8-6-2014

The Impact of Chronic Stress and Trauma on Psychological and Neuropsychological Functioning of College Students

Leslie R. SmithVarner
Indiana University of Pennsylvania

THE IMPACT OF CHRONIC STRESS AND TRAUMA ON PSYCHOLOGICAL AND NEUROPSYCHOLOGICAL FUNCTIONING OF COLLEGE STUDENTS

A Dissertation

Submitted to the School of Graduate Studies and Research

in Partial Fulfillment of the

Requirements for the Degree

Doctor of Psychology

Leslie R. Smith Varner

Indiana University of Pennsylvania

May 2014

Indiana University of Pennsylvania
School of Graduate Studies and Research
Department of Psychology

We hereby approve the dissertation of

Leslie R. Smith Varner

Candidate for the degree of Doctor of Psychology

_____ _____
William Meil, Ph.D.
Professor of Psychology, Advisor

_____ _____
David LaPorte, Ph.D.
Professor of Psychology

_____ _____
Pearl Berman, Ph.D.
Professor of Psychology

ACCEPTED

_____ _____
Timothy P. Mack, Ph.D.
Dean
School of Graduate Studies and Research

Title: The Impact of Chronic Stress and Trauma on Psychological and Neuropsychological Functioning of College Students

Author: Leslie R. Smith Varner

Dissertation Chair: Dr. William Meil

Dissertation Committee Members: Dr. David LaPorte
Dr. Pearl Berman

Neuropsychological impairments related to explicit memory, working memory, and executive functioning (response inhibition, cognitive flexibility, attention) have been established within the literature as a consequence of exposure to chronic stress or trauma. Increased symptoms of depression, anxiety, perceived stress, posttraumatic stress, and psychosis have also been noted as a function of the cumulative effect of stress and trauma. The current study evaluated the effects of both potentially traumatic and stressful life events upon neuropsychological and psychological functioning using a sample of 129 undergraduate college students. The current study utilized the Life Events Survey, Stressful Life Events Questionnaire, and the Perceived Stress Scale to determine previous exposure to stress and trauma, as well as current levels of stress. The Trail Making Test, Wisconsin Card Sort Test, N-back test, and Rey Auditory Verbal Learning Test were utilized to measure neuropsychological functioning. To determine levels of depression, anxiety, and posttraumatic stress disorder symptomology, the Beck Depression Inventory, Second Edition, State Trait Anxiety Inventory, and Impact of Event Scale-Revised were administered, respectively. Results indicate that, consistent with past literature, college students experiencing increased traumatic and life stressors had increased symptoms of depression, perceived stress, trait anxiety, and posttraumatic stress. Interpersonal types of trauma were associated with increased psychological difficulties compared to no trauma controls, but non-interpersonal types of trauma were not different from controls. PTSD

symptoms were associated with increased perceived stress and trait anxiety, above and beyond trauma exposure alone. Frequency and duration of trauma partially mediated the effect of numbers of events experienced on PTSD symptoms. Neuropsychological functioning, however, did not differ according to the number of traumatic or stressful experiences reported, type of trauma, level of distress, or frequency and duration of trauma. These results may suggest that the neurocognitive functioning of college students may be resilient to the damaging effects of stress and trauma.

ACKNOWLEDGEMENTS

Throughout this process, my committee chair Dr. Meil, has been exceedingly patient and understanding. It is with sincere and tremendous gratitude that I thank him for his encouragement to keep moving forward, especially in times where my persistence began to wane. His depth of understanding about neurophysiology and the influence of environmental factors on neuropsychological functioning has been instrumental in guiding me through the completion of this dissertation. I cannot thank him enough for all of his suggestions and his thorough review and advisement of my work.

This dissertation also would not have been possible without the dedication, time, and helpful insights of my other committee members, Dr. Berman and Dr. LaPorte. Dr. Berman's lessons in case conceptualizations and the effects of trauma on child development helped enormously throughout this project. My time in assessment classes and clinic with Dr. LaPorte piqued my interest in neuropsychological assessment and provided a solid foundation for my understanding of the topics covered in this dissertation. Thank you!

Thank you to the graduate and undergraduate students from IUP who helped me to collect data for this project.

I would also like to acknowledge the moral support provided to me by my wonderful husband, Jared. The journey through graduate school has been a long one and I am grateful for his patience and encouragement along the way. Not to be forgotten, thank you to my mother for her unwavering confidence that I would complete this successfully and her cheerleading when I was not so sure myself. Thanks as well to my sister and niece for helping to keep Oliver occupied during the early days of this project.

TABLE OF CONTENTS

Chapter Page

I LITERATURE REVIEW 1

History of Stress Science 1
Defining Stress and Trauma 10
The Acute Stress Response 18
Chronic Stress 45
Stress Related Disorders 56

II METHODS 95

Participants 95
Measures 96
Procedure 103

III RESULTS 110

Main Analyses 110
Exploratory Hypotheses 130

IV DISCUSSION 144

Summary 144
Prevalence of Trauma among College Students 146
Effects of Number of Lifetime Stressful and Traumatic Events on Neuropsychological and Psychological Functioning 147
Effects of Interpersonal Trauma on Neuropsychological and Psychological Functioning 153
Effects of PTSD Status on Neuropsychological and Psychological Functioning 157
Effect of Age at Time of Self-reported Traumatic Event on Neuropsychological and Psychological Functioning 162
Factors Potentially Contributing to or Protecting from Influence of Trauma and Stress on Neuropsychological and Psychological Functioning 165
Limitations 168
Conclusions, Implications, and Recommendations for Future Research 171

REFERENCES 176

Chapter Page

APPENDICES 211

Appendix A- Screening Questionnaire 211
Appendix B- Rey Auditory Verbal Learning Test 212
Appendix C- Trail Making Test 213
Appendix D- Impact of Event Scale 214
Appendix E- Life Experiences Survey 215
Appendix F- Stressful Life Experiences Screening Questionnaire 217
Appendix G- Demographic Questionnaire 222
Appendix H- Perceived Stress Scale 225
Appendix I- Beck Depression Inventory, Second Edition 226
Appendix J- State Trait Anxiety Inventory 228
Appendix K- Informed Consent Form 229
Appendix L- Debriefing Form 231

LIST OF TABLES

Table		Page
1	Demographic Information of Participants	96
2	Participant Reported Number of Traumas Experienced on the Pre-screening Measure	104
3	Number and Percentage of Participants Reporting Traumatic Events on the SLESQ	108
4	Number and Percentage of Participants Reporting Stressful Life Experiences on the LES	109
5	Multivariate ANOVA Testing Effects of Number of Negative Stressful/Traumatic Events Experienced on Anxiety, Perceived Stress, and Posttraumatic Stress	112
6	Univariate ANOVA Analyses Testing Effects of Number of Stressful/Traumatic Events Reported on PSS, IES-R, and STAI-B Scores	113
7	Mean Scores on PSS, BDI-II, IES-R, and STAI-B of Low, Medium, and High Groups of Numbers of Events Experienced (with Standard Deviations in Parentheses)	114
8	Multivariate ANOVA Testing Effects of Number of Negative Stressful/Traumatic Events Experienced on TMT-Attention, RAVLT-DR, WCST, and N-back Test	115
9	Mean Scores on N-back, WCST, TMT-Attention, and RAVLT-DR of Low, Medium, and High Groups of Numbers of Events Experienced (with Standard Deviations in Parentheses)	116
10	Multivariate ANOVA Testing Effects of Type of Trauma (Interpersonal, Non-Interpersonal, No Trauma Control) on Depression, Anxiety, Perceived Stress, and PTSD	117
11	Univariate ANOVA Analyses Testing Effects of Type of Trauma (Interpersonal, Non-Interpersonal, No Trauma Control) on Depression, Anxiety, Perceived Stress, and PTSD	118

Table		Page
12	Mean Scores on PSS, BDI-II, IES-R, and STAI-B of No Trauma Controls, Non-Interpersonal, and Interpersonal Groups of Type of Trauma Experienced (with Standard Deviations in Parentheses)	119
13	Multivariate ANOVA Testing Effects of Interpersonal, Non-Interpersonal Stressful/Traumatic Events Experienced, and No Trauma Controls on TMT-Attention, RAVLT-DR, and N-back Test	120
14	Mean Scores on TMT-Attention, WCST, RAVLT-DR, and N-back test of No Trauma Controls, Non-Interpersonal, and Interpersonal Groups of Type of Trauma Experienced (with Standard Deviations in Parentheses)	121
15	Multivariate ANOVA Testing Effects of PTSD Status on Anxiety and Depression	122
16	Univariate ANOVA Analyses Testing Effects of PTSD Status on PSS, IES-R, and STAI-B Scores	123
17	Mean Scores on BDI-II, PSS, and STAI-B of No Trauma Controls, Trauma Without PTSD, and PTSD Groups of PTSD Status (with Standard Deviations in Parentheses)	124
18	Multivariate ANOVA Testing Effects of Number of PTSD Status on TMT-Attention, RAVLT-DR, and N-back Test	125
19	Mean Scores on TMT-Attention, WCST, RAVLT-DR, and N-back test of No Trauma Controls, Trauma Without PTSD, and PTSD Groups of PTSD Status (with Standard Deviations in Parentheses)	126
20	Multivariate ANOVA Testing Effects of Age of Trauma Reported on IES-R on Depression, Anxiety, and Perceived Stress	127
21	Mean Scores on BDI-II, PSS, STAI-B, and IES-R of Early Childhood (5-9), Childhood (10-13), Adolescence (14-17), and Adult (18 and over) Groups of Age at Time of Trauma (with Standard Deviations in Parentheses)	128
22	Multivariate ANOVA Testing Effects of Number of Age at Time of Trauma on TMT-Attention, RAVLT-DR, and N-back Test	130

Table		Page
23	Mean Scores on TMT-Attention, WCST, RAVLT-DR, and N-back test of Early Childhood (5-9), Childhood (10-13), Adolescence (14-17), and Adult (18 and over) Groups of Age at Time of Trauma (with Standard Deviations in Parentheses)	131
24	Correlations Among Total Events on SLESQ and LES and Executive and Psychological Functioning	131
25	One way ANOVA Testing Effects of Number of Negative Stressful/Traumatic Events Experienced, Level of Distress, and Frequency/Duration on Psychological (BDI-II, STAI-II, IES-R, PSS) and Neuropsychological (TMT, WCST, RAVLT-DR, N-back Test) Functioning	133
26	Summary of Multiple Regression Analyses for Variables Predicting Variance in BDI-II Scores	135
27	Correlations Among Variables Within the Multiple Regression Analysis with Dependent Variable BDI-II Scores	135
28	Summary of Multiple Regression Analyses for Variables Predicting Variance in IES-R Scores	136
29	Correlations Among Variables Within the Multiple Regression Analysis with Dependent Variable IES-R Scores	137
30	Summary of Multiple Regression Analyses for Variables Predicting Variance in STAI Scores	138
31	Correlations Among Variables Within the Multiple Regression Analysis with Dependent Variable BDI-II Scores	139
32	Summary of Multiple Regression Analyses for Variables Predicting Variance in BDI-II, STAI-II, IES-R, and PSS Scores	140
33	Multivariate ANOVA Testing Interaction Effect of Number of Negative Stressful/Traumatic Events Experienced and Mental Health Treatment on Depression, Anxiety, Perceived Stress, and Posttraumatic Stress	141
34	Participant Reported Alcohol Use on Demographic Questionnaire	142

Table Page

35 Multivariate ANOVA Testing Interaction Effect of Number of Negative Stressful/Traumatic Events Experienced and Drinking Behavior on Depression, Anxiety, Perceived Stress, and Posttraumatic Stress 143

LIST OF FIGURES

Figure		Page
1	Means of psychological functioning scores by number of stressful/traumatic events	114
2	Means of psychological functioning scores by type of trauma	120
3	Means of psychological functioning scores by PTSD status	125
4	Means of psychological functioning scores by age at time of trauma	129

CHAPTER I

LITERATURE REVIEW

History of Stress Science

Stress has been a topic of interest for centuries (Cooper & Dewe, 2004). Early conceptualizations focused on "homeostasis," a concept that described biological processes of coping with environmental change. In the 1930s, Hans Seyle, the "father of stress science" detailed his theory of the bodies reaction to stress, called "general adaptation syndrome." Following this early theory, investigations into the production of hormones relevant in producing the stress response were initiated. Of these hormones, corticotrophin releasing hormone (CRH) was most relevant to the initiation of the stress response. As the understanding of the neurobiology of stress developed, individual differences relevant in the production of the stress response were identified. In addition, theories relating to the intersection of individual differences and the environmental context were detailed. As such, a refined concept of "homeostasis" emerged, which took these findings into account. This new conceptualization is termed, "allostasis." Allostasis describes the process in which the body provides resources for coping with environmental demands via predictive regulation, anticipating needs based on the integration of prior knowledge with sensory input to ensure efficient, multisystemic physiological responses (Sterling, 2011).

Homeostasis

In 1854, Claude Bernard discussed his conceptualization of the internal environment within the body. Specifically, he was interested in the blood and fluid surrounding the cells of the body. He discovered that, through this fluid, exchanges took place which allowed the blood glucose level to stay constant within cells (as reviewed in Gross, 1998). This led him to the

conceptualization that an internal organizing force ensures that a level of constancy is achieved, despite changing conditions within the external environment (as reviewed in Gross, 1998). This concept was later expanded, providing a biological framework for the understanding of how the body reacts to environmental changes.

The term "homeostasis" was coined in the 1930s by Walter Cannon in his book, "Wisdom of the Body." He described this as a dynamic process in which the human body self-regulated and adapted in response to changes within the external environment. The regulated variables, which were those that were meant to remain stable to ensure survival, were kept constant through the regulating mechanisms of negative and positive feedback. Sensory neurons detect a change in the levels of regulated variables (mainly gas content of blood, acidity or pH, temperature, glucose, blood pressure, asmotic pressure) and send feedback to the hypothalamus in the brain, which synthesizes information and coordinates a behavioral response. In negative feedback, the response negates or opposes the deviation from the balanced range. Positive feedback mechanisms are less common methods of maintaining homeostasis and involve further perpetuating the disturbance in steady state. Cannon first conceptualized the process of the fight-or-flight response as the body's mechanism for coping with exposure to stressors that produced increased attention, vigilance, and energy.

General Adaptation Syndrome

Hans Selye (1956) detailed the first model of stress, which he described as a reaction of the body upon exposure to a stimulus that threatened homeostasis. Through a series of experiments conducted in 1936, Seyle discovered that animals respond in similar, stereotyped manner upon exposure to a variety of environmental conditions (Seyle, 1956). He coined this

physiological phenomenon, stress. While this term had previously been used in physics to describe an "internal distribution of force exerted on a material body, resulting in strain," he used the term to describe "actions of forces that take place across any section of the body to threaten homeostasis." His conceptualization of stressors included any stimulus which promoted a change within the body's natural steady state.

Seyle's (1956) description of stress was based upon the body's physiological response to stimuli that threatened homeostasis, which he detailed in his concept of "general adaptation syndrome." This process described how individuals coped with stressors and set the ground work for explaining the individual differences observed in anxiety levels upon exposure to stressors. He described this as how individuals "navigate a steady course toward whatever they consider a worthwhile goal." This process was described as having three distinct stages, the alarm reaction, resistance, and exhaustion. Upon the recognition of a stressor or threat in the environment, the body begins to prepare resources for coping and reacting. This is described as the alarm reaction. He described the physiological components of the alarm reaction and discovered how the endocrine and nervous systems help to adapt to constant changes in and around the individual. He discovered that the stress response was largely mediated by the adrenal glands, which produced corticoids. His ablation experiments identified ACTH as the key hormone in triggering the alarm reaction. However, he discovered a number of hormones that played a role within the body in the stress response. If the stressor continues, it is necessary for the individual to attempt to develop coping mechanisms to reduce or eliminate the stressor and return to homeostasis. This stage is referred to as resistance and describes the attempt to adapt to the situation. If the stressor continues without resolution, the individual will experience a depletion of resources available to

cope efficiently with the stressor, resulting in the exhaustion phase. It is during this phase where vulnerability to disease and illness will present, identified as "diseases of adaptation."

Hormones and Stress

Early research into the hormonal system of the body focused on the role of the visceral organs in producing hormones (as reviewed in Sapolsky, 2004). Later findings discovered that, upon destroying the pituitary gland, these organs began to secrete hormones erratically. As a result, attention focused on the role of the pituitary as the "master gland" of the body, directing the hormonal release of organs in the periphery. Research emerged detailing the effects of damage to the area of the brain surrounding the pituitary gland, which resulting in sporadic release of hormones . Considering these findings, it was suggested that the brain secreted hormones, facilitating the release of hormones from the pituitary gland (Harris, 1944). Two researchers, Guillemin and Schally, independently discovered a hormone released by the brain that regulates the release of hormones by the thyroid via the pituitary gland. In addition, they concluded that the brain released this hormone via the hypothalamus (Goot & Guillemin, 1969; Schally, 1969).

Research continued into the discovery of hormones released by the hypothalamus and their effects upon the pituitary. The hormone that was released by the hypothalamus to stimulate the pituitary to release the hormone corticotrophin (ACTH) remained elusive. In 1981, the chemical structure of the hormone, corticotrophin releasing hormone (CRH) was isolated within hypothalamic tissue (Vale, Spiess, Rivier, & Rivier, 1981). In both the isolated and artificially created CRH samples, increased levels of ACTH were released from the pituitary (Vale et al., 1981). It was speculated that CRH was critical in mediating the body's response to stress.

Following the recognition of CRH, the hypothalamic-pituitary-adrenal (HPA) axis was articulated, broadening the understanding of the physiological stress response.

Allostasis and Allostatic Load

Along with the understanding of the role of physiological mechanisms of acute exposure to stress, researchers and theorists were also interested in the consequences of prolonged exposure to stress, a concept initially introduced by Seyle (1956). Homeostatic mechanisms rely on feedback systems within each system in the brain and body, which detect deviations from the set points established to maintain balance. Upon detection of deviations, positive or negative feedback systems are elicited to return the system to the previously established set point. In homeostasis, each organ within the body has its own regulatory system that functions independently from other organs and systems within the body (Sterling, 2011). To further understand the consequences of stress on functioning, the expansion of the concept of homeostasis was introduced by Sterling and Eyer (1988) and clarified further by McEwen (1998).

When faced with acute stressors, the physiological and psychological aspects of the stress response serve an adaptive function, promoting behavior likely to be effective in coping. Allostasis describes the process by which bodily systems respond in a manner that "promote adaptation to activities such as locomotion and to aversive stimuli-like noise and crowding, hostility, fatigue, isolation, hunger, excessive heat or cold- and threats to safety" (McEwen, 2002, p. 37). Allostasis provides a more systemic and efficient mechanism for adaptation to stressors or changes within the internal or external environment than does homeostasis (Sterling, 2011). The brain serves as a regulatory mediator which monitors conditions within the body and

the external environment, anticipates needs based upon this information, and coordinates complex adjustments through the coordination of multiple systems for coping with these anticipated changes (Sterling, 2011). When faced with stressors, multiple systems valuable in the response are coordinated so that they are matched for their intensity and initiation (Sterling, 2011). To ensure adequate resources are available for both responding and recovery, alternate systems are coordinated for arousal and relaxation. Finally, to increase efficiency, global activation of response systems occur via common messengers (hormones, neurotransmitters), which are then modulated via top-down regulation (Sterling, 2011). Overall, these predictive mechanisms operate on two levels, a shorter term process related to immediate anticipated changes and that which estimates the likely duration of the new state (Sterling, 2011). The second mechanisms allows for the prediction of recurrent or ongoing stressors, increasing efficiency of resource utilization in responding (Sterling, 2011).

Experience of a stressor of severe intensity or exposure to repeated stressors is likely to result in damage to the brain and body, discussed in the concept of allostatic load. McEwen (2004) details three types of physiological responses that contribute to the development of allostatic load, including the experience of frequent stress, failed shut down of stress response, and inadequate responses to the stressor. When exposed to repeated stressors, the physiological stress response occurs repeatedly and can lead to damage within the brain and body (McEwen, 2004). Stressors of this nature include, among others, poverty and childhood abuse. In another type of situation, the individual does not adjust to the stressor, and experiences prolonged activation of the HPA Axis, even when the stressor is no longer present (McEwen, 2004). Finally, if the response of the SAM and HPA axes is not appropriately inhibited, prolonged activation of the stress response is likely to occur. In all three of these situations, the stress

response is activated chronically, resulting in excess amounts of glucocorticoids and neurotransmitters to remain within the brain. The chronic activity of the stress response, and co-occurring increases in hormones and neurotransmitters, can result in negative health consequences and damage within the brain and body (McEwen, 2004). A final situation can result in the experience of allostatic load, the underproduction of glucocorticoids due to damage in negative feedback mechanisms in response to stress (McEwen, 2004). This prevents the action of cortisol in the regulation of the immune system and the reduction of inflammation, resulting in conditions like allergies, asthma, and autoimmune disorders (McEwen, 2004).

Psychological Factors

Concurrently, research investigating individual differences in the experience of stress was also being studied within the field of psychology. Traditional stress theories often defined stress as a biological condition that was non-specific, meaning that the stress response was similar regardless of the stressor that precipitated the response. Researchers began to observe individual variations in the situations that would trigger the stress response. Studies focused on a variety of different stressors and researchers began to urge the field to consider defining the concept according to the details of the stressor (Dohrenwend & Dohrenwend, 1974). Lazarus and Folkman (1984) drew attention to the role that cognitive appraisals play in determining if an environmental condition would become a stressor. Several factors were identified as being associated with increased levels of stress. They argued that stress should be considered a "complex rubric," in which the "person, in interaction with a given environmental situation, generates appraisals of harm/loss, threat, challenge" (Lazarus & Folkman, 1984). As such, definitions of stress that involve only one component, whether it is the individual or the environment, will fail to capture the complexity of the concept (Lazarus & Folkman, 1984).

Lazarus and Folkman's Cognitive Appraisal Theory. Lazarus and Folkman (1984) described the process by which the individual and the environment interact within the context of stress. These interactions, labeled transactions, are described as a reciprocal process in which the individual is influenced by environmental conditions and in which the individual engages in an active process serving to determine the perception of the environment and potential stressor. Cognitive appraisal is described as the process through which environmental conditions are determined to be stressors by the individual. In primary appraisal, the individual evaluated the potential danger or risk presented to the person by the potential stressor. According to the model, primary appraisals can produce three potential judgments: irrelevancy, benign-positive, and stressful. Upon appraising a situation as stressful, individuals further assess the stressor according to levels of harm-loss, threat, and challenge that are presented (Lazarus & Folkman, 1984). Processing also occurs through secondary appraisal. This process involves judgments regarding the perception of resources available to cope with the presented situation (Lazarus & Folkman, 1984). When a discrepancy between the estimated coping abilities and the presented demands is detected, the individual is likely to perceive it as stressful. Events are perceived as stressful according to personal and situational factors. Individuals with high self-esteem are likely to view an event as less stressful as they are more likely to view themselves as having adequate coping resources (Sarafino, 2008). Situational factors that impact stress appraisals include: ambiguity, desirability, and controllability (Sarafino, 2008). People tend to perceive events that are unclear, undesirable, and uncontrollable as more stressful than those that provide clear roles or outcomes, are pleasant, and are within behavioral and cognitive control (Sarafino, 2008).

Dynamic models of stress

In addition to the identification of physiological and psychological aspects of stress, research suggested the role of social situations in the production of symptoms of stress.

Biopsychosocial model. The bio psychosocial model of stress attempts to capture the multi-faceted nature of the concept of stress (Engle 1977; Schwartz, 1982). Stress is influenced by multiple domains, including biological, psychological, and social. This model attempts to describe the intersection of these domains in the experience of stress. Biological influences on the experience of stress include genetic factors, structural defects, and the quality of physical responses (Sarafino, 2008). As discussed in Lazarus's cognitive appraisal theory (1984), psychological factors, such as cognition, emotion, and motivation, also play a role in the experience of stress. The social context also influences the experience of stress. Engle (1980) applies the bio psychosocial model to the systems approach at conceptualization. In this approach, factors are viewed as influencing one another in a dynamic process to create a stimulus that would be considered a stressor by the individual (Engle, 1980). Research examining stress has uncovered many factors that mediate and moderate the experience of stress on multiple levels (Engle, 1980). Using a systems approach allows each of these factors to interact in a complex manner to produce an individual experience of stress (Engle, 1980).

Alternate coping mechanisms

As the understanding of stress and how it influences the body expanded, additional factors were identified as contributing the behavioral aspects of coping with a stressor, mainly alternates to the concept of "fight or flight" as was initially introduced by Walter Cannon (1932). The discovery of additional hormonal systems activated upon exposure to stressors allowed for

the expansion of different responses, mainly that of the sympathetic nervous system (SNS) and the HPA axis. This allowed for a greater understanding of the role of neurotransmitters like norepinephrine (NE) and hormones, like glucocorticoids secreted by the adrenal glands. These findings, however, were challenged by Taylor et al. (2000), who claimed that theories relating to the stress response were biased towards males, and that the response of females may be behaviorally distinct, a response labeled "tend and befriend."

Tend and befriend theory. As previous research regarding the stress response was mostly conducted on males, Taylor and colleagues (2000) argued that the understanding of the behavioral responses to stress was skewed. Noting evolutionary pressures unique to women in terms of childrearing responsibilities, it was discovered that females were more likely to respond to stress by focusing on assuring the safety of their children and by seeking social affiliation. As a result, the behavioral response of "flight" is inhibited via the mechanisms of the hormone oxytocin, secreted to a greater extent among females (Taylor et al., 2000). The understanding of this response to stress was expanded to suggest that males may also elicit this behavioral response (Geary & Flinn, 2002). Stress science expanded following these findings and the understanding of the unique response of the body to different stressors became an important focus.

Stress Signatures

Another important finding further illuminated the complexity of the response to stress, the identification of "stress signatures." The concept of stress signatures involves the diversity of the hormonal constellation of the stress response to varying types of stressors (Sapolsky, 2004). Research suggests that the HPA Axis and the SNS react differentially according to the type of

stressor presented, namely physical or psychological stressors (Dayas, Buller, Crane, Xu, & Day, 2001). Physical and psychological stressors are found to activate different pathways within the amygdala, a structure important in the modulation of the HPA Axis as well as differential recruitment of noradrenergic cells (Dayas et al., 2001). These differential responses are also proposed to translate into stress related disorder dichotomies as anxiety and vigilance are ascribed to sympathetic system function whereas depression is associated with primary alterations in glucocorticoid function (Sapolsky, 2004).

Defining Stress/Trauma

Stress is a common occurrence in the lives of many individuals, with a majority of Americans reporting experiencing moderate to high levels of stress (APA Stress Survey, 2010). Despite reports that stress is commonplace and frequently experienced, the scientific exploration of stress continues to struggle to determine a clear, consistent, and broad definition of the concept. In general, the progression of the scientific inquiry of stress has gone from "cells to society" (Contrada & Baum, 2010). Early conceptualizations of stress evolved from the biology of the stress response and the influence of stressful experience on the health of the individual (Seyle, 1956). Individual differences and mediating/moderating factors that contributed to variation within the physiological stress response broadened the scope of the concept and led to a greater focus on factors within the individual, like personality and coping strategies, and environmental factors, like the type of stressor, contributing to stress. Current conceptualizations focus on the role of cognition in the perception of stressors, the selection and implementation of coping responses, and resulting influence on physical, psychological, and emotional health and well-being (Contrada & Baum, 2010). The current focus of stress science involves the integration of the neurobiology of stress with cognitive, social, and emotional factors (Contrada

& Baum, 2010). In an effort to capture the complexity of the stress concept, the following definition has been proposed, "a process in which environmental demands tax or exceed the adaptive capacity of an organism, resulting in psychological and biological changes that may place persons at risk for disease" (Cohen, Kessler, & Gordon, 1995).

Stress research has historically been conducted in a fragmented manner, with different professions offering different research methodologies and focusing on different aspects of the broad concept. This is problematic when attempting to concisely or operationally define stress. In addition, the lack of consistency regarding the definition of key concepts within the field and the minimal level of communication present among stress researchers in different disciplines makes it difficult to compare findings. Several attempts to organize and summarize the aspects of stress research have been presented (Green, 1990; Khoozani & Hadzic, 2010). Many different terms are used to describe stress and stressors. Green (1990) attempted to provide a conceptual framework for the classification of these events and experiences. Three categories of terms that are commonly described within the literature of the stress process were included in this framework, including input/environmental, interaction/perceptual, and output/psychological. These categories mirror a three stage "stress process." The first stage involves environmental input. Terms used to describe this stage include, stimulus, event, stressor, loss, and catastrophic event. These are objective descriptions of the environmental conditions. During the second phase, the individual perceives the stimuli from the environment and uses judgment to complete an appraisal of the situation. Terms used within this phase include trauma, serious threat, and bereavement. These terms imply an interaction between the individual and the environmental context. Finally, in the third phase, a physiological or psychological response occurs. Included terms are distress, grief, and stress response.

In a recent article, Khoozani and Hadzic (2010) propose ontology to organize the multitude of research within the field of stress science. They categorized the literature into three main domains: causes, mediators, and effects. According to this model, causes of stress refer to aspects of the stimuli promoting the experience of stress within the individual, the stressor. Stressors are described as being divided into three categories, according to their relativity, objectivity, and duration (Khoozani et al., 2010). This category captures the distinction between different types of stressors proposed in the literature, including acute, chronic, episodic acute, and traumatic stress.

Types of Stressors

Interest in conceptualizing stress according to the type of stimuli experienced was prompted by the discovery that negative psychological and physical illness was positively correlated to the number and intensity of stressful life events (Masuda & Holmes, 1967). Three types of stress are frequently identified within the literature: acute, chronic, and traumatic.

Acute stressors. As the most frequent form of stress, acute stressors are generally brief and involve the activation of the biological stress response in an effort to resume a steady state. This type of stressor is generally brief, with the individual resuming normal functioning shortly following the resolution of the situation. Acute stressors generally are short-lived and trigger the stress response. Following exposure, the body inactivates the response (Sutton, 2007). Examples include noise, crowding, isolation, hunger, danger, infection, high technology effects (video games, ringing mobile phones), and imagining a threat or remembering a dangerous event.

Chronic stressors. Chronic stress refers to ongoing stressful situations, like ongoing highly pressured work, long-term relationship problems, loneliness, and persistent financial

worries (Sutton, 2007). Chronic stress is not limited to external events in the environment, but can also be a result of internal factors. For instance, internal stressors could refer to physical or psychological events, including worry about a harmful event, infections, or inflammation.

Traumatic stressors. Of the terms used to conceptualize stress, an area of considerable attention has been that of traumatic stress. Traumatic stress is defined as "psychological and physiological reactions to stressors that threaten the person's life or bodily integrity (or witnessing this happen to another person) and that involves the subjective experience of extreme fear, helplessness, or horror due to being beyond the person's ordinary capacity to cope"(Reyes, Elhai, & Ford, 2008). This concept is specified within the DSM-IV TR's diagnostic criteria of a traumatic event or "trauma", an essential component of the diagnosis of PTSD. As previously mentioned by Green (1990), the term "trauma" and "traumatic stress" typically refers to an interactional process between environmental stimuli and subjective appraisals and judgments.

van der Kolk et al. (1996) define traumatic stressors as "events that violate our existing ways of making sense of our reactions, structuring our perceptions of other people's behavior, and creating a framework for interacting with the world at large." As a result, traumatic stressors introduce the individual to a sense of intense fear and hopelessness and often lead them to seek meaning to resolve the overwhelming nature of the event. These types of stressors often involve a betrayal of their expectations of others and the world in which they live. As such, they often attempt to process the event and re-create a world view that would put it into context (Reyes et al., 2008).

Different subtypes can be identified among traumatic stressors. They may be time limited in nature, like natural disasters, motor vehicle accidents, or sexual assault. These events are

typically unanticipated and often intense (van der Kolk et al., 1996). Sequential stressors are described as those which are more chronic in nature and have a cumulative effect, like those experienced by emergency responders. The last type of stressor involves a chronic exposure to highly dangerous stressors, like those experienced by combat soldiers and in child abuse.

Current Conceptualization of Stress and Stress Related Disease

Throughout the course of its history, stress science has deepened and broadened our understanding of stress, including the biological mechanisms behind the body's response to a stressor and the role of individual differences, psychological factors, and social influences upon the stress response. The progression of this understanding is described as an evolution from "cells to society," indicating the expansion of the understanding of stress beyond basic biological mechanisms to include more complex influences, such as societal influences (Contrada & Baum, 2010).

Despite the progression of stress science, a consistent, widely accepted definition of the stress concept has yet to emerge. Many authors describing stress categorize the concept according to the "type" of stressor, including acute, chronic, and traumatic stress (American Psychological Association, 2011; Sutton, 2007). While this may be helpful in gaining a conceptual understanding of the different subjective experiences individuals face, it may also create misconceptions. First, research does not operationally define these concepts in a clear manner, making it difficult to validate which stressors actually belong within these categories. The previous review of the traditional categories of stress provided examples of stressors used within research to understand stress. Second, the activation of the stress response has been shown to vary depending on the type of stressor presented, making it important to distinguish between

physical and psychological stressors (Sapolsky, 2004). The following will review the implications the distinction between physical and psychological stressors has on current conceptualizations of stress and stress related disorders.

Sapolsky (2004) provides an overview of stress and how it relates to the development of stress related disease and disorders. In his synthesis of current research findings, he distinguishes three different categories of stressors, "acute physical", "chronic physical", and "psychological and social."

"Acute physical" stressors are those that induce immediate demands for protection and adaptation (Sapolsky, 2004). For example, these stressors are like those that would be presented to a zebra being chased by a lion (Sapolsky, 2004). It would be necessary to respond in a manner that would divert energy from processes un-necessary in fleeing or fighting the predator and channel it towards those that are essential, such as muscles, the cardiovascular system, and respiratory system. Our stress response system is ideal in coping with these stressors, as it was developed for that purpose (Sapolsky, 2004).

"Chronic physical" stressors are those that present prolonged or repeated circumstances in which the body must adapt to ensure survival (Sapolsky, 2004). Included in this category would be stressors similar to natural disasters, like drought or famine (Sapolsky, 2004). In these types of stressors, repeated physical energy is required to accommodate necessary coping strategies. Sapolsky (2004) comments that these are not commonly experienced among western human cultures, but are experienced in non-western human cultures and among other species of mammals.

Humans and other social primates are described as differing from other animals due to the advanced development of our cerebral cortex, particularly the frontal lobes (Sapolsky, 2004). As such, humans also experience "psychological or social" stressors (Sapolsky, 2004). Examples of these types of stressors include the emotion of worrying, social evaluative stressors, and anticipatory stress (Sapolsky, 2004). This distinction is important, according to Sapolsky (2004), as these types of stressors most frequently lead to disease. These stressors evoke the same stress response as do physical stressors, but can be damaging as they can be activated chronically (Sapolsky, 2004). The chronic activation of this stress response leads to disease in a multitude of systems and structures within the body, including diabetes, heart disease, psychological disorders, and neuropsychological dysfunction (Sapolsky, 2004).

The following sections of the literature review will discuss, in greater detail, the mechanisms involved in the acute stress response, how the activation of the stress response can produce damage when experiencing chronic stress or intense traumatic stress, and finally, how this relates to development of stress related disorders. As research within each of these areas in extensive, the review will focus primarily on the effects of acute, chronic, and traumatic stress on the neuropsychological functions of executive functions and memory. In addition, the impact of psychological disorders, including Posttraumatic Stress Disorder (PTSD) and depression, upon executive functioning and memory will be discussed. Finally, research involving factors shown to mediate or moderate the relationship between stress and psychological disorders and neuropsychological functioning will be briefly summarized, including gender, socioeconomic status (SES), personality traits, coping styles, social support/affiliation, early childhood environment, and spirituality/religiosity.

The Acute Stress Response

A product of evolution, the acute stress response developed as an adaptive mechanism for coping with threatening or dangerous encounters with stimuli in the environment (McEwen, 2007). Upon exposure to an acute stressor, the stress response is initiated to allow for behavioral reactions that will promote survival of the individual and, by extension, of the species (McEwen, 2007). This response is initiated, maintained, and terminated through a widespread network of structures, neurotransmitters, and hormones within the brain (Contrada & Baum, 2010). Contrada and Baum (2010) refer to these as "stress networks" and describe them as, "highly connected and conserved brain structures that are activated when real or imagined threats to the individual are perceived by vertebrate organisms, from fish to humans (p.11)." Interconnections among the various components of the network afford reciprocal communication that serves to facilitate optimal responding to acute stress. The acute stress response can be conceptually divided into four stages: a.) Initiation of the response, which includes the appraisal of events by the prefrontal cortex (PFC), amygdala, and hypothalamus b.) mobilization of resources, which includes activation of the hypothalamus-pituitary-adrenal (HPA) axis and the sympathetic adrenal medullary (SAM) axis, serving to ready the brain and body for active coping, c.) completion of complex cognitive processes, including decision making, emotional understanding, and learning, which are facilitated by the dopamine, norepinephrine, and serotonin systems, and d.) relaxation and response termination, which occurs via the parasympathetic nervous system and through negative feedback loops within the stress network (as reviewed in Contrada & Baum, 2010).

Following the detection of stressors, the brain coordinates a complex interplay of interactions which make up the stress response and facilitate coping behaviors aimed at survival.

Several pathways are hypothesized to facilitate the triggering of the stress response, including a direct pathway activated upon perception of immediate, physical danger and a more elaborate pathway activated when exposed to a stressor requiring cognitive appraisal from higher brain regions (Herman & Cullinan, 1997). A newer perspective on the maintenance of homeostasis in the body extends beyond the previous conception of the interplay between the peripheral and central nervous systems (PNS, CNS, respectively), which conceptualized processes as distinct relative to the modality of the sensory information (i.e. temperature, pain) (Craig, 2003). In a review, Craig (2003) details the structure and function of a more integrative system that achieves homeostasis through interoception, a process in which the brain achieves a "sense of the physiological condition of the entire body." This system details a pathway through which sensory information from the periphery can communicate with the ANS and the limbic system (hippocampus, amygdala, and prefrontal cortex).

The system and processes involved in the concept of interoception help explain the manner in which sensory information is received and communicated to the limbic system and ANS, which are key in the initiation of the acute stress response (as reviewed in Craig, 2003). Afferent fibers that innervate tissue throughout the entire body terminate within the lamina of the dorsal horn of the spinal cord. From the lamina, projections extend to the parabrachial nucleus(PB), described as a site of integration for afferent information important in maintaining homeostasis. Projections extend from the PB to three regions: a.) the pariaquaductal gray, important in motor responses that serve to ensure homeostasis and in defensive behaviors, b.) the hypothalamus, and c.) the ventromedial thalamus. From the thalamus, projections lead to the structures of the limbic system via the dorsal and anterior insula, which connect to the anterior cingulate cortex (ACC), amygdala, hypothalamus, and ventromedial prefrontal cortex (vmPFC).

As such, the anterior insula facilitates an emotional context for the afferent sensory information. The amygdala is described as the key initiator of the stress response and, upon input from a wide variety of structures, facilitates the release of neurohormones that regulate physiological and cognitive processes via its connection to the hypothalamus.

Amygdala

The stress response, coordinated principally by the HPA Axis, is initiated by a structure located within the rostromedial temporal lobe, the amygdala (Sapolsky, 2003). The amygdala is important in receiving, processing, and communicating sensory information (as reviewed in Ramachandran, 2002; as reviewed in Sapolsky, 2003). The amygdala receives information from all of the different sensory systems in the brain, especially the higher order sensory association cortex within the frontal lobe and the vmPFC. These connections serve to alert it to the need for physiological or emotional responding. The amygdala is described as part of the fear and anxiety circuit in the brain and, as such, is found to integrate information from multiple afferent connections and projects to multiple sites key in promoting survival, like the HPA axis, and areas important in learning and memory, the prefrontal cortex and hippocampus.

Due to the role of the amygdala as a mediator between higher and lower level brain structures, the amygdala facilitates multiple cognitive processes. Research relating the functioning of the amygdala has focused on its role in fear conditioning. Specifically, the amygdala is found to be important in the generation of automatic or reflexive behavioral responses to stimuli associated with fear (Bechara et al., 1995; LeDoux, 1996). In rodent studies, the pairing of the conditioned stimulus of a tone and the unconditioned stimulus of a foot shock, generated a conditioned response of freezing (LeDoux, 2000; Davis, Penschuck, Fritschy, &

McCarthy, 2000; Maren, 2001). The lateral amygdala was found to be important in the remembered pairing of these stimuli, whereas the central nucleus of the amygdala was found to be important in fear expression. Implication that vmPFC is responsible for inhibition of fear in extinction (Quirk, Garcia, & Gonzalez-Lima, 2006).

The amygdala plays a role in interpreting complex stimuli, like faces. In addition, it detects information related to novel stimuli and plays a role in the affective component of stimuli, especially those that are rewarding or aversive (as reviewed in Ramachandran, 2002). Within the structure, a complex system synthesizes these inputs and coordinates responses. From the amygdala, connections extend back to the sensory areas, including the primary visual cortex. Also, information is sent to the hypothalamus and the brain stem, contributing to vital functions like heart rate, blood pressure, gut/bowel functions, breathing, and bladder functioning. The amygdala sends projections to the orbital and medial frontal cortex, which suggest a role in assigning an "affective sign" (i.e. if something is rewarding or aversive) and determining mood (as reviewed in Ramachandran, 2002). In addition, it is involved in memory, including object recognition and assigning an affective quality to memory (Lezak, Howieson, Loring, Hannay, & Fischer, 2004).

The interconnections among the amygdala and the prefrontal cortex (PFC) and hypothalamus are important in understanding the acute stress response system. Connections with the PFC occur via the ventromedial PFC, the anterior cingulate cortex (ACC), and the anterior insula (Craig, 2003). These connections are reciprocal (Craig, 2003). In addition, the amygdala is reciprocally connected to the hypothalamus, an area key in initiating the stress response via the HPA Axis.

Prefrontal Cortex (PFC)

One of the challenges historically presented to the field of stress science is to explain the role of individual differences in stress responding (Sapolsky, 2004). Miller and Cohen (2001) provide a detailed theoretical model of the functioning of the PFC. An understanding of this model helps to conceptualize the role of the PFC in the regulation of the stress response.

According to the theoretical model presented by Miller and Cohen (2001), as an evolutionary advantage, the PFC developed in humans as a mechanism to coordinate a complex array of sensory inputs, cognitive stimuli, and emotional content in a manner that would promote the completion of goal directed behaviors (Miller & Cohen, 2001). The PFC is responsible for a variety of higher order cognitive processes, termed "executive functions," including emotion regulation, working memory, personality, and inhibition of other brain systems and processes (Miller & Cohen, 2001). The PFC is interconnected with other brain regions, including the amygdala, illustrating the widespread influence it has upon brain functions (Miller & Cohen, 2001). It is considered the control station for the brain, modulating the activity carried out by other areas. As such, under normal conditions, the PFC provides a "top-down regulation" of cognitive functioning. In "top down regulation," inappropriate responses, behaviors, or processes are inhibited in favor of those that are relevant to the achievement of goal directed behavior (Arnsten, 2009). When "the mappings between sensory inputs, thoughts, and actions either are weakly established relative to other existing ones or are rapidly changing," the PFC becomes important in regulating appropriate responses based on "internal representations of goals and the means to achieve them" (Miller & Cohen, 2001, p.168). This occurs via biasing signals within neurons of other brain regions and networks that govern behavioral, physiological, and emotional processes (Miller & Cohen, 2001). If necessary, the PFC can override hardwired, automatic

behaviors in favor of those more fitting with current goals and desires (Miller & Cohen, 2001). When faced with a novel situation, the PFC is able to facilitate a trial and error process in which responses are judged based on their success or failure, utilizing reinforcement signals (Miller & Cohen, 2001).

The PFC is divided into separate regions, which are shown to govern different processes related to the regulation of behavior, including the ventromedial PFC (vmPFC), dorsolateral PFC (dlPFC), orbitofrontal cortex, dorsomedial PFC (dmPFC), and the anterior cingulate cortex (ACC). Communication among these different areas occurs within the cortex and can project to subcortical regions via complex circuits (Miller & Cummins, 2007). These circuits utilize common neurotransmitters, including glutamate (excitatory), y-aminobutyric acid (GABA; inhibitory), dopamine, acetycholine, serotonin, and norepinephrine (Miller & Cummins, 2007). The medial region of the frontal cortex plays an important role in the initiation of the stress response via its interconnectivity to the amygdala. In particular, the vmPFC and the dmPFC are important in the understanding of the acute stress response.

Medial PFC. The mPFC is associated with a variety of functions important in regulating emotion and responses to stress. Damage to this area of the brain is found to produce "disinhibition syndrome," a condition associated with flat affect, emotional outbursts, poor decision making, and impulsivity (Barrash, Tranel, & Anderson, 2000). Research suggests that the medial region of the PFC provides an inhibitory function on regulating the stress response (Pascucci, Ventura, Latagliata, Cabib, & Puglisi-Allegra, 2007). Support for the role of the mPFC in the modulation of the stress response results from the study of patients with damage to this area of the brain. Individuals with damage to the mPFC often display hyper- or hypo-stress reactivity as they struggle to accurately identify social or emotional cues (Barrash et al., 2000).

To further evaluate this relationship, Buchanan et al. (2010) measured changes in affect, cortisol reactivity, and heart rate variability following exposure to a social stressor, the Trier Social Stress Test. In individuals with damage to the mPFC, a pronounced emotional, hormonal, and cardiovascular response was noted, indicating that damage to this region impairs the ability of the mPFC to inhibit the psychological and physiological stress response (Buchanan et al., 2010).

The mPFC is also suggested to regulate the neurophysiological response to stress by identifying stimuli as being a stressor and facilitating catecholamine release (Hansel & von Kanel, 2008). Following exposure to a stressor, an increase in NE within the mPFC occurs, which is theorized to facilitate the release of DA from the nucleus accumbens (NAc:Pascucci et al., 2007). To evaluate this theorized relationship, Pascucci and colleagues (2007) evaluated the dynamic relationship between NE and DA in rats exposed to restraint stress. Upon exposure to restraint, rats displayed an increase in both NE and DA within the mPFC (Pascucci et al., 2007). Following this initial exposure, a subsequent increase in DA release within the nucleus accumbens was noted (Pascucci et al., 2007). Throughout the duration of the stressor, catecholamine activity was monitored within the mPFC and the NAc. DA release in the NAc decreased throughout the duration of restraint (Pascucci et al., 2007). NE output from the mPFC also decreased, but cortical release of DA increased throughout the duration of the stressor (Pascucci et al., 2007). Upon administration of selective depletion of NE within the PFC, the increased outflow of NE by the mPFC was eliminated, along with the release of DA in the NAc (Pascucci et al., 2007). This suggests that, in addition to having an inhibitory role within the stress response, the mPFC also facilitates the release of catecholamines important in regulating the stress response.

The mPFC is divided into several regions that serve distinct functions. Two key areas related to the stress response include the ventromedial PFC (vmPFC) and the dorsomedial PFC (dmPFC). Stress and trauma research have noted the influence of connections between the amygdala and the medial PFC in relation of emotional processing, anxiety, and fear (Williams, Kemp, Felmingham, Barton, & Olivieri, 2006). The amygdala is responsible for many aspects of fear, including the appraisal of threatening stimuli, and the initiation and maintenance of the fear response (Williams et al., 2006). The mPFC has several subdivisions, including the anterior cingulate cortex (ACC) and more ventral areas that include the infralimbic and prelimbic cortices (Sotres-Bayon, Bush, & LeDoux, 2004). The ACC is further divided into dorsal and ventral regions. The ventral region has been associated with the regulation of emotions and has connections with the amygdala (Cohen, Botvinick, & Carter, 2000). Alternately, the dorsal region is associated with attention and cognition (Cohen et al., 2000). The amygdala and vmPFC are interconnected in a manner that allows the vmPFC inhibit the fear response initiated by the amygdala. Alternately, the amygdala can serve to reduce vmPFC activity (Garcia, Vouimba, Baundry, & Thompson, 1999). Functional neuroimaging studies have revealed a negative relationship between the amygdala and the ventrolateral PFC (vlPFC) in which amygdala activity decreased as vlPFC increased (Williams et al., 2006). It has been suggested that a lack of inhibition from the mPFC contributes to "hyperresponsivity to fear-related stimuli" (Williams et al., 2006).

Ventromedial prefrontal cortex. The ventromedial PFC (vmPFC) has reciprocal connections with key structures within the limbic system, which is important in long term memory and in emotional experience, including affect and motivation (Miller & Cohen, 2001; Arnsten, 2009). Specifically, the vmPFC is strongly connected to the amygdala and plays an

important role in the acute stress response (Arnsten, 2009). In addition, connections with the visual cortex allow for both visual input and selective attention directed towards visual details within the environment (Carretie, Hinojosa, Mercado, & Tapia, 2005). The vmPFC rapidly responds to visual information within the environment, particularly those that involve negative emotionality involving threat or danger, such as faces displaying fearful responses (Carretie et al., 2005). Following the detection of danger, the vmPFC facilitates the direction of attention towards threatening stimuli (Carretie et al., 2005).

The somatic marker hypothesis outlines the process by which signals from the body influence higher order cognitive processes via the ventromedial prefrontal cortex (vmPFC), which is a key structure in the emotional circuitry of the brain (Dunn, Dalgleish, & Lawrence, 2006). The vmPFC is connected to the limbic system (amygdala, hippocampus) and receives input regarding the environment from the periphery nervous system and helps to regulate decision making regarding responses to the environment in situations that are ambiguous or complex (Dunn et al., 2006). Past emotional experiences are used to predict long-term punishments and rewards based on previous experiences. Damage to this region is associated with decision making deficits as emotion biased information received from the body is unable to be utilized to guide response options and previous emotional experiences are unable to guide decision making (Dunn et al., 2006).

Dorsomedial prefrontal cortex. The dmPFC is also implicated in the modulation of the stress response via the PVN of the hypothalamus, the key player in the facilitation of the HPA axis, the hub of the stress response (Radley, Williams, & Sawchenko, 2008). As was previously discussed relating to the perception of a stressor within the environment, the process of interoception allows the individual to maintain a sense of self within their external environment

and provides a basis for the point of interaction between the two domains (Craig, 2003). The ability for interoception is mediated by the frontal lobes (as reviewed in Miller & Cummins, 2007). A related concept, phrenoception, refers to the ability to produce thoughts that are task independent and not necessarily related to features of the external environment . These thoughts are labeled, "stimulus-independent thoughts" (SITS) and have been shown to be mediated by the dmPFC (McGuire, Paulesu, Frackowiak, & Frith, 1996). Specifically, activation of the dmPFC correlates with self-reported frequency of SITS (McGuire et al., 1996). Further studies have alluded to the role of this area in the process of evaluating personal emotions. For instance, when reported emotions of guilt or embarrassment are elicited, the dmPFC is activated (Berthoz, Armony, Blair, & Dolan, 2002). In addition, activity within the dmPFC is noted when participants are asked to provide feedback related to provided evaluative judgments related to the self (Schmitz, Kawahara-Baccus, & Johnson, 2004).

In summary, the medial regions of the PFC, including the vmPFC and the dmPFC, play an important role in the perception of stimuli as a stressor. In addition, both of these regions are interconnected to the amygdala and serve to communicate information integrated across the frontal lobes to regions of the hypothalamus, with the amygdala as the mediating structure. The hypothalamus is integral in the generation of stress hormones important in fueling complex cognitive functioning critical in behavioral responses, glucocorticoids. The release of glucocorticoids is facilitated by the HPA Axis, of which the hypothalamus is the initiating structure.

Dorsolateral Prefrontal Cortex. The dorsolateral prefrontal cortex (dlPFC) coordinates a multitude of functions that relate to engaging in organized and purposeful responses to environmental contingencies, namely executive functions and working memory (Miller &

Cummings, 2007). Specifically, executive functions include volition, recalling information from memory and utilizing it for planning behavior or responses, programing motor activity, implementation of task relevant behaviors and minimization of distractions, monitoring behavior in attention, and set shifting (Miller & Cummings., 2007). Collectively, these functions are "involved in the control and direction of lower level, more autonomic functions" (Miller & Cummings, 2007, p.293).

Attention. The process by which stimuli are chosen to be preferentially processed at the expense of others is referred to as attention, an important component of executive functioning (as reviewed in Miller & Cummings., 2007). Attentional processes have several main functions, including sustained attention (vigilance), selective attention, and divided attention. In sustained attention, the frontal lobes and reticular activating system, located in the medial PFC and the brainstem, respectively, work to ensure that information is able to be attended to over a span of time. Also, it is important that this information is attended to with sufficient consistency. Selective attention refers to the ability to focus on relevant stimuli at the exclusion of other competing stimuli that are irrelevant to the required task or process. Finally, divided attention is the ability to simultaneously process different information or complete different tasks. In divided attention, processing speed becomes an important component as it contributes to the speed with which information is processed, making room for additional information. In addition, divided attention requires the ability to switch between subtasks when completing tasks that cannot be completed simultaneously. Upon exposure to stress, increased NE levels have been shown to increase processing of task relevant stimuli, while simultaneously reducing salience of non-relevant stimuli (Aston-Jones, Rajkowski, & Cohen, 1999).

Cognitive flexibility. Cognitive flexibility refers to the process by which a dominant, engrained response is inhibited to enable the search for more appropriate, task relevant, responses. Upon exposure to acute stress, performance on cognitive flexibility measures is impaired (Alexander, Hillier, Smith, Tivarus, & Beversdorf, 2007; Ishizuka, Hillier, Beversdorf, 2007; Renner & Beversdorf, 2010). For example, Alexander and colleagues (2007) facilitated the experience of acute psychosocial stress by having participants complete the Trier Social Stress Test. Following stress exposure, healthy male college students were required to complete a complex cognitive task requiring cognitive flexibility, visuospatial memory, and motor speed. Results indicated impaired performance on the cognitive flexibility task, but not the memory or motor speed task. In addition, a condition involved administering propranolol, a drug identified as a beta-adrenergic antagonist and comparing task performance with non-drug conditions. Results indicated improved performance in propranolol conditions, suggesting the role of norepinephrine (NE) activity during tasks requiring cognitive flexibility.

Working memory. Baddeley and Hitch (1974) developed a three component system of short term memory, which they labeled "working memory." In this model, a control system labeled the "central executive" managed two sub-systems, the phonological loop and the visuospatial sketch pad in the active maintenance of information for use in cognitive processing (Baddley, 2003). This model expanded the concept of working memory beyond that of short term memory.

As reviewed in Baddley (2003), the phonological loop is theorized as a short term storage mechanism that encodes and retains auditory information for immediate or short term use. Memory traces for immediately processed information are theorized to be held within a phonological storage loop, where content is rehearsed in order to be made available for

immediate recall. This process appears to involve auditory processes. Information from the environment is perceived through auditory pathways. Once perceived, the information creates a memory trace that is held temporarily within phonological short term storage, located within the inferior parietal lobe. If needed, the information is then relayed to an output buffer, which processes and prepares information for spoken output or recycles information for rehearsal. If entered into rehearsal, the information will be recycled through short term storage. If information is perceived in visual format, it is transformed into auditory format before being entered into short term storage.

Baddley's (2003) conceptualization of working memory also describes a mechanism that allows for the maintenance and manipulation of visual and spatial information, the visuospatial sketchpad. This mechanism is divided into two components, the visual cache and the inner scribe. The visual cache retains information regarding form and color. The inner scribe is a more complex component, as it retains spatial and movement information and also rehearses information presented in the visual cache and also transfers information to the central executive (Baddley, 2003). This aspect of working memory has been less studied.

Baddley (2003) describes the central executive as an attentional control system. Since the original theory of working memory, research regarding cognitive abilities has expanded the role of the central executive to include the control and regulation of a wide variety of functions. In regards to working memory, it is now considered to have two distinct processes, one that operates through the use of schemas and another that requires more extensive attentional control, the supervisory activating system (SAS). The Norman and Shallice model (1986) describes two processes through which the central executive controls actions, automatic and willed. Through automatic control mechanisms, routine cognitive tasks are able to be completed. This relies on

"sensorimotor schemas." For example, the routine behavior of opening a door would be controlled through the use of a schema, which would limit the cognitive resources required to oversee the completion of the task (Pezzulo, 2007). For more complex or novel actions, the SAS coordinates, synthesizes, and plans the components of the response.

Baddley (2003) also describes the functions of the episodic buffer, a mechanism for storage that synthesizes information from different modalities that is within conscious awareness. This is theorized to govern the cognitive ability of "chunking." In addition, the episodic buffer allows information to be entered into or retrieved from long term memory. Finally, it provides a mechanism for communication between phonological and visuospatial subsystems.

Research has attempted to locate areas of the brain important in the facilitation of working memory. Functional imaging studies have revealed activation of bilateral dorsolateral prefrontal cortex while completing the n-back test (Baddeley, 2003; Cohen 1997). A linear relationship is revealed, with increased activation noted upon increasing levels of load (Smith & Jonides, 1997; Cohen et al.,1997; Baddley, 2003; Braver et al. 1997). In addition, a meta-analysis of n-back test studies reveals consistent activation of several other areas, including bilateral and medial posterior parietal cortex, bilateral premotor cortex, dorsal/cingulate premotor cortex, and bilateral mid-ventrolateral prefrontal cortex (Owen, McMillan, Laird, & Bullmore 2005). Owen and colleagues (2005) further described the specific working memory related functions that were correlated with performance in these areas. The dorsolateral PFC was identified as serving an organizing function when completing the n-back test by selecting appropriate mechanisms that would serve to improve performance, like chunking. Neuroimaging studies reveal that, during a wide variety of tasks requiring the development of a plan or strategy to remember or recall information, the ventrolateral PFC is activated (Owen et al., 2005). When

examined in lesion studies, monkeys experiencing lesions in this region display difficulties in the "initiation and execution of many types of intended action." The rostral PFC or frontal pole is suggested to be involved in tasks that require the integration of several different cognitive tasks (Owen et al., 2005). The bilateral and medial PFC are engaged during the visuospatial maintenance of information (Owen et al., 2005). Finally, neuroimaging studies have shown activation within the bilateral and medial posterior parietal cortex during working memory tasks. The region to the left is associated with short term storage of verbal material, whereas the right region is associated with spatial material (Owen et al., 2005).

The proliferation of glucocorticoids within the PFC following activation of the HPA Axis has been found to have a dose dependent relationship on working memory functioning (Lupien, Gillin, & Hauger, 1999). At moderate doses of glucocorticoids, performance is enhanced. For instance, moderate doses of glucocorticoids have been associated with enhanced learning and classical conditioning and enhanced memory functioning (Yuen, Liu, Karatsoreos, Feng, McEwen, & Yan 2009). This enhanced functioning has been associated with the increase of glutamatergic transmission via increased numbers of NMDAR and AMPAR receptor subunits following exposure to a moderate stressor (Yuen et al., 2009). Studies have also described the shift between the DMN and the central executive processes within the PFC in response to acute stress (Daniels, McFarlane, Bluhm, Moores, & Clark, 2010). Typically, upon presentation with a task that would involve complex cognitive processing, like WM tasks, the DMN is disengaged and the executive networks are initiated (Daniels et al., 2010). This occurs during tasks that encourage a high WM load (Daniels et al., 2010).

Hypothalamus

While the stress response involves multiple networks within the brain, the initiation of the "fight or flight" response is thought to originate within the autonomic nervous system (ANS), specifically the periventricular nucleus (PVN) of the hypothalamus (Chrousos & Gold, 1992). The hypothalamus regulates autonomic functions of the body, like body temperature, sleep cycles, hunger, and thirst. In addition, the hypothalamus engages in three main processes that aim to maintain homeostasis within the body (as reviewed in Ramachandran, 2002). Specifically, it 1) regulates the autonomic nervous system (ANS), 2) facilitates the neuroendocrine system, and 3) organizes motivational states or behavioral responses. The hypothalamus has projections to multiple sites throughout the central nervous system (CNS). It projects to regions within the brain stem that are responsible for regulating autonomic reactions. One of these areas is the locus coreulus (LC), which plays a role in the distribution of neurochemicals important in the stress response, namely norepinephrine (NE) . It also projects to cortical and subcortical structures of the limbic system . In addition, it projects outside the CNS to the pituitary gland . As such, the hypothalamus serves as a communicator of information across a wide number of brain systems, including the HPA axis, the SAM Axis, and the limbic system.

Within the hypothalamus, the PVN is the "primary controller" of the HPA Axis and SAM Axis in response to stressors (as reviewed in Herman, Cullinan, Ziegler, & Tasker, 2002). The PVN is a small group of neurons that synthesize information received into a "triad of outputs" sent to the adrenal cortex, including dorsal parvocellular zone (dp), ventral extent of the medial parvocellular region (mpv), and lateral parvocellular region (Herman et al., 2002). The PVN is comprised of magnocellular and parvocellular nuclei (Herman, et al., 2002). Several subregions are located within the PVN, including the posterior magnocellular division (pm), dorsal

parvocellular zone (dp), dorsolateral medial parvocellular zone (mpd), and the medial parvocellular region (mpv) (Herman et al., 2002). The PVN is surrounded by a region that is rich in GABAenerigic neurons and that receives input from a multitude of regions within the brain, including the limbic system, brainstem, and other regions of the hypothalamus (Herman, et al., 2002). These regions provide input regarding the state of other brain areas active during stress exposure. Limbic structures projecting to the peri-PVN region include the ventral subiculum (vSub), mPFC, lateral septum, and the medial amygdala (Herman, et al., 2002). The vSub, an area of the hippocampus, is implicated in providing glutamanergic input to the peri-PVN (Herman, et al., 2002). The lateral septum and medial amygdala are primarily GABAenergic (Herman et al., 2002). Ascending connections from regions within the brainstem provide cholinergic and seretongergic inputs into the peri-PVN (Sawchenko, Swanson, Steinbusch, & Verhofstad, 1983). The raphe nucleus in the brainstem provides serotonin (5-HT) input (as reviewed in Herman, et al., 2002). Acytecholine (Ach) connections are received from the dorsolateral tagmental nucleus . In addition, GABAenergic inputs are received from other areas of the hypothalamus and the bed nucleus of stria terminalis (BST). Finally, the peri-PVN receives input from the NE system .

The PVN synthesizes information from a multitude of regions and systems within the brain that are important in the stress response. Projections from these systems appear to terminate within the peri-PVN, where they are synthesized and communicated to the PVN proper via GABAenergic transmission (Herman, et al., 2002). The mpd receives this input and facilitates intracellular communication among the subregions of the PVN (Ludwig, 1998). The dp, mpd, and mpv have direct connections to the brainstem and spinal cord, providing feedback for integration of sympathetic and parasympathetic responses (as reviewed in Herman, et al.,

2002). The pm produces direct release of vasopressin and oxytocin, which facilitate the balance of fluid and electrolytes and assist with balance and lactation (Swanson & Sawchenko, 1983). Finally, following the integration of input received from sites within and outside the PVN, the mpd projects to the median eminence, which releases corticotrophin releasing hormone (CRH) into circulation (Herman et al., 2002).Following the release of CRH into the circulation, two different systems are initiated to facilitate the direction of resources within the brain and periphery, in a manner suggested to optimize coping with stressors. These two systems are the SAM and HPA Axes.

Sympathetic adrenal medullary (SAM) Axis. Following exposure to a real or perceived stressor, regions of the posterior hypothalamus become activated, which in turn activates regions of the adrenal medulla (Foley & Kirschbaum, 2010). Upon exposure to a stressor, activation of the "preganglionic neurons in the intermediolateral cell column of the thoracolumbar spinal cord" results in activation of connections projecting to the adrenal medulla (Ulrich & Herman, 2009). Through direct innervation to the adrenal medulla, the sympathetic nervous system (SNS) facilitates the release of epinephrine and norepinephrine (NE) throughout the bloodstream through excitation of the preganglionic splanchic nerve (Foley & Kirschbaum, 2010). This connection, and the resulting EPI and NE release, is called the SAM Axis. Due to this direct connection, the SAM produces a much faster physiological response to stress than does the HPA Axis. Responses occur within seconds and are short lived (Ulrich-Lai & Herman, 2009). This response is activated during exposure to physical stressors, in which an immediate response is necessary to achieve optimal responding (Ulrich-Lai & Herman, 2009).

Activation of the SNS and the resulting influence of the SAM Axis result in the increase of monoamines in several structures throughout the brain, including the hippocampus, amygdala,

prefrontal cortex, and nucleus accumbens (Joels & Baram, 2010). Specifically, increased levels of NE (Morilak et al., 2005), serotonin (5-HT; Maier & Watkins, 2005), and dopamine (DA; Goto, Otani, & Grace, 2007) are noted following acute stress exposure.

Circulating levels of epinephrine and NE within the bloodstream produce several physiological effects key in the stress response, including increased oxygenation of the blood, increased heart rate, increased blood supply to brain and muscles, increased metabolism, and decreased digestion (Aston-Jones & Cohen, 2005). In addition, the release of NE has been found to result in a shift in attention for processing sensory information (Aston-Jones & Cohen, 2005). Attention is shifted from a more detailed focus on sensory information to a more global attention that allows for the ability to scan the environment for important stimuli to aid in processing (Aston-Jones & Cohen, 2005).

Serotonin is a widely circulated neurotransmitter within the brain, serving both excitatory and inhibitory functions (Southwick, 2010; Lowry, 2002). It plays a role in multiple functions, including sleep, aggression, cardiovascular and respiratory activity, motor output, anxiety, mood, and neuroendocrine activity (as reviewed in Southwick, 2010). Research suggests a functional correlation between exposure to stressful stimuli, behavioral and motor activity, and activation of the serotonin receptors within the caudate putamen (Imai & Steindler, 1986). This implicates this pathway in the facilitation of behavioral and motor responding to stressors. In addition, 5-HT plays a role in the facilitation and inhibition of the HPA and SAM axes, and the limbic system (Lowry, 2002). Finally, 5-HT has been shown to reduce post stress anxiety (Adamec, Holmes, & Blundell, 2008).

Hypothalamic-pituitary-adrenal (HPA) Axis. As previously reviewed, upon receiving information regarding the presence of a stressor, the hypothalamus releases CRH into the circulation. Following this, the interaction of the pituitary and adrenal gland results in the production of glucocorticoids, the primary stress hormone.

The pituitary gland is located near the brain stem and is connected to the hypothalamus through two types of connections (as reviewed in Ramachandran, 2002). Neural connections from the periventricular and supraoptic nuclei to the anterior portion of the pituitary gland serve to facilitate the release of hormones into blood circulation. In the second type of connection, blood vessels from the hypothalamus extend to the anterior pituitary and facilitate hormone release through chemical signals. This allows for a more precise and variable combination of hormones to be released by the pituitary. Upon the detection of corticotrophin releasing hormone (CRH) released by the hypothalamus, endocrine cells within the anterior pituitary release adrenocorticotrophic hormone (ACTH) into blood circulation. ACTH interacts with the adrenal gland, which releases stress hormones into circulation within the periphery.

The adrenal gland is located next to the kidneys in the body and has two distinct regions, the adrenal cortex and adrenal medulla (as reviewed in Winn, 2001). The adrenal cortex is comprised of neural crest cells, which can develop into one of two types of cells, those comprising the adrenal medulla or those making up the sympathetic nervous system. The presence of glucocorticoids facilitates the creation of adrenal medulla cells, able to secrete several hormones, including acetylcholine, epinephrine, norepinephrine, or cortisol. The adrenal gland is triggered to release hormones vital to the stress response by two separate mechanisms, projections from the hypothalamus via the sympathetic nervous system or through the release of ACTH from the pituitary gland. Circulating ACTH released by the pituitary gland activates the

adrenal cortex, which facilitates the release of mineralocorticoids, important in sodium retention and in the increase of blood pressure. Corticosteroids (cortisol) is also released through the adrenal cortex, facilitating transformation of fats and proteins into glucose, which helps create energy for the behaviors encouraged by the stress response. Also, cortisol plays a role in suppressing the immune system, creating more available energy. These hormones prepare the body for physical response, including increased heart rate, blood pressure, and circulation to muscles.

Unlike the monoamines released through activation of the SNS and SAM Axis, which target specific sites within the brain, corticosteroids are exposed globally to all cells within the CNS,PNS, and other organ systems (Joels & Baram, 2010). Two types of receptors allow binding with corticosteroids, mineralocorticoid receptors (MR) and glucocorticoid receptors (GR). These receptors have different levels of affinity for corticosteroids, with MR having higher affinity, making them likely to be occupied during low levels. (DeKloet, Joels, & Holsboer, 2005). Increased numbers of these receptors are found in the hippocampus, PVN of the hypothalamus, amygdala, and locus coeruleus (LC; DeKloet et al., 2005). By contrast, GRs have a much lower affinity for corticosteroids, becoming increasingly occupied upon stress (DeKloet et al., 2005). Increased numbers of GRs are found in the hippocampus and the PVN of the hypothalamus (DeKloet et al., 2005). Glucocorticoids refer to corticosteroids binding on GRs. Of these, cortisol is most widely recognized for its role in stress.

Hippocampus

Located in the temporal horn of the lateral ventricle, within the medial temporal lobe, the hippocampus is implicated in the integration of perceptual information and memory, particularly

spatial memory (Lezak, Muriel, Howieson, Diane, & Loringetal, 2004). Lezak cites the description provided by Rolls (1990) as "specialized to detect the best way in which to store information and then, by return paths to the neocortex, directs memory storage there." In addition, it places a time "stamp" upon information to be placed within long term memory storage (Lezak et al., 2004). The hippocampus projects to the sensory association cortex, the hypothalamus (but not the nuclei responsible for autonomic behavior), and the prefrontal and cingulate cortices (as reviewed in Ramachandran, 2002). The hippocampus has been reported to have an inhibitory response on the activity of the HPA axis (Pruessner et al., 2008). In addition, it is responsible for a variety of neurological functions, including behavioral inhibition, memory, and spatial memory or navigation (Lezak et al., 2004).

Upon exposure to acute stress, several changes in the neuro-chemical environment of the brain occur, including the release of monoamines, CRH, and corticosteroids (de Kloet et al., 2005). As previously reviewed, increased expression of MRs and GRs are found within the hippocampus, indicating that this area produces key effects of the release of corticosteroids during stress. While the implications of severe or chronic stress have been widely examined, review of these effects will be provided in later sections. Upon increase in levels of glucocorticoids, several effects occur within the hippocampus, including some that alter the function of gene transcription and others that do not (nongenomic). One of the rapid, nongenomic functions of acute stress on hippocampal functioning is the increase of glutamate release, an important excitatory neurotransmitter involved in learning and memory (Karst et al., 2005). Stress also activates the carbonic anhydrase 1 (CA1) area of the hippocampus, facilitating long term potentiation (LTP), which is the enhancement of communication among neurons in a manner that prolongs and enhances transmission (Wiegert, Joels, Krugers, 2006). The

mechanism behind this may be related to gene related changes prompted following increased voltage dependent calcium currents (Chameau, Qin, Spijker, Smit, & Joels, 2007). Increased intracellular calcium levels several hours following the initial rise in corticosteroids are shown to result in decreased ability to relay excitatory messages within the CA1 area (Joels & de Kloet, 1989). As a result, LTP is suppressed, resulting in the preservation of information encoded into memory following the initial increase in corticosteroids (as reviewed in Joels & de Kloet, 2008). In addition, increased calcium levels are shown to reduce functioning in N-methyl D-aspartate (NMDA) receptors. The changes in gene expression following stress exposure serve to alter subsequent responses to similar stressors (as reviewed in Joels, 2008).

Episodic memory. Episodic memory was initially described by Tulving (1984), who conceptualized it as memory for past experiences and their temporal and spatial context. In addition, it includes mental representations that allow for the personalization of these experiences and identify them as representing an aspect of themselves throughout time (Singer, 1995). Emotional arousal is necessary for information to be entered into episodic memory (Roozendaal, McEwen, & Chattarji, 2009). Tulving (1984) identifies three aspects that make episodic memory unique. First, it is the only memory system that includes "recollections of previous experiences of events, happenings, and situations" (Tulving, 1984). Second, upon recollection, it cues up aspects of the past experience and allows the individual to experience them in the present (Tulving, 1984). Third, it includes a distinction between what we remember and what we know (Tulving, 1984). As such, past experiences are regarded as memories for past events rather than as facts. Fourth, when one recalls an episodic memory, they cue up semantic knowledge about the experience (Tulving, 1984).

Tulving (1984) originally described two distinct types of memory, episodic and sematic. These different sources of memory were related to different types of awareness, which he labeled autonoetic and noetic. Autoneotic consciousness allowed for the identification of the original context in which the memory occurred (Tulving, 1984). Conversely, noetic consciousness was characterized by identifying information, but with no contextual markers (Tulving, 1984). Wixted (2007) describes models of recognition in memory, including the dual process and signal-detection models. The dual process model suggests that two processes underlie the recognition of information in memory, recollection and familiarity (Wixted, 2007). The process of recollection involves the identification of contextual cues relating to the remembered information (Wixted, 2007). In familiarity, information is not remembered in the original context in which they were remembered. Signal detection theory suggests that the distinction between "knowing" and "remembering" lies on a continuum (Wixted, 2007). The determination of "know" versus "remember" is based on the strength of the memory trace (Wixted, 2007). Information that is presented in a strong memory would cross the threshold and be considered "remembered" (Wixted, 2007). Lower levels of memory would be given the distinction of "known" (Wixted, 2007).

The amygdala and hippocampus are described as key components of episodic memory and are functionally interconnected (Dere, Pause, & Pietrowsky, 2010). The amygdala is found to be important in the processing of fear responses. In particular, it receives sensory information and determines the level of threat or danger it presents. Efferent connections to other structures and networks indicate that it facilitates behavioral responses to fear. Reciprocal connections with the hippocampus allow communication of the emotional context of memory both during encoding (Kensinger & Corkin, 2004) and during retrieval (Kensinger & Schacter, 2007). Other

key components to the episodic memory system include the anterior insular cortex, mesolimbic dopamine system, neuropeptide hormones, and neurokinins and cholecystokinin (as reviewed in Dere et al., 2010).

Exposure to acute stress is, under moderate conditions, associated with memory enhancement, especially of stress related information (Henckens, 2009). Upon exposure to a stressor, the LC facilitates the release of NE across a broad range of brain networks (Henckens, 2009). This encourages neural plasticity and results in a state of hypervigilance, increasing processing of information relevant to the stressor within the medial temporal lobe (Henckens, 2009). Henckens (2009) examined episodic memory for stressful video clips in humans while using fMRI. Results revealed that improved memory for details of the stressor occurred with lower levels of hippocampal activity. Authors suggest this may occur due to the co-occurring activation of the "ventral frontoparietal attention network," which facilitates the identification of salient information (Henckens, 2009). As a consequence, the information relayed to the hippocampus is likely to be fewer and more relevant. This is supported by the finding that better remembered information was coupled with less hippocampal activation (Henckens, 2009).

Alternatively, investigations into the retrieval of information encoded during conditions of emotional arousal reveal reports of enhanced recall, but decreased accuracy for contextual details (Rimmele, Davachi, Petrov, Dougal, & Phelps, 2011). Rimmele and colleagues (2011) extended previous research into the accuracy of recall for negative emotionally valanced stimuli through a study in which undergraduates were shown a sequence of scenes with neutral and negative emotional arousing content. As consistent with previous research, enhanced confidence in memory for the scenes eliciting negative emotion compared to neutral scenes was reported among participants. Accuracy for the contextual details of the pictures, such as background color

and peripheral objects, was lower than that for neutral scenes. Authors suggest this supports the hypothesis that emotional arousal enhances memory for information central to the stimuli, but decreases memory for contextual details.

HPA axis regulation. The role of the hippocampus in the regulation of the HPA Axis via a negative glucocorticoid feedback system has been studied extensively. Most of the research in this area has been completed utilizing animal models. This relationship was originally suggested as damage to the dorsal hippocampal region produced increased HPA Axis functioning, leading to the suggestion that it must perform an inhibitory function (Sapolsky, Krey, & McEwen, 1984). Follow-up studies revealed an influence of the ventral subiculum (vSUB), which is found to project from the hippocampus to the PVN of the hypothalamus (Herman, Cullinan, Morano, Akil, & Watson, 1995). Recent research describes the role of the anterior bed nucleus of the stria terminalis (aBST) in hippocampal mediated HPA Axis inhibition (Radley & Sawchenko, 2011). Radley & Sawchenko (2011) completed chemical ablation of GABAergic aBST neurons projecting from the hippocampus to the PVN in rats. Following this, they were subjected to 30 minutes of restraint stress. Two hours following stress exposure, HPA activation was measured and found to be increased, suggesting reduced inhibition. Furthermore, tracing revealed a common GABAergic pathway within the aBST where pathways from the mPFC and the vSUB projecting to the PVN. This suggests a point of integration where input from the mPFC and vSUB converge to promote inhibition of the HPA Axis.

Parasympathetic Nervous System

In contrast to the SNS, the parasympathetic nervous system (PNS) functions to return the body to a restful, relaxed state. The PNS is activated upon the detection of excess cortisol, NE,

and 5-HT in circulation. The PNS serves to facilitate responses which constrict pupils, stimulate salivation, reduce heart rate, reduce rate of breathing, stimulates digestion, and stimulates the bladder. These functions serve to re-establish homeostatic functioning following exposure to a stressor. Within the brain and the periphery, most sites receive innervation from both the sympathetic and parasympathetic systems (as reviewed in Winn, 2001). These connections provide reciprocal functions, with the sympathetic divisions displaying increased activity during stress and the parasympathetic displaying increased activation upon rest. Preganglionic neurons of the PNS are located within the brainstem and spinal cord. Axons extend from these neurons via cranial nerves 3, 7, 9, and 10. The most notable of these is the vagus nerve, which extends to the respiratory system, heart, gastrointestinal tract, liver, and kidneys.

As noted within Seyle's conceptualization of the stress process, negative feedback mechanisms serve to discontinue the stress response and return the body to a state of homeostasis. In the HPA axis, excess levels of cortisol inhibit the release of chemicals within the hypothalamus and the anterior pituitary. This is accomplished through the second, slower stress response mode, governed by the CRHR2 system (de Kloet et al., 2005). Glucocorticoid receptors are densely located within the PVN of the hypothalamus as well as pathways within the limbic system affiliated with the inhibitory GABA network surrounding the PVN (de Kloet et al., 2005). They respond to high levels of cortisol, inhibit the stress response, and facilitate recovery. In addition, they promote memory storage for use in future situations (de Kloet et al., 2005).

Glucocorticoid Negative Feedback System. Inhibition of the stress response and a return to homeostasis is achieved via a complex negative feedback system within several brain regions that play key roles in the response, including the mPFC and the hippocampus. A coordinated glucocorticoid negative feedback system allows these structures to inhibit the

activity of the HPA Axis, reducing the amount of glucocorticoid circulation (Ulrich-Lai & Herman, 2009). Research indicates the hippocampus as a key structure in the inhibition of the HPA Axis (Herman et al., 2003). Following hippocampal stimulation, a decrease in the secretion of glucocorticoids has been noted in studies with rodents and humans (as reviewed in Ulrich-Lai & Herman, 2009). Damage to the hippocampus has been associated with an increase in circulating cortisol levels (Ulrich-Lai & Herman, 2009). The mPFC also plays a role in the inhibition of the HPA Axis (Ulrich-Lai & Herman,2009). Evidence suggests that it is particularly sensitive to psychogenic stressors, inhibiting responses to those types of stressors with greater frequency than other types (as reviewed in Ulrich-Lai & Herman, 2009). The dmPFC is particularly implicated in the inhibition of the stress response (as reviewed in Ulrich-Lai & Herman, 2009). In addition, projections from the raphe nucleus, extending to the PVN region of the hypothalamus contribute to the inhibition of the HPA Axis (Lowry, 2002).

Chronic Stress

When faced with acute stressors, the physiological and psychological aspects of the stress response serve an adaptive function, promoting behavior likely to be effective in coping. As previously detailed, stress responses are most adaptive to short lived stressful situations of mild to moderate intensity. Modern life, however, often involves exposure to ongoing stressful circumstances without a definitive end that are often accompanied by frequent activations of the acute stress response. Examples of these types of stressors include, working in an unsatisfying job for a demanding boss or living in a poor neighborhood where frequent threats to safety occur. This frequent triggering of the physiological stress response results in damage to several systems within the body, including the cardiovascular system, immune system, and central nervous system. The term “chronic stress” refers to the plethora of reactions within the body following

exposure to these recurrent stressors, which increase vulnerability to disease of mental health disorders. Of particular interest in the study of neuropsychological functioning following exposure to chronic stress is the damaging effects produced on three structures, the hippocampus, amygdala, and prefrontal cortex.

McEwen (2002) discusses the process by which chronic stress increases vulnerability to disease in his theory of "allostasis" and "allostatic load." Allostasis expands the understanding of homeostasis mechanisms by further describing the brain as a central mediator for the stress response process. Ganzel, Morris, and Wethington (2010) describe the functioning of allostasis as a) an evaluation process involving perceived controllability and environmental conditions, b) a mechanism by which a response can occur in anticipation of a stressor, c) a process by which adaptations to the environmental conditions may occur over time, and d) allows for the prediction of a response given environmental demands. When presented with a stressor, allostasis allows for "allostatic accommodation," which describes the dynamic process in which different stressors elicit varying homeostatic set points. This allows for a variation of physiological responses based on the environmental conditions. When this process of allostatic accommodation breaks down or does not work effectively, "wear and tear" on the physiological mechanisms of stress responding become apparent (McEwen, 2004). These effects are described as "allostatic load."

Three types of physiological responses that contribute to the development of allostatic load, including the experience of frequent stress, failed shut down of stress response, and inadequate responses to the stressor. When exposed to repeated stressors, the physiological stress response occurs repeatedly and can lead to damage within the brain and body (McEwen, 2002). Stressors of this nature include, among others, poverty and childhood abuse. To illustrate this

process, McEwen (2002) describes research involving the effects of recurrent stressors on the development of heart disease. The introduction of a monkey into a novel social group results in the experience of stress as vying for a position in the social hierarchy is initiated (Kaplan, Petterson, Manuck, & Olsson, 1991). Monkeys were frequently moved into new social groups, continually producing an increase in blood pressure. Eventually, an increase in heart attack risk became evident among monkeys most frequently placed in novel social systems, evidencing the role of chronic stress on allostatic load (Kaplan et al., 1991).

In another type of situation, the individual does not adjust to the stressor, and experiences prolonged activation of the HPA Axis, even when the stressor is no longer present (McEwen, 2002). In these situations, the individual fails to initiate allostatic accommodation, resulting in continued stress response activation, despite having opportunities to adjust to the stressor (McEwen, 2002). Kirchbaum et al., (1995) conducted a study that suggested the role of personality factors in determining if a situation if perceived to be stressful. In this study, male college students completed a public speaking task and arithmetic task (Trier Social Stress Test) upon which their performance would be evaluated. Upon repeated administration of the task, most participants evidenced stabilization in circulating cortisol levels following the task. Individuals with lower scores on measures of self-confidence and self-esteem, however, did not display a reduction in cortisol levels upon repeated administration of the stressor.

Finally, if the response of the SAM and HPA axes is not appropriately inhibited, prolonged activation of the stress response is likely to occur (McEwen, 2002). Gerin and Pickering (1995) showed that mechanisms behind this may be related to genetic traits as undergraduate students provided with an arithmetic test that displayed elevated heart rate and

blood pressure following completion were found to have an increased likelihood of having two parents diagnosed with hypertension.

In all three of these situations, the stress response is activated chronically, resulting in excess amounts of glucocorticoids and catecholamines to remain within the brain (McEwen,2002). The chronic activity of the stress response, and co-occurring increases in hormones and catecholamines, can result in negative health consequences and damage within the brain and body (McEwen, 2002). Alternatively, a final situation can result in the experience of allostatic load, the underproduction of glucocorticoids in response to stress (McEwen, 2002). This prevents the action of cortisol in the regulation of the immune system and the reduction of inflammation, resulting in conditions like allergies, asthma, and autoimmune disorders (McEwen, 2002).

Contrada & Baum (2010) reviews the central and peripheral responses to chronic stressors in terms of a broad chronic stress response network, which details the differential response that occurs when exposed to chronic stressors. Chronic stress is defined as stressors that are "sustained" or "repeated," of very high intensity, or those that cannot be coped with according to the behavioral resources available (Contrada & Baum, 2010). The frequent or intense activation of the stress response results in increased or prolonged exposure of structures within the central and peripheral nervous system to hormones and chemicals utilized in the acute response, including glucocorticoids, 5-HT, DA, and NE (Contrada & Baum, 2010). Eventually, exposure leads to structural or functional changes that result in maladaptive behavioral correlates for the individual, such as anxiety, excessive fear, or emotional difficulties (as reviewed in Contrada & Baum, 2010). Damage to several key structures and systems are found in response to

chronic or intense stressors, including the amygdala, HPA Axis, PFC, and hippocampus (as reviewed in Contrada & Baum, 2010).

The following will review the effects of chronic stress upon structures and systems involved in neuropsychological functioning related to memory and executive functioning. Of particular interest are the hippocampus, amygdala, and prefrontal cortex.

Hippocampus

Studies have shown the effects of chronic stress on hippocampal volume, suggesting that increased levels of glucocorticoids result in cell loss, particularly in the CA3 region (Conrad, Jackson, & Wise, 2004). The mechanism behind these findings has been suggested to involve disturbance within the glutamate uptake process, as increased levels of glucocorticoids over the course of a sustained stressor result in the inhibition of glutamate transporters (Virgin, Ha, Packan, Tombaugh, Yang et al., 1991). This results in increased levels of glutamate present within the synapses of hippocampal neurons, which become vulnerable to "excitotoxin-induced calcium mobilization and calcium-triggered proteolytic events" (Virgin et al., 1991, p. 1427). These events serve to increase death or damage of neurons within the hippocampus.

Sapolsky and colleagues (1985) studied the effects of glucocorticoid neurotoxicity on the aging process within the hippocampus of rats. Prolonged exposure (over the course of 2 weeks to 3 months) of glucocorticoids at levels consistent with those found during realistic stressors produced structural changes in the CA3 region of the hippocampus. Differential effects of acute (2 weeks) versus chronic (3 months) administration were revealed. Following acute administration, decreased numbers of receptors within the CA3 region of the hippocampus were noted compared to controls. Levels subsequently returned to normal following one week without

stress. Among rats exposed to chronic levels of stress hormones, reduced numbers of both receptors and neuronal number were found within the CA3 region compared to controls. Unlike rats in the acute condition, those in the chronic condition did not evidence a reversibility of these damaging effects. Changes in hippocampal structure following chronic stress accelerate the natural aging process, which involves gradual loss of receptor numbers within the hippocampus (Kerr, Campbell, Applegate, Brodish, & Lanfield, 1991). In addition, these changes increase subsequent responsivity to stressors, due to reduced ability to inhibit the HPA Axis (Sapolsky, Krey, & McEwen, 1984; Sapolsky, Krey, & McEwen, 1985).

Conflicting results relating to elevations in glucocorticoids producing damage to the hippocampus lead to the introduction of the "glucocorticoid vulnerability hypothesis." This theory differs from the cascade hypothesis in that it suggests that glucocorticoid elevations present within the hippocampus during chronic stress increased vulnerability to subsequent damage, but these elevations do not need to be present at the time of the metabolic event (Conrad, 2008). In a study of the effects of chronic stress on rat hippocampi, rats were subjected to restraint stress for six hours per day, for 21 days (Conrad, Jackson, & Wise, 2004). Following a three to four day cessation of restraint, a neurotoxin, ibotenic acid (IBO) was administered. Rats that had been exposed to the chronic stress condition evidenced increased damage within the CA3 region of the hippocampus, suggesting that chronic stress and co-occurring glucocorticoid elevations result in an extended period of time in which the hippocampus is vulnerable to damage from subsequent events (Conrad et al., 2004).

Atrophy of dendritic spines among hippocampal neurons has been suggested as the mechanism behind the increased vulnerability produced following chronic stress (Conrad, 2008). Several animal studies indicate that, following exposure to chronic stressors, hippocampal

dendrites within the CA3 region of the hippocampus atrophy and retract (Wooley, Gould, & McEwen, 1990; Magarinos & McEwen, 1995; Magarinos, McEwen, Flugge, & Fuchs, 1996; Sunanda, Rao, & Raju, 1995). When this occurs, synapses within the brain are pulled apart, reducing the ability to communicate among neurons as dendrites are reduced in complexity and in length (Sapolsky, 2004). Evidence also suggests reduction in number of synapses within the CA3 region (Sousa, Lukoyonov, Madiera, Almeida, & Balbosa, 2000). These effects occur following exposure to extreme elevations in glucocorticoids or when experiencing chronic levels of stress, but are not shown in exposure to acute stress (Conrad, 2006). These effects are reversible, however, as regrowth emerges following four days without stress exposure (Vyas, Pillai, & Chattarji, 2004).

Increased levels of glucocorticoids have also been found to decrease long term potentiation within the hippocampus, which is key in memory functioning and learning (Diamond, Bennet, Fleshner, & Rose, 1992). Previous studies indicate that CA1 areas of the hippocampus and the dentate gyrus are responsible for the effects of long term potentiation (Pavlides, Nivon, & McEwen, 2002).

Research suggests that the hippocampus is a remarkably plastic structure, continually producing new cells via neurogenesis (Schloesser, Manji, & Martinowich, 2009). The process of neurogenesis is found to originate within the dentate gyrus, a site for the integration of sensory information innervating the hippocampus (Schloesser, 2009; Djavadian, 2004). New cells are created within the subgranular layers of the dentate gyrus and become incorporated into the structure (as reviewed in Djavadian, 2004). In addition, these new cells develop axons which project into the CA3 region of the hippocampus (as reviewed in Djavadian, 2004). Neurogenesis is largely dependent upon serotonin functioning. The dentate gyrus is densely innervated with

serotonergic fibers, which bind to a variety of serotonergic receptors (as reviewed in Djavadian, 2004). In rodents, serotonin depletion through administration of a neurotoxin results in a reduction in new cells created via neurogenesis (Brezun & Daszuta, 1999). These effects are long lasting, with some rodents taking up to three months for rates to return to that of controls (Brezun & Daszuta, 1999). Decreased proliferation of new cells has been related to the exposure to stressors in rodents (Schloesser et al., 2009). The hippocampus serves to inhibit the firing of the HPA axis. These new cells are implicated in this process (Schloesser et al., 2009). As a result, the reduction of neurogenesis resulting from excess levels of glucocorticoids has been suggested to reduce the hippocampus' ability to inhibit the initiation of the HPA axis (Schloesser et al., 2009). This further increases the circulating levels of cortisol, potentially contributing to hippocampal cell death. Recent research suggests that early childhood stress in rodents may serve to increase neural plasticity in adulthood when encountering stress (Oomen et al., 2010; Champagne, Bagot, van Hasselt, Ramakers, & Meaney, 2008). Functional performance on a learning and memory task was impaired relative to maternal separation, suggesting that reduced neurogenesis has functional implications in adulthood among rodents (Oomen et al., 2010).

Given the role of the hippocampus in memory related processes, the functional implications of damage to this region following the experience of chronic stress is important to examine. The following will review relevant research relating the memory functioning, specifically explicit memory, following chronic stress exposure.

Explicit memory. Research on memory functioning has revealed functional deficits in the performance of rodents on spatial memory tasks following chronic administration of glucocorticoids (Bodroff, Humphreys, Lehman, Diamond, Rose, Meaney, 1995; Luine, Villegas, Martinez, & McEwen, 1994; Bardgett, Taylor, Csernansky, Newcomer, & Nock, 1994; Endo,

Nshimura, & Kimura, 1996). Activation of minerocorticoid receptors (MR), which mediated the consolidation of stimuli into memory, at baseline level is necessary for new learning to occur (as reviewed by Alderson & Novak, 2002). This process is hindered, however, upon chronic activation of glucocorticoid receptors and new learning is impaired . While research has indicated deficits among rodents in the acquisition and storage of information in long term memory following glucocorticoid administration, evidence suggests that elevations of these stress hormones also influence retrieval of previously learned information (deQuervain, Roozendaal, & McGaugh, 1998). Functional imaging procedures suggest reduced blood flow to the medial temporal lobe may provide a mechanism for these effects noted within memory retrieval following increased glucocorticoid administration (deQuervain et al., 2003).

Studies have also revealed functional impairments on explicit memory tasks associated with elevations of glucocorticoids in humans. Newcomer et al. (1999) evaluated the effect of cortisol on declarative memory functioning among healthy, human volunteers recruited from the community. Two doses of cortisol were provided, which aimed to differentiate effects of mild and severe stress. Following four days of cortisol exposure, decrements in declarative memory were reported. Upon cessation of cortisol administration, performance returned to normal. Research also indicates impaired declarative memory functioning when exposed to more natural stressors, like social evaluation. Upon exposure to a psychosocial stressor, the Trier Social Stress Test, individuals evidencing cortisol elevations experienced deficits in declarative memory (Kirshcbaum, Wolf, May, Wippich, & Hellhammer, 1996). These impairments evidenced a linear relationship with glucocorticoid levels.

Discrepancies within the literature are evident as other studies have failed to find relationships between glucocorticoid levels and memory performance (Schmidt, Fox, Goldberg,

Smith, & Schulkin, 1999). It is unclear if they represent different effects on memory processes (encoding, storage, retrieval), length of glucocorticoid elevation, and if the elevation occurred through exposure to a natural stressor or was administered (Alderson & Novak, 2002). In addition, research indicates several factors that represent individual vulnerability to the damaging effects of stress, including personality and developmental stage.

In animal models, individual traits similar to that of personality in humans have been shown to influence the level of vulnerability to stress related impairments in memory functioning including novelty reactivity and anxiety. Novelty reactivity is measured in rodents via level of loco motor activity displayed when placed in a novel environment (Kabbaj, Devine, Savage, & Akil, 2000). Touyarot, Venero, & Sandi (2004) examined the role of novelty reactivity upon chronic stress adaptation in both high and low reactivity rats. Both groups of rats were exposed to a psychosocial stress condition in which they were introduced into a novel environment with another rat on a daily basis for 21 days. Rodents identified as highly reactive displayed decreased performance on a spatial memory task. In addition, anxiety levels have been shown to increase risk for detrimental effects on memory functioning following chronic stress exposure in rodents (Herrero, Sandi, & Venero, 2006).

Research with rodents also indicates that early childhood stress may result in deficits in performance on hippocampal dependent memory tasks later in life (Avishai-Eliner, Brunson, Sandman, & Baram, 2002). Chronic stress occurring during childhood may contribute to late emerging deficits in memory functioning as a result of reduced long term potentiation and dendritic atrophy during aging (Brunson, Kramár, Lin, Chen, & Colgin,2006). Evidence suggests that the mechanism behind these effects is corticotrophin releasing hormone (CRH) within the

hippocampus, as antagonists of these receptors reduce the late onset development of neurological damage in rodents (Ivy et al., 2010).

Prefrontal Cortex

Medial prefrontal cortex. Within the brain, the medial prefrontal cortex (mPFC) has a high concentration of glucocorticoid receptors (Sánchez, Young, Plotsky, Insel, 2000). As such, it is sensitive to damaging effects following chronic elevations of glucocorticoids. Animal models have revealed that, in rodents, chronic restraint stress (six hours a day for three weeks) is associated with dendritic changes within the mPFC (Cook & Wellman, 2004). Specifically, reduced dendritic branch number and length were noted (Cook & Wellman, 2004; Radley et al., 2004). In addition, repeated stress among rodents results in dendritic spine loss, reducing the number of synapses within the mPFC (Radley et al., 2004). Damage to the mPFC , particularly the anterior cingulate is found to reduce functioning on complex attention tasks involving set shifting in rodents (Liston et al., 2006). Among humans, levels of perceived stress have been associated with decreased performance on measures of set shifting among outpatients (Ohman, Nordin, Bergdahl, Birgander, & Stigsdotter, 2007) and college students (Orem, 2008). Orem (2008) assessed college students according to self-reported levels of stress, as measured by the Perceived Stress Scale (PSS), and a measure of set shifting, Trails B on the Trail Making Test. Results indicated a negative relationship among scores on the PSS and Trails B, such that increased levels of perceived stress predicted impaired performance on the set shifting ability. Specifically, individuals with increased PSS scores took longer to complete the set shifting task.

Dorsolateral prefrontal cortex. The experience of chronic stress, like childhood poverty, is associated with reduced working memory performance in adulthood (Evans &

Shamberg, 2009). Further research has indicated that the relationship among childhood stress and working memory is moderated by the estimated level of maternal responsiveness reported by participants on a responsiveness measure (Doan & Evans, 2011). In addition, trauma exposure is related to working memory performance. In a study of working memory functioning among psychiatric outpatients, trauma exposure was predictive of working memory performance, independent of anxiety or depression levels (El-Hage & Gaillard, 2006). Familial exposure to trauma may present an increased risk for executive functioning deficits, including working memory, compared to non-familial trauma (DePrince, Weinzierl, & Combs, 2009). Among college women with childhood sexual abuse histories, decreased performance on a response inhibition task has been noted compared to a control group of non-trauma exposed students (Navalta, Polcari, Webster, Boghossian, & Teicher, 2006).

In addition to neuropsychological deficits, exposure to chronic stress or trauma contributes to the vulnerability in developing mental health disorders, like PTSD, depression, and anxiety. Stress plays a role in the development of these mental health disorders through structural and hormonal changes.

Stress Related Disorders

Following exposure to chronic stressors, a multitude of negative health consequences are possible due to the effects of allostatic load. Theories surrounding the mechanism behind these effects have focused on the role of cortisol (Miller, Chen, & Zhou, 2007). In most of these disorders, excess levels of cortisol are brought about by the stress response mediated by the HPA Axis, resulting in damage to systems within the brain and body (Miller et al., 2007). Chronic stress is typically defined as a stressor that has occurred for a period of at least one month (Miller

et al., 2007). In addition, chronic stress could be defined as an event shorter in duration, but likely to provide longer lasting distress (Miller et al., 2007). In a meta-analysis review, Miller et al. (2007) attempted to delineate the consequences of chronic activation of the HPA axis, as seen in exposure to chronic stress. Results suggest a complex relationship between the type of stressor experienced, factors within the individual, and the increase or decrease of activity within the HPA Axis (Miller at al., 2007). These factors include the time since onset of the stressor, nature of stressor, level of controllability, experience of shame or loss, and individual psychological symptomology (Miller et al., 2007).

Post- Traumatic Stress Disorder (PTSD)

PTSD is a complex disorder, characterized by dysfunctional reactions to stressful events. Responses to trauma in this disorder include symptoms of re-experiencing the event, behaviors of avoidance or numbing of emotions, and increased levels of arousal (American Psychiatric Association, 2000). These symptoms present following events of extreme trauma, in which individuals perceive themselves or others at risk for death, injury, or diminished Integrity (APA, 2000). During the trauma, they experience intense fear, helplessness, or horror.

Diagnosis. With the inclusion of the classification of the diagnosis of PTSD in the DSM-III (1980), conceptualizations of traumatic experience were advanced. The diagnosis included specific criterion regarding the experienced event, which shifted the focus of the cause of trauma to an external force, rather than a representation of an intrapsychic phenomenon. Specifically, the criterion to be considered a traumatic event included that it would "evoke significant symptoms of distress in almost everyone." In addition, it listed three clusters of symptoms, including re-experiencing, avoidance/numbing, and arousal. Debate regarding the criterion for the stressor

resulted in restricting included events to those that are "outside the range of usual human experience" in the DSM-III-R (1987). The number of required symptoms was also expanded in the revised version.

Following its original inclusion in the Diagnostic and Statistical Manual in 1980, the criteria for PTSD has been altered due to a vast amount of research evidence uncovered within the past three decades. In the DSM-IV (American Psychological Association, 2000), stressors sufficient in the triggering of PTSD were no longer defined as "outside the range of usual human experience." The criterion describing traumatic events became more objective with the specification of two distinct factors, the qualifying stressor and the subjective experience of intense fear, helplessness, or horror. These stressors can occur through direct experience, witnessing, or other means of confrontation. This change reflected research findings that pointed to the importance of the individual interpretation of the event. In addition, the current diagnostic criteria for PTSD includes three categories of symptoms, re-experiencing, avoidance/numbing, and arousal.

Re-experiencing. Individuals diagnosed with PTSD experience vivid memories of the event, termed re-experiencing. These memories are characterized by their intrusiveness, intensity, and distortion (APA, 2000). Trauma memories can be triggered by minute reminders of aspects of the event and may be distorted in that, individuals may feel as though they are actually experiencing the trauma event in the present and may not recognize it as happening in the past. They may re-experience, with intensity, many aspects of the trauma, including emotions, localized pain or discomfort, hallucinations, or dissociation. Traumatic memories may produce psychological and physiological distress, often as a result of the external and internal cues resembling the event. In an attempt to better describe these symptoms, Kekledze (2011) describe

four distinct types of flashback symptoms: “1) dreams and nightmares; 2) dreams whose content after waking “leave the person under such a strong impression that they are disorientated in relation to their surroundings for some period of time;” 3) visualizations on the background of clear consciousness, without loss of contact with the surroundings (“intrusive memories”); 4) “unconscious flashbacks” in which the person again “experiences” the traumatic event, losing connection with ongoing activities for a short time.”

Traumatic memories intrude upon the life of the individual by interrupting the function of everyday life. In particular, the sleep patterns of those with PTSD are disturbed (Harvey, Jones, & Scmidt, 2003). They often have difficulty falling asleep and experience intense and upsetting nightmares of the trauma, which disrupts their sleep cycles and makes it difficult to fall back asleep. One possibility for this disturbance may be related to a decreased capacity to suppress intrusive images or memories when in a pre-sleep and sleep state (Harvey et al., 2003). The result may be more intrusive memories, which may increase anxiety and arousal and make it difficult to fall and remain asleep.

Avoidance/numbing. Clients with PTSD experience difficulty completely controlling the occurrence of trauma memories or flashbacks. As a means to obtain control over their presence, people often exercise avoidance behaviors. These behaviors involve avoiding potential trauma related reminders. They may avoid discussion, thoughts, and feelings associated with the trauma (APA, 2000). In addition, their memories of the event may be incomplete, evidenced by gaps or an inability to recall certain aspects of the trauma. They may also shy away from activities, people, and places that may serve as reminders.

Individuals with PTSD may display a restricted range of affect, often labeled emotional numbing. This emotional state is characterized by detachment from others, disinterest in pleasurable activities, and deficient ability to experience and relate emotion (Miller & Litz, 2004). This state is not viewed as a constant deficiency in expressing positive emotion, but rather a reaction to the presentation of trauma related cues (Litz, Orsillo, Kaloupek, & Weathers, 2000). Following trauma reminders, the tendency to emotionally disengage may serve a protective function, shielding the individual from further exposure to negative experiences or emotions (Miller & Litz, 2004). Those with PTSD may also have an incomplete or pessimistic view about their future. For example, they may not expect to have a career, marriage, or a normal life span (APA, 2000).

Arousal. People with PTSD are often in a state of hyperarousal, indicated by their rigid reactions to stimulus in their environment with little interpretation of their content (van der Kolk, McFarlane, & Weisaeth, 1996). Hyperarousal is an important factor of PTSD as it has been suggested as a key factor in the maintenance of posttraumatic stress symptomology (Schell, Marshall, & Jaycox, 2004). They exhibit increased arousal, noted by an accelerated heart rate, in response to trauma related stimuli (Elsesser, Sartory, & Tackeburg, 2004). In addition, higher levels or corticotrophin-releasing-factor (CRF), which initiates the stress response, have been associated with PTSD, as well as an increased level of adrenaline and noradrenaline (Scott & Stradling, 2001). As a result of excess CRF, clients with PTSD will often respond to neutral stimuli in their environment and perceive them as potential threats. They will experience "false alarms," evidenced by intense negative emotions (fear, anxiety, anger) when no true threat exists. These feelings may be maintained following the traumatic event and reminder cues, as a result of the increased rate of adrenaline. As a result, these individuals may react with their environment

by becoming irritable with others. They may also have difficulty concentrating, may be hyper vigilant, and may experience difficulties with sleep due to their increased arousal (Scott et al., 2001).

The diagnostic criterion for PTSD set a standard for the number of symptoms in each core cluster that are sufficient to receive diagnosis. Criterion B requires the event to be persistently relived by at least one symptom of re-experiencing. In addition, the client must evidence three or more symptoms of avoidance or numbing, as required by Criterion C. Two or more symptoms of increased arousal are needed to fulfill Criterion D. These symptoms must be present for more than three months in order to receive diagnosis. Symptoms must also result in significant impairment in social, occupational, or other important areas of functioning. In addition to diagnosis, it is helpful to specify whether the symptoms are considered acute (present less than three months), chronic (present three months or more), or if they are delayed in their onset (symptoms presented at least six months after the stressor).

Differential Diagnosis. With the DSM-IV-TR, additional stress related disorders are presented. These help to further distinguish among trauma related symptom presentations.

Adjustment Disorder. The diagnostic features of Adjustment Disorder account for psychological responses to an identifiable stressor, evidenced by emotional or behavioral symptoms. Unlike PTSD, the stressor can be of any severity and the reactions to the stressor can be less severe. This diagnosis is used for stressors of a lesser severity than the required extreme traumatic event for PTSD. The classic symptoms of PTSD, like re-experiencing, are not present. Adjustment Disorder can be displayed by a wide range of symptoms, including those which are considered to be abnormal given the minimal severity of the event, those which cause significant

distress, or those which are a result of multiple stressors (marital problems, work difficulties, etc.). This diagnosis can only be applied for symptoms lasting less than six months after the occurrence of the stressor. A diagnosis of chronic Adjustment Disorder can be given beyond six months in the presence of multiple or chronic stressors.

Acute Stress Disorder. A diagnosis of Acute Stress Disorder can be given during the month following the stressor when a diagnosis of PTSD cannot be given. This diagnosis can be given if symptoms are present two days after traumatic event, but cannot extend beyond one month. If the symptoms persist beyond this time span, a diagnosis of PTSD may be given. The stressors considered sufficient for the disorder are the same as those for PTSD. During or following the event, the individual must have experienced three dissociative symptoms, such as subjective sense of numbing or absence of emotional response, reduction in awareness of their surroundings, derealization, depersonalization, or dissociative amnesia. In addition, the individual must experience at least one symptom from each other cluster of symptoms of PTSD (re-experiencing, avoidance, and hyperarousal). Like PTSD, to be diagnosed with Acute Stress Disorder, symptoms must result in significant impairment in areas of functioning.

Neurobiology of PTSD. A complex interaction of neurobiological systems produce a unique constellation of effects that increase the risk of developing psychological symptoms found in PTSD. PTSD is characterized by alterations in the systems responsible for regulating the stress response within the brain, namely the HPA Axis and the LC/NE system.

Symptoms of PTSD involve overactivity of the stress response within the body, including symptoms of hypervigilance, agitation, and anxiety. This occurs largely through overactivity of the HPA Axis, which controls the physiological stress response via neurochemical interactions

between the hypothalamus, pituitary gland, and adrenal gland. In addition, the limbic system, the guiding force for self-preservation, functions abnormally in those with PTSD (Fieldman et al., 2009). In particular, the hippocampus and amygdala deviate in their response to stress when posttraumatic stress symptomology is present. Neurohormonal abnormalities in the production of catecholamine's, corticosteroids, serotonin, and opioids have been proposed to interfere with the effectiveness of the stress response. Taken together, these anomalies have been evidenced to play a role in the development and maintenance of PTSD.

HPA Axis. Studies of the effects of stress and trauma on the functioning of the HPA Axis have revealed mixed results. While the mechanisms of the acute stress response suggest an elevation in the circulating level of cortisol should be noted following exposure to a stressor, this is not always the pattern revealed. This suggests that abnormalities of reactivity in the HPA Axis are observed in response to chronic stress, PTSD, depression, and trauma. Decreased levels of cortisol have been reported in some studies (Mason, 1986; Yehuda, Southwick, Nussbaum, Wahby, & Giller, 1990; Yehuda et al., 1995; Trestman, Levengood, & Siever, 1996;), whereas increased levels have been reported in others (Pitman & Orr, 1990; Bremner et al., 1997; Lemieux & Coe, 1995; De Bellis, Baum, Birmaher, Kashavan, & Eccard, 1999). Several factors have been suggested to play a role in producing this variability, including psychiatric diagnosis status, pre-existing vulnerabilities, and methodological limitations of research.

Studies reporting the influence of PTSD status upon HPA Axis reactivity, as measured by circulating levels of cortisol, have produced mixed results. Overall, studies evaluating HPA Axis reactivity among those diagnosed with PTSD have suggested cortisol suppression (Miller et al., 2007; Mason et al., 1986; Yehuda et al., 1990, 1995, 1996). This suggests decreased activation of the HPA Axis. Delahanty, Ramonde, Spoonster, and Cullado (2003) evaluated motor vehicle

accident victims within the emergency room by monitoring cortisol and catecholamine levels throughout a 15 hour period. Results indicated that, among those developing PTSD, a decrease in urinary cortisol levels was noted. In addition, epinephrine and norepinephrine levels were lower in the PTSD group. Regression analysis revealed that initial cortisol levels predicted 9% of variance related to the diagnosis of PTSD, suggesting that pre-existing levels of cortisol presented a vulnerability to symptom development.

Low baseline levels of cortisol are not unique to the diagnosis of PTSD as these effects are also seen in those experiencing chronic stressors, but who do not exhibit psychiatric symptoms (Fries, Hesse, Hellhammer, & Hellhammer, 2005; Carpenter, 2007). Carpenter et al. (2007) evaluated ACTH levels in response to a psychosocial stressor, the Trier Social Stress Test, in individuals with a history of moderate to severe child abuse. Results indicated reduced ACTH levels in response to CRH stimulation, possibly suggesting a permanent down regulation in receptors on the pituitary gland.

In response to these findings, researchers began focusing on the medical condition of hypocortisolism. Hypocortisolism is characterized by inefficiency of cortisol production. It is hypothesized to occur following hyperactivity of the HPA Axis prompted by chronic stress and is thought to be a result of an "overcorrection," in which high levels of cortisol are counteracted by a continuous drop in concentration (Fries et al., 2005). This may serve a protective and allow for a compensatory effect in the wake of frequent HPA Axis activation (Fries et al., 2005). As a result, it would help to compensate for frequent increases in levels of cortisol and buffer against the negative effects of hypercortisolism (Fries et al., 2005).

Research suggests that childhood trauma, including physical and sexual abuse and neglect, result in future ACTH and cortisol abnormalities (as reviewed in Watts-English, Fortson, Gibler, Hooper, & de Bellis, 2006). Studies of HPA Axis functioning in children exposed to maltreatment generally reveal an increase in cortisol levels (de Bellis et al., 1999; de Bellis, Keshavan, & Harenski, 2001; El-Sheikh & Harger, 2001; Gunnar & Donzella, 2002; Flinn & England, 1995). Studies involving adolescents and adults, however, often report decreased levels of cortisol (as cited in De Bellis, 2005). Child abuse likely contributes to overresponsivity to future stressors rather than elevated base levels. Elzinga et al. (2003) found increased levels of cortisol upon exposure to trauma related cues in women diagnosed with PTSD who had been abused in childhood. Upon recovery from the stressor, cortisol levels returned to normal baseline levels. Before and after exposure to trauma related cues, PTSD symptoms did not correlate with cortisol levels. This suggests that PTSD symptomology may not predict cortisol during rest. Authors suggest that measurement of cortisol levels may be misleading as they rely on conceptualizations of a static hyper or hypocortisolism. In reality, cortisol levels may fluctuate widely due to exposure to traumatic reminders or other stressors that elicit HPA Axis reactivity. As such, reduced levels may be captured during a refractory period.

Discrepancies within the literature in relation to HPA Axis effects may be due, in part, to methodological anomalies. In particular, differences in the time course since the occurrence of trauma or stress may account for some of the variability. Miller et al. (2007) indicated that, according to a review of the available literature, the time since onset of the traumatic stressor likely plays a role in determining cortisol levels and is negatively correlated with cortisol levels. When chronic stressors are found to still be occurring, cortisol levels continue to be high.

Conflicting results within the literature may represent a failure to take these timing issues into consideration.

Variability among findings in regards to HPA Axis reactivity have created confusion regarding the role of stress, particularly chronic stress and trauma, upon the release of cortisol. Miller et al. (2007) attempt to address these issues through a meta-analysis on possible mediating or moderating factors between stress and cortisol reactivity. They suggest that the variability may be accounted through examining the time since the onset of the stressor, nature of the threat, emotional factors, perceived controllability, and psychiatric factors.

Activity of the HPA Axis has been proposed to follow a time course in which high levels of cortisol are released following exposure to a stressor and these levels rebound to a level that is below that of normal baseline levels over the course of time following the stressor. In a meta-analysis of studies evaluating cortisol levels in response to stress, Miller et al. (2007) report a negative relationship between cortisol levels and time since the onset of a stressor. As most studies evaluate cortisol levels at only one point in time, it is difficult to capture a picture of the time course of cortisol reactivity following stress exposure. Several longitudinal studies evaluating the impact of stressors on cortisol levels have been completed. Anisman (2001) evaluated salivary cortisol levels among those experiencing a severe ice storm. The experienced distress of the event was correlated with increased cortisol levels one month following the event. At a one year follow-up, cortisol levels were similar to controls.

Miller et al. (2007) also suggests the role of the type of stressor on cortisol levels. According to results of the meta-analysis, a high and flat pattern of cortisol concentrations are reported following events that threaten physical well-being, like combat. This pattern was also

confirmed in studies of traumatic stress. Results indicate that social stressors result in peak cortisol levels occurring during different times of the day, like the morning and the afternoon or evening. Social evaluative threats are shown to increase day time cortisol levels, possibly as individuals are encountering social situations or engaged in rumination regarding social performance.

Norepinephrine (NE). The LC facilitates the release of NE following exposure to stress, which facilitates vigilance, attention to novel stimuli, and cardiovascular responsivity (as reviewed in Vasterling & Brewin, 2005). Upon receiving sensory information, the LC sends signals throughout a wide network of NE receptors located in the amygdala, hippocampus, hypothalamus, and PFC. Activation of these regions is associated with "perceiving, evaluating, remembering, and responding to potentially threatening situations" (Vasterling & Brewin, 2005, p.28).

Within animal models, frequent exposure to uncontrollable stressors served to increase the chemical release of NE throughout the brain, resulting in symptoms of anxious arousal (as reviewed in Vasterling & Brewin, 2005). High levels of NE serve to potentiate the response of the amygdala to stress, which facilitates processes of fear conditioning and memory consolidation for information with high emotional content . Studies suggest that NE activity within the basolateral nucleus of the amygdala plays a central role in learning and consolidation of memory for emotional content (Ferry, Roozendal, & McGaugh, 1999). Evidence also suggests that epinephrine released from the periphery potentiates the release of NE within the amygdala (Ferry et al., 1999). Enhancement of memory functioning occurs via activation of postsynaptic beta and alpha-1 adrenoceptors within the amygdala(Ferry et al., 1999). Presynaptic alpha-2 adrenoreceptors located within this region are shown to impair memory functioning when

activated (Ferry et al., 1999). Evidence of this effect comes from research in which administration of yohimbine, an alpha-2 adrenorecptor antagonist, within the BLA produced enhanced retention of information (Ferry et al., 1999). Other research has found that administration of an alpha-2 adrenoreceptor agonist is correlated with reduced NE activity within the amygdala (Ferry, et al., 1999).

NE functioning in PTSD is measured via several different methods, including acquiring baseline levels, 24 hour plasma levels, receptor numbers, and catecholamine challenge paradigms. The following will review the findings from these various methodologies in relation to PTSD.

Although baseline levels of NE are not found to be elevated among those with PTSD, evidence suggests that individuals diagnosed with PTSD display increased noradrenergic reactivity in response to stressors (Southwick, 2010; Onur, Walter, Schlaepfer, Rehme, Schmidt, et al., 2009). Previous research indicates a reciprocal relationship between the BLA and the LC in facilitating the stress response (as cited in Oner et al., 2009). As such, it is suggested that, when both become disinhibited, a hyper-responsivity to stress occurs (Onur et al., 2009). In those diagnosed with PTSD, heightened activity of the BLA and LC circuitry is hypothesized to lead to symptoms of exaggerated fear responsivity (Oner et al., 2009). To evaluate this postulation, Onur and colleagues (2009) evaluated the activity within the amygdala and LC of non-clinical participants following exposure to a series of emotionally laden pictures of faces, previously implicated in triggering amygdala activity. Researchers pharmacologically simulated a hyperresponsiveness to fear by providing participants with a dose of reboxetine, which blocks presynaptic uptake of NE, resulting in increased levels circulating in the synapse (Onur et al., 2009). Following this increase in NE levels, the amygdala displayed hyperresponsiveness upon

exposure to fearful images, as measured by functional magnetic resonance imaging (fMRI). Also, the amygdala displayed hyporesponsiveness to neutral stimuli (Onur et al., 2009). Authors suggest these findings as a potential mechanism for the noted symptom of hyperresponsivity experienced by those diagnosed with PTSD. They propose that the BLA-LC system performs a bottom-up regulation of the stress response by creating an increased startle response to fearful stimuli in the BLA via the disinhibition of the BLA-LC system and increased levels of NE (Onur et al., 2009). This would contribute to increased responses to fearful stimuli, promoting the acquisition of conditioned fear responses, and increased memory consolidation of traumatic recollections (Onur et al., 2009).

While mixed results have been noted among studies investigating the baseline levels of NE activity within those diagnosed with PTSD, 24-hour plasma level estimations have revealed elevated NE levels within those diagnosed with PTSD compared to those diagnosed with depression and controls (as reviewed in Yehuda, 1998). In addition, abnormalities within the alpha-2 adrenergic receptors is noted in PTSD (as reviewed in Yehuda, Siever, Teicher, Levengood, &Gerber, 1998). Specifically, fewer binding sites are noted on alpha-2 adrenergic receptors in combat veterans and traumatized children with PTSD.

In response to stressors, individuals diagnosed with PTSD display increased noradrenergic reactivity (as reviewed in Friedman et al., 2010). This has been implicated in contributing to symptoms of arousal and re-experiencing. These increased levels are found only in response to stressors, as no baseline differences in NE activity are noted. When NE levels are measured via 24 hour plasma concentrations, however, increased levels of NE are found among those with PTSD.

Amygdala. As previously discussed, research indicates the presence of hyperactivation of the BLA in PTSD, which is implicated in the increased responsivity to fear stimuli frequently noted. In a meta-analysis of functional neuroimaging studies related to anxiety disorders, Etkin and Wager (2007) noted that, in addition to hyperactivity within the BLA, other areas of the structure evidenced hypoactivation. Specifically, hypoactivation of the dorsal amygdala, anterior hippocampus, rostral anterior cingulate, and vmPFC were noted (Etkin et al., 2007). Impairments in the vmPFC have been related to deficits in extinguishing fear responses (Etkin et al., 2007).

Serotonin (5-HT). As 5-HT has been shown to play a role in the regulation of stress and anxiety, its influence on the symptom presentation found within PTSD is not surprising. Upon exposure to stress, an increase in 5-HT throughout several brain regions occurs (as reviewed in Krystal & Neumeister, 2009). In addition, 5-HT has been shown to play a role in neuroplasticity, which is noted within the context of PTSD. The 5-HT1b receptor has received considerable research attention and plays an important role in theories of resilience to the development of PTSD following stress exposure (Krystal et al., 2009). Previous studies using knockout procedures of this receptor indicate an increase in anxiety (as cited in Krystal et al., 2009). Situated within the "somatodendritic or axon terminal regions" of serotonergic neurons, 5-HT1b receptors serve to inhibit the release or synthesis of 5-HT (as reviewed in Neumaier, Edwards, & Plotsky, 2002). Increased receptor expression is related to a decrease in 5-HT release within the PFC, contributing to symptoms of stress and anxiety. Evidence suggests that individuals with PTSD display reduced down regulation of 5-HT1b receptors, creating reduced levels of 5-HT within the amygdala (as reviewed in Krystal et al., 2009). Reduced levels of 5-HT within the amygdala produce a reduced firing threshold, leading to increased activation upon exposure to stressors (Morgan & Krystal, 2003). This occurs as reduced levels of 5-HT fail to inhibit the

excitation of 5-HT1a and 5-HT3 receptors, which inhibit the creation of an action potential and stimulate GABA release, respectively (Krystal et al., 2009). Also, reduced activation of 5-HT1b receptors has been shown to result in inhibition of 5-HT release within the PFC, contributing to a failed inhibition of the amygdala via top-down regulatory processes (Neumaier et al., 2002).

Ventromedial PFC (vmPFC). Numerous studies have suggested that the fear circuitry centers within the brain are altered within PTSD. Reduced volume and activity of the vmPFC has been noted within PTSD (Koenigs & Grafman, 2009). The vmPFC has been shown to be important in the regulation of fear conditioning (Koenigs, et al., 2009). Research involving fear conditioning involves pairing neutral stimuli (conditioned stimulus) with an aversive stimuli (unconditioned stimulus), resulting in physiological changes labeled the fear response (Koenigs, et al., 2009). Eventually, presentation of the conditioned stimulus (CS)elicits the fear response. Extinction occurs when the CS is presented several times without the unconditioned stimulus (US), resulting in the elimination of the fear response (Koenigs et al., 2009). Studies implicate the vmPFC in the extinction of the fear response in fear conditioning (Koenigs et al., 2009). The mechanism of the extinction process is thought to be the inhibition of the amygdala by the vmPFC (Koenigs et al., 2009). Abnormalities within the vmPFC and amygdala circuitry has been implicated within the PTSD. Research indicates, upon presentation of trauma related cues, those with PTSD experience reduced activation of the vmPFC and overactivity in the amygdala (Koenigs et al., 2009). Lesion studies investigating the role damage to this region of the brain has upon functioning suggest that damage in this region is related to several symptoms present within PTSD, including the "misinterpretation of emotionally laden cues, impulsivity, aggression, and enhanced emotional memory" (as cited in Vasterling et al., 2005). In a study of veterans with documented damage to the vmPFC, the presentation of PTSD symptoms was

found to be lower, calling into question the causal hypothesis presented to explain the role of damage to this region noted among those with PTSD (Koenigs et al., 2009). Authors suggest an alternate explanation for the role of the vmPFC in PTSD, focusing on the role of this area in cognitive functions of self-awareness and reflection (Koenigs et al., 2009). Impairment in self-awareness may serve to buffer against PTSD symptoms, as a diminished capacity for self-reference may interfere with autobiographical memory, found to be hyperactive in those with PTSD (Koenigs et al., 2009). The finding of hypoactivity in this region among those with PTSD may relate to damage to the amygdala, which has been found to impair functioning in this region (as cited in Koenigs et al., 2009).

Chronic psychosocial stress decreases the receptor density in areas important within the limbic system (Southwick, 2010). Decrease in serotonin is related to exaggerated startle response and high responsivity to novel stimuli (van der Kolk, 1996). Abnormalities within the serotonin system have been associated with impairment in the PFC, amygdala, LC, and hippocampus. Within the PFC, serotonin deficiencies negatively impact the orbitofrontal cortex, responsible for processing social and emotional information (Southwick, 2010). Performance on neuropsychological tasks tapping orbitofrontal functioning is impaired in individuals diagnosed with PTSD (as cited in Southwick, 2010). Decreased serotonin increases the firing of the amygdala, shown to be active in the regulation of fear and anxiety responses (as cited in Southwick, 2010). This effect is shown to be dependent upon the presence of corticosterone, which is increased during exposure to stressors. In addition, serotonin has an inhibitory influence on the LC, critical in NE circulation. With a decrease in serotonin levels, the LC produces higher levels of NE (Southwick, 2010). Finally, serotonin has been found to influence the hippocampus. In chronic stress, low levels of serotonin have been correlated with decreases in hippocampal cell

neurogenesis and proliferation (Southwick, 2010). Traumatized individuals and those with PTSD have been found to have decreased hippocampal volume and to display impaired performance on measure of declarative, verbal memory (Southwick, 2010).

Dopamine (DA). Research suggests abnormalities within the DA system in PTSD. In particular, elevated urinary and plasma levels are noted within the PFC and amygdala associated with exposure to acute and chronic stress (Vermetten & Bremner, 2002). When evaluating urinary and plasma cortisol levels among Vietnam veterans, those diagnosed with PTSD evidenced elevated levels of DA compared to trauma exposed controls (Yehuda, Southwick, Giller, Ma, & Mason, 1992; Hamner & Diamond, 1993). These increased levels of DA may contribute to symptoms of dissociation and depersonalization commonly associated with PTSD (Weiss, 2007).

***Working memory*.** Executive functioning, including working memory, has been shown to be defective in PTSD patients (Weber, Clark, McFarlane, Moores, & Morris, 2005). In those diagnosed with PTSD, reduced neuronal firing is evidenced within the dorsolateral and inferior parietal network (Weber at al., 2005). To measure abnormalities in working memory processing, studies commonly utilize electroencephalography (EEG), which measures the electrical activity of the brain through the use of probes on the scalp or skull. The EEG provides a measurement of event related potentials (ERPs), which indicates neuronal firing and communication among neurons within the brain. This allows for an understanding of the timing of communications within the brain during the completion of information processing tasks. Past studies utilizing this technique have discovered abnormal stimulus processing responses to both neutral and trauma-related stimuli (as reviewed in Weber et al., 2005). PTSD has been associated with delayed N2 responses, suggesting difficulty with stimulus discrimination (Veltmeyer, 2009). Also,

diminished P3 amplitudes indicate difficulty with selective attention processes, suggesting deficits in determining the significance of stimuli, distinguishing among relevant and irrelevant information, and completing the processes of memory updating (Weber et al., 2005). Researchers have suggested that these findings are suggestive of difficulty in maintaining information within working memory (Weber et al., 2005).

Stress and trauma research have noted the influence of connections between the amygdala and the medial PFC in relation of emotional processing, anxiety, and fear (Williams, Kemp, Felmingham, Barton, & Olivieri, 2006). The amygdala is responsible for many aspects of fear, including the appraisal of threatening stimuli, and the initiation and maintenance of the fear response (Williams et al., 2006). The mPFC has several subdivisions, including the anterior cingulate cortex and more ventral areas that include the infralimbic and prelimbic cortices (as reviewed in Sotres-Bayon, 2004). The anterior cingulate is further divided into dorsal and ventral regions. The ventral region has been associated with the regulation of emotions and has connections with the amygdala. Alternately, the dorsal region is associated with attention and cognition. The amygdala and ventromedial PFC are interconnected in a manner that allows the vmPFC inhibit the fear response initiated by the amygdala. Alternately, the amygdala can serve to reduce vmPFC activity (Williams et al., 2006). Functional neuroimaging studies have revealed a negative relationship between the amygdala and the ventrolateral PFC in which amygdala activity decreased as vlPFC increased (Williams et al., 2006). It has been suggested that abnormalities within this interconnections contribute to symptoms found in PTSD, particularly symptoms of re-experiencing, avoidance, and hyperarousal. Specifically, it has been suggested that a lack of inhibition from the mPFC contributes to "hyperresponsivity to fear-related stimuli" (Williams et al., 2006). The amygdala, responsible for assigning emotional meaning to stimuli

and initiating fear responses, displays heightened activity in response to trauma cues in people with PTSD (Brunello, Davidson, Deahl, Jessler, Mendlewicz, et al., 2001). Bremner, Narayan, Staib, Southwick, & McGlashan (1999) further describe the mechanism through which these deficits occur through the use of fMRI. Upon exposure to trauma related scripts, PTSD patients with histories of childhood abuse evidenced reduced blood flow to the mPFC coupled with a failure to activate the anterior cingulate. This is suggestive of a neural response following trauma related cues that fails to inhibit the amygdala, resulting in fear responding, which, in turn, interferes with working memory ability. In individuals with prior exposure to trauma, functional imaging techniques reveal abnormal temporal firings within the subregions of the amygdala in response to presentation of fearful faces (Williams et al., 2006).

***Hippocampus*.** The damaging effects of trauma to the hippocampus have been widely studied in a variety of populations, including veterans, childhood trauma victims, and in individuals with borderline personality and post-traumatic stress disorders. Results of these studies suggest a stress related reduction in volume, particularly in the CA3 region (Bremner, 2005). Damage occurs to the hippocampus through three mechanisms: elevations of the stress hormone cortisol, reductions in brain derived neurotropic factor, and the inhibition of neurogenesis (as reviewed in Bremner, 2005). In a meta-analysis, Bremner et al. (2005) discovered that, relative to controls without a history of trauma, individuals with PTSD and with exposure to trauma evidence reductions in hippocampal tissue bilaterally. Individuals with PTSD, however, evidence a more pronounced reduction in volume, particularly in the left hemisphere (Karl, 2006). This suggests that hippocampal atrophy is a result of trauma exposure itself and not necessarily dependent upon diagnosis of PTSD. The role of trauma in the creation of reduced hippocampal volume has been further called into question by the study of twins, with

one who developed PTSD and the other healthy. Preexisting decreases in hippocampal volume suggests that this may present a vulnerability factor for the development of PTSD, rather than a consequence of the disorder (Gilbertson, Shenton, Ciszewski, Kasai, & Lasko, 2002; Malberg, 2004).

Neuropsychological functioning in PTSD. While neurobiological abnormalities have been noted among those with PTSD, it is important to consider the functional deficits that may be evident as these individuals attempt to complete tasks within their everyday lives. To make this determination, neuropsychological assessment of cognitive functioning reveals performance of individuals completing specific information processing and response generation tasks.

Evaluation of executive functions reveals decreased performance among individuals with PTSD. Research regarding neuropsychological functioning among those with PTSD has often focused on the discovery of possible pre-trauma exposure variables that would suggest potential risk or reliance to the development of the disorder (Aupperle, Melrose, Stein, & Paulus, 2011). To this end, several factors have been suggested as possible risk/resiliency factors, including intelligence quotient (IQ) score, education level, verbal recall, working memory, and visuomotor speed (Aupperle et al., 2011). Specifically, an increased level of intelligence (particularly verbal intelligence), a higher level of educational attainment, and lower scores on measures of verbal memory, attention, and executive functioning are found to have an inverse relationship with PTSD diagnosis (as reviewed in Aupperle et al., 2011). Evidence regarding these findings has been mixed in the literature, with some studies reporting decreased functioning in these domains prior to the development of PTSD and others reporting evidence that deficits occur in these domains as a function of PTSD status (as reviewed in Aupperle et al., 2011).

To further investigate the role of this relationship, Gilberton, Paulus, Williston, Gurvits, Lasko, et al. (2006) completed neuropsychological testing with a group of male, monozygotic twin pairs. The pairings consisted of one twin who was a veteran of the Vietnam war and one who had no history of trauma exposure (Gilbertson et al., 2006). Among these pairings, some of the veterans also had developed symptoms consistent with a diagnosis of PTSD (Glibertson et al., 2006). The design of the study allowed for three different analyses which tested the competing theories related to lowered functioning: 1. Comparisons of neuropsychological performance among veterans with PTSD and without PTSD, 2. Comparisons of veterans with a diagnosis of PTSD and their non-trauma exposed twins, and 3. Comparisons of the non-trauma exposed twins with brothers with and without PTSD (Gilbertson et al., 2006). Results were consistent with previous studies in that veterans with PTSD evidenced lower scores on measures of intelligence, attention, and verbal declarative memory compared to trauma-exposed veterans who did not develop the disorder (Gilbertson et al., 2006). Comparisons of the non-trauma exposed co-twins revealed that co-twins of veterans who had PTSD did not differ significantly in their performance on neuropsychological measures (Gilbertson et al., 2006). In addition, their performance was lower than that of both co-twins in the pair with a non-PTSD veteran (Gilbertson et al., 2006). The findings suggest that the neurocognitive deficits reported in studies of those with PTSD are a function of pre-existing traits. The veterans utilized in this sample were diagnosed with severe cases of PTSD, suggesting that pre-existing deficits in cognitive performance may serve as a vulnerability factor within severe forms of the disorder (Gilbertson et al., 2006).

Selective attention and inhibition. Research indicates that processes related to selectively attending to information and the inhibition of automatic responses are impaired in those with

PTSD (Falconer, Bryant, Felmingham, Kemp, & Gordon, 2008; Shucard, McCabe, Szymanski, 2008, Jenkins, Langlais, Delis, and Cohen, 2000). Studies evaluating selective attention typically utilize continuous performance tests (CPTs) or go-no go tasks. In a study of inhibitory control ability among PTSD patients, Falconer and colleagues (2008) utilized fMRI to evaluate the neural activity of individuals while completing a "go-no go" task, the auditory odd ball task. As with other go-no go measures, this task requires participants to respond to select stimuli behaviorally until the response is established and automatic (as cited in Falconer et al., 2008). Once established, an alternate stimulus is presented and the participant is expected to inhibit their behavioral response (Falconer et al., 2008). Performance is measured by the number of commission errors, instances of failed inhibition to non-target stimuli (Falconer et al., 2008). The sample utilized included individuals with PTSD, a matched comparison group with no trauma exposure or PTSD, and a group of trauma exposed individuals without PTSD (Falconer et al., 2008). Unlike previous studies evaluating performance utilizing stimuli of an emotionally evocative nature, neutral stimuli were used (Falconer et al., 2008). Results indicate reduced activity within the "right-lateralized frontotemporoparietal cortical inhibitory network," which includes the right inferior cortex, dlPFC, mPFC, and cerebellum among those with PTSD compared to controls (Falconer et al., 2008). In addition, increased commission error rates among those with PTSD compared to controls were noted (Falconer et al., 2008). These factors were related to symptom severity, with increased severity associated with decreased activity within inhibitory networks and increased error rates (Falconer et al., 2008). In addition, increased activity was noted among regions associated with processing sensory information (Falconer et al., 2008). Authors suggest that increased processing within the postcentral gyrus, parrahippocampal, striatum, and visual cortical regions likely interferes with inhibition ability by

monopolizing cognitive resources and biasing the individual towards the identification of threat related stimuli (Falconer et al., 2008). While PTSD participants differed in neurological activation of regions important to the functional correlates of inhibition from both healthy and trauma-exposed controls, performance on neuropsychological assessment differed only from healthy controls (Falconer et al., 2008).

Attention and concentration deficits evident in PTSD have been proposed as a potential source of symptom development as impaired abilities in this area may influence performance in other domains, such as memory (Bressan et al., 2009). Symptoms of PTSD are proposed to result from the occurrence of increased activation of arousal mechanisms functioning within the physiological stress response functioning in conjunction with impairment among the "top-down" inhibitory system (Falconer et al.,2008). As such, PTSD is characterized by hyperreactivity of subcortical structures regulating arousal functioning with co-occurring decrease in mechanisms that serve to inhibit these regions, like the mPFC and the right anterior cingulate cortex (rACC) (as cited in Falconer et al., 2008). Symptoms of re-experiencing and hyperarousal may relate to difficulties in the inhibition of intrusive internal stimuli emerging following trauma related cues and reminders presented in the external environment (Leskin & White, 2007). Similarly, symptoms of numbing may relate to deficits in the ability to differentiate relevant from irrelevant stimuli (Leskin & White, 2007).

In a more comprehensive assessment of performance on neuropsychological measures of attention, Jenkins, Langlais, Delis, and Cohen (2000), studied selective, sustained, and focused attention abilities among victims of rape diagnosed with PTSD, rape victims without PTSD, and healthy controls. Prior studies had indicated deficits in sustained and focused attention, but intact selective attention abilities on Posner's Visual Selective Attention Test (Jenkins et al., 2000).

Women diagnosed with PTSD scored lower on sustained attention tasks presented in auditory (Paced Auditory Serial Addition Test and Digit Span) and visual (Continuous Performance Test, Digit Symbol Coding, Trail Making Test-B) formats compared with trauma exposed and healthy controls (Jenkins et al., 2000). No significant differences in performance were noted on a measure of visual selective attention, consistent with prior research (Jenkins et al., 2000). This effect remained when depression and substance abuse were controlled for in analysis (Jenkins et al., 2000).

Several studies have reported deficits in response inhibition and attention measured by the Trail Making Test, Trail B in those diagnosed with PTSD (Beckham, Crawford, & Feldman, 1998; Jenkins et al., 2000; Stein, Kennedy, & Twamley, 2002). In this task, participants are presented with a page of circles containing either a number or a letter. They are instructed to alternate between numbers and letters and, starting with numbers create a sequence in ascending order (1-A-2-B-3-C, etc.). As such, it is a measure of sustained attention, sequencing, set shifting, and inhibition (Lezak, 2004). This contrasts with Trail A, which only contains circles with numbers that the participant is instructed to connect in ascending disorder. While some studies report deficits among those with PTSD, others do not report differences in performance on executive function measures compared to trauma exposed and healthy controls (Crowell, Kieffer, Siders, & Vanderploeg, 2002; Twamley, Hami, & Stein, 2004). In a study of Vietnam veterans with PTSD and without PTSD, Crowell and colleagues (2002) administered a broad neuropsychological assessment battery and found no differences in functioning compared to controls. To evaluate the possible effect of distress level upon functioning, authors included a measure of distress and found no differences among those experiencing current distress and those without (Crowell et al., 2002). Findings may have been limited as they did not evaluate

effects of other traumas (beyond combat related stress) and did not include females within their sample. Twamley and colleagues (2004) evaluated neuropsychological performance on Trails B among a sample of college students and uncovered no differences in performance relative to non-traumatized controls. Few studies have evaluated functioning among non-clinical populations, which introduces the possibility that confounds may account for differences reported among studies, including distress levels, coping abilities, and social support (Crowell et al., 2002). Leskin and White (2007) evaluated neuropsychological performance among college students meeting criteria for PTSD, as well as trauma-exposed and healthy controls. No differences in performance emerged on traditional trail making tests, however, performance on the executive control task of the ANT was lower among those with PTSD (Leskin & White, 2007). Authors suggest that sensitivity in the utilized measures ability to assess response inhibition may contribute to discrepancies noted within previous research. Also, as no measure of intellectual functioning or distress levels were utilized, it is suggested that future studies include these factors (Leskin & White, 2007).

Response inhibition has also been measured utilizing the Stroop test paradigm. The task requires the participant to read words of colors, printed in various colored inks, or to say the color of ink in which the word is printed. This requires inhibition as the participant must complete a task that is counter to their automatic response. In a sample of individuals presenting in emergency rooms following trauma exposure, deficits in performance on the Stroop test indicate deficits in inhibition among those with PTSD compared to trauma exposed individuals not meeting diagnostic criteria (LaGarde, Doyon, & Brunet, 2010). Results support the suggestion that PTSD, not trauma exposure alone, is related to impaired attentional ability (LaGarde et al., 2010).

***Cognitive flexibility and planning*.** In addition to inhibition, the TMT also measures cognitive flexibility as the participant is required to switch between sets of instructions. As such, studies previously reviewed related to attention and inhibition are applicable to the functional implications of cognitive flexibility as well. The Wisconsin Card Sort Test (WCST) is another measure of flexibility and provides a more comprehensive assessment of the level of flexibility in switching among different categories rather than flexibility in switching attention (Aupperle et al., 2011). Research utilizing this measure indicates increased response times among those diagnosed with PTSD during the initial trial compared to controls, but no differences on future trials (Kanagaratnam & Asbjornsen, 2007; Twamley, 2009). In a study evaluating executive functioning among women experiencing intimate partner violence (IPV), Twamley, Allard, Thorp, Norman, and Cissell (2009) found that participants diagnosed with PTSD did not differ from controls in their performance on the WCST. Participants did display lower scores on measures of processing speed, indicating the presence of cognitive slowing during problem solving (Twamley et al., 2009).

Major Depressive Disorder

Major Depressive Disorder (MDD) is considered the most common psychiatric disorder, with a lifetime prevalence of 16.5% and a yearly prevalence of 6.7% of the population in the United States (as reviewed in National Institute of Mental Health, 2011). Research suggests a relationship between stress and depression as 80% of individuals diagnosed with a major depressive episode indicate experiencing a major life event or stressor prior to onset of symptoms (as reviewed in Robbins, 2009). In addition, the type of stressor experienced relates to increased likelihood of symptom development, with internal and uncontrollable stressors being more likely to result in depression.

Diagnosis. Diagnosis of Major Depressive Disorder (MDD) involves the presence of a single Major Depressive Episode that is not attributed to Schizoaffective Disorder, Schizophrenia, Delusional Disorder, or Psychotic Disorder, Not otherwise specified (American Psychological Association, 2000). In addition, no history of a manic episode occurrence can be reported (APA, 2000). MDD can be considered either as a single episode or recurrent. To be considered recurrent, the presence of two or more Major Depressive Episodes must be reported with lapses in symptoms of no less than 2 consecutive months occurring between episodes (APA, 2000).

Major Depressive Episode. Diagnosis of a Major Depressive Episode involves the presence of five or more symptoms within the diagnostic criteria (APA, 2000). At least one of these symptoms must include either depressed mood or loss of interest or pleasure in previously enjoyed activities (APA, 2000). In addition, it is necessary for these symptoms to produce significant levels of impairment or distress in social, occupational, or other areas of functioning, like personal relationships (APA, 2000). The symptoms also cannot be a result of the physiological effects of a substance (APA, 2000). Also, other diagnoses must be ruled out before the diagnosis of depression can be made, including Mood Disorder due to a general medical condition, Substance-induced Mood Disorder, Dysthymic Disorder, Schizoaffective Disorder, Schizophrenia, and Psychotic Disorder (APA, 2000).

Biology of Depression. Major depression is a complex disorder that appears to affect several regions within the brain. As such, several theories related to the biological underpinnings of the disorder have been proposed. Current models of depression emphasize the role of dysregulation within the CNS following responses to stress that are longer enduring or that are more intense (Thase, Jindal, & Howland, 2002). Research reveals deficits in several key

chemicals within the brain among individuals diagnosed with depression, including DA (Korf & van Proag, 1971), NE (Ressler & Nemeroff, 1999), and 5-HT (Maes & Meltzer, 1995). A current hypothesis regarding the etiology and treatment of depression, the neurogenic theory of depression, integrates findings related to the high co-occurrence of stress with depression, abnormalities within the 5-HT system, and abnormalities within the hippocampus in those with depression.

Research indicates impaired functioning of the 5-HT system among those with depression. Reduced plasma levels of 5-hydroxyindole acetic acid (5-HIAA), a 5-HT metabolite, has been found among individuals with depression (as reviewed in Vinkers, Olivier, Bouwknecht, Groenink, & Olivier, 2010). In addition, low levels of tryptophan, a precursor to 5-HT, is reported among those with depression. Studies evaluating these effects generally utilize dietary tryptophan depletion paradigms in which participants are measured both on a depleted diet and under normal dietary conditions (Toker et al.2010). Gender related differences have been noted within the effects that depletion procedures have on serotonin synthesis among men and women (Toker et al., 2010). Specifically, women display greater reductions in serotonin synthesis following tryptophan depletion (Toker et al., 2010).

Research suggests the process by which this deficiency occurs and how it relates to subsequent depressive symptoms. In this theory, emphasis is placed on inflammation following stress exposure in the development of depression (Maes, Leonard, Myint, Kubera, & Verkerk, 2011; Scheipers, Wichers, & Maes, 2005). Depressive symptoms are thought to be produced by the initiation of immune system activation, mediated by the release of cytokines (as reviewed in Scheipers et al., 2005). The mechanism behind the reduction of tryptophan levels is described as increased levels of interleukin-1B (IL-1) B, IL-6, tumor necrosis factor-a (TNF a), and interferon

(INF)-y, which result in indolamine2,3-dioxygenase (IDO) to be released in the blood and brain (as reviewed in Maes et al., 2011). This results in a reduction of tryptophan, which relates to a subsequent reduction in 5-HT levels. Increased levels of cytokines in the circulation also have implications for HPA activity, which is shown to be hyperactive among those with depression. Cytokines produce increased activity of the HPA axis.

Research has focused on the role of systems regulating the stress response in the occurrence of depression, namely the HPA axis. Elevations in circulating cortisol levels, the end product of the HPA axis, have been noted among those diagnosed with depression, an occurrence labeled "hypercortisolism" (Holsboer, 2000; Thase, Jindal, & Howland, 2002). Hypercortisolism occurs in conjunction with dysfunction among NE and 5-HT systems (Holsboer, 2000; Maes & Meltzer, 1995; Schatzberg & Schildkraut, 1995). Evidence suggests that individuals with depression may display a heightened responsivity to stress, rather than baseline elevations in cortisol levels (as reviewed in Barden et al., 2004). In addition, individuals with depression evidence increased numbers of CRH cells within the PVN of the hypothalamus (as reviewed in Barden, 2004).

In major depression, elevated cortisol concentrations have been shown to produce damage within the hippocampus (Sheline, 2000). Specifically, a 15% reduction in left hippocampal volume and 12% reduction in right hippocampal volume was measured in individuals with depression using an mRI (as reviewed in Sheline, 2000). Findings related to the role of the dentate gyrus, located within the hippocampus, in making new neurons throughout the life span suggested that depression may result from dysregulation within the process of neurogenesis (Jacobs, van Praag, & Gage, 2000). Upon exposure to chronic stress, increased levels of cortisol, along with co-occurring reductions in 5-HT levels, inhibits neurogenesis within

the DG (Jacobs et al., 2000). This reduces the ability to form new memories and impairs cognition (Jacobs et al., 2000). Damage to this area also impairs its regulation of the HPA axis, resulting in increased activation and release of cortisol, which exacerbates the negative effects occurring in the DG (as reviewed in Jacobs et al., 2000).

Interest in the role of neurogenesis in the development of depression was enhanced upon the discovery that treatment with selective serotonin reuptake inhibitors (SSRIs) resulted in an increase in of neurogenesis within the hippocampus (Malberg, Eisch, Nestler, & Duman, 2000). Furthermore, the effectiveness of SSRIs, evidenced in the behavioral reduction of depressive symptoms in rodents, were shown to be dependent upon the occurrence of neurogenesis (Santarelli et al., 2003). Research further suggests the role of 5-HT in the process of neurogenesis as the activation of the 5-HT1a receptor is shown to coincide with increased cell proliferation within the DG (Jacobs et al., 2000). In addition, 5-HT depletion is associated with decreases in neurogenesis (Brezun & Daszuta, 1999).

Additional Findings. Studies of the effects of antidepressant medications have contributed to research in the domain of intracellular mechanisms, such as second messengers and gene transcription in depression. Initial findings related to the effectiveness of antidepressants, which focused on tricyclic (TCAs) and monoamine oxidase inhibitors (MAOIs) medications, revealed that levels of NE and 5-HT were elevated within the synapse shortly following drug administration, however, reductions in depression symptoms took several weeks to occur (Duman, Heninger, & Nestler, 1997; Shelton, 2000). Gene activity has since been shown to underlie this delay as the increase in monoamines in the synapse facilitates a sequence of intracellular mechanisms (Duman et al., 1997; Shelton, 2000).

Additional neurotransmitter disturbances have been noted in depression, including abnormalities in acetylcholine (ACH), gamma-aminobutyric acid (GABA), and glutamate (as reviewed in Thase et al., 2002). In chronic stress, GABA levels have been shown to be reduced (Weiss & Kilts, 1998). Reduced levels of GABA have been found within the plasma and CSF of depressed individuals (Petty, 1995). Glutamate, a widely distributed neurotransmitter binding to the N-methyl-D-aspartate (NMDA) receptor, may be important in depression (Mathew et al., 2001).

Neuropsychological findings in depression. Research focusing on neuropsychological functioning among individuals diagnosed with depression has uncovered deficits in functioning that occur predominately within the acute phase of the disorder (as reviewed in Hammer & Ardal, 2009). Research indicates two main findings related to cognitive functioning among those with depression, cognitive biases and cognitive deficits (Murrough, Iacoviello, Neumeister, Charney, & Iosifescu, 2011). Cognitive biases involve the tendency for those with depression to utilize schemas that channel cognition to negative beliefs about "themselves, the world, and the future" (Murrough et al., 2011, p.2). Cognitive deficits describe dysfunction within executive functioning, attention, and memory among those diagnosed with depression (Murrough et al., 2011).

Hammer and Ardal (2009) summarize neuropsychological findings in depression according to three separate theoretical interpretations of findings related to acute depression, stating that cognitive impairment in depression is 1) globally reduced across a broad range of functions, 2) associated with specific deficits in executive functioning and memory, and 3) related to deficits in functions that require sustained effort rather than automatic responses (as cited in Hammer & Ardal, 2009). In contrast, long term cognitive functioning among those with

depression can be separated into two distinct hypotheses, 1) impairment continues even following remission of depression symptoms and 2) the relapses produce further cognitive impairment (as cited in Hammer & Ardal, 2009).

Executive functioning. Research indicates deficits in executive functioning ability among individuals within the acute phase of major depression (as reviewed in Hammer & Ardal, 2009). Specifically, deficits in set shifting, working memory, response inhibition, and mental flexibility have been reported (as reviewed in Hammer & Ardal, 2009).

Set shifting-Cognitive flexibility. Research indicates that findings related to dysfunction within the domain of cognitive flexibility are the most prominent among those with depression (Austin, Mitchell, & Goodwin, 2001). As mental flexibility aids individuals in selecting coping responses, the rigidity characteristic of those with depression limits potential problem solving solutions, increasing vulnerability to stress (Marazziti, Consoli, Picchetti, Carlini, & Faravelli, 2010). Studies investigating set shifting abilities among those with depression have generally relied on the Wisconsin Card Sorting Test (WCST) or Trail Making Test (TMT),Trails B (as reviewed in Austin et al., 2001). In a population based study of executive functioning in Sweden, those with depression took increased periods of time to complete Trails B than did healthy controls (Airaksinen, Larsson, Lundberg, & Forsell, 2004). In follow-up analyses, it was revealed that these effects were only found within a subtype of depressed individuals, those with dysthymia (Airaksinen et al., 2004). Depressed individuals performed similar to controls on Trails A, indicating intact psychomotor speed abilities (Airaksinen et al., 2004).

Past research indicates deficits in performance on the WCST among those with major depression relative to healthy controls (Merriam et al., 1999). Purcell, Maruff, Kyrios, and

Pantelis (1997) examined the role of depression in neuropsychological performance among outpatients utilizing the intradimensional/extradimensional (ID/ED) task, a measure similar to the WCST. Results indicated that depressed individuals performed more poorly on the task, indicated by an increase in the number of trials necessary to learn the correct strategy (Purcell et al., 1997). Additional analyses revealed that only a percentage of those with depression displayed these deficits, those with a history of inpatient psychiatric placement (Purcelli et al., 1997).

Attention. Evidence suggests that difficulties in concentration and attention are common among those with major depression (Lyche, Jonassen, Stiles, Ulleberg, & Landro, 2010). Research related to attention ability, as measured via neuropsychological assessment, has produced variable results (Lyche et al., 2010). Several studies indicate a tendency to focus on emotional aspects of stimuli and struggle to disengage from this focus (as reviewed in Lyche et al., 2010). Hammer and Ardal (2009) reviewed research related to attentional ability among those with depression and concluded that individuals display abnormal functioning on tasks requiring effortful processing, but adequate performance on those that rely on automatic behavior. Authors suggest that findings related to attention in depression are inconsistent, in part, due to difficulty in teasing apart effects related to processing speed and those related to impaired ability to maintain attention in the selection of relevant information for processing (Hammer & Ardal, 2009).

Research relating to the effects of depression on attentional ability often utilizes the Stroop interference test as a measure of attention (as reviewed in Lyche et al., 2010). Poor performance among those diagnosed with depression is reported among individuals diagnosed with major depression (as reviewed in Lyche et al., 2010). Difficulty in identifying consistent findings related to effects on attention may relate to the complexity of the concept, in which

three aspects of functioning have been identified, alerting, orienting, and executive control (as reviewed in Lyche et al., 2010). In addition, conflicting results may be attributed to the presence of co-morbid disorders, like anxiety. Lyche and colleagues (2010) measured attention utilizing a Stroop test measure in participants meeting criteria for major depression and for co-morbid depression and anxiety. Results indicate that performance on the attention measure was impaired, as indicated by slower reaction times, in individuals with a dual diagnosis of attention and anxiety relative to healthy controls (Lyche, 2010). This suggests a possible explanation for conflicting findings within previous research as individuals in samples may have varied according to their level of co-occurring anxiety.

Krompinger and Simons (2010) measured Stroop test performance in undergraduate college students while also measuring their event related potentials (ERPs). Performance on the measure did not differ among college students with depression and healthy controls (Krompinger & Simons, 2010). Differences emerged, however, in ERPs (Krompinger & Simons, 2010). Those with depression evidenced increased amplitude on an ERP measure, suggesting the possibility of the use of compensatory measures and the increased activity of areas of the brain responsible for conflict detection (Krompinger & Simons, 2010). These results indicate that, in college students, deficits in functioning may not be present despite alterations in cognitive processing styles. Differences in functioning may be too subtle to be revealed on provided paper and pencil tests.

Inhibition. Individuals with depression display an impaired ability to inhibit distracting information while engaging in simultaneous processing of other information (as reviewed in Hammar & Ardal, 2009). Hugdahl et al. (2009) utilized a dichotic listening paradigm to evaluate the inhibition ability of individuals diagnosed with depression. In this paradigm, semantically meaningless syllables are presented in both right and left ears. Participants are instructed to

attend to different syllables when presented. As this occurs, variations in the salience of the presentation can occur, resulting in cognitive conflict in which the individual must inhibit a natural response to achieve a task relevant response (Hugdahl et al., 2009). Individuals with depression evidenced impaired ability in inhibition, suggesting increased conflict in top-down and bottom up conflict regarding the regulation of behavior (Hugdahl et al., 2009). Other studies reveal impaired ability in the inhibition of neutral information and suppression of processing task irrelevant stimuli while completing working memory (WM) tasks (Gohier, Ferracci, Surguladze, Lawrence, Hage et al., 2009).

Working Memory (WM). Findings related to functioning of WM within individuals with depression have been mixed with some studies reporting deficits (Beats, 1996; Elliot, 1996; Landro, 2001; Nebes, 2000; Porter, 2003) and others finding no differences in levels of functioning (Barch, 2003; MacQueen, 2000). Deficits in functioning related to WM ability in depressed individuals is suggested to occur within the process of updating contents as new stimuli are presented and disregarding irrelevant information (Harvey et al., 2004). In addition, difficulties in filtering irrelevant, emotionally charged information from entering into WM have been reported (as cited in Joorman, Levens, & Gotlib, 2011). The role of cognitive inflexibility and WM abilities in depressed individuals has recently been related to symptoms of rumination characteristic of the disorder. Mechanisms behind rumination may be contributed to by difficulties in removing negative emotional information from WM processing (as cited in Joorman et al., 2011). Difficulties in manipulating information in WM, especially negatively charged emotional content, is likely to contribute to cognitive symptoms of depression, like rumination (Joorman et al., 2011).

Research Rationale and Hypotheses

The impact of traumatic stress is complex and is associated with several potential negative consequences, including the development of posttraumatic stress disorder (PTSD), depression or anxiety, abnormal stress reactions within the HPA axis. In addition, research has focused on neuropsychological deficits associated with anomalies in brain structure and hormone distribution. Specifically, abnormalities within HPA Axis functioning (hyper or hypo activity), working memory, and explicit memory have been noted. Few studies have evaluated these effects on non-clinical populations and an even smaller number have examined the impact of trauma upon college students specifically. College students have been shown to have high incidence of trauma exposure, with 67- 84% being exposed to at least one significant event and over one third being exposed to four or more events (Bernat, Ronfeldt, Calhoun, & Arias, 1998; Vrana & Lauterbach,1994). Only a small percentage of students exposed to trauma develop PTSD symptomology and evidence neuropsychological deficits, suggesting that they possess factors that make them resilient to the effects of trauma exposure (Bernat et al., 1998; Twamley, Hami, & Stein, 2004). Despite this suggestion, there is little understanding of the specific factors that would potentially contribute to their resilience.

The purpose of the current study was to extend previous research related to the relationship between chronic stress and trauma and the development of stress related disorders, including depression, anxiety, and PTSD. In addition, this study aimed to clarify the relationship stressful and/or traumatic experience has upon functional abilities of attention, working memory, explicit memory, response inhibition, and cognitive flexibility. Previous studies involving these relationships have traditionally focused on clinical populations. This study aimed to determine if these effects were shown among college students. While previous research suggests few

neuropsychological deficits occurring within this population, little emphasis was placed on duration, intensity, or frequency of stressors. The current study examined the role of current perceived stress, historical report of stressful events, and a measure of traumatic experiences. In addition, factors previously shown to influence the effects of stress and trauma were included to further clarify factors that may mediate or moderate the relationship between stress, trauma, and measured levels of functioning.

Primary research hypotheses

Hypothesis 1: Increased number of experienced traumatic events (measured on the Life Experiences Scale and Stressful Life Event Screening Questionnaire) will be predictive of variability of performance on neuropsychological measures of executive functioning (as measured by the Trail Making Test, N-back test, and Wisconsin Card Sort Test), explicit memory (Rey Auditory Verbal Learning Test), and psychopathology, including PTSD (as measured by the Impact of Event Scale), depression (as measured by the Beck Depression Inventory, Second Edition), and anxiety (as measured by the State Trait Anxiety Inventory).

Hypothesis 2: Individuals experiencing interpersonal trauma will display increased psychopathology (as measured by scores on BDI-II, STAI, and IES) and increased deficits on executive functioning (as measured by the Trail Making Test, N-back test, and Wisconsin Card Sort Test) and explicit memory (Rey Auditory Verbal Learning Test) than will individuals experiencing other types of trauma or no trauma.

Hypothesis 3: A continuum of scores will be displayed, such that individuals with PTSD will score lower on measures of executive functioning (as measured by the Trail Making Test, N-back test, and Wisconsin Card Sort Test) and explicit memory (Rey Auditory Verbal Learning

Test), and higher on measures of psychopathology (as measured by scores on BDI-II, STAI) than will individuals with trauma exposure and no PTSD, but those with no trauma exposure will display higher scores on measures of executive functioning (as measured by the Trail Making Test, N-back test, and Wisconsin Card Sort Test) and explicit memory (Rey Auditory Verbal Learning Test) and lower on measures of psychopathology (as measured by scores on BDI-II, STAI) than both those with PTSD and those with trauma exposure, without PTSD.

Hypothesis 4: Earlier age at time of trauma or stress will be associated with lower scores on measures of executive functioning (as measured by the Trail Making Test, N-back test, and Wisconsin Card Sort Test) and explicit memory (Rey Auditory Verbal Learning Test), and higher scores on measures of psychopathology (as measured by scores on BDI-II, STAI) than will those experiencing trauma at later ages.

CHAPTER II

METHODS

Participants

The current results are based upon a final sample of 129 college students enrolled in sections of the Introductory Psychology course offered at Indiana University of Pennsylvania. The sample consisted of 76 females (58.9%) and 53 males (41.1%). Demographic information for participants is listed in Table 1. A majority of participants, 86.5%, were within the age range of 18-20 years old, with the range being 18-32 years of age. Eighty eight percent of participants (n = 114) indicated that they were currently Single, 2% (n = 3) report being married, and 8% (n = 11) report being a member of an unmarried couple. The largest religious affiliation reported by participants was Catholic (n=48; 37.2%), followed by Protestant (n = 21; 16.3%). Twenty three percent of participants (n = 30) indicated that they had no preference/no religious affiliation and 17.8% (n = 23) indicated that they have an other religious preference. As previous studies have indicated substance use is a potential moderating factor in terms of executive functioning deficits, participants were asked questions regarding their substance use behavior. Most participants indicated they used alcohol (n =93; 72.1%), 31% (n = 41) reported using alcohol weekly, 18% (n = 24) reported using monthly, 19% (n = 25), and 4.7% (n = 6) reported using three or more times per week. Ten percent of participants (n = 14) indicated that they use street drugs or misuse prescription medications. As previous research has suggested that social support serves as a mediating factor in the effects of stress on functioning, participants were asked how many community organizations in which they were involved. Thirty four percent of participants indicate that they are involved in no community organizations (n = 44), 10% indicated being

involved in 1-2 organizations (n = 14), 3% indicated being involved in 3-4 organizations, and 3% indicated being involved in 5 or more organizations.

Table 1

Demographic Information of Participants

Variable	Number	Percentage
Ethnicity		
Caucasian	97	75.2%
African American	18	14%
Hispanic/Latino	3	14%
South American	1	0.8%
Asian/Pacific	3	3.2%
Other	3	2.3%
Class		
Freshman	84	65.1%
Sophomores	30	23.3%
Juniors	9	7%
Seniors	6	4.7%

Measures

Working Memory: N-back test

Previous studies have utilized n-back tests to evaluate working memory. During the test, a series of stimuli are presented sequentially. The participant is instructed to monitor the stimuli according to a given dimension, like content, color, identification, or position and indicate when the given stimuli matches one presented two numbers before the currently presented number. The span can be altered, which makes the task more difficult. The task requires a complex array of functions, including abilities to encode information from the environment into working memory and maintain for use in problem solving. The total number of correct responses is the dependent variable used within analyses. For this study, "N-backer" software was utilized (Monk, Jackson, Nielson, Jefferies, & Olivier, 2011).

Explicit Memory: Rey Auditory Verbal Learning Test (RAVLT)

Research relating to deficits in hippocampal functioning have utilized measures that assess learning through recall and recognition procedures. The RAVLT measures immediate memory span for verbal information, short-term and long-term storage capacity, and learning strategies (Lezak, 1995). It allows for both recall and recognition performance to be measured (Lezak, 1995). The measure includes five presentations of a 15-word list. The recall score is determined by the number of words correctly remembered. In addition, an alternate 15-word list is presented as a distractor. Another recall trial is administered following the presentation of this distractor list. Following a 30 minute delay, a final recall trial is administered. A recognition trial may also be administered, if the score of the final delayed recall trial is less than 13. A copy of this measure is included in Appendix A.

Multiple scores are calculated according to responses provided on the RAVLT, including the recall score for each trial, a total recall score, repetition scores, and error scores. In addition to the recall score calculated for each trial, a total recall score is calculated by summing the scores on trials I thru IV.

The RAVLT is reported to be sensitive to neuropsychological impairment and correlates with other measures of memory and learning (Lezak, 1996). Test-retest reliability, concurrent validity, and criterion-related validity are reported to be adequate (Schmidt, 1996). Performance correlates with gender, with women obtaining higher scores than men. Scores also correlate with education, as higher education levels have been associated with improved performance.

Executive Functioning: Trail Making Test (TMT), Trails A and B; Wisconsin Card Sorting Test (WCST)

As measures of executive functioning, the TMT and the WCST assess skills related to functioning within the prefrontal cortex. The TMT primarily measures visual conceptual and visuomotor tracking, but also measures abilities related to responding to complex visual information, sequencing, processing multiple stimuli or cognitive processes, and cognitive flexibility (Lezak, 1996). The test includes two trails, A and B. In Trail A, numbers are printed within circles that are randomly scattered on the page. The participant is instructed to draw a line connecting the circles of numbers in a sequential fashion. In Trail B, circles with numbers are provided along with circles with letters written in them. The participant is instructed to draw a line connecting the numbers and letters alternately and sequentially. For instance, a line would be drawn from 1 to A, A to 2, 2 to B, and so on until the letter H is reached. Scores on this measure include time of completion of the task. In addition, a difference score is calculated to determine

the difference in speed of performance following the addition of a more complicated and demanding task (Trail B-A).

The TMT is sensitive to brain damage and dysfunction, particularly difficulties with frontal lobe functioning (Lezak, 1996). Performance decreases with age, with completion times increasing incrementally with age (Lezak, 1996). Education also influences scores, with improved performance being correlated with increased level of education (Lezak, 1996). Reliability estimates indicate that the measure is stable (r = 0.6-0.9). Reliability for Trail B, and for the difference score of Trail B-A, appears to be less stable than Trail A (Lezak, 1996). A copy of this measure is provided in Appendix B.

The WCST is also noted to be sensitive to the measure of frontal lobe dysfunction and assesses cognitive flexibility, set shifting, and problem solving (Lezak, 1996). Reliability estimates indicate that the WCST is stable upon retesting (r =.05) and has concurrent validity with Trails B (r = -0.52).

PTSD Status: Impact of Event Scale-Revised (IES-R)

The IES is a 15-item self-report measure created to measure psychological responses to stressful situations, including responses related to intrusion or avoidance (Horowitz, Wilner, & Alvarez, 1979). The IES is made up of three scales, intrusion, avoidance, and hypervigilance, as well as a total score. Respondents are instructed to list responses over the course of the past 7 days on a 4 point scale (0 = not at all to 5 = often). Authors suggest that, when using the total score as a measure of likelihood of PTSD diagnosis, the following categories should be used, low < or = 8.5, medium = 8.6-19, and high > or = 19. In addition, a revised version that includes hyperarousal symptoms was used in the current study (IES-R, Weiss and Marmar, 1996).

Internal consistency scores reveal good to high levels of reliability (0.79 to 0.92). Discriminate validity related to diagnosis of PTSD has been established by comparisons with the Structured Clinician-administered Interview Schedule (SCID), PTSD section (r= 0.48) and the PTSD scale of the MMPI(r = 0.33) (Rush et al., 2008). A chronbach's alpha was calculated and indicated high internal consistency of this measure with this sample ($\alpha = 0.95$). The IES-R takes approximately 5-10 minutes to administer. A copy of this measure is provided in Appendix C.

Stressful Life Experiences: Life Experiences Survey (LES) and Stressful Life Events Screening Questionnaire- Revised (SLESQ-R)

The LES measures the occurrence and desirability or undesirability of life events (Sarason, Johnson, & Siegel, 1978). It includes 47 items in which respondents describe their ratings to a variety of events on a 7-point Likert scale (-3 = extremely negative; 0 = no impact; +3 = extremely positive). Three scores are derived from the LES, including positive change, negative change, and total change. Positive change is calculated by summing items rated as slightly (+1), moderately (+2), or extremely (+3) positive, with possible scores ranging from 1-150. Negative change is calculated by summing items rated as slightly (-1), moderately (-2), or extremely (-3) negative, with possible scores ranging from -1 to -150. Test-retest reliability among college students has revealed low to moderate indices of change scores (r = 0.19-0.88). Authors suggest these lower scores are a function of the dynamic nature of stress experiences, as respondents may experience stressors between testing. Negative change scores display a strong correlation with established measures of anxiety, like the State-Trait Anxiety Inventory (STAI; r = 0.29-0.46; Speilberg et al., 1970). In addition, it is correlated with grade point average (GPA) among college students (r = -0.38). The LES takes approximately 10 minutes to administer. A copy of this measure is included in Appendix D.

The SLESQ-R is a 13-item self-report measure of trauma exposure history. Each item include follow-up questions related to when the event occurred, how long it occurred, and questions aimed to determine the severity of the event. The measure was normed with a college population, making it appropriate for use with non-clinical populations (Goodman, Corcoran, Turner, Yuan, & Green, 1998). Test-retest reliability among college students is reported to be good, 0.89 (Goodman et al., 1998). Convergent validity with clinical interviews relating to the number of traumatic events reported was adequate, 0.77. A copy of this measure is provided in Appendix E.

Depression: Beck Depression Inventory, Second Edition (BDI-II)

The BDI-II is a 21-item self-report measure evaluating symptoms of Major Depressive Disorder. Each item contains four possible responses, including "not present" (score = 0) to "severe" (score = 3). Estimates of reliability indicate that the BDI-II holds together as a unitary measure (r = 0.92). Test-retest reliability estimates indicate that performance on the measure is stable across time (r =0.93). Convergent validity estimates suggest the BDI-II correlates well with other measures of depressive symptoms, including the Revised Hamilton Psychiatric Rating Scale (r = 0.71). The scores are cutoff into four levels of severity: 0-13 (minimal depression), 14-19 (mild depression), 20-28 (moderate depression), and 29-63 (severe depression). In addition to the total score, responses are further divided into two main categories, reflective of different components of Major Depressive Disorder, affective and vegetative (physical) symptoms. This factor structure is found to be stable among different samples (Beck, 1996). A chronbach's alpha was calculated and indicated good internal consistency of this measure with this sample (α = 0.81). A copy of this measure is provided in Appendix G.

Anxiety: State-Trait Anxiety Inventory (STAI)

The STAI is a 40-item measure of two subtypes of anxiety, state and trait. State anxiety represents a "transitory state of arousal" in response to perceived dangerous or stressful stimuli (Hedberg, 1972). On this measure, 20 items measure this construct by assessing symptoms the participant is currently experiencing. Trait anxiety refers to a more stable and long lasting behavioral response style to ongoing stress (Hedberg, 1972). The 20 items making up the trait anxiety subscale measure how the participant "generally feels." (Hedberg, 1972). The measure has good internal consistency (r = 0.83-0.96) and test-retest reliability (0.73-0.86). Scores correlate with other measures of anxiety, suggesting adequate concurrent validity (r = 0.75-0.85). A chronbach's alpha was calculated and indicated high internal consistency of this measure with this sample ($\alpha = 0.94$). A copy of this measure is provided in Appendix H.

Perceived Stress: Perceived Stress Scale (PSS)

The perceived stress scale measures degree to which life events are considered unpredictable, uncontrollable, or overwhelming (Cohen, Kamarck, & Mermelstein, 1983). As a result, an increased understanding of stress is obtained beyond traditionally used stressful events checklists. The PSS is a 14-item measure that includes four distinct domains: unpredictability, lack of control, burden overload, and stressful life circumstances. Responses to items rate the frequency of provided feelings, thoughts, or circumstances on a 5-point Likert scale (0 = never; 4 = very often). Respondents were asked to provide answers based on experiences within the past month. A single score may be obtained on the measure, with a range from 0-56 and higher scores indicating greater levels of perceived stress. Average scores for college students range from 23.2 (SD = 7.3) to 23.7 (SD = 7.8). Internal consistency among two college student samples (N = 332;

N = 114) reveal Cronbach alpha values of 0.84 and 0.85, respectively (Rush, First, & Blacker, 2008). As evidence of the distinct subjective and experiential aspects of experiences measured by the PSS, low correlations among scores on the PSS and life events questionnaires have been reported (r = 0.25-0.35; Rush et al., 2008). Scores on the PSS scale have also been linked to salivary cortisol levels (Rush et al., 2008). Administration takes approximately 3 minutes to complete. A copy of the measure is provided in Appendix I.

Demographic Questionnaire

A survey created by the researcher asked questions regarding the individuals' gender, age, and the number of clubs and organizations in which they were involved. In addition, questions inquired about their alcohol and substance use. Also, a question allowed for the report of any psychological diagnosis. A copy of this measure is provided in Appendix F. As previous research has identified that the level of the stress response is mediated by social support and substance use, the questionnaire asked information about these factors.

Procedure

Participants were selected from a pool of students enrolled in introductory psychology courses at Indiana University of Pennsylvania (IUP). Prior to selection from the subject pool, all potential participants completed a brief pretest that inquired how many traumatic and/or stressful events they had experienced. This was designed to increase the possibility of inclusion within the sample those students reporting experiencing high levels of trauma and chronic stress. A total of 741 potential participants completed the prescreening measure. All students indicating that they had experienced more than 5 events were recruited for participation. A random sample was pulled from those indicating experiencing the following numbers of events, 3-4 events (80

participants), and 1-2 events (50 participants). A random sample of 55 participants was also recruited from participants indicating that they have experienced no traumatic events (55). Table 2 provides demographic information relating to participants responding to the prescreening measure (see Appendix J).

Table 2

Participant Reported Number of Traumas Experienced on the Pre-screening Measure

Number of Traumas Experienced	Frequency	Percentage
0	305	41.2%
1	144	19.4%
2	123	16.6%%
3	72	9.7%
4	46	6.2%
5	22	3.0%
6	6	0.8%
7	7	0.9%
8	4	0.5%
9	5	0.7%
10	3	0.4%
11	2	0.3%
23	1	0.1%

A total of 235 participants were initially selected from the subject pool. All were contacted for participation in the study. Due to a low rate of students agreeing to volunteer, an additional 50 participants were chosen from the subject pool to be recruited for participation. Volunteering to participate in a research project fulfilled a course requirement. Alternately, students were given the opportunity to complete a read and review assignment. Recruitment and assignment of participants were coordinated by the IUP subject pool. Upon assignment of participants to the study, the researcher contacted participants via e-mail or telephone to schedule participation times. Participants completed the study in the psychology research room, located in the basement of Uhler Hall on the IUP campus.

Upon arrival, participants were given an informed consent form, which detailed the nature of the study (see Appendix K). In addition, this form provided information regarding participant rights, including the ability to discontinue participation at any point without penalty. No deception was utilized in the completion of this study. Information regarding the specific nature of the research was withheld to ensure responses on measures and performance on provided assessments were un- biased. Specifically, it was not discussed that the focus was on the neuropsychological effects of chronic or traumatic stress exposure. Participants were informed that all information obtained would be free from identifiers and would be stored in a locked office on campus. The only information associated with their identity was the results of the prescreening measure. Following participation, their identity would no longer be associated with their responses.

Following assent, participants proceeded with the study by completing counterbalanced neuropsychological tests and self-report questionnaires. Surveys inquiring about trauma were administered after neuropsychological measures, and were also counterbalanced in their

presentation. Administration was completed by doctoral Clinical Psychology and undergraduate Psychology students, who completed training in the administration and scoring of the neuropsychological instruments.

Following completion of all aspects of the study, participants were provided with a debriefing form (see Appendix L) that briefly described the research study. Given the sensitive nature of some of the information asked within the self-report measures, participants were provided with the contact information for a trained counselor to discuss any concerns or distress that may have arisen. In addition, contact information was provided should the participant have further questions or concerns related to the research study.

CHAPTER III

RESULTS

Descriptive Analyses

Trauma Events

The range of total overall traumatic and stressful life events was 0-32 events, with 19% (n = 25) of participants indicating that they experienced 9 or 10 stressful events ($\overline{X}$ = 11.68; sd = 5.69). Tables 3 and 4 provide the frequency and percentage of participants experiencing each traumatic event inquired about on the SLESQ and LES, respectively.

Psychopathology

Participants reported current levels of perceived stress on the PSS ranging from 12-38 ($\overline{X}$ = 21.55; sd = 4.19). Overall, participant scores on the BDI-II were within the range of 0-34 ($\overline{X}$ = 10.50; sd = 6.75). This is consistent with norms among college students ($\overline{X}$ = 9.27; sd = 8.07). On this measure, total scores of 0-13 is considered minimal range (77.8%), 14-19 is mild (12.5%), 20-28 is moderate (7.2%), and 29-63 is severe (2.4%). Participants report a score range of 20-72 ($\overline{X}$ = 36.74; sd = 10.22) on State Anxiety (STAI-A) and a range of 21-67 ($\overline{X}$ = 39.36; sd = 10.26) on Trait Anxiety (STAI-B). Previous research suggests that an average score range for college students would be approximately 26.47 to 46.47 (36.47 ± 10.02). The average score provided by participants on the IES, which rates symptoms consistent with PTSD, was 23.36 (sd = 18.55). Thirty percent of participants scored above 33, which is considered the cutoff score for probable diagnosis of PTSD.

Table 3

Number and Percentage of Participants Reporting Traumatic Events on the SLESQ

Traumatic Event	Number of Participants	Percentage
Family Member, Partner, Close Friend Dying due to suicide, homicide, or accident	37	28.68%
Emotional Abuse	31	24.03%
Life Threatening Accident	19	11.62%
Other	17	13.17%
Life Threatening Illness	15	11.62%
Sexual Assault/Molestation	15	11.62%
Witness Assault or Murder of Others	15	11.62%
Threatened with Weapon (knife, gun, etc.)	14	10.85%
Physical Abuse as Child, Victim	13	10.07%
Intimate Partner Violence	13	10.07%
Physical Force or Weapon in Robbery	11	8.52%
Sexual Assault as Victim	7	5.42%

Table 4

Number and Percentage of Participants Reporting Stressful Life Experiences on the LES.

Stressful Life Experience	Number of Participants	Percentage
Failing an Important Exam	59	50.00%
Minor Law Violations (tickets, citations)	34	28.81%
Financial Problems Concerning School	29	24.57%
Failing a Course	24	20.33%
Death of Close Friend	22	18.64%
Academic Probation	14	11.86%
Sexual Difficulties	13	11.01%
Dismissed from Dormitory or Residence	9	7.62%
Trouble with Employer	7	5.93%
Foreclosure on Mortgage or Loan	6	5.08%
Detention in Jail	6	5.08%
Trouble with In-laws	4	3.38%
Separation from Spouse (work, travel, etc.)	2	1.69%
Death of Spouse	1	0.84%

Neuropsychological functioning

The mean completion time among participants on Trail A was 22.58 (sd = 9.45) and Trail B was 58.57 (sd =27.16). The mean number of errors among participants for Trail A was 0.50 (sd = 1.2) and for Trail B was 0.91 (sd = 1.60). The mean score for the Attention score of the TMT (Trail B-A) was 31 (sd = 16.67). The mean normed scores among healthy adults are Trail A= 21.48 (sd = 6.44), Trail B= 48.77 (sd = 18.66), and Attention=26.03 (sd = 12.08; Strauss, Sherman, & Spreen, 2006). Within this sample, Trail B and Attention scores are higher than in normed populations. The mean number of correct words remembered on the RAVLT for immediate recall was 10.63 (sd = 2.31) and 10.19 (sd = 2.79) for delayed recall. Metanorms estimate an average score within healthy adults as 11.5 (sd = 2.3) for immediate recall and 11.3 (sd = 2.5) for delayed recall. The mean number of errors on the WCST among participants was 15.33 (sd = 8.33). The mean number of correct responses on the N-back test was 53.14 (sd = 21.81).

Main Analyses

Prior to completion of analyses for the main hypotheses, preliminary correlational analyses revealed that psychological measures and neuropsychological measures were not moderately correlated (r = 0.3-0.6; Pallant, 2010), suggesting that it would be more appropriate to conduct separate multivariate analyses for these two factors in subsequent analyses. To control for increased family wise Type 1 error rate, Bonferroni corrected p-values of p = .03 and p = .005 (.05/2 and .01/2, respectively) were utilized to judge statistical significance for each hypothesis. For each analysis, preliminary testing was completed to ensure assumptions of the

statistical model were met. In addition, sample size to ensure an adequate level of power (.8) was calculated using G*Power sample size calculation software.

Hypothesis 1: Increased number of traumatic/stressful life experiences will be associated with increased scores on measures of psychological functioning (BDI-II, STAI-B, IES-R, PSS) and with impaired scores on neuropsychological measures (WCST, TMT, RAVLT-DR, N-back test)

Psychological Functioning. To evaluate the hypothesis that participants reporting higher numbers of traumatic/stressful life experiences had higher scores on measures of psychological symptoms of depression (BDI-II), anxiety (STAI-B), posttraumatic stress (IES-R), and current perceived stress (PSS), a one way multivariate analysis of variance (MANOVA) was completed. The independent variable was the total number of negative stressful/traumatic experiences reported by the participant on the SLESQ and LES. When evaluating the assumption of equality of error variance, the dependent variable of BDI-II displayed unequal error variance across groups, as judged by a Levene test, $F(2,125) = 4.03$, $p = .02$. As described in Pallant (2010), violations of this assumption when having more than 30 cases in each cell of the variable are not likely to influence the results of the analysis given the robustness of the statistic at sufficient sample sizes. As suggested in Myers, Gamsti, and Guarino (2013), this variable was included in the analysis, but a more stringent alpha level was utilized to judge statistical significance of univariate effects for this variable ($p = .006$). In addition, follow up univariate analyses utilized the Dunnett T3 t-test in SPSS, which does not assume equal error variance across levels of the number of traumas/stressful events experienced. Results of the MANOVA are listed below in Table 5.

Table 5

Multivariate ANOVA Testing Effects of Number of Negative Stressful/Traumatic Events Experienced on Depression, Anxiety, Perceived Stress, and Posttraumatic Stress.

	Value	F	Hypothesis df	Error df	Sig.	Eta squared
Wilks Lambda	.81	3.39*	8	240	.001	.10

* significant at the p = .01 level

As listed in Table 5, a one-way MANOVA revealed a significant main effect for the number of stressful/traumatic events experienced, Wilks Lambda = .81, F (8,240) = 3.39, p = 0.001. To further evaluate the effect of the number of events experienced, a series of univariate analyses were completed, using Bonferroni corrected alpha levels to determine statistical significance, with a critical value of p = .01 (.05/4) Univariate main effects were observed in scores on the PSS, $F(2,123) = 6.21$, p = .00, partial eta squared = .09, BDI-II, F (2,123) = 10.47, p = .00, partial eta squared = .15, and IES-R, $F(2,123) = 4.60$, p = .01, partial eta squared = .07 (see Table 6).

Post hoc analyses reveal significant differences between participants reporting experiencing 0-2 traumatic/stressful life events and those reporting experiencing 6 or more events on the PSS, p = .00, with those experiencing 6 or more events reporting increased scores than those experiencing 0-2 events. Participants experiencing 6 more events also report higher IES-R scores than those experiencing 0-2 events, p = .01. Significant differences in BDI-II scores among participants reporting 0-2 and 3-5 events were also observed, p = .00, with those experiencing 3-5 events reporting increased scores. Participants reporting 6 or more events also

scored significantly higher on the BDI-II than did those reporting 0-2 events, $p = .00$. Overall, the more traumatic/stressful events reported by the participant, the higher the reported symptoms of depression, posttraumatic stress, and perceived stress. Significant differences emerged between individuals reporting 0-2 events and 6 or more events, but those experiencing 3-5 events were not significantly different than those reporting 6 or more events. Figure 1 displays the mean scores across the PSS, BDI-II, IES-R, and STAI-B measures and reveals the linear trend in scores across events experienced.

Table 6

Univariate ANOVA Analyses Testing Effects of Number of Stressful/Traumatic Events Reported on PSS, IES-R, and STAI-B Scores.

Dependent Variable		Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
PSS	Contrast	217.06	2	108.53	6.21**	.00	.09
	Error	2149.93	123	17.48			
BDI-II	Contrast	837.45	2	418.57	10.47**	.00	.15
	Error	4918.82	123	39.99			
IES-R	Contrast	2994.12	2	1497.06	4.60*	.01	.07
	Error	40013.41	123	363.30			
STAI-B	Contrast	678.55	2	339.28	3.30	.04	.05
	Error	40013.41	123	102.90			

* significant at the p = .01 level, **- significant at the p<.003 level.

Table 7

Mean Scores on PSS, BDI-II, IES-R, and STAI-B of Low, Medium, and High Groups of Numbers of Events Experienced (with Standard Deviations in Parentheses)

	Number of Events Experienced		
Psychological Measures	0-2	3-5	6 or more
PSS	19.98 (0.57)	21.82 (0.60)	23.46 (0.78)
BDI-II	6.87 (0.71)	10.80 (0.84)	13.19 (1.36)
IES-R	18.37(2.66)	22.75 (2.44)	30.49 (3.36)
STAI-B	36.50 (1.50)	40.66 (1.53)	41.89 (1.69)

Note. N = 126.

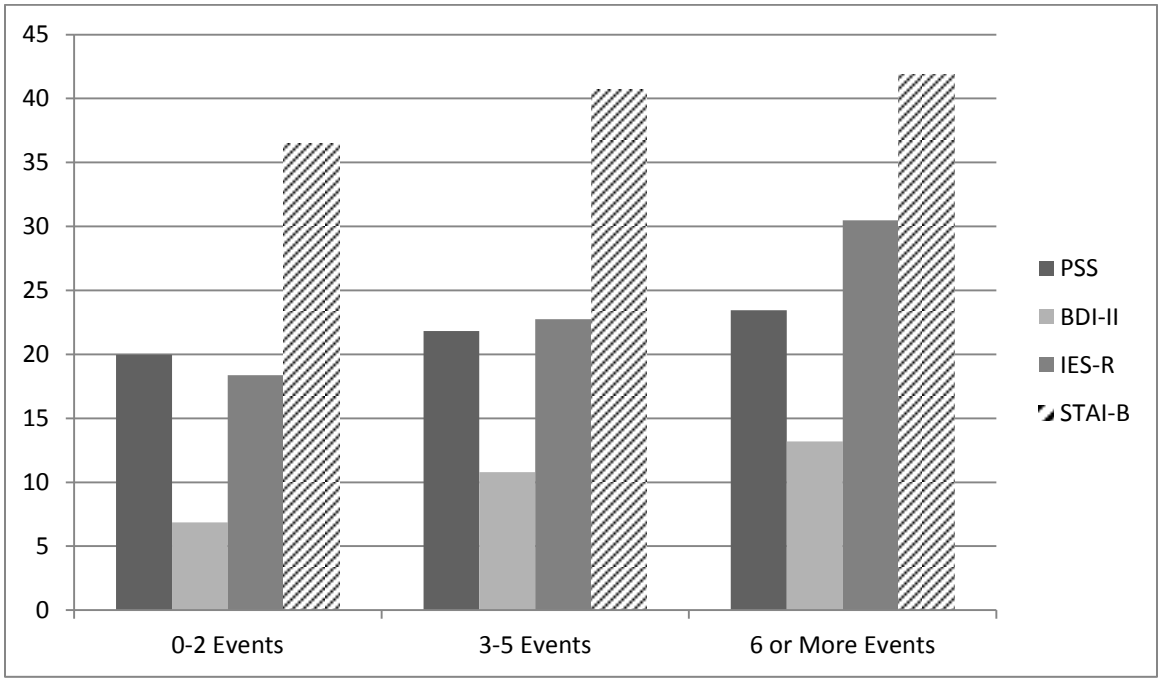


Figure 1. Means of psychological functioning scores by number of stressful/traumatic events.

Neuropsychological Functioning. To evaluate the hypothesis that participants reporting higher numbers of traumatic/stressful life experiences will have impaired scores on measures of neuropsychological functions of attention (TMT), working memory (N-back test), cognitive flexibility (WCST),and explicit memory (RAVLT-DR), a one way MANOVA was completed. Results of the multivariate analysis are listed below in Table 8 and indicate that the number of reported traumatic and stressful events was not associated with variance in scores on neuropsychological measures. Means and standard deviations of the neuropsychological measures across groups of traumatic events are listed in Table 9.

Table 8

Multivariate ANOVA Testing Effects of Number of Negative Stressful/Traumatic Events Experienced on TMT-Attention, RAVLT-DR, WCST, and N-back Test.

	Value	F	Hypothesis df	Error df	Sig.	Eta squared
Wilks Lambda	.96	.60	8	246	.77	.02

* significant at the p = .01 level, **- significant at the p<.002 level.

Table 9

Mean Scores on N-back, WCST, TMT-Attention, and RAVLT-DR of Low, Medium, and High Groups of Numbers of Events Experienced (with Standard Deviations in Parentheses)

Neuropsychological Measures	Number of Events Experienced		
	0-2	3-5	6 or more
N-Back	56.77 (2.90)	52.80 (3.20)	48.95 (3.10)
WCST	15.72 (1.18)	15.60 (1.40)	14.51 (1.21)
TMT-Attention	36.34(3.16)	37.40 (4.77)	35.51 (3.49)
RAVLT-DR	9.89 (0.40)	10.47 (0.40)	10.22 (0.53)

Note. N = 129.

Hypothesis 2: Participants experiencing interpersonal relationship trauma/stressors will report increased scores on measures of psychological functioning (BDI-II, STAI-B, IES-R, PSS) and will have impaired scores on neuropsychological measures (WCST, TMT, RAVLT-DR, N-back test)compared to those experiencing non-interpersonal relationship trauma/stressors

To determine the effects of experiencing interpersonal traumatic stress on psychological and neuropsychological functioning, participants were divided into categories based on their self-reported traumatic events experienced on the SLESQ. Those reporting experiencing sexual assault, molestation, physical abuse as a child or adult, or emotional abuse were placed within the interpersonal trauma group. Those reporting experiencing life threatening illness or injury, mugging, traumatic loss, witnessing serious injury or death of another person, or military combat

were assigned into the non-interpersonal trauma group. Participants indicating that they had not experienced an event on the SLESQ were assigned into the no trauma control group.

Psychological Functioning. To evaluate the hypothesis that participants experiencing interpersonal trauma would report higher scores on measures of psychological symptoms of depression (BDI-II), anxiety (STAI-B), posttraumatic stress (IES-R), and current perceived stress (PSS) than would participants experiencing non-interpersonal traumas (i.e. serious illness/injury, car accidents, mugging, witnessing another individual being killed or injured) or no trauma controls, a one way MANOVA analysis was completed. Results are listed below in Table 10.

Table 10

Multivariate ANOVA Testing Effects of Type of Trauma (Interpersonal, Non-Interpersonal, no Trauma Control) on Depression, Anxiety, Perceived Stress, and PTSD.

	Value	F	Hypothesis df	Error df	Sig.	Eta squared
Wilks Lambda	.82	3.17	8	240	0.002	0.10

* significant at the p<0.05 (p = .025) level, **- significant at the p< 0.01 (p<.005) level.

As listed in Table 10, a one-way MANOVA revealed a significant main effect for the type of trauma experienced (interpersonal, non-interpersonal, no trauma) on psychological functioning, Wilks Lambda=0.82, F (8,240) = 3.17, p = 0.002. To further evaluate the effect observed in the multivariate analysis, a series of univariate analyses were completed, using a Bonferroni corrected alpha level of *p* = .01 (.05/4). Univariate main effects were observed in scores on the BDI-II, PSS, and IES-R (see Table 11). Participants experiencing interpersonal relationship trauma reported higher mean scores on the BDI-II than did participants in the no

trauma control group, p = .000, but did not differ from those reporting non-interpersonal types of trauma, p = .35. Individuals experiencing interpersonal trauma also report significantly higher scores on the PSS than do no trauma controls, p = 0.001. Scores among those reporting interpersonal trauma trended towards being significantly higher than those experiencing non-interpersonal trauma, p =.03. In addition, participants experiencing interpersonal trauma reported higher mean scores on the IES-R than did no trauma controls, p = .01. Means and standardization for each group across measures are included in Table 12 and Figure 2.

Table 11

Univariate ANOVA Analyses Testing Effects of Type of Trauma (Interpersonal, Non-Interpersonal, No Trauma Control) on Depression, Anxiety, Perceived Stress, and PTSD.

Dependent Variable		Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
PSS	Contrast	276.64	2	138.31	8.14**	.00	.12
	Error	2090.35	123	16.99			
BDI-II	Contrast	674.33	2	337.17	8.16**	.00	.12
	Error	5081.64	123	41.31			
IES-R	Contrast	3119.12	2	1559.56	4.81*	.01	.07
	Error	39888.40	123	324.30			
STAI-B	Contrast	840.13	1	420.07	4.14	.02	.06
	Error	12495.36	123	101.59			

* significant at the p<0.05 (p = .01) level, **- significant at the p< 0.01 (p<.002) level.

Table 12

Mean Scores on PSS, BDI-II, IES-R, and STAI-B of No Trauma Controls, Non-Interpersonal, and Interpersonal Groups of Type of Trauma Experienced (with Standard Deviations in Parentheses)

	Type of Trauma Experienced		
Psychological Measures	No Trauma	Non-Interpersonal	Interpersonal
PSS	20.13 (3.6)	20.88 (3.53)	23.48 (4.91)
BDI-II	7.20 (4.89)	10.18 (6.47)	12.57 (7.61)
IES-R	18.73(17.08)	20.75 (16.48)	29.73 (19.82)
STAI-B	36.46 (8.32)	39.48 (10.02)	42.47 (11.58)

Note. $N = 126$.

A one way MANOVA was completed to evaluate the hypothesis that participants experiencing interpersonal relationship trauma would have impaired scores on measures of neuropsychological functions of attention (TMT), working memory (N-back test), cognitive flexibility (WCST),and explicit memory (RAVLT-DR) relative to those experiencing non-interpersonal trauma or no trauma controls. Results of the multivariate analysis are listed below in Table 13 and indicate non-significant effects of the interpersonal nature of trauma on neuropsychological functioning measures, $F(8,246) = .94$, $p = .49$. Table 14 provides the means and standard deviations across groups of trauma type.

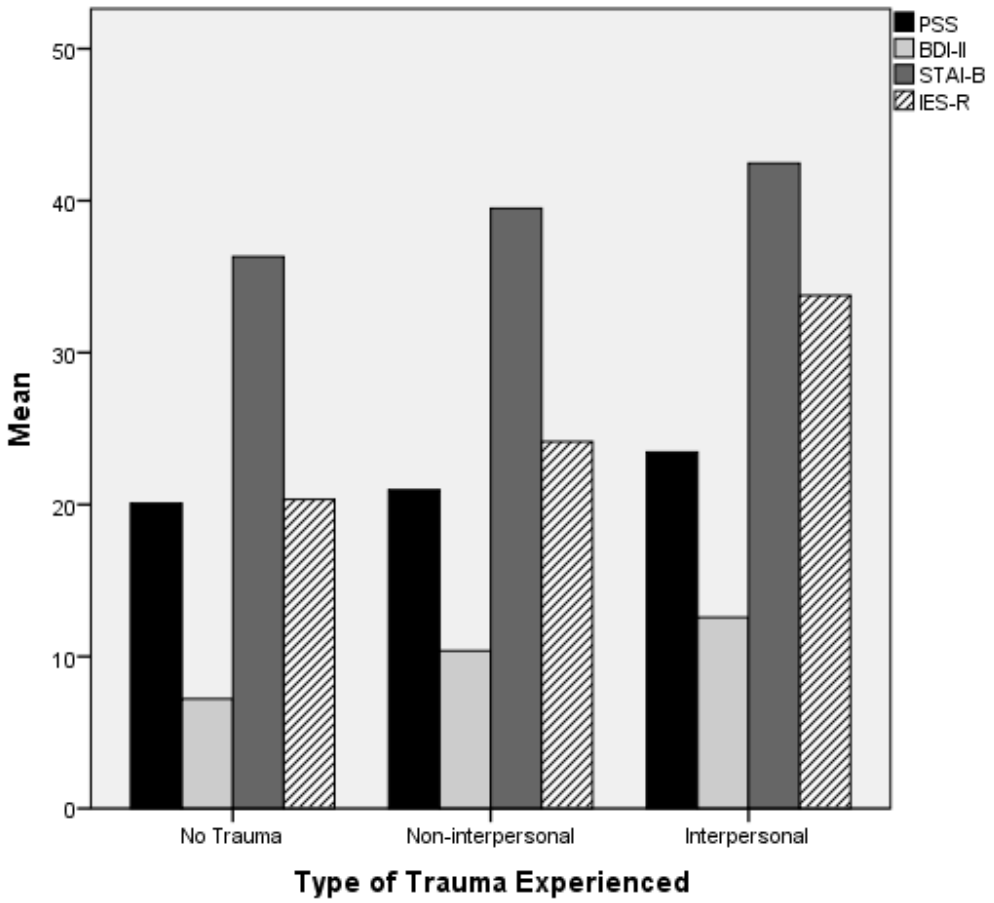


Figure 2. Means of psychological functioning scores by types of trauma experienced.

Table 13

Multivariate ANOVA Testing Effects of Interpersonal, Non-Interpersonal Stressful/Traumatic Events Experienced, and No Trauma Controls on TMT-Attention, WCST, RAVLT-DR, and N-back Test.

	Value	F	Hypothesis df	Error df	Sig.	Eta squared
Wilks Lambda	.94	.94	8	246	.49	.03

* significant at the p = .01 level, **- significant at the p<.002 level.

Table 14

Mean Scores on TMT-Attention, WCST, RAVLT-DR, and N-back test of No Trauma Controls, Non-Interpersonal, and Interpersonal Groups of Type of Trauma Experienced (with Standard Deviations in Parentheses)

	Type of Trauma Experienced		
Psychological Measures	No Trauma	Non-Interpersonal	Interpersonal
TMT-Attention	32.89 (15.10)	36.00 (15.35)	33.44 (19.07)
WCST	15.64 (8.71)	14.82 (8.08)	15.40 (8.29)
RAVLT-DR	10.15(2.57)	10.68 (2.18)	9.88 (3.31)
N-Back	55.49 (21.25)	47.03 (19.99)	55.17 (23.16)

Note. N = 129.

Hypothesis 3: PTSD reported symptomology will be associated with increased scores on measures of psychological functioning (BDI-II, STAI-II, PSS) and with impaired scores on neuropsychological measures (WCST, TMT, RAVLT-DR, N-back test) relative to no trauma controls

To quantify PTSD status, participants were divided into 3 groups based on their reported experience of a potentially traumatic event (an event listed on the SLESQ) and their scores on the IES-R, with those having experienced an event on the SLESQ and scoring above 33 placed in the PTSD group and those experiencing an event on the SLESQ and scoring below 33 being placed in the trauma no PTSD group (Creamer et al., 2003). Participants not experiencing an event on the SLESQ were placed into the no trauma control group.

Psychological Functioning. To evaluate the hypothesis that participants reporting increased symptomology of posttraumatic stress would have higher scores on measures of psychological symptoms of depression (BDI-II), anxiety (STAI-II), and current perceived stress (PSS), a one way MANOVA was completed. Results of the multivariate analysis are listed below in Table 15.

Table 15

Multivariate ANOVA Testing Effects of PTSD Status on Anxiety, Perceived Stress, and Depression.

	Value	F	Hypothesis df	Error df	Sig.	Eta squared
Wilks Lambda	.72	7.35**	6	248	.00	.15

* significant at the p = .01 level, **- significant at the p<.002 level.

As listed in Table 15, a one-way MANOVA revealed a significant main effect for the reported PTSD status, Wilks Lambda = .72, F (6,248) = 7.35, p = 0.00. To further evaluate the effect observed in the multivariate analysis, a series of univariate analyses were completed, using Bonferroni corrected alpha levels to determine statistical significance, with a critical value of *p* =.02 (.05/3) Univariate main effects were observed in scores on the BDI, *F* (2,126) = 12.10, *p* =.00, partial eta squared = .16 (see Table 16). Post hoc analyses revealed significantly higher BDI-II scores in participants in the PTSD group compared to no trauma controls, *p* = .00, and to experiencing trauma but without PTSD symptoms, *p* =.04. No significant differences in BDI-II scores were found among those in the no trauma control group and those exposed to trauma without PTSD level symptoms, *p* =.14. Follow-up analyses of the univariate effect on the PSS

scores, F (2,126) = 17.58, p .00, partial eta squared = .22, reveal that participants in the PTSD group had significantly higher PSS scores than did those within the no trauma control group, p =.00, and the trauma without PTSD group, p =.00. No significant differences in PSS scores were found among those in the no trauma control group and those exposed to trauma without PTSD level symptoms, p =.43. Post hoc analyses exploring the univariate effect of STAI-B scores, F (2,126)=4.82, p =.01, partial eta squared .07, revealed significantly higher scores among the PTSD group compared to the no trauma control group, p =.01, but no differences between the PTSD group and the trauma no PTSD group, p =.15, or the no trauma control group and the trauma no PTSD group, p = 1.00. Table 17 and Figure 3 provide means and standard deviations for the BDI-II, PSS, and STAI-B across groups of PTSD status.

Table 16

Univariate ANOVA Analyses Testing Effects of PTSD Status on BDI-II, PSS, and STAI-B Scores.

Dependent Variable		Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
BDI-II	Contrast	938.56	2	469.28	12.10**	.00	.16
	Error	4886.07	126	38.78			
PSS	Contrast	535.99	2	267.99	17.58**	.00	.22
	Error	1920.40	126	15.24			
STAI-B	Contrast	958.75	2	479.38	4.82	.01*	.07
	Error	12521.12	126	99.37			

* significant at the p = .02 level, **- significant at the p<.003 level.

Table 17

Mean Scores on BDI-II, PSS, and STAI-B of No Trauma Controls, Trauma Without PTSD, and PTSD Groups of PTSD Status (with Standard Deviations in Parentheses)

	Type of Trauma Experienced		
Psychological Measures	No Trauma	Trauma Without PTSD	PTSD
BDI-II	8.14 (5.73)	10.89 (6.28)	15.21 (7.60)
PSS	20.34 (3.64)	21.61 (3.56)	25.75 (4.97)
STAI-B	37.64 (8.88)	39.39 (11.18)	44.87 (11.71)

Note. N = 129.

Neuropsychological Functioning. To evaluate the hypothesis that participants reporting increased symptomology of posttraumatic stress would have impaired scores on measures of neuropsychological functions of attention (TMT), working memory (N-back test), cognitive flexibility (WCST),and explicit memory (RAVLT-DR), a one way MANOVA was completed. Results of the multivariate analysis are listed below in Table 18 and indicate non-significant effect of PTSD status on scores on TMT, RAVLT, WCST, and the N-back test, $F(8,246) = 1.15$, $p = .33$. Table 19 and Figure 4 provide the means and standard deviations across groups of PTSD status.

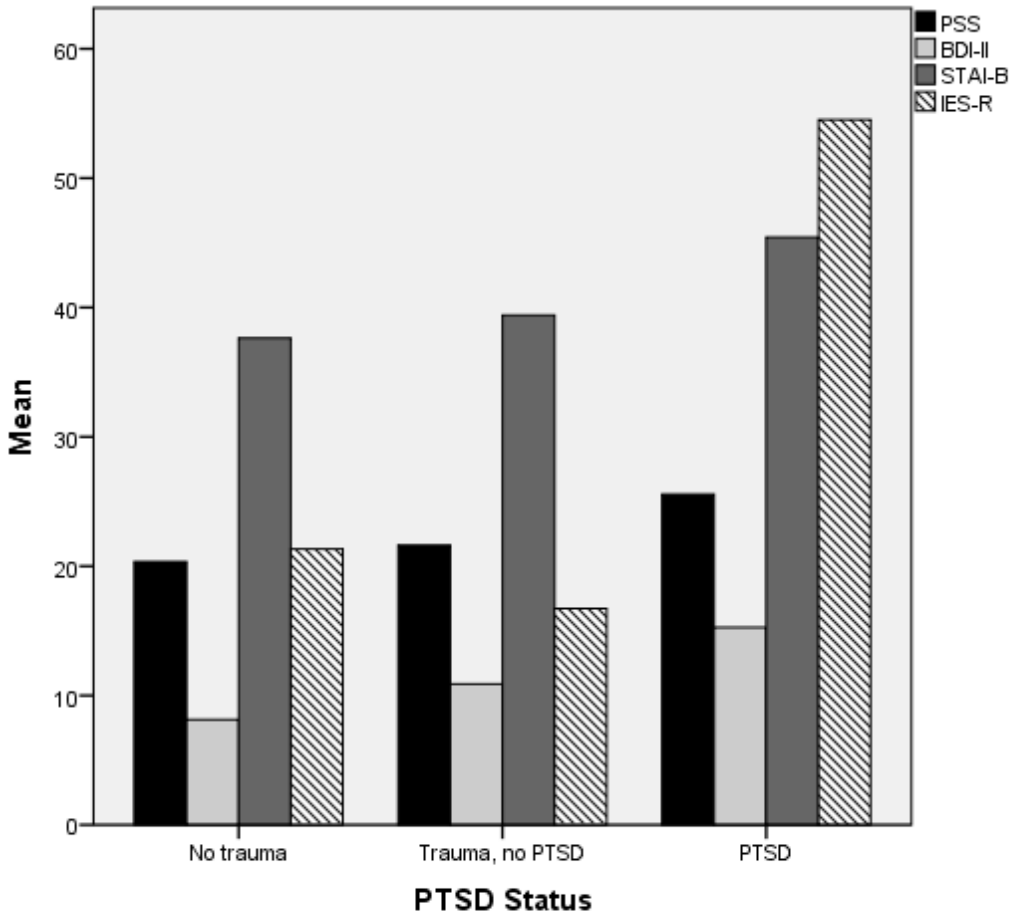


Figure 3. Means of psychological functioning by PTSD status.

Table 18

Multivariate ANOVA Testing Effects of PTSD Status on TMT-Attention, RAVLT-DR, and N-back Test.

	Value	F	Hypothesis df	Error df	Sig.	Eta squared
Wilks Lambda	.93	1.15	8	246	.33	.04

* significant at the p = .01 level, **- significant at the p<.002 level.

Table 19

Mean Scores on TMT-Attention, WCST, RAVLT-DR, and N-back test of No Trauma Controls, Trauma Without PTSD, and PTSD Groups of PTSD Status (with Standard Deviations in Parentheses)

	PTSD Status		
Psychological Measures	No Trauma	Trauma Without PTSD	PTSD
TMT-Attention	34.62 (16.26)	33.96 (17.46)	31.58 (17.54)
WCST	15.52 (8.40)	13.71 (6.22)	16.63 (10.13)
RAVLT-DR	10.18 (2.52)	10.36 (2.63)	10.00 (3.72)
N-Back	53.57 (22.04)	59.07 (19.70)	44.85 (21.71)

Note. $N = 129$.

Hypothesis 4: Younger age at the time of trauma reported on IES-R will be associated with increased scores on measures of psychological functioning (BDI-II, STAI-II, IES-R, PSS) and with impaired scores on neuropsychological measures (WCST, TMT, RAVLT-DR, N-back test)

Psychological Functioning. To evaluate the hypothesis that participants reporting younger age at time of traumatic/stressful life experiences would have higher scores on measures of psychological symptoms of depression (BDI-II), anxiety (STAI-B), posttraumatic stress (IES-R), and current perceived stress (PSS), a one way MANOVA analysis was completed. Results of the multivariate analysis are listed below in Table 20 and indicate a significant multivariate effect of age at time of trauma on psychological functioning, F (12,241) = 2.02, p = .02.

Table 20

Multivariate ANOVA Testing Effects of Age of Trauma Reported on IES-R on Depression, Anxiety, Posttraumatic Stress, and Perceived Stress.

	Value	F	Hypothesis df	Error df	Sig.	Eta squared
Wilks Lambda	.78	2.02*	12	241	.02	.08

* significant at the p = .02 level, **- significant at the p<.005 level.

After utilizing bonferroni corrected alpha values for follow-up analyses (.05/4 = .01), none of the univariate effects were significant. A trend towards significance was observed in the IES-R score variable, F (3,94) = 2.67, p =.05, partial eta squared = .06. Post hoc tests indicate that participants reporting experiencing the event above age 18 had higher IES-R scores than did those reporting the event occurred during early childhood (ages 5-9), p =.03. Means and standard deviations for age groups are listed in Table 21.

Neuropsychological Functioning. To evaluate the hypothesis that participants reporting younger ages at the time of the traumatic event occurred on the IES-R will have impaired scores on measures of neuropsychological functions of attention (TMT), working memory (N-back test), cognitive flexibility (WCST),and explicit memory (RAVLT-DR), a one way MANOVA was completed. Results of the multivariate analysis are listed below in Table 22 and indicate non-significant effect of age at time of trauma on scores on TMT, RAVLT, and N-back test, F(12,241) = .52, p = .90. Means and standard deviations are provided in Table 23.

Table 21

Mean Scores on BDI-II, PSS, STAI-B, and IES-R of Early Childhood (5-9), Childhood (10-13), Adolescence (14-17), and Adult (18 and over) Groups of Age at Time of Trauma (with Standard Deviations in Parentheses)

	Age at Time of Trauma			
Psychological Measures	Age 5-9	Age 10-13	Age 14-17	Age 18+
BDI-II	10.44 (2.28)	8.50 (2.16)	9.29 (1.17)	9.64 (1.02)
PSS	20.78 (1.38)	22.50 (1.31)	21.18 (0.71)	21.33 (0.62)
STAI-B	43.33 (3.33)	32.40 (3.16)	39.06 (1.71)	37.29 (1.49)
IES-R	11.22 (7.07)	18.50 (6.70)	24.65 (3.64)	30.76 (3.16)

Note. N = 129.

Table 22

Multivariate ANOVA Testing Effects of Number of Age at Time of Trauma on TMT-Attention, RAVLT-DR, and N-back Test.

	Value	F	Hypothesis df	Error df	Sig.	Eta squared
Wilks Lambda	.93	.52	12	241	.90	.02

* significant at the p = .01 level, **- significant at the p<.002 level.

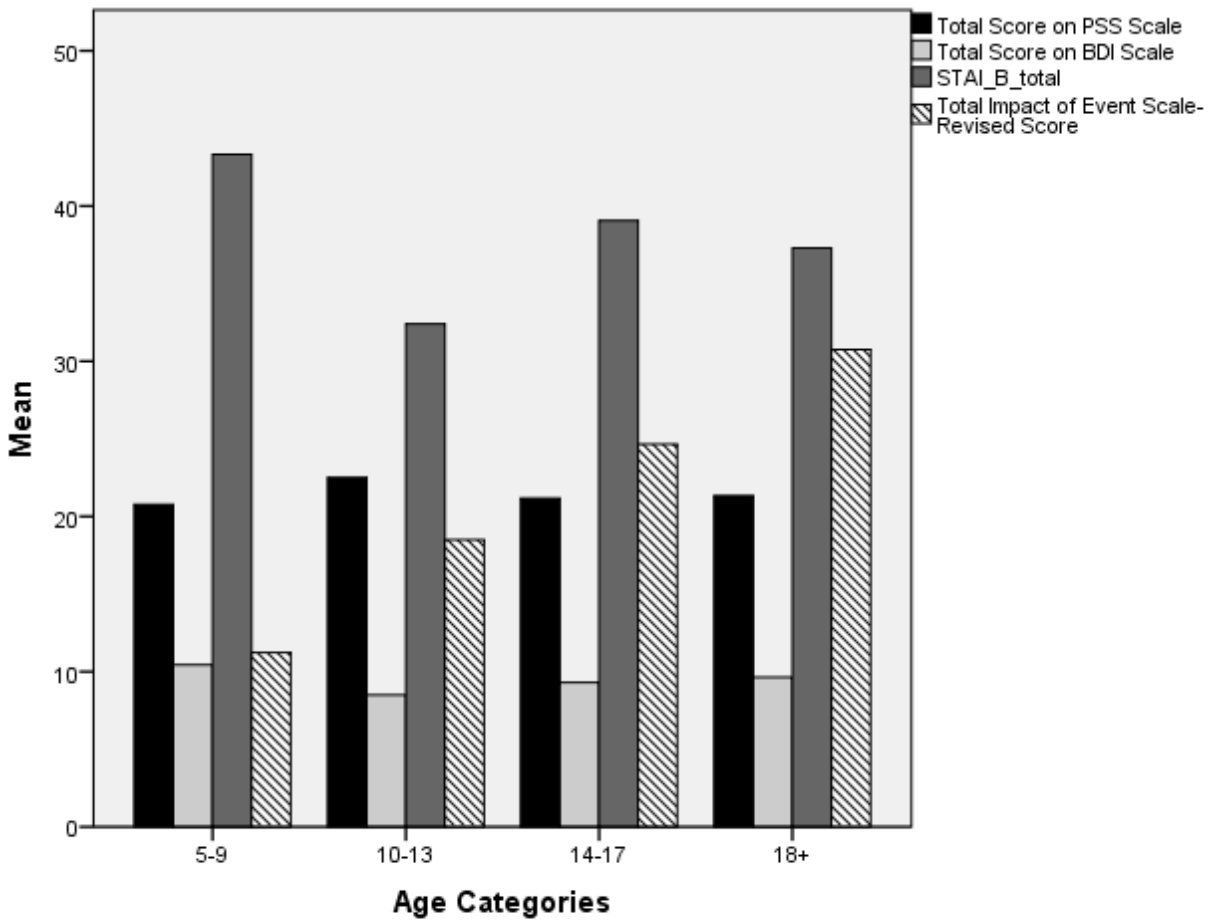


Figure 4. Means of psychological functioning scores by age at time of trauma.

Table 23

Mean Scores on TMT-Attention, WCST, RAVLT-DR, and N-back test of Early Childhood (5-9), Childhood (10-13), Adolescence (14-17), and Adult (18 and over) Groups of Age at Time of Trauma (with Standard Deviations in Parentheses)

Psychological Measures	Type of Trauma Experienced			
	Age 5-9	Age 10-13	Age 14-17	Age 18+
TMT-Attention	35.56 (5.16)	32.30 (11.80)	36.56 (2.66)	15.98 (1.21)
WCST	16.22 (2.71)	15.10 (2.57)	13.27 (1.40)	15.98 (1.21)
RAVLT-DR	11.00 (0.93)	10.60 (0.88)	10.15 (0.48)	10.04 (0.41)
N-Back	55.78 (7.03)	52.00 (6.67)	49.34 (3.62)	54.98 (3.14)

Note. $N = 129$.

Exploratory Hypotheses

Exploratory Hypothesis 1: Reported levels of distress and the frequency/duration of traumatic experiences would mediate or moderate the effect of the total number of traumas on psychological (BDI-II, STAI-II, IES-R, PSS) and neuropsychological (TMT, WCST, RAVLT-DR, N-back test) functioning

While few studies have explored the influence of trauma on functioning in college students, those that have did not include measure of the subjective levels of distress experienced by the participants in response to events or the frequency and duration of the trauma events. Within this study, two measures of stressful and traumatic experiences were included, the SLESQ and LES. Correlational analyses reveal differing effects of these two surveys on psychological and neuropsychological functioning (see Table 24). The SLESQ includes events

that are more atypical in everyday experiences (i.e. child abuse, sexual assault, robbery, etc.), while the LES includes events that are more common (i.e. death of loved ones, financial struggles, failing an exam). As such, scores on the SLESQ were included in this analysis to further explore the effects of these less common traumatic events on functioning. To explore the impact of the level of distress, additional questions were added to the SLESQ asking participants to rate their level of distress following each trauma that they experienced.

Table 24

Correlations Among Total Events on SLESQ and LES and Executive and Psychological Functioning.

	BDI	STAI-B	IES	PSS	TMT	DR	WCST	N-back
SLESQ								
r value	.44**	.35**	.37**	.39**	.07	-.03	.05	.10
p-value	.01	.00	.00	.00	.43	.78	.57	.24
LES								
r value	.27**	.36**	.22*	.09	.00	.04	-.03	.01
p-value	.00	.00	.02	.36	.97	.65	.71	.95

* significant at the p = .05 level, **significant at the p<.01 level.

To examine the hypothesis that the level of distress experienced and the frequency and duration of the traumatic events mediate or moderate the relationship between the number of lifetime events reported, a series of multiple regression analyses were completed. These analyses were completed to determine which of the criterion variables were significantly explained by the model of variables of total number of SLESQ events experienced, frequency/duration, and

distress. To reduce aggregate Type I error, Bonferroni adjusted p-values were utilized to determine significance of findings (p = .007). Table 25 lists the results of these analyses and shows that only scores on the BDI, IES-R, STAI-B, and PSS were significantly explained by the model. As such, additional analyses for mediating and moderating effects were only completed with those variables.

Mediating variables are prediction variables that, once added into the regression equation, reduce the effect of another predictor variable in the model on the criterion or dependent variable. In effect, they explain the effect of that variable by partitioning out the variance. Using a pathway analysis approach, the initial relationship between the number of events experienced on the SLESQ was measured for the dependent variable (Myers et al., 2013). Then the relationship between the number of events experienced and the potential mediator (frequency/duration or distress), as well as the relationship between the potential mediator and the criterion/dependent variable (BDI, STAI-B, IES scores) were measured. If after controlling for the effects of the mediator, the original criterion variable of the number of events experienced was reduced to a non-significant level of effect, then the presence of a mediating effect was confirmed. To ensure that any mediating effects were statistically significant, Sobel tests were also completed for all models. The predictors were the number of traumas reported on the SLESQ, total distress reported on the SLESQ, and the combined total frequency and duration of events reported on the SLESQ.

Table 25

One way ANOVA Testing Effects of Number of Negative Stressful/Traumatic Events Experienced, Level of Distress, and Frequency/Duration on Psychological (BDI-II, STAI-II, IES-R, PSS) and Neuropsychological (TMT, WCST, RAVLT-DR, N-back test) Functioning.

Dependent Variable		Sum of Squares	df	Mean Square	F	Sig.
BDI-II	Regression	14.62	3	4.89	5.64*	.002
	Residual	65.92	77	.87		
IES-R	Regression	62.32	3	20.78	5.59*	.002
	Residual	286.15	77	3.72		
PSS	Regression	268.34	3	89.45	5.44*	.002
	Residual	1266.91	77	16.45		
STAI-B	Regression	1283.71	3	427.91	4.61*	.005
	Residual	7141.21	77	92.74		
RAVLT-DR	Regression	64.79	3	21.60	2.85	.043
	Residual	568.77	75	7.58		
N-back	Regression	1610.39	3	536.80	1.09	.360
	Residual	36506.97	74	493.34		
TMT-Attn	Regression	4.61	3	1.54	.74	.530
	Residual	159.56	77	2.07		
WCST	Regression	.03	3	.01	.255	.858
	Residual	3.27	76	.04		

* significant at the p = .006 level, **- significant at the p<.001 level.

Moderation in regression is similar to interaction effects within ANOVA equations. A moderating variable is a predictor variable that, when added within the regression equation,

influences the strength and/or direction of the relationship that another predictor variable has on the criterion/dependent variable. In effect, a moderating variable influences the strength of the relationship of a predictor (independent) variable on a criterion (dependent) variable. To test for these effects within a regression model, interaction terms between the main predictor variable (number of traumas experienced) and the predicted moderator (frequency/duration or distress) were computed and then added into the regression model. If these were significant, it suggested the variable influenced the strength of the effect of the total numbers of traumas experienced on the criterion variable.

BDI-II. The results of the multiple regression equation indicated that a significant portion of the variance in BDI-II scores (transformed using square root transformation) was predicted by total traumas reported, total distress, and combined frequency and duration, F (3,77) = 5.64, p = 0.002. Multiple R squared indicated that 20% of the variance in the total number of traumatic and stressful life events experienced was accounted for by these factors. It was found that, among those factors, SLESQ total scores significantly predicted BDI-II scores (B = 0.85, p = 0.001) and independently accounted for 15% of the variance in BDI-II scores. The addition of total distress accounted for an additional 3.8% of variance (R square change = .038), but this was not a significant change (p = .06). The addition of total frequency/duration contributed to an additional 0.5% of the variance in BDI-II scores, but this was not significant (p = .46). Table 26 lists the results of the multiple regression model analysis. Table 27 provides a listing of Pearson product moment correlations among predictor variables with the dependent variable, total number of traumatic and stressful life events experienced. When the total number of SLESQ events experienced was controlled for, the effects of distress and frequency/duration are found to contribute to a small, non-statistically significant amount of variance in depression scores. This

suggests that these variables were not mediating or moderating factors in depression scores. A Sobel test indicated no significant mediating effect for distress (z = 1.40, *p* = .25) or frequency/duration (z = 1.84, *p* =.07). Regression equations of interaction effects with SLESQ total were non-significant for distress, *t* (3,79) = .94, *p* =.35 or frequency/duration, *t* (3,79) = .13, *p* = .47.

Table 26

Summary of Multiple Regression Analyses for Variables Predicting Variance in BDI-II Scores

Variable	B	SE(B)	Beta	t	Sig. (p)
SLESQ Total	0.51	0.15	0.85	3.50	0.00
SLESQ Total Distress	-0.07	0.04	-0.42	-1.85	0.07
SLESQ Total Frequency/Duration	-0.03	0.04	-0.11	-0.72	0.48

Note. R Square = 0.20

Table 27

Correlations Among Variables Within the Multiple Regression Analysis with Dependent Variable BDI-II Scores

Variable	Pearson r	Sig. (p)
SLESQ Total	0.40	0.000
SLESQ Total Distress	0.27	0.007
SLESQ Total Frequency/Duration	0.23	0.019

IES-R. The results of the multiple regression equation indicated that a significant portion of the variance in IES-R scores (transformed using square root transformation) was predicted by total traumas reported, total distress, and combined frequency and duration, F (3,77) = 5.59, *p* = 0.002. Multiple R squared indicates that 18% of the variance in IES-R scores is accounted for by these factors (R squared = .18). When initially entered into the model, SLESQ total scores significantly predicted IES-R scores (*B* = 0.38, *p* = 0.002) and independently accounted for 12% of the variance in IES-R scores (R squared = .12). The addition of total frequency/duration, however, reduced the individual effects of total events to being non-significant (*B* =.14, *p* = .41). A Sobel test indicates that frequency/duration significantly partially mediated the relationship between the total events experienced and posttraumatic symptomology, measured by IES-R (*z* = 1.96, *p* = .05). A Sobel test indicated no significant mediating effect for distress (z = -1.38, *p* = .17). Regression equations of interaction effects with SLESQ total were non-significant for distress, *t* (80) = -.80, *p* = .42 or frequency/duration, *t* (80) = -1.02, *p* = .31. Table 28 lists the results of the multiple regression model analysis. Table 29 provides a listing of Pearson product moment correlations among predictor variables with the dependent variable, scores on the IES-R.

Table 28

Summary of Multiple Regression Analyses for Variables Predicting Variance in IES-R Scores

Variable	B	SE(B)	Beta	t	Sig. (p)
SLESQ Total	-1.43	2.44	-.14	-.59	.61
SLESQ Total Distress	.09	.07	.29	1.27	.21
SLESQ Total Frequency/Duration	.15	.08	.29	1.92	.05

Note. R Square = 0.18

Table 29

Correlations Among Variables Within the Multiple Regression Analysis with Dependent Variable IES-R Scores

Variable	Pearson r	Sig. (p)
SLESQ Total	.34	.000
SLESQ Total Distress	.37	.000
SLESQ Total Frequency/Duration	.39	.000

STAI-II. The results of the multiple regression equation indicated that a significant portion of the variance in STAI-II scores (transformed using log transformation) was predicted by total traumas reported, total distress, and combined frequency and duration, F (3,77) = 2.82, *p* = 0.04. Multiple R squared indicates that 9% of the variance in the total number of traumatic and stressful life events experienced was accounted for by these factors (R squared = .09). It was found that, among those factors, SLESQ total scores significantly predicted STAI-II scores (*B* = 0.03, *p* = 0.04) and independently accounted for 8% of the variance in STAI-II scores. The addition of total distress accounted for an additional 0.6% of variance (R square change = .006), which was not a significant change (*p* = .47). The addition of total frequency/duration contributed to an additional 1% of the variance (R squared change = .01) in STAI-II scores, but this was not significant (*p* = .37). Table 30 lists the results of the multiple regression model analysis. Table 31 provides a listing of Pearson product moment correlations among predictor variables with the dependent variable, total number of traumatic and stressful life events experienced. When the total number of SLESQ events experienced was controlled for, the effects

of distress and frequency/duration were found to contribute to a small, non-statistically significant amount of variance in depression scores. This suggested that these variables were not mediating or moderating factors in depression scores. A Sobel test indicated no significant mediating effect for distress (z = .75, *p* = .45) or frequency/duration (z = .99, *p* = .32). A regression equation for an interaction effect between SLESQ total and frequency/duration revealed a significant interaction between the two factors, *t* (80) = 2.27, *p* = .03. Further exploration of the effect revealed that, for those individuals with higher scores on frequency/duration, experiencing more traumatic events as measured by the SLESQ was moderately correlated (r = .4) with increased scores on the STAI-II, but for those reporting low scores on frequency/duration, increased traumatic events were associated with decreased STAI-II scores (r = .1). Regression equations of interaction effects with SLESQ total were non-significant for distress, *t* (80) = 1.83, *p* = .07.

Table 30

Summary of Multiple Regression Analyses for Variables Predicting Variance in STAI-II Scores

Variable	B	SE(B)	Beta	t	Sig. (p)
SLESQ Total	.03	.02	.53	2.07	.04
SLESQ Total Distress	-.00	.00	-.16	-.64	.52
SLESQ Total Frequency/Duration	-.03	.04	-.11	-.72	.48

Note. R Square = 0.09

Table 31

Correlations Among Variables Within the Multiple Regression Analysis with Dependent Variable BDI-II Scores

Variable	Pearson r	Sig. (p)
SLESQ Total	.29	.01
SLESQ Total Distress	.22	.24
SLESQ Total Frequency/Duration	.13	.12

Exploratory Hypothesis 2: Reported levels of community involvement and religious affiliation would influence the effect of the total number of traumas on psychological (BDI-II, STAI-II, IES-R, PSS) and neuropsychological (TMT, WCST, RAVLT-DR, N-back test) functioning

To examine the hypothesis that community involvement and religious affiliation influence the relationship between the number of lifetime events reported, a series of multiple regression analyses were completed. The predictors were the number of traumas reported on the SLESQ, number of community organizations involved in, and the reported level of religious involvement. Table 32 lists regression equations for each predictor on each dependent variable. To reduce aggregate Type I error, only psychological measures were included in these analyses, as previous analyses have not revealed significant effects on neuropsychological measures. Bonferroni adjusted p-values were utilized to determine significance of findings ($p = .01$). As noted in the table below, the reported number of community organizations and reported level of

religious involvement did not significantly influence scores on the BDI-II, STAI-II, IES-R, or PSS.

Table 32

Summary of Multiple Regression Analyses for Variables Predicting Variance in BDI-II, STAI-II, IES-R, and PSS Scores

Variable	B	SE(B)	Beta	t	Sig. (p)
BDI-II Sqrt					
SLESQ Total	.20	.05	.38	4.44	.000
Community Organizations	-.02	.09	-.01	-.16	.871
Religious Involvement	.05	.08	.06	.65	.52
STAI-II Log					
SLESQ Total	.02	.01	.29	3.34	.001
Community Organizations	-.00	.01	-.03	-.33	.742
Religious Involvement	-.00	.01	-.00	-.00	.99
IES-R Sqrt					
SLESQ Total	.40	.09	.36	4.29	.000
Community Organizations	-.29	.20	-.12	-1.45	.149
Religious Involvement	-.23	.16	-.12	-1.45	.49
PSS Log					
SLESQ Total	.02	.00	.33	3.84	.000
Community Organizations	.00	.01	.02	.17	.865
Religious Involvement	.01	.01	.06	.74	.461

Exploratory Hypothesis 3: Receiving mental health services will contribute to differences in the effects of the number of traumatic events on psychological functioning (BDI-II, STAI-II, IES-R, and PSS)

A MANOVA was conducted to test the hypothesis that receiving mental health services would influence the impact of the number of traumatic/ stressful life events on psychological functioning. The dependent variables used in the analysis were BDI-II, STAI-II, IES-R, and PSS scores. The independent variables were mental health treatment (yes, no) and the number of events experienced divided into 3 groups, low (0-2), medium (3-5), and high (6 plus). As noted in Table 33, the multivariate interaction effect indicated that mental health treatment does not interact with the total number of events to contribute to variance in psychological functioning.

Table 33

Multivariate ANOVA Testing Interaction Effect of Number of Negative Stressful/Traumatic Events Experienced and Mental Health Treatment on Depression, Anxiety, Perceived Stress, and Posttraumatic Stress.

	Value	F	Hypothesis df	Error df	Sig.	Eta squared
Wilks Lambda	.95	.77	8	238	.63	.03

* significant at the p = .05 level, **- significant at the p = .01 level.

Exploratory Hypothesis 4: Increased drinking behavior would contribute to differences in the effects of the number of traumatic events on psychological functioning (BDI-II, STAI-II, IES-R, and PSS)

Table 34 provides the number and percentage of participants indicating various amounts of alcohol typically consumed while drinking. Most participants indicated they used alcohol (n = 93; 72.1%), 31% (n = 41) reported using alcohol weekly, 18% (n = 24) reported using monthly, 19% (n = 25), and 4.7% (n = 6) reported using three or more times per week. Ten percent of participants (n = 14) indicated that they use street drugs or misuse prescription medications. As the sample included so few participants reporting drug use, no analysis was completed to explore the effect of drug use on functioning.

Table 34

Participant Reported Alcohol Use on Demographic Questionnaire

Amount of Use	Number of Participants	Percentage
0-2 drinks	29	22.0%
3-5 drinks	44	34.1%
6-10 drinks	17	13.2%
11-15 drinks	11	8.5%
16-20 drinks	2	2.3%

A MANOVA was conducted to test the hypothesis that engaging in increased drinking behavior would influence the impact of the number of traumatic/ stressful life events on psychological functioning. The dependent variables used in the analysis were BDI-II, STAI-II,

IES-R, and PSS scores. The independent variables were the number of drinks typically consumed when drinking, divided into 3 categories (0-2, 3-5, and 6 or more) and the number of events experienced. As noted in Table 35, the multivariate interaction effect indicated that drinking behavior did not interact with the total number of events to contribute to variance in psychological functioning.

Table 35

Multivariate ANOVA Testing Interaction Effect of Number of Negative Stressful/Traumatic Events Experienced and Drinking Behavior on Depression, Anxiety, Perceived Stress, and Posttraumatic Stress.

	Value	F	Hypothesis df	Error df	Sig.	Eta squared
Wilks Lambda	.77	1.56	16	281	.08	.06

* significant at the p = .05 level, **- significant at the p = .01 level.

Exploratory Hypothesis 5: Total number of traumatic events would be associated with increased academic problems

To evaluate the hypothesis that increased number of traumatic/stressful events would be associated with the reported experience of academic problems (being placed on academic probation or failing a class), a t-test was conducted comparing those that experienced academic problems to participants that denied experiencing difficulties according to the number of traumatic/stressful events that have experienced. Participants reporting having academic problems indicated having experienced increased numbers of traumatic/stressful life events than did those denying academic problems, t (126) = -4.39, p =.000.

CHAPTER IV

DISCUSSION

Overall, results in this study conclude that evidence of neuropsychological deficits in attention, cognitive flexibility, inhibition, working memory, and explicit memory are not found among college students experiencing chronic stress and/or trauma within the current sample. College students do, however, evidence differences in psychological functioning in relation to their exposure to traumatic and stressful events. The current study also indicates that college students do experience considerable histories of traumatic and stressful life experiences, possibly increasing their vulnerability to stress related disorders of depression, anxiety, and PTSD.

Results of this study indicate that experiencing an increased number of negative traumatic and/or stressful life events is associated with significantly increased psychological symptoms of perceived stress, depression, and posttraumatic stress. This is especially true of individuals reportedly experiencing more than six traumatic or stressful life events. This supports theories detailing that stressful life events and trauma have a cumulative effect on contributing to symptoms of stress related disorders of depression and PTSD (McEwen et al., 1996). They also suggest that individuals experiencing increased numbers of stressful events are likely to experience increased stress reactivity, or a current subjective experience of stress. Neuropsychological functioning, however, was not found to differ based upon the number of past events experienced. This is similar to past research, which has suggested that college students do not display impairment in the domains of attention and executive functioning relative to trauma exposure, as is found in clinical populations (Twamley et al., 2004). The current study extends these findings by exploring the influence of trauma on memory performance as well,

concluding that memory impairment is not evident among college students according to the frequency of trauma and stress exposure.

Previous studies have identified trauma occurring in the context of a social situation or relationship (i.e. sexual assault, intimate partner violence, child abuse, etc.) to be more damaging than other types of traumas (i.e. car accidents, serious illness/injury, etc.; DePrince et al., 2009). Results of this study indicate that college students experiencing trauma occurring within the context of a social situation or relationship (i.e. sexual assault, intimate partner violence, child abuse, etc.) have increased symptoms of depression, perceived stress, anxiety, and posttraumatic stress than no trauma controls. While results trended towards those experiencing relationship trauma having higher scores on measures of psychological symptoms than did those reporting non-relationship traumas (i.e. car accidents, serious illness/injury, etc.), they were not significantly different. Individuals reporting experiencing traumas not occurring within the context of a relationship did not differ in scores on measure of psychological symptoms from no trauma controls. This suggests that, among college students, the type of trauma experienced matters in terms of the level of psychological symptoms experienced, with interpersonal trauma history placing them at increased risk for the development of psychological symptoms. Experiencing relationship trauma did not, however, contribute to differences in neuropsychological functioning in the domains of attention, cognitive flexibility, working memory, or explicit memory.

Results of this study indicate that college students experiencing traumatic stress and reporting symptoms of posttraumatic stress to a level that might be considered clinical level on a screening questionnaire for PTSD experience increased symptoms of depression, perceived stress, and anxiety relative to no-trauma controls and when compared to individuals experiencing

trauma, but reporting posttraumatic symptoms below the clinical level for a diagnosis of PTSD. Consistent with previous research among college students, PTSD symptomology among college students in this sample did not contribute to impairments in attention, working memory, executive functioning, or explicit memory (Twamley et al, 2004).

The following will discuss findings of this study in greater detail, including potential mediating and moderating factors. Limitations and suggestions for future research will also be discussed.

Prevalence of Trauma among College Students

Previous research suggests that exposure to potentially traumatic events is not uncommon among college students, with an estimated 66-84% of students being exposed to at least one event (Read, Ouimette, White, & Colder, 2011; Bernat et al., 1998: Vrana et al., 1994). Definitions of trauma or stressors have varied across studies. Within the current study, two measures of stressful events were included. The SLESQ measures what would be more consistent with Criterion A events in the DSM-IV TR diagnostic category for PTSD and includes potentially traumatic events. The LES, however, measures more common life stressors that are stressful, but not necessarily traumatic. Consistent with previous research, 66% of participants in the current study reported experiencing at least one event on the SLESQ, a traumatic event. Previous research notes that the most common events provided by students were life threatening illness, death of a loved one, and being involved in an accident. Participants in the current study reported that death of a loved one, emotional abuse, and life threatening accident were the most commonly experienced traumatic events. While this is somewhat consistent with previous literature, it appears that the current sample included more individuals reporting having

experienced emotional abuse than previous samples. The overall number of events reported is consistent with other studies, suggesting that the current sample is representative of previous work.

Effects of Number of Lifetime Stressful and Traumatic Events on Neuropsychological and Psychological Functioning

The current study defined the cumulative effects of negative stressful events and trauma using both a measure of events more typical to everyday life (LES) and a measure more specific to trauma (SLESQ). The number of events experienced on both measures were combined to operationally define the number of negative stressful and/or traumatic events experienced. Previous research has studied stressful life events using either a traumatic experience checklist or a measure of everyday stressors. As both of these types of events have been linked with effects in neuropsychological and psychological functioning, combining these factors allows for a more complete understanding of the cumulative effect of experiencing numerous stressful life events. Chronic stress and stress related disorders are linked to the abnormal activation of the stress response system, producing structural and neurochemical changes which result in functional deficits. Experiencing frequent stressors sensitizes individuals to the detrimental effects of future stressful events. As such, the current study contributes to the understanding of the influence of stressful events on college students by including a broader base of potentially stressful events. In addition, the current study explores both neuropsychological and psychological functioning, expanding the understanding of the potential functional implications relevant to stress and trauma to include a comparison of mental health and cognitive domains.

Physiologically, the stress response has developed in a manner that provides an adaptive and protective function when implemented in response to short lived stressful events of mild to moderate intensity (Sapolsky, 2007). Through the mechanism of allostasis, the body is able to respond to these stressors efficiently and effectively by utilizing systemic responses that promote adaptive coping strategies and responses (McEwen, 2002). When individuals are exposed to stressful or traumatic events repeatedly, increased risk for damage to the brain and body occurs through the theoretical components of allostatic load, which result in recurrent activation of the stress response system (McEwen, 2004). Recurrent exposure to increased glucocorticoids, 5-HT, DA, and NE, facilitated by the repeated activation of the SAM and HPA axes, results in structural and functional changes that contributes to impairment in functioning and the development of stress related disorders (Contrada & Baum, 2010). Areas of the brain particularly vulnerable to the effects of repeated activation of the stress response system are the hippocampus (Virgin et al, 1991; Sapolsky et al., 1985; Kerr et al, 1991; Conrad et al., 2004, 2008; Schloesser et al., 2009), medial prefrontal cortex (Cook et al., 2004; Radley et al., 2004, Liston et al., 2006, Jett et al., 2013), dorsolateral prefrontal cortex (Evans et al., 2009; Kim et al., 2013), amygdala (Onur et al., 2009), and the hypothalamus (Contrada et al., 2010) .

Neuropsychological Functioning

Previous research has generally focused on the influence of specific traumas and life events on the effects of neuropsychological functioning of individuals, and a majority of those studies have been conducted using clinical populations. Within these studies, it has been suggested that trauma and chronic or perceived stress is likely to have a unique contribution to executive functioning difficulties (El-Hage et al., 2006; Stein et al., 2002; Ohman et al., 2007; Majer et al., 2010; Evans et al., 2009; Nixon et al., 2004). Among non-clinical populations, brain

imaging studies suggest that report of greater numbers of negative lifetime events has been associated with reduced volume within the ventromedial prefrontal cortex and anterior cingulate (Ansell et al., 2012), a key area in the regulation of the stress response through innervation with the amygdala (Williams et al., 2006; Cohen et al., 2000), as well as regulation of emotional processing and integrating information from the periphery to guide decision making (Dunn et al., 2006). The experience of childhood sexual abuse has been associated with decreased response inhibition ability within a community sample (Navalta et al., 2006). Among healthy individuals experiencing chronic life stress, reduced hippocampal volume has been associated with the number of events experienced among women (Gianaros, et al., 2002). Effects of chronic stress or cumulative stressful experiences do not consistently indicate reductions in volume within the hippocampus for individuals within non-clinical populations, suggesting that this may be a consequence of psychiatric disorders or a possible pre-disposing factor for the development of clinical level symptomology (Ansell et al. 2012). Ongoing brain development and maturation, associated with neuroplasticity, may also explain the inconsistencies within the literature and suggest a possibility that damage resulting from early stress and trauma may be repaired through neurogenesis (Fumagalli, Molteni, Racagni, & Riva, 2007).

Few studies relating to the experience of chronic stress or traumatic events have been conducted using college student populations and those that have been completed typically focus on the experience of specific events. The experience of increased life stressors also has been associated with decreased working memory performance in college students (Klein et al., 2006; Wilding et al., 2007), although these studies have been completed with small sample sizes and did not also look at the history of traumatic events. Twamley and colleagues (2004) completed a comprehensive neuropsychological battery with college students and examined the influence of

the number of lifetime traumas as reported on a checklist for events that would meet the DSM-IV criteria for a trauma on executive functioning. Results indicated no relationship between the number of traumatic events reported and executive functioning or working memory. They did not measure explicit memory performance. Other studies have suggested that current levels of perceived stress have been associated with decreased set shifting ability among college students, suggesting that current stress levels and the number of current stressors experienced contribute to executive functioning difficulties (Orem, 2008). Current stress levels have been associated with decreased working memory performance within college females and reduced activation of the dorsolateral PFC, as measured by the N-back test (Qin et al., 2009). This suggests that working memory performance among college students may be influenced by current perceived stress levels, even if they are not influenced by historical events.

The current study did not find differences in working memory, explicit memory, executive functioning, or cognitive flexibility and inhibition in college students as a function of the number of past stressors and traumatic events experienced. While these effects have not been explored extensively in past literature, one study looking at the impact of the number of traumatic events experienced on neuropsychological functioning produced similar results and suggested that college students may be somewhat resilient to the effects of trauma documented among clinical populations (Twamley et al., 2004). The current study extended that study by additionally measuring explicit memory, as that has been an area of deficit documented among clinical populations. No effects of trauma and stress on explicit memory functioning were found in this study. Previous studies finding effects relating to stress on neuropsychological functioning utilized a measure of perceived stress to operationally define chronic stress, rather than utilizing the lifetime number of events experienced. College students may represent a

resilient population. As some of the traumatic and stressful events may have occurred when students were younger, it is also a possibility that the effects of these events had been corrected through neuroplasticity. Neuroplasticity refers to the growth and development of new axon branches and synapses, which can be enhanced through environmental enrichments and positive experiences (Kolb, Mychasiuk, Muhammad, & Gibb, 2013). College students may have experienced factors, such as positive peer relationships, positive parent-child relationships, experiences encouraging learning, that may have promoted brain development that helped repair damage from stress exposure (Kolb et al., 2013).

Alternatively, it is also possible that the standard measures of working memory, explicit memory, executive functioning, and set shifting are not sensitive enough to pick up on subtle differences in performance within this population. In a recent study by Yang and colleagues (2013), the use of both neuropsychological measures of executive functioning and rating scales relating to the self-perceived level of behavioral symptoms indicating executive functioning (Behavior Rating Inventory of Executive Function-Adult Version, BRIEF-A) were included in one study to compare the influence of polyvictimization on functioning. While no significant differences emerged among college students on neuropsychological tests relating to their trauma status, participants reported significantly greater difficulties relating to executive functioning on the BRIEF-A. This indicates that college students may demonstrate behavioral deficits in functioning relating to their abilities to inhibit responses, think flexibly, engage in problem solving, and make decisions, but that these difficulties may be too subtle to be detected on standard neuropsychological measures.

Psychological Functioning

Studies relating to the influence of cumulative stress and trauma on functioning in college students have also focused on psychological symptoms, college adjustment, academic performance, and retention. Vrana and Lauterbach (1994) measured the number of traumatic events reported by college students and compared this with reports of psychological symptoms. Students reporting increased number of events reported increased symptoms of depression, trait anxiety, and posttraumatic stress than did no trauma controls. The cumulative effect of lifetime trauma and stress has also been measured within the social-emotional adjustment of college students (Banyard & Cantor, 2004) and in symptoms of depression (Turner & Butler, 2003).

The current study did find moderate to large effects of the number of lifetime events experienced on measures of psychological functioning, including perceived stress, depression, and posttraumatic stress. A linear trend in psychological measure scores emerged where increased stressful events experienced were associated with increased depression, perceived stress, and posttraumatic symptoms. An increase in anxiety was also noted among individuals reporting increased events, but this was only true of trait anxiety, as the combined score using the STAI-II did not reveal a significant result, but did trend in the expected direction. Previous studies have used this measure and have utilized the trait anxiety subscale to examine the impact of traumatic events (Vrana et al., 1994). This would be consistent with the chronic stress and trauma literature, which describes accumulative events as producing increased reactivity and anxiety as a trait of an individual's personality. As a trend also emerged for higher levels of current perceived stress among those experiencing 6 or more events, a measure correlated with stress response reactivity, these results suggest that college students who have experienced several traumatic and/or stressful life events throughout their lifetime experience higher levels of

stress compared to other college students, and are likely to have a more anxious approach to their world view then are those that experience minimal stressful life experiences. They are also more likely to experience symptoms of depression and are likely to be more vulnerable to the development of PTSD or to present with current symptoms of posttraumatic stress.

The current study contributed to a growing body of literature relating to understanding stress among college students that suggests that stressful life experiences that do not meet the DSM-IV TR criteria of a trauma also produce changes in mental health functioning (Anders, Frazier, & Shallcross, 2012). This was accomplished by including both traumatic and stressful life events on measures. This indicates that negative life events, when occurring accumulatively, also produce increased depression and posttraumatic stress. Recent studies also identify factors associated with stress reactivity at the genetic level, BDNF and 5-HT gene variants, as moderating the influence of stressful and traumatic life experiences in the development of depression symptoms of negativity among college students, and supports that the experience of current and personal stress is particularly related to mental health symptoms (Perea, Paternina, Gomez, & Lattig, 2012). This indicates that stressful life events are meaningful to include in an understanding of the accumulative effects of stress and are likely linked to differences within the stress response system.

Effects of Interpersonal Trauma on Neuropsychological and Psychological Functioning

Traumatic stressors have been defined as events that betray an individual's world view and expectations about the behaviors of others (van der Kolk et all., 1996). While a consistent, complete definition of traumatic stress has yet to be agreed upon, research has focused on numerous types of traumatic experience, including those that occur within the context of

interpersonal attachment relationships (e.g. abandonment, death, divorce, affairs, etc.) and those that affect an individual's personal identity (physical and sexual abuse, intimate partner violence, sexual assault, etc.; Kira, 2001). As humans are social beings, they create ongoing value systems which guide beliefs about the self and the self in relation to others, which are undermined during the experience of traumatic stressors that occur within the context of an interpersonal relationship (Kira, 2001; van der Kolk et al., 1996). As such, traumatic stressors occurring within a personal relationship context are shown to be associated with increased difficulties compared with other traumatic or stressful events across the lifespan. Previous research with children experiencing potentially traumatic events suggests that events that occur within the context of family relationships, like child physical and sexual abuse, are particularly distressing and related to long term deficits in executive functioning, including working memory, inhibition, attention, and processing speed (DePrince et al., 2009). These events produced greater effects on neuropsychological functioning than did non- familial types of trauma. Research supports the influence of child physical, sexual, and emotional abuse upon the development of abnormal stress responding, reduced memory, attention, and inhibition abilities, as well as an increase of psychological symptoms of PTSD, depression, and anxiety (Glaser, 2000; Watts-English et al., 2006; Miskovic et al., 2010; Majer et al., 2010). Several studies have found that, relative to children experiencing non-familial or relationship traumas, children experiencing physical, sexual, or emotional abuse indicate an increased risk for neuropsychological and psychological functioning difficulties (Cromheeke, Herpoel, & Mueller, 2013; Fishbein, Warner, Krebs, Travarthen, Flannery et al., 2009). Studies examining the effects of accumulative trauma throughout the lifespan have identified a more significant impact of personal, relationship oriented, stressors and traumas compared to other life stressors on symptoms of depression,

quality of life ratings, and loneliness within elderly populations (Keinan, Shrira, & Shmotkin, 2012; Palgi, Shrira, Ben-Ezra, Shiovitz-Ezra, & Ayalon, 2012).

Among college students, experiencing traumas occurring within the context of interpersonal relationships have been shown to be increasingly distressing compared with non-relationship traumas and/or life stressors. Studies comparing the influence of relationship context trauma compared with other types of traumas have focused on betrayal or attachment trauma theory, which posit that events that involve the undermining of trust and attachment within an interpersonal relationship are particularly damaging (Freyd, 1996). College students who experienced interpersonal trauma have been shown to report increased PTSD and mental health symptoms than those experiencing other types of trauma (Hetzel-Riggin & Roby, 2013; Lancaster, Melka, & Rodriguez, 2009). Interpersonal trauma is particularly likely to contribute to posttraumatic difficulties among college students, compared with students with no trauma history or who have experienced non-interpersonal types of trauma as measured by the SLESQ (Ford, Stockton, Kaltman, & Green, 2006; Krupnick et al., 2004). Sexual abuse and assault has been shown to be increasingly associated with depression, anxiety, and PTSD symptoms when compared to other types of trauma among college students (Vrana et al., 1994; Krupnick, et al., 2004).

The current study contributed to the literature in that it explored both neuropsychological and psychological functioning among college students experiencing interpersonal versus non-interpersonal types of traumas, and compared to no trauma controls. Research with the college student population has previously focused on the influence of interpersonal trauma on psychological functioning, school achievement, and life satisfaction. Experiencing interpersonal trauma was not associated with variance in executive functioning, memory, or cognitive

flexibility and a small effect size was noted. This was a contrast to the large effect size observed on measures of depression, perceived stress, and posttraumatic stress. It may be that students vary according to the type of trauma experienced, but that the effects may be more subtle than can be observed on the measures provided within this study.

Given the contrast of effects found for neuropsychological and mental health functioning, it may be that the effects of past interpersonal trauma may be related to differences in alternative executive functioning tasks not measured within this study, like emotional regulation. Studies among college students have revealed that emotional regulation difficulties have been associated with increased mental health symptoms, re-victimization, and increased drinking behavior among those experiencing sexual assault (Messman-Moore, Ward, & Zerubavel, 2013). Emotional regulation difficulties have been shown to be associated with mental health symptomology (Bradley et al., 2011). As such, it is possible that interpersonal trauma influences executive functioning abilities that require the use of emotional regulation and measuring executive functions while processing emotional content may uncover differences in functioning among the college student population.

Students making it into college or those choosing to pursue a degree may have pre-existing resiliency factors that have helped them to succeed in the past and may help to buffer them from the negative effects of interpersonal trauma on neuropsychological functioning. Also, it is possible that they have experienced environmental supports or positive relationships and experiences that have helped to encourage neuroplasticity relating to cognitive functioning following trauma. The contrasting large effect size in mental health symptoms suggests that emotional processing differences would be advantageous to explore within future studies.

Participants who reported experiencing interpersonal trauma within the current study (sexual assault, molestation, physical abuse as a child or adult, or emotional abuse) reported higher levels of perceived stress, depression, and posttraumatic stress than did no trauma controls. Differences were not found, however, among individuals who reported interpersonal and non-interpersonal types of traumas or among individuals experiencing non-interpersonal types of trauma and no trauma controls. Within the current study, it appears that those experiencing non-interpersonal types of trauma are within a middle range on measures of perceived stress, depression, and posttraumatic symptoms, between scores of no trauma controls and those experiencing interpersonal trauma. Consistent with previous research, this suggests that interpersonal trauma is particularly detrimental to the emotional well-being of college students (Lancaster et al., 2009; Ford et al., 2006). This suggests that the type of trauma experienced is an important factor to consider among college students, and may contribute to increased difficulties with symptoms of depression, stress, and anxiety specific to past traumas. This is consistent with trauma literature relating to betrayal trauma and attachment based trauma theories, which posit that traumatic events occurring within the context of a relationship with another person are likely to undermine the sense of connection and security a victim has in future interpersonal relationships, making them vulnerable to future negative outcomes.

Effects of PTSD Status on Neuropsychological and Psychological Functioning

Previous research has found abnormalities within the stress response system in individuals diagnosed with PTSD, as indicated by levels of neurotransmitters and hormones circulating in the body and produced through the HPA Axis. Measures of cortisol levels within those diagnosed with PTSD have produced mixed results, with studies reporting increased levels (Pitman et al., 1990; Bremner et al., 1997; Lemieux et al., 1995; De Bellis et al., 1999) and

others reporting decreased levels (Mason, 1986; Yehuda et al., 1990, 1995; Trestman et al., 1996). Individuals with PTSD have also been suggested to have increased reactivity of the HPA Axis in response to future stressors (Elzinga et al., 2003). Review of the literature suggests that cortisol levels could be discrepant due to varying lengths of time from the trauma or the type of trauma experienced (Miller et al., 2007). Increased norepinephrine among those with PTSD following exposure to stressors has been shown upon exposure to emotionally laden images and has been suggested to play a role in increased memory for aspects of trauma (Southwick, 2010; Onur et al., 2009; Friedman et al., 2010). Levels of serotonin have also been shown to be elevated among those with PTSD (Morgan et al., 2003; Krystal et al., 2009), contributing to decreased inhibition of the amygdala (Neumaier et al.,2002). Reduced activity and volume within the vmPFC among those with PTSD further reduces the inhibition of the amygdala, which results in increased activation of the HPA Axis (Koenigs et al., 2009). Similar to literature relating to chronic stress and allostatic load, the repeated activation of the HPA Axis and increased levels of neurohormones in the nervous system contributes to structural and functional damage among those diagnosed with PTSD.

Within the trauma research literature, the role of PTSD diagnosis in contributing to neuropsychological and psychological deficits in functioning has been debated, with some studies noting that the diagnosis of PTSD contributes to deficits in functioning and others noting that the deficits found in those with PTSD represent pre-existing vulnerability factors (Gilbertson et al., 2006). Individuals diagnosed with PTSD display increased abnormalities in neurological activity when engaging in a selective attention and inhibition task compared with no trauma controls and when compared with those exposed to trauma, but not diagnosed with PTSD (Falconer et al., 2008). In addition, reduced performance on neuropsychological assessment for

selective attention and inhibition was noted among those diagnosed with PTSD and no trauma controls, but no differences in performance were revealed among those exposed to trauma without PTSD (Falconer et al., 2008). Other studies have found deficits in performance in selective attention and inhibition, as measured by Trails B of the Trail Making Test (Beckham et al., 1998; Jenkins et al., 2000; Stein et al., 2002). Those diagnosed with PTSD are also found to have decreased performance in cognitive flexibility, as measured by the WCST (Kanagaratnam et al., 2007; Twamley et al., 2009).

Few studies have been completed exploring the effects of PTSD symptoms on neuropsychological functioning among college students (Leskin et al., 2007; Twamley et al., 2004). Twamley and collegues (2004) measured neuropsychological performance on traditional measures, including the TMT, WCST, FAS test, and Digit Span, among college students. Students were classified into groups based on trauma exposure and level of PTSD symptomology, including a group exposed to trauma and considered to have PTSD levels symptoms, those exposed to trauma without clinical level PTSD symptoms, and a no trauma control group. Participants did not differ in performance on TMT, Digit Span, or the FAS test according to PTSD status. Differences were found among those exposed to trauma, without PTSD and no trauma controls on the WCST score measuring the number of responses until a correct category was completed. Leskin and collegues (2007) also explored the effect of PTSD symptoms on neuropsychological performance among college students. They utilized the TMT and the Amsterdam Neuropsychological Test (ANT) to assess attention and inhibition. While no differences were found in performance on the TMT, the PTSD group performed worse on the ANT test than did a control group of low trauma with no PTSD and those with high numbers of trauma, but no PTSD symptoms. Authors suggest that traditional paper-and-pencil tests, like the

TMT may fail to uncover differences in college students due to an inadequate level of specificity to detect differences, due to the minimal level of variability within that group (Leskin et al., 2007). Also, it was suggested that the lack of differences could be related to the lower severity of clinical symptoms found among the college student population.

The current study expanded upon previous findings by including a broader measure of neuropsychological functioning by including a measurement of memory ability and also including an alternative to a traditional measure of working memory and sustained attention, the N-back test. The additional measurement of memory expanded results as previous PTSD research has shown deficits within the region of the hippocampus and memory functioning. The N-back test is used commonly in neurophysiological studies and has been shown to be associated with functioning in the dorsolateral PFC, an area also shown to be less active among those diagnosed with PTSD. The use of this measure addressed previous suggestions that traditional tests are not shown to be sensitive enough to overcome the limited variability within the college student population. As in previous studies, the current study did not find differences in performance on the TMT, N-back, WCST, or RAVLT-DR tests among no trauma controls, those exposed to trauma without symptoms of PTSD, and those considered to have high levels of PTSD symptoms. There are several possible reasons that effects were not found in the current study. PTSD symptoms are likely less extreme among college students than within a clinical population, which possibly limits the effects on cognitive functioning. The findings confirm previous studies that find no differences in scores on the TMT for the PTSD group. Also, assignment of participants into the PTSD group was made using a self-report measure of PTSD type symptoms. While the measure is shown to have adequate convergent validity with semi-structured interviews for PTSD, it is still not as thorough a diagnosis as may have been

completed in previous clinical population research. This may have allowed for some individuals being placed within the PTSD group that would not be considered to have a clinical diagnosis of PTSD if provided a complete clinical interview, making it more difficult to find differences among groups. Another consideration for the lack of differences in neuropsychological functioning is that college students likely present with higher cognitive abilities, providing further support for research suggesting that deficits in functioning among individuals diagnosed with PTSD represent pre-existing vulnerability factors.

Previous studies have found that PTSD symptoms among college students is associated with higher scores on measures of psychopathology and lower life satisfaction. Students considered to have PTSD symptoms have been found to have a greater number of depression symptoms (Leskin et al., 2007; Twamley et al., 2004). Co-morbid depression is common among individuals diagnosed with PTSD (Brady, Killeen, Brewerton, & Lucerini, 2000). Studies have focused on other negative outcomes relating to diagnosis of PTSD among college students, including increased drug and alcohol use (Read, Colder, Merrill, & Ouimette, 2012), lower grade point averages (Bachrach & Read, 2012), and drop out (Boyraz, Horne, Owens, & Armstrong, 2013).

The current study expands upon previous research by including a more broad assessment of mental health functioning, including depression, anxiety, and perceived stress. The current study supports previous studies finding that diagnosis of PTSD among college students is associated with increased report of depression symptoms. Specifically, those considered to have clinical levels of PTSD symptoms had higher depression scores than did no trauma controls and those with a history of trauma without PTSD level symptoms. A similar pattern of scores was observed for perceived stress, as those in the PTSD group reported higher levels of current stress

than did those within the no trauma control group and those exposed to trauma without PTSD. Trait anxiety was also higher among individuals with PTSD compared to no trauma controls, but did not differ from individuals exposed to trauma without PTSD. This suggests that, among college students, high levels of PTSD symptoms are associated with increased symptoms of depression and perceived stress and these scores are independent of what could be explained by trauma exposure alone. Those experiencing a high number of PTSD symptoms are likely to report an increase in depression, perceived stress, and anxiety proneness. These symptoms may place them at risk for future academic failure, dropout, or substance use.

Effect of Age at Time of Self-reported Traumatic Event on Neuropsychological and Psychological Functioning

Conflicting evidence has been presented related to the role of age at the time of experiencing traumatic events and deficits to neurocognitive and psychological outcomes. One hypothesis relating to the effects of age at time of trauma is that early stress exposure undermines neural development, impairing neurological functioning and putting individuals at risk for psychological disorders. Another hypothesis would be that experiencing trauma at earlier ages would be inconsistently linked to neuropsychological and psychological functioning in adulthood due to the dynamic process of neuroplasticity. Several studies have described neurobiological changes in structure and function following exposure to early childhood trauma (Champagne, 2010; Anda, Felitti, Bremner, Walker, & Whitfield, 2006; Watts-English et al., 2006; Glasser, 2000). Childhood trauma is associated with increased mental health symptoms in adulthood (Chapman et al., 2004). Cumulative childhood trauma, as compared to cumulative adulthood trauma, is found to have more substantial influence on the development of PTSD symptoms, suggesting that early trauma may make individuals vulnerable upon exposure to future stressors

(Cloitre et al., 2009). One of the mechanisms proposed to explain the influence of childhood stressors on adulthood outcomes is through stress sensitization, by which early childhood stress contributes to abnormalities in HPA axis functioning that persist throughout development and contribute to vulnerability to damage from future exposure to stressors by increasing stress reactivity (McLaughlin, Conron, Koenen, & Gilman, 2010). Another is through epigenetics, in which environmental experiences transcribe upon DNA to make lasting effects genetically (Champagne, 2010). Others have suggested that periods of development are sensitive to the development of certain functions and that, trauma or stress occurring during these times will contribute to long term deficits (Watts-English, et al., 2006). For executive functioning, this developmentally sensitive period is between ages 7-16 (Watts-English, et al., 2006). Ogle and collegues (2013) explored life satisfaction and PTSD symptoms among an elderly population and found that individuals experiencing childhood traumas were more likely to experience depression, low life satisfaction, and increased PTSD symptoms than were individuals exposed to trauma at other times in their lives.

Given the strong research background of early childhood trauma on neuropsychological and psychological functioning, the current study hypothesized that younger age at the time of the trauma event provided on the IES-R would be associated with increased symptoms of psychological difficulties and decreased neuropsychological performance. This was not the trend that emerged, however, as the only effect for age was related to posttraumatic stress symptomology, and it reached only marginal significance once alpha adjusted critical values were utilized to control for increased error rate. The trend in PTSD symptoms was that events occurring after age 18 were associated with increased PTSD symptoms than were those occurring between the ages of 5-9 years. Research also suggests that more recent traumatic

events are likely to be associated with increased distress scores than would those from earlier in childhood among college students. Specifically, events occurring after the age of 18 years (recent events) have been shown to be associated with increased PTSD scores among college students (Leskin et al., 2007). Given the nature of posttraumatic symptomology, it is possible that those who listed a more recent traumatic event were still within the acute stress phase and potentially likely to reduce their reported symptoms as time remitted. It is also possible that, due to the recency of trauma exposure, the cognitive effects relating to stress exposure may not have yet emerged, but may in the future. Students reporting events for earlier in childhood may have rated their symptoms as less severe due to them not being as salient. Research regarding neuroplasticity and epigenetics also discusses the role of enriching environments in assisting with reducing damaging effects of HPA system activation following stress (Champagne, 2010). These mechanisms restore functioning and are described as an intersection between genetics and the environment (Champagne, 2010). Changes in the expression of genes can occur through stimulation from the environment (Champagne, 2010). It is possible that college students have previous environmental strengths and enrichments that have helped them to be less damaged by the effects of early childhood trauma. Individuals making it into college likely have increased supports and resources that have helped them to gain admittance into school and to set the goal for themselves to obtain a degree, potentially beyond what would be anticipated within a clinical or community based sample.

The lack of effects found for age on neuropsychological and psychological factors could also be related to the categorization assigned to the age groups. The groups were assigned based on frequencies that would allow for analysis, paired as best to developmental stages as possible. Limited representation in childhood age ranges may undermine the representativeness of the

results to other samples that include a larger percentage of individuals endorsing traumatic events at a younger age range. The current study expands upon previous research among college students with trauma histories by exploring the effect of age in greater detail, however, further exploration of these effects and possible relationships to resiliency factors that may have promoted neurogenesis and/or neuroplasticity would be recommended for future research.

Factors Potentially Contributing to or Protecting from Influence of Trauma and Stress on Neuropsychological and Psychological Functioning

Subjective Distress

Previous research suggests that the subjective experience of distress relating to a traumatic event contributes to higher chance of negative outcome and psychological symptoms (O'Hare & Shrerrer, 2013). Those who report increased subjective distress relating to a traumatic event are found to be increasingly vulnerable to the development of PTSD symptoms than are those reporting less distress (Pineles et al., 2013; Sugar & Ford, 2012; Frazier et al., 2011). As previous studies with college students exposed to potentially traumatic events have suggested that they may represent a resilient group to the effects of trauma, it is possible that one of the mediating or moderating factors between experiencing trauma and the development of mental health symptoms is related to the level of subjective distress reported by college students about the event. To this author's knowledge, no other study has explored the role of subjective distress in explaining mental health symptoms among college students while comparing the number of traumatic events experienced. As such, the results of this study contributed to the literature through a better understanding of the role of subjective distress relating to traumatic events on the development of mental health symptoms among college students.

In the current study, subjective distress relating to potentially traumatic experiences did not contribute to differences in scores on depression, anxiety, or posttraumatic stress. Previous research finding minimal effects of trauma exposure on functioning have suggested that one of the possible reasons for the lack of findings could be that the students that have been exposed to trauma and have made it into college possibly are less likely to be distressed by their past experiences (Twamley et al., 2004). The current study provides support for this, as students indicate that past traumatic experiences influence them to only a mild-moderate degree. An additional question relating to how distressing the trauma was to the individual was added to all of the events on the SLESQ, using a 5 point scale. The mode response among this sample was 3 out of 5. Twenty nine percent of participants indicated an average subjective distress of 1 or 2 out of 5. This may speak to a potential resiliency factor among college students relating to their perceived level of distress relating to the traumas they have experienced.

Frequency and Duration

Repeated traumatic events over a longer period of time are more likely to result in negative mental health consequences, like depression and PTSD, than are those occurring less frequently and for shorter periods of time (Eshelman & Levendosky, 2012). This is likely a consequence of greater abnormality in cortisol levels among those with increased trauma duration and frequency (Bevans, Cerbone, & Overstreet, 2008). Exploration of the influence of frequency and duration of traumatic stressors on subsequent functioning has not been completed among college students, making examination of these factors within the current study an important contribution to the understanding of trauma within this population. As such, the current study utilized the SLESQ, which includes additional questions relating to the duration and frequency of traumatic events.

Results of the current study suggest that the number of potentially traumatic events reported on the SLESQ significantly predicted posttraumatic stress symptoms, but that these effects were partially mediated by the frequency and duration of these events. This suggests that trauma exposure alone among college students is not able to predict posttraumatic stress scores as the development of symptoms is partially dependent upon the frequency and duration of the event. Frequency and duration did not, however, further explain differences in depression and anxiety scores after controlling for the total number of events experienced. This indicates that frequency and duration of traumatic events most influences PTSD symptomology among college students. This may suggest that college students may struggle to process and cope with traumatic events which have occurred frequently or are of longer duration, leading to increased PTSD symptoms being reported.

Social support and religious involvement

Social support has been found to provide a buffering effect to those exposed to potentially traumatic events against the development of PTSD, depression, and anxiety (Evans, Steel, & DiLillo, 2013; Etter, Gauthier, McDade-Montez, Cloitre, & Carlson, 2013; Grasso et al., 2012). Perceived social support is shown to be associated with reduced symptoms following the experience of childhood trauma (Evans et al., 2013). This is suggested to be explained by reducing low positive affect, which is shown to be a cognitive and attentional contributing factor to depression, anxiety, and posttraumatic stress (Etter et al., 2013). Among college students, those who report low perceived social support are found to have an increased severity of PTSD symptoms (Grasso et al., 2012).

Religious involvement and coping following trauma has also been associated with a protective effect from negative mental health outcomes (Bryant-Davis & Wong, 2013; Tran, Kuhn, Walser, & Drescher, 2012). Individuals coping with traumatic events by focusing on spirituality and religion are shown to be associated with decreased symptoms of depression and anxiety (Bryant-Davis et al., 2013). The current study explored the role of community involvement and religious affiliation as potential mediating or moderating influences on trauma exposure in explaining mental health symptoms. Neither factor was found to influence functioning of college students in this sample. Measurement of each factor included only one question, making it possible that the variables were not defined in a manner that would uncover effects.

Limitations

The results of the current study indicate strong relationships between the experience of stress and trauma on psychological functioning, but reveal no differences among neuropsychological performance. Few studies have explored neurocognitive consequences of trauma and stress within the college student population and those that have used specific trauma types, like rape, child abuse, or intimate partner violence. When a large sample of college students experiencing a range of different traumatic events are studied, little difference emerges in neuropsychological performance relative to the number of traumatic events experienced or the presence of PTSD symptoms (Twamley et al., 2004). One of the possibilities in explaining effects found in some studies are the limited sample sizes used when measuring more specific types of trauma. It is possible that students have pre-existing strengths that have helped them to become enrolled within an academic institution. Academic achievement may also serve as a protective factor for those experiencing childhood trauma, offering them a chance to establish

positive relationships with others that may encourage resiliency. Also, those included in the sample were able to adequately schedule their participation and to follow through with their appointment. Several students did not complete their participation, despite reminder phone calls and emails. It is possible that the students within the sample were a subset of college students that were able to successfully manage their numerous responsibilities. As this would be a potential indication of having organizational and planning skills, it is possible that the students with deficits in these areas were unable to complete participation. These same strengths may help to buffer them from the effects of trauma and stress found in clinical populations in cognitive functioning. The current study does not allow for the identification of these strengths beyond screening questions relating to community involvement and religious affiliation, meant to measure engagement within the community and social support. It is possible that the questions used to measure these factors were not adequate in measuring these variables. As such, a limitation of the current study is the limited understanding of factors that might help protect students from the effects found in clinical studies.

The measures of neurocognitive functioning used in the current study may not have been sensitive enough to subtle difficulties and differences in performance that may have been evidenced relative to trauma or stress exposure. Attention within the literature has begun to focus on the ecological validity of neuropsychological tests, developed to detect gross differences in functioning, in identifying deficits in everyday behavior (Chaytor & Schmitter-Edgecombe, 2003). As previously stated, a recent study has found that students reporting trauma histories did not evidence differences in paper and pencil tests, but did on self-report measures of their cognitive abilities. The current study did not include self-report of more behavioral indicators of difficulties in functioning, such as grades, study habits, etc. As such, important differences that

might help to guide intervention and prevention strategies within this population may have been overlooked by the current neuropsychological measures utilized. An exploratory analysis indicates that those reporting more trauma and stress are more likely to report academic problems, suggesting that the use of more behavioral or functional measures of cognitive difficulties may be important to include in future studies. Alternatively, the use of self-report measures of neurocognitive functioning, like the BRIEF or the Frontal Systems Behavior Scale (FrSBe), would likely allow for the identification of difficulties related to stress exposure. As individuals experiencing difficulties in effectively utilizing executive functioning often display reduced self-awareness, the inclusion of caregiver or other self-report about the individuals functioning may aide in further determining these effects in future studies.

The use of self-report surveys to measure trauma exposure and psychological symptoms and functioning is another limitation within the current study. A brief screening measure relating to PTSD symptomology was utilized to operationally define and categorize participants into groups according to their frequency and intensity of reported PTSD symptoms. As a clinical interview was not utilized, the representation of the PTSD group within this study was likely limited and may not be representative of individuals diagnosed within a clinical setting. For the purpose of this study, a broad look at PTSD symptomology among college students was chosen to better accommodate the likely less severe and persistent presentation. Generalizing beyond the scope of a college student population is likely to be questionable. The strength of relationship found among trauma and stress exposure and PTSD symptomology suggests, however, that further study using more stringent diagnostic criteria would likely be beneficial in further exploring the results of this study. Also, as students were asked to recall their past histories of exposure to trauma and stressors, the results are limited to the accuracy of their ability to recall

information accurately. It is possible that the report of trauma may have been skewed by levels of stress the individual was currently experiencing. As such, individuals who were feeling stressed may have been more likely to recall traumatic experiences and stressful events than were others who were feeling less stressed (Pachana, Brilleman, & Dobson, 2011).

In this study and in others exploring the influence of trauma and stress on neuropsychological functioning of college students, it has been suggested that they are a resilient population. While the current study aimed primarily to further add to the literature within this area, limited inclusion of resiliency factors to explore what may make this population less likely to evidence impairment limits the unique contribution of this study. Possible factors identified in exploratory analyses (social support, religious affiliation, mental health treatment) were explored through the use of brief questions on the demographic questionnaire and may not have operationally defined the factors of resilience optimally. Further investigation through the use of standardized measures would likely be advantageous to contribute to the understanding of resilience among college students. Also, small sample sizes in some variables, like drug use and past mental health treatment, limit the confidence in results with these variables.

Conclusions, Implications, and Recommendations for Future Research

The current study contributed to the literature of the effects of traumatic and stressful life events among college students by updating, expanding, and supporting a previous study of neuropsychological functioning relating to trauma and PTSD (Twamley et al., 2004). It expanded upon that study by including a measure of memory function and by including a working memory task used within neuropsychiological research and found to be activated in brain networks facilitating working memory, the N-back test. While no differences in

performance were noted in domains of attention, working memory, executive functioning, or memory, increased mental health symptoms of depression, trait anxiety, posttraumatic stress, and perceived stress were strongly related to experiencing increased numbers of stressful life or potentially traumatic events. It should be noted, however, that while these effects were statistically significant, the severity of mental health symptoms remained low within this sample, suggesting that there may be limitations to the practical significance of these effects. This study was unique in that it explored both cognitive and psychological domains. Research is now suggesting that college students may perform adequately on basic neuropsychological measures, but that they report increased cognitive and learning difficulties on self-report measures of their neurocognitive functional abilities as a function of stress and trauma exposure (Yang et al., 2013). Future research should continue to explore these differences by including more ecologically valid and sensitive measures of neuropsychological abilities, like the BRIEF or FrSBe.

The finding that increased numbers of traumatic and/or stressful life events were associated with increased mental health symptoms suggests that, while they may have cognitive strengths that allow them to complete basic tasks of cognitive functioning adequately, they feel more anxious, are bothered by event specific anxiety, and are less happy and satisfied with their lives. As previous studies suggest that mental health symptoms, particularly those related to PTSD, are associated with decreased retention rates and lower grade performance, these findings are important and speak to the need to develop programs that will assist students in coping with stressful life experiences as well as improve their current stress management skills. Including measures in future research that help to identify how past traumatic events influences current life satisfaction and learning abilities would likely be helpful in guiding these programs. The current

study found that students with increased numbers of stressful events were more likely to indicate that they had academic difficulties than were students with fewer stressful experiences. While this was only measured as a tertiary hypothesis, future research focusing on the academic impact of stress and trauma would likely be helpful. A further understanding of the long term impact of increased stress among college students would be helpful to explore in future research. Many of the participants in this sample were within their freshman or junior year of college. It is possible that the effects of stress and trauma on their functioning would become detectable as they reached higher grade levels, or possibly as they entered into the work force. While they may be able to function adequately to obtain a degree, it may also be that they take longer to do so than other students with less frequency of stressors or trauma.

The current study further explored the influence of PTSD symptoms on neuropsychological and psychological functioning. Findings suggest that PTSD symptoms contribute to levels of current perceived stress and personality traits of anxiety proneness above and beyond what would be explained by trauma alone. This contributes to previous literature relating to understanding if PTSD symptoms contribute to deficits independently of just trauma exposure alone. It also suggests the usefulness of screening students for PTSD symptoms to allow for intervention that might help build adaptive coping strategies prior to the development of academic difficulties and also to help buffer against poor decision making that might result in further trauma exposure. Future studies might benefit from including a broader, more detailed clinical interview of PTSD. The current study utilized a screening measure to allow for the measurement of additional factors. This likely compromised the validity of the diagnostic assumptions made and interpretations about the effects of PTSD should be interpreted cautiously as the influence of PTSD symptoms, rather than the diagnosis of the disorder itself. As the DSM-

5 has recently been released, including diagnosis using the new criteria would be helpful to update the literature.

The extent of trait anxiety and perceived stress among those experiencing increased stressful events suggests that programs on college campuses aimed at assisting students experiencing trauma and stress would likely help to reduce diminished academic performance, although further exploration of possible behavioral or academic difficulties is warranted to better guide development of programs that would target meaningful areas of impairment. Given the high number of traumatic events reported within this study, having trauma related therapy or psychoeducational groups on campuses would likely be beneficial and may allow those experiencing past trauma to develop more adaptive coping strategies that would afford them increased life satisfaction while in college. Future research further focusing on resiliency factors among college students, compared directly to those within clinical settings, would additionally assist in identifying factors that might be targeted in early preventative methods.

Given the strong effect of stress and trauma on emotional functioning, future studies might explore executive functioning relating to emotional regulation, processing, and appraisal. Previous research indicates that the areas of the brain governing these functions are also shown to evidence impairment following exposure to chronic or traumatic stress. The current study found that experiencing interpersonal trauma contributed to increased symptoms of depression, perceived stress, and posttraumatic stress. It is possible that experiencing interpersonal trauma provides deficits to areas of emotional processing and regulation, particularly of an interpersonal nature. Additional research relating to these factors would be beneficial, as it is an overlooked area within the literature.

College students have been considered to be a resilient population to the cognitive effects of trauma and stress observed in clinical and, in some cases, community populations. While this is suggested, a direct comparison of college students with community and clinical populations has yet to be completed. To better understand how college students differ and what strengths they may have that reduce their cognitive deficits relative to trauma and stress, a direct comparison by including these different groups within one study is recommended for future studies within this area.

References

Adamec R., Holmes A., & Blundell J. (2008). Vulnerability to lasting anxiogenic effects of brief exposure to predator stimuli: Sex, serotonin and other factors-relevance to PTSD. *Neuroscience and Biobehavioral Reviews, 32*, 1287-1292.

Airaksinen, E., Larsson, M., Lundberg, I., & Forsell, Y. (2004). Cognitive functions in depressive disorders: Evidence from a population-based study; cognition and depression. *Psychological Medicine, 34*(1), 83-91.

Anda, R. F., Felitti, V. J., Bremner, J. D., Walker, J. D., Whitfield, C., Perry, B. D. . Giles, W. (2006). The enduring effects of abuse and related adverse experiences in childhood: A convergence of evidence from neurobiology and epidemiology. *European Archives of Psychiatry and Clinical Neuroscience, 256*(3), 174-186.

Anders, S. L., Frazier, P. A., & Shallcross, S. L. (2012). Prevalence and effects of life event exposure among undergraduate and community college students. *Journal of Counseling Psychology, 59*(3), 449-457.

Alexander, J. K., Hillier, A., Smith, R. M., Tivarus, M. E., & Beversdorf, D. Q. (2007). Beta-adrenergic modulation of cognitive flexibility during stress. *Journal of Cognitive Neuroscience, 19*(3), 468-478.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders: DSM-IV-TR.* Washington, DC: Author.

Amoss, M.S. & Guillemin, R. (1969). Elevation of plasma LH concentrations induced by LH-releasing factor as measured by radioimmunoassay in the sheep. *Endocrinology*, *64*, 1517-1520.

Anisman, H., Griffiths, J., Matheson, K., Ravindran, A. V., & Merali, Z. (2001). Posttraumatic stress symptoms and salivary cortisol levels. *American Journal of Psychiatry, 158*(9), 1509-1511.

Arnsten, A. F. T. (2009). Stress signaling pathways that impair prefrontal cortex structure and function. *Nature Reviews Neuroscience, 10*(6), 410-422.

Aston-Jones, G., Rajkowski, J., & Cohen, J. (1999). Role of locus coeruleus in attention and behavioral flexibility. *Biological Psychiatry, 46*(9), 1309-1320.

Aupperle, R. L., Melrose, A. J., Stein, M. B., & Paulus, M. P. (2011). Executive function and PTSD: Disengaging from trauma. *Neuropharmacology, 62*(2), 686-694.

Austin, M., Mitchell, P., & Goodwin, G. M. (2001). Cognitive deficits in depression: Possible implications for functional neuropathology. *British Journal of Psychiatry, 178*, 200-206.

Avitsur, R., Stark, J. L., & Sheridan, J. F. (2001). Social stress induces glucocorticoid resistance in subordinate animals. *Hormones and Behavior, 39*(4), 247-257.

Avishai-Eliner, S., Brunson, K.L., Sandman, C.A., & Baram, T.Z. (2002). Stressed-out, or in (utero)? *Trends in Neuroscience, 25*, 518-524.

Bachrach, R. L., & Read, J. P. (2012). The role of posttraumatic stress and problem alcohol involvement in university academic performance. *Journal of Clinical Psychology*, *68*(7), 843-859.

Baddeley, A., & Hitch, G. (1974). Working memory. In G. Bower, *The psychology of learning and motivation: Advances in research and theory* (pp. 47-87). New York, NY: Academic Press.

Baddeley, A. (2003). Working memory: Looking back and looking forward. *Nature Reviews Neuroscience, 4*(10), 829-839.

Banyard, V. L., & Cantor, E. N. (2004). Adjustment to college among trauma survivors: An exploratory study of resilience. *Journal of College Student Development, 45*(2), 207-221.

Barden, N. (2004). Implication of the hypothalamic-pituitary-adrenal axis in the physiopathology of depression. *Journal of Psychiatry & Neuroscience, 29*, 185-193.

Bardgett, M. E., Taylor, G. T., Csernansky, J. G., & Newcomer, J. W. (1994). Chronic corticosterone treatment impairs spontaneous alternation behavior in rats. *Behavioral & Neural Biology, 61*(2), 186-190.

Barrash, J., Tranel, D., & Anderson, S. W. (2001). Acquired personality disturbances associated with bilateral damage to the ventromedial prefrontal region. *Developmental Neuropsychology, 18*(3), 355-381.

Beats, B. C., Sahakian, B. J., & Levy, R. (1996). Cognitive performance in tests sensitive to frontal lobe dysfunction in the elderly depressed. *Psychological Medicine, 26*(3), 591-603.

Bechara, A., Tranel, D., Damasio, H., & Adolphs, R. (1995). Double dissociation of conditioning and declarative knowledge relative to the amygdala and hippocampus in humans. *Science, 269*(5227), 1115-1118.

Beck, A.T., Steer, R.A., & Brown, G.K. (1996). *Manual for the Beck Depression Inventory-II.* San Antonio, TX: Psychological Corporation.

Beckham, J. C., Crawford, A. L., & Feldman, M. E. (1998). Trail making test performance in Vietnam combat veterans with and without posttraumatic stress disorder. *Journal of Traumatic Stress, 11*(4), 811-819.

Berthoz, S., Armony, J. L., Blair, R. J. R., & Dolan, R. J. (2002). An fMRI study of intentional and unintentional (embarrassing) violations of social norms. *Brain: A Journal of Neurology, 125*(8), 1696-1708.

Bevans, K., Cerbone, A., & Overstreet, S. (2008). Relations between recurrent trauma exposure and recent life stress and salivary cortisol among children. *Development and Psychopathology, 20*(1), 257-272.

Botvinick, M. M., Cohen, J. D., & Carter, C. S. (2004). Conflict monitoring and anterior cingulate cortex: An update. *Trends in Cognitive Sciences, 8*(12), 539-546.

Boyraz, G., Horne, S. G., Owens, A. C., & Armstrong, A. P. (2013). Academic achievement and college persistence of african american students with trauma exposure. *Journal of Counseling Psychology, 60*(4), 582-592.

Bradley, B., DeFife, J. A., Guarnaccia, C., Phifer, J., Fani, N., Ressler, K. J., & Westen, D. (2011). Emotion dysregulation and negative affect: Association with psychiatric symptoms. *Journal of Clinical Psychiatry, 72*(5), 685-691.

Brady, K. T., Killeen, T. K., Brewerton, T., & Lucerini, S. (2000). Comorbidity of psychiatric disorders and posttraumatic stress disorder. *Journal of Clinical Psychiatry, 61,* 22-32.

Braver, T.S., Cohen, J.D., Nystrom, L.E., Jonides, J., Smith, E.E., & Noll, D.C. (1997). A parametric study of prefrontal cortex involvement in human working memory. *Neuroimage, 5*, 49-62.

Bremmer, J. D., Narayan, M., Staib, L. H., Southwick, S. M., McGlashan, T., & Charney, D. S. (1999). Neural correlates of memories of childhood sexual abuse in women with and without posttraumatic stress disorder. *American Journal of Psychiatry, 156*(11), 1787-1795.

Bremner, J. D., Licinio, J., Darnell, A., & Krystal, J. H. (1997). Elevated CSF corticotrophin-releasing factor concentrations in posttraumatic stress disorder. *The American Journal of Psychiatry, 154*(5), 624-629.

Bremner, J. D. (2005). Effects of traumatic stress on brain structure and function: Relevance to early responses to trauma. *Journal of Trauma & Dissociation, 6*(2), 51-68.

Bressan, R. A., Quarantini, L. C., Andreoli, S. B., Araújo, C., Breen, G., Guindalini, C. . . . Mari, J. J. (2009). The posttraumatic stress disorder project in Brazil: Neuropsychological, structural and molecular neuroimaging studies in victims of urban violence. *BMC Psychiatry, 9*, 9-30.

Brezun, J. M. J., & Daszuta, A. A. (1999). Depletion in serotonin decreases neurogenesis in the dentate gyrus and the subventricular zone of adult rats. *Neuroscience, 89*(4), 999-1002.

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

Brunson, K. L., Kramár, E., Lin, B., Chen, Y., Colgin, L. L., & Yanagihara, T.K. (2005). Mechanisms of late-onset cognitive decline after early-life stress. *The Journal of Neuroscience, 25*(41), 9328-9338.

Bryant-Davis, T., & Wong, E. C. (2013). Faith to move mountains: Religious coping, spirituality, and interpersonal trauma recovery. *American Psychologist, 68*(8), 675-684.

Buchanan, T. W., Driscoll, D., Mowrer, S. W., Sollers, J. J., Thayer, J. F., Clemens, K., & Tranel, D. (2010). Medial prefrontal cortex damage affects physiological and psychological stress responses differently in men and women. *Psychoneuroendocrinology, 35*, 56-66.

Cannon, W. B. (1932). *The wisdom of the body.* New York, NY: Norton.

Carpenter, L. L., Carvalho, J. P., Tyrka, A. R., Wier, L. M., Mello, A. F., Anderson, G. M., & Price, L. H. (2007). Decreased adrenocorticotropic hormone and cortisol responses to stress

in healthy adults reporting significant childhood maltreatment. *Biological Psychiatry, 62*(10), 1080-1087.

Carretie, L., Hinojosa, J.A., Mercado, F., & Tapia, M. (2005). Cortical response to subjectively unconscious fear. *Neuroimage, 24*, 615-623.

Champagne, F. A. (2010). Early adversity and developmental outcomes: Interaction between genetics, epigenetics, and social experiences across the life span. *Perspectives on Psychological Science, 5*(5), 564-574.

Champagne, D. L., Bagot, R. C., van Hasselt, F., Ramakers, G., Meaney, M. J., de Kloet, E. R., . Krugers, H. (2008). Maternal care and hippocampal plasticity: Evidence for experience-dependent structural plasticity, altered synaptic functioning, and differential responsiveness to glucocorticoids and stress. *The Journal of Neuroscience, 28*(23), 6037-6045.

Chapman, D. P., Whitfield, C. L., Felitti, V. J., Dube, S. R., Edwards, V. J., & Anda, R. F. (2004). Adverse childhood experiences and the risk of depressive disorders in adulthood. *Journal of Affective Disorders, 82*(2), 217-225.

Chaytor, N., & Schmitter-Edgecombe, M. (2003). The ecological validity of neuropsychological tests: A review of the literature on everyday cognitive skills. *Neuropsychology Review, 13*(4), 181-197.

Chrousos G.P. & Gold P.W. (1992). The concepts of stress and stress system disorders: Overview of physical and behavioral homeostasis. *JAMA: The Journal of the American Medical Association, 267*, 1244-1252.

Cloitre, M., Stolbach, B. C., Herman, J. L., van, d. K., Pynoos, R., Wang, J., & Petkova, E. (2009). A developmental approach to complex PTSD: Childhood and adult cumulative trauma as predictors of symptom complexity. *Journal of Traumatic Stress, 22*(5), 399-408.

Cohen, S. S., Kamarck, T. T., & Mermelstein, R. R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24*(4), 385-396.

Cohen, S.S., Perlstein, W.M., Braver, T.S., Nystrom, L.E., Noll, D.C. & Jonides, J. (1997). Temporal dynamics of brain activation during a working memory task. *Nature, 386*, 604-608.

Cohen, S.S., Botvinick, M., & Carter, C.S. (2000). Anterior cingulate and prefrontal cortex: Who's in control? *Nature Neuroscience, 3*, 421-423.

Cohen, S., Kessler, R.C., & Gordon, L.U. (1995). *Measuring stress: A guide for health and social scientists.* New York, NY: Oxford University Press.

Conrad, C. D. C., Jackson, J. L. J., & Wise, L. S. L. (2004). Chronic stress enhances ibotenic acid-induced damage selectively within the hippocampal CA3 region of male, but not female rats. *Neuroscience, 125*(3), 759-767.

Conrad, C. D. (2006). What is the functional significance of chronic stress-induced CA3 dendritic retraction within the hippocampus? *Behavioral and Cognitive Neuroscience Reviews, 5*(1), 41-60.

Conrad, C. D. (2008). Chronic stress-induced hippocampal vulnerability: The glucocorticoid vulnerability hypothesis. *Reviews in the Neurosciences, 19*(6), 395-411.

Contrada, R.J. & Baum, A. (2011). *The handbook of stress science: Biology, psychology, and health.* New York, NY: Springer Publishing Co.

Cook, S.C. & Wellman, C.L. (2004). Chronic stress alters dendritic morphology in rat medial prefrontal cortex. *Journal of neurobiology, 60*, 236-248.

Cooper, C. L. & Dewe, P. (2004). *Stress: A brief history*. Malden: Blackwell Publishing.

Craig, A. D. (2003). Interoception: The sense of the physiological condition of the body. *Current Opinion in Neurobiology, 13*(4), 500-505.

Cromheeke, S., Herpoel, L., & Mueller, S.C. (2013). Childhood abuse is related to working memory impairment for positive emotion in female university students. *Child Maltreatment,* DOI: 10.1177/1077559513511522.

Crowell, T. A., Kieffer, K. M., Siders, C. A., & Vanderploeg, R. D. (2002). Neuropsychological findings in combat-related posttraumatic stress disorder. *Clinical Neuropsychologist, 16*(3), 310-321.

Daniels J. K., McFarlane A. C., Bluhm R. L., Moores K. A., & Clark C. R. (2010). Switching between executive and default mode networks in posttraumatic stress disorder: alterations in functional connectivity. *Journal of Psychiatry & Neuroscience, 35*, 258-266.

Davis, A. M., Penschuck, S, Fritschy, J. M., & McCarthy, M. M. (2000). Developmental switch in the expression of GABA(A) receptor subunits alpha(1) and alpha(2) in the hypothalamus and limbic system of the rat. *Brain Research: Developmental brain research, 119*, 127-138.

Dayas, C. V., Buller, K. M., Crane, J. W., Xu, Y., & Day, T. A. (2001). Stressor categorization: Acute physical and psychological stressor elicit distinctive recruitment patterns in the amygdala and in medullary noradrenergic cell groups. *European Journal of Neuroscience, 14*(7), 1143-1152.

De Bellis, M. D., Keshavan, M. S., & Harenski, K. A. (2001). Anterior cingulate N-acetylaspartate/creatine ratios during clonidine treatment in a maltreated child with posttraumatic stress disorder. *Journal of Child and Adolescent Psychopharmacology, 11*(3), 311-316.

De Bellis, M. D., Baum, A. S., Birmaher, B., Keshavan, M. S., Eccard, C. H., Boring, A. M., . Ryan, N. D. (1999). Developmental traumatology part I: Biological stress systems. *Biological Psychiatry, 45*(10), 1259-1270.

De Groot, J. & Harris, G.W. (1950). Hypothalamic control of the anterior pituitary gland. *Journal of Physiology, 111*, 335-346.

de Kloet, E. R., Joëls, M., & Holsboer, F. (2005). Stress and the brain: From adaptation to disease. *Nature Reviews Neuroscience, 6*(6), 463-475.

de Quervain, Dominique J. -F., Henke, K., Aerni, A., Treyer, V., McGaugh, J. L., Berthold, T., & Hock, C. (2003). Glucocorticoid-induced impairment of declarative memory retrieval is associated with reduced blood flow in the medial temporal lobe. *European Journal of Neuroscience, 17*(6), 1296-1302.

de Quervain, D. J., Roozendaal, B., & McGaugh, J. L. (1998). Stress and glucocorticoids impair retrieval of long-term spatial memory. *Nature, 394*(6695), 787-790.

Delahanty, D. L., Raimonde, A. J., Spoonster, E., & Cullado, M. (2003). Injury severity, prior trauma history, urinary cortisol levels, and acute PTSD in motor vehicle accident victims. *Journal of Anxiety Disorders, 17*(2), 149-164.

Deprince, A. P., Weinzierl, K. M., & Combs, M. D. (2009). Executive function performance and trauma exposure in a community sample of children. *Child Abuse & Neglect, 33*(6), 353-361.

Dere, E., Pause, B. M., & Pietrowsky, R. (2010). Emotion and episodic memory in neuropsychiatric disorders. *Behavioral Brain Research, 215*(2), 162-171.

Diamond, D. M., Bennett, M. C., Fleshner, M., & Rose, G. M. (1992). Inverted-U relationship

between the level of peripheral corticosterone and the magnitude of hippocampal primed burst potentiation. *Hippocampus, 2*, 421-430.

Djavadian, R.L. (2004). Serotonin and neurogenesis in the hippocampal dentate gyrus of adult mammals. *Acta Neurobiologiae Experimentals, 64*, 189-200.

Doan, S. N. & Evans, G. W. (2011). Maternal responsiveness moderates the relationship between allostatic load and working memory. *Development and psychopathology, 23,* 873-880.

Dohrenwend, B. S., & Dohrenwend, B. P. (1974). *A brief historical introduction to research on stressful life events.* Oxford, England: John Wiley & Sons.

Duman, R. S., Heninger, G. R., & Nestler E. J. (1997). A molecular and cellular theory of depression. *Archives of General Psychiatry, 54*, 597-606.

Dunn, B. D., Dalgleish, T., & Lawrence, A. D. (2006). The somatic marker hypothesis: A critical evaluation. *Neuroscience and Biobehavioral Reviews, 30*(2), 239-271.

El-hage, W., Gaillard, P., Isingrini, M., & Belzung, C. (2006). Trauma-related deficits in working memory. *Cognitive Neuropsychiatry, 11*(1), 33-46.

Elliott, R., Sahakian, B. J., McKay, A. P., & Herrod, J. J. (1996). Neuropsychological impairments in unipolar depression: The influence of perceived failure on subsequent performance. *Psychological Medicine, 26*(5), 975-989.

Elsesser, K., Sartory, G., & Tackenberg, A. (2004). Attention, heart rate, and startle response during exposure to trauma-relevant pictures: A comparison of recent trauma victims and patients with posttraumatic stress disorder. *Journal of Abnormal Psychology, 113*(2), 289-301.

El-Sheikh, M., & Harger, J. (2001). Appraisals of marital conflict and children's adjustment, health and physiological reactivity. *Developmental Psychology, 37*(6), 875-885.

Elzinga, B. M., Schmahl, C. G., Vermetten, E., van Dyck, R., & Bremner, J. D. (2003). Higher cortisol levels following exposure to traumatic reminders in abuse-related PTSD. *Neuropsychopharmacology, 28*(9), 1656-1665.

Endo, Y., & Kimura, F. (1996). Impairment of maze learning in rats following long-term glucocorticoid treatments. *Neuroscience Letters, 203*(3), 199-202.

Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science, 196*(4286), 129-136.

Engel, G. L. (1980). The clinical application of the biopsychosocial model. *The American Journal of Psychiatry, 137*(5), 535-544.

Eshelman, L., & Levendosky, A. A. (2012). Dating violence: Mental health consequences based on type of abuse. *Violence and Victims, 27*(2), 215-228.

Etkin, A., & Wager, T. D. (2007). Functional neuroimaging of anxiety: A meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. *American Journal of Psychiatry, 164*(10), 1476-1488.

Etter, D. W., Gauthier, J. R., McDade-Montez, E., Cloitre, M., & Carlson, E. B. (2013). Positive affect, childhood adversity, and psychopathology in psychiatric inpatients. *European Journal of Psychotraumatology, 4.*

Evans, G. W., & Schamberg, M. A. (2009). Childhood poverty, chronic stress, and adult working memory. *Proceedings of the National Academy of Sciences of the United States of America, 106*(16), 6545.

Evans, S. E., Steel, A. L., & DiLillo, D. (2013). Child maltreatment severity and adult trauma symptoms: Does perceived social support play a buffering role? *Child Abuse & Neglect, 37*(11), 934-943.

Falconer, E., Bryant, R., Felmingham, K. L., Kemp, A. H., Gordon, E., Peduto, A., . Williams, L. M. (2008). The neural networks of inhibitory control in posttraumatic stress disorder. *Journal of Psychiatry & Neuroscience : JPN, 33*(5), 413-422.

Ferry, B., Roozendaal, B., & McGaugh, J. L. (1999). Role of norepinephrine in mediating stress hormone regulation of long-term memory storage: A critical involvement of the amygdala. *Biological Psychiatry, 46*(9), 1140-1152.

Fishbein, D., Warner, T., Krebs, C., Travarthen, N., Flannery, B., & Hammond, J. (2009). Differential relationships between personal and community stressors and children's neurocognitive functioning. *Child Maltreatment, 14*, 299-315.

Flinn, M. V., & England, B. G. (1995). Childhood stress and family environment. *Current Anthropology, 36*(5), 854-866.

Foley P., & Kirschbaum C. (2010). Human hypothalamus-pituitary-adrenal axis responses to acute psychosocial stress in laboratory settings. *Neuroscience and biobehavioral reviews, 35*, 91-96.

Ford, J. D., Stockton, P., Kaltman, S., & Green, B. L. (2006). Disorders of extreme stress (DESNOS) symptoms are associated with type and severity of interpersonal trauma exposure in a sample of healthy young women. *Journal of Interpersonal Violence, 21*(11), 1399-1416.

Frazier, P. A., Gavian, M., Hirai, R., Park, C., Tennen, H., Tomich, P., & Tashiro, T. (2011). Prospective predictors of posttraumatic stress disorder symptoms: Direct and mediated relations. *Psychological Trauma: Theory, Research, Practice, and Policy, 3*(1), 27-36.

Fries, E., Hesse, J., Hellhammer, J., & Hellhammer, D. H. (2005). A new view on hypocortisolism. *Psychoneuroendocrinology, 30*(101).

Freyd, J. J. (1996). *Betrayal trauma: The logic of forgetting childhood abuse.* Harvard University Press, Cambridge, MA.

Fumagalli, F., Molteni, R., Racagni, G., & Riva, M. A. (2007). Stress during development: Impact on neuroplasticity and relevance to psychopathology. *Progress in Neurobiology, 81*, 197-217.

Ganzel, B. L., Morris, P. A., & Wethington, E. (2010). Allostasis and the human brain: Integrating models of stress from the social and life sciences. *Psychological Review, 117*(1), 134.

Garcia, R., Vouimba, R. M., Baudry, M., & Thompson, R. E. (1999). The amygdala modulates prefrontal cortex activity relative to conditioned fear. *Nature, 402*, 294-296.

Geary, D. C., & Flinn, M. V. (2002). Sex differences in behavioral and hormonal response to social threat: Commentary on Taylor et al. (2000). *Psychological Review, 109*(4), 745-750.

Gerin, W., & Pickering, T. G. (1995). Association between delayed recovery of blood pressure after acute mental stress and parental history of hypertension. *Journal of Hypertension, 13* (6), 603-610.

Gilbertson, M. W., Shenton, M. E, Ciszewski, A. A., Kasai, K. K., Lasko, N. B., Orr, S. P., & Pitman, R. K. (2002). Smaller hippocampal volume predicts pathologic vulnerability to psychological trauma. *Nature Neuroscience, 5*(11), 1242-1247.

Gilbertson, M. W., Paulus, L. A., Williston, S. K., Gurvits, T. V., Lasko, N. B., Pitman, R. K., & Orr, S. P. (2006). Neurocognitive function in monozygotic twins discordant for combat exposure: Relationship to posttraumatic stress disorder. *Journal of Abnormal Psychology, 115*(3), 484-495.

Gohier, B., Ferracci, L., Surguladze, S. A., Lawrence, E., ElHage, W., Allain, P., . LeGall, D. (2009). Cognitive inhibition and working memory in unipolar depression. *Journal of Affective Disorders, 116*(1-2), 100-105.

Goto Y., Otani S., & Grace A. A. (2007). The Yin and Yang of dopamine release: a new perspective. *Neuropharmacology, 53*, 583-587.

Grasso, D. J., Cohen, L. H., Moser, J. S., Hajcak, G., Foa, E. B., & Simons, R. F. (2012). Seeing the silver lining: Potential benefits of trauma exposure in college students. *Anxiety, Stress & Coping: An International Journal*, *25*(2), 117-136.

Green, B.L. (1990). Defining trauma: Terminology and generic stressor dimensions. *Journal of Applied Social Psychology, 20*, 1632-1642.

Goodman, L. S., Corcoran, C., Turner, K., Yuan, N., & Green, B. L. (1998). Assessing traumatic event exposure: General issues and preliminary findings for the Stressful Life Events Screening Questionnaire. *Journal of Traumatic Stress, 11*, 521-542.

Gross, C.G. (1998). Claude Bernard and the constancy of the internal environment. *The Neuroscientist,4*, 380-385.

Gunnar, M. R., & Donzella, B. (2002). Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology, 27*(1-2), 199-220.

Hamner, M. B., & Diamond, B. I. (1993). Elevated plasma dopamine in posttraumatic stress disorder: A preliminary report. *Biological Psychiatry, 33*(4), 304-306.

Hänsel, A., & von Känel, R. (2008). The ventro-medial prefrontal cortex: A major link between the autonomic nervous system, regulation of emotion, and stress reactivity? *BioPsychoSocial Medicine, 2*, 1751-1759.

Harvey, A. G., Jones, C., & Schmidt, D. A. (2003). Sleep and posttraumatic stress disorder: A review. *Clinical Psychology Review, 23*(3), 377-407.

Herman, J. P., Cullinan, W. E., Morano, M. I., Akil, H., & Watson, S. J. (1995). Contribution of the ventral subiculum to inhibitory regulation of the hypothalamo-pituitary-adrenocortical axis. *Journal of Neuroendocrinology, 7*, 475-482.

Herman, J. P., Figueiredo, H., Mueller, N. K., Ulrich-Lai, Y., Ostrander, M. M., et al. (2003). Central mechanisms of stress integration: Hierarchical circuitry controlling hypothalamo-pituitary-adrenocortical responsiveness. *Frontiers in Neuroendocrinology, 24* (3), 151-180.

Hermans, E. J., & Pu, Z. (2009). Stressed memories: How acute stress affects memory formation in humans. *Journal of Neuroscience, 29*(32), 10111-10119.

Herrero, A. I., & Sandi, C. (2006). Individual differences in anxiety trait are related to spatial learning abilities and hippocampal expression of mineralocorticoid receptors. *Neurobiology of Learning and Memory, 86*(2), 150-159.

Hetzel-Riggin, M., & Roby, R. P. (2013). Trauma type and gender effects on PTSD, general distress, and peritraumatic dissociation. *Journal of Loss and Trauma, 18*(1), 41-53.

Holsboer F. (2000). The stress hormone is back on the map. *Current Psychiatry Reports, 2*, 454-456.

Horowitz, M. J., Wilner, N. R., & Alvarez, W. A. (1979). Impact of event scale: A measure of subjective stress. *Psychosomatic Medicine, 41*(3), 209-218.

Hugdahl, K., Westerhausen, R., Alho, K., Medvedev, S., Laine, M., & Hämäläinen, H. (2009). "Attention and cognitive control: Unfolding the dichotic listening story": Corrigendum. *Scandinavian Journal of Psychology, 50*(2), 191.

Ishizuka, K., Hillier, A., & Beversdorf, D. Q. (2007). Effect of the cold pressor test on memory and cognitive flexibility. *Neurocase, 13*(3), 154-157.

Imai, H., Steindler D. A., & Kitai S. T. (1986). The organization of divergent axonal projections from the midbrain raphe nuclei in the rat. *The Journal of Comparative Neurology, 15*, 363-380.

Ivy, A. S., Rex, C. S., Chen, Y., Dube, C., Maras, P. M., Grigoriadis, D. E., . . Baram, T. Z. (2010). Hippocampal dysfunction and cognitive impairments provoked by chronic early-life stress involve excessive activation of CRH receptors. *Journal of Neuroscience, 30*(39), 13005-13015.

Jacobs B. L., van Praag, H., & Gage F. H. (2000). Adult brain neurogenesis and psychiatry: a novel theory of depression. *Molecular Psychiatry, 5*, 262-269.

Jaeggi, S. M., Buschkuehl, M., Jonides, J., & Perrig, W. J. (2008). Improving fluid intelligence with training on working memory. *Proceedings of the National Academy of Sciences of the United States of America, 105*, 6829-6833.

Jenkins, M. A., Langlais, P. J., Delis, D., & Cohen, R. A. (2001). Attentional dysfunction associated with posttraumatic stress disorder among rape survivors. *Clinical Neuropsychologist, 14*(1), 7-12.

Joëls, M., & Baram, T. Z. (2009). The neuro-symphony of stress. *Nature Reviews Neuroscience, 10*(6), 459-466.

Joels, M., Krugers, H., & Wiegert, O. (2006). Timing is essential for rapid effects of corticosterone on synaptic potentiation in the mouse hippocampus. *Learning & Memory, 13*(2), 110-113.

Joels & de Kloet (1989). Effects of glucocorticoids and norepinephrine on the excitability in the

hippocampus. *Science, 245,* 1502-1505.

Joormann, J., Levens, S. M., & Gotlib, I. H. (2011). Sticky thoughts: Depression and rumination are associated with difficulties manipulating emotional material in working memory. *Psychological Science, 22*(8), 979-983.

Kabbaji, M., Devine, D. P., Savage, V. R., & Akil, H. (2000). Neurobiological correlates of individual differences in novelty-seeking behavior in the rat: Differential expression of stress-related molecules. *The Journal of Neuroscience, 20*(18), 6983-6988.

Kaplan, J.R., Pettersson, K., Manuck, S.B., & Olsson, G. (1991). Role of sympathoadrenal medullary activation in initiation and progression of atherosclerosis. *Circulation, 84* (6, suppl), 123-132.

Karst, H., Berger, S., Turiault, M., Tronche, F., Schutz, G, & Joels, M. (2005). Mineralocorticoid receptors are indispensable for nongenomic modulation of hippocampal glutamate transmission by corticosterone. *Proceedings of the National Academy of Science of the United States of America, 102*, 19204-19207.

Keinan, G., Shrira, A., & Shmotkin, D. (2012). The association between cumulative adversity and mental health: Considering dose and primary focus of adversity. *Quality of Life Research: An International Journal of Quality of Life Aspects of Treatment, Care & Rehabilitation, 21*(7), 1149-1158.

Kensinger, E. A., & Schacter, D. L. (2007). Remembering the specific visual details of presented objects: Neuroimaging evidence for effects of emotion. *Neuropsychologia, 45*(13), 2951-2962.

Kensinger, E. A., & Corkin (2004). Two routes to emotional memory: Distinct neural processes for valence and arousal. *Proceedings of the National Academy of Science of the United*

States of America, 101, 3310-3315.

Kerr, D. S., Campbell, L. W., Applegate, M. D., Brodish, A., & Lanfield, P. W. (1991). Chronic stress-induced acceleration of electrophysiologic and morphometric biomarkers of hippocampal aging. *Journal of Neuroscience, 11*, 1316-1324.

Khoozani, E. N., & Hadzic, M. (2010). Designing the human stress ontology: A formal framework to capture and represent knowledge about human stress. *Australian Psychologist, 45*(4), 258-273.

Kirschbaum, C., Wolf, O. T., May, M., & Wippich, W. (1996). Stress- and treatment-induced elevations of cortisol levels associated with impaired declarative memory in healthy adults. *Life Sciences, 58*(17), 1475-1483.

Kirschbaum, C., Prüssner, J. C., Stone, A. A., & Federenko, I. (1995). Persistent high cortisol responses to repeated psychological stress in a subpopulation of healthy men. *Psychosomatic Medicine, 57*(5), 468-474.

Koenigs, M., & Grafman, J. (2009). Posttraumatic stress disorder: The role of medial prefrontal cortex and amygdala. *Neuroscientist, 15*(5), 540-548.

Kolb, B., Mychasiuk, R., Muhammad, A., & Gibb, R. (2013). Brain plasticity in the developing brain. *Progress in Brain Research, 207*, 35-64.

Korf, J., & Van Praag H.M. (1971). Amine metabolism in the human brain: further evaluation of the probenecid test. *Brain Research, 35*, 221-230.

Krompinger, J. W., & Simons, R. F. (2011). Cognitive inefficiency in depressive undergraduates: Stroop processing and ERPs. *Biological Psychology, 86*(3), 239-246.

Krupnick, J. L., Green, B. L., Stockton, P., Goodman, L., Corcoran, C., & Petty, R. (2004). Mental health effects of adolescent trauma exposure in a female college sample: Exploring

differential outcomes based on experiences of unique trauma types and dimensions. *Psychiatry: Interpersonal and Biological Processes, 67*(3), 264-279.

Krystal, J. H., & Neumeister, A. (2009). Noradrenergic and serotonergic mechanisms in the neurobiology of posttraumatic stress disorder and resilience. *Brain Research, 1293,* 13-23.

Lancaster, S. L., Melka, S. E., & Rodriguez, B. F. (2009). An examination of the differential effects of the experience of DSM-IV defined traumatic events and life stressors. *Journal of Anxiety Disorders, 23*(5), 711-717.

Lazarus, R. S. & Folkman, S. (1984). *Stress, appraisal, and coping.* New York, NY: Springer Publishing Company, Inc.

LaGarde, G., Doyon, J., & Brunet, A. (2010). Memory and executive dysfunctions associated with acute posttraumatic stress disorder. *Psychiatry Research, 177*(1-2), 144-149.

LandrØ, N. I., Stiles, T. C., & Sletvold, T. (2001). Neuropsychological function in nonpsychotic unipolar major depression. *Neuropsychiatry, Neuropsychology and Behavioral Neurology, 14*(4), 233-240.

LeDoux, J. E. (2000). Emotion circuits in the brain. *Annual Review of Neuroscience, 23*, 155-184.

Lemieux, A. M., & Coe, C. L. (1995). Abuse-related posttraumatic stress disorder: Evidence for chronic neuroendocrine activation in women. *Psychosomatic Medicine, 57*(2), 105-115.

Lezak, M. D., Howieson, D. B., Loring, D. W., Hannay, H. J., & Fischer, J. S. (2004). *Neuropsychological assessment (4th ed.).* New York, NY: Oxford University Press.

Li, Y., Dong, F., Cao, F., Cui, N., Li, J., & Long, Z. (2013). Poly-victimization and executive functions in junior college students. *Scandinavian Journal of Psychology, 54*(6), 485-492.

Liston, C., Miller, M. M., Goldwater, D. S., Radley, J. J., Rocher, A. B., Hof, P. R., . McEwen, B. S. (2006). Stress-induced alterations in prefrontal cortical dendritic morphology predict selective impairments in perceptual attentional set-shifting. *The Journal of Neuroscience, 26*(30), 7870-7874.

Litz, B. T., Orsillo, S. M., Kaloupek, D., & Weathers, F. (2000). Emotional processing in posttraumatic stress disorder. *Journal of Abnormal Psychology, 109*, 26-39.

Lowry, C. A. (2002). Functional subsets of serotonergic neurones: implications for control of the hypothalamic-pituitary-adrenal axis. *Journal of Neuroendocrinology, 14*, 911-923.

Ludwig, M. (1998). Dendritic release of vasopressin and oxytocin. *Journal of Neuroendocrinology, 10*, 881-895.

Luine, V., Villegas, M., Martinez, C., & Mcewen, B. S. (1994). Repeated stress causes reversible impairments of spatial memory performance. *Brain Research, 639*(1), 167-170.

Lupien, S. J., Gillin, C. J., & Hauger, R. L. (1999). Working memory is more sensitive than declarative memory to the acute effects of corticosteroids: a dose-response study in humans. *Behavioral Neuroscience, 113*, 420-430.

Lyche, P., Jonassen, R., Stiles, T. C., Ulleberg, P., & LandrØ, N. I. (2011). Attentional functions in major depressive disorders with and without comorbid anxiety. *Archives of Clinical Neuropsychology, 26*(1), 38-47.

MacQueen, G. M., Tipper, S. P., Young, L. T., Joffe, R. T., & Levitt, A. J. (2000). Impaired distractor inhibition on a selective attention task in unmedicated, depressed subjects. *Psychological Medicine, 30*(3), 557-564.

Maes, M., Meltzer, H.Y., D'Hondt, P., Cosyns P.,. Blockx P. (1995). Effects of serotonin

precursors on the negative feedback effects of glucocorticoids on hypothalamic-pituitary-adrenal axis function in depression. *Psychoneuroendocrinology, 20*, 149-167.

Maes M., Leonard B. E., Myint A. M., Kubera M., & Verkerk R. (2011). The new '5-HT' hypothesis of depression: cell-mediated immune activation induces indoleamine 2,3-dioxygenase, which leads to lower plasma tryptophan and an increased synthesis of detrimental tryptophan catabolites (TRYCATs), both of which contribute to the onset of depression. *Progress in Neuropsychpharmacology & Biological Psychiatry, 35*, 702-721.

Magarinos, A. M., & McEwen, B. S. (1995). Stress-induced atrophy of apical dendrites of hippocampal CA3c neurons: involvement of glucocorticoid secretion and excitatory amino acid receptors. *Neuroscience, 69*, 89-98.

Magariños, A. M., McEwen, B. S., Flügge, G., & Fuchs, E. (1996). Chronic psychosocial stress causes apical dendritic atrophy of hippocampal CA3 pyramidal neurons in subordinate tree shrews. *The Journal of Neuroscience, 16*(10), 3534-3540.

Malberg, J. E., Eisch, A. J., Nestler, E. J., & Duman, R. S. (2000). Chronic antidepressant treatment increases neurogenesis in adult rat hippocampus. *The Journal of Neuroscience: The official journal for the society for neuroscience,20*, 9104-9110.

Malberg, J. E. (2004). Implications of adult hippocampal neurogenesis in antidepressant action. *Journal of Psychiatry & Neuroscience, 29*(3), 196-205.

Marazziti, D., Consoli, G., Picchetti, M., Carlini, M., & Faravelli, L. (2010). Cognitive impairment in major depression. *European Journal of Psychiatry, 626*(1) 83-86.

Maren, S. (2001). Is there savings for pavlovian fear conditioning after neurotoxic basolateral amygdala lesions in rats? *Neurobiology of Learning and Memory, 76*(3), 268-283.

Mason, J. W. (1986). Urinary free-cortisol levels in posttraumatic stress disorder patients. *Journal of Nervous and Mental Disease, 174*(3), 145-149.

Masuda, M. & Holmes, T. H. (1967). Magnitude estimations of social readjustments. *Journal of Psychosomatic Research, 11*, 219-225.

McEwen, B. S. (1998). *Stress, adaptation, and disease: Allostasis and allostatic load.* New York, NY: New York Academy of Sciences.

McEwen, B. S. (2004). *Protective and damaging effects of the mediators of stress and adaptation: Allostasis and allostatic load.* New York, NY: Cambridge University Press.

McEwen, B. S., & Lasley, E. N. (2002). *The end of stress as we know it.* Washington, DC: Joseph Henry Press.

McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews, 87*, 873-904.

McGuire, P. K., Paulesu, E., Frackowiak, R. S., & Frith, C. D. (1996). Brain activity during stimulus independent thought. *Neuroreport, 7*, 2095-2099.

McLaughlin, K. A., Conron, K. J., Koenen, K. C., & Gilman, S. E. (2010). Childhood adversity, adult stressful life events, and risk of past-year psychiatric disorder: A test of the stress sensitization hypothesis in a population-based sample of adults. *Psychological Medicine, 40*(10), 1647-1658.

Messman-Moore, T., Ward, R. M., & Zerubavel, N. (2013). The role of substance use and emotion dysregulation in predicting risk for incapacitated sexual revictimization in women: Results of a prospective investigation. *Psychology of Addictive Behaviors, 27*(1), 125-132.

Maier S. F. & Watkins L. R. (2005). Stressor controllability and learned helplessness: the roles of the dorsal raphe nucleus, serotonin, and corticotrophin-releasing factor. *Neuroscience and Biobehavioral Reviews, 29*, 829-841.

Merriam, E. P., Thase, M. E., Haas, G. L., Keshavan, M. S., & Sweeney, J. A. (1999). Prefrontal cortical dysfunction in depression determined by Wisconsin card sorting test performance. *The American Journal of Psychiatry, 156*(5), 780-782.

Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience, 24*, 167-202.

Miller, G. A. (1956). The magical number seven, plus or minus two: Some limits on our capacity for processing information. *Psychological Review, 63*(2), 81-97.

Miller, M. W., & Litz, B. T. (2004). Emotional-processing in posttraumatic stress disorder II: Startle reflex modulation during picture processing. *The Journal of Abnormal Psychology, 113*(3), 451-463.

Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin, 133*, 25-45.

Morgan, C. A., Krystal, J. H., & Southwick, S. M. (2003). Toward early pharmacological posttraumatic stress intervention. *Biological Psychiatry, 53*(9), 834-843.

Morilak, D. A., Barrera, G., Echevarria, D. J., Garcia, A. S., & Hernandez, A. (2005). Role of brain norepinephrine in the behavioral response to stress. *Progress in Neuro-pharmacology& Biological Psychiatry, 29*, 1214-1224.

Müller, M. B., Lucassen, P. J., Yassouridis, A., Hoogendijk, W. J. G., Holsboer, F., & Swaab, D. F. (2001). Neither major depression nor glucocorticoid treatment affects the cellular

integrity of the human hippocampus. *European Journal of Neuroscience, 14*(10), 1603-1612.

Murrough, J. W., Iacoviello, B., Neumeister, A., Charney, D. S., & Iosifescu, D. V. (2011). Cognitive dysfunction in depression: Neurocircuitry and new therapeutic strategies. *Neurobiology of Learning and Memory,* 1074-7427.

Navalta, C. P., Polcari, A., Webster, D. M., Boghossian, A., & Teicher, M. H. (2006). Effects of childhood sexual abuse on neuropsychological and cognitive function in college women. *The Journal of Neuropsychiatry and Clinical Neurosciences, 18*(1), 45-53.

Nebes, R. D., Butters, M. A., Mulsant, B. H., Pollock, B. G., Zmuda, M. D., Houck, P. R., & Reynolds, C. F. (2000). Decreased working memory and processing speed mediate cognitive impairment in geriatric depression. *Psychological Medicine, 30*(3), 679-691.

Neumaier, J.F., Edwards, E., & Plotsky, P.M. (2002). 5-HT(1B) mrna regulation in two animal models of altered stress reactivity. *Biological Psychiatry, 51*, 902-908.

Oehman, L., Nordin, S., Bergdahl, J., Birgander, L. S., & Neely, A. S. (2007). Cognitive function in outpatients with perceived chronic stress. *Scandinavian Journal of Work, Environment & Health, 33*(3), 223-232.

Ogle, C. M., Rubin, D. C., & Siegler, I. C. (2013). The impact of the developmental timing of trauma exposure on PTSD symptoms and psychosocial functioning among older adults. *Developmental Psychology, 49*(11), 2191-2200.

Onur, O. A., Walter, H., Schlaepfer, T. E., Rehme, A. K., Schmidt, C., Keysers, C., . Hurlemann, R. (2009). Noradrenergic enhancement of amygdala responses to fear. *Social Cognitive and Affective Neuroscience, 4*(2), 119-126.

Oomen, C. A., Soeters, H., Audureau, N., Vermunt, L., van Hasselt, F. N., Manders, E. M. M., . . Krugers, H. (2010). Severe early life stress hampers spatial learning and neurogenesis, but improves hippocampal synaptic plasticity and emotional learning under high-stress conditions in adulthood. *The Journal of Neuroscience, 30*(19), 6635-6645.

Orem, D. M., Petrac, D. C., & Bedwell, J. S. (2008). Chronic self-perceived stress and set-shifting performance in undergraduate students. *Stress: The International Journal on the Biology of Stress, 11*(1), 73-78.

Owen, A. M., McMillan, K. M., Laird, A. R., & Bullmore, E. (2005). N-back working memory paradigm: A meta-analysis of normative functional neuroimaging studies. *Human Brain Mapping, 25*(1), 46-59.

Pachana, N. A., Brilleman, S. L., & Dobson, A. J. (2011). Reporting of life events over time: Methodological issues in a longitudinal sample of women. *Psychological Assessment, 23*(1), 277-281.

Palgi, Y., Shrira, A., Ben-Ezra, M., Shiovitz-Ezra, S., & Ayalon, L. (2012). Self- and other-oriented potential lifetime traumatic events as predictors of loneliness in the second half of life. *Aging & Mental Health, 16*(4), 423-430.

Pascucci, T., Ventura, R., Latagliata, E. C., Cabib, S., & Puglisi-Allegra, S. (2007). The medial prefrontal cortex determines the accumbens dopamine response to stress through the opposing influences of norepinephrine and dopamine. *Cerebral Cortex, 17*(12), 2796-2804.

Pavlides, C., Nivón, L. G., & McEwen, B. S. (2002). Effects of chronic stress on hippocampal long-term potentiation. *Hippocampus, 12*(2), 245-257.

Perea, C. S., Paternina, A. C., Gomez, Y., & Lattig, M. C. (2012). Negative affectivity moderated by BDNF and stress response. *Journal of Affective Disorders, 136*(3), 767-774.

Petty, F. (1995). GABA and mood disorders: a brief review and hypothesis. *Journal of Affective Disorders, 34*, 275-281.

Pineles, S. L., Suvak, M. K., Liverant, G. I., Gregor, K., Wisco, B. E., Pitman, R. K., & Orr, S. P. (2013). Psychophysiologic reactivity, subjective distress, and their associations with PTSD diagnosis. *Journal of Abnormal Psychology, 122*(3), 635-644.

Pitman, R. K., & Orr, S. P. (1990). Twenty-four hour urinary cortisol and catecholamine excretion in combat-related posttraumatic stress disorder. *Biological Psychiatry, 27*(2), 245-247.

Porter, R. J., Gallagher, P., Thompson, J. M., & Young, A. H. (2003). Neurocognitive impairment in drug-free patients with major depressive disorder. *British Journal of Psychiatry, 182*(3), 214-220.

Pruessner, J. C., Dedovic, K., Khalili-Mahani, N., Engert, V., Pruessner, M., Buss, C., . . . Lupien, S. (2008). Deactivation of the limbic system during acute psychosocial stress: Evidence from positron emission tomography and functional magnetic resonance imaging studies. *Biological Psychiatry, 63*(2), 234-240.

Quirk, G. J., Garcia, R., & González-Lima, F. (2006). Prefrontal mechanisms in extinction of conditioned fear. *Biological Psychiatry, 60*(4), 337-343.

Radley, J. J., Sisti, H. M., Hao, J., Rocher, A. B., McCall, T., Hoff, P., . Morrison, J. (2004). Chronic behavioral stress induces apical dendritic reorganization in pyramidal neurons of the medial prefrontal cortex. *Neuroscience, 125*, 1-6.

Radley, J. J., Williams, B., & Sawchenko, P. E. (2008). Noradrenergic innervation of the dorsal medial prefrontal cortex modulates hypothalamo-pituitary-adrenal responses to acute emotional stress. *The Journal of Neuroscience, 28*(22), 5806-5816.

Radley, J. J., & Sawchenko, P. E. (2011). A common substrate for prefrontal and hippocampal inhibition of the neuroendocrine stress response. *Journal of Neuroscience, 29*, 9683-9695.

Ramachandran, V.S. (2002). *Encyclopedia of the human brain.* Academic Press.

Read, J. P., Colder, C. R., Merrill, J. E., Ouimette, P., White, J., & Swartout, A. (2012). Trauma and posttraumatic stress symptoms predict alcohol and other drug consequence trajectories in the first year of college. *Journal of Consulting and Clinical Psychology*, *80*(3), 426-439.

Read, J. P., Ouimette, P., White, J., Colder, C., & Farrow, S. (2011). Rates of DSM–IV–TR trauma exposure and posttraumatic stress disorder among newly matriculated college students. *Psychological Trauma: Theory, Research, Practice, and Policy*, *3*(2), 148-156.

Renner, K. H., & Beversdorf, D. Q. (2010). Effects of naturalistic stressors on cognitive flexibility and working memory task performance. *Neurocase, 16*(4), 293-300.

Ressler K. J., & Nemeroff C. B. (1999). Role of norepinephrine in the pathophysiology and treatment of mood disorders. *Biological Psychiatry*, *46*, 1219-1233.

Reyes, G., Elhai, J. D., & Ford, J. D. (2008). *The encyclopedia of psychological trauma.* Hoboken, NJ: John Wiley & Sons Inc.

Sanchez, M. M., Young, L. J., Plotsky, P. M., & Insel, T. R. (2000). Distribution of corticosteroid receptors in the rhesus brain: relative absence of glucocorticoid receptors in the hippocampal formation. *The Journal of Neuroscience: The official journal for the Society of Neuroscience*, *20*, 4657-4668.

Santarelli, L., Saxe, M., Gross, C., Surget, A., & Battaglia, F. (2003). Requirement of hippocampal neurogenesis for the behavioral effects of antidepressants. *Science, 301*, 805-809.

Sapolsky, R. M. R., Krey, L. C. L., & McEwen, B. S. B. (1984). Glucocorticoid-sensitive hippocampal neurons are involved in terminating the adrenocortical stress response. *Proceedings of the National Academy of Sciences of the United States of America, 81*(19), 6174-6177.

Sapolsky, R. M., Krey, L. C., & McEwen, B. S. (1985). Prolonged glucocorticoid exposure reduces hippocampal neuron number: implications for aging. *Journal of Neuroscience, 5,* 1222-1227.

Sapolsky, R. M. R. (1985). A mechanism for glucocorticoid toxicity in the hippocampus: Increased neuronal vulnerability to metabolic insults. *The Journal of Neuroscience : The Official Journal of the Society for Neuroscience, 5*(5), 1228-1232.

Sapolsky, R. M., Krey, L. C., & McEwen, B. S. (1986). The neuroendocrinology of stress and aging: The glucocorticoid cascade hypothesis. *Endocrine Reviews, 7,* 284-301.

Sapolsky, R. M. (2003). Taming stress. *Scientific American, 289,* 86-95.

Sapolsky, R. M. (2007). *Why zebras don't get ulcers: Stress, metabolism, and liquidating your assets.* Westport, CT: Praeger Publishers/Greenwood Publishing Group.

Sarafino, E. P. (2008). *Health psychology: Biopsychosocial interactions, Sixth Edition*. Hoboken, NJ: Wiley & Sons, Inc.

Sarason, I. G., Johnson, J. H., & Siegel, J. M. (1978). Assessing the impact of life changes: Development of the life experiences survey. *Journal of Consulting and Clinical Psychology, 46*(5), 932-946.

Sawchenko P. E., & Swanson L. W.(1983). The organization of forebrain afferents to the paraventricular and supraoptic nuclei of the rat. *The Journal of Comparative Neurology, 218,* 121-144.

Schally, A. V., Redding, T. W., Bowers, C. Y., & Barrett, J. F. (1969). Isolation and Properties of Porcine Thyrotropin-releasing Hormone. *The Journal of Biological Chemistry, 244*, 4077- 4088.

Schiepers, O. J., Wichers, M. C., & Maes, M.(2005). Cytokines and major depression. *Progress in Neuropsychopharmacology & Biological Psychiatry, 29*, 201-217.

Schell, T. L., Marshall, G. N., & Jaycox, L. H. (2004). All symptoms are not created equal: the prominent role of hyperarousal in the natural course of posttraumatic psychological distress. *Journal of Abnormal Psychology, 113*, 189-197.

Schloesser, R. J., Manji, H. K., & Martinowich, K. (2009). Suppression of adult neurogenesis leads to an increased hypothalamo-pituitary-adrenal axis response. *NeuroReport: For Rapid Communication of Neuroscience Research, 20*(6), 553-557.

Schmidt, L. A. L., Fox, N. A. N., Goldberg, M. C. M., Smith, C. C. C., & Schulkin, J. J. (1999). Effects of acute prednisone administration on memory, attention and emotion in healthy human adults. *Psychoneuroendocrinology, 24*(4), 461-483.

Schmitz, T. W., Kawahara-Baccus, T. N., & Johnson, S. C. (2004). Metacognitive evaluation, self-relevance, and the right prefrontal cortex. *Neuroimage, 22*, 941-947.

Schwartz, G. E. (1982). Testing the biopsychosocial model: the ultimate challenge facing behavioral medicine? *Journal of consulting and clinical psychology, 50*, 1040-1053.

Scott, M. J., & Stradling, S. G. (2001). Translating the psychobiology of post-traumatic stress disorder into clinically useful analogy. *British Journal of Medical Psychology, 74*(2), 249-254.

Seyle, H. (1956). *The stress of life.* New York, NY: McGraw Hill

Sheline, Y. I. (2000). 3D MRI studies of neuroanatomic changes in unipolar major depression: The role of stress and medical comorbidity. *Biological Psychiatry, 48,* 791-800.

Shelton, R. C. (2000). Cellular mechanisms in the vulnerability to depression and response to antidepressants. *The Psychiatric Clinics of North America, 23,* 713-729.

Shucard, J. L., McCabe, D. C., & Szymanski, H. V. (2008). An event-related potential study of attention deficits in posttraumatic stress disorder during auditory and visual Go/NoGo continuous performance tasks. *Biological Psychology, 79*(2), 223-233.

Smith, E. E., & Jonides, J. (1997). Working memory: A view from neuroimaging. *Cognitive Psychology, 33,* 5-42.

Sousa, N., Lukoyanov, N. V., Madeira, M. D., Almeida, O. F., & Paula-Barbosa, M. M. (2000). Reorganization of the morphology of hippocampal neurites and synapses after stress-induced damage correlates with behavioral improvement. *Neuroscience, 97,* 253-266.

Sotres-Bayon, F., Bush, D. E. A., & LeDoux, J. E. (2004). Emotional perseveration: An update on prefrontal-amygdala interactions in fear extinction. *Learning & Memory, 11*(5), 525-535.

Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). *Manual for the State-Trait Anxiety Inventory.* Palo Alto, CA: Consulting Psychologists Press

Stein, M. B., Kennedy, C. M., & Twamley, E. W. (2002). Neuropsychological function in female victims of intimate partner violence with and without posttraumatic stress disorder. *Biological Psychiatry, 52*(11), 1079-1088.

Sterling, P., & Eyer, J. (1988). *Allostasis: A new paradigm to explain arousal pathology.* Oxford, England: John Wiley & Sons.

Sterling, P. (2011). Allostasis: A model of predictive regulation. *Physiology & Behavior, June.*

Stevenson, C. W., & Gratton, A. (2003). Basolateral amygdala modulation of the nucleus accumbens dopamine response to stress: Role of the medial prefrontal cortex. *European Journal of Neuroscience, 17*(6), 1287-1295.

Sugar, J., & Ford, J. D. (2012). Peritraumatic reactions and posttraumatic stress disorder in psychiatrically impaired youth. *Journal of Traumatic Stress, 25*(1), 41-49.

Sunanda, M. S. R., & Raju, T. R. (1995). Effect of chronic restraint stress on dendritic spines and excrescences of hippocampal CA3 pyramidal neurons: A quantitative study. *Brain Research, 694*(1-2), 312-317.

Sutton, A. (2007). *Stress-related disorders sourcebook.*

Taylor, S. E., Klein, L. C., Lewis, B. P., Gruenewald, T. L., Gurung, R. A. R., & Updegraff, J. A. (2000). Biobehavioral responses to stress in females: Tend-and-befriend, not fight-or-flight. *Psychological Review, 107*(3), 411-429.

Thase, M. E., Jindal, R., & Howland, R. H. (2002). *Biological aspects of depression.* New York, NY: Guilford Press.

Touyarot, K., Venero, C., & Sandi, C. (2004). Spatial learning impairment induced by chronic stress is related to individual differences in novelty reactivity: Search for neurobiological correlates. *Psychoneuroendocrinology, 29*(2), 290-305.

Toker, L., Amar, S., Bersudsky, Y., Benjamin, J., & Klein, E. (2010). The biology of tryptophan depletion and mood disorders. *The Israel Journal of Psychiatry and related sciences, 47*, 46-55.

Tran, C. T., Kuhn, E., Walser, R. D., & Drescher, K. D. (2012). The relationship between religiosity, PTSD, and depressive symptoms in veterans in PTSD residential treatment. *Journal of Psychology and Theology, 40*(4), 313-322.

Turner, H. A., & Butler, M. J. (2003). Direct and indirect effects of childhood adversity on depressive symptoms in young adults. *Journal of Youth and Adolescence, 32*(2), 89-103.

Twamley, E. W., Allard, C. B., Thorp, S. R., Norman, S. B., Cissell, S. H., Berardi, K. H., . . . Stein, M. B. (2009). Cognitive impairment and functioning in PTSD related to intimate partner violence. *Journal of the International Neuropsychological Society, 15*(6), 879-887.

Twamley, E. W., Hami, S., & Stein, M. B. (2004). Neuropsychological function in college students with and without posttraumatic stress disorder. *Psychiatry Research, 126*(3), 265-274.

Ulrich-Lai, Y. M., & Herman, J. P. (2009). Neural regulation of endocrine and autonomic stress responses. *Nature reviews: Neuroscience,10*, 397-409.

Vale, W., Spiess, J., Rivier, C., & Rivier J. (1981). Characterization of a 41-residue ovine hypothalamic peptide that stimulates secretion of corticotrophin and beta-endorphin. *Science, 213*, 1394-1397.

van der Kolk, B. A., McFarlane, A. C., & Weisaeth, L. (1996). *Traumatic stress: The effects of overwhelming experience on mind, body, and society.* New York, NY: The Guilford Press.

Vasterling, J. J., & Brewin, C. R. (2005). *Neuropsychology of PTSD: Biological, cognitive, and clinical perspectives* New York, NY: Guilford Press.

Veltmeyer, M. D., Clark, C. R., McFarlane, A. C., Moores, K. A., Bryant, R. A., & Gordon, E. (2009). Working memory function in post-traumatic stress disorder: An event-related potential study. *Clinical Neurophysiology, 120*(6), 1096-1106.

Vermetten, E., & Bremner, J. D. (2002). Circuits and systems in stress: II, applications to neurobiology and treatment in posttraumatic stress disorder. *Depression and Anxiety, 16*(1), 14-38.

Virgin, C. E., Ha, T. P., Packan, D. R., Tombaugh, G. C., Yang, S. H., et al. (1991). Glucocorticoids inhibit glucose transport and glutamate uptake in hippocampal astrocytes: Implications for glucocorticoid neurotoxicity. *Journal of Neuroschemistry, 57,* 1422-1428.

Vollman-Honsdorf, G. K., Flugge, G., & Fuchs, E. (1997). Chronic psychosocial stress does not affect the number of pyramidal neurons in tree shrew hippocampus. *Neuroscience Letters, 233*(2-3), 121-124.

Vyas, A., Pillai, A. G., & Chattarji, S. (2004). Recovery after chronic stress fails to reverse amygdaloid neuronal hypertrophy and enhanced anxiety-like behavior. *Neuroscience, 128,* 667-673.

Watts-English, T., Fortson, B. L., Gibler, N., Hooper, S. R., & DeBellis, M. D. (2006). The psychobiology of maltreatment in childhood. *Journal of Social Issues, 62*(4), 717-736.

Weber, D. L., Clark, C. R., McFarlane, A. C., Moores, K. A., Morris, P. L. P., & Egan, G. F. (2005). Abnormal frontal and parietal activity during working memory updating in post-traumatic stress disorder. *Psychiatry Research: Neuroimaging, 140*(1), 27-44.

Weiss, D. S., & Marmar, C. R. (1997). *The impact of event Scale—Revised.* New York, NY: Guilford Press.

Weiss, S. J. (2007). Neurobiological alterations associated with traumatic stress. *Perspectives in Psychiatric Care, 43*(3), 114-122.

White, P. M. (2007). Attentional networks reveal executive function deficits in posttraumatic stress disorder. *Neuropsychology, 21*(3), 275-284.

Williams, L. M., Kemp, A. H., Felmingham, K., Barton, M., & Olivieri, G. (2006). Trauma modulates amygdala and medial prefrontal responses to consciously attended fear. *Neuroimage, 29,* 347-357.

Winn, P. (2001). *Dictionary of biological psychology.* New York, NY: Routledge.

Wixted, J. T. (2007). Dual-process theory and signal-detection theory of recognition memory. *Psychological Review, 114*(1), 152-176.

Woolley, C. S., Gould, E., & McEwen, B. S. (1990). Exposure to excess glucocorticoids alters dendritic morphology of adult hippocampal pyramidal neurons. *Brain Research, 531,* 225-231.

Yehuda, R., Southwick, S. M., Nussbaum, G., & Wahby, V. S. (1990). Low urinary cortisol excretion in patients with posttraumatic stress disorder. *Journal of Nervous and Mental Disease, 178*(6), 366-369.

Yehuda, R., Southwick, S. M., Giller, E. L., Ma, X., & Mason, J. W. (1992). Urinary catecholamine excretion and severity of PTSD symptoms in Vietnam combat veterans. *Journal of Nervous and Mental Disease, 180*(5), 321-325.

Yehuda, R., Kahana, B., Binder-Brynes, K., & Southwick, S. M. (1995). Low urinary cortisol excretion in holocaust survivors with posttraumatic stress disorder. *The American Journal of Psychiatry, 152*(7), 982-986.

Yehuda, R., Teicher, M. H., Trestman, R. L., Levengood, R. A., & Siever, L. J. (1996). Cortisol regulation in posttraumatic stress disorder and major depression: A chronobiological analysis. *Biological Psychiatry, 40*(2), 79-88.

Yehuda, R., Siever, L. J., Teicher, M. H., Levengood, R. A., Gerber, D. K., Schmeidler, J., & Yang, R. (1998). Plasma norepinephrine and 3-methoxy-4-hydroxyphenylglycol

concentrations and severity of depression in combat posttraumatic stress disorder and major depressive disorder. *Biological Psychiatry*, *44*(1), 56-63.

Yuen, E.Y., Liu, W., Karatsoreos, I. N., Feng, J., McEwen, B. S., & Yan, Z. (2009). Acute stress enhances glutamatergic transmission in prefrontal cortex and facilitates working memory. *Proceedings of the National Academy of Sciences of the United States of America, 106*, 14075-14079.

Appendix A

Screening Questionnaire

Below are listed several events or circumstances that may be experienced by college students. Please review this list and answer the questions that follow related to any of your experiences with these events. Please note: it is NOT necessary to indicate which of these events you have experienced. Also, your response to this question will only be reviewed by administrators within the subject pool.

Homelessness

Robbery

Poverty

Serious Personal Illness

Childhood abuse (sexual, physical, emotional)

Serious Injury/Accident

Death of a close family member or friend

Witnessing Domestic Violence

Sexual Assault

Military Combat

Serious illness or injury of family member

Being threatened with a weapon

Physical Assault (including domestic violence as victim)

Witnessing Crime

Have you experienced one or more of these events? Yes No

Please list the number of these events you have experienced. _____

Appendix B
Rey Auditory Verbal Learning Test
(RAVLT)

REY AUDITORY VERBAL LEARNING TEST

INSTRUCTIONS: (Trial 1 List A) I am going to read a list of words. Listen carefully, for when I stop you are to say back as many as you can remember. It doesn't matter in what order you repeat them. Just try to remember as many as you can.

(Trials 2,3,4,5 List A) Now I am going to read the same list again. When I stop tell me as many words as you can remember, including any words you said last time.

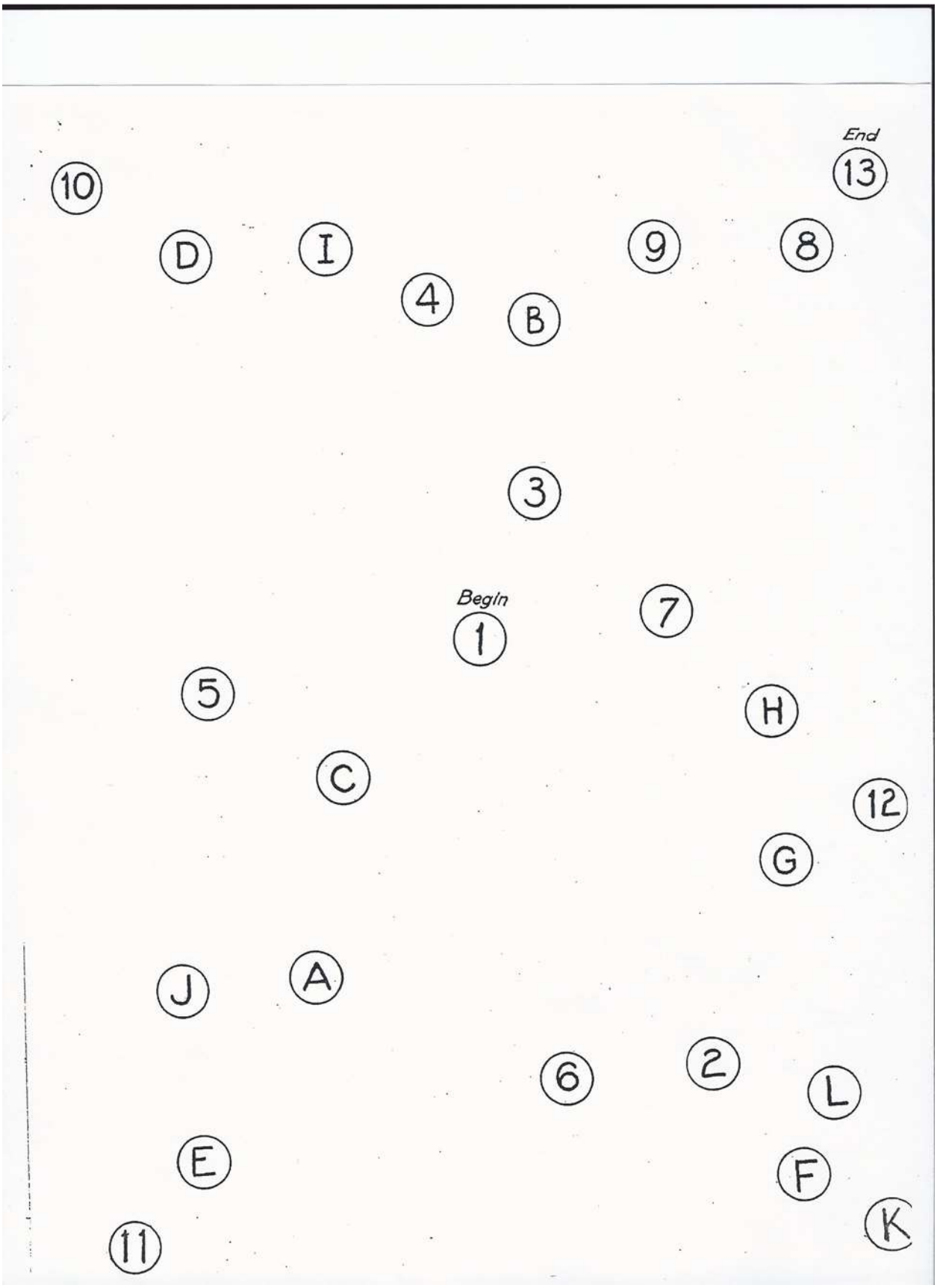
(Trial 6 List B) Now I am going to read a different list of words. This list I will read only once, but try to remember as many words as you can.

(Trial 7) Now tell me as many words as you can remember from the first list that I read to you.

I	II	III	IV	V	VI	VII	DELAYED RECALL
DRUM ___	DRUM ___	DRUM ___	DRUM ___	DRUM ___	DESK ___	DRUM ___	___
CURTAIN ___	CURTAIN ___	CURTAIN ___	CURTAIN ___	CURTAIN ___	RANGER ___	CURTAIN ___	___
BELL ___	BELL ___	BELL ___	BELL ___	BELL ___	BIRD ___	BELL ___	___
COFFEE ___	COFFEE ___	COFFEE ___	COFFEE ___	COFFEE ___	SHOE ___	COFFEE ___	___
SCHOOL ___	SCHOOL ___	SCHOOL ___	SCHOOL ___	SCHOOL ___	STOVE ___	SCHOOL ___	___
PARENT ___	PARENT ___	PARENT ___	PARENT ___	PARENT ___	MOUNTAIN ___	PARENT ___	___
MOON ___	MOON ___	MOON ___	MOON ___	MOON ___	GLASSES ___	MOON ___	___
GARDEN ___	GARDEN ___	GARDEN ___	GARDEN ___	GARDEN ___	TOWEL ___	GARDEN ___	___
HAT ___	HAT ___	HAT ___	HAT ___	HAT ___	CLOUD ___	HAT ___	___
FARMER ___	FARMER ___	FARMER ___	FARMER ___	FARMER ___	BOAT ___	FARMER ___	___
NOSE ___	NOSE ___	NOSE ___	NOSE ___	NOSE ___	LAMB ___	NOSE ___	___
TURKEY ___	TURKEY ___	TURKEY ___	TURKEY ___	TURKEY ___	GUN ___	TURKEY ___	___
COLOR ___	COLOR ___	COLOR ___	COLOR ___	COLOR ___	PENCIL ___	COLOR ___	___
HOUSE ___	HOUSE ___	HOUSE ___	HOUSE ___	HOUSE ___	CHURCH ___	HOUSE ___	___
RIVER ___	RIVER ___	RIVER ___	RIVER ___	RIVER ___	FISH ___	RIVER ___	___
LS ___	___	___	___	___	___	___	___

NAME _____

DATE _____


End
13
10
D
I
9
8
4
B
3
Begin
1
7
5
H
C
12
G
J
A
6
2
L
E
F
K
11

Impact of Event Scale- Revised

INSTRUCTIONS: Below is a list of difficulties people sometimes have after stressful life events. Please read each item, and then indicate how distressing each difficulty has been for you **DURING THE PAST SEVEN DAYS** with respect to _____ _____, which occurred on _____. How much were you distressed or bothered by these difficulties?

Item Response Anchors are 0 = Not at all; 1 = A little bit; 2 = Moderately; 3 = Quite a bit; 4 = Extremely.
The Intrusion subscale is the mean item response of items 1, 2, 3, 6, 9, 14, 16, 20.
The Avoidance subscale is the mean item response of items 5, 7, 8, 11, 12, 13, 17, 22.
The Hyperarousal subscale is the mean item response of items 4, 10, 15, 18, 19, 21.

1. Any reminder brought back feelings about it.
2. I had trouble staying asleep.
3. Other things kept making me think about it.
4. I felt irritable and angry.
5. I avoided letting myself get upset when I thought about it or was reminded of it.
6. I thought about it when I didn't mean to.
7. I felt as if it hadn't happened or wasn't real.
8. I stayed away from reminders of it.
9. Pictures of it popped into my mind.
10. I was jumpy and easily startled.
11. I tried not to think about it.
12. I was aware that I still had a lot of feelings about it, but I didn't deal with them.
13. My feelings about it were kind of numb.
14. I found myself acting or feeling like I was back at that time.
15. I had trouble falling asleep.
16. I had waves of strong feelings about it.
17. I tried to remove it from my memory.
18. I had trouble concentrating.
19. Reminders of it caused me to have physical reactions, such as sweating, trouble breathing, nausea, or a pounding heart.
20. I had dreams about it.
21. I felt watchful and on-guard.
22. I tried not to talk about it.

Total IES-R Score: _____

Contact information: Daniel S. Weiss, Ph.D., Professor of Medical Psychology, Department of Psychiatry, University of California San Francisco, CA 94143-0984, (415) 476-7557, Mail CodeL UCSF Box 0984-F, daniel.weiss@ucsf.edu

Appendix E
Life Experiences Survey

The Life Experiences Survey

Listed below are a number of events which sometimes bring about change in the lives of those who experience them and which necessitate social readjustment. *Please check those events which you have experienced in the recent past and indicate the time period during which you have experienced each event.* Be sure that all check marks are directly across from the items they correspond to.

Also, for each item checked below, *please indicate the extent to which you viewed the event as having either a positive or negative impact on your life* at the time the event occurred. That is, *indicate the type and extent of impact that the event had.* A rating of −3 would indicate an extremely negative impact. A rating of 0 suggests no impact either positive or negative. A rating of +3 would indicate an extremely positive impact.

Section 1

	0 to 6 mo	7 mo to 1 yr	extremely negative	moderately negative	somewhat negative	no impact	slightly positive	moderately positive	extremely positive
1. Marriage			−3	−2	−1	0	+1	+2	+3
2. Detention in jail or comparable institution			−3	−2	−1	0	+1	+2	+3
3. Death of spouse			−3	−2	−1	0	+1	+2	+3
4. Major change in sleeping habits (much more or much less sleep)			−3	−2	−1	0	+1	+2	+3
5. Death of close family member:									
a. mother			−3	−2	−1	0	+1	+2	+3
b. father			−3	−2	−1	0	+1	+2	+3
c. brother			−3	−2	−1	0	+1	+2	+3
d. sister			−3	−2	−1	0	+1	+2	+3
e. grandmother			−3	−2	−1	0	+1	+2	+3
f. grandfather			−3	−2	−1	0	+1	+2	+3
g. other (specify)			−3	−2	−1	0	+1	+2	+3
6. Major change in eating habits (much more or much less food intake)			−3	−2	−1	0	+1	+2	+3
7. Foreclosure on mortgage or loan			−3	−2	−1	0	+1	+2	+3
8. Death of close friend			−3	−2	−1	0	+1	+2	+3
9. Outstanding personal achievement			−3	−2	−1	0	+1	+2	+3
10. Minor law violations (traffic tickets, disturbing the peace, etc.)			−3	−2	−1	0	+1	+2	+3
11. *Male*: Wife/girlfriend's pregnancy			−3	−2	−1	0	+1	+2	+3
12. *Female*: Pregnancy			−3	−2	−1	0	+1	+2	+3
13. Changed work situation (different work responsibility, major change in working conditions, working hours, etc.)			−3	−2	−1	0	+1	+2	+3
14. New job			−3	−2	−1	0	+1	+2	+3
15. Serious illness or injury of close family member:									
a. father			−3	−2	−1	0	+1	+2	+3
b. mother			−3	−2	−1	0	+1	+2	+3
c. sister			−3	−2	−1	0	+1	+2	+3
d. brother			−3	−2	−1	0	+1	+2	+3
e. grandfather			−3	−2	−1	0	+1	+2	+3
f. grandmother			−3	−2	−1	0	+1	+2	+3
g. spouse			−3	−2	−1	0	+1	+2	+3
h. other (specify)			−3	−2	−1	0	+1	+2	+3
16. Sexual difficulties			−3	−2	−1	0	+1	+2	+3

Reprinted with permission of Irwin G. Sarason, Ph.D. Source: Sarason IG, Johnson JH, Siegel JM: "Assessing the Impact of Life Changes: Development of the Life Experiences Survey." *Journal of Consulting and Clinical Psychology* 46(5):932–946, 1978

	0 to 6 mo	7 mo to 1 yr	extremely negative	moderately negative	somewhat negative	no impact	slightly positive	moderately positive	extremely positive
40. Retirement from work			−3	−2	−1	0	+1	+2	+3
41. Son or daughter leaving home (due to marriage, college, etc.)			−3	−2	−1	0	+1	+2	+3
42. Ending of formal schooling			−3	−2	−1	0	+1	+2	+3
43. Separation from spouse (due to work, travel, etc.)			−3	−2	−1	0	+1	+2	+3
44. Engagement			−3	−2	−1	0	+1	+2	+3
45. Breaking up with boyfriend/girlfriend			−3	−2	−1	0	+1	+2	+3
46. Leaving home for the first time			−3	−2	−1	0	+1	+2	+3
47. Reconciliation with boyfriend/girlfriend			−3	−2	−1	0	+1	+2	+3
Other recent experiences which have had an impact on your life. List and rate.									
48._____			−3	−2	−1	0	+1	+2	+3
49._____			−3	−2	−1	0	+1	+2	+3
50._____			−3	−2	−1	0	+1	+2	+3
Section 2: Student Only									
51. Beginning a new school experience at a higher academic level (college, graduate school, professional school, etc.)			−3	−2	−1	0	+1	+2	+3
52. Changing to a new school at same academic level (undergraduate, graduate, etc.)			−3	−2	−1	0	+1	+2	+3
53. Academic probation			−3	−2	−1	0	+1	+2	+3
54. Being dismissed from dormitory or other residence			−3	−2	−1	0	+1	+2	+3
55. Failing an important exam			−3	−2	−1	0	+1	+2	+3
56. Changing a major			−3	−2	−1	0	+1	+2	+3
57. Failing a course			−3	−2	−1	0	+1	+2	+3
58. Dropping a course			−3	−2	−1	0	+1	+2	+3
59. Joining a fraternity/sorority			−3	−2	−1	0	+1	+2	+3
60. Financial problems concerning school (in danger of not having sufficient money to continue)			−3	−2	−1	0	+1	+2	+3

STRESSFUL LIFE EVENTS SCREENING QUESTIONNAIRE - REVISED

The items listed below refer to events that may have taken place at any point in your entire life, including early childhood. **If an event or ongoing situation occurred more than once, please record all pertinent information about additional events on the last page of this questionnaire. (**Please print or write neatly).

1. Have you ever had a life-threatening illness?

No _____ Yes _____ If yes, at what age? _____

Duration of Illness _____

Describe specific illness _____

2. Were you ever in a life-threatening accident?

No _____ Yes _____ If yes, at what age? _____

Describe accident_____

Did anyone die? ____ Who? (Relationship to you)_____

What physical injuries did you receive? _____

Were you hospitalized overnight? No_____ Yes _____

3. Was physical force or a weapon ever used against you in a robbery or mugging?

No _____ Yes _____ If yes, at what age? _____

How many perpetrators?_____

Describe physical force (e.g., restrained, shoved) or weapon used against you.

_____

Did anyone die? _____

Who?_____

What injuries did you receive? _____

Was your life in danger? _____

4. Has an immediate family member, romantic partner, or <u>very close</u> friend died because of accident, homicide, or suicide?

No _____ Yes _____ If yes, how old were you? _____

How did this person die? _____

Relationship to person lost _____

In the year before this person died, how often did you see/have contact with him/her? _____

Have you had a miscarriage? No _____ Yes _____ If yes, at what age?_____

5. At any time, has anyone (parent, other family member, romantic partner, stranger or someone else) ever <u>physically forced</u> you to have intercourse, or to have oral or anal sex against your wishes, or when you were helpless, such as being asleep or intoxicated?

No _____ Yes _____ If yes, at what age? _____

If yes, how many times? 1 _____, 2-4 _____, 5-10 _____, more than 10_____

If repeated, over what period? 6 mo. or less _____, 7 mos.-2 yrs. _____, more than 2 yrs. but less than 5 yrs. _____, 5 yrs. or more _____.

Who did this? (Specify stranger, parent, etc.) _____

Has anyone **else** ever done this to you? No_____ Yes_____

6. Other than experiences mentioned in earlier questions, has anyone ever touched private parts of your body, made you touch their body, or tried to make you to have sex against your wishes?

No _____ Yes _____ If yes, at what age? _____

If yes, how many times? 1 _____, 2-4 _____, 5-10 _____, more than 10_____

If repeated, over what period? 6 mo. or less _____, 7 mos.-2 yrs. _____, more than 2 yrs. but less than 5 yrs. _____, 5 yrs. or more _____.

Who did this? (Specify sibling, date, etc.) _____

What age was this person? _____

Has anyone **else** ever done this to you? No_____ Yes_____

7. When you were a child, did a parent, caregiver or other person ever slap you repeatedly, beat you, or otherwise attack or harm you?

No _____ Yes_____ If yes, at what age _____

If yes, how many times? 1 _____, 2-4 _____, 5-10 _____, more than 10 _____

If repeated, over what period? 6 mo. or less _____ , 7 mos.- 2 yrs. _____, more

than 2 yrs. but less than 5 yrs _____, 5 yrs. or more _____.

Describe force used against you (e.g., fist, belt)_____

Were you ever injured? _____ If yes, describe _____

Who did this? (Relationship to you) _____

Has anyone **else** ever done this to you? No _____ Yes _____

8. As an adult, have you ever been kicked, beaten, slapped around or otherwise physically harmed by a romantic partner, date, family member, stranger, or someone else?

No _____ Yes _____ If yes, at what age? _____

If yes, how many times? 1 _____, 2-4 _____, 5-10 _____, more than 10_____

If repeated, over what period? 6 mo. or less _____, 7 mos.- 2 yrs. _____, more

than 2 yrs. but less than 5 yrs. _____ , 5 yrs. or more _____.

Describe force used against you (e.g., fist, belt) _____

Were you ever injured?_____ If yes, describe_____

Who did this? (Relationship to you) _____

If sibling, what age was he/she_____

Has anyone **else** ever done this to you? No_____ Yes _____

9. Has a parent, romantic partner, or family member repeatedly ridiculed you, put you down, ignored you, or told you were no good?

No _____ Yes _____ If yes, at what age? _____

If yes, how many times? 1 _____, 2-4 _____, 5-10 _____, more than 10_____

If repeated, over what period? 6 mo. or less _____, 7 mos.- 2 yrs. _____, more

than 2 yrs. but less than 5 yrs. _____ , 5 yrs. or more _____.

Who did this? (Relationship to you) _____

If sibling, what age was he/she_____

Has anyone **else** ever done this to you? No_____ Yes _____

10. Other than the experiences already covered, has anyone ever <u>threatened</u> you with a weapon like a knife or gun?

No _____ Yes _____ If yes, at what age? _____

If yes, how many times? 1 _____ , 2-4 _____ , 5-10 _____, more than 10_____

If repeated, over what period? 6 mo. or less _____, 7 mos.- 2 yrs. _____, more

than 2 yrs. but less than 5 yrs. _____, 5 yrs. or more _____.

Describe nature of threat _____

Who did this? (Relationship to you) _____

Has anyone **else** ever done this to you? No_____ Yes _____

11. Have you ever been present when another person was killed? Seriously injured? Sexually or physically assaulted?

No _____ Yes _____ If yes, at what age? _____

Please describe what you witnessed _____

Was your own life in danger? _____

12. Have you ever been in any other situation where you were seriously injured or your life was in danger (e.g., involved in military combat or living in a war zone)?

No_____ Yes_____

If yes, at what age? _____ Please describe. _____

_____

13. Have you ever been in any other situation that was extremely frightening or horrifying, or one in which you felt extremely helpless, that you haven't reported?

No_____ Yes_____

If yes, at what age? _____ Please describe. _____

_____

The interviewer should determine if the respondent is reporting the same incident in multiple questions, and should record it in the most appropriate category.

Goodman, Corcoran, Turner, Yuan, & Green, 1998

Appendix G
Demographic Survey

Demographic Questionnaire

Please respond to the following questions by providing the most appropriate response.

Gender: Male Female

Ethnicity: African American Caucasian Asian/Pacific Islander
Latino South American Native American Other: _____

Age: _____

What academic year are you currently?

Freshman
Sophmore
Junior
Senior

Not including religious organizations, how many civic or community organizations are you a part of (ex: clubs, student leadership organizations, etc.).

None
1-2
3-4
5 or more

What, if any, is your religious preference?

Protestant
Catholic
LDS / Mormon
Jewish
Other
No Preference / No religious affiliation
Prefer not to say

How active do you consider yourself in the practice of your religious preference?

Very active
Somewhat active
Not very active
Not active
Does not apply / Prefer not to say

Marital status

Are you:

Married
Divorced
Widowed
Separated
Never been married
A member of an unmarried couple

Please circle from the following list any diagnosis you have previously received (from doctor or psychologist).

Major Depression

Posttraumatic Stress Disorder (PTSD

Attention Deficit Hyperactivity Disorder (ADHD)

Anxiety Disorder

Impulse Control Disorder

Substance Abuse/Dependence

Autism Spectrum Disorder

Schizophrenia

Oppositional Defiant Disorder (ODD)

Bipolar Disorder

Do you currently use alcohol? Yes No

If yes, please answer the following questions by placing an "X" in the box that best represents the frequency of each behavior:

Question:	Never	Less than monthly	Monthly	Weekly	Three time per week or more
How often do you drink alcohol?					
How often do you have more than 6 drinks?					
How often do you have guilt or					

remorse about drinking?					
How often have you been unable to remember the night before due to drinking?					

Please indicate below the number of drinks you typically consume when drinking.

0-2 drinks
3-5 drinks
6-10 drinks
11-15 drinks
16-20 drinks
20 or more drinks

Do you currently use illegal/street drugs or prescription drugs not prescribed for you by a medical doctor? Yes No

If yes, please list the drugs you take: _____

How often? _____ How much? _____

Have you ever experienced a concussion or traumatic brain injury? Yes No

If yes, did you experience unconsciousness? Yes No

Appendix H
Perceived Stress Scale

Perceived Stress Scale

The questions in this scale ask you about your feelings and thoughts **during the last month**. In each case, you will be asked to indicate by circling *how often* you felt or thought a certain way.

Name _____Date _____

Age _____ Gender (*Circle*): **M F** Other _____

0 = Never 1 = Almost Never 2 = Sometimes 3 = Fairly Often 4 = Very Often

1. In the last month, how often have you been upset?
because of something that happened unexpectedly?..... ….**0 1 2 3 4**
2. In the last month, how often have you felt that you were unable?
to control the important things in your life?..... **0 1 2 3 4**
3. In the last month, how often have you felt nervous and "stressed"? **0 1 2 3 4**
4. In the last month, how often have you felt confident about your ability
to handle your personal problems?..... …**0 1 2 3 4**
5. In the last month, how often have you felt that things
were going your way?..... …**0 1 2 3 4**
6. In the last month, how often have you found that you could not cope
with all the things that you had to do? ..... …**0 1 2 3 4**
7. In the last month, how often have you been able
to control irritations in your life?..... …**0 1 2 3 4**
8. In the last month, how often have you felt that you were on top of things?..... **0 1 2 3 4**
9. In the last month, how often have you been angered
because of things that were outside of your control? ..... ….**0 1 2 3 4**
10. In the last month, how often have you felt difficulties
were piling up so high that you could not overcome them?..... ….**0 1 2 3 4**

References

The PSS Scale is reprinted with permission of the American Sociological Association, from Cohen, S., Kamarck, T., and Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24*, 386-396.

Appendix I
Beck Depression Inventory, Second Edition

BDI-II

Date:

Name: _____ Marital Status: _____ Age: _____ Sex: _____

Occupation: _____ Education: _____

Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the **one statement** in each group that best describes the way you have been feeling during the **past two weeks, including today**. Circle the number beside the statement you have picked. If several statements in the group seem to apply equally well, circle the highest number for that group. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. Sadness
- 0 I do not feel sad.
- 1 I feel sad much of the time.
- 2 I am sad all the time.
- 3 I am so sad or unhappy that I can't stand it.

2. Pessimism
- 0 I am not discouraged about my future.
- 1 I feel more discouraged about my future than I used to be.
- 2 I do not expect things to work out for me.
- 3 I feel my future is hopeless and will only get worse.

3. Past Failure
- 0 I do not feel like a failure.
- 1 I have failed more than I should have.
- 2 As I look back, I see a lot of failures.
- 3 I feel I am a total failure as a person.

4. Loss of Pleasure
- 0 I get as much pleasure as I ever did from the things I enjoy.
- 1 I don't enjoy things as much as I used to.
- 2 I get very little pleasure from the things I used to enjoy.
- 3 I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings
- 0 I don't feel particularly guilty.
- 1 I feel guilty over many things I have done or should have done.
- 2 I feel quite guilty most of the time.
- 3 I feel guilty all of the time.

6. Punishment Feelings
- 0 I don't feel I am being punished.
- 1 I feel I may be punished.
- 2 I expect to be punished.
- 3 I feel I am being punished.

7. Self-Dislike
- 0 I feel the same about myself as ever.
- 1 I have lost confidence in myself.
- 2 I am disappointed in myself.
- 3 I dislike myself.

8. Self-Criticalness
- 0 I don't criticize or blame myself more than usual.
- 1 I am more critical of myself than I used to be.
- 2 I criticize myself for all of my faults.
- 3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes
- 0 I don't have any thoughts of killing myself.
- 1 I have thoughts of killing myself, but I would not carry them out.
- 2 I would like to kill myself.
- 3 I would kill myself if I had the chance.

10. Crying
- 0 I don't cry anymore than I used to.
- 1 I cry more than I used to.
- 2 I cry over every little thing.
- 3 I feel like crying, but I can't.

_____ Subtotal Page 1

Continued on Back

THE PSYCHOLOGICAL CORPORATION®
Harcourt Brace & Company
SAN ANTONIO
Orlando • Boston • New York • Chicago • San Francisco • Atlanta • Dallas
San Diego • Philadelphia • Austin • Fort Worth • Toronto • London • Sydney

0154018392

11. Agitation

0 I am no more restless or wound up than usual.
1 I feel more restless or wound up than usual.
2 I am so restless or agitated that it's hard to stay still.
3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

0 I have not lost interest in other people or activities.
1 I am less interested in other people or things than before.
2 I have lost most of my interest in other people or things.
3 It's hard to get interested in anything.

13. Indecisiveness

0 I make decisions about as well as ever.
1 I find it more difficult to make decisions than usual.
2 I have much greater difficulty in making decisions than I used to.
3 I have trouble making any decisions.

14. Worthlessness

0 I do not feel I am worthless.
1 I don't consider myself as worthwhile and useful as I used to.
2 I feel more worthless as compared to other people.
3 I feel utterly worthless.

15. Loss of Energy

0 I have as much energy as ever.
1 I have less energy than I used to have.
2 I don't have enough energy to do very much.
3 I don't have enough energy to do anything.

16. Changes in Sleeping Pattern

0 I have not experienced any change in my sleeping pattern.
1a I sleep somewhat more than usual.
1b I sleep somewhat less than usual.
2a I sleep a lot more than usual.
2b I sleep a lot less than usual.
3a I sleep most of the day.
3b I wake up 1–2 hours early and can't get back to sleep.

17. Irritability

0 I am no more irritable than usual.
1 I am more irritable than usual.
2 I am much more irritable than usual.
3 I am irritable all the time.

18. Changes in Appetite

0 I have not experienced any change in my appetite.
1a My appetite is somewhat less than usual.
1b My appetite is somewhat greater than usual.
2a My appetite is much less than before.
2b My appetite is much greater than usual.
3a I have no appetite at all.
3b I crave food all the time.

19. Concentration Difficulty

0 I can concentrate as well as ever.
1 I can't concentrate as well as usual.
2 It's hard to keep my mind on anything for very long.
3 I find I can't concentrate on anything.

20. Tiredness or Fatigue

0 I am no more tired or fatigued than usual.
1 I get more tired or fatigued more easily than usual.
2 I am too tired or fatigued to do a lot of the things I used to do.
3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex

0 I have not noticed any recent change in my interest in sex.
1 I am less interested in sex than I used to be.
2 I am much less interested in sex now.
3 I have lost interest in sex completely.

14 15 16 17 18 19 20 A B C D E

Appendix J
State Trait Anxiety Inventory

mind garden

SELF-EVALUATION QUESTIONNAIRE

STAI Form Y-1

Please provide the following information:

Name_____ Date_____ S_____

Age_____ Gender (*Circle*) **M** **F** T_____

DIRECTIONS:

A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you feel *right* now, that is, *at this moment*. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

	NOT AT ALL	SOMEWHAT	MODERATELY SO	VERY MUCH SO
1. I feel calm	1	2	3	4
2. I feel secure	1	2	3	4
3. I am tense	1	2	3	4
4. I feel strained	1	2	3	4
5. I feel at ease	1	2	3	4
6. I feel upset	1	2	3	4
7. I am presently worrying over possible misfortunes	1	2	3	4
8. I feel satisfied	1	2	3	4
9. I feel frightened	1	2	3	4
10. I feel comfortable	1	2	3	4
11. I feel self-confident	1	2	3	4
12. I feel nervous	1	2	3	4
13. I am jittery	1	2	3	4
14. I feel indecisive	1	2	3	4
15. I am relaxed	1	2	3	4
16. I feel content	1	2	3	4
17. I am worried	1	2	3	4
18. I feel confused	1	2	3	4
19. I feel steady	1	2	3	4
20. I feel pleasant	1	2	3	4

Published by MIND GARDEN 1690 Woodside Road Suite 202, Redwood City California 94061 (650) 261-3500

STAIE-AD Scoring Key

Appendix K
Informed Consent Form

Informed Consent Form

Indiana University of Pennsylvania

Title of Project: Neuropsychological functioning in college students: Investigating the effects of stress and trauma on cognitive ability.

Student Researcher: Leslie Smith Varner
Doctoral Candidate, Clinical Psychology
Uhler Hall
1020 Oakland Ave
Indiana, PA 15705

Dissertation Chair: William Meil, Ph.D.
Faculty, Psychology Dept.
Uhler Hall
1020 Oakland Ave.
Indiana, PA 15705

You have been invited to participate in this research study. The following will provide additional details about the study to assist you in making an informed decision regarding if you would or would not like to participate. Eligibility to participate in this study is determined by your enrollment in an introductory psychology course. Please do not hesitate to ask any questions regarding this form or the research study.

The purpose of this study is to determine the effects, if any, of stressful or traumatic experiences on neuropsychological functioning among college students, as determined through a series of brief, non-invasive tests provided in a paper and pencil format.

For this research study, you will be asked to sign two Informed Consent Forms (you will retain one copy for your records), complete three neuropsychological assessments administered by a research assistant, and six brief self-report surveys. Participation in this study will take approximately 30 minutes. Once you have completed the study, you will be eligible to receive extra course credit.

All information and data collected will be kept confidential. Only an impersonal identification number, not your name, will be attached to the information. Data will be kept in a locking filing cabinet on university property and will be destroyed after the completion of the study. Your participation in this research study is voluntary. If you do agree to participate, you have the right to stop participating at any time or to decline answering any questions. Withdrawal from the study will involve no penalty or loss of benefits. If you chose to discontinue participation, all information provided by in the context of this research study will be destroyed. Information provided will only be considered in the context of the present study and will not be utilized to identify individuals eligible for psychological services.

While procedures relating to research participation are not anticipated to pose risk beyond what would be encountered in everyday life, the questions and/or statements contained in the instruments may be sensitive in nature. If you feel discomfort at any time, you can decline to

answer any question or choose to discontinue participation. Benefits associated with participation include the opportunity to contribute to the understanding of the experience of stress and trauma among college students.

If you are willing to participate in this study, please sign the statement provided below and submit it to the research assistant administering the survey. In addition, please collect a blank copy of the form for your records. These will be provided by the research assistant. Following participation, you will be provided with a form that will provide additional information including who to contact should you be interested in obtaining the results of the study and with referral sources for counseling should any issues arise from participation.

VOLUNTARY CONSENT FORM:

I have read and understand the information on the form and I consent to volunteer to be a subject in this study. I understand that my responses are completely confidential and that I have the right to withdraw at any time. I have received an unsigned copy of this informed Consent Form to keep in my possession.

_____

Participant Name

_____ _____

Participant Signature Date

I certify that I have explained to the above individual the nature and purpose, the potential benefits, and possible risks associated with participating in this research study, have answered any questions that have been raised, and have witnessed the above signature.

_____ _____

Principal Investigator's Signature Date

This project has been approved by the Indiana University of Pennsylvania Institutional Review Board for the Protection of Human Subjects (Phone: 724/357-7730).

Debriefing Form

Neuropsychological functioning in college students:
Investigating the effects of stress and trauma on cognitive ability.

Stressful and traumatic experiences are an unfortunate occurrence in the lives of many individuals. Evidence suggests that frequent or intense exposure to these experiences can contribute to difficulties in completing certain cognitive tasks, like shifting attention, concentration, and the ability to be flexible in switching among responses. On addition, these experiences have also been correlated with depression and anxiety symptoms. These difficulties may have implications in everyday functioning, however, college students appear to be somewhat resilient to these effects. The tests administered were designed to evaluate the relationship among previous or current stressful or traumatic experiences and neuropsychological functioning and symptoms of depression and anxiety.

As stated earlier, your responses to all of the questionnaires will be absolutely confidential. Your name will not be associated in any way with your responses and the researcher will not be evaluating the results of the surveys until the completion of the study.

Your participation in this study is greatly appreciated. If you'd be interested in obtaining a copy of the results once the study is complete, you may contact the primary researcher, Leslie Smith Varner at vhhp@iup.edu.

As some of the measures have inquired about potentially upsetting past or present experiences, some individuals may have concerns relating to their own well-being or their own abilities in relation to completion of the tests administered today. If this is the case, the following resources are available to you, should you be interested in obtaining counseling services. Please note, you will be responsible for contacting these resources, setting up appointments, and paying necessary fees. The IUP Student Counseling Center is free to students.

IUP Student Counseling Center: Suites on Maple East, G31, 901 Maple Street
Indiana, PA 15705
Phone: 724-357-2621

Center for Applied Psychology: 210 Uhler Hall
IUP
Indiana, PA 15705
Phone: (724) 357-6228

Indiana County Guidance Center: 699 Philadelphia St.

Indiana, PA 15705
(724) 465-5576

In the event that you have any complaints, concerns, or questions about this research, please feel free to contact the faculty sponsor of this research, William Meil, Ph.D. (meil@iup.edu).

Thank you very much for participating!