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Military Veteran Students Transition to Academic Life with PTSD, Trauma, and Potential for Freezing Response.

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MILITARY VETERAN STUDENTS TRANSITION TO ACADEMIC LIFE
WITH PTSD, TRAUMA, AND POTENTIAL FOR FREEZING RESPONSE

by

Alicia M. Erchul

A Thesis submitted to the Department of Psychology
in partial fulfillment of the requirements for the degree of

Master of Science in Psychological Science

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THESIS CERTIFICATE OF APPROVAL

The thesis of Alicia M. Erchul is approved:

Dr. Lori Lange
Department and Committee Chair

Date

Dr. Angela Mann

Date

Dedication

I dedicate this work to my beloved sons, Aidan and Gabriel. Aidan has walked the journey of traumatic loss and healing with me in the most courageous ways possible. Aidan has watched me struggle and work long hours to achieve this degree. His sweet spirit has filled my hours with laughter, joy, and fun.

Gabriel, my sweet baby, who will be forever four months old. You will always be loved, forever in our hearts, and never forgotten. Your legacy of love helped to save two lives who continue to share your love with their families.

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Table of Contents

THESIS CERTIFICATE OF APPROVAL.....	2
Dedication.....	3
Acknowledgements.....	4
Abstract	10
Introduction.....	11
Posttraumatic stress disorder (PTSD)	12
Individual Differences in the Development of PTSD.....	15
Neurobiology of PTSD	16
Neurotransmitters and PTSD.....	16
Developmental Differences in Response to Trauma.....	17
Environmental Factors	19
Gene X Environment Interactions.....	20
Traumatic Life Events and PTSD.....	22
Military Veterans and PTSD.....	23
PTSD and Stress Response System	28
Heart Rate Variability and PTSD.....	29
Fear Response.....	32
“Fight-Flight or Freeze” Avoidance and Tonic Immobility	33
Tonic Immobility and PTSD.....	35
Fear Inhibition	36
Hypotheses and Research Questions.....	37
Method	39
Participants	39
Procedure.....	40
Materials.....	41
Post-Traumatic Stress Disorder Checklist for Civilians	41
Brief Trauma Questionnaire	42
Perceived Stress Scale.....	42
Depression Patient Health Questionnaire	42

Social Connectedness Scale.....	42
UCLA Loneliness Scale.	43
Physical Health Questionnaire.....	43
MOS 36-Item Short Form Survey Instrument.....	43
Heart Rate Variability (HRV), Electrocardiogram (ECG).....	44
Stabilometer.....	44
Pictorial stimuli	45
Results.....	45
Sociodemographic, Trauma and Stress, Mental and Physical Health, and Social Constructs	46
Deployment Status	46
Gross Income.....	47
PTSD Diagnosis	47
VA Disability Rating	47
Bivariate Relationships.....	48
Hypotheses	49
Hypotheses 1-3	49
Hypotheses 4.....	51
Discussion.....	53
Hypotheses 1-3.....	54
Hypothesis 4.....	56
Limitations	57
Conclusion	58
Tables and Figures	59
Figure 1.....	59
Theoretical gene x environment model for PTSD symptomology effects on freezing and avoidance fear response.....	59
Table 1	60
Average PTSD, Trauma and Stress Constructs by Sociodemographic Variable.	60
Table 2.....	61
Average Mental Health Constructs by Sociodemographic Variables	61
Table 3.....	62
Average Physical Health Constructs by Sociodemographic Variables	62

Table 4.....	63
Average Social Constructs by Sociodemographic Variables	63
Table 5.....	64
Pearson Correlations on PTSD, Trauma, Stress, Psychological, Social and General Health Outcome Variables.....	64
Table 6.....	65
Pearson Correlations on PTSD Diagnosis, PCL Score, Trauma, and Heart Rate During Emotional Stimuli.....	65
Table 7.....	66
PTSD Symptomology Groups' Response to Emotional Stimuli via Anterior-Posterior Body Sway (cm).....	66
Table 8.....	66
PTSD Symptomology Groups' Response to Emotional Stimuli via Heart Rate (beats per minute)	66
Table 9.....	66
PTSD Symptomology Groups' (covaried with BTQ) Response to Emotional Stimuli via Anterior-Posterior Body Sway (cm).....	66
Table 10.....	66
PTSD Symptomology Groups' (covaried with BTQ) Response to Emotional Stimuli via Heart Rate (beats per minute).....	66
Figure 2.....	67
PTSD Symptomology Groups' Response to Emotional Stimuli via Anterior-Posterior Body Sway	67
Figure 3.....	67
PTSD Symptomology Groups' Response to Emotional Stimuli via Heart Rate (beats per minute)	67
Figure 4.....	68
PTSD Symptomology Groups' (covaried with BTQ) Response to Emotional Stimuli via Anterior-Posterior Body Sway (cm).....	68
Figure 5.....	68
PTSD Symptomology Groups' (covaried with BTQ) Response to Emotional Stimuli via Heart Rate (beats per minute).....	68
Table 11.....	69
BTQ Groups' Response to Emotional Stimuli via Anterior-Posterior Body Sway (cm)	69

Table 12.....	69
BTQ Groups' Response to Emotional Stimuli via Heart Rate (beats per minute)	69
Figure 6.....	70
BTQ Groups' Response to Emotional Stimuli via Anterior-Posterior Body Sway (cm)	70
Figure 7.....	70
BTQ Groups' Response to Emotional Stimuli via Heart Rate (beats per minute)	70
Table 13.....	71
PTSD Symptomology Groups' Response split by BTQ Groups to Emotional Stimuli via Anterior-Posterior Body Sway (cm).....	71
Figure 8.....	72
Low PTSD Symptomology Group's Response Split by BTQ Groups to Emotional Stimuli via Anterior-Posterior Body Sway (cm).....	72
Figure 9.....	72
Moderate PTSD Symptomology Group's Response Split by BTQ Groups to Emotional Stimuli via Anterior-Posterior Body Sway (cm)	72
Figure 10	73
Severe PTSD Symptomology Group's Response Split by BTQ Groups to Emotional Stimuli via Anterior-Posterior Body Sway (cm).....	73
Table 14.....	74
PTSD Symptomology Groups' Response split by BTQ Groups to Emotional Stimuli via Heart Rate (beats per minute).....	74
Figure 11	75
Low PTSD Symptomology Group's Response Split by BTQ Groups to Emotional Stimuli via Heart Rate (beats per minute).....	75
Figure 12	75
Moderate PTSD Symptomology Group's Response Split by BTQ Groups to Emotional Stimuli via Heart Rate (beats per minute).....	75
Figure 13	76
Severe PTSD Symptomology Group's Response Split by BTQ Groups to Emotional Stimuli via Heart Rate (beats per minute).....	76
Figure 14	77
Pictorial Representation of Body Sway, Freezing, and Avoidance Responses to Pleasant and Unpleasant Stimuli by Moderate and Severe PTSD symptomology Groups	77
Figure 15	78

Graphic Representation of Body Sway, Freezing, and Avoidance Responses to Pleasant and Unpleasant Stimuli by Moderate and Severe PTSD symptomology Groups	78
Table 15.....	79
Occurrence of Trauma (BTQ) for Participants	79
References	80
Curriculum Vitae.....	100

Abstract

The current study is the first known research to investigate the association of the freezing response with PTSD and traumatic stress in the military veteran student population. Current understanding of the freezing response are primarily based in comparative psychology, with some studies extending to human participants (Azevedo et al., 2005; Facchinetti et al., 2006; Hagedaars et al., 2012; Volchan et al., 2017). Models generally agree that the freezing response consists of reduced body sway with decreased heart rate (Porges, 2003, 2007; Hagedaars et al., 2014).

Thirty-eight military veteran students (n=38; 18 female; 20 male) with ages ranged from 19 to 49 participated in the study. Participants completed self-report assessments administered through Qualtrics, which include the PTSD Checklist (PCL-5; Weathers et al., 2013), Brief Trauma Questionnaire (BTQ; Schnurr et al., 1999), and additional demographic, health, and military questions. To objectively measure the freezing response, participants stood on a stabilometric platform (Tekscan Inc., South Boston, MA) with a heart rate variability (HRV) monitor attached. Participants then completed four 60-second trials. The first trial was a baseline with eyes open, followed by three trials of randomized emotional stimuli of neutral, pleasant, and unpleasant images from the International Affective Picture System (IAPS; Lang et al., 1997).

Results reveal evidence of a freezing response that includes reduced body sway and bradycardia when veteran students are presented with imminent threat in the form of unpleasant stimulus. Participants with severe PTSD symptomology and a history of traumatic events measured a freezing response across all emotional stimuli except the unpleasant stimulus where they had an avoidant response. This may indicate the cumulative effect of traumatic life events on the defense system. This study contributes to the body of knowledge on the freezing response in humans that indicates an increased risk for the development of PTSD and increased severity of symptoms.

Keywords: PTSD, traumatic life events, military veterans, university students, stress response, fear response, freezing, avoidance, tonic immobility, body sway, heart rate, posturography, stabilometer, stress, trauma

Introduction

Military veterans with combat exposure have experienced a tripling of the rate of posttraumatic stress disorder (PTSD) relative to the general population. However, the rate of veterans with PTSD varies across campaigns ranging from 11-20% for Operations Iraqi Freedom (OIF) and Enduring Freedom (OEF) and 15-30% for the Vietnam War based on lifetime prevalence of PTSD for the Vietnam veterans (Gradus, 2017; Hoge et al., 2004; Smith et al., 2008; True et al., 1993). Even though a higher percentage of veterans have experienced traumatic combat, only 11-30% of veterans experience PTSD, revealing high variability in the impacts of exposure to trauma and combat across the lifespan (Breslau, 2001; Kessler et al., 1995). Veterans who have endured longer combat or higher combat intensity have an increased risk for PTSD (Goldberg et al., 1990; Yehuda et al., 2010). Veterans develop PTSD in varying degrees of severity with different treatment outcomes (Goldberg et al., 1990; Yehuda et al., 2010). The same level of combat does not always affect individuals in the same way. How and why do individuals react differently to the same trauma? Why do some individuals freeze, fight, or take flight in the face of threat?

PTSD is the only disorder that requires exposure to a traumatic environmental trigger as a prerequisite for diagnosis by the Diagnostic and Statistical Manual of Mental Disorders (DSM-V). Individual differences in the severity of PTSD symptoms may be the result of gene-environment (G X E) interactions, where traumatic experiences in childhood and throughout the lifespan create dysregulation of neurobiological pathways (Delahanty, 2008). Additional genetic factors include the biological response to stress, cognitive abilities, personality, and other autonomic physiological factors (Koenen et al., 2003; Plomin et al., 2001; Stein et al., 2002; True et al., 1993). This study expands the understanding of individual differences in how people respond to threatening situations, differing levels of PTSD, and the variations in the freezing or flight response to passive threats as measured by body sway. Individual differences will be explored in regards to the heart rate variability (Porges, 1995), physiological transformations (Streeter et al., 2012), tonic immobility, behavioral neuroscience, social-behavioral outcomes, and developmental theories. Additionally, bottom-up

methodologies that discuss how the body changes in regards to psychophysiological changes and increased self-regulation will also be discussed (Ogden, Pain and Fischer, 2006; Van der Kolk, 2006).

Posttraumatic stress disorder (PTSD)

PTSD is the only disorder within the American Psychiatric Association's (2013) *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5) that requires a traumatic event as a requirement for diagnosis (DSM-5; American Psychological Association, 2013). A major change to the diagnosis of PTSD has been a shift of the disorder away from being listed under anxiety disorders to a new diagnostic category based on "Trauma and Stressor-related Disorders." This shift away from anxiety disorders recognizes the effects of trauma and stress on the brain and the body. PTSD involves many emotions (guilt, shame, or anger) and symptoms that do not align with anxiety or depression disorders (DSM-5; American Psychological Association, 2013). The diagnosis for PTSD requires the following that a person be exposed to a life stressor and experience symptoms from the following criteria for longer than a month that affects their daily life in a negative way.

A life stressor qualifies as a traumatic event if a person feels as though their life was in danger of serious injury, major illness, or death regardless of whether the person experienced the event directly or if they were a witness, indirectly exposed or learned about their family member or friend experiencing the trauma (DSM-5; American Psychological Association, 2013). Another criterion regards symptoms that surround intrusion of unwanted and upsetting dreams, flashbacks, memories, night terrors, emotional distress, or physical reactivity after exposure to traumatic reminders (DSM-5; American Psychological Association, 2013). Symptoms that satisfy the avoidance criteria include avoidance of trauma-related stimuli that induce feelings, thoughts, or reminders of trauma (DSM-5; American Psychological Association, 2013).

In order to qualify as having met the criteria for negative alterations in cognitions and mood, the individual must at least two of the following symptoms: exaggerated blame of self or others for trauma, overly negative thoughts about oneself or the world inability to recall key features of trauma, increased negative affect, decreased interest in activities, feeling isolated, and limited ability to experience positive affect (DSM-

5; American Psychological Association, 2013). The last criterion is that the individual experiences alterations in arousal and reactivity that began or worsened after trauma and includes irritability, aggression, risky behavior, hypervigilance, heightened startle reaction, difficulty sleeping, and difficulty concentrating. PTSD is a complex disorder that impacts so many areas of an individual's life. If only one criterion is met, the individual may be suffering from major depression or anxiety depending on the symptoms (DSM-5; American Psychological Association, 2013). Often PTSD is co-morbid with anxiety and major depression due to the overlapping nature of the symptoms (DSM-5; American Psychological Association, 2013).

Stressful life events often have an additive effect resulting in increased vulnerability to psychopathology and stress reactivity (Ozer et al., 2008). Individuals may be at an increased risk to develop PTSD if they have experienced the following: repeated traumatic events, life-threatening illness, child abuse, psychopathology, dissociation, and lack of social support (Ozer et al., 2008). The greatest risk factor is peritraumatic dissociation due to the numerous memory impairments that change the perception of the traumatic experience resulting in misperceptions and decreased awareness of the true impacts of the trauma (Ozer et al., 2008).

Typically, to qualify as PTSD, one has to feel as though one feared their life was in danger or they witnessed someone close to them die or experience severe trauma and they have no control over that situation (Bisson et al., 2007; Ehring et al., 2014). Traumatic events are a common experience with 50-70% of adults in the United States experiencing a traumatic event during their lifetime however only 5-12% of the population will develop posttraumatic stress disorder (PTSD) (Kessler et al., 1995). Military veterans with combat exposure have experienced a threefold increase in the incidence of PTSD (Smith et al., 2008; Hoge et al., 2004). Due to the nature of combat and isolation after returning to civilian life, military combat veterans experience PTSD at a higher rate than the general population.

Military veterans who experienced greater social isolation upon returning to civilian life have a higher lifetime prevalence rate of PTSD than other combat veterans. Vietnam Veterans experience the highest rate of PTSD at 30.9%, versus 12.1% for veterans of the Gulf War (Gradus, 2017). Additionally, traumatic life

events may prime veterans to be more vulnerable and at greater risk of developing PTSD (Hertsgaard et al., 1995; Nachmias et al., 1996; Willemsen–Swinkels et al., 2000).

Research findings reflect that childhood trauma impairs brain development with impacts related to emotion regulation and effective interpersonal behaviors (Shipman et al., 2000; Shipman et al., 2005). Developmental changes to the brain from child abuse often do not show up until adulthood, where decreases in hippocampal volumes and frontal-limbic system structures reveal the long lasting impacts of child abuse (Van der Kolk, 2006). An individual's original stress reactivity determines the impact that traumatic events have on the brain and individual differences vary. Chronic stress and acute trauma affect several areas of the brain, including the amygdala, hippocampus, and medial prefrontal cortex (Bremner, 2006).

Individual diagnosis and treatment of PTSD may often be delayed. Many individuals will experience symptoms of PTSD within three months of a traumatic incident, but PTSD may not develop for several years or the individual may not recognize their behavior as relating to PTSD. PTSD symptoms range in severity with impacts upon daily life or around anniversaries of the trauma.

PTSD symptoms and impacts to brain functions are as wide and varied as trauma that the survivor experienced. Chronic PTSD affects daily life with negative feelings including isolation, shame, and despair, unrealistic beliefs about their trauma, survivor guilt, trouble concentrating, addiction, difficulties sleeping due to flashbacks and nightmares (Aupperle et al., 2012; Ford & Kidd, 1998; Miller et al, 2014). The most common areas of the brain that are impacted are hippocampal volume, overactive amygdala, and prefrontal cortex impairments. With so many areas of the brain impaired by trauma, effective treatment is often hard to achieve.

Successful treatment that includes training of the executive functioning, mindfulness meditation, memory, and emotional recognition in addition to drug protocols may increase neurogenesis of impaired brain structures (Bremner, 2006). When PTSD presents with symptoms related to both serotonin and norepinephrine, selective serotonin reuptake inhibitor (SSRI) have proven effective in decreasing symptoms related to fear response, intrusive memories and hypervigilance (Davis, et al., 1997; Vermetten & Bremner,

2002). Research continues to find so many differences in how individuals respond to stress and trauma. Future research should focus on finding ways to maximize treatment by implementing a multi-measure approach that includes self-report, physiological markers, fMRI for brain functioning and genotyping to produce better treatment results based on the individual's stress reactivity.

Individual Differences in the Development of PTSD

Individual differences in the severity of PTSD symptoms may also be the result of gene by environment (GXE) interactions that create dysregulation of neurobiological pathways (Delahanty, 2011). The genetic influences for PTSD vulnerability and the genetic changes that occur after trauma that results in PTSD symptoms are incredibly similar (Stein et al., 2002). Genetic and epigenetic factors, particularly reactivity to environmental stimuli may explain approximately 30-70% of these individual differences found in phenotypic stress responses and the development of PTSD (Afifi et al., 2010; Boyce & Ellis, 2005; Matthews, 1986; Pitman et al., 2012).

Recent neuroimaging studies on PTSD development reveal that individual differences in brain structures, function, genetics, epigenetics, and overall brain function reveal several genes and phenotypes that have been associated with increased vulnerability to stress, trauma and the development of PTSD (Lebois et al., 2016; Stark et al., 2015; Yehuda et al., 2005). The genes and phenotypes impact the develop of the physiological stress response, memory, executive function, emotion regulation, fear inhibition, threat detection and pain perception within the brain resulting in dysregulation and vulnerability in the individual's response to stress and trauma (Lebois et al., 2016). Even when trauma does not lead to PTSD, there are significant and long lasting effects on brain activity and function resulting from GXE interactions (Stark et al., 2015). Family, twin, and adoption studies have revealed that there are genetic factors with over 30% of PTSD cases connected to the heritability of the genetic vulnerability to PTSD that is independent of the effects of combat exposure (Koenen et al., 2008; Merikangas & Swendsen, 1997; Stein et al., 2002; True et al., 1993). Family history provides an understanding of both the heritability of PTSD vulnerability and vicarious exposure to trauma due to family members living with PTSD. Parents with PTSD have an increased risk of

transmitting trauma-related genetic changes that increase the PTSD vulnerability in their children (Chemtob et al., 2010; Leen-Feldner et al., 2013; Yehuda et al., 2005).

Neurobiology of PTSD

Individuals with PTSD have smaller hippocampal and anterior cingulate areas, impairments to medial prefrontal and anterior cingulate functions and overactive amygdala functioning (Bremner, 2006). Additional changes that occur in the neurochemical systems, brain structure, and functions may determine the severity of PTSD symptoms (Bremner, 2006). Neuroimaging of individuals with PTSD and those without reveal significant differences in the prefrontal cortex, temporal, basal ganglia, amygdala, anterior cingulate cortex, thalamus and the sensorimotor cortex that support the pathology of PTSD in regards to memory, emotion and fear regulation (Lebois et al., 2016; Mahan & Ressler, 2012; Stark et al., 2015). Decreased gray matter density in the prefrontal cortex leads to deficits in emotion regulation (Lebois et al., 2016). Impacts to the hippocampus result in deficits in both explicit and contextual memories related to traumatic experiences (Mahan & Ressler, 2012). The hippocampus mediates the extinction of conditioned fear; stress and trauma decrease hippocampal volume and impairs fear extinction particularly to every day stimuli (Brocke et al., 2006; Mahan & Ressler, 2012).

Neurotransmitters and PTSD

Differences in the brain's interactions with neurotransmitters results in the varying degrees of symptom severity and the risk factors that surround the development of PTSD. Two of the main neurotransmitters connected to PTSD are serotonin and norepinephrine (Davis et al., 1997). These neurotransmitters correspond to the brain areas involved in the body's response to stress, including subcortical structures (amygdala, hippocampus, and hypothalamus) and cortical regions (anterior cingulate, insula, orbitofrontal, and prefrontal cortices) receive these transmissions (Lebois et al., 2016; Mahan & Ressler, 2012; Newport & Nemeroff, 2000; Rauch et al., 2006, Stark et al., 2015). Trauma results in both over activation and dysregulation of the amygdala, which results in dysfunction in fear processing and fear

inhibition impairment (Brocke et al., 2006). The overactive amygdala points to dysregulation in brain serotonin (5-HT) systems and serotonin levels (Davis et al., 1997).

The serotonin neurotransmitter assists with numerous functions related to PTSD symptomology, including aggression, fear response, appetite, alcoholism, sleep disturbances, heart rate variability, impulsivity, depression, anxiety, and suicidality (Davis et al.; Southwick et al., 1999; Vermetten & Bremner, 2002). Dysregulation of the serotonergic system, particularly the polymorphic region (5-HTTLPR), terminates the production of serotonin (5-HT) in the brain and has been observed in participants with PTSD, anxiety and MDD (Sangkuhl et al., 2009; Southwick et al., 1999; van Praag, 2004). This dysregulation of the serotonergic system results in the overreaction to both threatening and safe stimuli, how trauma memories are processed and the coordination of fear-based behaviors (Murrrough et al., 2011).

Additionally, controlling for combat exposure, age, race and sex, the 5-HTTLPR alleles have been connected to the development of PTSD, symptom severity, treatment outcomes, and quality of life for OEF/OIF veterans with individual differences reported for the S' allele and the L' allele (Grabe et al., 2009; Gressier et al., 2013; Kimbrel et al., 2014 & Wang et al., 2011). The S' allele impacts the amygdala resulting in both over activation and dysregulation of the amygdala response which results in dysfunction in fear processing and fear inhibition impairment while the L' allele does not impair the amygdala (Brocke et al., 2006). The impairment caused by the S' allele explains some of the variation for the severity of PTSD symptoms and FPS response for some of the veterans with PTSD.

Developmental Differences in Response to Trauma

The probabilistic epigenesis (PE) model allows for differences in developmental outcomes by assuming that the same genotype can lead to different behavioral and neural outcomes based on different life experiences (Gottlieb & Halpern, 2002). Family, twin, and adoption studies have all revealed genetic and environmental factors that result in the development of PTSD (Koenen et al., 2008; Merikangas & Swendsen, 1997). Different genetic expressions may be due to variances in biological sensitivity to context (BSC) which can result in highly reactive phenotypes that create maladaptive responses to stress and trauma (Boyce &

Ellis, 2005). For example, child abuse and trauma at an early age results in negative changes to the stress-responsive biological systems resulting in high reactivity to stress (Hertsgaard et al., 1995; Meyer et al., 2001; Nachmias et al., 1996; Willemsen–Swinkels et al., 2000). This high reactivity to stress may then result in PTSD, MDD or other disorders in the face of additional trauma or combat.

Genetic factors may provide protection or vulnerabilities in the face of stress and trauma. These genetic factors include executive functioning, cognitive abilities, personality, and other autonomic physiological factors (Koenen et al., 2003; Plomin et al., 2001; Stein et al., 2002; True et al., 1993). The genetic influences for PTSD vulnerability and the genetic changes that occur after trauma that results in PTSD symptoms are incredibly similar (Stein et al., 2002). Twin and family studies have revealed that there are genetic factors and an increased genetic vulnerability to PTSD that is independent of the effects of combat exposure. Over 30% of PTSD cases are connected to the heritability of the genetic vulnerability (Stein et al., 2002; True et al., 1993).

Studies on family history and heritability of reactivity phenotypes stress the importance of GXE interactions that result in PTSD (Bartels et al., 2003; Busjahn et al., 1999; Merikangas & Swendsen, 1997). Phenotypes associated with MDD have also been found in individuals with PTSD, with common genetic liability related to MDD at 15% of total variation and 58% related to the genetic variation in PTSD (Koenin et al., 2008). This genetic overlap of phenotypes demonstrates the epigenetic modifications that occur within the brain due to environmental factors of neglect, child abuse, trauma and combat which result in several systems being impaired.

There are many gene expression profiles, additional alleles, and phenotypes associated with PTSD; this experiment focuses on the phenotypes and alleles connected to the impaired FPS as it distinguishes PTSD from MDD. Genetic factors are only part of the equation for understanding PTSD development. Environmental factors and their interactions with the above-mentioned genetic factors must be considered to fully understand PTSD development.

Stressful life events correspond with changes in the 5-HTTLPR genotype (Duman & Canli, 2015). In studies that controlled for combat exposure, age, race and sex, the 5-HTTLPR alleles are correlated with PTSD development, symptom severity, treatment outcomes, and quality of life for OEF/OIF veterans with differences between those with the S' allele and the L' allele (Grabe et al., 2009; Gressier et al., 2013; Kimbrel et al., 2014; Wang et al., 2011). The S' allele impacts the amygdala resulting in both over activation and dysregulation of the amygdala response which results in dysfunction in fear processing and fear inhibition impairment while the L' allele does not impair the amygdala (Brocke et al., 2006).

Social support and environment influence the effect of 5-HTTLPR genotype on PTSD vulnerability, particularly for individuals with the S' allele (Bronfenbrenner, 1994; Bronfenbrenner & Morris, 2007; Jovanovic & Ressler, 2010). The S' allele is associated with a lower risk of PTSD when the individual is in a low-risk environment with high social support and a higher risk for PTSD when in a high-risk environment with low social support confirming that the social environment moderates the effect of 5-HTTLPR genotype on PTSD risk (Armbruster et al., 2009; Koenin et al., 2008). Individual differences in stress reactivity in response to trauma leads to several different disorders and changes to the brain. Genetic factors, early environmental factors, and epigenetic modifications influence stress reactivity either directly or in interaction with each other. While genetic markers may exist, PTSD is not purely biological in nature.

Environmental Factors

Interindividual differences within the microsystem (family, university, work, military, veteran services), mesosystem (interactions between the veteran and the microsystem and the exosystem), and the exosystem (government, military branches, politics, and veteran affairs) may affect the development and severity of PTSD (Bronfenbrenner, 1979; Bronfenbrenner & Morris, 2006). Assessments on family history, trauma type, timing, intensity, and duration of the trauma must be included in any study to understand why some veterans are more at risk to develop PTSD. For example, veterans who have endured longer combat or higher combat intensity have an increased risk for PTSD (Goldberg et al., 1990; Yehuda et al., 2010).

Family history provides an understanding of both the heritability of PTSD vulnerability and vicarious exposure to trauma due to family members living with PTSD. Parents with PTSD have increased risk of transmitting trauma-related genetic changes that increase the PTSD vulnerability in their children (Chemtob et al., 2010; Leen-Feldner et al., 2013; Yehuda et al., 2005). Children who had parents who were veterans with PTSD have higher rates of exposure to domestic violence and stress from caring for a parent with PTSD leading to greater risk of developing PTSD as an adult (Margolin & Vickerman, 2007; Taft et al., 2005). Environmental factors surrounding childhood maltreatment and other trauma significantly increase an individual's vulnerability to develop PTSD later in life (Duman & Canli, 2015).

Individual differences in stress response are due to the high variability of biological changes that accompany stressful life events (Boyce & Ellis, 2005). Early developmental trauma may predispose some individuals to high stress reactivity as dysregulation impaired the stress response system. Stressful life events often have an additive effect resulting in increased vulnerability to psychopathology and stress reactivity (Hertsgaard et al., 1995; Nachmias et al., 1996; Willemsen–Swinkels et al., 2000). Individuals with a trauma history may experience one traumatic event to several traumatic events including child abuse, emotional abuse, neglect, absent parents and other life stressors (Kessler, 2000). PTSD increases in complexity when individuals have experienced multiple traumas throughout their lifespan (Cloitre et al., 2009). Complex PTSD occurs when an individual is exposed to sustained, repeated or multiple traumas, with greater impact given to childhood trauma due to the persistent impacts on overall development.

Gene X Environment Interactions

PTSD is the result of genetic factors interacting with environmental factors. While an individual may have a genetic vulnerability to develop PTSD, without trauma or combat, that individual would never develop PTSD. Overlapping comorbidities and symptoms between MDD, anxiety, and PTSD can occur with trauma, making diagnosis of PTSD difficult. Our theoretical model suggests that the relationship between genetic factors and environmental factors is bidirectional in nature, such that early childhood trauma and other traumatic events could influence stress reactivity and increase vulnerability in the development of PTSD.

Individual differences in the severity of PTSD symptoms may also be the result of gene by environment (GXE) interactions that create dysregulation of neurobiological pathways (Delahanty, 2008). The complex phenotype of PTSD emerges from bidirectional interactions of multiple genetic, behavioral, and environmental factors that influence changes at a genetic level (Zannas et al., 2015). GXE interactions change the expression of the reactive phenotypes resulting in anxiety, MDD, or PTSD (Alkon et al., 2003; Allen & Matthews, 1997). Individual differences in reactivity to environmental stimuli may explain some of the variance found in phenotypic stress responses to trauma (Boyce & Ellis, 2005; Matthews, 1996). The complexity of psychiatric traits makes it difficult to claim that an individual gene would have the same impact as an interaction between many genes and the environment (Newton-Cheh & Hirschhorn, 2005).

Epigenetic studies in PTSD reveal that trauma results in genetic changes and these changes may be allele-specific based on the GXE interactions and the bidirectional nature of those interactions (Zannas et al., 2015). These GXE interactions affect how an individual responds to stress, neurotransmitter function, immune regulation, phenotypes, and endophenotypes that increase vulnerability or resilience after trauma. An individual's original stress reactivity determines the impact of traumatic events have on the brain and individual differences vary by the length of deployment, type of combat, intensity of combat and additional traumatic events (Boyce & Ellis, 2005). Each traumatic event further modifies the brain's response to stress and fear. Stressful life events correspond with changes in the 5-HTTLPR genotype (Duman & Canli, 2015).

PE and GXE interactions related to stress reactivity may be able to answer the how and why behind individual differences in the development of PTSD (Boyce & Ellis, 2005; Gottlieb, 2007). These changes occur in all individuals, as stressful life events may be normative or non-normative. Childhood maltreatment, particularly when it occurs in the first 5 years of life, has lasting effects on stress response and dysregulation that lasts into adulthood (Duman & Canli, 2015). Childhood maltreatment influences the development of the S' allele which has been associated with more severe PTSD symptoms and MDD (Caspi et al., 2003). Additionally, social support and environment influence the effect of 5-HTTLPR genotype on PTSD vulnerability, particularly for individuals with the S' allele (Bronfenbrenner, 1994; Bronfenbrenner & Morris,

2007; Jovanovic & Ressler, 2010). The S' allele is associated with a lower risk of PTSD when the individual is in a low-risk environment with high social support and a higher risk for PTSD when in a high-risk environment with low social support confirming that the social environment moderates the effect of 5-HTTLPR genotype on PTSD risk (Armbruster et al., 2009; Koenin et al., 2008; Koenin et al., 2009). The variation in self-regulation and dysregulation for veterans stem from the dynamic interplay of genes and the early environment, which consist of parental relationships, prior experiences, and interpretation of childhood events (Sameroff, 2010). All of these GXE interactions affect fear inhibition resulting in its dysregulation and impairment for veterans with PTSD.

Traumatic Life Events and PTSD

Traumatic life events, particularly in childhood are connected to physiological changes that influence development of the mind, body, and spirit. These changes affect emotion regulation, fear response with increased freezing responses. Additionally, traumatic life events predispose individuals to have increased vulnerabilities to develop PTSD or other mental illnesses after trauma and combat. At least 80% of military veterans have experienced at least one traumatic life event and at least 40% of military veterans have experienced four or more traumatic life events during their childhood and adolescence (Brodskey & Stanley, 2008; Bryan et al., 2015; Carroll et al., 2017). Numerous traumatic life events increase these risk factors and lessens the likelihood that veterans will seek treatment for PTSD due to variations in PTSD symptomology and the increased likelihood of late-onset PTSD symptoms (Andersen et al., 2014). Studies of civilian and military populations indicate strong correlations between adult suicide and physical abuse, neglect and other traumatic events that occur during childhood (Carroll et al., 2017).

Multiple animal studies report that rats exposed to multiple stressors have impaired fear response with increases in freezing response and contextual fear conditioning (Imanaka et al., 2006). Additionally, freezing behavior was found in rats that were separated from their mother and nest during the first weeks of life with the freezing behavior continuing into adulthood (Sanders & Knoepfler, 2008). Animal studies have found that there is a compounding effect of multiple traumas than a singular incident. Rats that experienced

multiple traumatic events exhibited increased fear learning, however, rats that only had one traumatic experience did not (Rau & Fanselow, 2009).

Childhood trauma leads to continual dysregulation of the hypothalamic-pituitary-adrenal axis stress response, with the stress response system being chronically reactive or nonresponsive to stimuli (Frodl & O'Keane, 2013; McCrory et al., 2011; McLaughlin et al., 2014). Human studies have found that early traumatic life events disrupt the automatic stress response by maintaining elevated levels of epinephrine and cortisol, which fail to return to normal levels after trauma (Elzinga et al., 2008). Traumatic life events in childhood significantly correlate with cognitive impairments, social-emotional delays, developmental delays, learning, executive memory, and attentional disorders (Frodl & O'Keane, 2013; McCrory et al., 2011; McLaughlin et al., 2014).

Even for humans, single vs. multiple traumatic experiences have different impacts on physiological systems. Individuals who have experienced multiple traumas displayed higher levels of dissociation and shame while also display increased social dependency (Allen & Lauterbach, 2007; Hageaars et al., 2011). In comparison to individuals who only experienced one traumatic event, those that experienced multiple traumatic life events have a decreased ability to cope or respond to current life stressors or threats (Boscarino & Adams, 2009).

Military Veterans and PTSD

Traumatic life events and combat exposure result in different PTSD symptomatology. Childhood trauma and combat need to be examined together to determine risk factors, symptom severity, and other disorders. Military veterans often seek out military service as a way to survive in a structured environment after growing up in chaos and neglect with military veterans experiencing far more traumatic life events than their non-military peers (Afifi et al., 2016; Blosnich et al., 2014). Furthermore, there is a positive correlation between the number of traumatic life events and the severity of motor dysfunctions in conversion disorder (Roelofs et al., 2002). Chronic abuse or continual stress desensitizes the neuroendocrine stress response system, which may lead to enhanced fear responses, particularly the freezing response (Santa Ana et al., 2006).

While traumatic life events are not positive, military veterans may carry both benefits and disadvantages from childhood trauma. While resilience may be seen as a strength or benefit of surviving traumatic life events, there are psychophysiological changes that occur in the brain development of individuals who have experienced traumatic life events. These neurological changes affect how individuals respond to treatment for PTSD and the severity of symptoms (Sachs-Ericsson et al., 2009). Pre or post screening of military veterans for traumatic life events would assist in treatment efforts as the severity of symptoms and onset of PTSD impacts how individuals respond to medicine and therapy.

Previous research regarding military populations focus on older populations of veterans who have served in prior wars and not currently active duty or who have recent combat service Bound & Turner, 2002; Cohen & Wills, 1985; DiRamio et al., 2008; Rumann & Hamrick, 2010). One population that requires further study due to the uniqueness of their profession is military-connected students. The reintegration of military into civilian life presents a complex array of stressors on the mind, body, and spirit of soldiers making the transition to academic life (Goldberg et al., 1990; Green et al., 1990; Yehuda et al., 2010). There have been an increased number of military personnel transitioning to academic life due to the GI Bill across the country representing a unique and easily tapped resource on the university campus (Bound & Turner, 2002; Cohen & Wills, 1985; DiRamio et al., 2008; Rumann & Hamrick, 2010).

Military-connected students face significant challenges that set them apart from the regular student population. These veteran students may have undiagnosed PTSD, anxiety, depression and social isolation that present barriers to living within the academic community due to their traumatic combat exposure (DiRamio et al., 2008) Veteran students have strained social interactions with family, friends, and classmates that lead them to delay seeking help for PTSD, feel further isolated, and excluded from every aspect of civilian life (Hoge et al., 2004; O'Herrin, 2011; Rumann & Hamrick, 2009). Other differences result from maturity levels, role inconsistencies, social relationships, disabilities and health issues (DiRamio et al., 2008; O'Herrin, 2011; Rumann & Hamrick, 2009). In addition, military-connected students face several transitional phases that create increased stress throughout their lives. These life phases "Moving In, Moving Through,

and Moving Out” represent a transitional phase that correspond to life events that military-connected students face that reflect the decision to join the military, continues throughout their military appointment and transition back to civilian life (DiRamio et al., 2008; Schlossberg et al., 1989). While each phase represents a choice that the individual has made, they also present stressful transition periods. The “Moving In” phase describes the transition from civilian life to military life where the individual separates from their family and friend circle and joins a new unit. During the “Moving Through” phase, the individual is often presented with many returns to the “Moving In” phase as they transition from boot camp to various deployments (DiRamio et al., 2008). Each new assignment results in forming new bonds and relationships for support and stressful transitions. The compounding stressors of the “Moving Out” phase include leaving behind the comradery of their unit, life experiences that family, fellow students, and faculty have not, changes to their family with possibility of divorce upon return from deployment, and mental health disorders from traumatic combat experiences (DiRamio et al., 2008; O’Herrin, 2011; Rumann & Hamrick, 2009). The new civilian phase of “Moving In” to academic life presents further stressors on an individual who has already faced many traumatic events. The “Moving In” phase for a veteran to transition to academic life, presents new challenges with less support, PTSD multiplying the impacts of social isolation and feelings of loneliness, and physical disabilities that also further separate veterans from traditional college students (DiRamio et al., 2008; O’Herrin, 2011; Rumann & Hamrick, 2009). In an attempt to help military-connected students acclimate to campus life, many universities have military student resource centers and other training programs provided to faculty and staff to help these students be successful (O’Herrin, 2011; Rumann & Hamrick, 2009).

Military-connected students may face greater transitional periods if they have served on several deployments that result in greater transitions between home life and combat (DiRamio et al., 2008). Military-connected students who have been deployed face great interpersonal difficulties that can be quite isolating (DiRamio et al., 2008). Many veterans experience emotional, physical, and behavioral changes due to their

deployment. The stress caused by the transition to civilian life results in loss of sleep, frequent unwanted memories and aggressiveness or irritability (NCPTSD, 2010).

Stress exacerbates underlying trauma from combat that includes ambushes, attacks and enemy fire (NCPTSD, 2010). Combat results in serious injuries or witnessing nearby death or injury of another from bombing, blasts, or random improvised explosive devices (IED) all of which cause life-threatening trauma to the mind. Additionally, survival often depends on how the veteran responds to their deployment, the impacts of estrangement from family, concerns about job security after deployment, and the hostile combat environment. This trauma remains long after the danger passes and continues to cause damage to the mental states of those who returned home.

Trauma affects every aspect of life: physical, mental, emotional, and social (DMS-V). Whether at home, work, or university, social relationships are challenged by the impacts of trauma that result in social isolation. Military veterans are often not the same age as other university students and may be the same age as their professors (DiRamio et al., 2008). These individual differences sometimes result in strained interactions with faculty and fellow students that result in greater stress and further social isolation for the military-connected student (DiRamio et al., 2008; O'Herrin, 2011; Rumann & Hamrick, 2009). Research that focuses on the stress caused by this transition helps to identify ways to assist military-connected students for a successful transition to academic life (DiRamio et al., 2008; O'Herrin, 2011; Rumann & Hamrick, 2009). Due to the extensive military training for combat that involves not reacting to unfamiliar and unsafe surroundings, veterans often do not seek help or acknowledge when stress has become unhealthy (DiRamio et al., 2008; O'Herrin, 2011; Rumann & Hamrick, 2009). Many military-connected students do not display that they are experiencing challenges in their transition due to the belief that revealing stress or asking for help is a sign of weakness and they are unfit to serve (DiRamio et al., 2008; O'Herrin, 2011; Rumann & Hamrick, 2009)e.

Social isolation and transitional stress on military connected students compound the challenges that they already face, including suicidal ideation, depression, anxiety, and PTSD. Suicide rates for all branches of the military have steadily increased each year, with more than 325 active-duty personnel taking their lives in

2018 (Department of Veteran Affairs and Department of Defense, 2019). Suicide risk is not limited to veterans who have been deployed; the suicide rate was highest for divorced military who have worked in administrative, mechanical, or electrical repair roles and those who have not deployed (Department of Veteran Affairs and Department of Defense, 2019). As stress levels increase, military-connected students who suffer from PTSD may experience an increase in symptoms that include intrusive recollection of traumatic events, sleep difficulties, hopelessness, and depression (Bryan & Anestis, 2011). Intrusive memories of trauma increase veterans' suicidality due to not only the severity of PTSD symptoms, but also the perceived burden on caregivers, partners, and family members (Bryan & Anestis, 2011; Calhoun et al., 2002).

The transition to civilian life reveals many obstacles for healthy relationships and connections to family, friends, fellow students, and faculty. Veterans with PTSD often experience increased levels of stress in regards to social situations resulting in further isolation and subsequent decreased overall mental health and wellbeing (Batten et al., 2009; Baumeister & Leary, 1995; Smith et al., 1999). Social stress increases social isolation with feelings of insecurity, loneliness, and instability creating further stress (Baumeister & Leary, 1995; Hawkey et al., 2003; Hawkey & Cacioppo, 2010; Lee et al., 2002). Much of this stress surrounds their family unit: divorce papers served upon return home, knowledge of a spouse being unfaithful, children avoid them, household self-sufficiency, no role for the veteran in the household, and lack of belongingness and connection to family members and friends (Moore & Kennedy, 2011; Bell & Schumm, 2011; Ellison et al., 2012). Each social interaction with family members and friends, reinforces the veteran's belief that they are alone, isolated and excluded from daily civilian life at a time when they also lack the camaraderie of their fellow troop members (Hoge et al., 2004). Military-connected students transitioning to academic life face greater stress as they attempt to fit into a new environment with new social connections with peers and faculty, possible financial challenges, disabilities, mental health disorders including PTSD and other health outcomes (DiRamio et al., 2008).

This study focuses on how the challenges presented by the transition to academic life impacts military-connected students' physical and mental health particularly in regards to PTSD as it correlates with response to a threat stimulus. Greater emphasis is placed on the impacts of the degree of PTSD symptomology and flight, fight or freezing response mediates their overall health and affects their transition into successful academic life.

PTSD and Stress Response System

PTSD impairs the stress response system with increased levels of cortisol and norepinephrine in response to every day events often leading to impairments in the hippocampus, chronic stress, impaired fear inhibition, and high anxiety (Bremner, 2006). These impairments and deficits result in a failure to regulate their emotions, recognize real threats, and respond to daily life events without lashing out at small irritations (Van der Kolk, 2006). The elevated levels of norepinephrine affect the hippocampus resulting in powerful memories of the trauma in long-term memory storage, flashbacks, and hypervigilance associated with PTSD (Davis et al., 1997). Veterans with PTSD showed higher fear responses, impaired fear inhibition, and greater amygdala activation than veterans with no disorders (Jovanovic & Ressler, 2010; Lissek et al., 2005). Greater amygdala activity increases the severity of PTSD symptoms, particularly in regards to intrusive memories, nightmares, sympathetic, and parasympathetic nervous system responses (Jovanovic & Ressler, 2010). Hyperarousal symptoms may be due to impaired connections between the prefrontal cortex (PFC) and the amygdala as exposure to fearful stimuli results in amygdala activation (Jovanovic & Ressler, 2010). Fear in response to stimuli can overpower the PFC so much that it is not able to inhibit activity in the amygdala (Liberzon et al., 1999; Rauch et al., 2006; Van der Kolk, 2006). Greater amygdala activity results in increased severity of the PTSD symptoms of intrusive memories, nightmares, FPS, sympathetic, and parasympathetic nervous system responses (Jovanovic & Ressler, 2010). The resulting hyperactivity in the amygdala pushes further activation of the autonomic nervous system, strengthens the endocrine response of hypothalamic-

pituitary-adrenal (HPA) axis, and increases the pathways that control the fight-flight-freeze response to stress (Van der Kolk, 2006).

Heart Rate Variability and PTSD

Heart rate variability (HRV) goes beyond the simple measurement of heart rate. Heart rate is the number of heartbeats per minute and presents information about the overall cardiovascular health of an individual. HRV is the measurement of the variability of interbeat intervals (IBIs) of consecutive heartbeats. The measurement of HRV provides a precise measurement of the health of the cardiovascular system, respiratory system, and nervous system and reveals whether these systems are impacted by mental, physical, or environmental stressors. While heart rate provides a picture at what is happening to the heart during a specific moment in time, HRV reveals how a person is able to tolerate stress and the overall health and resilience of an individual.

HRV reveals the overall health and functioning of the cardiovascular system and the autonomic nervous system (ANS) which helps to maintain and regulate the body's organs (Shaffer and Ginsberg, 2017). The ANS is comprised of two branches: the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). Both the SNS and the PNS respond to stress and trauma and result in dysfunction (Porges, 2007). HRV demonstrates how well the heart and brain work together, whether there is autonomic balance, health of the heart, vascular tone as it regulates blood pressure, digestion and facial expressions due to changes in facial muscles (Shaffer and Ginsberg, 2017).

HRV relates to how emotions are regulated, with higher levels of HRV connected to emotional and behavioral flexibility that provide greater physiological reactivity that results in better physical and mental health outcomes and greater physiological reactivity that (Beckham et al., 1990; Berntson et al., 1997; Foa & Kozak, 1986; Lang et al., 1970; Porges, 2007; Sack et al., 2004; Sloan et al., 2005; Thayer & Lane, 2000; Thayer et al., 2009). Higher levels of HRV correspond with self-regulation and skills necessary to manage thoughts, emotions, and goals. HRV provides a physiological measure that is correlated with emotion regulation with higher levels connected to greater emotion regulation and better health outcomes (Porges,

2007). Lower levels of HRV correspond with dysregulation in emotion regulation and inability to adapt to one's environment. This dysregulation leads to poorer mental and physical health outcomes and limited emotion regulation (Porges, 2007). HRV measurements are dependent on the time-domain used to record the HRV and are not interchangeable (Shaffer & Ginsberg, 2017).

Trauma or combat place the body under stress and it responds by activating the SNS with a rush of norepinephrine and epinephrine (Yehuda et al., 1998). These hormones are related to the 'fight, flight, or freeze' response and include dilated pupils, increased heart rate, muscle contraction, and inhibiting the digestion system (Yehuda et al., 1998). In response to the SNS, the PNS will respond after the stressor is over with 'rest and digest', which slows down the heart rate, relaxes the body, and focuses on digestion. Both SNS and PNS work together to manage heart rate, stress response, digestion and other systems to keep the body healthy and protected with health measured by the variability and adaptability to stressors within the environment (Berntson et al., 1997).

The Polyvagal theory describes how the vagus nerve affects the cardiac parasympathetic system in great detail (Porges, 2007). The vagus nerve begins in the nucleus ambiguus in the brain and connects to the cardiac sinoatrial node and the visceral organs that control emotional expression and communication (Porges, 1995). The vagus nerve conserves energy by slowing the heart rate and inhibiting sympathetic activity while increasing parasympathetic activity when a person is at rest. Stress or other cognitive challenges influence the way that the vagal system responds by decreasing the parasympathetic activity. The polyvagal theory presents the evolution of the self-regulatory functions and why they adapt to the environment, particularly for the parasympathetic system in regards to social functioning, emotion regulation, and expression (Porges, 1995). All of these areas are impacted when a person experiences life-threatening trauma. The Polyvagal theory supports the fight, flight or freeze defensive response to danger and aversive threats via the SNS by targeting the cardiovascular system for increased oxygen and glucose in the blood (Porges, 2007).

The Neurovisceral Integration Model builds upon the Polyvagal Theory by identifying a vast network of constructs that are influenced by the vagus nerve, central nervous and cardiovascular systems (Thayer &

Lane, 2000). The Neurovisceral Integration Model attempts to explain how the cardiac vagal tone, as measured by HRV, indicates the overall functioning of affective information processing, attentional regulation, classical conditioning, cognitive functioning, emotion regulation, self-regulation, and physiological flexibility (Sack et al., 2004; Thayer & Lane, 2000; Thayer et al., 2009). The model describes how individuals emotionally respond to the world around them and direct their behaviors toward meeting a goal. If everything is working properly, the individual's emotions will adapt and respond to the environment via self-regulation. Self-regulation allows an individual to choose one response over another and inhibit less appropriate responses to the current environment (Thayer & Lane, 2000; Thayer et al., 2009). The Neurovisceral Integration Model continues to expand on how HRV is a significant marker for the cognitive processing of emotional stimuli for emotion regulation, attentional awareness to process environmental stimuli, and stress response via behavioral and neuroimaging studies that have identified the neural networks connected to these processes (Thayer & Lane, 2000; Thayer et al., 2009)

PTSD inhibits processing of environmental cues and self-regulation for appropriate responses to the environment resulting in dysregulation, mental and physical pathologies (Thayer & Lane, 2000; Thayer et al., 2009). Emotion dysregulation results in rigidity and decreased adaptability to changes in the environment, which in turn lead to poor physical and mental outcomes (Thayer & Lane, 2000; Thayer et al., 2009). Individuals who have higher HRV tend to be better at regulating their emotions, adapting to their environment and healthier physical and mental states. Those with lower HRV tend to be more rigid, hyper-vigilant, and maladaptive in their response to changes in their environment (Thayer & Lane, 2000; Thayer et al., 2009).

Both the Polyvagal Theory and the Neurovisceral Integration Model have presented a possible connection between HRV and PTSD. Many significant studies have found evidence that there is a relationship between lower HRV and PTSD diagnosis. A few studies that did not include any manipulations have found that individuals with PTSD have lower resting HRV than healthy individuals (Blechert et al., 2007; Cohen et al., 1997). Additional studies added manipulations to measure baseline HRV and HRV while

recalling a traumatic or stressful event that may have led to PTSD (Cohen et al., 1998). For participants with PTSD, there were no significant changes found between the baseline HRV and recall event HRV. However, for the non-PTSD group, low variability was recorded for the recall event HRV while the baseline HRV had significantly higher variability than the PTSD sample. Prior confirmation was found when comparing baseline HRV and HRV during recall for individuals with PTSD, individuals with panic disorders, and healthy control group (Cohen et al., 2000). The individuals with PTSD showed significantly lower variability in baseline HRV measurement when compared to the other groups. While the individuals with PTSD did not show any significant difference between baseline HRV and the recall event HRV, the other groups experienced lower variability HRV while performing the recall task and higher variability in baseline HRV measurements. Additional studies have all found that individuals with a clinical diagnosis of PTSD had lower variability in their baseline HRV than healthy individuals (Blechert et al., 2007; Cohen et al., 1997; Cohen et al., 1998; Cohen et al., 2000).

Fear Response

One of the key symptoms that differentiate PTSD from other mental disorders is fear response to everyday activities. The amygdala controls fear response in the brain. Trauma results in both over activation and dysregulation of the amygdala response, which results in dysfunction in fear processing and fear inhibition impairment (Brocke et al., 2006). Brain serotonin (5-HT) systems and serotonin have been studied extensively in connection with PTSD due to the increased activity in the amygdala (Davis et al., 1997). The serotonin neurotransmitter assists with numerous functions related to PTSD symptomology, including aggression, fear response, appetite, alcoholism, sleep disturbances, heart rate variability, impulsivity, depression, anxiety, and suicidality (Davis et al.; Southwick et al., 1999; Vermetten & Bremner, 2002). Dysregulation of the serotonergic system results in the overreaction to both threatening and safe stimuli, how trauma memories are processed and the coordination of fear-based behaviors (Murrough et al., 2011).

In addition to serotonin, there are elevated levels of norepinephrine in response to trauma impacts the hippocampus resulting in realistic memories of the trauma in long-term memory storage (Davis et al.,

1997). Elevated levels of norepinephrine result in the flashback triggers, intrusive memories, and hypervigilance associated with PTSD (Davis et al., 1997). When PTSD presents with symptoms related to both serotonin and norepinephrine, selective serotonin reuptake inhibitor (SSRI) have proven effective resulting in a decrease of those symptoms related to fear response, intrusive memories and hypervigilance (Davis, et al., 1997; Vermetten & Bremner, 2002).

“Fight-Flight or Freeze” Avoidance and Tonic Immobility

Research provides many conflicting definitions in regards to ‘fight flight and freeze’, avoidance, and tonic immobility. Numerous animal studies have attempted to distinguish different responses to threat (Kalin et al., 1998). Research on humans tends to be harder to control and manipulate. Some newer studies focus on human threat response with past studies focusing on avoidance or flight (Brewin & Holmes, 2003; Chen & Bargh, 1999) and other new studies focus on tonic immobility or freezing (Blanchard et al., 2001; Volchan et al., 2017). Humans and other mammals exhibit a freezing response in a wide variety of forms that include tonic immobility, attentive immobility and immobility under attack (Volchan et al., 2017). Freezing is a defense response that occurs after risk assessment, enhances attentional processes, and prepares the body for action once escape can happen (Blanchard et al., 2001; Lang et al., 1997). Current research on freezing defines this phenomenon in several ways, including tonic immobility, attentive immobility, or immobility under attack when both humans and other animals (Hagenaars et al., 2014; Volchan et al., 2017) exhibit freezing. Since freezing is often used synonymously with these types of immobility, particularly for humans, it is difficult to compare studies and constructs of immobility. In animal studies, immobility is easier to study for various immobility responses to make the distinction between these behaviors for prey and predators (Eilam, 2005; Marks, 1987; Ratner, 1967). Ethically, humans cannot be subjected to the same types of threats that would illicit these variable automatic responses. Freezing as a defense response that occurs after risk assessment, optimizes attentional processes, and prepares the body for action once escape can happen (Blanchard et al., 2001; Lang et al., 1997).

Automatic threat response helps us to survive when are under a perceived attack. The perceived attack may be physical, mental or emotional and not always a direct, imminent threat. Research uses aversive stimuli to provoke automatic threat response, which can include avoidance and tonic immobility and contract that response. The threat response is contrasted to a response to pleasant or neutral stimuli that could provoke an approach response. The automatic defensive response that is chosen depends on the form of aversive stimuli and options for escape. The premotor cortex reveals a connection between peripersonal space and the defense response that is chosen (Graziano & Cooke, 2006). Peripersonal space is the area around the body that will allow for fight or flight and changes in different situations and surroundings (Cléry et al., 2015; Hayduk, 1978; Holt et al., 2014; Horowitz et al., 1964; Lloyd, 2009). Miniscule motor reactions present challenges when measuring in research studies. Avoidance or freezing result in tiny body movements in the split of a second that may be indiscernible to an observer.

Psychometric instruments have been used to record tonic immobility via retrospective reports from female survivors of sexual assault, students, police officers and patients with PTSD (Abrams et al., 2009; Bados et al., 2008; Bovin et al., 2008; Fiszman et al., 2008; Fusé et al., 2007; Heidt et al., 2005; Humphreys et al., 2010; Lima et al., 2010; Maia et al., 2015; Portugal et al., 2012; Rocha-Rego et al., 2009; TeBockhorst et al., 2015). Individuals with PTSD scored twice as high for tonic immobility than individuals without PTSD. As the severity of PTSD symptomology increases, the retrospective reports of tonic immobility also increases and decreases the effectiveness of medicine used to treat PTSD (Fiszman et al., 2008; Lima et al., 2010; Rocha-Rego et al., 2009). These retrospective studies reveal a connection between the freezing response and development of PTSD (Bovin et al., 2008; Galliano, Noble, Puechl, & Travis, 1993; Heidt, Marx, & Forsyth, 2005). Retrospective reports are not always reliable nor do they allow for the capture of various responses to a similar threat. Numerous studies have employed a stabilometer to measure approach, avoidance and freezing responses to stimuli (Azevedo et al., 2005; Bastos et al., 2016; Facchinetti et al., 2006; Fiszman et al., 2008; Lima et al., 2010; Rocha-Rego et al., 2009; Volchan et al., 2011). Stabilometry provides a way to measure body sway via a controlled method. A stabilometer is a machine that is calibrated to measure

miniscule movements made during a standing pose or stride across the machine (Volchan, et al., 2017). Stabilometers record the continuous rebalancing of weight, center of gravity and redistribution of equilibrium on the soles of the feet (Volchan, et al., 2017). Stabilometers are sometimes referred to as force plates, balance boards, or other terms to references the recording of body sway movements. This study uses a MobileMat by Tekscan that is defined as a low-profile pressure sensing mat that captures static and dynamic pressure measurement data for foot function and gait analysis, as well as balance, sway and postural data. Stabilometry reveals a freezing response that has been correlated with increased PTSD symptomology and severity (Fizman et al., 2008; Lima et al., 2010; Rocha-Rego et al., 2009; Volchan et al., 2011; Volchan et al., 2017). When peripersonal space allows for escape, studies reveal that humans engage in avoidance or flight as indicated by increase body sway (Bastos et al., 2016). However, when there is no possibility for escape, a freezing response with bradycardia has been recorded (Bastos et al., 2016).

Tonic Immobility and PTSD

Individual differences in fear response and defensive strategies may be due to physiological, neurobiological, and neuroendocrinological differences (Korte et al., 2005). While fear response is primarily an evolutionary adaptation and beneficial for survival in an ever changing environment, defensive strategies may be maladaptive and result in great harm for the individual. Animal studies revealed that repeated inductions of tonic immobility resulted in long-term health issues and mortality (Liberson et al., 1961; Ratner, 1967). Individuals suffering from PTSD may experience tonic immobility or similar freezing responses to every day stimuli. The freezing response may be to internal or external cues and the freezing response is often a daily occurrence that is coupled with sustained tachycardia, reduced HRV, and reduced body sway (Volchan et al., 2011).

Individuals who experience freezing from a traumatic event tend to have more severe symptoms than those who do not experience freezing with greater risk of brain dysregulation for multiple freezing responses (Fizman et al., 2008; Monroe & Harkness, 2005; Kario et al., 2003; Lima et al., 2010; McEwen & Gianaros, 2010; Rocha-Rego et al., 2009). The long-term reduction in HRV has been associated with

increased risk of cardiovascular disease, inflammation and a compromised immune system (Kemp & Quintana, 2013). Greater understanding of why certain individuals respond to threats with freezing will allow for more effective therapies and medicines to help individuals with PTSD.

PTSD diagnosis depends almost entirely on retrospective and self-reports. The inclusion of information about tonic immobility and fear response would allow for more treatments that are effective. Often PTSD is treated with selective serotonin reuptake inhibitors (SSRIs) as depression is a co-morbidity. Unfortunately, individuals who have a freezing response to threat and trauma do not respond to traditional SSRIs and are often resistant to SSRIs (Berger et al., 2009; Fiszman et al., 2008; Houlihan, 2010; Lima et al., 2010; Naylor et al., 2015). Further study that features fMRI or other brain imaging would allow for greater understanding on how avoidance and tonic immobility affects different areas of the brain and why SSRIs are not effective for tonic immobility.

Fear Inhibition

PTSD is a complex disorder with one of the stress responses being the inability to control fear. Individual differences in fear inhibition may lead to understanding how certain individuals are at an increased risk to develop PTSD. Normal development of fear inhibition occurs via the learning of safety and danger signals. Fear inhibition allows people to understand the difference between danger and safety and will inhibit fear response to safe situations (Jovanovic & Ressler, 2010).

Veterans with PTSD showed higher fear responses, impaired fear inhibition, and greater amygdala activation than veterans with no disorders (Jovanovic & Ressler, 2010; Lissek et al., 2005). Impairment of the fear potentiated startle (FPS) response occurs primarily in PTSD and could be a specific biological marker for diagnosing PTSD. Fear in response to stimuli can overpower the prefrontal cortex (PFC) so much that it is not able to inhibit activity in the amygdala (Liberzon et al., 1999; Rauch et al., 2006). Hyperarousal symptoms may be due to impaired connections between the PFC and the amygdala as exposure to fearful stimuli results in amygdala activation (Jovanovic & Ressler, 2010). Greater amygdala activity results in increased severity of the PTSD symptoms of intrusive memories, nightmares, FPS, sympathetic, and parasympathetic nervous

system responses (Jovanovic & Ressler, 2010). Fear inhibition measures may both identify veterans who are at risk for developing PTSD and interventions that increase resilience.

The FPS response has been defined as an increase in the startle response when aroused by a conditioned stimulus (CS) combined with a dangerous unconditioned stimulus (US) (Grillon & Morgan, 1999; Jovanovic et al., 2005). A normal FPS responds only to dangerous or aversive situations. An impaired FPS responds to any situation whether safe or dangerous revealing an impairment to fear inhibition. The acoustic startle paradigm used to investigate FPS response reveals the impact of genetic variations in serotonergic function, fear inhibition and fear potentiation (Brocke et al., 2006). The FPS response measurement allows for verification of the differences between individuals with the S' allele and the L' allele in the 5-HTTLPR, as the S' allele impairs serotonin uptake and results in greater amygdala activity when faced with terror or threat (Brocke et al., 2006). Fear inhibition impairment may correspond with the S' allele, confirming that impairment of fear inhibition is an intermediate phenotype for PTSD. The impaired fear inhibition phenotype may be both a predictor for PTSD development and a deficit caused by PTSD (Jovanovic & Ressler, 2010).

Hypotheses and Research Questions

The current investigation examines the freezing and avoidance response as it corresponds to pleasant and threatening stimuli in military-connected students. The study investigates normal and impaired fear response in controlled conditions that mimic exposure to pleasant, neutral, and unpleasant stimuli to determine if an increased freezing response corresponds to students with increased PTSD symptomology. This study explores how these differences relate to physical, psychological, and stress-related health as students transition from military to civilian life. Primary emphasis of this study revolves around the effects of PTSD on mental, physical, and social outcomes.

My primary hypothesis (H1) was that individuals with severe levels of PTSD symptomology would demonstrate a freezing response when presented with all stimuli and an increased freezing response when

presented with unpleasant stimulus. This hypothesis as well as the third hypothesis is considered an attempt to replicate previous studies that have observed the freezing response in various human populations: young men (Azvedo et al., 2005); young women (Hagenaars et al., 2011); PTSD diagnosed participants (Volchan, 2011). These prior studies build upon animal studies that elicited the freezing response from animals (Lang et al., 1997; Maser & Gallup, 1997; Stevens & Gerzog, 1977) in an effort to measure the freezing response in humans.

Our second hypothesis (H2) was that individuals with moderate levels of PTSD symptomology would react with avoidance of unpleasant stimulus and a natural body sway when presented with neutral and pleasant stimuli. This is an exploratory hypothesis that builds upon previous freezing studies (Azvedo et al., 2005; Hagenaars et al., 2011; Volchan, 2011) to see if the stabilometer will provide accurate measurements to determine avoidance movements.

My third hypothesis (H3) was that individuals with little to no PTSD symptomology would demonstrate a freezing response when tested with unpleasant stimulus and natural body sway when presented with neutral and pleasant stimuli. This hypothesis is supported by prior studies (Azvedo et al., 2005; Hagenaars et al., 2011; Volchan, 2011) and is a replication of their methods with a new population.

My final hypothesis (H4) is that an increase in traumatic events experienced by an individual will correspond with an increased freezing response to all stimuli. This hypothesis is supported by prior research that found that individuals with numerous traumatic life events will have reduced body sway and more likely to respond to stimuli with a freezing response (Hagenaars et al., 2011).

Both freezing response and avoidance are connected to PTSD, the physical measurement of body sway allows for the diagnosis of PTSD symptoms that goes beyond current self-report measures. Currently, clinicians diagnose PTSD via self-report measures only. Self-report measures are lacking, as many veterans will not respond honestly, as they do not want to appear weak or show that they have a mental illness. Physiological markers like body sway and tonic immobility would allow clinicians to diagnose PTSD without a lengthy interview or self-report measure and allow the clinicians to separate PTSD from other disorders.

This study observed veteran students with varying levels of PTSD symptomology with the following aims: (i) to replicate the freezing and avoidance responses to various stimuli, (ii) to identify an objective measure for these phenomena, and (iii) to determine how a freezing response correlates to the severity of PTSD symptomology and traumatic life events. This study's hypothetical model is reported in Figure 1.

Method

Participants

Thirty-eight University of North Florida students ($n=38$; 18 female; 20 male) were recruited from the Military Veterans Resource Center to capture veteran and military-connected students. The students' ages ranged from 19 to 49. While all branches of the military were represented, almost half (18) students served in the navy. Time of service ranged from 1 year to 24 years. Over half (23) of the students were deployed to a combat zone, including Iraq (9), Afghanistan (5), Kuwait (2), Southwest Asia (3), Africa (3), or on a ship (14). Seven veterans were deployed on peacekeeping missions. Thirteen veterans suffer from a permanent physical injury related to military service, including traumatic brain injury, chronic cough, chronic pain, and orthopedic injuries. Over half (22) of the veterans have a VA disability rating. Only eight veterans revealed that they have been diagnosed with PTSD. Two participants had body sway measurements excluded due to physical disabilities. Four participants have missing body sway measurements due to technical difficulties with saved data. Three participants have missing heart rate data due to instrument errors.

Upon approval from the Institutional Review Board of the University of North Florida, participants signed the informed consent documentation before assessment. Participants could withdraw from the experiment at any time. Based on a power analysis using G*Power (Faul et al., 2007), for data analysis with multiple regression, with a medium effect size, $f = .35$, significance (p-value) set at $p < .05$, and power established at .80, the minimum required sample size was 43. The recommended good practices for analysis of this type of study with statistical tests including ANOVAs, ANCOVAs, correlations and multiple regression require at least 30-50 participants for effective power (Lancaster et al., 2004). This sample size

would allow for 80% power and a medium effect size. Unfortunately, due to the coronavirus (COVID-19) data collection was abruptly stopped during the spring 2020. This has resulted in less than optimal power effects and will reveal trends in the data for this novel approach.

Procedure

The participant met a trained research assistant in the atrium of the UNF Psychology Department and then be escorted back to the laboratory suite. After providing a short description of the study, the researcher presented the participant with an informed consent document. After reviewing the informed consent document, the researcher reviewed whether the participant had any questions. If the participant consented and signed the informed consent document, they continued with the study. If the participant did not consent to the study, they were escorted back to the atrium.

The participant completed several self-report assessments while seated in front of a computer. Self-report assessments were administered via Qualtrics. Survey instruments include: the PTSD Checklist (PCL-5; Weathers et al., 2013), Brief Trauma Questionnaire (BTQ; Schnurr et al.,1999), Perceived Stress Scale (PSS; Cohen et al., 1983), Social Connectedness Scale - Revised (Lee et al., 2001), UCLA Loneliness Scale - 8 (ULS-8; Hays & DiMatteo), Patient Health Questionnaire (PHQ-8; Kroenke et al., 2009), General Anxiety Disorder Questionnaire (GAD-7; Spitzer et al., 2006), Physical Health Questionnaire (PHQ; Schat et al., 2005), MOS SF-36; Ware & Sherbourne, 1992. In addition, demographic, health, and military questions were administered.

Heart Rate Variability (HRV) Baseline. After the participant completed the assessments, they were asked to apply a heart rate monitor based on a display with instructions in a private room. HRV baseline measurements were taken while participant sat quietly and motionless for 2 minutes. HRV baseline measurements were taken before and after each task (Stabilometer). Additionally, heart rate measurements were taken simultaneously with HRV.

Stabilometer. In order to objectively measure the body sway and the freezing response, participants stood on a Tekscan pressure mat (Tekscan Inc., South Boston, MA) to measure posterior-anterior and left-to-

right body sway with the following stimuli presented in 60 second increments: eyes open with a white cross on a black background for a baseline, and 3 video stimuli presented in random order (pleasant, unpleasant and neutral). Participants completed a 60-second baseline trial to familiarize themselves with the stabilometric platform while viewing a black screen with a fixation cross on a monitor positioned one meter away at eye level. After the baseline trial, participants completed three 60-second intervals of randomized sets of neutral, pleasant, and unpleasant images from the International Affective Picture System (IAPS; Lang, P., Bradley, M., & Cuthbert, B., 1997) while standing on the stabilometric platform with a randomized order for the image set type. Between each interval, the participants relax while a black screen is shown for 10 seconds followed by a 5-second black screen with a white fixation cross to refocus attention...

Materials

Post-Traumatic Stress Disorder Checklist for Civilians (PCL-5; Weathers et al., 2013). The PCL-5 was administered to measure PTSD symptom levels in participants. The PCL-5 captures the 20 DSM-5 symptoms in a short and easy 20-item self-report measure. This PCL-5 has a high reliability (Cronbach's $\alpha = .90$) for PTSD diagnosis and screening (Weathers et al., 2013). This study found good internal consistency (Cronbach's $\alpha = .94$). It is also used to monitor symptom levels before and after treatment to determine effectiveness. A score of 40 or higher reflecting a severe degree of PTSD symptoms; while lower scores allow for greater detection of PTSD (Weathers et al., 2013). Participants respond to the degree that they are experiencing post-trauma symptomatology based on 5-point scale for each item ranging from zero to four. The suggested PCL cutoff score for screening purposes is 30-35 (PCL-5). Scale scores for the total PCL-5 severity score ranged from 17 to 85 points, with a higher score denoting higher or more severe PTSD symptomatology. This study factored participants into three categories based on PTSD symptomatology: no to low symptomatology (scores 0 to 14), moderate symptomatology (scores 15 to 27), and severe symptomatology (scores 28 to 62). The internal reliability remained high for the symptomatology groups (Cronbach's $\alpha = .95$).

Brief Trauma Questionnaire (BTQ; Schnurr et al., 1999). The BTQ is a 10-item questionnaire with yes or no answers, which measures the lifetime history of trauma exposure. Trauma exposure includes the frequency of traumatic events that incited fear of death, terror, horror, and/or helplessness due to both child abuse and other traumatic events. The BTQ will be administered to measure lifetime history of traumatic life events. The BTQ provides a sum score reflecting the total frequency of different types of trauma (e.g., child abuse, combat, car accident, sexual assault) that a participant has experienced throughout their lifespan. The BTQ score is a covariate with the PTSD designation and affects the freezing response.

Perceived Stress Scale. (PSS-10; Cohen et al., 1983). The PSS-10 contains 10 questions about perceived stress during the past month. The scores are summed with higher scores corresponding with higher levels of perceived stress. This study found good internal consistency (Cronbach's alpha = .90). The PSS-10 has good reliability of (Cronbach's alpha = 0.84-.85) across several populations (Cohen et al., 1983).

Depression Patient Health Questionnaire. (PHQ-8; Kroenke et al., 2009). The PHQ-8 is an 8-item questionnaire that measures the level of depressive symptoms experienced by participants for the last 30 days. The scores are summed to reveal higher levels of depressive symptoms. This study found good internal consistency (Cronbach's alpha = .96). Test-retest correlations confirmed validity for periods of 48 hours (Cronbach's alpha = 0.84-.95) and 7 days (Cronbach's alpha = 0.81-.96) (Smarr & Keefer, 2011).

Generalized Anxiety Disorder-7. (GAD; Spitzer et al., 2006). The GAD contains seven items that provide a generalized anxiety screening that review the past two weeks. Scores are summed to reveal the severity of anxiety symptoms with a score of ten or higher serving as a general cutoff. This study found good internal consistency (Cronbach's alpha = .89). The GAD has good internal consistency, test-retest reliability and across clinical and general populations, there is good convergent, construct, criterion, and factorial validity (Cronbach's alpha = 0.89) and cross-cultural validity (Löwe et al., 2008; Spitzer et al., 2006).

Social Connectedness Scale. (SCS; Lee & Robbins, 1995). The SCS measures three aspects of an individual's social connectedness across eight items affiliation (3 items), companionship (1 item), and connectedness (4 items). This study found good internal consistency (Cronbach's alpha = .96). There is

good test-retest reliability (Cronbach's alpha = .96) with strong internal reliability and stability (Lee & Robbins, 1995). The responses are summed to provide a score that reveals the perceived level of social connectedness. The higher the score the higher the level of social connectedness. Higher scores of social connectedness positively correlate to collective self-esteem and independent self-construct. Lower scores of social connectedness negatively correlate with depression, hostility, social discomfort and social distress (Lee et al., 2001).

UCLA Loneliness Scale. (ULS; Russell, 1996). The ULS contains 20 items that are related to loneliness and feelings of isolation. The responses are summed so that higher scores reveal an increased degree of loneliness. This study found this scale to be highly reliable (Cronbach's alpha = 0.87). There is a high degree of internal consistency and reliability (Cronbach's alphas = 0.89 to 0.94) across several populations (Russell, 1996).

Physical Health Questionnaire. (PHQ; Schat et al., 2005). The PHQ captures 11 items relating to physical health over the participant's last 30 days. After the items are summed, a higher score corresponds with negative health outcomes. This study found good overall internal consistency (Cronbach's alpha = .83). There is good internal consistency and reliability for gastrointestinal problems (Cronbach's alpha = .83), headaches (Cronbach's alpha = .88), and sleep disturbances (Cronbach's alpha = .80). These physical symptoms often result from stress and correspond to PTSD.

MOS 36-Item Short Form Survey Instrument. (MOS SF-36; Ware & Sherbourne, 1992). The MOS SF-36 assesses general health domains that limit physical, social, and usual activities due to physical, mental, emotional, or psychological problems. Physical pain and fatigue levels are also assessed. This study found good internal consistency (Cronbach's alpha = .88). There is good validity for the scales with Cronbach's alpha in the range of .78-93 (McHorney et al., 1994). A general overall score is provided for all health outcomes and subdivided into the domains of general health, emotional problems, social functioning, and energy levels. The ratings are averaged together with higher scores indicating higher functioning and favorable health outcomes. Lower scores indicate diminished functioning and poorer health outcomes.

Heart Rate Variability (HRV), Electrocardiogram (ECG). Self-adhesive ECG electrodes placed on a three-lead configuration on the participant's chest. The ECG and heart rate period data from the monitoring devices (eMotion Faros ECG sensor, Mega Electronics, Ltd, Kupio, Switzerland) will be visually inspected and calculated with Kubios HRV software (<http://kubios.uef.fi>). This method is an accurate and sensitive quantitative indicator of HRV (Lewis et al., 2012). HRV provides a noninvasive way to assess the variation between heartbeat intervals and provides a quantitative measurement for the functioning of the autonomic nervous system (Minassian et al., 2014; Pole, 2007). Of particular relevance to the current study, HRV is an established measure of vagal influence on the heart, with decreased HRV associated with higher stress and with PTSD (Porges, 2009; Tan et al., 2011). Additionally, a freezing response is categorized by reduced body sway and bradycardia (Blanchard et al., 2001; Volchan et al., 2017). A deceleration in heart rate corresponds with the freezing response. Participants with severe PTSD symptomology may exhibit tachycardia or a higher heart rate and reduced HRV than other participants across all stimuli. This may indicate the cumulative effect of traumatic life events on the defense system.

Stabilometer. Participants stand on a Matscan pressure mat (Tekscan Inc., South Boston, MA), which contains pressure sensors (sensors) to measure applied force. The stabilometer provides measurements of body sway, freezing, anterior-posterior and left-right movements using the SAM™ (Sway Analysis Module) software module. Body sway variability reveals unmyelinated vagal activity that promotes freezing behavior. This measure is relatively new in the area of stress and PTSD, although there is some evidence that PTSD and stress is associated with tonic immobility (Fragkaki et al., 2016; Roelofs et al., 2002; Volchan et al., 2011). During each stimuli, anterior-posterior (AP) body sway, left-right (LR) body sway, body sway variability (VAR), and area was captured. Freezing was defined as lower scores indicating an increased immobility or freezing response. Avoidance was defined as an increase in AP body sway, with variance in foot pressure, revealing a movement away from the stimuli. Body sway variability has been defined as overall variability, area, AP body sway, and LR body sway. Regardless of measure, a decrease in variability reveals an increase

immobility and freezing response. For this study, the AP body sway has been used to define freezing, avoidance, and body sway. Examples of the body sway measurements may be found in Figures 14 and 15.

Pictorial stimuli. Three sets of stimuli were selected based on previous studies for replication purposes. These stimuli were chosen from the International Affective Picture System (IAPS; Center for the Study of Emotion and Attention, 1999). The pleasant video contained 20 pictures of various energetic sports (IAPS catalog 8021, 8032, 8034, 8040, 8041, 8090, 8161, 8186, 8190, 8192, 8200, 8210, 8300, 8370, 8400, 8460, 8465, 8467, 8470, and 8620). The unpleasant video contained 20 pictures of blood, mutilated bodies, and death (IAPS catalog 3000, 3010, 3030, 3051, 3053, 3060, 3061, 3062, 3063, 3064, 3069, 3080, 3100, 3102, 3110, 3130, 3140, 3150, 3261, and 3400). The neutral video contained 20 pictures of neutral objects (IAPS catalog 7000, 7002, 7004, 7006, 7009, 7010, 7020, 7025, 7030, 7031, 7035, 7040, 7050, 7052, 7060, 7080, 7090, 7150, 7175, and 7211). These images were displayed on a television monitor roughly 1 meter away from the participant at eye height. A baseline measurement was taken on the stabilometer with a white cross on a black background. An additional stress inducing measurement was taken by having the videos and images were presented in a random order.

Results

Preliminary analysis of the data with means and standard deviations for the variables utilized in these analyses. Pearson correlations were utilized to discover patterns of associations between predictor variables, with significant correlations and their coefficients. Further analysis was performed via repeated measures ANOVAs and ANCOVAs on the hypotheses and tested in support of the proposed model. Due to the size of the sample, mediators and moderators were not included in this analysis as significance was lacking. The bootstrap approach was not employed as the novelty of the body sway measurements made this option less than appropriate. All statistical analysis was performed using the SPSS software package (IBM – version 22).

For some of the analysis, participants were grouped by levels of PTSD symptomology to observe trends that correspond to the hypotheses. The participants were broken into three groups based on responses to the PCL-5 rather than how they responded to the PTSD diagnosis question. Only eight

participants responded as being formally diagnosed with PTSD. Questions from the PCL-5 focus on symptoms experienced in the past 30 days rather than on the chronic nature of PTSD. Participants were grouped based on severity of PTSD symptomology, with 13 participants with no symptoms to low symptoms (scores 0 to 14), 12 participants with moderate symptomology (15 to 27), and 14 participants with severe symptomology (28 to 62). The suggested PCL cutoff score for screening purposes is 30-35 (PCL-5). However, when reviewing the initial data, symptom severity seemed to have an effect on the freezing response. These group ranges provided almost equal groups with a decent range of symptom severity.

Sociodemographic, Trauma and Stress, Mental and Physical Health, and Social Constructs

Individual differences between sociodemographic and various scale variables were computed via analyses of variance (ANOVAs) to determine significant differences. All means and standard deviations for the groups with significance differences are reported in Tables 1, 2, 3 and 4. Individual differences were not observed as significant for the following demographics: sex, race or ethnicity, marriage status, first time in college, first generation college student, or deployment location. These variables assist in understanding how environmental factors influence PTSD development. Due to the small sample size, mediation and moderation effects could not be calculated in regards to the overarching hypotheses; however, there are some interesting patterns to report.

Deployment Status

Deployment status revealed significant individual differences regardless of deployment location or if the deployment included combat. Participants who deployed have increased PTSD scores [$F(1, 37) = 5.335, p = .027$]. Deployed participants also reported to have increased perceptions of stress [$F(1,37) = 6.786, p = .013$], decreased social connectedness [$F(1,37) = 8.054, p = .007$], increased perceptions of loneliness [$F(1,37) = 6.507, p = .015$], increased symptoms of depression [$F(1,37) = 6.881, p = .013$], increased generalized anxiety [$F(1,37) = 4.215, p = .047$], and decreased physical health [$F(1,37) = 8.926, p = .005$].

Gross Income

Participants' gross income also produced significant differences across many of the variables.

Participants with incomes less than \$10,000 have increased PTSD scores [$F(2, 37) = 3.295, p = .049$], increased incidence of trauma [$F(2, 37) = 5.801, p = .007$], and decreased physical health [$F(2, 37) = 3.889, p = .030$].

PTSD Diagnosis

Additionally, if participants revealed their PTSD diagnosis that they received from Veterans' Affairs after military service, there were significant individual differences revealed across all scale variables.

Participants with PTSD have significant differences in mental health with reported increased symptoms of PTSD [$F(1,37) = 13.414, p = .001$], increased perceptions of stress [$F(1,37) = 4.529, p = .040$], increased symptoms of depression [$F(1,37) = 14.450, p = .001$], and increased symptoms of generalized anxiety [$F(1,37) = 10.257, p = .003$]. Participants with a PTSD diagnosis have significantly increased incidence of trauma [$F(1, 37) = 27.797, p \leq .000$]. They experience decreased physical health [$F(1, 37) = 7.236, p = .011$] and worse medical outcomes [$F(1, 37) = 15.805, p \leq .000$]. Participants with a PTSD diagnosis also report significant differences with decreased perceptions of social connectedness [$F(1, 37) = 8.393, p = .006$] and increased perceptions of loneliness [$F(1, 37) = 5.237, p = .028$].

VA Disability Rating

Participants with a VA disability rating have significant differences across the variables than participants without. While participants with a VA disability rating reported increased PTSD symptomology [$F(1, 37) = 8.626, p = .006$] there was no significant difference in incidence of trauma. These participants did have increased perceptions of stress [$F(1, 37) = 9.729, p = .004$], reported more symptoms of depression [$F(1, 37) = 10.265, p = .003$], and increased symptoms of generalized anxiety [$F(1, 37) = 9.166, p = .005$]. Participants with a VA disability rating also reported worse medical outcomes [$F(1, 37) = 6.145, p = .018$]. Participants

with a VA disability rating also report decreased social connectedness [$F(1, 37) = 6.412, p = .016$] and increased loneliness [$F(1, 37) = 10.865, p = .002$].

Bivariate Relationships

Bivariate relationships were assessed via Pearson correlation coefficients to analyze the relationships between PTSD and the following variables: brief trauma questionnaire (BTQ), perceived stress, depression, generalized anxiety, physical health, medical outcomes, social connectedness, and loneliness. Significant correlations and their coefficients may be found in Table 5. Overall, PTSD is a strong predictor for all of the stress, mental, physical and social outcome variables.

PTSD had a statistically significant positive correlation with previous incidence of trauma [$r(38) = .573, p \leq .000$], perceptions of stress [$r(38) = .700, p \leq .000$], depression [$r(38) = .805, p \leq .000$], generalized anxiety [$r(38) = .745, p \leq .000$], and loneliness [$r(38) = .593, p \leq .000$]. PTSD had a statistically significant negative correlation with social connectedness [$r(38) = -.646, p \leq .000$] and general medical outcomes [$r(38) = -.695, p \leq .000$]. While the correlation is positive, PTSD had a significant correlation with physical health outcomes [$r(38) = .689, p \leq .000$] which means that PTSD increases problems with physical health as a higher score reveals poorer physical health.

PTSD had statistically significant positive correlations with heart rate during the emotional stimuli for the body sway measurements: pleasant stimuli [$r(31) = .413, p = .021$], unpleasant [$r(31) = .019$], and neutral stimuli [$r(30) = .373, p = .042$]. There was no significant correlation with the eyes open stimuli. PTSD did not have any significant correlations with the body sway measurements. However, participants who were diagnosed with PTSD did have significant correlations with anterior-posterior measurements during two stimuli. Being diagnosed with PTSD after military service negatively correlated with anterior-posterior body sway during the eyes open stimuli [$r(37) = -.354, p = .032$] and positively correlated with anterior-posterior body sway during the unpleasant stimulus [$r(37) = .363, p = .027$]. PTSD did have a significant correlation with heart rate during all of the emotional stimuli. These correlations are reported in Table 6.

Hypotheses

The initial hypotheses surrounded PTSD symptomology and the effects on body sway in response to various stimuli. Given the novel nature of the body sway measurements and small sample size, many of the hypotheses did not reveal any significance. Analysis via group splitting was attempted but that did not reveal any significance. While bootstrapping is appropriate when sample sizes are small to help adjust for power, the body sway measure is fairly unknown and bootstrapping could result in false positive bias. Additional analysis and graphing was performed to reveal any trends that may be of interest for future research.

Hypotheses 1-3

H1: Severe PTSD symptomology would demonstrate a freezing response across all stimuli, with more pronounced freezing response to unpleasant stimulus.

H2: Moderate PTSD symptomology would react with avoidance of unpleasant stimulus and body sway when presented pleasant and neutral stimuli.

H3: No to low PTSD symptomology would demonstrate a freezing response to the unpleasant stimulus and body sway when presented with pleasant and neutral stimuli.

A repeated measures ANOVA was performed by PTSD symptomology groups against the four emotional stimuli with the anterior-posterior (AP) body sway measurements. There was no significant interaction between PTSD symptomology and the AP body sways measurements. In regards to anterior-posterior (AP) body sway, no significance was found in any groups response to the emotional stimuli. Means and standard deviations for the above hypotheses are reported in Table 7. Trends from the means have been graphed in Figure 2. There is a marked trend where all groups respond with freezing towards the unpleasant stimulus. The freezing response is qualified by the reduced body sway and reduced heart rate during the unpleasant stimulus. The means and standard deviations for heart rate during the emotional stimuli are reported in Table 8. Regardless of the severity of PTSD symptomology, all groups had a reduction in heart rate and reduction of body sway during the unpleasant stimulus, as seen in Figure 3. While the severe PTSD symptomology group responded with decreased body sway towards the unpleasant stimulus, their freezing

response was greater in response to the neutral stimuli. When only considering PTSD symptomology, no group revealed any tendency to avoid the unpleasant stimulus. Further analysis for H4 revealed that there was avoidance for the some individuals in the severe PTSD symptomology group.

A repeated measures ANCOVA was performed by PTSD symptomology groups and covariance of the BTQ against the four emotional stimuli with the anterior-posterior (AP) body sway measurements. There was no significant interaction between PTSD symptomology and the AP body sway measurements. Means and standard errors for the AP body sway when covaried with BTQ are reported in Table 9. With the variance from the number of traumatic events is removed, the trend lines for the PTSD symptomology groups is clearer, as seen in Figure 4. When based solely on PTSD symptom level, the low and moderate groups respond with a reduction in body sway during the unpleasant stimulus. The reduced body sway combined with their reduced heart rate during the unpleasant response reveals their freezing response. The heart rate means are graphed in Figure 5. The severe PTSD symptomology group expresses freezing across all stimuli. The severe PTSD symptomology group had a slight reduction in heart rate during the unpleasant stimulus to qualify the freezing response. The means and standard errors for the heart rate measurements when covaried with BTQ are reported in Table 10.

As reported above, the heart rate measurements provided qualification for the freezing response when combined with the reduction in body sway. A repeated measures ANCOVA was performed by PTSD symptomology groups and covariance of the BTQ against the heart rate (per minute) for the four emotional stimuli. No significance was found between PTSD symptomology and the heart rate measurements. The heart rate trends are reported in Figure 5 where the severe PTSD symptomology group has a high heart rate across all emotional stimuli and a reduced heart rate during the emotional stimulus to qualify the freezing response. The heart rate reduction is most pronounced during the unpleasant stimulus by the moderate PTSD symptomology group to qualify the freezing response. Overall, the no to low PTSD symptomology group with a slight reduction in heart rate during the unpleasant response to qualify the freezing response.

The means and standard errors between the symptomology groups are reported in Table 10 and graphed in Figure 5.

Hypotheses 4

H4: An increase in traumatic events experienced by an individual will correspond with more freezing and avoidance response to stimuli.

A repeated measures ANOVA was performed by BTQ groups against the four emotional stimuli with the anterior-posterior (AP) body sway measurements. BTQ groups were based off the number of traumatic events reported by participants as reported in Table 14. The BTQ group based on zero to four traumatic events did not have any traumatic events that caused injury or danger to their lives. The BTQ group based on five to nine events had traumatic events that placed their lives in danger or had serious injuries or illness. The BTQ group was based on 10 to 13 traumatic events that included serious illness, injury, or life threatening situations. Homogeneity of the covariance matrices is assumed. While the main effect of the emotional stimuli on the AP body sway measurements is not statistically significant, Wilks' Lambda = .964, $F(3, 29) = .362, p = .781$. This effect, however, is qualified by a significant AP body sway X traumatic events group interaction, Wilks' Lambda = .589, $F(6, 58) = 2.927, p = .015$. The within-subject effect of the emotional stimuli on the AP body sway measurements was not significant but has sphericity assumed $F(3, 93) = .335, p = .800$. The AP body sway X traumatic events group interaction effect was significantly significant, sphericity assumed $F(6, 93) = 2.458, p = .030$. The higher-order quadratic component was significant [$F(2, 31) = 4.404, p = .021$] suggesting that the mean level of traumatic events exhibited a quadratic trend over the four measurements.

By grouping the BTQ by the number of traumatic events, a new trend in the heart rate data is revealed. When based on BTQ event groups, the low and middle groups respond with a reduction in body sway during the unpleasant stimulus. The reduced body sway combined with their reduced heart rate during the unpleasant response reveals their freezing response. The high BTQ group does not express freezing across all stimuli. The high BTQ group's pattern is completely different from the severe PTSD

symptomology group during the unpleasant stimulus. While this group, had a reduction in heart rate during the unpleasant stimulus, the AP body sway exhibited an avoidance response instead as the measurement increased compared to the other stimuli. The means and standard deviations for the BTQ groups are reported in Table 11. These new trends are graphed in Figure 6.

A repeated measures ANOVA was performed by BTQ groups against the four emotional stimuli with the heart rate (beats per minute) measurements. While no significance was found, there are some interesting trends to be reported from the means and standard deviations. The means and standard deviations are reported in Table 12 and graphed in Figure 7. The heart rates for all BTQ groups reduce during the unpleasant stimulus. The notable feature of the heart rate measurements is that the high BTQ group has a consistently higher heart rate when compared with the other BTQ groups.

Further analysis was performed to determine how trauma effects PTSD and the freezing response as measured by anterior-posterior (AP) body sway and heart rate (beats per minute). When mapping the BTQ trauma groups onto the PTSD symptomology groups, there are some interesting trends in the data. The means and standard deviations are reported in Table 13 and Table 14. The overall small sample size did not allow for significance of these findings but the trends are compelling. Within the low PTSD symptomology group, there are only participants who have under 9 traumatic events. For the BTQ 5 to 9 traumatic events group with low PTSD symptomology, there was high body sway observed for the pleasant stimulus and a reduced body sway for the unpleasant stimulus, as seen in Figure 8. Only those who had under 4 traumatic events had a reduction in heart rate to qualify the freezing response, as seen in Figure 11. The moderate PTSD symptomology group appeared to have the most common response between the low and middle BTQ groups with similar body sway observed across all stimuli, as seen in Figure 9. However, only the low BTQ group had a reduced heart rate to qualify the freezing response, as seen in Figure 12. The severe PTSD symptomology group reveals the difference that trauma plays in response to unpleasant stimulus. While the low and middle BTQ groups have a reduced body sway in response to the unpleasant stimulus, the high BTQ group has an opposite reaction with increased body sway as seen in Figure 10. The medium BTQ group had

a reduced heart rate in response to the unpleasant stimulus to qualify the freezing response, as seen in Figure 11. The high BTQ group also had a reduced heart rate to qualify the avoidant fear response, as seen in Figure 11.

Discussion

Military veteran students with varying levels of PTSD symptomology were studied with the following aims: (i) to replicate the freezing and avoidance responses to various stimuli, (ii) to identify an objective measure for these phenomena, and (iii) to determine how a freezing response correlates to the severity of PTSD symptomology and traumatic life events. Almost all of these aims were satisfied except for the avoidance response. My hypothesis that those with moderate PTSD would have an avoidant response to unpleasant stimulus was proven false when analyzing the trends across all trauma groups. However, there is some evidence that there could be a possible avoidance response by individuals with severe PTSD symptomology and a high number of traumatic events. While the results were not always significant, there are important trends that were revealed and confirmation that utilizing the stabilometer is an appropriate body sway measurement for freezing in response to various stimuli. Further study is needed to determine the significance of using the stabilometer for the measurement of avoidance. This tool has numerous measurements that are taken in micro measurements. The anterior-posterior body sway measurement seems to correspond with the avoidance response when viewed in relation to the freezing response. Both fear responses occurred with a reduction in heart rate during the unpleasant stimulus. A larger sample size is required to better test this measurement. Additional testing with multiple unpleasant stimulus might prove useful but could potentially harm already compromised individuals.

The current investigation examined the connections between PTSD, traumatic events and the freezing response with military veteran students. The examination of these factors leads to further understanding of how the transition from military life to civilian life at a university differs from other students. The stress from transitioning from military life to civilian life exacerbates PTSD symptomology. Even those participants who did not report they had previously been diagnosed with PTSD did have

moderate to severe symptom levels. Additionally, this research confirmed that deployment has lasting impacts on veterans overall health. Across all factors, deployment status plays a significant part in how veteran students perceive stress, experience anxiety, depression, and lack social connections that affect levels of loneliness.

Social isolation is increased when someone suffers from PTSD as the individual is on high alert to potential threats. Individuals with PTSD navigate the world differently than their peers. The trends from the body sway measurements revealed that those with more severe PTSD symptomology regard even neutral stimuli as a threat with an increased freezing response. When an individual is unable to process whether a person is a friend or a foe, it increases loneliness and isolation.

Hypotheses 1-3

While the hypotheses were not significant for an interaction between PTSD and body sway measurements, there are key findings that correspond with the current literature that supported the hypotheses. The overarching hypothesis was that individual differences in PTSD symptomology would result in differences in response to unpleasant stimulus. That as symptom severity increased, so too, would the freezing response also increase across all of the emotional stimuli regardless of valence. The trends of the data reveal that these hypotheses might be correct but the data lacks significance.

When the variance for traumatic life events is removed from the analysis, the low, moderate, and severe PTSD symptomology groups no longer had similar responses to the emotional stimuli. This allowed the true influence of PTSD symptomology on body sway to be revealed. As shown in Figure 2, the symptomology groups are no longer similar in their responses to the emotional stimuli. Participants with more severe PTSD symptomology exhibited a freezing response across all emotional stimuli. Participants with moderate PTSD symptomology exhibited more body sway in response to pleasant and neutral stimuli and a freezing response to unpleasant stimulus. Participants with no to low PTSD symptomology exhibited a slight freezing response to only the unpleasant stimulus and no reduction in body sway across the other emotional stimuli. These trends may be seen in Figure 2.

The use of the stabilometer to measure body sway and freezing response in humans is still in its infancy. While tonic immobility, avoidance, and the freezing response have been extensively studied in animals, the first successful human study was published in 2011 (Volchan et al., 2011). The field is still growing to determine if there are differences in types of tonic immobility, freezing, and avoidance responses in humans. The ability to test these responses is often limited to the type of stimuli that can be employed in the laboratory setting. Often the emotional stimuli used to elicit the freezing response are vastly limited to pictures, videos, loud sounds, or other not necessarily threatening stimuli. This study compares to the work of (Azevedo et al., 2005; Facchinetti et al., 2006; Hagenaars et al., 2012; Volchan et al., 2011; Volchan et al., 2017) in the use of stabilometer and its ability to measure the fight, flight, or freeze reactions to emotional pictorial stimuli. Overall, variations in responsiveness point to individual differences in symptom severity and number of traumatic events. Often the results are non-linear, with more defensive reactions and freezing response correlated to symptom severity and traumatic events, similar to this study's findings shown in Figure 10. Pictorial representations of how the body sway measures are reported are shown in Figures 14 and 15. These figures highlight differences between the freezing response (moderate PTSD symptomology group) and the avoidant response (severe PTSD symptomology group) when measured against the unpleasant stimulus. Also featured in these figures are natural body sway (moderate PTSD symptomology group) and the freezing response (severe PTSD symptomology group) when measured against the pleasant stimulus. When viewing these figures, the differences between the red and green circles provide greater insight into the individual differences in response.

PTSD did have a significant positive correlation with heart rate during all of the emotional stimuli as seen in Table 6. The severe PTSD symptomology group expressed higher heart rates than the other groups regardless of emotional stimuli. Additionally, those with more 10 or more traumatic events also had higher heart rates across all emotional stimuli. All PTSD symptomology groups expressed a deceleration of heart rate during the unpleasant stimulus, which confirmed the freezing response with the reduced body sway. This deceleration of the heart rate along with the reduced body sway, suggest that there was high arousal to

the unpleasantness of the pictorial stimuli. Research testing only heart rate in response to emotional stimuli, heart rate reactivity increased depending on symptom severity (McTeague et al., 2010). Prior research proposed a similar model of testing the fight, flight or freeze response with the hypothesis that individuals with multiple traumas would exhibit a deceleration of heart rate (Schaer & Elbert, 2010).

When a reduced heart rate occurs with the reduced body sway in response to the unpleasant stimulus, it qualifies the response and represents a defensive action, particularly a freezing response. When comparing the means between emotional stimuli, a reduced heart rate during the unpleasant stimulus confirms the freezing response. The means and SD for heart rate during the emotional stimuli are reported in Tables 8 and 10. Prior research concurs with this finding where a similar model was used (Azevedo et al., 2005; Hagenaaers et al., 2012; Volchan et al., 2011). Human biological indicators of the freezing are exhibited by a reduced body sway, tachycardia, and low heart rate variability (Azevedo et al., 2005; Facchinetti et al., 2006; Volchan et al., 2017). The freezing response is associated with trauma, stress, and the development of PTSD (Fragkaki et al., 2016). Individuals with PTSD that exhibit a freezing response typically have more severe PTSD symptoms that do not respond to traditional PTSD pharmacological treatment due to neural differences (Bovin et al., 2008; Fiszman et al., 2008; Fragkaki et al., 2016; Hagenaaers et al., 2012; Heidt et al., 2005; Lima et al., 2010; Marx et al., 2008).

Hypothesis 4

This hypothesis included the addition of the number of traumatic events that participants experienced when measuring freezing response to the emotional stimuli. Although the previous hypotheses did not achieve significant results, the number of traumatic events did reveal a significant interaction across the emotional stimuli. The key finding concerns the effect of traumatic events on the freezing response. Many of our participants have experienced numerous traumatic events in their lives. These have been reported in Table 15.

While this is not surprising, as trauma is a criterion for diagnosis with PTSD, the significance of traumatic events on the freezing response is important. For participants with up to 9 traumatic events, the

overall body sway is reduced in response to unpleasant stimulus with reduced heart rate to qualify the freezing response. However, the participants with more than 10 traumatic events, there is an increased body with a reduced heart rate to qualify an avoidant response as seen in Figures 6 and 7.

Further conclusions might be that as the number of traumatic events increase the more alert an individual becomes to dangerous stimuli in the environment. The mind becomes conditioned to be on high alert and braced to freeze in response to triggers. The severe PTSD symptomology group generalized the freezing across the emotional stimuli point to this conclusion. The inability to determine threat from neutral objects is present in the freezing response to all stimuli. Furthermore, the increased freezing response from the other PTSD symptomology groups may reveal that as the number of trauma events increase, the more likely a person is to freeze in response to unpleasant stimulus regardless of having PTSD. When the impact of trauma is not removed, individuals who do not suffer from PTSD do not freeze as much as others, as seen in Figure 4. Other studies have found similar decreased abilities to determine threat from everyday situations as the number of traumatic events experienced increases (Hagenaars et al., 2012). Further study across multiple stimuli is needed to explore the differences between freezing and avoidance responses.

Limitations

Limitations of the present study include an in-person laboratory setting that lacked neuroimaging possibilities and longitudinal design elements. Due to the numerous components of this study and its overall extended time commitment of more than one hour, participants may experience fatigue before the end of the study and affected the results. The sample is limited, as all of the participants were between the ages of 20-40, university students, and resided in Florida. The sample was further limited by the closing of the University of North Florida campus due to the coronavirus pandemic. This closure halted all research and ability to conduct studies in a laboratory setting.

Reliance on self-report methods may be a limitation even if physiological tasks are also included. Self-presentation biases are a concern for any survey on this population. PTSD research has difficult replication issues due to changes in diagnosis and self-report measures that continually change over the years.

PTSD research tends to be highly intensive in efforts with mixed results due to lack of specificity and numerous conditions that are comorbid with PTSD. The vast differences between how PTSD symptoms present themselves in both clinical and everyday settings leads to further confusion when attempting to understand the biology behind this disorder (Nemeroff et al., 2013).

Conclusion

Despite limitations, the present study will contribute important findings regarding the diagnosis and treatment of PTSD in military veterans by expanding the understanding of the freezing response. Further research via brain imaging to examine neurological activity would allow for greater understanding on how freezing and avoidance fear responses affects different areas of the brain during trauma. This would help to understand why SSRIs are not effective for treating PTSD with severe symptoms. Empirical evidence of the neurobiological connections for how individuals who suffer from PTSD react to threat would help improve treatment recommendations and efficacy. Military, police officers, and first responders often face threat repeatedly that has lasting impacts on the amygdala and the development of abnormal brain patterns (Fragkaki et al., 2016). Combat veterans, first responders, and victims of sexual abuse often report intense guilt, shame and failure for their freezing response to trauma (Fizman et al., 2008; Hagedaars et al., 2014; Lima et al., 2010; Marx et al., 2008; Rocha-Rego et al., 2009; Volchan et al; 2012). Both guilt and shame increase negative outcomes for both mental and physical health due to increased loneliness from social isolation. Future research should include self-report measurements to understand the levels of guilt and shame that their participants are facing in their life.

Understanding that the freezing response is just as valid as flight or fight in response to danger or trauma will assist in the therapeutic treatment of PTSD. Normalizing the freezing response will relieve veterans of the burden of guilt and shame that isolates them from their peers and families. This study and future research will help inform mental health professionals and the general public about how fear response is automatic and involuntary reaction to danger and trauma. Understanding the freezing response is important as it is linked with the development of PTSD and other trauma-related disorders.

Tables and Figures

Figure 1

Theoretical gene \times environment model for PTSD symptomology effects on freezing and avoidance fear response.

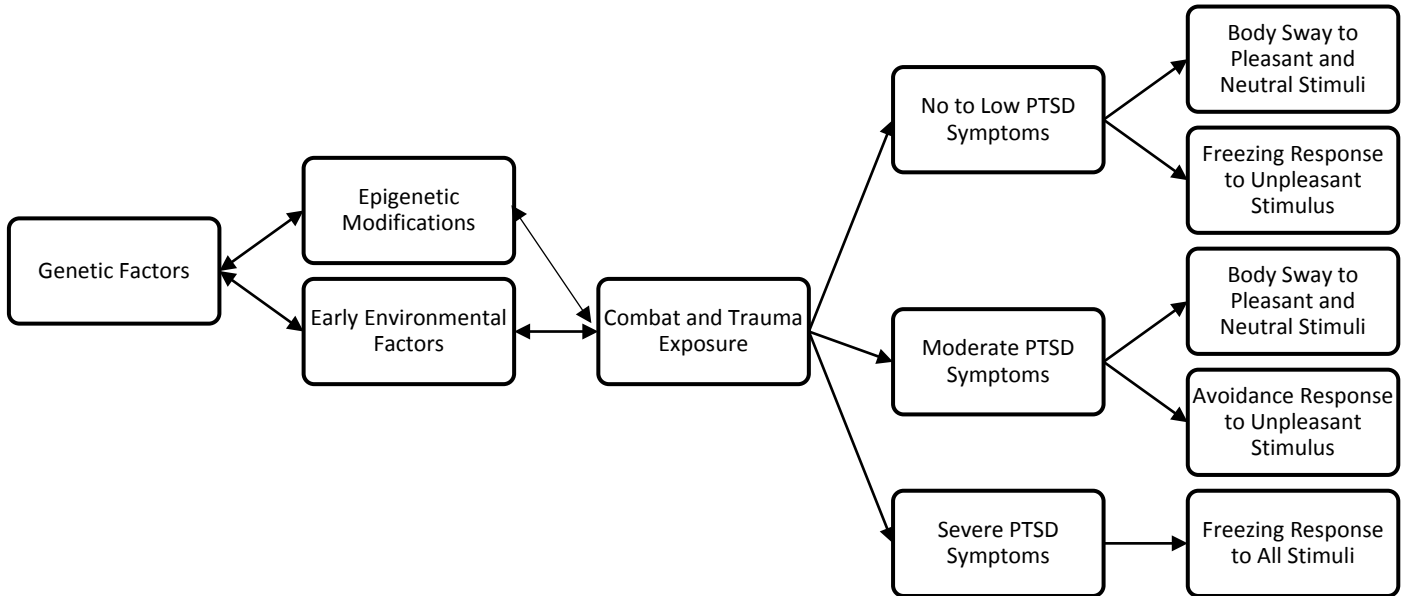


Table 1*Average PTSD, Trauma and Stress Constructs by Sociodemographic Variable.*

Variable	n	PTSD via PCL-5		Brief Trauma Questionnaire		Perceived Stress	
		M	SD	M	SD	M	SD
Gender	38	24.21	17.49	4.79	3.34	17.79	6.98
Female	18	27.33	18.21	5.00	3.79	18.50	7.07
Male	20	21.40	16.78	4.60	2.96	17.15	7.02
PTSD Diagnosis	38	24.21***	17.49	4.79***	3.34	17.79*	6.99
Yes	8	41.63	16.43	9.00	3.30	22.25	5.39
No	30	19.57	14.80	16.60	6.95	16.60	6.95
VA Disability Status	38	24.21**	17.49	4.79	3.34	17.79**	6.98
Yes	22	30.68	16.10	5.55	3.19	20.50	4.97
No	16	15.31	15.70	3.75	3.36	14.06	7.75
Service Branch	38	24.21	17.49	4.79	3.34	17.79	6.98
Air Force	5	10.80	7.92	3.00	2.55	12.80	6.18
Army	9	35.00	21.72	5.89	4.31	19.00	8.02
Navy	18	22.33	13.99	4.06	2.60	18.67	7.19
Marine Corps	5	26.60	21.57	6.80	3.90	16.80	4.82
Other	1	16.00		7.00		21.00	
Deployment	38	24.21*	17.49	4.79	3.34	17.78*	6.98
Yes	26	28.42	16.70	5.50	3.11	19.65	6.17
No	12	15.08	16.21	3.25	3.41	13.75	7.17
Location							
Combat Zone	23	30.74	15.94	5.91	3.04	20.39	5.31
Peace-keeping Mission	7	15.43	14.85	3.57	2.37	13.43	5.19
Gross Income	38	24.21*	17.49	4.79**	3.34	17.79	6.98
Less than \$10,000	1	62.00		13.00		23.00	
\$10-50,000	23	20.87	14.75	3.83	2.35	17.26	6.55
Greater than \$50,000	14	27.00	19.09	5.79	3.81	18.29	7.96

Note: * p<.05, **p<.01, ***p<.001

Table 2*Average Mental Health Constructs by Sociodemographic Variables*

Variable	<i>Depression - PHQ-8</i>			<i>Generalized Anxiety Disorder</i>	
	n	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Gender	38	8.71	5.66	9.11	5.72
Female	18	10.17	5.91	10.56	6.20
Male	20	7.40	5.23	7.80	5.04
PTSD Diagnosis	38	8.71***	5.66	9.11**	5.72
Yes	8	14.50	4.24	14.25	4.46
No	30	7.17	4.98	7.73	5.26
VA Disability Status	38	8.71**	5.67	9.11**	5.72
Yes	22	10.95	4.90	11.27	4.85
No	16	5.63	5.28	6.13	5.60
Service Branch	38	8.71	5.66	9.11	5.72
Air Force	5	4.20	2.39	4.20	3.49
Army	9	8.89	5.73	9.89	5.44
Navy	18	9.22	5.95	9.78	6.21
Marine Corps	5	11.00	6.44	9.80	4.51
Other	1	9.00		11.00	
Deployment	38	8.71*	5.66	9.11*	5.72
Yes	26	10.23	5.50	10.35	5.11
No	12	5.42	4.66	6.42	6.26
Location					
Combat Zone	23	10.74	5.39	10.83	4.89
Peace-keeping Mission	7	5.29	5.15	6.71	5.15
Gross Income	38	8.71	5.66	9.11	5.72
Less than \$10,000	1	19.00		16.00	
\$10-50,000	23	8.22	4.88	8.83	5.58
Greater than \$50,000	14	8.79	6.51	9.07	6.04

Note: * $p < .05$, ** $p < .01$, *** $p < .001$

Table 3*Average Physical Health Constructs by Sociodemographic Variables*

Variable	<i>Physical Health Questionnaire</i>			<i>Medical Outcomes Study SF-36</i>	
	n	M	SD	M	SD
Gender	38	37.79	13.06	51.95	25.04
Female	18	40.28	14.35	43.96	23.54
Male	20	35.55	11.70	59.14	24.70
PTSD Diagnosis	38	37.79*	13.06	51.95***	25.04
Yes	8	48.00	13.28	25.52	13.43
No	30	35.07	11.78	59.00	22.63
VA Disability Status	38	37.79	13.06	51.95*	25.04
Yes	22	40.86	13.08	43.90	21.37
No	16	33.56	12.18	63.01	26.11
Service Branch	38	37.79	13.06	51.95	25.04
Air Force	5	28.20	6.61	70.00	20.80
Army	9	39.67	12.67	43.78	27.25
Navy	18	37.94	13.29	53.52	25.71
Marine Corps	5	43.80	17.14	46.59	19.60
Other	1	36.00		33.75	
Deployment	38	37.79**	13.06	51.95	25.04
Yes	26	41.69	13.15	46.76	22.74
No	12	29.33	8.17	63.20	27.04
Location					
Combat Zone	23	42.04	13.31	43.21	21.09
Peace-keeping Mission	7	34.57	13.33	72.29	12.97
Gross Income	38	37.79*	13.06	51.95	25.04
Less than \$10,000	1	66.00		13.13	
\$10-50,000	23	34.70	11.35	57.78	21.70
Greater than \$50,000	14	40.86	13.39	45.14	27.58

Note: * p<.05, **p<.01, ***p<.001

Table 4*Average Social Constructs by Sociodemographic Variables*

Variable	<i>Social Connectedness Scale</i>			<i>Loneliness Scale</i>	
	n	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Gender	38	77.16	22.15	18.18	5.60
Female	18	74.17	22.10	18.89	5.67
Male	20	79.85	22.42	17.55	5.61
PTSD Diagnosis	38	77.16**	22.15	18.18*	5.60
Yes	8	58.75	18.53	22.00	4.24
No	30	82.07	20.61	17.17	5.53
VA Disability Status	38	77.16**	22.15	18.18**	5.60
Yes	22	69.91	16.00	20.45	4.69
No	16	87.13	25.87	15.06	5.36
Service Branch	38	77.16	22.15	18.18	5.60
Air Force	5	93.20	19.93	15.00	5.52
Army	9	75.89	27.15	17.78	6.10
Navy	18	73.89	21.70	19.28	5.83
Marine Corps	5	77.80	15.83	17.60	4.51
Other	1	64.00		21.00	
Deployment	38	77.16**	22.15	18.18*	5.60
Yes	26	70.81	17.98	19.65	4.80
No	12	90.92	24.79	15.00	6.09
Location					
Combat Zone	23	67.61	15.54	20.17	4.51
Peace-keeping Mission	7	86.00	17.32	15.71	4.27
Gross Income	38	77.16	22.15	18.18	5.60
Less than \$10,000	1	77.00		20.00	
\$10-50,000	23	79.65	19.27	17.70	5.23
Greater than \$50,000	14	73.07	27.20	18.86	6.47

Note: * $p < .05$, ** $p < .01$

Table 5

Pearson Correlations on PTSD, Trauma, Stress, Psychological, Social and General Health Outcome Variables

Variable	1	2	3	4	5	6	7	8	9	10
1 PTSD via PCL-5	1									
2 PSTD Symptomology Groups	.904**	1								
3 Brief Trauma Questionnaire	.573**	.420**	1							
4 Perceived Stress	.700**	.589**	.432**	1						
5 Depression - PHQ-8	.805**	.722**	.556**	.801**	1					
6 Generalized Anxiety Disorder	.745**	.677**	.515**	.807**	.899**	1				
7 Physical Health Questionnaire	.689**	.642**	.305	.412*	.603**	.507**	1			
8 Medical Outcomes Study	-.695**	-.602**	-.622**	-.787**	-.742**	-.692**	-.427**	1		
9 Social Connectedness Scale	-.646**	-.614**	-.493**	-.834**	-.768**	-.756**	-.377*	.751**	1	
10 Loneliness Scale	.593**	.563**	.482**	.846**	.769**	.753**	.289	-.709**	-.895**	1

Note: * p<.05, **p<.01

Table 6

Pearson Correlations on PTSD Diagnosis, PCL Score, Trauma, and Heart Rate During Emotional Stimuli.

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1 PTSD Diagnosis - Military Related	1														
2 PTSD via PCL-5 Brief Trauma Questionnaire	.521**	1													
3 Heart Rate (beats per min) Eyes Open	.660**	.573**	1												
4 Heart Rate (beats per min) Pleasant	.274	.362	.272	1											
5 Heart Rate (beats per min) Unpleasant	.320	.413*	.336	.945**	1										
6 Heart Rate (beats per min) Neutral	.292	.420*	.319	.947**	.964**	1									
7 A-P Exc (cm) During Eyes Open	.277	.373*	.275	.964**	.988**	.962**	1								
8 L-R Exc (cm) During Eyes Open	-.354*	-.272	-.267	-.207	-.205	-.168	-.203	1							
9 A-P Exc (cm) During Pleasant	-.300	-.160	-.278	-.255	-.302	-.210	-.288	.535**	1						
10 L-R Exc (cm) During Pleasant	.175	-.216	.202	-.198	-.134	-.091	-.196	.147	.109	1					
11 A-P Exc (cm) During Unpleasant	.156	-.128	.232	-.135	-.047	.006	-.107	-.140	.127	.641**	1				
12 L-R Exc (cm) During Unpleasant	.363*	.189	.370*	.217	.276	.265	.249	.095	.119	.179	.208	1			
13 A-P Exc (cm) During Neutral	.143	.036	.141	.102	.051	.068	.037	-.107	.100	.021	.106	.692**	1		
14 L-R Exc (cm) During Neutral	-.235	-.308	-.116	.055	.018	.100	.020	.328	.370*	.172	-.009	.310	.270	1	
15 A-P Exc (cm) During Neutral	-.124	-.188	.005	-.053	-.131	-.086	-.123	.098	.357*	.129	.070	.254	.526**	.737**	1

Note: * p<.05, **p<.01

Table 7

PTSD Symptomology Groups' Response to Emotional Stimuli via Anterior-Posterior Body Sway (cm)

PTSD Group	<i>n</i>	<i>Eyes Open</i>		<i>Pleasant</i>		<i>Unpleasant</i>		<i>Neutral</i>	
		<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
No to Low (0-14)	11	7.06	1.19	5.67	1.02	4.82	1.05	6.55	1.12
Moderate (15-27)	12	6.55	1.14	6.74	0.98	4.38	1.00	6.16	1.07
Severe (28-62)	11	5.08	1.19	4.44	1.02	4.28	1.05	3.31	1.12
Total	34	6.24	3.92	5.65	3.43	4.49	3.37	5.36	3.88

Table 8

PTSD Symptomology Groups' Response to Emotional Stimuli via Heart Rate (beats per minute)

PTSD Group	<i>n</i>	<i>Eyes Open</i>		<i>Pleasant</i>		<i>Unpleasant</i>		<i>Neutral</i>	
		<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
No to Low (0-14)	10	81.58	11.99	79.01	11.94	77.14	12.93	80.52	10.75
Moderate (15-27)	9	77.11	11.81	76.32	12.32	75.67	11.44	76.33	11.75
Severe (28-62)	10	88.29	12.73	87.34	14.70	86.75	13.72	88.08	13.43
Total	29	82.50	12.64	81.05	13.47	80.00	13.30	81.82	12.60

Table 9

PTSD Symptomology Groups' (covaried with BTQ) Response to Emotional Stimuli via Anterior-Posterior Body Sway (cm)

PTSD Group	<i>n</i>	<i>Eyes Open</i>		<i>Pleasant</i>		<i>Unpleasant</i>		<i>Neutral</i>	
		<i>M</i>	<i>SDE</i>	<i>M</i>	<i>SDE</i>	<i>M</i>	<i>SDE</i>	<i>M</i>	<i>SDE</i>
No to Low (0-14)	11	6.60	1.27	6.38	1.05	5.76	1.03	6.62	1.21
Moderate (15-27)	12	6.75	1.16	6.44	0.95	3.98	0.93	6.13	1.10
Severe (28-62)	11	5.33	1.22	4.05	1.00	3.77	0.98	3.27	1.16
Total	34	6.23	1.22	5.63	1.00	4.51	0.98	5.34	1.16

Table 10

PTSD Symptomology Groups' (covaried with BTQ) Response to Emotional Stimuli via Heart Rate (beats per minute)

PTSD Group	<i>n</i>	<i>Eyes Open</i>		<i>Pleasant</i>		<i>Unpleasant</i>		<i>Neutral</i>	
		<i>M</i>	<i>SDE</i>	<i>M</i>	<i>SDE</i>	<i>M</i>	<i>SDE</i>	<i>M</i>	<i>SDE</i>
No to Low (0-14)	10	84.56	4.20	82.28	4.49	80.26	4.41	83.09	4.20
Moderate (15-27)	9	75.62	4.35	74.70	4.35	74.12	4.26	75.05	4.06
Severe (28-62)	10	88.29	12.73	87.34	14.70	86.75	13.72	88.08	13.43
Total	29	82.50	12.64	81.05	13.47	80.00	13.30	81.82	12.60

Figure 2

PTSD Symptomology Groups' Response to Emotional Stimuli via Anterior-Posterior Body Sway



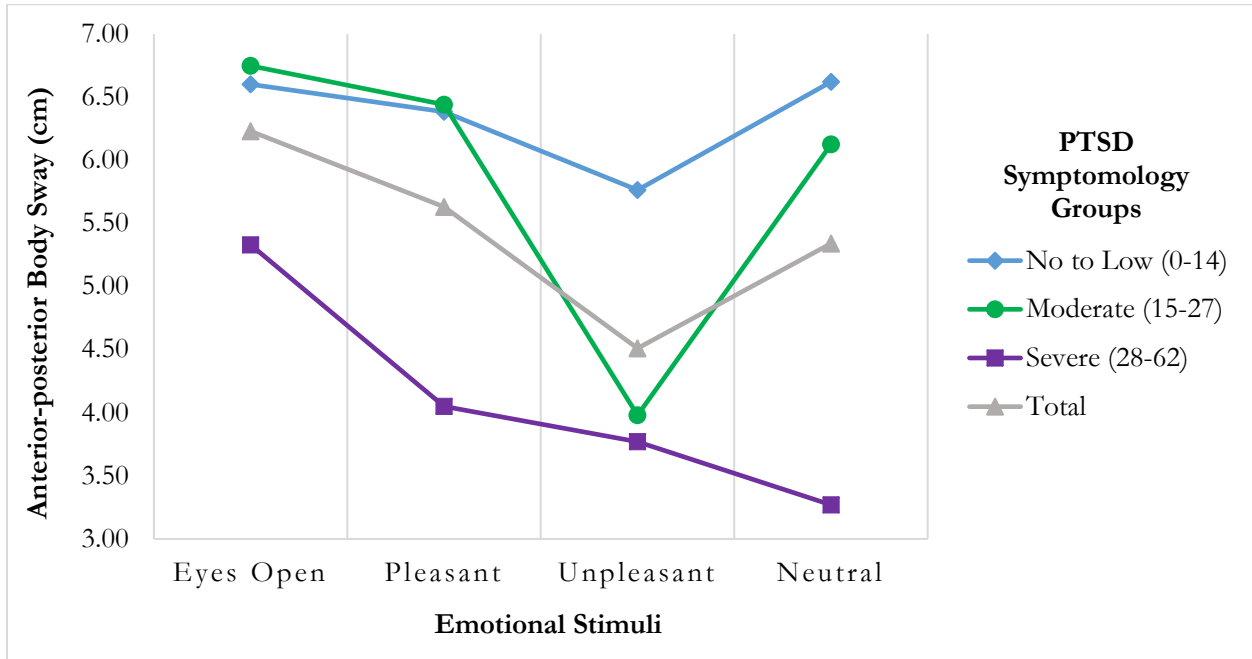
Figure 3

PTSD Symptomology Groups' Response to Emotional Stimuli via Heart Rate (beats per minute)



Figure 4

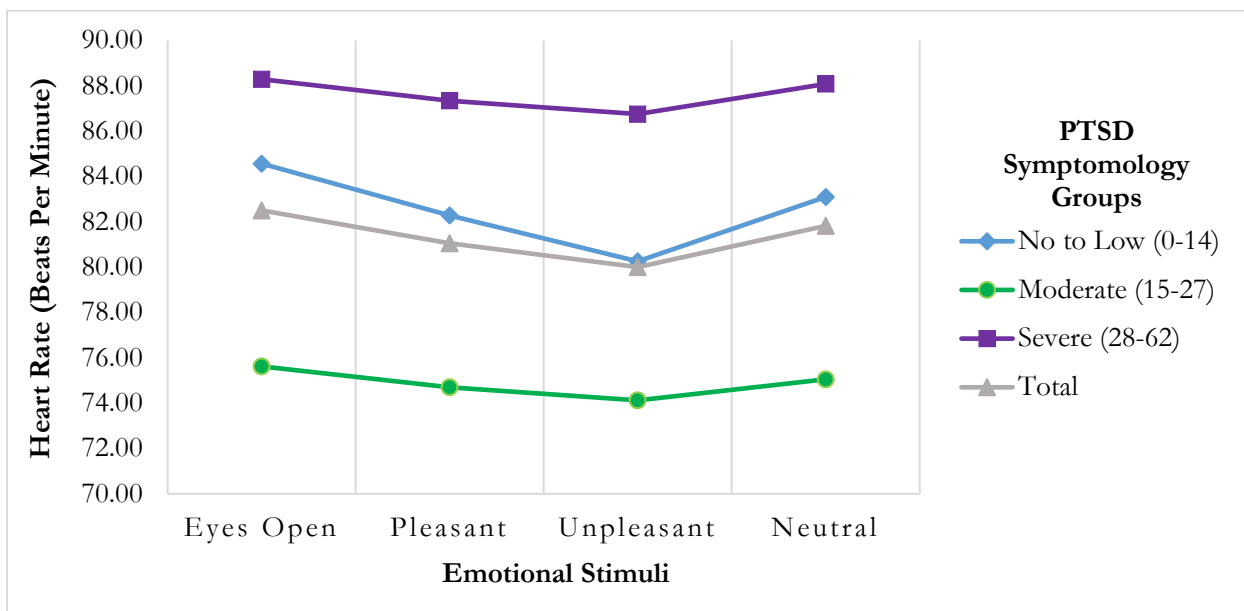
PTSD Symptomology Groups' (covaried with BTQ) Response to Emotional Stimuli via Anterior-Posterior Body Sway (cm)



Note: Covariates appearing in the model are evaluated at the following values: Sum of BTQ answers = 4.62.

Figure 5

PTSD Symptomology Groups' (covaried with BTQ) Response to Emotional Stimuli via Heart Rate (beats per minute)



Note: Covariates appearing in the model are evaluated at the following values: Sum of BTQ answers = 4.48.

Table 11

BTQ Groups' Response to Emotional Stimuli via Anterior-Posterior Body Sway (cm)

BTQ Group	<i>n</i>	<i>Eyes Open</i>		<i>Pleasant</i>		<i>Unpleasant</i>		<i>Neutral</i>	
		<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
0 to 4 Events	19	6.59	4.41	5.05	3.34	4.06	2.49	5.09	4.19
5 to 9 Events	12	6.55	3.29	6.64	3.63	4.00	3.34	6.32	3.67
10 to 13 Events	3	2.81	0.54	5.53	3.48	9.17	5.83	3.25	1.68
Total	34	6.24	3.92	5.65	3.43	4.49	3.37	5.36	3.88

Table 12

BTQ Groups' Response to Emotional Stimuli via Heart Rate (beats per minute)

BTQ Group	<i>n</i>	<i>Eyes Open</i>		<i>Pleasant</i>		<i>Unpleasant</i>		<i>Neutral</i>	
		<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
0 to 4 Events	16	81.35	12.20	79.51	12.74	77.91	12.87	80.82	11.96
5 to 9 Events	10	79.20	11.47	77.83	12.26	77.79	11.64	78.43	11.91
10 to 13 Events	3	99.63	4.75	100.03	6.00	98.48	7.19	98.53	5.16
Total	29	82.50	12.64	81.05	13.47	80.00	13.30	81.82	12.60

Figure 6

BTQ Groups' Response to Emotional Stimuli via Anterior-Posterior Body Sway (cm)

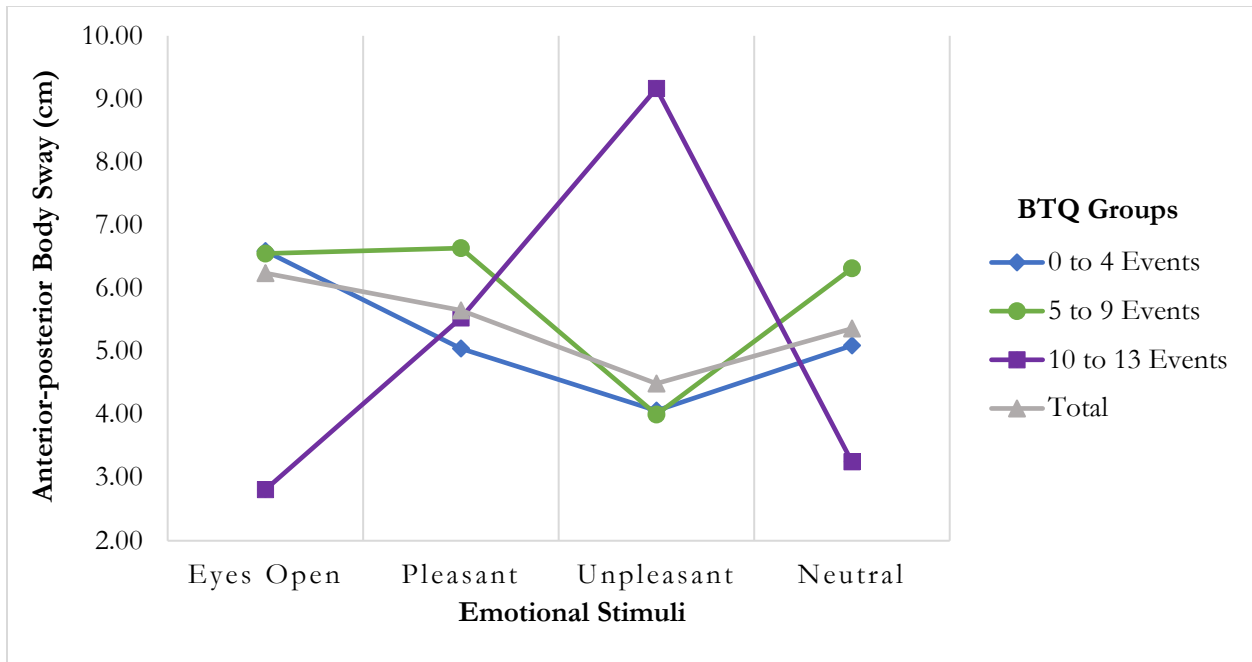


Figure 7

BTQ Groups' Response to Emotional Stimuli via Heart Rate (beats per minute)

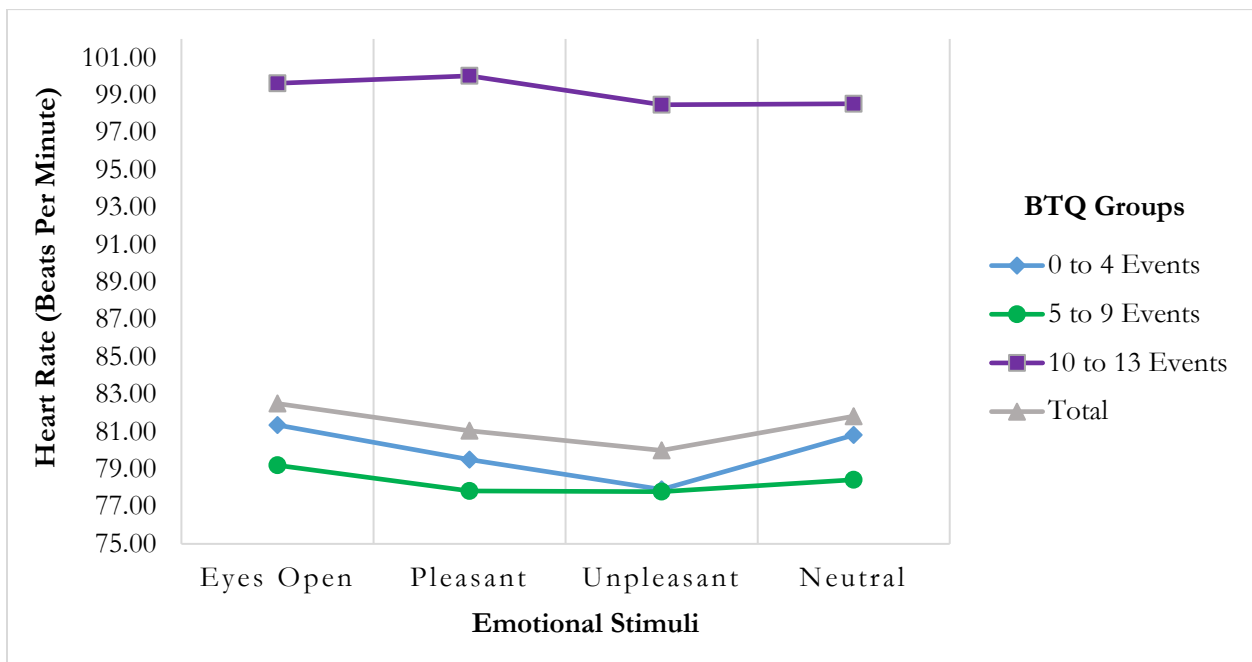


Table 13*PTSD Symptomology Groups' Response split by BTQ Groups to Emotional Stimuli via Anterior-Posterior Body Sway (cm)*

<i>PTSD Symptomology Group</i>	<i>BTQ Group</i>	<i>n</i>	<i>Eyes Open</i>		<i>Pleasant</i>		<i>Unpleasant</i>		<i>Neutral</i>	
			<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Low (0-14)	No to 4 Trauma Events	9	7.61	4.59	4.91	3.39	5.26	2.45	6.74	4.50
	5 to 9 Trauma Events	2	4.61	1.11	9.12	2.73	2.85	0.05	5.67	1.91
	Total	11	7.06	4.29	5.67	3.58	4.82	2.40	6.55	4.09
Moderate (15-27)	No to 4 Trauma Events	5	6.78	5.20	5.92	3.43	3.10	2.27	4.71	4.48
	5 to 9 Trauma Events	7	6.39	2.69	7.33	3.81	5.30	3.93	7.19	4.56
	Total	12	6.55	3.72	6.74	3.57	4.38	3.40	6.16	4.50
Severe (28-62)	No to 4 Trauma Events	5	4.58	3.28	4.43	3.75	2.87	2.16	2.51	1.97
	5 to 9 Trauma Events	3	8.20	5.41	3.36	1.15	1.74	0.38	4.72	1.76
	10 to 13 Trauma Events	3	2.81	0.54	5.53	3.48	9.17	5.83	3.25	1.68
	Total	11	5.08	3.85	4.44	3.00	4.28	4.33	3.31	1.91
Total	No to 4 Trauma Events	19	6.59	4.41	5.05	3.34	4.06	2.49	5.09	4.19
	5 to 9 Trauma Events	12	6.55	3.29	6.64	3.63	4.00	3.34	6.32	3.67
	10 to 13 Trauma Events	3	2.81	0.54	5.53	3.48	9.17	5.83	3.25	1.68
	Total	34	6.24	3.92	5.65	3.43	4.49	3.37	5.36	3.88

Figure 8

Low PTSD Symptomology Group's Response Split by BTQ Groups to Emotional Stimuli via Anterior-Posterior Body Sway (cm)

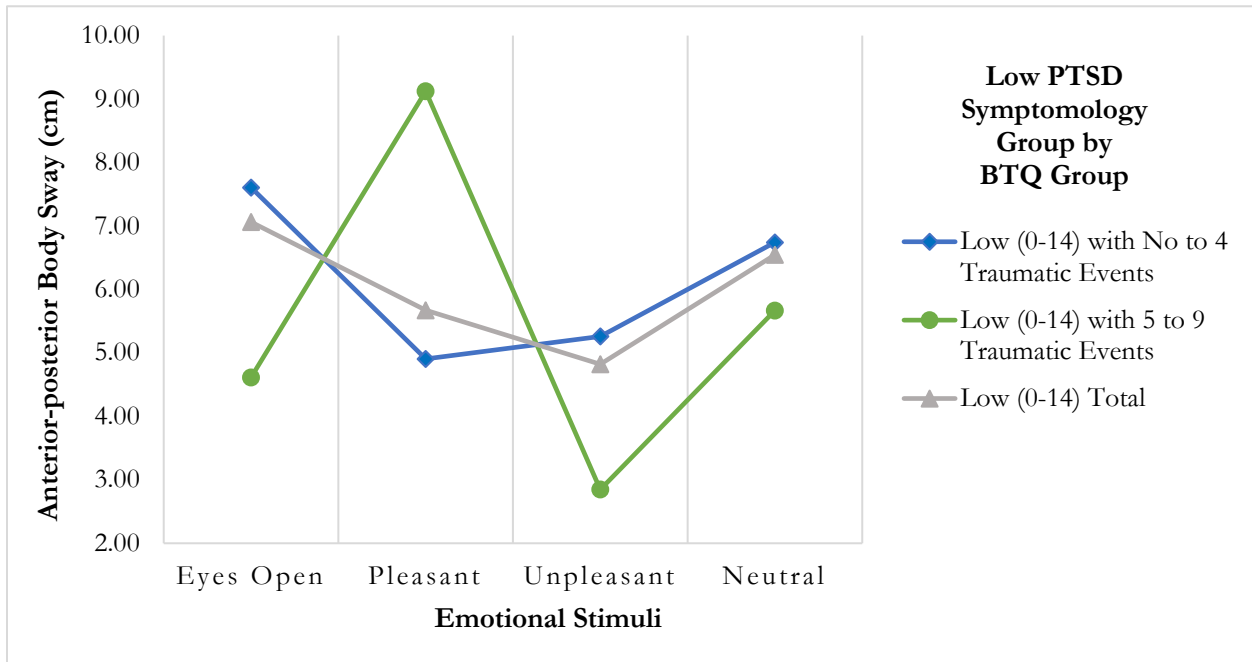


Figure 9

Moderate PTSD Symptomology Group's Response Split by BTQ Groups to Emotional Stimuli via Anterior-Posterior Body Sway (cm)

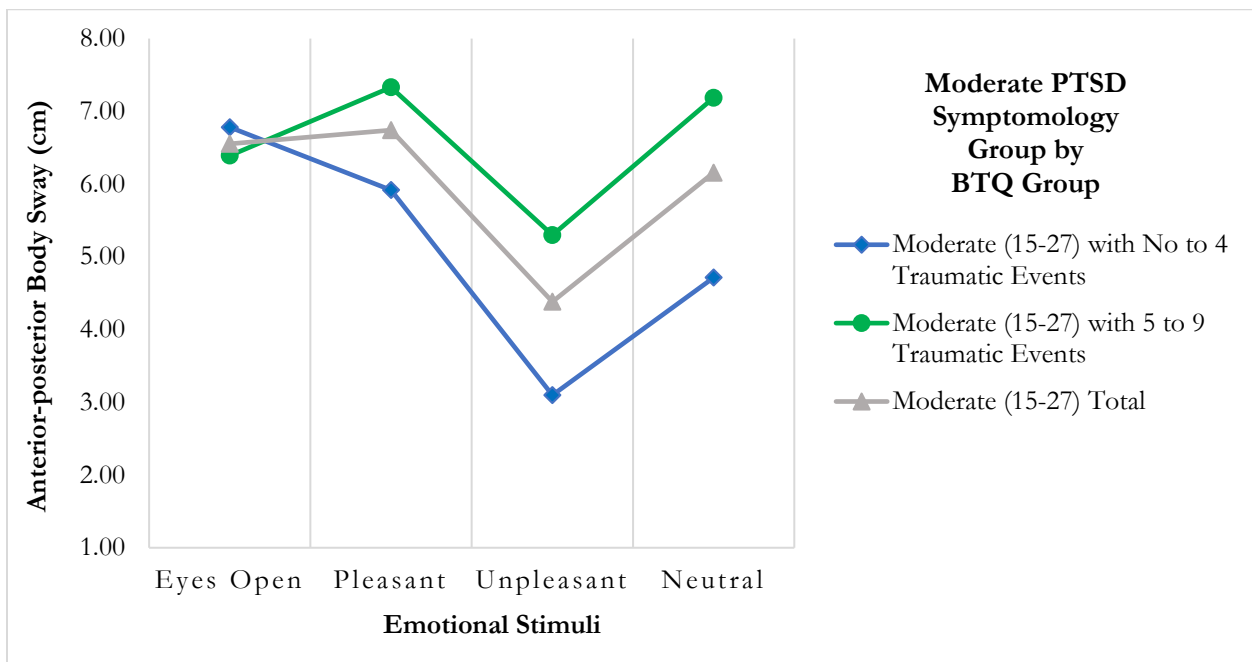


Figure 10

Severe PTSD Symptomology Group's Response Split by BTQ Groups to Emotional Stimuli via Anterior-Posterior Body Sway (cm)

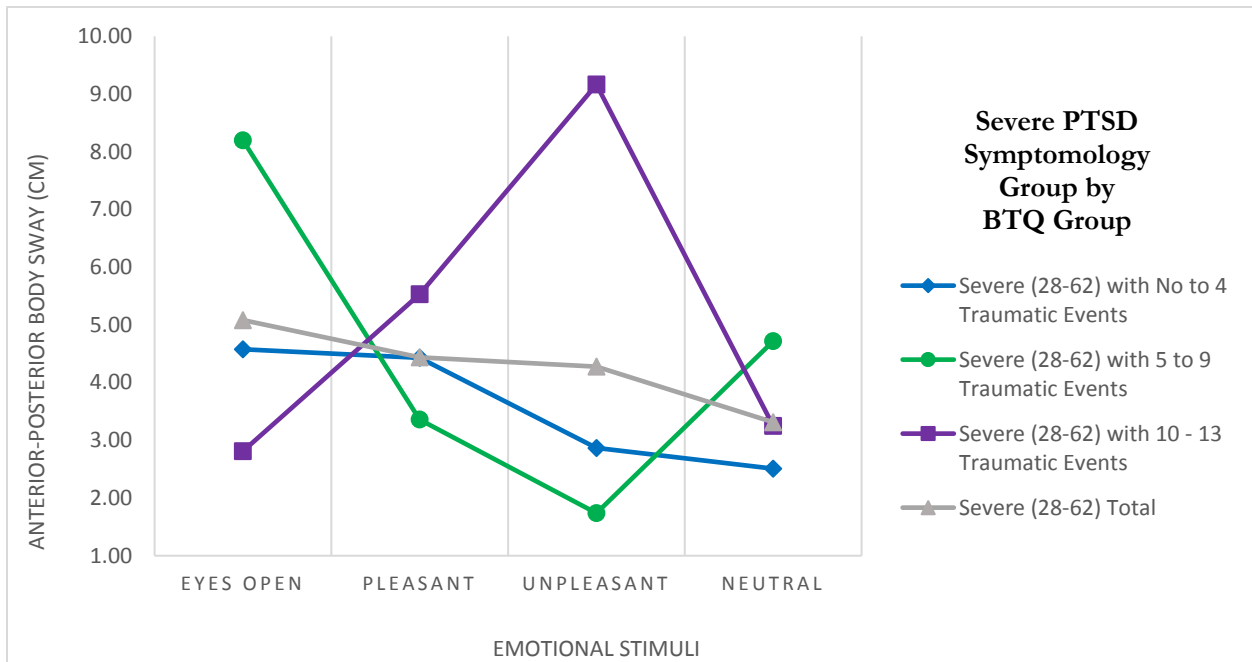


Table 14*PTSD Symptomology Groups' Response split by BTQ Groups to Emotional Stimuli via Heart Rate (beats per minute)*

<i>PTSD Symptomology Groups</i>		<i>Eyes Open</i>		<i>Pleasant</i>		<i>Unpleasant</i>		<i>Neutral</i>		
<i>PTSD Symptomology Groups</i>	<i>BTQ Groups</i>	<i>n</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Low (0-14)	No to 4 Trauma Events	9.00	80.39	12.08	77.79	11.98	75.59	12.69	79.48	10.85
	5 to 9 Trauma Events	1.00	92.24		90.03		91.07		89.89	
	Total	10.00	81.58	11.99	79.01	11.94	77.13	12.93	80.52	10.75
Moderate (15-27)	No to 4 Trauma Events	3.00	75.17	8.41	77.09	4.94	73.72	3.14	76.53	4.65
	5 to 9 Trauma Events	6.00	78.07	13.84	75.94	15.25	76.65	14.22	76.23	14.56
	Total	9.00	77.11	11.81	76.32	12.32	75.67	11.44	76.33	11.75
Severe (28-62)	No to 4 Trauma Events	4.00	88.16	14.33	85.19	18.83	86.28	16.28	87.04	17.66
	5 to 9 Trauma Events	3.00	77.11	4.25	77.53	3.18	75.65	2.42	79.00	5.20
	with 10 - 13	3.00	99.63	4.75	100.03	6.00	98.48	7.19	98.53	5.16
	Total	10.00	88.28	12.73	87.34	14.70	86.75	13.72	88.08	13.43
Total	No to 4 Trauma Events	16.00	81.35	12.20	79.51	12.74	77.91	12.87	80.82	11.96
	5 to 9 Trauma Events	10.00	79.20	11.47	77.83	12.26	77.79	11.64	78.43	11.91
	with 10 - 13	3.00	99.63	4.75	100.03	6.00	98.48	7.19	98.53	5.16
	Total	29.00	82.50	12.64	81.05	13.47	80.00	13.30	81.82	12.60

Figure 11

Low PTSD Symptomology Group's Response Split by BTQ Groups to Emotional Stimuli via Heart Rate (beats per minute)

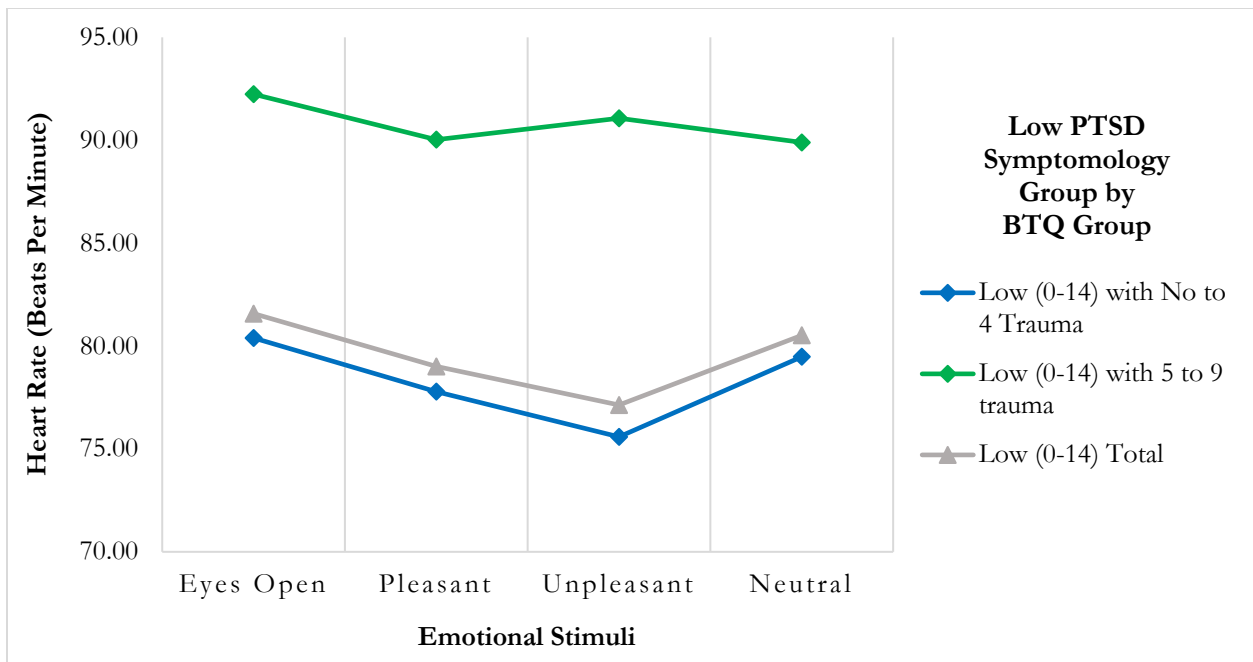


Figure 12

Moderate PTSD Symptomology Group's Response Split by BTQ Groups to Emotional Stimuli via Heart Rate (beats per minute)

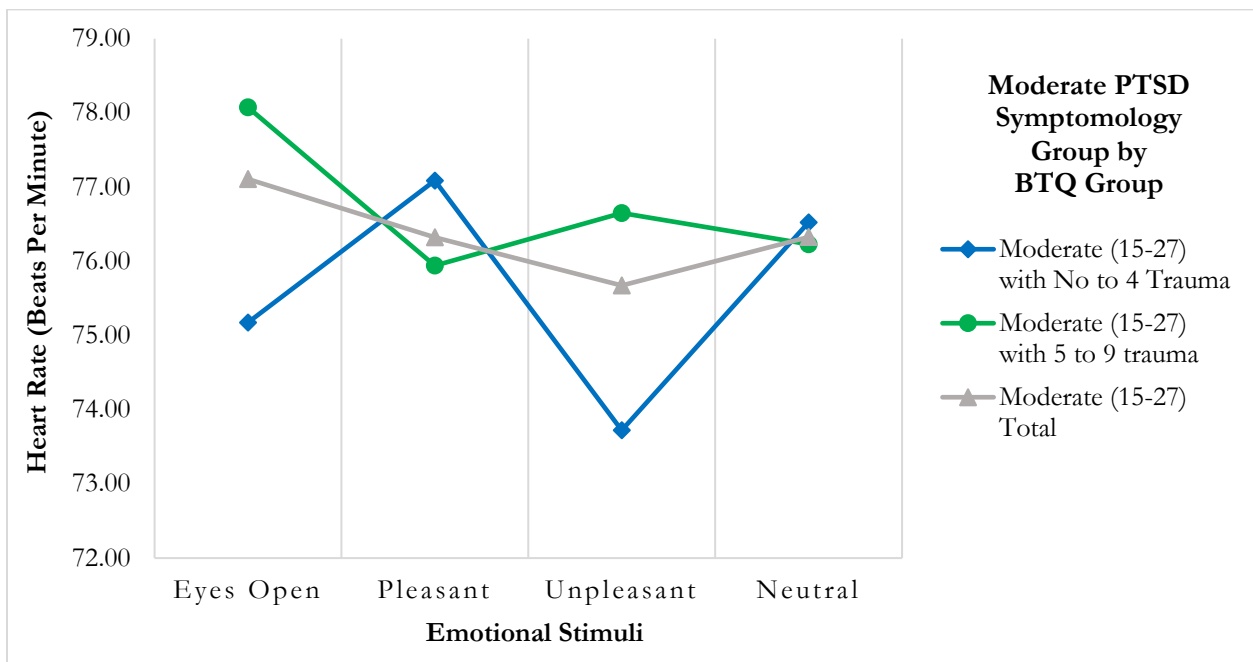


Figure 13

Severe PTSD Symptomology Group's Response Split by BTQ Groups to Emotional Stimuli via Heart Rate (beats per minute)

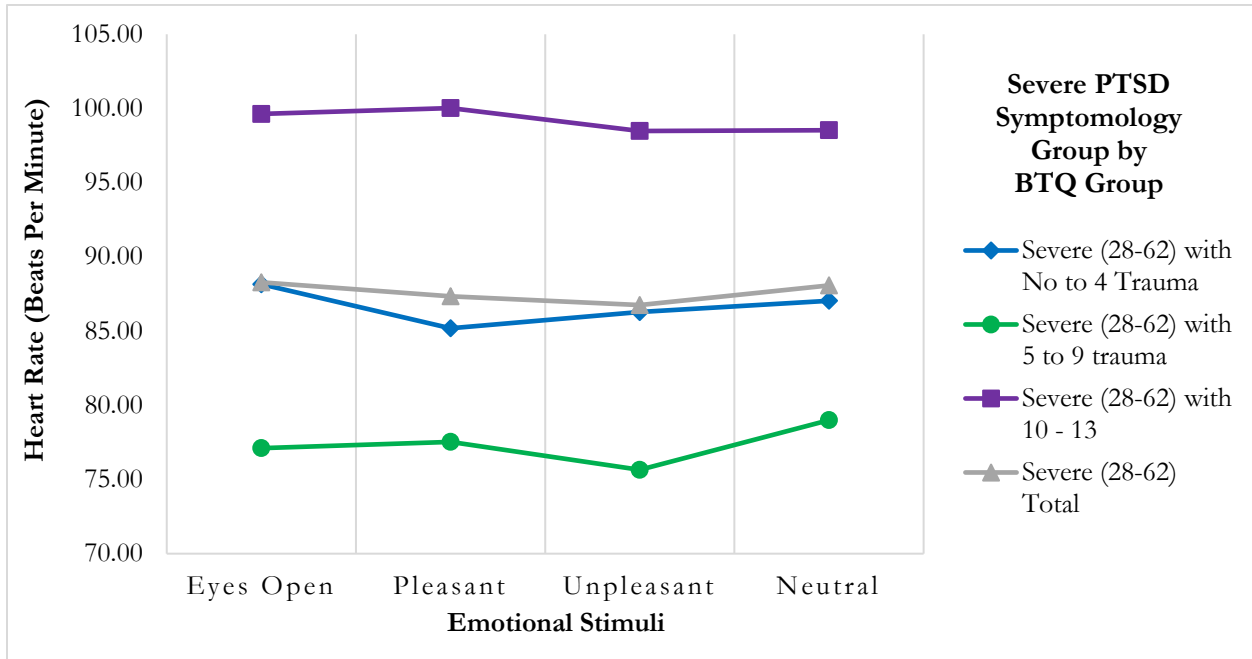
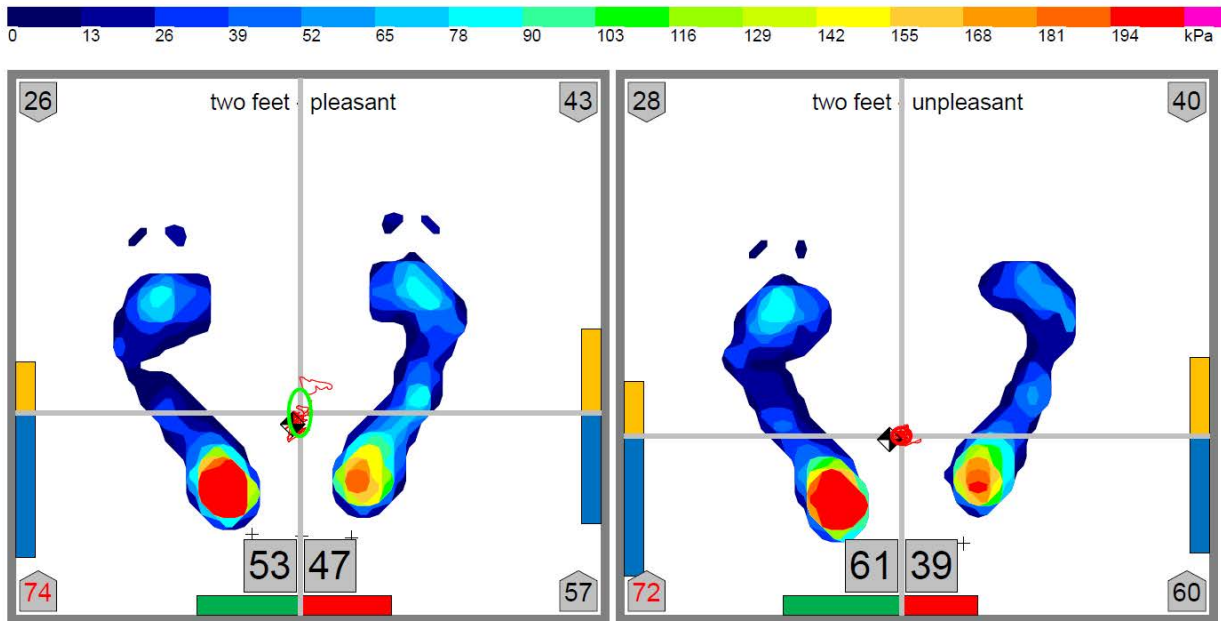


Figure 14

Pictorial Representation of Body Sway, Freezing, and Avoidance Responses to Pleasant and Unpleasant Stimuli by Moderate and Severe PTSD symptomology Groups

Moderate PTSD Symptomology Group



Severe PTSD Symptomology Group

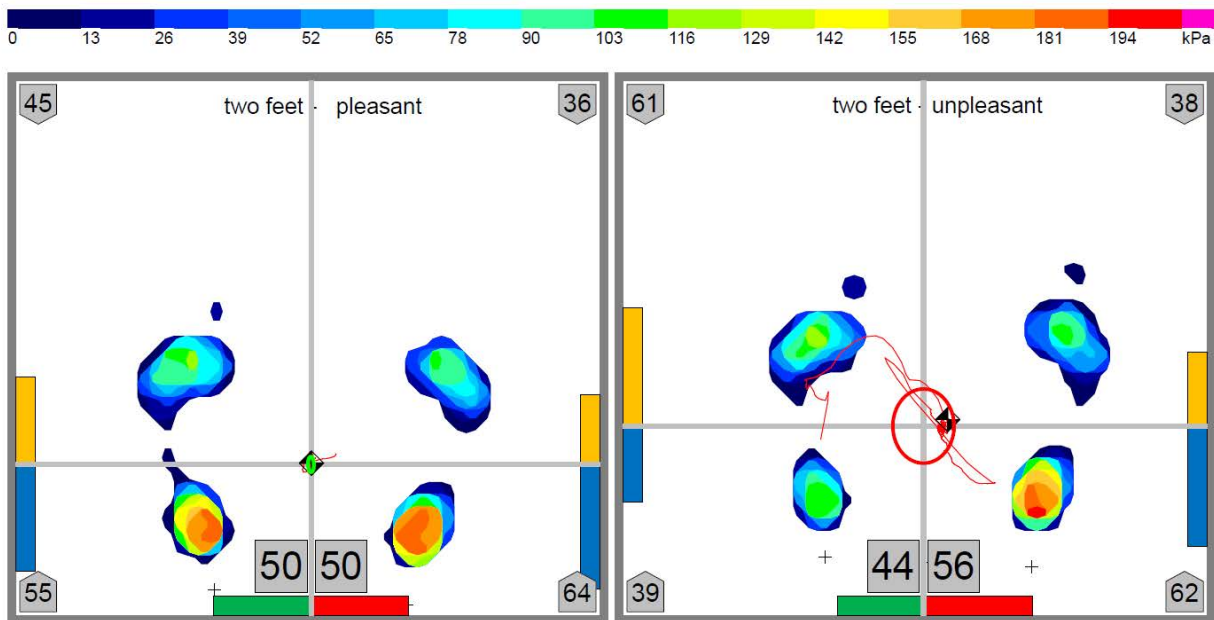
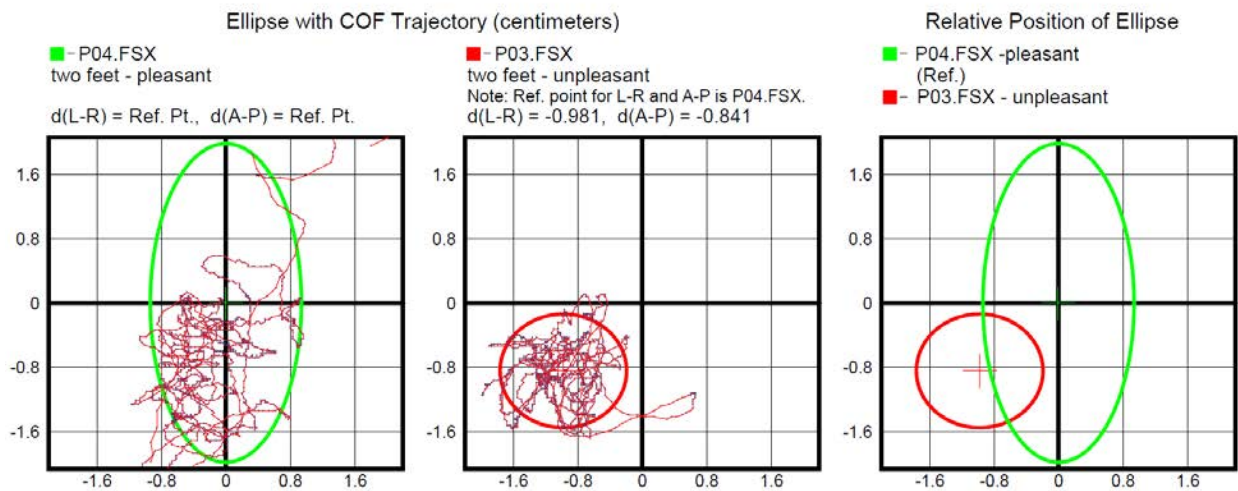


Figure 15

Graphic Representation of Body Sway, Freezing, and Avoidance Responses to Pleasant and Unpleasant Stimuli by Moderate and Severe PTSD symptomology Groups

Moderate PTSD Symptomology Group



Severe PTSD Symptomology Group

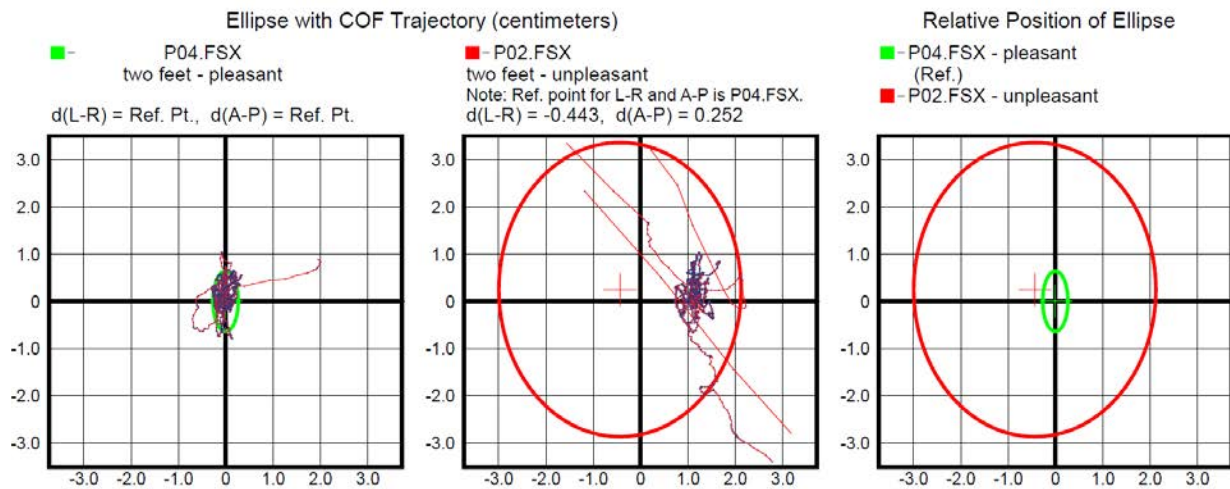


Table 15*Occurrence of Trauma (BTQ) for Participants*

<i>Trauma Type</i>	<i>n</i>	<i>M</i>	<i>SD</i>
Served in a war zone or in noncombat job with exposure to war-related casualties	14	0.37	0.49
Life threatened or in danger	4	0.71	0.47
Seriously injured	1	0.07	0.27
Serious car accident or other severe accident	11	0.30	0.46
Life threatened or in danger	5	0.45	0.52
Seriously injured	2	0.18	0.40
Major natural or technological disaster (fire, tornado, hurricane, flood, earthquake, or chemical spill)	22	0.58	0.50
Life threatened or in danger	9	0.41	0.50
Seriously injured	16	0.00	0.00
Life-threatening illness	4	0.11	0.31
Life threatened or in danger	4	1.00	0.00
Physical Child Abuse	13	0.34	0.48
Life threatened or in danger	7	0.46	0.52
Seriously injured	4	0.31	0.48
Physical assault by known or unknown person	13	0.35	0.48
Life threatened or in danger	5	0.38	0.51
Seriously injured	4	0.31	0.48
Sexual assault by known or unknown person	13	0.34	0.48
Life threatened or in danger	6	0.46	0.52
Any other traumatic life-threatening situation	10	0.26	0.45
Seriously injured	2	0.20	0.42
Violent death of close family member or friend	8	0.21	0.41
Seriously injured	0	0.00	0.00
Witnessed a situation in which someone was seriously injured or killed, or feared someone would be seriously injured or killed	16	0.42	0.50
<hr/>			
<i>Trauma Occurrence Summary</i>	<i>n</i>		
No trauma	2		
Single trauma	3		
Multiple traumas	33		
Child abuse and multiple traumas	13		
Life threatened or danger of injury	26		
Seriously injured	13		

References

- Afifi, T., Asmundson, G., Taylor, S., & Jang, K. (2010). The role of genes and environment on trauma exposure and posttraumatic stress disorder symptoms: a review of twin studies. *Clinical Psychology Review, 30*, 101–112.
- Alkon, A., Goldstein, L., Smider, N., Essex, M., Kupfer, D., & Boyce, W. (2002). Developmental and contextual influences on autonomic reactivity in young children. *Developmental Psychobiology, 42*(1), 64-78.
- Allen, B., & Lauterbach, D. (2007). Personality characteristics of adult survivors of childhood trauma. *Journal of traumatic stress, 20*(4), 587-595.
- Allen, M., & Matthews, K. (1997). Hemodynamic responses to laboratory stressors in children and adolescents: The influences of age, race, and gender. *Psychophysiology, 34*(3), 329-339.
- American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders: Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition*. Arlington, VA: American Psychiatric Association.
- Andersen, S, Karstoft K., Bertelsen M., & Madsen T. (2014). Latent trajectories of trauma symptoms and resilience: the 3-year longitudinal prospective USPER study of Danish veterans deployed in Afghanistan. *Journal of Clinical Psychiatry 75*, 1001–1008.
- Armbruster, D., Moser, D., Strobel, A., Hensch, T., Kirschbaum, C., Lesch, K., & Brocke, B. (2009). Serotonin transporter gene variation and stressful life events impact processing of fear and anxiety. *The international journal of neuropsychopharmacology, 12*(3), 393–401.
- Aupperle, R., Melrose, A., Stein, M., & Paulus, M. (2012). Executive Function and PTSD: Disengaging from Trauma. *Neuropharmacology, 62*(2), 686–694.

- Azevedo, T., Volchan, E., Imbiriba, L., Rodrigues, E.C., Oliveira, J., Oliveira, L., Lutterbach, L., & Vargas, C. (2005). A freezing-like posture to pictures of mutilation. *Psychophysiology*, *42*(3), 255-260.
- Batten, S., Drapalski, A., Decker, M., DeViva, J., Morris, L., Mann, M., & Dixon, L. (2009). Veteran interest in family involvement in PTSD treatment. *Psychological Services*, *6*(3), 184–189.
- Bartels, M., Van den Berg, M., Sluyter, F., Boomsma, D., & de Geus, E. (2003). Heritability of cortisol levels: review and simultaneous analysis of twin studies. *Psychoneuroendocrinology*, *28*, 121-137.
- Baumeister, R., & Leary M. (1995). The need to belong: desire for interpersonal attachments as a fundamental human motivation. *Psychology Bulletin*, *117*(3), 497–529.
- Beckham, J., Vrana, S., May, J., Gustafson, D., & Smith, G. (1990). Emotional processing and fear measurement synchrony as indicators of treatment outcome in fear of flying. *Journal of Behavior Therapy and Experimental Psychiatry*, *21*(3), 153–162.
- Bell, D., & Schumm, W. (2005). Balancing Work and Family Demands in the Military: What Happens When Your Employer Tells You to Go to War? In D. Halpern, S. Murphy (Eds.), *from work-family balance to work-family interaction: Changing the metaphor* (pp. 83-97). Mahwah, NJ US: Lawrence Erlbaum Associates Publishers.
- Bernsten, D., Rubin, D., & Siegler, I. (2011). Two versions of life: Emotionally negative and positive life events have different roles in the organization of life story and identity. *Emotion*, *11*(5), 1190-1201.
- Bisson, J., Ehlers, A., Matthews, R., Pilling, S., Richards, D., & Turner, S. (2007). Psychological treatments for chronic post-traumatic stress disorder: Systematic review and meta-analysis. *British Journal of Psychiatry*, *190*(2), 97-104.

- Blanchard, D., Hynd, A., Minke, K., Minemoto, T., & Blanchard, R. (2001). Human defensive behaviors to threat scenarios show parallels to fear- and anxiety-related defense patterns of non-human mammals. *Neuroscience & Biobehavioral Reviews*, 25 (7-8), 761–770.
- Blechert, J., Michael, T., Grossman, P., Lajtman, M., & Wilhelm, F. (2007). Autonomic and respiratory characteristics of posttraumatic stress disorder and panic disorder. *Psychosomatic Medicine* 69(9), 935-43.
- Blosnich, J., Gordon, A., & Bossarte, R. (2014). Suicidal ideation and mental distress among adults with military service history: results from 5 US States. *American Journal of Public Health*, 104, S595-S602.
- Boscarino, J., & Adams, R. (2009). PTSD onset and course following the World Trade Center disaster: findings and implications for future research. *Social psychiatry and psychiatric epidemiology*, 44(10), 887–898.
- Bound, J., & Turner, S. (2002). Going to war and going to college: Did World War II and the GI bill increase educational attainment for returning veterans? *Journal of Labor Economics*, 20(4), 784-815.
- Bovin, M., Jager-Hyman, S., Gold, S., Marx, B., & Sloan, D. (2008). Tonic immobility mediates the influence of peritraumatic fear and perceived inescapability on posttraumatic stress symptom severity among sexual assault survivors. *Journal of Trauma Stress*, 21, 402–409.
- Boyce, W. & Ellis, B. (2005). Biological sensitivity to context: I. An evolutionary–developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology*, 17(2), 271-301.
- Bremner, J. (2006). Traumatic stress and the brain. *Dialogues in Clinical Neuroscience*, 8(4), 445-461.
- Breslau, N. (2001). The epidemiology of posttraumatic stress disorder: what is the extent of the problem? *Journal of Clinical Psychiatry*, 62(17), 16-22.

- Brewin, C. & Holmes, E. (2003). Psychological theories of posttraumatic stress disorder. *Clinical Psychology Review* 23(3), 339-76.
- Brodsky, B. & Stanley, B. (2008). Adverse childhood experiences and suicidal behavior. *Psychiatric Clinics of North America*, 31(2), 223- 235.
- Brocke, B., Armbruster, D., Muller, J., Hensch, T., Jacob, C., Lesch, K., Kirschbaum, C. & Strobel, A. (2006). Serotonin transporter gene variation impacts innate fear processing: acoustic startle response and emotional startle. *Molecular Psychiatry*, 11(12), 1106-1112.
- Bronfenbrenner, U. (1979). *The Ecology of Human Development: Experiments by Nature and Design*. Harvard University Press.
- Bronfenbrenner, U. (1994). Ecological models of human development. In *International Encyclopedia of Education*, (Vol. 3, 2nd. Edition). Oxford: Elsevier.
- Bronfenbrenner, U. & Morris, P. (2006). The Bioecological Model of Human Development. *Handbook of Child Psychology, 6th ed., vol. 1, Theoretical Models of Human Development*, ed. Richard M. Lerner. (p. 793–828). John Wiley & Sons.
- Bronfenbrenner, U. and Morris, P. (2007). The Bioecological Model of Human Development. In *Handbook of Child Psychology* (eds, Damon, W., Lerner, R., & Lerner, R.).
- Bryan, C., & Anestis, M. (2011). Re-experiencing symptoms and the interpersonal psychological theory of suicidal behavior among deployed service members evaluated for traumatic brain injury. *Journal of Clinical Psychology*, 67(9), 856-865.
- Busjahn, A., Faulhaber, J., Frier, K., & Luft, F. (1999). Genetic and Environmental Influences on Coping Styles: a twin study. *Psychosomatic Medicine*. 61(4), 469-475.
- Calhoun, P., Beckham, J., & Bosworth, H. (2002). Caregiver burden and psychological distress in partners of veterans with chronic posttraumatic stress disorder. *Journal of Traumatic Stress*, 15, 205-212.

- Carroll, T., Currier, J., McCormick, W., & Drescher, K. (2017). Adverse childhood experiences and risk for suicidal behavior in male Iraq and Afghanistan veterans seeking PTSD treatment. *Psychological Trauma: Theory, Research, Practice, and Policy*, *9*(5), 583–586.
- Caspi, A., Sugden, K., Moffitt, T., Taylor, A., Craig I., & Harrington, H. (2003). Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science*, *301*, 386–9.
- Chemtob, C., Nomura, Y., Rajendran, K., Yehuda, R., Schwartz, D., Abramovitz, R. (2010). Impact of maternal posttraumatic stress disorder and depression following exposure to the September 11 attacks on preschool children's behavior. *Child Development*, *81*, 1129—1141.
- Chen, M., & Bargh, J. (1999). Consequences of automatic evaluation: Immediate behavioral predispositions to approach or avoid the stimulus. *Personality and Social Psychology Bulletin*, *25*(2), 215–224.
- Cloitre, M., Stolbach, B., Herman, J., van der Kolk, B., Pynoos, R., Wang, J., & Petkova, E. (2009). A developmental approach to PTSD: Childhood and adult cumulative trauma as predictors of symptom complexity. *Journal of Traumatic Stress* *22*(5), 399-408.
- Cohen, H., Kotler, M., Matar, M., Kaplan, Z., Miodownik, H., & Cassuto, Y. (1997). Power spectral analysis of heart rate variability in posttraumatic stress disorder patients. *Biological Psychiatry* (*41*), 627–629.
- Cohen, H., Kotler, M., Matar, M., Kaplan, Z., Loewenthal, U., & Miodownik, H. (1998). Analysis of heart rate variability in posttraumatic stress disorder patients in response to a trauma-related reminder. *Biological Psychiatry* (*44*), 1054–1059.
- Cohen, H., Benjamin, J., Geva, A., Matar, M., Kaplan, Z., & Kotler, M. (2000). Autonomic dysregulation in panic disorder and in post-traumatic stress disorder: application of power spectrum analysis of heart rate variability at rest and in response to recollection of trauma or panic attacks. *Psychiatry Research* (*96*), 1–13.

- Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of health and social behavior*, 385-396.
- Cohen, S., & Wills, T. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin*, 98(2), 310.
- Davis, L., Suris, A., Lambert, M., Heimberg, C. & Petty, F. (1997). Post-traumatic stress disorder and serotonin: new directions for research and treatment. *Journal of Psychiatric Neuroscience*, 22(5), 318-26.
- Department of Veterans Affairs & Department of Defense. (2019). *VA/DoD clinical practice guideline for the management of posttraumatic stress*. Washington, DC: Veterans Health Administration, Department of Defense.
- Delahanty, D. (2011). *The psychobiology of trauma and resilience across the lifespan*. Lanham: Jason Aronson.
- DiRamio, D., Ackerman, R., & Mitchell, R. (2008). From combat to campus: Voices of student-veterans. *NASPA Journal*, 45(1), 73-102.
- Duman, E. & Canli, T. (2015). Influences of life stress, 5-HTTLPR genotype, and SLC6A4 methylation on gene expression and stress response in healthy Caucasian males. *Biology of Mood and Anxiety Disorders* 5(20), 1-14.
- Eilam, D. (2005). Die-hard: a blend of freezing and fleeing as a dynamic defense –implications for the control of defensive behavior. *Neuroscience & Biobehavioral Reviews*. 29, 1181–1191,
- Ellison, M., Mueller, L., Smelson, D., Corrigan, P., Stone, R., Bokhour, B., & Drebing, C. (2012). Supporting the education goals of post-9/11 veterans with self-reported PTSD symptoms: A needs assessment. *Psychiatric Rehabilitation Journal*, 35(3), 209-217.
- Elzinga, B., Roelofs, K., Tollenaar, M., Bakvis, P., van Pelt, J. & Spinhoven, P. (2008). *Psychoneuroendocrinology*, 33, 227-237.

- Facchinetti, L., Imbiriba, L., Azevedo, T., Vargas, C., & Volchan, E. (2006). Postural modulation induced by pictures depicting prosocial or dangerous contexts. *Neuroscience Letters*, *410*, 52-56.
- Faul, F., Erdfelder, E., Lang, A., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, *39*(2), 175-191.
- Fiszman, A., Mendlowicz, M., Marques-Portella, C., Volchan, E., Coutinho, E., Souza, W., Rocha, V., Lima, A., Salomão, F., Mari, J., & Figueira, I. (2008). Peritraumatic tonic immobility predicts a poor response to pharmacological treatment in victims of urban violence with PTSD. *Journal of Affective Disorder*, *107*, 193–197.
- Foa, E., & Kozak, M. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin*, *99*(1), 20–35.
- Ford, J. & Kidd, P. (1998). Early Childhood Trauma and Disorders of Extreme Stress as Predictors of Treatment Outcome with Chronic Posttraumatic Stress Disorder. *Journal of Traumatic Stress*, *11*(4), 743–761.
- Fragkaki, I., Thomaes, K. & Sijbrandij, M. (2016) Posttraumatic stress disorder under ongoing threat: a review of neurobiological and neuroendocrine findings. *European Journal of Psychotraumatology*, *7*(1), 30915.
- Frodl, T. & O’Keane, V. (2013). How does the brain deal with cumulative stress? A review with focus on developmental stress, HPA axis function and hippocampal structure in humans. *Neurobiology of Disease*, *52*, 24-37.
- Goldberg J., True W., Eisen S., & Henderson W. (1990) A Twin Study of the Effects of the Vietnam War on Posttraumatic Stress Disorder. *Journal of the American Medical Association*. *263*(9):1227–1232.

- Gottlieb, G. (2007). Probabilistic epigenesis. *Developmental Science*, *10*(1), 1-11.
- Gottlieb, G. & Halpern, C. (2002). A relational view of causality in normal and abnormal development. *Development and Psychopathology*, *14*, 421–435.
- Grabe, H., Spitzer, C., Schwann, C., Marcinek, A., Frahnow, A., Barnow, S., & Rooskopf, D. (2009). Serotonin transporter gene (SLC6A4) promoter polymorphisms and the susceptibility to posttraumatic stress disorder in the general population. *American Journal of Psychiatry*, *166*(8), 926–933.
- Gradus, J. (2017). Epidemiology of PTSD. U.S. Department of Veterans Affairs.
- Graziano, M. & Cooke, D. (2006). Parieto-frontal interactions, personal space, and defensive behavior. *Neuropsychologia* *44*, 845–859
- Gressier, F., Calati, R., Balestri, M., Marsano, A., Alberti, S., Antypa, N., & Serretti, A. (2013). The 5-HT₁LPR polymorphism and posttraumatic stress disorder: A meta-analysis. *Journal of Traumatic Stress*, *26*, 645–653.
- Grillon, C., Morgan, 3rd C. (1999). Fear-potentiated startle conditioning to explicit and contextual cues in Gulf War veterans with posttraumatic stress disorder. *Journal of Abnormal Psychology*, *108*, 134–142.
- Hagenaars, M., Fisch, I. & van Minnen, A. (2011). The effect of trauma onset and frequency of PTSD-associated symptoms. *Journal of Affective Disorders*, *132*(1-2), 192-9.
- Hagenaars, M.A., Oitzl, M., & Roelofs, K. (2014). Updating freeze: Aligning animal and human research. *Neuroscience & Biobehavioral Reviews*, *47*, 165-176.
- Hagenaars, M.A., Stins, J.F., & Roelofs, K. (2012). Aversive life events enhance human freezing responses. *Journal of Experimental Psychology: General*, *141*(1), 98.

- Hawkley, L., Burleson, M., Berntson, G., & Cacioppo, J. (2003). Loneliness in everyday life: Cardiovascular activity, psychosocial context, and health behaviors. *Journal of Personality and Social Psychology, 85*(1), 105.
- Hawkley, L., & Cacioppo, J. (2010). Loneliness matters: a theoretical and empirical review of consequences and mechanisms. *Annals of Behavioral Medicine, 40*(2), 218-227.
- Hayduk, L. (1978). Personal space: an evaluative and orienting overview. *Psychological Bulletin, 85* (1), 117-134.
- Heidt, J., Marx, B., & Forsyth, J. (2005). Tonic immobility and childhood sexual abuse: a preliminary report evaluating the sequela of rape-induced paralysis. *Behavior Research and Therapy, 43*, 1157-1171.
- Hertsgaard, L., Gunnar, M., Erickson, M., & Nachmias, M. (1995) Adrenocortical responses to the strange situation in infants with disorganized disoriented attachment relationships. *Child Development, 66*, 1100-1106.
- Hoge, C., Castro, C., Messer, S., McGurk, D., Cotting, D., & Coffman, R. (2004). Combat duty in Iraq and Afghanistan, mental health problems, and barriers to care. *The New England Journal of Medicine, 351*, 13-22.
- Holt, D., Cassidy, B., Yue, X., Rauch, S., Boeke, E., Nasr, S., Tootell, R., & Coombs, G. III. (2014). Neural correlates of personal space intrusion. *The Journal of Neuroscience, 34*(12), 4123-4134.
- Horowitz, M., Duff, D., & Stratton, L. (1964). Body-buffer zone. *Archives of General Psychiatry, 11*(6), 651-656.
- Imanaka, A., Morinobu, S., Toki, S., & Yamawaki, S. (2006). Importance of early environment in the development of post-traumatic stress disorder-like behaviors. *Behavioural Brain Research, 173*(1), 129-137

- Jovanovic, T. & Ressler, K. (2010). How the Neurocircuitry and Genetics of Fear Inhibition May Inform Our Understanding of PTSD. *American Journal of Psychiatry*, *167*, 648-662.
- Jovanovic, T., Keyes, M., Fiallos, A., Myers, K., Davis, M., Duncan, E. (2005). Fear potentiation and fear inhibition in a human fear-potentiated startle paradigm. *Biological Psychiatry*, *57*, 1559–1564.
- Kalin, N., Shelton, S., Rickman, M., & Davidson, R. (1998). Individual differences in freezing and cortisol in infant and mother rhesus monkeys. *Behavioral Neuroscience*, *112*(1), 251–254.
- Kessler, R. (2000). Posttraumatic stress disorder: The burden to the individual and to society. *Journal of Clinical Psychiatry*, *61* (Suppl 5), 4–12.
- Kessler, R., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Arch. Gen. Psychiatry* *52*, 1048–1060.
- Kimbrel, N., Morissette, S., Meyer, E., Chrestman, R., Jamroz, R., Silvia, P., & Young, K. (2014). Effect of the 5-HTTLPR polymorphism on posttraumatic stress disorder, depression, anxiety, and quality of life among Iraq and Afghanistan veterans. *Anxiety, Stress, & Coping*, *28*(4), 456-466.
- Koenen, K., Aiello, A., Bakshis, E., Amstadter, A., Ruggiero, K..., Acierno, R., & Galea, S. (2009). Modification of the association between serotonin transporter genotype and risk of posttraumatic stress disorder in adults by county-level social environment. *American Journal of Epidemiology*, *169* (6), 704-711.
- Koenen, K., Fu, Q., Ertel, K., Lyons, M., Eisen, S., True, W., & Tsuang, M. (2008). Common genetic liability to major depression and posttraumatic stress disorder in men. *Journal of Affective Disorders*, *105*, 109–115.

- Koenen, K., Lyons, M., Goldberg, J., Simpson, J., Williams, W., Toomey, R., & Tsuang, M. (2003). Co-twin control study of relationships among combat exposure, combat-related PTSD, and other mental disorders. *Journal of Traumatic Stress, 16*(5), 433-438.
- Kroenke, K., Strine, T., Spitzer, R., Williams, J., Berry, J., & Mokdad, A. (2009). The PHQ-8 as a measure of current depression in the general population. *Journal of affective disorders, 114*(1), 163-173.
- Lancaster, G., Dodd, S., & Williamson, P. (2004). Design and analysis of pilot studies: recommendations for good practice. *Journal of Evaluation in Clinical Practice, 10*(2): 307-12.
- Lang, P., Bradley, M., & Cuthbert, B. (1997). Motivated attention: Affect, activation, and action. In P. J. Lang, R. F. Simons, & M. T. Balaban (Eds.), *Attention and orienting: Sensory and motivational processes* (p. 97–135). Lawrence Erlbaum Associates Publishers.
- Lang, P., Bradley, M., & Cuthbert, B. (1999). *International Affective Picture System (IAPS): Instruction Manual and Affective Ratings. Technical Report A-4*. Center for Research in Psychophysiology, University of Florida: Gainesville, FL.
- Lebois, L., Wolff, J. & Ressler, K. (2016). Neuroimaging genetic approaches to Posttraumatic Stress Disorder. *Experimental Neurology, 284*, 141-152.
- Lee, R., Draper, M., & Lee, S. (2001). Social connectedness, dysfunctional interpersonal behaviors, and psychological distress: Testing a mediator model. *Journal of Counseling Psychology, 48*(3), 310.
- Lee, R., Keough, K., & Sexton, J. (2002). Social connectedness, social appraisal, and perceived stress in college women and men. *Journal of Counseling & Development, 80*(3), 355-361.
- Lee, R., & Robbins, S. (1995). Measuring belongingness: The social connectedness and the social assurance scales. *Journal of Counseling Psychology, 42*(2), 232.

- Leen-Feldner, E., Feldner, M., Knapp, A., Bunaciu, L., Blumenthal, H., & Amstadter, A. (2013). Offspring psychological and biological correlates of parental posttraumatic stress: review of the literature and research agenda. *Clinical Psychology Review, 12*, 1106—1133.
- Lewis G., Furman S., McCool M., & Porges S. (2012). Statistical strategies to quantify respiratory sinus arrhythmia: Are commonly used metrics equivalent? *Biological Psychology, 89*, 349–364.
- Liberzon, I., Taylor, S., Amdur, R., Jung, T., Chamberlain, K., Minoshima, S., Koeppe, R., & Fig, L. (1999). Brain activation in PTSD in response to trauma-related stimuli. *Biological Psychiatry, 45*, 817–826.
- Lima, A., Fiszman, A., Marques-Portella, C., Mendlowicz, M., Coutinho, E., Maia, D., Berger, W., Rocha-Rego, V., Volchan, E., Mari, J., & Figueira, I. (2010). The impact of tonic immobility reaction on the prognosis of posttraumatic stress disorder. *Journal of Psychiatric Research, 44*, 224–228.
- Lissek, S., Powers, A., McClure, E., Phelps, E., Woldehawariat, G., Grillon, C., & Pine, D. (2005). Classical fear conditioning in the anxiety disorders: a meta-analysis. *Behavior Research and Therapy, 43*, 1391–1424.
- Lloyd, D. (2009). The space between us: a neurophilosophical framework for the investigation of human interpersonal space. *Neuroscience & Biobehavioral Reviews 33*(3), 297–304.
- Löwe B., Decker O., Müller S., Brähler E., Schellberg D., & Wolfgang H. (2008). Validation and standardization of the Generalized Anxiety Disorder Screener (GAD-7) in the general population. *Medical Care, 46* (3):266–274
- Mahan, A. & Ressler, K. (2012). Fear conditioning, synaptic plasticity, and the amygdala: implications for posttraumatic stress disorder. *Trends in Neuroscience. 35*(1):24-35.

- Margolin, G., & Vickerman, K. (2007). Posttraumatic stress in children and adolescents exposed to family violence: I. Overview and issues. *Professional Psychology: Research and Practice, 38*, 613-619.
- Marx, B., Forsyth, J., Gallup, G., & Lexington, J. (2008). Tonic immobility as an evolved predator defense: implications for sexual assault survivors. *Clinical Psychology: Science and Practice, 15*, 74-90.
- Matthews, K. (1986). Summary, conclusions, and implications. In K. Matthews, S. Weiss & T. Detre (Eds.) *Handbook of stress, reactivity and cardiovascular disease*. New York: Wiley-Interscience.
- McCrory, E., De Brito, S., & Viding, E. (2011). The impact of childhood maltreatment: a review of neurobiological and genetic factors. *Frontiers in Psychiatry, 2*, 1-14.
- McHorney, C., Ware Jr, J., Lu, J., & Sherbourne, C. (1994). The MOS 36-item Short-Form Health Survey (SF-36): III. Tests of data quality, scaling assumptions, and reliability across diverse patient groups. *Medical care, 40*-66.
- McLaughlin, K., Sheridan, M. & Lambert, H. (2014). Childhood Adversity and Neural Development: Deprivation and Threat as Distinct Dimensions of Early Experience. *Neuroscience and Biobehavioral Reviews, 47*, 578-591.
- McTeague, L., Lang, P., Laplante, M., Cuthbert, B., Shumen, J., & Bradley, M. (2010). Aversive imagery in posttraumatic stress disorder: trauma recurrence comorbidity, and physiological reactivity. *Biological Psychiatry 67*, 346-356.
- Mega Electronics, Limited. (2016). eMotion Faros 360 Series electrocardiograph recorder and transmitter. Switzerland.
- Merikangas, K. & Swendsen, J. (1997) Genetic epidemiology of psychiatric disorders. *Epidemiology Reviews, 19*(1), 144-155.

- Meyer, S., Chrousos, G., & Gold, P. (2001). Major depression and the stress system: A life span perspective. *Development and Psychopathology*, *13*(3), 565–580.
- Miller, M., Wolf, E., & Keane, T. (2014). Posttraumatic Stress Disorder in DSM-5: New Criteria and Controversies. *Clinical Psychology: Science and Practice*, *21*, 208–220.
- Monroe, S. & Harkness, K. (2005). Life stress, the "kindling" hypothesis, and the recurrence of depression: considerations from a life stress perspective. *Psychological review*, *112*(2), 417–445.
- Moore, B., & Kennedy, C. (2011). *Wheels down: Adjusting to life after deployment*. Washington, DC: American Psychological Association.
- Murrough, J., Huang, Y., Hu, J., Henry, S., Williams, W., Gallezot, J.D., Bailey, C., Krystal, J., Carson, R. and Neumeister, A. (2011). Reduced amygdala serotonin transporter binding in posttraumatic stress disorder. *Biological Psychiatry*, *70*(11), 1033-1038.
- Nachmias, M., Gunnar, M., Mangelsdorf, S., Parritz, R., & Buss, K. (1996). Behavioral inhibition and stress reactivity: The moderating role of attachment security. *Child Development*, *67*, 508–522.
- National Center for PTSD. (2010). *Returning from the war zone: A guide for military personnel*. Retrieved from <http://www.ptsd.va.gov/public/reintegration/guide-pdf/FamilyGuide.pdf>
- Nemeroff, C., Weinberger, D., Rutter, M., Macmillan, H., Bryant, R., Wessely, S, Stein, D., Pariante, C., Seemüller, F., Berk, M., Malhi, G., Preisig, M., Brüne, M., & Lysaker, P. (2013). DSM-5: a collection of psychiatrist views on the changes, controversies, and future directions. *BMC Medical*, *11*, 202.
- Newport, J. & Nemeroff, C. (2000). Neurobiology of posttraumatic stress disorder. *Neurobiology*, *10*, 211-218.
- Newton-Cheh, C. & Hirschhorn, J. (2005) Genetic association studies of complex traits: design and analysis issues. *Mutation Research*, *573* (1-2), 54–69.

- Ogden, P., Minton, K., & Pain, C. (2006). *Trauma and the Body: A Sensorimotor Approach to Psychotherapy*. New York, N.Y.: W.W. Norton & Company, Inc.
- O'Herrin, E. (2011). Enhancing veteran success in higher education. *Peer Review, 13*(1).
- Ozer, E., Best, S., Lipsey, T., & Weiss, D. (2008). Predictors of posttraumatic stress disorder and symptoms in adults: A meta-analysis. *Psychological Trauma: Theory, Research, Practice, and Policy, 5*(1), 3–36.
- Pitman, R., Rasmusson, A., Koenen, K., Shin, L., Orr, S., Gilbertson, M., Milad, M., & Liberzon, I. (2012). Biological studies of post-traumatic stress disorder. *Nature Reviews Neuroscience, 15*(11), 769–787.
- Plomin, R., Asbury, K., Dunn, J. (2001). Why are children in the same family so different? Nonshared environment a decade later. *Canadian Journal of Psychiatry, 46*(3), 225-233.
- Porges, S. W. (1995). Orienting in a defensive world: Mammalian modifications of our evolutionary heritage. A polyvagal theory. *Psychophysiology, 32*(4), 301-318.
- Porges, S. (2003). The polyvagal theory: Phylogenetic contributions to social behavior. *Physiology & Behavior, 79*(3), 503-513.
- Porges, S. (2007). The polyvagal perspective. *Biological psychology, 74*, 116-143.
- Ratner, S. (1967). Comparative aspects of hypnosis. In: Gordon, J.E. (Ed.), *Handbook of Clinical and Experimental Hypnosis*. Macmillan, New York, pp. 550–587.
- Rau, V. & Fanselow, M. (2009). Exposure to a stressor produces a long lasting enhancement of fear learning in rats. *Stress, 12*(2), 125-33.
- Rauch, S., Shin, L., & Phelps, E. (2006). Neurocircuitry models of posttraumatic stress disorder and extinction: human neuroimaging research-past, present, and future. *Biological Psychiatry, 60*, 376–382.

- Rocha-Rego, V., Fiszman, A., Portugal, L., Garcia Pereira, M., de Oliveira, L., Mendlowicz, M., Marques-Portella, C., Berger, W., Freire Coutinho, E., Mari, J., Figueira, I., & Volchan, E. (2009). Is tonic immobility the core sign among conventional peritraumatic signs and symptoms listed for PTSD? *Journal of affective disorders, 115*(1-2), 269–273
- Roelofs, K., Keijsers, G., Hoogduin, K., Naring, G., & Moene, F. (2002). Childhood abuse in patients with conversion disorder. *The American Journal of Psychiatry, 159*, 1908–1913.
- Rumann, C., & Hamrick, F. (2010). Student veterans in transition: Re-enrolling after war zone deployments. *The Journal of Higher Education, 81*(4), 431-458.
- Russell, D. (1996). UCLA Loneliness Scale (Version 3): Reliability, validity, and factor structure. *Journal of personality assessment, 66*(1), 20-40.
- Sachs-Ericsson, N, Cromer, K. & Hernandez, A. (2009). A review of childhood abuse, health, and pain-related problems: the role of psychiatric disorders and current life stress. *Journal of Trauma & Dissociation, 10*, 170-188.
- Sack, M., Hopper, J., & Lamprecht, F. (2004). Low respiratory sinus arrhythmia and prolonged psychophysiological arousal in posttraumatic stress disorder: heart rate dynamics and individual differences in arousal regulation. *Biological Psychiatry, 55* (3), 284-290.
- Sameroff, A. (2010). A unified theory of development: a dialectic integration of nature and nurture. *Child Development, 81*(1), 6–22.
- Sanders, B. & Knoepfler, J. (2008). Neonatal handling increases cardiovascular reactivity to contextual fear conditioning in borderline hypertensive rats (BHR). *Physiology & Behavior, 95*(1-2), 72–76.
- Sangkuhl, K., Klein, T., & Altman, R. (2009). Selective serotonin reuptake inhibitors (SSRI) pathway. *Pharmacogenetics and Genomics, 19*, 907–909.

- Santa Ana, E., Saladin, M., Back, S., Waldrop, A., Spratt, E. McRae, A., LaRowe, S., Timmerman, M., Upadhyaya, H., & Brady, K. (2006). PTSD and the HPA axis: differences in response to the cold pressor task among individuals with child vs. adult trauma. *Psychoneuroendocrinology*, *31*, 501–509.
- Schat, A., Kelloway, E., & Desmarais, S. (2005). The Physical Health Questionnaire (PHQ): construct validation of a self-report scale of somatic symptoms. *Journal of occupational health psychology*, *10*(4), 363.
- Schauer, M., & Elbert, T. (2010). Dissociation following traumatic stress: Etiology and treatment. *Zeitschrift Fur Psychologie-journal of Psychology*, *218*, 109-127.
- Schlossberg, N. Lynch, A., & Chickering, A. (1989). *Improving higher education environments for adults. Responsive programs and services from entry to departure*. San Francisco: Jossey-Bass.
- Shaffer, F. & Ginsberg, J. (2017). An overview of heart rate variability metrics and norms. *Frontiers in Public Health*, *5*: 258, 1-41.
- Shipman, K., Edwards, A., Brown, A., Swisher, L., & Jennings, E. (2005). Managing emotion in a maltreating context: A pilot study examining child neglect. *Child Abuse & Neglect*, *29*, 1015–1029.
- Shipman, K., Zeman, J., Penza, S., & Champion, K. (2000). Emotion management skills in sexually maltreated and nonmaltreated girls: A developmental psychopathology perspective. *Development and Psychopathology*, *12*, 47–62.
- Sloan, D., Marx, B., & Epstein, E. (2005). Further Examination of the Exposure Model Underlying the Efficacy of Written Emotional Disclosure. *Journal of Consulting and Clinical Psychology*, *73*(3), 549–554.
- Smarr, K. & Keefer, A. (2011). Measures of depression and depressive symptoms: Beck Depression Inventory-II (BDI-II), Center for Epidemiologic Studies Depression Scale (CESD),

- Geriatric Depression Scale (GDS), Hospital Anxiety and Depression Scale (HADS), and Patient Health Questionnaire-9 (PHQ-9). *Arthritis care & research*, 63(S11), S454-S466.
- Smith, E., Murphy J., & Coats S. (1999). Attachment to groups: theory and measurement. *Journal of Personality and Social Psychology* 77, 94–110.
- Smith, T., Ryan, M., Wingard, D., Slymen, D., Sallis, J., & Kritz-Silverstein, D. (2008). New onset and persistent symptoms of posttraumatic stress disorder self-reported after deployment and combat exposures: Prospective population based US military cohort study. *British Medical Journal*, 336, 366–371.
- Southwick, S., Paige, S., Morgan III, C., Bremner, J., Krystal, J., & Charney, D. (1999). Neurotransmitter alterations in PTSD: catecholamines and serotonin. *Seminars in Clinical Neuropsychiatry*, 4, 242–248.
- Spitzer, R., Kroenke, K., Williams, J., & Löwe, B. (2006). A brief measure for assessing generalized anxiety disorder: the GAD-7. *Archives of internal medicine*, 166(10), 1092–1097.
- Stark, E., Parsons, C., Van Hartevelt, T., Charquero-Ballester, M., McManners, H., Ehlers, A., Stein, A., & Kringelbach, M. (2015). Post-traumatic stress influences the brain even in the absence of symptoms: A systematic, quantitative meta-analysis of neuroimaging studies. *Neuroscience and Biobehavioral Reviews*, 56, 207-221.
- Stein, M., Jang, K., Taylor, S., Vernon, P., & Livesley, W. (2002). Genetic and environmental influences on trauma exposure and posttraumatic stress disorder symptoms: a twin study. *American Journal of Psychiatry*, 159 (10), 1675-1681.
- Streeter C., Gerbarg P., Saper R., Ciraulo D., Brown R. (2012). Effects of yoga on the autonomic nervous system, gamma-aminobutyric-acid, and allostasis in epilepsy, depression, and post-traumatic stress disorder. *Medical Hypotheses* 78, 571–579.

- Taft, C., Pless, A., Stalans, L., Koenen, K., King, L., & King, D. (2005). Risk factors for partner violence among a national sample of combat veterans. *Journal of Consulting and Clinical Psychology, 73*, 151-159.
- Thayer, J. & Lane, R. (2000). A model of neurovisceral integration in emotion regulation and dysregulation. *Journal of Affective Disorders 61 (3)*: 201-16.
- Thayer, J., Hansen, A., Saus-Rose, E., & Johnsen, B. (2009). Heart rate variability, prefrontal neural function, and cognitive performance: the neurovisceral integration perspective on self-regulation, adaptation, and health. *Annals of behavioral medicine: a publication of the Society of Behavioral Medicine, 37(2)*, 141–153.
- True, W., Rice, J., Eisen, S., Heath, A., Goldberg, J., Lyons, M., & Nowak, J. (1993). A twin study of genetic and environmental contributions to liability for posttraumatic stress symptoms. *Archives of General Psychiatry, 50*, 257–264. doi:10.1001/archpsyc.1993.01820160019002
- Van der Kolk, B. (2006). Clinical implications of neuroscience research in PTSD. *Annals of New York Academy of Sciences, 1071*, 277-293.
- Van Praag, H. (2004). Can stress cause depression? *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 28*, 891–907.
- Vermetten, E. & Bremner, J. (2002). Circuits and systems in stress. II. Applications to neurobiology and treatment in posttraumatic stress disorder. *Depression and Anxiety, 16*, 14-38.
- Volchan, E., Souza, G., Franklin, C., Norte, C., Rocha-Rego, V., Oliveira, J., David, I., Mendlowicz, M., Coutinho, E., Fiszman, A., Berger, W., Marques-Portella, C., & Figueira, I. (2011). Is there tonic immobility in humans? Biological evidence from victims of traumatic stress. *Biological Psychology, 88*, 13–19.

- Volchan, E., Rocha-Rego, V., Bastos, A., Oliveira, J., Franklin, C., Gleiser, S., & Figueira, I. (2017). Immobility reactions under threat: A contribution to human defensive cascade and PTSD. *Neuroscience & Biobehavioral Reviews*, *76*, 29–38.
- Wang, Z., Baker, D., Harrer, J., Hamner, M., Price, M., & Amstadter, A. (2011). The relationship between combat-related posttraumatic stress disorder and the 5-HTTLPR/rs25531 polymorphism. *Depression & Anxiety*, *28*, 1067–1073.
- Ware Jr, J. & Sherbourne, C. (1992). The MOS 36-item short-form health survey (SF-36): I. Conceptual framework and item selection. *Medical care*, 473-483.
- Weathers, F., Litz, B., Keane, T., Palmieri, P., Marx, B., & Schnurr, P. (2013). The PTSD Checklist for DSM-5 (PCL-5) – Standard [Measurement instrument].
- Willemsen-Swinkels, S., Bakermans-Kranenburg, M., Buitelaar, J., Ijzendoorn, M., & Engeland, H. (2000). Insecure and disorganized attachment in children with a pervasive developmental disorder: relationship with social interaction and heart rate. *Journal of Child Psychology and Psychiatry*, *41*(6), 759-767.
- Yehuda, R., Engel, S., Brand, S., Seckl, J., Marcus, S., & Berkowitz, G. (2005). Transgenerational effects of posttraumatic stress disorder in babies of mothers exposed to the World Trade Center attacks during pregnancy. *Journal of Clinical Endocrinology Metabolism*, *90*, 4115—4118.
- Zannas, A., Provençal, N. & Binder, E. (2015). Epigenetics of posttraumatic stress disorder: Current evidence, challenges, and future directions. *Biological Psychiatry*, *78*(5), 327-33.

Alicia Erchul - Curriculum Vitae**Curriculum Vitae****Summary of Qualifications**

- Over 10 years of experience in higher education management with a record of accomplishment leading projects and maintaining budgetary control and both the college and departmental levels.
- Exemplary visionary for initiating programs that improve efficiency, cost effectiveness, resource management, and retention of faculty and students.
- Established and utilized measurements, statistics, and metrics to track program and student success.
- Cost forecasting, budget management and payroll processes for budgets totaling over \$33 million.
- Excellent organizational and leadership skills
- Supervised and managed over 85 administrative staff and student assistants.
- Proactive, accountable, autonomous, continual professional improvement and solutions oriented.

Education

Masters of Science Candidate Psychological Sciences	University of North Florida Summer 2020
<ul style="list-style-type: none"> • Served as Distance Learning Academic Coach • Thesis: Military Veteran Students Transition to Academic Life with PTSD, Trauma, and Potential For Freezing Response 	
Bachelor of Arts, (BA) double major International Business and Russian ACM Study Abroad in Krasnodar, Russia	Cornell College

Professional Experience

University of North Florida	Coordinator, Budgets, College of Arts and Sciences	January 2019 to present
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- **Budgetary oversight for College of Arts and Sciences (COAS) and over 25 departments, programs and centers contained within COAS.**
- Manage college fiscal operations by providing budgetary support, resource analysis, and strategic planning for databased decision making with the dean and associate deans.
- Design, develop, implement, and interpret college level processes and programs.
- Provides budgetary oversight for all COAS departments to ensure policy compliance, including: review of E&G, Auxiliary, Carry Forward, Concession Indexes and Foundation accounts; support of Travel Authorizations and Reimbursements, transfer requests, and payments to vendors, faculty and staff.

Alicia Erchul - Curriculum Vitae

- Facilitate budget development and estimation for department office managers, chairs, and directors to provide best financial resources for cost of continuance and program enhancement.
- Facilitate year-end financial close out with department office managers, chairs, and directors of COAS in conjunction with the Academic Affairs budget director.
- Coordinate training and on-going support for office managers in COAS who are responsible for fiscal activities in their departments.
- Fostered open and transparent communication between college departments, university divisions, faculty, and community partners.
- Attended College Business Management Institute (CBMI) presented by Southern Association of College and University Business Officers (SACUBO) for intensive course of study in business and financial management for administrators of colleges and universities.

**University of
North Florida**

Office Manager, Psychology Department

February 2011-December 2019

- Formulated, forecasted, and administered annual operating budgets that required understanding of requirements and funding policies on usage of federal, state and sponsored research monies, including quarterly reports and annual closeout of budgets for the department.
- Provides budgetary assistance for department to ensure policy compliance, including: review of E&G, Auxiliary, Carry Forward, and Foundation accounts; support of Travel Authorizations and Reimbursements, transfer requests, and payments to vendors, faculty and staff.
- Design, develop, implement, and interpret departmental processes and programs.
- Assisted in providing administrative direction, coordination, and execution of departmental activities for course scheduling, program evaluation, strategic planning, and scholarships.
- Advised in the planning, directing and reporting of the activities of the programs and assisted in developing and implementing procedures for the programs
- Manage (process, maintain, and track) all departmental personnel actions, including all new hire and termination paperwork, contracts, leave, appraisals, and sabbatical requests; following SACS, state and, university policies and procedures.
- Manage personnel budgets and payroll, and assist in managing departmental fiscal operations by providing budgetary support and resource analysis for the chair and department in support of strategic planning and data-based decision making
- Supervised, trained, and delegated departmental activities to office secretary and student office assistants.

**Community
Action of
Northeast
Indiana**

Head Start Office Systems Coordinator

November 2006 -October 2008

- Organized and conducted annual Self-Assessment audit of the Head Start program at 11 centers with over 80 participants ranging from Head Start parents, community volunteers, agency partners, and staff. Streamlined assessment process to take 2 days rather than a month to complete.
- Maintained \$40,000 federal grant budget for program operations with secondary approval for purchase orders.

Alicia Erchul - Curriculum Vitae

- Created quantitative and qualitative data analysis reports of Self-Assessment data, organized action plans with department supervisors for program areas that required improvement.
- Organized and conducted annual calendar planning retreat at an off-site location, which included the creation of motivation and team building exercises resulting in completion and publication of annual calendar.
- Investigated various data management software programs, coordinated data migration of old data into new software program and organized trainings for staff on new software.
- Established and ensured maintenance of efficient and effective record keeping and reporting systems through the computer and other tracking systems for all program data and reporting needs.
- Attended several trainings and conferences relating to Head Start and professional development including NAEYC National Conference, 2008 Governor's Conference on Service and Volunteerism and the Indiana Black Expo.

Publications**Presentations**

- **Professional Conference**
 - **Southeastern Psychological Association (SEPA) 65th Meeting**
 - *Poster –Threat-Related Freezing Response in Military and Veteran Students*
 - Nominated for Committee on Equality of Professional Opportunity (CEPO) Graduate Research Award
 - *Poster - Auditory Processing in Relation to Stress and PTSD in Veterans*
 - Alicia Erchul, Lori Lange, Lyndsey Johnson, Jessica Ledwith, Paige King, and Donnalea Goelz
 - **Association for Psychological Science (APS) 32nd Annual Convention**
 - *Poster – Freezing Reactions Under Threat: Body Sway Variations in Military Student Populations*
 - Alicia Erchul, Lori Lange, Jessica Ledwith, Paige King, and Lyndsey Johnson
 - *Poster - Auditory Processing As an Indicator of Stress and PTSD*
 - Lyndsey Johnson, Lori Lange, Alicia Erchul, Jessica Ledwith, and Paige King

Peer Review**Stress and Health****January 2018**

- *Models of First Responder Coping: Police Officers as a Unique Population*
- Keywords: Coping, Coping strategies, Job stress, Respite/recovery resilience, Stress
- Conducted peer review of methodology, statistical analysis and significance.

Book Chapter**Grief Diaries: Victim Impact Statements****July 2017****Editor: Lynda Cheldelin Fell**

- Gabriel Ray Crystalus, pages 27-40

Alicia Erchul - Curriculum Vitae**Research Experience**

- | | | |
|--------------------------------|--|-----------------------------|
| University of
North Florida | Graduate Research Assistant
Physiology and Resilience Lab
<u>Principal Investigator:</u> Dr. Lori Lange | January 2017-present |
| | <ul style="list-style-type: none"> • Current project: Reliability of bio-behavioral measures on stress with military-connected students. • Received Graduate Research Grant for research project, \$750 • Assisted with research proposal, literature review, study design, IRB document preparation, survey questionnaire design, statistical analysis, paper writing, and report creation. • Trained in heart rate variability (HRV) monitoring with several monitoring devices and software, including Kubios, Faros and certification provided for proficiency with CardioEdit and CardioBatch software. • Trained in the use of TekScan Mobile Mat for capturing body sway recordings. • Trained in the use of the SCAN-3 auditory processing testing program. • Trained in the use of audiometer for testing participants' hearing ranges. | |
| Stress and Health | Peer Review of Article | January 2018 |
| | <ul style="list-style-type: none"> • Models of First Responder Coping: Police Officers as a Unique Population • Keywords: Coping, Coping strategies, Job stress, Respite/recovery resilience, Stress • Conducted peer review of methodology, statistical analysis and significance. | |
| University of
North Florida | Coordinator , Research Programs,
Public Opinion Research Laboratory (PURL)
<u>Principal Investigator:</u> Dr. Paul Harwood | June 2009-February 2011 |
| | <ul style="list-style-type: none"> • Managed 15 sponsored research projects through entire grant life; including budget preparation, project estimates of deliverables and costs, project timeline, instrument creation, survey fielding and report writing. • Maintained departmental budgets and budgets for 15 projects with over \$750,000 received from businesses and local, state, and federal agencies, with all projects completed at or below budget estimate. • Wrote grant proposals with project objectives and high-level estimates, following policies and guidelines for state and federal agencies including the Department of Defense, Department of Homeland Security, Department of Transportation and Department of Education that resulted in receiving grant awards. • Supervised and trained 85 research assistants in 27-seat survey call center, utilizing Sawtooth WinCati, Ci3, and Sensus programs for data collection and SPSS for data analysis. • Assisted with research proposal, literature review, study design, IRB document preparation, survey questionnaire design, focus group discussion guides, statistical analysis, paper writing, and report creation. • Assisted with survey questionnaire design, focus group discussion guides, statistical analysis, and report creation. • Traveled to meet with clients to present project estimates and deliverables, discuss future project plans, and present research findings once projects were completed. • Attended national grant writer training program, passed examination, and received Certified Grant Writer credential. | |

Alicia Erchul - Curriculum Vitae**Research Projects with the Public Opinion Research Lab**

National Consortium for the Study of Terrorism and Responses to Terrorism (START) and Department of Homeland Security (DHS)	<ul style="list-style-type: none"> • START Radicalization Survey 2008-2011 grant for \$175,000 	2008-2011
National Consortium for the Study of Terrorism and Responses to Terrorism (START) and Federal Emergency Management Agency (FEMA) projects	<ul style="list-style-type: none"> • "Rapid Response" Surveys START/FEMA University of Maryland Center of Excellence grant for \$70,702.45 • "Rapid Response" Surveys START/FEMA University of Maryland Center of Excellence grant for \$35,352 	2009 2010
US Department of Transportation/ Florida Department of Transportation projects	<ul style="list-style-type: none"> • "Click It or Ticket" Survey 2009 Florida Department of Transportation grant for \$57,299 • "Over the Limit Under Arrest" DUI Program Evaluation and Data grant for \$46,405 • Program Evaluation and Data grant for \$136,364 • "Click It or Ticket" Survey 2010 grant for \$57,299 • "Over the Limit Under Arrest" DUI Program Evaluation and Data grant for \$66,945 • Program Evaluation and Data grant for \$150,000 	2009 2009 2009 2010 2010 2010
University of North Florida	<ul style="list-style-type: none"> • Transformation Learning Opportunity Election 2008 Study grant for \$28,600 • Graduate Program Awareness Survey grant for \$8,087 	2009 2009
Elkins Constructors	<ul style="list-style-type: none"> • Elkins Satisfaction Surveys grant for \$12,250 	2009
Jacksonville Aviation Authority	<ul style="list-style-type: none"> • Focus Group grant for \$6,400 	2009
Jacksonville Human Rights Commission	<ul style="list-style-type: none"> • LGBT Awareness Survey grant for \$8,800 	2009
Jacksonville Port Authority projects	<ul style="list-style-type: none"> • Community Awareness Survey 2009 grant for \$10,911 • Customer Satisfaction Survey 2009 grant for \$1,810 	2009 2009

Alicia Erchul - Curriculum Vitae**Research Interests**

Broad	trauma, resilience, stress, social, cognitive, neuroscience, spirituality, wellness
Specific	Trauma and stress disorders, self-regulation, PTSD, grief, depression, anxiety, religiosity, peace, compassion, conflict transformation, positive psychology, learning disorders, autism.

Professional Affiliations

- Southeastern Psychological Association 2018 to present
- Association for Psychological Science 2018 to present
- CITI (Collaborative Institutional Training Initiative) Group 2 Social Behavioral Research Investigators 2009, renewed 2013, 2016
- American Grant Writers' Association Certified Grant Writer 2009 to present
- Alpha Phi Omega – national service and leadership fraternity 1996 to present