of all victims. China alone reported almost 65 million victims which is 46.1% of the annual total of disaster victims (Guha-Sapir et al., 2015).

The top 10 countries that reported the most damage represented 56.2% of the total disaster figure of \$99.2 billion (Guha-Sapir et al., 2015). Unlike the previous categories, the seven countries in the top 10 that reported the most damage are classified as higher and upper-middle income economies. China, India, and the U.S.A., accounted for 79.6% of the reported disaster damage (China: 29.2%; India: 23.4%; U.S.A.: 16.9%). These numbers indicate that natural disasters are a widespread event that affect numerous individuals and economies, across all income ranges, and are a struggle for all of humanity.

### *Prevalence*

Knowing that the number of natural disasters that occur each year is quite high and that thousands of people are killed and even more are affected by these events, it is

important to determine how many of these individuals are at risk for and subsequently do develop PTSD in order to assure that they have access to the necessary treatment. In an attempt to study multiple natural disasters and determine a prevalence of PTSD Neria, Nandi and Galea (2007) conducted a literature review including reports published between 1980 (when PTSD was first introduced in DSM-III) to February 2007. They included 116 studies from 40 natural disasters starting with the Vajont landslide and tidal wave flood disaster in Northeast Italy in 1963 and ending with Hurricane Katrina in August of 2005. The percentage range for the prevalence of PTSD that they found was surprisingly broad, ranging from 3% to 60%. However, most studies reported prevalence estimates in the lower half of that range. Another study based on 10 disasters found the prevalence of PTSD to be 19% (North & Oliver, 2013). Fortunately, results indicated that at follow-up 39% of those that had PTSD at baseline were no longer experiencing symptoms.

Several studies focusing on one specific natural disaster have also been conducted. One study conducted in Thailand after the 2004 Indian Ocean Disaster, estimated 36.5% of individuals developed PTSD after the tsunami (Hussain, Weisaeth, & Heir, 2011). However, only 11.1% still had PTSD two and a half years after the disaster. Approximately 37% of individuals with PTSD sought treatment which might have an influence on the decreased prevalence values two and a half years later. Another study investigated the effects of the 2004 tsunami (Arnberg, Bergh Johannesson, & Michel, 2013). Although approximately 73% of the participants fulfilled part of the PTSD criteria from the DSM-IV, only 11.3% met the criteria for a lifetime diagnosis of PTSD and only 4.2% fulfilled the criteria for a current PTSD diagnosis. Notably, 37% of participants

reported having received treatment for psychological concerns related to the disaster and 3.5% were currently in treatment. Therefore, it seems early treatment may have prevented some individuals that initially met some PTSD criteria from reaching a severity level demanding a diagnosis of PTSD.

On May 12<sup>th</sup>, 2008 an earthquake struck Wenchuan in China's Sichuan province. Guo et al. (2014) conducted a study from July 2008 to January 2012 that sampled from the population five times over these years. The samples included 1,066; 1,344; 1,210; 1,174; and 1,281; individuals, respectively. However, only 45 individuals were included in every successive sample of the survey, resulting in each sample being largely independent. The prevalence of PTSD was highest two months after the earthquake with 58.2% of the participants likely having PTSD. The prevalence value dropped significantly over time ending at the fifth sample at 44 months after the earthquake at 8.0%.

Another study utilized a sample of adult Chileans that had survived an earthquake measuring 8.8 on the Richter Scale and/or a tsunami that occurred on February 27<sup>th</sup>, 2010 (Fernandez et al., 2016). The study included 1,708 individuals and results indicated that 10% of the total sample reported clinically significant post-disaster PTSD.

The prevalence of PTSD after a natural disaster appears to range from as low as 3% to as high as 60% (Neria et al., 2007). However, even though numerous individuals seek treatment for psychological problems related to a traumatic event, several still experience persistent symptoms of PTSD. Also, because natural disasters occur all over the world, including isolated and lower-income areas, it is important to be aware of the

risk of developing PTSD to ensure that treatment is available to everyone, regardless of economic status.

### *Predictors*

Because natural disasters affect such a large number of individuals it is important to identify risk factors that predict the onset of PTSD after natural disaster. These predictors provide the means necessary to identify individuals that are at the highest risk of developing PTSD from experiencing a natural disaster. A great deal of effort has gone into identifying some of these risk factors, with some success.

Rosellini, Coggey, Tracy, and Galea (2014) analyzed 810 individuals that were residing in southern Mississippi at the time of Hurricane Katrina in order to determine predictors of PTSD. The factors that they found were having less than a high school education, experiencing greater severity of depression as well as suicidal ideation, having experienced more pre-hurricane traumatic events, having less social support, experiencing more post-hurricane stressors, and belief that the hurricane was a punishment from God. Similar cognitive variables have also been identified for other trauma types (Dunmore et al., 2001). Other notable predictors were belonging to a non-White racial group, being single, being personally injured by or knowing others who were injured or killed by the storm, seeing dead bodies during or after the storm, feeling helpless or terrified during the storm, and experiencing more Katrina-related traumatic events (Rosellini et al., 2014). Prior psychological disorders have also been identified as predictors of PTSD in natural disaster survivors (Fernandez et al., 2016).

Identified predictors should enable providers to identify individuals at high risk of developing PTSD which might allow for careful monitoring and prompt treatment to limit symptomology and disorder duration.

### *Cognitive Deficits*

Severe PTSD has been shown to cause neurological change. Because structure is so closely linked to function, it is important to determine if different trauma types may give rise to different types of neurological impairment. When examining the white matter in 88 victims of the Chinese earthquake in Sichuan Province, Li and colleagues (2016) found, based on voxel analysis, that the left dorsolateral prefrontal cortex (DLPFC) and the left forceps major of the corpus callosum showed significantly increased fractional anisotropy, indicating adverse tissue changes. They also observed decreased radial diffusivity and axial diffusivity in PTSD subjects relative to controls, specifically in the left superior and middle frontal gyri, these also being signs of tissue change.

Studies have also shown significant gray matter loss associated with PTSD from natural disaster exposure. Chen et al. (2012) examined 10 survivors from a coal mine flood disaster and compared them to healthy controls. Results indicated that individuals with PTSD had smaller gray matter volume in the left dorsal anterior cingulate cortex. They also found that PTSD subjects had greater gray matter volume in the right pulvinar and left pallidum. Different gray matter deficits were observed in a study conducted by Zhang et al. (2011) from a sample of coal mine flood disaster survivors with PTSD. Results indicated that PTSD subjects had smaller gray matter volumes in left anterior hippocampus, left parahippocampal gyrus, and bilateral calcarine cortex. They also found that PTSD symptom severity was associated with reduced gray matter density.

To test brain responses to symptom provocation as well as short-term memory recall in subjects with PTSD, Hou et al. (2007) examined 10 individuals that had survived a coal mining accident and then in their study underwent fMRI while performing various tasks. They observed that during symptom provocation, diminished responses were evident in right anterior cingulate gyrus, left inferior frontal gyrus, and bilateral middle frontal gyrus. They also noted enhanced left parahippocampal gyrus response relative to controls, but in a memory recall task, diminished right parahippocampal gyrus response was observed. During short-term memory recall, results showed reduced responses in right inferior frontal gyrus as well as right middle frontal and left middle occipital gyrus. These findings suggest that neural abnormalities in systems important for memory function occur in subjects suffering from PTSD resulting from experiencing a natural disaster.

Many behavioral and cognitive deficits are characteristic of PTSD. These deficits have also been directly related to brain abnormalities. A common neural deficit described in the currently reviewed studies is reduced brain volume or functioning in areas important to fear learning and memory. These results are not surprising as subjects with PTSD are known to have difficulties with fear and suffer the effects of increased fear on learning and memory. Knowing what deficits result from PTSD helps researchers and clinicians develop the best treatments to target these deficits.

### *Treatment*

Identifying predictors of PTSD and determining cognitive and neural impairments are important but developing efficacious treatments is obviously the next step. One form of treatment that has been tested on a population of subjects with PTSD from a natural

disaster is CBT (Lopes et al., 2014). In a review of the current literature, Lopes and colleagues identified 11 studies that utilized CBT as a treatment for PTSD. Of the 11 studies, seven used exposure techniques and two used problem solving techniques. All of the 11 studies reported a significant reduction of PTSD symptoms after treatment.

Interpersonal Psychotherapy (IPT) is another form of treatment that has been used to reduce symptoms in individuals suffering from PTSD (Jiang et al., 2014). IPT focuses on one of four areas: interpersonal disputes, role transitions, grief/loss, or interpersonal sensitivity/deficit and attempts to change existing relationships and social support in order to improve mood and anxiety symptoms. Twenty-two individuals with PTSD resulting from a Chinese earthquake underwent 12 weeks of IPT. Treatment was delivered in one hour weekly individual sessions. At baseline, 66.7% of the individuals in the IPT group had PTSD, at the end of treatment, however, this number was reduced to 13.6% (Jiang et al., 2014). A significant decrease in depression symptoms was also observed. Results indicate that IPT is a viable treatment to reduce the symptoms and diagnosis of PTSD among survivors of natural disasters.

Having identified at least two treatments that are efficacious in reducing PTSD symptoms in victims of natural disasters, it is also important to make these treatments readily available to individuals that need them. As indicated previously, the majority of countries in the top 10 list with the most fatalities and most individual victims have economic challenges. That means that individuals in those countries likely cannot afford expensive treatment and if they cannot afford it, they will not seek it. Therefore, it is crucial that when a country experiences a natural disaster, regardless of economic conditions, the victims should have access to the treatment they need.

## CHAPTER FIVE

### PTSD from Motor Vehicle Accidents

Another trauma type that can lead to the development of PTSD is motor vehicle accidents. According to the U.S. Department of Transportation and the National Highway Traffic Safety Administration (2014), in 2013, 32,719 people died in motor vehicle accidents in the U.S. alone. An additional 2.31 million people were estimated to have been injured in motor vehicle accidents, as well (National Center for Statistics and Analysis, 2014).

In 2013, there were 30,057 fatal crashes and 5,657,000 non-fatal crashes. Of the non-fatal crashes, approximately 28% resulted in injury to someone involved in the crash. The remaining 72% of the total non-fatal crashes caused property damage only (National Center for Statistics and Analysis, 2014). These numbers indicate that although a vast number of individuals are involved in and die in car crashes each year, there are far more non-fatal crashes—including non-fatal crashes that result in serious injuries and can lead to the development and diagnosis of PTSD. Also, these numbers represent the United States alone and there are undoubtedly millions of individuals from other countries that are involved in motor vehicle accidents that experience these same debilitating effects. Because there is such a large population of individuals that experience non-fatal injuries from car crashes, there is also a large population that is at risk of developing PTSD from their trauma experience.

### *Prevalence*

With so many injuries occurring due to motor vehicle accidents, it is important to determine an approximate prevalence of PTSD among these survivors. In a 2013 review of current literature across 35 papers, results varied greatly across studies resulting in a prevalence range from 6% to 45% (Heron-Delaney, Kenardy, Charlton, & Matsuoka, 2013). Of motor vehicle accident victims admitted to a level-1 trauma center for their injuries in 2014, 10% of the sample met PTSD diagnostic status six-weeks after the traumatic experience (Hruska et al., 2014). Ehring, Ehlers, and Glucksman (2006), in a study of the greater London area, found that 21.8% of participants met criteria for PTSD diagnosis after being treated at King's College Hospital's Accident and Emergency Department. The prevalence is higher than other reported rates, which could be influenced by the severity of the injury that was required as an inclusion criterion.

PTSD prevalence was also calculated by Karl and colleagues (2009) who recruited 110 individuals that had survived a motor vehicle accident that had occurred in the past six months. All participants were from Germany. From the total sample, 30% of the subjects were considered to have full PTSD. There were also 32 individuals that presented with some PTSD symptoms but did not qualify for a full diagnosis of PTSD. Thus, 59% of the total sample experienced noteworthy PTSD symptoms—even if they did not qualify for a full diagnosis. These high prevalence rates can be explained, in part, by the criterion used in this study: all participants were seeking treatment related to their motor vehicle accident (Karl et al., 2009).

As found with other trauma, PTSD may improve over time, as indicated by a decrease in prevalence as time passes post-accident. In one study, at one-month post-

accident, prevalence rates ranged from 8% to 45%, at three months 8% to 30%, at six months 6% to 28%, and at one year 7% to 26%. The decrease in PTSD prevalence over time is a trend often observed in other longitudinal studies (Kupchik et al., 2007; Neria et al., 2007).

These studies demonstrate that there isn't a single agreed upon value that represents the prevalence of PTSD amongst individuals that experience motor vehicle accidents. A broad range is to be expected because there are a variety of outcomes from a motor vehicle accident as well as varying degrees of severity of injuries incurred. Another variable that contributes to the differences in prevalence values is the various inclusion criteria used across studies. Prevalence ranges found in these studies range from as low as 6% (Heron-Delaney et al., 2013) to 30% (Karl et al., 2009). However, values greater than 50% have also been found in particular samples (Kupchik et al., 2007). These high prevalence rates reflect individuals experiencing one or more of several significant risk factors or who were seeking treatments. These high prevalence values prompt the need for early identification as well as the development and utilization of effective treatments.

### *Predictors*

Several factors may influence the development of PTSD after a motor vehicle accident. A 2006 study of individuals that had experienced a motor vehicle accident in the past six months and who reported experiencing feelings of fear, helplessness, horror, and/or the perception that they would die, reported high prevalence of PTSD diagnoses. Gender differences were also observed. For men, a significant predictor was losing their

job. Those who were unemployed were 9.94 times more likely to develop PTSD than those men who were employed (Beck et al., 2006).

Many other predictors have also been identified. Based on a literature review, twelve variables were found to be consistent predictors of PTSD in motor vehicle accident survivors. The factors were: rumination about the trauma; perceived threat to life; lack of social support; higher Acute Stress Disorder symptom severity; persistent physical problems contributing to limitations at work and in social life; previous emotional problems including the presence of PTSD, anxiety, depression, travel phobia, or irritability prior to the accident; previous anxiety disorder; and involvement in litigation/compensation (Heron-Delaney et al., 2013). Several of these predictors have been identified in other studies (Kaczmarek & Zawadzki, 2012; Yasan, Guzel, Tanam, & Ozkan, 2009; Zawadzki & Popiel, 2012). Other significant predictors identified in these studies are amount of material loss and experiencing additional stressful events.

Several factors have been found to significantly predict the severity of PTSD symptomology. Therefore, it is important to evaluate individuals post-accident for these traits. By identifying victims early, the severity of PTSD symptoms experienced as well as the diagnosis of PTSD might be reduced by early treatment. In order to provide treatment early, first the most efficacious treatments need to be identified. Also, treatments need to be readily available to individuals that have suffered motor vehicle accidents and show signs of experiencing PTSD symptoms.

### *Cognitive Deficits*

A number of brain abnormalities have been exhibited by subjects with PTSD resulting from different trauma types. It is important to determine the frequency of neural

impairments in survivors of motor vehicle accidents suffering with PTSD as well. Some alterations in white matter have been documented (Sun et al., 2013). Specifically, decreased fractional anisotropy in the right anterior cingulate, right inferior occipital gyrus, right middle temporal gyrus, left superior temporal gyrus, right midbrain, and left gyrus rectus/medial OFC. Subjects also showed decreased diffusivity in bilateral superior frontal gyrus, and increased diffusivity in the left subcallosal gyrus. Similar results have been suggested in other studies (Hu et al., 2016; Li et al., 2016). Increased radial diffusivity has also been observed in brain areas involving corpus callosum, cingulum, and hippocampus in PTSD subjects relative to controls in these studies.

Alterations in cortical thickness have been observed as well in individuals suffering from PTSD after experiencing a motor vehicle accident. In one study, Bing and colleagues (2013) examined 20 participants with PTSD using high resolution structural MRI as well as resting-state fMRI and compared them to healthy controls. Results indicated that in the left hemisphere, decreased cortical thickness was observed in the medial prefrontal cortex, the triangular part of the inferior frontal gyrus, and the anterior cingulate cortex (Bing et al., 2013). Significant cortical thinning was observed in the right hemisphere, specifically in the superior temporal gyrus relative to the controls. Increased amplitude of low-frequency fluctuation values was found in the left medial prefrontal cortex, the anterior cingulate, and the right cerebellum in PTSD subjects relative to controls. Several of these areas were also indicated in a previous study (Sun et al., 2013) and have an established relationship with PTSD.

As is apparent from these studies, several brain regions have been implicated as being abnormal in individuals with PTSD from motor vehicle accidents. Also, many of

the brain regions that exhibit alterations in subjects suffering from PTSD after motor vehicle accidents have been indicated in previous PTSD studies resulting from different trauma types. Since the brain is clearly affected by PTSD, no matter the precipitating trauma, it is important to develop treatments that target these areas and that are effective in reducing PTSD symptomology because PTSD clearly leads to brain dysfunction.

### *Treatment*

Since so many individuals experience motor vehicle accidents and a large percentage incur injuries it is not surprising that a number develop PTSD. Thus, it is crucial to identify treatments that have been shown to be effective in treating PTSD resulting from a motor vehicle accident.

As we have seen before, EMDR therapy is considered to be an effective treatment of PTSD. A meta-analysis was conducted to examine the efficacy of EMDR amongst individuals with PTSD specifically from motor vehicle accidents (Boccia et al., 2015). The study included six papers and reported nine individual experiments on 440 subjects with PTSD resulting from traffic accidents. Results indicated that EMDR activated the same brain regions that show alterations in PTSD subjects. Since it activates the same brain areas, and was observed to decrease symptomology, EMDR seems to be an efficacious treatment for PTSD resulting from involvement in a motor vehicle accident.

CBT is another treatment that has commonly been used to reduce PTSD symptom severity. Beck and colleagues (2009) examined the effects of group cognitive behavior therapy for chronic PTSD in 26 individuals that had survived motor vehicle accidents. Participants engaged in 14 weekly group sessions, each two hours long (Beck et al., 2009). To assure that each group was receiving the same treatment, the treatment was

manualized and followed for each group. Results showed that after completion of treatment, individuals in the CBT group experienced a significant reduction in PTSD symptoms. At posttreatment, 88.3% of the patients that received CBT no longer met criteria for a PTSD diagnosis and stability was still noted at a 3-month follow-up. It bears observing however that nine participants dropped out, for whatever reason, and were not helped and they represented one third of the initial sample.

Another common treatment for PTSD is exposure treatment. In a recent experiment, written exposure therapy was examined for 46 participants with a current diagnosis related to motor vehicle accident (Sloan et al., 2012). The written exposure therapy consisted of five weekly sessions. The first session lasted one hour and each subsequent session lasted approximately forty minutes. However, actual therapist time ranged from ten to twenty-five minutes per session. The remainder of the session time was spent writing about the same motor vehicle accident event each time. An emphasis was placed upon focusing on deep emotions and thoughts at the time of the accident. Results indicated that participants that underwent written exposure therapy experienced significant reductions in PTSD symptom severity both immediately posttreatment and at three- and six-month follow-ups. Thus exposure therapy, specifically written exposure therapy, appears efficacious in reducing PTSD symptoms resulting from a motor vehicle accident.

Many traffic accidents occur every year resulting in numerous deaths and even more injuries. After enduring a traumatic motor vehicle accident and potentially sustaining or witnessing an injury, many individuals are at risk of developing PTSD.

Thus it is crucial that predictors are used to identify individuals most at risk and ensure that effective treatment is available to those who suffer.

## CHAPTER SIX

### Discussion

Although various estimates have been suggested for the prevalence of trauma-induced PTSD, the disorder is clearly established as a potential result of exposure to various trauma types. The classes of trauma discussed in this thesis are military trauma, personal trauma (including sexual and physical abuse), exposure to natural disaster, and/or involvement in a motor vehicle accident. For these trauma types prevalence estimates range from 2% to nearly 60%, however many estimates reside in the lower end of this range. Nonetheless, these estimates are often much greater than the value presented in the DSM-5. This discrepancy underlines the need for identifying predictors useful for identifying individuals that are at an elevated risk of developing PTSD. Several predictors have been reflected in the current literature for each trauma type. Many of the predictors are conserved across trauma types and are present in the DSM-5, but there are also several unique predictors for each trauma type that should be on the mind of healthcare professionals who encounter potential sufferers of PTSD.

Individuals with PTSD struggle with many deficits due to the disorder. Several neural abnormalities have been discussed and these seem to often result in observable behavioral deficits. Specifically, PTSD affects performances such as memory, emotional function, and job performance. PTSD also results in other pathology including depression and anxiety. Notably, PTSD does not appear to be trauma specific and various traumas, such as those discussed in this review, all lead to similar characteristic signs and

symptoms. The numerous symptoms demand the availability of effective treatments in order to help reduce PTSD and its behavioral symptomology.

There are a number of treatments that appear to reduce symptom severity and have benefit if available and applied. Among these are CBT, EMDR, PE, virtual reality, CPT, and IPT. Variations of several of these treatments are represented in the literature and research continues to improve upon the existing treatments. However, as this enhancement of treatment continues, a more concerted effort needs to be made in order to make treatments available to every individual at risk of developing PTSD after exposure to a traumatic event. A special focus should be concentrated on making treatments available to individuals of low economic status.

A vast amount of research has been focused on PTSD including the various trauma types that result in the disorder and specific factors related to these trauma types such as their respective prevalence values, predictors, cognitive deficits, and treatments. Future research should focus on continuing to define cognitive deficits and improving treatments in order to target these specific deficits. Also, future direction should include making treatment more available, especially for those that currently do not have access to adequate care.

## BIBLIOGRAPHY

- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders*, (3<sup>rd</sup> ed.). Washington, D.C.: American Psychiatric Association
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders: DSM-5*. Washington, D.C.: American Psychiatric Association.
- Arnberg, F., Bergh Johannesson, K., & Michel, P. (2013). Prevalence and duration of PTSD in survivors six years after a natural disaster. *Journal of Anxiety Disorders*, 27(3), 347-352.
- Beck, J. G., Coffey, S. F., Foy, D. W., Keane, T. M., & Blanchard, E. B. (2009). Group cognitive behavior therapy for chronic posttraumatic stress disorder: An initial randomized pilot study. *Behavior Therapy*, 40, 82-92.
- Beck, J. G., Palyo, S. A., Canna, M. A., Blanchard, E. B., & Gudmundsdottir, B. (2006). What factors are associated with the maintenance of PTSD after a motor vehicle accident? The role of sex differences in a help-seeking population. *Journal of Behavior Therapy and Experimental Psychiatry*, 37, 256-266.
- Bing, X., Ming-guo, Q., Ye, Z., Jing-na, Z., Min, L., Han, C., ...& Shao-xiang, Z. (2013). Alterations in the cortical thickness and the amplitude of low-frequency fluctuation in patients with post-traumatic stress disorder. *Brain Research*, 1490, 225-232.
- Bisson, J. I., Ehlers, A., Matthews, R., Pilling, S., Richards, D., & Turner, S. (2007). Psychological treatments for chronic post-traumatic stress disorder. *British Journal of Psychiatry*, 190, 97-104.
- Boccia, M., D'Amico, S., Bianchini, F., Marano, A., Giannini, A. M., & Piccardi, L. (2016). Different neural modifications underpin PTSD after different traumatic events: An fMRI meta-analytic study. *Brain Imaging and Behavior*, 10, 226-237.
- Boccia, M., Piccardi, L., Cordellieri, P., Guariglia, C., & Giannini, A. M. (2015). EMDR therapy for PTSD after motor vehicle accidents: meta-analytic evidence for specific treatment. *Frontiers in Human Neuroscience*, 9(213), 1-9.
- Booth-Kewley, S., Larson, G. E., Highfill-McRoy, R. M., Farland, C. F., & Gaskin, T. A. (2010). Correlates of posttraumatic stress disorder symptoms in Marines back from war. *Journal of Traumatic Stress*, 23, 69-77.

- Bremner, J. D., Vythilingam, M., Vermetten, E., Southwick, S. M., McGlashan, T., Staib, L. H., ...& Charney, D. S. (2003). Neural correlates of declarative memory for emotionally valenced words in women with posttraumatic stress disorder related to early childhood sexual abuse. *Society of Biological Psychiatry*, *53*, 879-889.
- Broekman, B. F. P., Olf, M., & Boer, F. (2007). The genetic background to PTSD. *Neuroscience and Biobehavioral Reviews*, *31*, 348-362.
- Chard, K. M. (2005). An evaluation of cognitive processing therapy for the treatment of posttraumatic stress disorder related to childhood sexual abuse. *Journal of Consulting and Clinical Psychology*, *73*(5), 965-971.
- Chen, Y., Fu, K., Feng, C., Tang, L., Zhang, J., Huan, Y., ...& Yin, H. (2012). Different regional gray matter loss in recent onset PTSD and non PTSD after a single prolonged trauma exposure. *PLOS one*, *7*(11), 1-8.
- Cloitre, M., Koenen, K. C., Cohen, L. R., & Han, H. (2002). Skills training in affective and interpersonal regulation followed by exposure: A phase-based treatment for PTSD related to childhood abuse. *Journal of Consulting and Clinical Psychology*, *70*(5), 1067-1074.
- Cloitre, M., Stovall-McClough, K. C., Nooner, K., Zorbas, P., Charry, S., Jackson, C. L., ...& Petkova, E. (2010). Treatment of PTSD related to childhood abuse: A randomized controlled trial. *American Journal of Psychiatry*, *167*(8), 915-924.
- Creamer, M., Wade, D., Fletcher, S., & Forbes, D. (2011). PTSD among military personnel. *International Review of Psychiatry*, *23*, 160-165.
- Cukor, J., Spitalnick, J., Difede, J., Rizzo, A., & Rothbaum, B. O. (2009). Emerging treatments for PTSD. *Clinical Psychology Review*, *29*, 715-726.
- Dunmore, E., Clark, D. M., & Ehlers, A. (2001). A prospective investigation of the role of cognitive factors in persistent posttraumatic stress disorder (PTSD) after physical or sexual assault. *Behaviour Research and Therapy*, *39*, 1063-1084.
- Ehlers, A. & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy*, *38*, 319-345.
- Ehring, T., Ehlers, A., & Glucksman, E. (2006). Contribution of cognitive factors to the prediction of post-traumatic stress disorder, phobia and depression after motor vehicle accidents. *Behaviour Research and Therapy*, *44*, 1699-1716.
- Fernandez, C. A., Vicente, B., Marshall, B. D. L., Koenen, K. C., Arheart, K. L., Kohn, R., ...& Buka, S. L. (2016). Longitudinal course of disaster-related PTSD among a prospective sample of adult Chilean natural disaster survivors. *International Journal of Epidemiology*, *00*, 1-13.

- Fisher, M. (2014). PTSD in the U.S. military, and the politics of prevalence. *Social Science & Medicine*, *115*, 1-9.
- Francati, V., Vermetten, E., & Bremner, J. D. (2007). Functional neuroimaging studies in posttraumatic stress disorder: Review of current methods and findings. *Depression and Anxiety*, *24*, 202-218.
- Frans, O., Rimmo, P. A., Aberg, L., Fredrikson, M. (2005). Trauma exposure and post-traumatic stress disorder in the general population. *Acta Psychiatr Scand*, *111*, 291-299.
- Geuze, E., Westenberg, H. G. M., Heinecke, A., de Kloet, C. S., Goebel, R., & Vermetten, E. (2008). Thinner prefrontal cortex in veterans with posttraumatic stress disorder. *NeuroImage*, *41*, 675-681.
- Guha-Sapir, D., Hoyois, P., & Below, R. (2015). *Annual disaster statistical review 2014: The numbers and trends*. Brussels: CRED.
- Guo, J., Wu, P., Tian, D., Wang, X., Zhang, W., Zhang, X., & Qu, Z. (2014). Post-traumatic stress disorder among adult survivors of the Wenchuan Earthquake in China: A repeated cross-sectional study. *Journal of Anxiety Disorders*, *28*, 75-82.
- Heron-Delaney, M., Kenardy, J., Charlton, E., & Matsuoka, Y. (2013). A systematic review of predictors of posttraumatic stress disorder (PTSD) for adult road traffic crash survivors. *Injury, Int. J. Care Injured*, *44*, 1413-1422.
- Hruska, B., Irish, L. A., Pacella, M. L., Sledjeski, E. M., & Delaganty, D. L. (2014). PTSD symptom severity and psychiatric comorbidity in recent motor vehicle accident victims: A latent class analysis. *Journal of Anxiety Disorders*, *28*, 644-649.
- Hou, C., Liu, J., Wang, K., Li, L., Liang, M., He, Z., ...& Jiang, T. (2007). Brain responses to symptom provocation and trauma-related short-term memory recall in coal mining accident survivors with acute severe PTSD. *Brain Research*, *1144*, 165-174.
- Hu, H., Zhou, Y., Wang, Q., Su, S., Qiu, Y., Ge, J., ...& Xiao, Z. (2016). Association of abnormal white matter integrity in the acute phase of motor vehicle accidents with post-traumatic stress disorder. *Journal of Affective Disorders*, *190*, 714-722.
- Hussain, A., Weisaeth, L., & Heir, T. (2011). Psychiatric disorder and functional impairment among disaster victims after exposure to a natural disaster: A population based study. *Journal of Affective Disorders*, *128*, 135-141.

- Jiang, R. F., Tong, H. Q., Delucchi, K. L., Neylan, T. C., Shi, Q., & Meffert, S. M. (2014). Interpersonal psychotherapy versus treatment as usual for PTSD and depression among Sichuan earthquake survivors: A randomized clinical trial. *Conflict and Health, 8*(14), 1-10.
- Kaczmarek, M. & Zawadzki, B. (2012). Exposure to trauma, emotional reactivity, and its interaction as predictors of the intensity of PTSD symptoms in the aftermath of motor vehicle accidents. *Journal of Russian and East European Psychology, 50*(3), 47-64.
- Karl, A., Rabe, S., Zollner, T., Maercker, A., & Stopa, L. (2009). Negative self-appraisals in treatment-seeking survivors of motor vehicle accidents. *Journal of Anxiety Disorders, 23*, 775-781.
- Kim, S., Chung, Y., Kim, B. S., Lee, S. J., Yoon, J., & An, Y. (2012). Resting cerebral glucose metabolism and perfusion patterns in women with posttraumatic stress disorder related to sexual assault. *Psychiatry Research: Neuroimaging, 201*, 214-217.
- Kintzle, S., Schuyler, A. C., Ray-Letourneau, D., Ozuna, S. M., Munch, C., Xintarianos, E., & ...Castro, C. A. (2015). Sexual trauma in the military: Exploring PTSD and mental health utilization in female veterans. *Psychological Services, 12*, 394-401.
- Kupchik, M., Strous, R. D., Erez, R., Gonen, N., Weizman, A., & Spivak, B. (2007). Demographic and clinical characteristics of motor vehicle accident victims in the community general health outpatient clinic: A comparison of PTSD and non-PTSD subjects. *Depression and Anxiety, 24*, 244-250.
- Lopes, A. P., macedo, T. F., Coutinho, E. S. F., Figueira, I., & Ventura, P. R. (2014). Systematic review of the efficacy of cognitive-behavior therapy related treatments for victims of natural disasters: A worldwide problem. *PLoS ONE, 9*(10), 1-11.
- McDonagh, A., Griedman, M., McHugo, G., Ford, J., Sengupta, A., Mueser, K., ...& Descamps, M. (2005). Randomized trial of cognitive-behavioral therapy for chronic posttraumatic stress disorder in adult female survivors of childhood sexual abuse. *Journal of Consulting and Clinical Psychology, 73*(3), 515-524.
- McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine, 338*, 171-179.
- Meewisse, M., Reitsma, J. B., De Vries, G., Gersons, B. P. R., & Olf, M. (2007). Cortisol and post-traumatic stress disorder in adults. *British Journal of Psychiatry, 191*, 387-392.

- Morris, M. C., Compas, B. E., & Garber, J. (2012). Relations among posttraumatic stress disorder, comorbid major depression, and HPA function: A systematic review and meta-analysis. *Clinical Psychology Review, 32*(4), 301-315.
- Myers, C. E., VanMeenen, K. M., & Servatius, R. J. (2012). Behavioral inhibition and PTSD symptoms in veterans. *Psychiatry Research, 196*, 271-276.
- National Center for Statistics and Analysis. (2014). 2013 motor vehicle crashes: Overview. Washington, DC: National Highway Traffic Safety Administration.
- Neria, Y., Nandi, A., & Galea, S. (2007). Post-traumatic stress disorder following disasters: A systematic review. *Psychological Medicine, 1*-14.
- Nemeroff, C. B., Bremner, J. D., Foa, E. B., Mayber, H. S., North, C. S., & Stein, M. B. (2006). Posttraumatic stress disorder: A state-of-the-science review. *Journal of Psychiatry Research, 40*, 1-21.
- North, C. S. & Oliver, J. (2013). Analysis of the longitudinal course of PTSD in 716 survivors of 10 disasters. *Social Psychiatry and Psychiatric Epidemiology, 48*, 1189-1197.
- Olf, M., Langeland, W., Draijer, N., & Gersons, B. P. R. (2007). Gender differences in posttraumatic stress disorder. *Psychological Bulletin, 133*(2), 183-204.
- Rauch, S. A. M., Defever, E., Favorite, T., Duroe, A., Garrity, C., Martis, B., & Liberzon, I. (2009). Prolonged exposure for PTSD in Veterans Health Administration PTSD Clinic. *Journal of Traumatic Stress, 22*, 60-64.
- Rees, S., Silove, D., Chey, T., Ivancic, L., Steel, Z., Creamer, M., & ...Forbes, D. (2011). Lifetime prevalence of gender-based violence in women and the relationship with mental disorders and psychosocial function. *JAMA, 306*(5), 513-521.
- Resick, P. A. & Schnicke, M. K. (1992). Cognitive processing therapy for sexual assault victims. *Journal of Consulting and Clinical Psychology, 60*(5), 748-756.
- Richardson, L. K., Frueh, B. C., & Acierno, R. (2010). Prevalence estimates of combat-related post-traumatic stress disorder: Critical review. *Australian & New Zealand Journal of Psychiatry, 44*, 4-19.
- Roberts, A. L., Gilman, S. E., Breslau, J., Breslau, N., & Koenen, K. C. (2011). Race/ethnic differences in exposure to traumatic events, development of post-traumatic stress disorder, and treatment-seeking for post-traumatic stress disorder in the United States. *Psychological Medicine, 41*, 71-83.
- Rogers, M. A., Yamsue, H., Abe, O., Yamada, H., Ohtani, T., Iwanami, A...& Kasai, K. (2009). Smaller amygdala volume and reduced anterior cingulate gray matter

density associated with history of post-traumatic stress disorder. *Psychiatry Research: Neuroimaging*, 174, 210-216.

- Rosellini, A. J., Coffey, S. F., Tracy, M., Galea, S. (2014). A person-centered analysis of posttraumatic stress disorder symptoms following a natural disaster: Predictors of latent class membership. *Journal of Anxiety Disorders*, 28, 16-24.
- Rothbaum, B. O., Price, M., Jovanovic, T., Norrholm, S. D., Gerardi, M., Dunlop, B., ... & Ressler, K. J. (2014). A randomized, double-blind evaluation of D-cycloserine or alprazolam combined with virtual reality for posttraumatic stress disorder (PTSD) in Iraq and Afghanistan war veterans. *American Journal of Psychiatry*, 171(6), 640-648.
- Sapolsky, R. M., Romero, L. M., Munck, A. U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative action. *Endocrine Reviews*, 21, 5-89.
- Simmons, A. N., Paulus, M. P., Thorp, S. R., Matthews, S. C., Norman, S. B., & Stein, M. B. (2008). Functional activation and neural networks in women with posttraumatic stress disorder related to intimate partner violence. *Society of Biological Psychiatry*, 64, 681-690.
- Sloan, D. M., Marx, B. P., Bovin, M. J., Feinstein, B. A., & Gallagher, M. W. (2012). Written exposure as an intervention for PTSD: A randomized clinical trial with motor vehicle accident survivors. *Behaviour Research and Therapy*, 50, 627-635.
- Smith, E. R., Porter, K. E., Messina, M. G., Beyer, J. A., Defever, M. E., Foa, E. B., & Rauch, S. A. M. (2015). Prolonged exposure for PTSD in a Veteran group: A pilot effectiveness study. *Journal of Anxiety Disorders*, 30, 23-27.
- Sun, Y., Wang, Z., Ding, W., Wan, J., Zhuang, Z., Zhang, Y., ... & Xu, J. (2013). Alterations in white matter microstructure as vulnerability factors and acquired signs of traffic accident-induced PTSD. *PLoS ONE*, 8(12), 1-12.
- Tiet, Q. Q., Leyva, Y. E., Blau, K., Turchik, J. A., & Rosen, C. S. (2015). Military sexual assault, gender, and PTSD treatment outcomes of U.S. veterans. *Journal of Traumatic Stress*, 28, 92-101.
- Veer, I. M., Oei, N. Y. L., van Buchem, M. A., Spinhoven, P., Elzinga, B. M., & Rombouts, S. A. R. B. (2015). Evidence for smaller right amygdala volumes in posttraumatic stress disorder following childhood trauma. *Psychiatry Research: Neuroimaging*, 233, 436-442.
- Voelkel, E., Pukay-Martin, N. D., Walter, K. H., & Chard, K. M. (2015). Effectiveness of cognitive processing therapy for male and female U.S. veterans with and without military sexual trauma. *Journal of Traumatic Stress*, 28, 174-182.

- Walter, K. H., Buckley, A., Simpson, J. M., & Chard, K. M. (2014). Residential PTSD treatment for female veterans with military sexual trauma: Does a history of childhood sexual abuse influence outcome? *Journal of Interpersonal Violence, 29*(6), 971-986.
- Walters, M. L., Chen, J., & Breiding, M. J. (2013). National Intimate Partner and Sexual Violence Survey (NISVS): 2010 findings on victimization by sexual orientation. *National Center for Injury Prevention and Control of the Centers for Disease Control and Prevention, 1-48*.
- Woon, F. L., Sood, S., & Hedges, D. W. (2010). Hippocampal volume deficits associated with exposure to psychological trauma and posttraumatic stress disorder in adults: A meta-analysis. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 34*, 1181-1188.
- Xue, C., Ge, Y., Tang, B., Liu, Y., Kang, P., Wang, M., & Zhang, L. (2015). A meta-analysis of risk factors for combat-related PTSD among military personnel and veterans. *PLOS one, 10*, 1-21.
- Yasan, A., Guzel, A., Tamam, Y., & Ozkan, M. (2009). Predictive factors for acute stress disorder and posttraumatic stress disorder after motor vehicle accidents. *Psychopathology, 42*, 236-241.
- Zawadzki, B. & Popiel, A. (2012). Temperamental traits and severity of PTSD symptoms: Data from longitudinal studies of motor vehicle accident survivors. *Journal of Individual Differences, 33*(4), 257-267.
- Zhang, J., Tan, Q., Yin, H., Zhang, X., Huan, Y., Tang, L., ...& Li, L. (2011). Decreased gray matter volume in the left hippocampus and bilateral calcarine cortex in coal mine flood disaster survivors with recent onset PTSD. *Psychiatry Research: Neuroimaging, 192*, 84-90.