5-2020

The Relationship Between Adverse Childhood Experiences, Acute Stress, and Working Memory

Marissa R. Jones
East Tennessee State University

The Relationship Between Adverse Childhood Experiences, Acute Stress, and Working Memory

_____

A dissertation

presented to

the faculty of the Department of Psychology

East Tennessee State University

In partial fulfillment

of the requirements for the degree

Doctor of Philosophy in Psychology

_____

by

Marissa R. Jones

May 2020

_____

Dr. Eric W. Sellers, Chair

Dr. Wallace Dixon

Dr. Julia Dodd

Dr. Matthew McBee

Dr. David Ryan

Keywords: Working Memory, Acute Stress, ACEs, EEG, Theta, Alpha

ABSTRACT

The Relationship Between Adverse Childhood Experiences, Acute Stress, and Working Memory

by

Marissa Jones

Working memory (WM) is a crucial component of cognitive function that affects learning, reasoning, and problem solving, all of which are important for daily functioning. Therefore, addressing factors that can impact working memory, such as stress, are incredibly relevant to understanding WM efficiency. WM is an important component of higher order cognitive function and high WM capacity has been shown to be important for academic and occupational performance. Thus, understanding the relationship between stress-related factors and WM could aid in identifying strategies to mitigate the deleterious effects of stress on working memory. Although some previous research has indicated a negative impact of acute stress on WM, other research has indicated no impact or even a positive impact of stress on WM. As the relationship between acute stress and WM is mixed, examining other stress-related factors may provide further insight into the relationship. The current study examines how adverse childhood experiences (ACEs) and acute stress influence WM, and how frontal theta and alpha activity are affected by WM task demands. Participants completed a working memory task while their EEG was recorded. Participants then completed the PANAS to assess their current emotional state. Following the PANAS, participants viewed a stressful or neutral video as an acute stress induction, followed by a second PANAS to ensure effectiveness of stress induction. Participants then completed the WM task a second time. Finally, the participants completed the ACEs

questionnaire. Bayesian linear mixed effects models were used to examine the relationships between ACEs, acute stress, WM, and frontal theta and alpha frequencies. Findings suggest there is not enough evidence to support a relationship between acute stress, ACEs, WM, and WM-related theta and alpha. While the current study did not reveal a relationship, future research should explore how acute stress and exposure to specific stressors during childhood could explain individual differences in WM.

DEDICATION

For my husband Brandon.

Thank you for all of your love and support, and for encouraging me to see this through.

ACKNOWLEDGEMENTS

I would like to thank the members of my dissertation committee, David Ryan, Matt McBee, Wallace Dixon, and Julia Dodd for their expertise and feedback in designing and executing this research. I would especially like to thank my mentor Dr. Eric Sellers, who has been incredibly patient with me through this process, and who has provided me with excellent support and guidance. I have grown both as a researcher and as a person from his mentorship.

I would also like to thank the faculty in the Department of Psychology who have all contributed to my training. Thank you for giving me so many opportunities. Finally, I would like to thank both the students who have worked in in the BCI lab, and those who have participated in our research.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

Chapter 1. Introduction

As an adaptive and dynamic structure, the development of the brain can shape and be shaped by experience. An organism's ability to acquire and manipulate information through interactions with the environment can impact future experiences. The combined process of synaptic pruning and synaptogenesis during brain development serves to fine-tune networks and communication to better adapt to the environment. With new experiences that can influence developmental trajectories, the body's response to these experiences are regulated through the process of allostasis. The allostatic process attempts to maintain homeostasis, which involves communication between the brain and the body's stress response systems.

For example, experiencing a state of stress, in response to either internal or external events, is associated with the activation of the sympathetic nervous system (SNS) and the limbic-hypothalamic-pituitary-adrenal (HPA) axis through neuroendocrine systems (Sapolsky & Meaney, 1986; Teicher, Andersen, Polcari, Anderson, & Navalta, 2002; Tsigos & Chrousos, 2002). The activation of this stress response system has been linked with changes in heart rate, metabolic rate, blood pressure, and alertness (Bellis & Zisk, 2014; Chrousos & Gold, 1992). This allostatic process is necessary to be able to continuously adapt to constant changes in the environment (Frodl & O'Keane, 2013).

Although this adaptability is one of the brain's greatest strengths, the adaptation through experience has the potential to lead to detriments in brain development. Exposure to excessively stressful or threatening stimuli is associated with increased allostatic load (Frodl & O'Keane, 2013; Juster et al., 2010; McEwen, 2000). Experiencing severe stress and trauma during childhood, during which critical periods of development occur, have been shown to be associated with the disruption of the stress response system (Bellis & Zisk, 2014; McEwen, 2000; Tsigos &

Chrousos, 2002). While the stress response can be adaptive and beneficial for reacting to a stressful event, prolonged stress exposure or severity of stress is associated with long-lasting impacts on brain development and future reactions to stress (Lupien et al., 2009; McEwen, 2007).

The activation of the human stress response system can result in the secretion of cortisol. It has been demonstrated that the secretion of cortisol is associated with changes in several brain regions, specifically the prefrontal cortex (PFC) and hippocampus (Bellis & Zisk, 2014; Diamond, Fleshner, Ingersoll, & Rose, 1996; Qin et al., 2012; Qin, Hermans, van Marle, Luo, & Fernández, 2009). The communication between these two structures appears to be involved in memory functioning. The hippocampus is considered to be a crucial structure for declarative memory encoding and consolidation (Oei, Everaerd, Elzinga, van Well, & Bermond, 2006), and the retrieval of declarative memory appears to be mediated by the PFC (Buckner & Wheeler, 2001; Oei et al., 2006; Ranganath, Johnson, & D'Esposito, 2003; Simons & Spiers, 2003). It is widely acknowledged that the PFC is a crucial structure that is linked to higher-order, complex cognition such as reasoning, planning, and problem solving (Levy & Goldman-Rakic, 2000; Roberts & Pennington, 1996). The dorsolateral PFC specifically is considered to be involved in the planning and execution of goal-directed behaviors (McEwen & Morrison, 2013) and is implicated as a "top down" influencer of executive functions (Miller, 2000). As these structures have the potential to be affected by stress or high cortisol levels, there may be a relationship between the experience of an acute stressor and memory impairment.

Memory functioning has been broken down into several systems, each related to a different function in processing information. The working memory (WM) system has been identified as a set of mechanisms that involve taking integrated information that has been

attended to and manipulating the information to complete a task or achieve a goal (Luck & Vogel, 1997). A relationship between stress exposure and memory impairment, specifically in the form of decreased WM performance, has been demonstrated in several studies (Gärtner, Rohde-Liebenau, Grimm, & Bajbouj, 2014; Luethi, Meier, & Sandi, 2009; Lupien, Gillin, & Hauger, 1999; Oei et al., 2006; Schoofs, Preuß, & Wolf, 2008). For example, Gärtner and colleagues (2015) investigated changes in WM-related frontal theta activity and cortisol on the n-back task in a sample of 31 males. All participants experienced both a neutral and stress condition by being shown either a series of video clips with neutral content or stressful content. Following each video clip within the two conditions, participants completed the n-back task with 3 levels of difficulty. The findings of Gärtner and colleagues (2015) indicated a decrease in WM-related frontal theta related to the stress condition, which was supported by increased cortisol levels associated with the stress induction. Their findings also demonstrated that behavioral performance on the n-back task was impaired at task difficulty levels that indicated decreases in frontal theta activity. Therefore, Gärtner and colleagues (2015) suggest that this supports evidence for frontal theta in the prefrontal cortex serving to improve task performance, and that the experience of acute stress can influence frontal theta activity.

Furthermore, increased arousal and changes in cortisol levels have been shown to be associated with greater impact on WM than other types of memory, such as declarative memory (Lupien et al., 1999). Considering that WM is a higher-order and complex cognitive system (Baddeley, 1992; Baddeley, Logie, Bressi, Sala, & Spinnler, 1986; Baddeley, 2003; Dong et al., 2015; Levy & Goldman-Rakic, 2000), detriments to WM performance can potentially impact daily functioning in contexts such as academic or occupational settings. Therefore, identifying

and understanding factors that impact WM can be incredibly relevant for psychological well-being.

In addition to the relationship between acute stress induction and WM performance (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007; Qin et al., 2012, 2009 Gärtner et al., 2014) research has also indicated a strong relationship between adverse childhood experiences and cognitive functioning (Bellis & Zisk, 2014; Bick & Nelson, 2016; Hughes, Karen et al., 2017). Furthermore, it has been shown that early stressors experienced during childhood are associated with decreased WM performance when compared to participants without exposure to such experiences (Philip et al., 2016). Research conducted by Philip and colleagues (2016) examined effects of early life stressors on WM performance using the N-back task using functional magnetic resonance imaging (fMRI). In comparing 14 participants with who had experienced early life stressors to 13 participants who had not, Philips and colleagues (2016) demonstrated that participants with early life stressors had increased activation in several regions of the brain that were associated with decreased performance on the n-back task. The authors suggest that this supports previous research that has indicated increased recruitment of cognitive resources in WM tasks as difficulty increases for those who have experienced early life stressors.

The current study seeks to provide a bridge for these findings. By examining the relationship between adverse childhood experiences or life event stressors and the experience of an acute stressor, the current study may provide insight into the relationship between previous exposure to stress, the experience of an acute stressor, and WM performance. The following sections will examine how the construct of WM is defined and measured, as well as its relevance for daily functioning. This will be followed by a discussion of literature documenting the negative impact that experiencing stress can have on WM. Research indicating the cumulative,

negative impact of traumatic and adverse stressors during childhood on psychological well-being and cognitive functioning will then be addressed. Measurement of these experiences using the Adverse Childhood Experiences (ACEs) questionnaire has contributed to the understanding of the long-term impact of childhood stressors on several outcomes well into adulthood, therefore background on ACEs will be provided. These adverse experiences and stressors have consistently been shown to impact brain development, which could influence cognitive functioning and psychological well-being. Therefore, a review of the impact of adverse childhood experiences on brain development will be provided. Finally. the findings of a study examining the relationship between ACEs, acute stress, and working memory will be presented and discussed in light of previous research.

Working Memory

The temporary storage of information in working memory (WM) allows for either the manipulation of information to complete a task, or storage of information into long-term memory for future use (Baddeley, 1992; Baddeley, 2003; Dong et al., 2015; Luck & Vogel, 1997; Roux & Uhlhaas, 2014). Research examining the construct of WM has consistently demonstrated that these mechanisms are associated with temporary storage, maintenance, or retrieval of information. The WM system has been shown to be associated with several higher-order cognitive functions, such as learning, reasoning, planning, and problem solving (Baddeley, 1992; Baddeley et al., 1986; Baddeley, 2003; Dong et al., 2015; Duncan et al., 2000; Goel & Grafman, 1995; Levy & Goldman-Rakic, 2000; Prabhakaran, Rypma, & Gabrieli, 2001).

The construct of WM has been correlated with measures of general intelligence (Dempster & Cooney, 1982; Dong et al., 2015), and has even been shown to be a stronger predictor of academic performance than other intelligence measures (Alloway, 2009; Alloway &

Alloway, 2010; Dong et al., 2015). Furthermore, children who demonstrate low working memory performance have been shown to demonstrate cognitive deficits such as inattention and distractibility, and difficulties with problem solving in academic settings (Alloway, 2009; Simmering & Perone, 2013). The research findings examining this relationship indicate that efficient WM is necessary for cognitive functioning and influences several outcome measures such as the ability to perform academically.

Integrating multiple features of stimuli allows for more information to be held in working memory, which can then be manipulated during a given task (Luck & Vogel, 1997). This integration through WM is generally thought to involve a representation of past events and executive systems that sustain and transform said representation (Posner, 1994). How WM integrates and stores information has often been explored in the context of WM capacity. The amount of information that can be held in visual WM appears to be limited at a given time, with a typical limit load between 4 and 12 items or integrated objects (Cowan, 2001; Luck & Vogel, 1997; Sauseng et al., 2010). For example, Luck and Vogel (1997) asked participants to view a series of arrays displaying several simple color items (colored squares). Participants were then shown a blank delay interval for 900-ms, followed by the presentation of another array of colored squares. The participants were then asked to recall the previous array of square and determine whether the current array matched the previous array. This stimulus presentation required participants to hold several items, containing multiple features, in their WM at the same time, i.e. the shape of the items, the color of the items, the number of items, and the location of the items in the array. It was observed that participants were able to maintain WM performance for arrays that contained between 1-3 visual items. However, performance was reported to decrease systematically as the number of items to be held in WM increased between 4 and 12

(Luck & Vogel, 1997). This decrease in performance has been observed across different types of items or stimuli, including alphanumeric, spatial information, and numeric.

As task demands increase (i.e., the amount of information to be held in WM) performance accuracy on a WM task may decrease once an individual's limit is reached. It has been suggested that the amount of information that can be held in WM during a given task may be dependent upon the availability of cognitive resources (Gevins, 1997; Luck & Vogel, 1997, 2013). As a task increases in difficulty, more cognitive resources are recruited to maintain performance, and it is suggested that there is a limited amount of cognitive resources that are allocated for WM. Once the limit of cognitive resources has been reached, an individual is not able to hold all of the information in WM. When processing information sequentially and capacity has been reached, this may result in higher recall for the first few items, which is referred to as the primacy effect. Or, it may result in a higher recall for the last few items, which is referred to as the recency effect (Stephane et al., 2010).

Having demonstrated a relationship between working memory (WM) and other higher-order cognitive functions, understanding individual differences in WM performance and factors that may be related to an individual's WM performance could be used to understand outcome measures such as academic or occupational performance (Alloway & Alloway, 2010; Alan Baddeley, 2010). Individuals who have lower WM capacity may be required to use more cognitive resources when performing an easy task, as compared to individuals with higher WM capacity. In contrast, individuals with higher WM capacity are suggested to require less effort to complete a task, as compared to individuals with lower WM capacity (Gevins, 1997).

Working Memory and EEG: Alpha and Theta Frequency

The identification and understanding of cognitive mechanisms such as WM have greatly benefited from the use of electroencephalography (EEG) technology. Using EEG, researchers are able to link neurophysiological changes to cognitive psychological constructs (Wolfgang Klimesch, 1996). The EEG detects the oscillations that are formed by synchronous firing of cell assemblies, which have been implicated in communication between brain regions (Klimesch, 1996). EEG provides information regarding the resting state of the brain, synchrony between regions (coherence) or spectral changes in response to a cognitive event (event-related synchronization/desynchronization). Frequency refers to the number of oscillations (or cycles) within a given time period, typically one second (Loo & Barkley, 2005). Oscillatory activity may be a general mechanism for the coordination of activity within neural circuits, and disruptions of synchronization among neurons could impact a wide range of cognitive processes (Kim et al., 2013). Large neural networks have been suggested to be reflected in oscillations in slow frequencies, (<20 Hz), allowing for communication between brain structures that may not be within close spatial proximity (Hanslmayr, 2011).

Communication between different brain regions has been shown to be related to changes in frequency in response to an internal or external event (Klimesch, 1996; Pfurtscheller & Lopes da Silva, 1999; Pfurtscheller, Neuper, Pichler-Zalaudek, Edlinger, & Lopes da Silva, 2000). This communication may also be reflected through amplitude changes, event-related synchronization (ERS) or event-related desynchronization (ERD). An increase in frequency, or increase in frequency power, is reflected by ERS. In contrast, a decrease in frequency power, or suppression, is reflected by ERD (Pfurtscheller, 1999; Klimesch, 1996; Pfurscheller, 1982). These frequency changes can provide insight into changes in an individual's state of alertness and the dynamics of

functional network formation (Dong et al., 2015). Two commonly examined frequency bands during WM tasks are referred to as alpha (8-12Hz) and theta (4-8Hz), (Dong et al., 2015; Gevins, 1997; Roux & Uhlhaas, 2014; Sauseng et al., 2010; Scheeringa et al., 2008).

Alpha frequency has been shown to be associated with the process of region inhibition, or suppression (Clayton, Yeung, & Kadosh, 2015; Foxe & Snyder, 2011; Hanslmayr, Gross, Klimesch, & Shapiro, 2011; Herrmann & Knight, 2001; Klimesch, Doppelmayr, Russegger, Pachinger, & Schwaiger, 1998; Klimesch, 2012). When alpha is observed in a specific region of the brain, it is assumed that the brain region is not currently processing information. It is suggested that the presence of alpha in a specific brain region may be associated with early inhibition of sensory input mechanisms (Cooper, Croft, Dominey, Burgess, & Gruzelier, 2003). For example, when an individual is engaged in a visual task that does not involve processing auditory information, there would be an observed increase in alpha activity at the auditory cortices, and a decrease in alpha activity in the occipital cortex where visual information is processed (Clayton et al., 2015; Foxe & Snyder, 2011; Hanslmayr et al., 2011; Herrmann & Knight, 2001; Klimesch et al., 1998). In this situation, the auditory cortex is not necessary for processing the visual information during the task, therefore alpha functions to actively inhibit the unnecessary region. Therefore, alpha activity may be related to an active process of inhibition that prevents information that is not relevant when completing a given task.

Theta activity can be observed at the fronto-midline (FM) and is generally maximal at electrode location Fz. Increases in theta power in this region have shown to be associated with memory functions (Asada, Fukuda, Tsunoda, Yamaguchi, & Tonoike, 1999; Bastiaansen & Hagoort, 2003; Cavanagh & Frank, 2014; Clayton et al., 2015; Sauseng et al., 2010). An increase in FM theta ERS can be observed during a task that requires more attention (Gevins, 1997). In

the context of WM tasks, as memory load increases (i.e., more information is held in WM), an increase in frontal theta activity is observed (Klimesch, 2012; Sauseng et al., 2010; Scheeringa et al., 2008). Therefore, this phasic FM theta may be modulated by task-related requirements (McNaughton, Flanagan, & Kirk, 2008). Particularly, FM theta may be modulated by tasks that are considered to involve sustained, internally directed cognition (Gevins, 1997; Hsieh & Ranganath, 2014; Raghavachari et al., 2001)

While EEG is considered to have high temporal resolution, it has very low spatial resolution in comparison to other neuroimaging technologies such as fMRI or PET (Yonelinas, 2013). Due to this limitation in spatial resolution, identifying the neural generators of a given frequency has proven to be a challenge in psychophysiological research (Lagerlund, 1982; Nunez & Srinivasan, 2006). Frontal and midline theta activity has been suggested to originate from activation of the prefrontal cortex (PFC) and anterior cingulate cortex (ACC), which reflect attentional control and performance monitoring processes (Asada et al., 1999; Cavanagh & Frank, 2014; Cohen, 2011; Gärtner et al., 2014; Onton, Delorme, & Makeig, 2005; Roberts & Pennington, 1996).

Within cognitive psychology, WM and long-term memory have historically been considered distinct constructs (Yonelinas, 2013). This is primarily due to lesion cases such as the well-known case of patient HM (Levy & Murdock, 1968; Scoville & Milner, 2000), in which damage to the medial temporal lobe resulted in impaired long-term memory functioning, while other cognitive functioning remained intact. Damage in such cases has primarily been used to make the case for the crucial role of the hippocampus in the formation of new memories (i.e. transfer to long-term memory).

In a study conducted by Drachman and Arbit (1966) with patients with memory deficits, including patient HM, participants were instructed to hear and repeat back digit strings of increasing length. In this digit span task, participants were presented each string of digits until they repeated the whole string correctly (Drachman & Arbit, 1966; Jeneson & Squire, 2012). Then, the string would increase by one digit. For participants with no damage, the first errors were consistently made for strings with eight digits. However, when allowed to repeat strings until correct, participants with no damage were able to recall up to 20 digits. For patients with damage to the medial temporal lobe (MTL), difficulties in performance were observed with each increase in string size. Furthermore, in the case of patient H.M., once a certain limit had been reached (six digits), he was unable to recall the next string size despite many repetitions of the same string (Drachman & Arbit, 1966; Jeneson & Squire, 2012). Thus, it appears that with these patients who are able to retrieve previous memories from long-term memory, they were no longer able to store new memories into long-term memory to be retrieved for later use. It is suggested that one may interpret these findings as evidence that once WM capacity has been reached, WM performance may then also depend on long-term memory functioning (Jeneson & Squire, 2012).

In recent years it has been shown that in certain conditions, patients who have hippocampal damage display deficits in working memory and perception in addition to long-term memory deficits (Cowell et al., 2010; Lee et al., 2012; Ranganath & Blumenfeld, 2005). Thus, the relationship between the hippocampus, long-term memory, and WM remains an area of interest for research. Therefore, in recent years there has been growing interest in examining the relationship between recollection of information, (i.e. retrieval of information from long-term memory) and WM functioning. There has been a number of inconsistent findings that

demonstrate that performance on some WM tasks benefit from recollection or familiarity of stimuli, which are processes that are thought to involve retrieval processes through long-term memory (Yonelinas, 2013).

Therefore, during a WM task, as the task becomes more difficult and WM capacity is reached, information may then need to be stored into long-term memory. WM performance with large amounts of information may then involve retrieval from long-term memory that is associated with hippocampal functioning. This process may also involve attentional and performance monitoring associated with the prefrontal cortex, suggesting that increased communication between the hippocampus and the prefrontal cortex may be necessary for optimal WM performance as cognitive load increases and WM capacity has been reached. Therefore, measurement of frontal and midline theta activity may be related to this communicative process, reflecting the changes in communication during WM once a capacity has been reached. As the task becomes more difficult and the WM capacity is reached, an increase in midline theta activity may reflect that more cognitive resources are being recruited to help maintain WM performance. Once the task becomes too difficult to maintain performance, even with the recruitment of more cognitive resources, midline theta activity may decrease. This may reflect that as the task is too difficult to complete, that recruitment of more cognitive resources is not necessary, as it no longer assists in completing the task.

Impact of Acute Stress on Working Memory

As WM has been shown to be associated with higher order cognitive functions, such as problem solving and learning, WM is important in domains such as academic or occupational settings (Alloway & Alloway, 2010; Baddeley, 2010). Research examining potential influencers on WM have identified a relationship between acute and chronic stress and WM performance

(Gärtner et al., 2014; Mizoguchi et al., 2000). As an organism attempts to maintain homeostasis, this homeostasis is constantly confronted by adverse forces, or stressors, that threaten the maintenance of homeostasis (Tsigos, 2002). Allostasis, which is the process of regulating homeostasis, allows for an organism to respond and adapt to stressors in the environment. The process of allostasis involves coriticotrophin-releasing hormones (CRH), the locus-coeruleus-norepinephrine (LC-NE)/autonomic systems, and the limbic-hypothalamic-pituitary-adrenal (HPA) axis (Chrousos, 1992). The communication between these systems allows for humans to adapt to constant changes, including stressors, in the environment (Frodl & O'Keane, 2013)

The HPA axis is a system that involves hormonal communication between the hypothalamus and the pituitary gland located in the brain, and the adrenal gland located in the kidneys. When an acute stressor is processed by sensory systems, CRH results in the secretion of cortisol, transmitting signals to the prefrontal cortex (PFC), hypothalamus, and hippocampus (Bellis & Zisk, 2014). Depending on the emotional relevance of the stressor, the LC-NE sympathetic system, releases NE throughout the brain, which causes increased arousal (Tsigos & Chrousos, 2002). This process also activates the HPA axis and sympathetic nervous system, which causes glucose, heart rate, and blood pressure to increase (Chrousos & Gold, 1992).

The advantageous qualities of allostasis allows for flexible adaptability to internal or external stressors. However, when responding to an acute stressor, these stress response systems are intended to be exercised for only a limited duration (Tsigos, 2002, Chrousos 1992). Prolonged activation of the stress system due to chronic stress can lead to extremes of high or low sensitivity of the system, causing hyper-arousal or hypervigilance in an individual (Chrousos, 1992). Furthermore, the HPA axis has been linked to growth and immunity, and

prolonged activation of the HPA axis has been shown to be related to detriments in immune response, as well as overall growth and development (Chrousos & Gold, 1992).

Evidence spanning decades of research has indicated that damage to the hippocampus may be associated with memory impairments, both in human and animal research (Diamond, Park, Heman, & Rose, 1999; Diamond et al., 1996). The effects of stress, specifically, on memory that is dependent on hippocampus and functioning follow an inverted U-function (Yerkes & Dodson, 1908). Moderate levels of stress exposure may facilitate memory functions, but high levels of stress exposure may lead to impairment of memory functioning (Luethi, 2009; Oei 2006; Schoofs 2008). This inverted U-shaped relationship between stress and cognitive performance has been further supported by an inverted U-shaped dose-response relationship with cognitive performance and LC-NE activity, glucocorticoids as reflected in elevations in cortisol levels, and catecholamines such as dopamine (DA), (Qin 2009; Arnsten 2004; Arnsten 2007; Aston-Jones 2005; Aston-Jones 1999), which all have been shown to impact the PFC. This neuro-modulation due to acute stress has been shown impact WM processing in the dorsolateral PFC, as increased presence of stress-sensitive catecholamines result in the suppression of neural firing (Arnsten, 2007; Arnsten & Li, 2005; Porcelli et al., 2008; Qin et al., 2012).

Possibly due to this observed inverted U-shaped relationship between acute stress and cognition, findings of the impact of acute stress specifically on WM performance have been mixed (Porcelli, 2008). Some stress manipulations have demonstrated negative effects (Porcelli et al., 2008; Kirschbaum, Wolf, May, Wippich, & Hellhammer, 1996; Kuhlmann, Piel, & Wolf, 2005; Oei et al., 2006; Patil, Apfelbaum, & Zacny, 1995), whereas others have demonstrated no impact on WM performance (Domes et al., 2002; McMorris et al., 2006; Smeets et al., 2006).

Therefore, understanding the relationship between acute stress, WM, and differences in environmental contexts could benefit from further research.

Impact of Acute Stress on Working Memory-Related Theta Activity

The impact of neurochemical changes associated with stress induction on behavioral measure of WM performance has received some support. However, the examination of the relationship between stress and frontal theta activity related to WM has received limited attention. Gärtner et al. (2014) investigated the effects of acute psychological stress on WM-related frontal theta activity. Noting the close relationship between frontal theta activity and WM (Gevins et al., 1997), the synchronization of theta activity reflecting network connections to the prefrontal cortex (Anderson et al., 2010; Cohen, 2011), and the impact of stress on the prefrontal cortex (Arnsten, 2007, 2009; Arnsten et al., 1999; Arnsten & Li, 2005), Gartner et al. (2014) suggests that acute stress should be associated with reduced synchronization of frontal theta activity. Using male participants, Gärtner et al. (2014) measured WM performance and frontal theta under acute psychological stress. Baseline measures of WM and frontal midline theta were collected during the n-back task. Samples of cortisol were collected and subjective measures using the Positive Affect Negative Affect Schedule (PANAS) were used to examine changes in stress response. Participants completed a stress condition and a neutral condition, which were counter-balanced, separated by a 20 minute break. For each condition, the participants completed a WM task followed by a video that contained either stressful or neutral content. For their stress induction, the researchers used a clip from a French movie depicting violent acts against other humans. This allowed for Gärtner et al. (2014) to examine the relationship between experience of acute stress and WM-related frontal theta.

Gärtner et al. (2014) observed the expected decrease in frontal theta activity associated with decreases in WM performance that were observed on more difficult trials. During tasks following the stress induction, Gärtner et al. (2014) observed stress related decreases in WM recall performance during high workload WM trials, as well as decreases in frontal theta. This indicated that the experience of acute stress is associated with an observed decrease in WM-related theta. According to Gärtner et al. (2014), these findings support that frontal theta activity may reflect the functioning of prefrontal cortex network connections, which can be disrupted by experience of acute stressors.

Adverse Childhood Experiences

Examining the relationship between exposure to abuse/neglect and household dysfunction experienced during childhood and several health outcomes has increased in interest over the years. Particularly, the development of the Adverse Childhood Experiences (ACE) questionnaire by Felitti et al. in 1998 has served as a catalyst. The initial ACE study (Felitti et al., 1998) assessed the negative, long-term impact of a range of abuse exposure during childhood. The long term outcomes that were assessed range from potential disease risk factors, overall quality of life, use of health care services, and mortality outcomes in adults. The ACE questionnaire utilizes items that were pulled from other scales to evaluate several types of abuse or dysfunction that could occur in the household, such as psychological and physical abuse, exposure to substance abuse, whether other family members exhibited signs of mental illness, if the mother figure in the family experienced violent treatment, or if any family members engaged in criminal behavior. The development of this questionnaire allowed researchers to inquire about participants' experiences prior to the age of 18. The measurement of these experiences allowed for Felitti et al. (1998) to examine the long-term impact that adverse childhood experiences,

reported retrospectively, could potentially have on health outcomes well into adulthood. The authors' findings indicated a graded relationship between exposure to ACEs and negative health outcomes such as illness and risk-taking behaviors. In other words, as exposure to ACEs increased, the chronic illness and risk-taking behaviors also increased. This graded relationship between ACE exposure and negative outcomes has consistently been observed over consecutive years of research (Bick & Nelson, 2016; Hughes et al., 2017; Koss et al., 2003; Mersky, Topitzes, & Reynolds, 2013).

Impact of Adverse Experiences on Brain Development

Prior to the development of the ACEs questionnaire, the relationship between exposure to early stress in childhood, development, and health outcomes had gained overwhelming empirical support. The experience of trauma or chronic early life stress during development is thought to have maladaptive impact on brain development, as it is associated (Bellis & Zisk, 2014; De Bellis, 2001; Teicher et al., 2002). In human and animal research, exposure to severe stress during childhood can result in the disruption of stress response regulation across the lifespan (Lupien, McEwen, Gunnar, & Heim, 2009; McEwen, 2007; Meaney, 2001; Plotsky & Meaney, 1993; Sánchez, Ladd, & Plotsky, 2001).

Research has indicated that children who have more adverse experiences have increased risk for several cognitive delays, memory problems, and learning difficulties that have the potential to negatively impact academic performance and adjustment to the school environment (Anda et al., 2006; Levy & Goldman-Rakic, 2000). Furthermore, studies that have examined early childhood trauma and maltreatment have indicated that compared to children who experienced no maltreatment, children who experienced maltreatment may have lower IQs, deficits in language, and problems with academic performance (Bellis & Zisk, 2014; Carrey,

Butter, Persinger, & Bialik, 1995; Culp, Little, Letts, & Lawrence, 1991; Eckenrode, Laird, & Doris, 1993; McFadyen & Kitson, 1996; Trickett & McBride-Chang, 1995). There are not only observed problems in cognitive functioning, but also increased risk for developing various psychopathologies, such as depression, PTSD, borderline personality disorder, and substance use disorder (Philip et al., 2016; Teicher, 2006; Teicher & Samson, 2013). Epidemiological research has indicated that adults with history of ACEs have an increased risk for suicidal ideation and suicide attempts (Fuller-Thomson, Baird, Dhrodia, & Brennenstuhl, 2016).This increased risk is observed even among adults without any diagnosable psychological disorders who self-report moderate to severe maltreatment during childhood exhibit dysregulation of the stress-response systems (Carpenter et al., 2007).

By the time children have reached preschool age, basic structures of the brain that are necessary for sensory processing have been myelinated; however, connections to the prefrontal cortex (PFC) are not yet fully myelinated (Brody, Kinney, Kloman, & Gilles, 1987; Yakovlev & Lecours, 1967), as well as some regions of the hippocampus (Jabès & Nelson, 2015). This allows for adaptability of circuitry through gained experiences. Therefore, the early experiences during childhood greatly impact the shaping of cognitive processing into adolescence and early adulthood (Bick et al., 2016). The experience of stress, which can lead to increased levels of stress hormones and neurotransmitters, can result in delayed myelination (Bellis & Zisk, 2014; Dunlop, Archer, Quinlivan, Beazley, & Newnham, 1997; Teicher et al., 2002)

It is suggested that a lack of expected information, such as attention from caregivers, during certain sensitive periods of brain development can prevent proper development (Bick et al., 2016). As well as the impact that neglect could have on brain development, the addition of abusive experiences can have detrimental impact on shaping neural circuitry formation. As

experiences of neglect and abuse often co-occur it is difficult to examine the impact that each individually has on brain development (Bick, 2016). In describing the impact of experience of early stress on development, Teicher et al. (2002) presents a model of cascading effects of exposure to stress early in life. The activation of the stress-response systems alters molecular organization. Teicher et al. (2002) suggests this alteration in molecular structure functions to increase sensitivity to future, stressful stimuli. The resulting release in hormones impacts the myelination, neural morphology, neurogenesis, and synaptogenesis (Teicher et al. 2002). This in turn impacts the developmental trajectory of different brain regions, and potentially impacts efficiency of communication between brain structures and pathways. The neurobiological changes associated with exposure to early stress may result in increased risk for several psychological disorders or behavioral problems. Teicher et al. (2002) suggests that the mechanisms by which early stress exposure alters brain development may reflect the adaptive nature of the brain to cope with levels of stress in one's environment. Thus, in response to early stress the developmental trajectory of the brain is altered to produce vigilance for detecting of danger.

Some research has indicated reductions in adult hippocampal (Bremner et al., 1997; Stein, Koverola, Hanna, Torchia, & McClarty, 1997; Whittle, Vijayakumar, Simmons, Yucel, & Lubman, 2016) and amygdala (Hanson et al., 2015) growth in relation to childhood maltreatment, as well as reduced thickness of the anterior cingulate cortex (Kelly et al., 2013). However, these findings have not been consistent across studies (Bick, 2016). Hippocampal volumes, for example, measured in children exposed to early stress showed no reduction compared to children with no early stress exposure (Carrion et al., 2001; De Bellis et al., 1999; De Bellis et al., 2002). Heterogeneity among findings may be explained by the impact that

differences among severities and types of maltreatment could potentially result in detriments to brain development (Bick, 2016). Thus, the understanding of the impact of stressful or adverse experiments on brain development is further complicated.

Current Study

The objective of the current study is to examine if previous exposure to stressful events during childhood may impact the relationship between exposure to acute stress and working memory (WM) performance. The literature examining impact of acute stress has been mixed; some studies have observed a negative relationship (Porcelli et al., 2008; Kirschbaum et al., 1996; Kuhlmann et al., 2005; Oei et al., 2006; Patil et al., 1995), and others have observed no impact (Domes et al., 2002; McMorris et al., 2006; Smeets et al., 2006). However, the relationship between WM performance and experiencing stress in an individual's current environment has the potential to be influenced by early stress and adverse experiences during childhood. Therefore, including the ACEs survey may be able to elucidate the mixed findings in the acute stress and working memory literature.

As demonstrated extensively in ACEs literature, the more exposure to stressful, adverse childhood experiences, the greater the potential impact on brain development (Bellis & Zisk, 2014; Bick & Nelson, 2016; M. D. De Bellis, 2001; Martin H Teicher et al., 2002). These detriments to healthy brain development, in turn, have an impact on individuals' ability to process information efficiently, ultimately having an impact on behavior. For example, previous findings have indicated that those who have experienced stress early in life have impaired WM compared to those who have not experienced such events (Philip et al., 2016).

The current study examined the relationship between early stress and adverse experiences prior to age 18 and working memory performance. It was hypothesized that participants who have higher scores on the ACEs questionnaire, indicating more adverse events in childhood, would perform lower on a baseline working memory task than participants with low scores on the ACEs questionnaire. Furthermore, due to the relationship between working memory and frontal theta activity, it was predicted that baseline theta will be lower for participants with low ACEs scores. Given the inverse relationship between theta and alpha, it was predicted that baseline alpha activity would be higher for participants with low ACEs scores. In addition to differences in baseline WM and frequency, it was hypothesized that a stress induction would result in lower WM performance, decreased theta synchronization, and increased alpha for all participants who experience the stress induction. Furthermore, the proposed study will examine how exposure to adverse experiences during childhood will impact the influence of an acute stressor on WM performance. Therefore, it is also hypothesized that an acute stressor will have less impact on WM performance, theta and alpha power for those with high scores on the ACEs questionnaire.

For the current study, participants were placed in one of two groups, Stress or Neutral. Participants in both groups were first be given a measure of their subjective emotional state. Participants were then asked to complete a WM memory task known as the digit span task. The participants in the Stress group then viewed a video containing potentially stressful content (acute stress induction). The participants in the Neutral group viewed a video containing no stressful content. Following the video, participants in both groups completed the WM task a second time using a different list of digits. Following the completion of the second WM task, participants were asked to complete the ACEs questionnaire to determine the amount of

exposure to early life stress or adverse experiences. Thus, current study sought to shed light on the potential relationship between previous early stress exposure during childhood, WM performance, and acute stress on WM performance.

Chapter 2. Methods

Participants

A total of 57 participants (23 male/34 female) were recruited using the East Tennessee State University SONA participant recruitment system. This study was approved by the East Tennessee State Institutional Review Board and each participant gave informed consent. Five participants were unable to complete the study, leaving 52 of the participants in the final analysis. Prior to obtaining informed consent, participants were place into one of two groups, Stress (N=28) or Neutral (24). Participants in the Stress group had a mean ACEs score of 2.89 (SD=2.57) and participants in the Neutral group had a mean ACEs score of 2.58 (SD=2.08). Two participants in the Stress group were not included in the analyses of frequency power due to abnormally high values in in power.

EEG Data Acquisition

Electroencephalograph (EEG) was recorded using a cap (Electro-Cap International, Inc.) embedded with 32 tin electrodes. Channels were referenced to the right mastoid and grounded to the left mastoid. The EEG was digitized at 256 Hz and bandpass filtered to [0.5 Hz, 30 Hz] by two 16-channel g.tec g.USBamp amplifiers. EEG data acquisition and collection was performed using g.Recorder, and stimuli were presented electronically using E-Prime 3.0 software.

Survey Measures

Adverse Childhood Experiences Questionnaire. The Adverse Childhood Experiences Questionnaire (ACEs) was first developed and validated by Felitti et al. (1998). The questionnaire was adapted from several scales that evaluate several types of abuse or dysfunction that could occur in the household, such as psychological and physical abuse, exposure to substance abuse, whether other family members exhibited signs of mental illness, if the mother

figure in the family experienced violent treatment, or if any family members engaged in criminal behavior (Appendix A).

Positive Affect Negative Affect Schedule. To assess subjective emotional state, positive and negative ratings were obtained using the Positive Affect Negative Affect Schedule (PANAS; Watson, Clark, & Carey, 1988). This measure has previously been used in studies examining the impact of a stress induction on WM (Gärtner et al., 2014). The PANAS is a questionnaire that contains a list of 20 descriptive words (for example, excited, nervous, etc.) that convey emotional states. Each emotional word is paired with a 5-point Likert-scale, ranging from 1 (not at all) to 5 (extremely) to indicate whether the word describes the current emotional experience. Half of the words contained in the PANAS are associated with subjectively positive emotions (positive affect; PA), and half of the words are associated with subjectively negative emotions (negative affect; NA). If a participant scores high on the PA, they are considered to have higher levels of positive affect. If a participant scores high on the NA, they are considered to have higher levels of negative affect. The PANAS was administered at three time points, before the first WM task, before viewing video footage (stress/ non-stress), and after viewing the video. The PANAS was used to serve as a manipulation check to ensure the effectiveness of the stress induction. Manipulation was determined by whether the stress induction produced an increase in negative affect and a decrease in positive affect compared to participants who did not undergo stress induction (Appendix B).

Working Memory Task

The working memory (WM) task used for this study is commonly referred to as the digit span task (Conway et al., 2005; Dempster & Cooney, 1982; Dong et al., 2015). The digit span task is a measure of WM, as it involves the active maintenance of a mental representation of information within the temporary storage of WM. During the digit span task, participants are presented with a series of digits and are then asked to recall the digits after stimulus presentation. The digit span task is designed to begin with a small number of digits, or pieces of information, to be held in WM. Then, after a pre-determined number of trials, the number of digits the participant will be required to hold in WM, and subsequently asked to recall, is increased by one digit. The increase in task demand requires allocation of additional cognitive resources, and progressively becomes too difficult for the WM system to hold all the information presented. Therefore, as the amount of information to be held in WM increases, WM performance is suggested to decrease.

Experimental Stress Induction

Viewing strongly aversive and violent footage has been shown to elicit both physiological and psychological stress (Henckens, Hermans, Pu, Joels, & Fernandez, 2009; Hermans et al., 2011; Qin et al., 2012, 2009; Gärtner et al., 2014; Hermans et al., 2011; Ossewaarde et al., 2010; Qin et al., 2009). To induce a stress response in the stress condition, participants were shown a video containing footage from school shootings that are both publicly available and have been shown either on the news or included in news articles covering school shootings. The footage was obtained from public domain websites and includes footage from school shootings at the American School of Northeast in Colegio Americano del Noreste, Mexico, and Marjory Stoneman Douglas High School, in Parkland, Florida. The video also

contains interviews with survivors from the school shooting in Columbine High School in Columbine, Colorado. The footage for the stress induction was chosen due to its relevance to the study sample of college undergraduate students. For the neutral condition, participants viewed a clip of non-aversive classroom activities. Participants were informed prior to the start of the video which video they would view and were reminded that they could ask to end the video and the study at any time.

Experiment Procedure and Design

Each participant completed one experimental session that lasted approximately 2 hours. Participants were placed into one of two groups, Stress or Neutral, and group placement was counter-balanced. Each participant was seated in a chair approximately 90cm from a computer monitor. Participants were then asked to complete an informed consent form with demographic information and were then fully debriefed on the experiment procedure, including the potential for stress induction. Once the informed consent was completed, participants were asked to complete the positive affect negative affect schedule (PANAS) for the first time (see Table 2). Participants were then fitted with a 32 electrode EEG cap and asked to focus their attention on the computer screen to complete the first working memory (WM) digit span task. Due to the difficult nature of the task, each participant completed two practice trials of the set size of 4 to ensure that they understand the instructions of the task. The practice trials were used to ensure that any errors on future trials are due to limitations of WM and not a misunderstanding of the task.

During the experimental session participants completed two practice digit span trials, followed by six, digit span sets. Each "set size" included four trials, beginning with a set size of four digits to be held in WM. After every four trials, or each set size, the number of digits to be

held in WM was increased by one until they have completed a set size of nine digits. For each WM task, participant completed all six set sizes three times for a total of 12 trials per set size. Between each block of set size four through set size of nine, participants were given a short break. Each digit within a trial was presented for 1000 ms (see Figure 1). For each trial, participants were prompted to recall the digits that had appeared on the screen and to repeat the digits by pressing the corresponding digits on the keyboard. After each digit span set (four trials), participants were asked to provide a Likert scale measure of the amount of effort they felt was required to complete the task, ranging from 1 (little to no effort) to 7 (as much effort as possible). The digits presented in the WM task were produced by a random number generator. Four different versions of the WM task were created with each version containing a different list of digits. Each trial contained a different order of digits to ensure participants did not see the same set of digits more than once. Participants completed two of the four separate versions, one for the first WM task and the other for the second WM task. The order of which versions of the WM task were used was counter-balanced across participants.

Following the first WM task, participants were asked to complete the PANAS for a second time (see Table 2). After completing the PANAS, participants were asked to view video footage on the computer screen based on the condition they have been assigned, Stress or Neutral. Participants were informed immediately before viewing the video which video they will be viewing, footage from school shootings (stress condition) or footage of students in a classroom (neutral condition). Each participant agreed to continue with the experiment and viewed a video that is approximately 4 minutes in length. Upon viewing the video, participants were asked to complete the PANAS again (see Table 2) to inquire whether viewing the video impacted their emotional state. Participants were then asked to complete the WM task again to

examine whether viewing the video, specifically the stress induction, impacted their WM performance.

The procedure for the first instance of the WM task was repeated for the second WM task using a different version of containing different digits. Finally, upon completing the second task, participants were handed an envelope containing the ACEs questionnaire. Each envelope was labeled with the participant ID only, so that participant information is kept unidentifiable. The experimenter then left the room to allow the participant to complete the ACEs questionnaire, as the items inquired about potentially sensitive information.

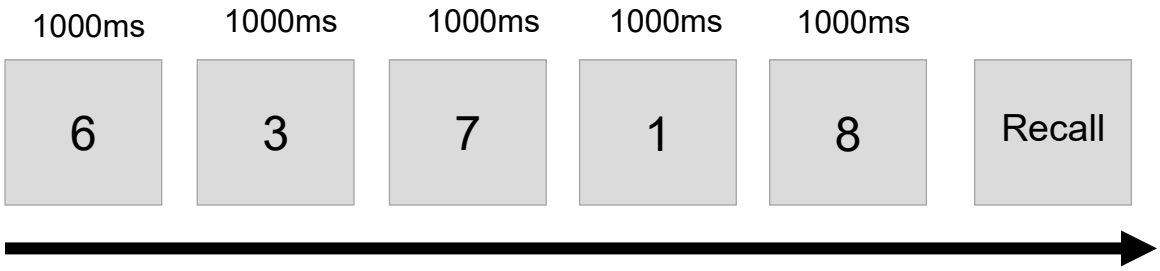

Figure 1: Time course of stimulus presentation for a set size of 5 digits.

Chapter 3. Analyses

EEG Data Processing

The EEG data analysis was conducted using MATLAB (Version 2016b, The MathWorks, Inc., Massachusetts, USA) and EEGLAB, Version 14.1.2b (Delorme & Makeig, 2004). Customized scripts were created and used to carry out analyses. An independent components analysis (ICA; using the EEGLAB plugin ICLabel) was then applied to the clean data. ICA components including eye movements, muscle artifacts, line noise, and channel noise were then identified and removed from the data. The EEG data was then extracted in epochs from the onset of the first digit to the participant's offset of the second digit, i.e. the first 2000 msec of the stimulus presentation (see Figure 2). Spectral analysis was conducted using EEGLAB and spectral power was calculated in 1Hz bins for each frequency in the range of 8-12Hz at electrode locations Fz, and FCz to examine theta activity. In addition to examining power at the two individual electrodes, mean power for the two electrodes was collapsed to create one Frontal Theta variable. To examine theta activity, power was calculated in 1Hz bins ranging from 4-8Hz at electrode locations Cz, Pz, and POz. In addition to examining power at the three individual electrodes, mean power for the three electrodes was collapsed to create one Parietal Alpha variable.

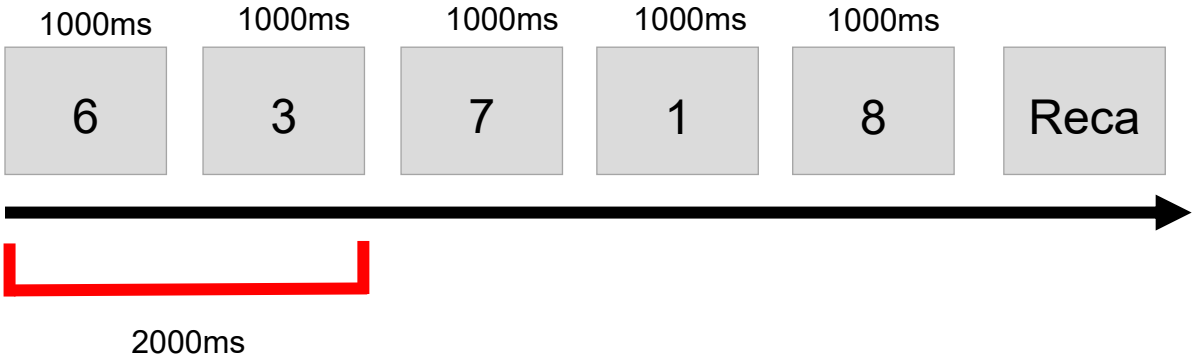

Figure 2: Time course of stimulus presentation and epoch window from beginning from the onset of the first digit to the offset of the last digit for a set size of 5 digits.

Statistical Analyses

Statistical analyses were performed using R 3.52 (R Core Team, 2019) using the package BRMS. A Bayesian t-test was used to examine differences in PANAS scores at times 2 and 3 to measure change in affect following stress induction. A series of Bayesian linear mixed effects models (LMM) were used to examine the fixed effects of acute stress condition (neutral vs stress) and adverse childhood experiences (ACEs), and random effects of subject on the dependent variables of working memory performance (WM), frontal theta activity, and alpha activity. The Bayes Factor provides a ratio of the likelihood of obtaining the data (**D**) given under the alternative (H_1) and null hypothesis (H_0).

$$B_{10} = P(D|H_1)/ P(D|H_0)$$

Interpretations of a given Bayes factor based on the recommendations of Kass and Raftery (1995) are provided in Table 1. For the current study, a Bayes factor of 15 for the null was chosen as the criterion to determine whether each of the alternative hypotheses were supported.

Table 1.
Interpretation of Bayes Factors

Evidence for H0		**Evidence for H1**
0.01	Extreme	100<
0.03	Very Strong	30
0.1	Strong	10
0.33	Moderate	3
1	Weak/Anecdotal	1

Using a Bayesian LMM allows for each model to reflect variations in intercept and slope for each participant (i) across time (t), and whether ACEs scores and acute stress (s) may predict WM performance, and changes in frontal theta and parietal alpha.

$$M0: Yi = b0 + \mu_i + e_i$$

To test the main effect of time

$$M1: Yti = b0 + b1(time_{ti}) + \mu_i + e_{ti}$$

To test the main effect of stress

$$M2: Yti = b0 + b1(stress_{si}) + \mu_i + e_{si}$$

To test the main effect of ACEs

$$M3: Yti = b0 + b1(ACE_{ai}) + \mu_i + e_{ai}$$

To test the joint main effects

$$M4: Yti = b0 + b1(time_{ti}) + b2(stress_i) + b3\ (ACE_i) + \mu_i + e_{ti}$$

To test stress by time interaction

$$M5: Yti = b0 + b1(time_{ti}) + b2(stress_i) + b3\ (time_{ti} \times stress_i) + \mu_i + e_{ti}$$

To test 2-way interactions

$$M6: Yti = b0 + b1(time_{ti}) + b2(stress_i) + b3\ (time_{ti} \times stress_i) + b4(ACE_i) + b5\ (time_{ti} \times ACE_i) + b6\ (ACE_{ti} \times stress_i)\ \ \mu_i + e_{ti}$$

To test 3-way interactions

$$M7: Yti = b0 + b1(time_{ti}) + b2(stress_i) + b3\ (time_{ti} \times stress_i) + b4(ACE_i) + b5\ (time_{ti} \times ACE_i) + b6\ (ACE_{ti} \times stress_i) + b7(time_{ti} \times stress_i \times ACE_i) + \mu_i + e_{ti}$$

A likelihood ratio to test the main effect of time was calculated by comparing the Bayes factor obtained for M1 by M0. To test the main effect of stress, a likelihood ratio was calculated by comparing M2 to M0. Likewise, to test the main effect of ACEs, a likelihood ratio was calculated by comparing M3 to M0. For examining joint main effects, a comparison was made between M4 and M0. To test the stress by time interaction, M5 was compared to M4. For examining all two-way interactions, comparisons were made between M6 and M4. Lastly, to examine all three-way interaction, comparisons were made between M7 and M4.

To determine WM performance for each participant, a baseline WM capacity score was calculated. This capacity score was determined by first calculating performance accuracy for each set size. Accuracy for each set size was determined by the percentage of correct response trials for each set size. Then, the smallest set size in which accuracy for set size trials was less than 50% was determined for each participant. The set size prior to the set size in which each participant reached less than 50% accuracy was then used as the WM capacity score. For example, if a participant reached less than 50% accuracy for the first time at set size 6, the participant would receive a WM capacity score of 5. Peak power for frontal theta and parietal was expected to occur at the set size in which participants reached their WM capacity. By determining which set size is considered to be the WM capacity for each participant, the measurement of power at frontal and parietal locations should reflect peak power changes related to limits in cognitive resources being reached. It is predicted that frequency power should increase until that limit is reached, then decrease once the limit has been reached.

Chapter 4. Results

Measure of Stress Induction

Changes in positive affect (PA) and negative affect (NA) on the PANAS before viewing video footage in each condition, Stress or Neutral, were used to ensure effectiveness of stress induction. Change scores were calculated by subtracting scores obtained before viewing the video from scores obtained after viewing the video for both PA and NA for each group. A one-tailed Bayes factor t-test showed strong evidence (B_{10} = 43.89 ±0.01%) suggesting that there was a larger decrease in PA scores for the Stress group than the Neutral group (see means in Table 2). A one-tailed Bayes factor t-test also showed strong evidence (B_{10} = 3012035) suggesting that there was a larger increase in NA scores for the Stress group than the Neutral group (see means in Table 2). These results indicate that the stress induction successfully reduced positive affect and increased negative affect.

Table 2.
ACEs and PANAS scores

	ACEs	PA 1	PA 2	PA 3	NA 1	NA 2	NA 3
Stress							
M	2.89	29.36	24.96	21.00	13.50	14.46	18.96
SD	2.57	5.98	6.55	5.36	3.32	3.70	5.10
N	28	28	28	28	28	28	28
Neutral							
M	2.58	30.92	24.54	22.83	11.62	13.67	11.33
SD	2.08	6.40	7.43	7.59	2	5.25	2.85
N	24	24	24	24	24	24	24

Working Memory Performance

For model comparisons, Bayes factor analysis using the default prior of the BRMS package to examine whether changes in WM performance from the first WM task to the second WM task were related to condition of stress and ACEs scores (means and standard deviations provided in Table 3). Therefore, a Bayes factor was calculated for the model examine the main effect of time, with weak evidence for the model $B_{10} = 2.35 \pm 0.73\%$. Thus, there is not enough evidence to suggest that WM performance changes from WM task 1 to WM task 2 are due to the main effect of time. A Bayes factor was also calculated for the model examine the main effect of stress, $B_{10} = 0.33 \pm 1.71\%$, with moderate evidence in favor for the null. Thus, there is not enough evidence to suggest that WM performance changes from WM task 1 to WM task 2 are due to the main effect of stress. To examine the main effect of ACEs, a Bayes factor of $B_{10} = 0.29 \pm 1.1\%$ indicated moderate evidence in support of the null, suggesting no main effect of ACEs on WM performance. Examining the joint main effects of time, subject, stress, and ACEs also indicated weak evidence in favor of the null ($B_{10} = 0.52 \pm 2.36\%$).

The examination of the interaction of stress by time also indicated weak evidence in favor of the null ($B_{10} = 0.52 \pm 3.66\%$), suggesting no effect of the interaction on WM performance. When examining all possible 2-way interactions, strong evidence in favor of the null ($B_{10} = 0.18 \pm 4.35\%$) suggests no effect of 2-way interactions on WM performance. Finally, the examination of the three-way interaction indicated weak evidence in favor of the null ($B_{10} = 0.92 \pm 5.4\%$), indication there is not enough evidence to suggest an effect of the 3-way interaction on WM performance.

Frontal Theta

A Bayes factor was calculated for the model examining the main effect of time on frontal theta and demonstrated moderate evidence in favor of the null (B_{10} = 0.41 ±0.67%). A Bayes factor was also calculated for the model examine the main effect of stress on frontal theta, with moderate to weak evidence in favor of the null (B_{10} = 0.52 ±2.24%). Thus, there is not enough evidence to suggest that theta changes at frontal theta from WM task 1 to WM task 2 are due to the main effect of stress. To examine the main effect of ACEs, moderate evidence in favor of the null (B_{10} = 0.49 ±0.61%) also suggests no main effect of ACEs on frontal theta. Examining the joint main effects of time, subject, stress, and ACEs also indicated strong evidence in favor of the null (B_{10} = 0.09 ±5.31%).

The examination of the interaction of stress by time also indicated moderate evidence in favor of the null (B_{10} = 0.31 ±4.44%), suggesting no effect of the interaction on frontal theta. When examining all possible 2-way interactions, weak evidence in favor of the model (B_{10} = 2.31 ±6.09%), suggests there is not enough evidence for an effect of 2-way interactions on frontal theta. Finally, the examination of the three-way interaction indicated weak evidence in favor of the null (B_{10} = 0.74 ±3.55%), suggesting no effect of the 3-way interaction on theta frontal theta (means and standard deviations provided in Table 3).

Fz

A Bayes factor was calculated for the model examining the main effect of time on theta at electrode Fz and demonstrated moderate evidence in favor of the null (B_{10} = 0.35 ±0.79%). A Bayes factor was also calculated for the model examine the main effect of stress on theta at Fz, with moderate to weak evidence for the null (B_{10} = 0.42 ±0.92%). Thus, there is not enough evidence to suggest that theta changes at Fz from WM task 1 to WM task 2 are due to the main

effect of stress. To examine the main effect of ACEs, moderate evidence in favor of the null (B_{10} = 0.35 ±1.37%) also suggests the is not enough evidence for the main effect of ACEs on theta at Fz. Examining the joint main effects of time, subject, stress, and ACEs also indicated strong evidence in favor of the null (B_{10} = 0.06 ±3.82%).

The examination of the interaction of stress by time also indicated moderate evidence in favor of the null (B_{10} = 0.39 ±4.41%), suggesting no effect of the interaction on theta at Fz. When examining all possible 2-way interactions, weak to moderate evidence in favor of the model (B_{10} = 2.88 ±3.54%), suggesting there is not enough evidence to conclusively say there is an effect of 2-way interactions on theta at Fz. Finally, the examination of the three-way interaction indicated weak evidence in favor of the null (B_{10} = 0.86 ±3.14%), suggesting there is not enough evidence to say there is an effect of the 3-way interaction on theta at Fz (means and standard deviations provided in Table 3).

FCz

A Bayes factor was calculated for the model examining the main effect of time on theta at electrode FCz and demonstrated weak evidence in favor of the model (B_{10} = 1.13 ±2.82%). A Bayes factor calculated for the model examining the main effect of stress on theta at FCz demonstrated moderate to weak evidence in favor of the null (B_{10} = 0.61 ±2.88%). Thus, there is not enough evidence to suggest that theta changes at FCz changes from WM task 1 to WM task 2 are due to the main effect of stress. To examine the main effect of ACEs, moderate to weak evidence in favor of the null (B_{10} = 0.96 ±5.84%) suggests there is not enough evidence to suggest a main effect of ACEs on theta at FCz. Examining the joint main effects of time, subject, stress, and ACEs indicated weak evidence for the null (B_{10} = 0.75 ±3.01%).

The examination of the interaction of stress by time indicated moderate to weak evidence in favor of the null (B_{10} = 0.49 ±4.91%), suggesting no effect of the interaction on theta at FCz. When examining all possible 2-way interactions, moderate evidence in favor of the null (B_{10} = 0.32 ±5.93%) indicates there is not enough evidence to suggest an effect of 2-way interactions on theta at FCz. Finally, the examination of the three-way interaction indicated moderate to weak evidence in favor of the null (B_{10} = 0.51 ±7.61%), suggesting there is not enough evidence to demonstrate an effect of the 3-way interaction on theta at FCz (means and standard deviations provided in Table 3).

Parietal Alpha

A Bayes factor was calculated for the model examining the main effect of time on parietal alpha and demonstrated moderate to weak evidence in favor of the null (B_{10} = 0.59 ±2.19%). A Bayes factor was calculated for the model examining the main effect of stress on alpha at Cz and demonstrated moderate to weak evidence in favor of the null (B_{10} = 0.53 ±2.17%). Thus, there is not enough evidence to suggest that parietal alpha changes from WM task 1 to WM task 2 are due to the main effect of stress. To examine the main effect of ACEs, moderate evidence in favor of the null (B_{10} = 0.47 ±0.81%) suggests no main effect of ACEs on parietal alpha. Examining the joint main effects of time, subject, stress, and ACEs also indicated strong evidence in favor of the null (B_{10} = 0.14 ±3%).

The examination of the interaction of stress by time also indicated moderate evidence in favor of the null (B_{10} = 0.46 ±4.55%), therefore there is not enough evidence to suggest an effect of the interaction parietal alpha. When examining all possible 2-way interactions, strong evidence in favor of the null (B_{10} = 0.17 ±5.5u%) suggests no effect of 2-way interactions on parietal alpha. Finally, the examination of the three-way interaction indicated moderate evidence

in favor of the null (B_{10} = 0.48 ±7.03%), suggesting an effect of the three-way interaction on parietal alpha (means and standard deviations provided in Table 3).

Cz

A Bayes factor was calculated for the model examining the main effect of time on alpha at electrode Cz and demonstrated moderate evidence in favor of the null (B_{10} = 0.41 ±2.09%). A Bayes factor calculated for the model examining the main effect of stress on alpha at Cz, indicated moderate to weak evidence in favor of the null (B_{10} = 0.54 ±1.54%). Thus, there is not enough evidence to suggest that alpha changes at Cz changes from WM task 1 to WM task 2 are due to the main effect of stress. To examine the main effect of ACEs, moderate evidence in favor of the null (B_{10} = 0.44 ±1.12%) also indicates there is not enough evidence to suggest that there is a main effect of ACEs on alpha at Cz. Examining the joint main effects of time, subject, stress, and ACEs indicated strong evidence in favor of the null (B_{10} = 0.09 ±2.79%).

The examination of the interaction of stress by time also indicated moderate evidence (B_{10} = 0.42 ± 4.28%) in favor of the null, suggesting no effect of the interaction on alpha at Cz. When examining all possible 2-way interactions, moderate evidence in favor of the null (B_{10} = 0.29 ±4.48%) suggests no effect of 2-way interactions on alpha at Cz. Finally, the examination of the three-way interaction indicated weak evidence in favor of the null (B_{10} = 0.69 ±5.82%), suggesting there is not enough evidence to suggest an effect of the 3-way interaction on alpha at Cz (means and standard deviations provided in Table 3).

Pz

A Bayes factor was calculated for the model examining the main effect of time on alpha at electrode Pz and demonstrated moderate evidence in favor of the null (B_{10} = 0.51 ±2.13%). A

Bayes factor was calculated for the model examining the main effect of stress on alpha at Pz, and indicated weak evidence for the null (B_{10} = 0.79 ±2.11%). Thus, there is not enough evidence to suggest that alpha changes at Pz changes from WM task 1 to WM task 2 are due to the main effect of stress. To examine the main effect of ACEs, moderate evidence in favor of the null (B_{10} = 0.44 ±0.75%) also suggests no main effect of ACEs on alpha at Pz. Examining the joint main effects of time, subject, stress, and ACEs also indicated strong evidence in favor of the null (B_{10} = 0.18 ±2.71%).

The examination of the interaction of stress by time also indicated moderate evidence in favor of the null (B_{10} = 0.46 ±2.99%), suggesting no effect of the interaction on alpha at Pz. When examining all possible 2-way interactions, moderate to strong evidence in favor of the null (B_{10} = 0.13 ±5.33%) suggests no effect of 2-way interactions on alpha at Pz. Finally, the examination of the three-way interaction indicated moderate to weak evidence in favor of the null (B_{10} = 0.64 ±7%), indicating that there is not enough evidence to suggest an effect of the 3-way interaction on alpha at Pz (means and standard deviations provided in Table 3).

POz

A Bayes factor was calculated for the model examining the main effect of time on alpha at electrode POz and demonstrated moderate evidence in favor of the null (B_{10} = 0.32 ±0.61%). A Bayes factor calculated for the model examining the main effect of stress on alpha at POz indicated moderate to weak evidence in favor of the null (B_{10} = 0.58 ±0.59%). Thus, there is not enough evidence to suggest that alpha changes at POz changes from WM task 1 to WM task 2 are due to the main effect of stress. To examine the main effect of ACEs, moderate evidence in favor of the null (B_{10} = 0.40 ±0.55%) also suggests no main effect of ACEs on alpha at POz.

Examining the joint main effects of time, subject, stress, and ACEs also indicated strong evidence in favor of the null (B_{10} = 0.07 ±3.63%).

The examination of the interaction of stress by time also indicated moderate evidence in favor of the null (B_{10} = 0.43 ±1.92%), indicating there is not enough evidence to suggest an effect of the interaction on alpha at Pz. When examining all possible 2-way interactions, strong evidence in favor of the null (B_{10} = 0.17 ±16.37%) suggests no effect of 2-way interactions on alpha at POz. Finally, the examination of the three-way interaction moderate to weak evidence in favor of the null (B_{10} = 0.52 ±5.32%) indicates no effect of the 3-way interaction on alpha at POz (means and standard deviations provided in Table 3).

Table 3.

Working Memory Means and Standard Deviations

	Accuracy	Fz Theta	FCz Theta	Cz Alpha	Pz Alpha	Poz Alpha	Frontal Theta	Parietal Alpha
	Stress WM1							
M	69.94	5.42	1.06	5.30	1.68	0.92	3.24	2.63
SD	13.48	2.17	0.81	1.68	1.22	0.58	1.29	0.96
N	28	26	26	26	26	26	26	26
	Stress WM2							
M	60.57	7.12	1.07	5.89	2.19	1.04	4.10	3.04
SD	21.68	6.63	0.74	3.12	1.79	0.65	3.49	1.53
N	28	26	26	26	26	26	26	26
	Neutral WM1							
M	65.28	4.64	0.89	4.94	3.28	1.42	2.77	3.21
SD	12.69	1.94	0.68	1.79	5.04	1.94	1.17	2.49
N	24	24	24	24	24	24	24	24
	Neutral WM2							
M	61.81	5.75	0.97	5.24	2.83	1.29	3.36	3.12
SD	20.40	3.78	0.77	1.73	3.12	1.66	1.97	1.94
N	24	24	24	24	24	24	24	24

Chapter 5. Exploratory Analyses

PANAS

As the results indicated that the PANAS measured a change in affect due to the stress manipulation, exploratory analyses were conducted to examine whether changes in PA and NA scores could be more sensitive to detect the effects of stress on the outcome variables of interest than the categorical variable of stress (Stress or Neutral group). Therefore, PA change scores were calculated by subtracting the PA score after viewing video footage from the PA score prior to watching the video and NA change scores were calculated by subtracting the NA score after viewing video footage from the NA score prior to watching the video. The PA change score was then used to replace the variable of stress in each of the models to determine if the change in PA was more specific than the coding of stress or neutral. The same procedure was followed by replacing the variable of stress with the NA change score.

PANAS - Working Memory Performance

A Bayes factor was also calculated for the model examining the main effect of stress using the PA change score, with weak evidence for in favor of the null ($B_{10} = 0.37 \pm 1.55\%$). Thus, there is not enough evidence to suggest that WM performance changes from WM task 1 to WM task 2 are due to the main effect of stress using the PA change score. Examining the joint main effects of time, subject, stress using the PA change score, and ACEs also indicated moderate evidence in favor of the null ($B_{10} = 0.29 \pm 3.84\%$).

The examination of the interaction of stress using PA change score by time also indicated moderate evidence in favor of the null ($B_{10} = 0.32 \pm 5.42\%$) suggesting no effect of the interaction on WM performance. When examining all possible 2-way interactions, extreme

evidence in favor of the null (B_{10} = 0.004 ±16.87%) suggests no effect of 2-way interactions on WM performance. Finally, the examination of the three-way interaction indicated weak evidence in favor of the null (B_{10} = 0.49 ±30.57%), thus there is not enough evidence to suggest an effect of the 3-way interaction on WM performance.

A Bayes factor was also calculated for the model examining the main effect of stress using the NA change score, with moderate evidence in favor of the null (B_{10} = 0.26 ±1.17%). Thus, there is no evidence to suggest that WM performance changes from WM task 1 to WM task 2 are due to the main effect of stress using the NA change score. Examining the joint main effects of time, subject, stress using the NA change score, and ACEs also indicated moderate evidence in favor of the null (B_{10} = 0.20 ±3.13%).

The examination of the interaction of stress using NA change score by time also indicated weak evidence in favor of the null (B_{10} = 0.46 ±8.17%), indicating there is not enough evidence to suggest an effect of the interaction on WM performance. When examining all possible 2-way interactions, very strong evidence in favor of the null (B_{10} = 0.03 ±12.84%) suggests no effect of 2-way interactions on WM performance. Finally, the examination of the three-way interaction indicated moderate evidence in favor of the null (B_{10} = 0.33 ±21.16%), suggesting no effect of the 3-way interaction on WM performance.

PANAS-Frontal Theta

A Bayes factor was calculated for the model examining the main effect of stress using the PA change score on frontal theta, with moderate evidence in favor of the model (B_{10} = 3.12 ±1.57%). Thus, there is evidence to suggest that changes in frontal theta from WM task 1 to WM task 2 are may be associated with the main effect of stress using the PA change score. Examining

the joint main effects of time, subject, stress, and ACEs also indicated weak evidence in favor of the null (B_{10} = 1.00 ±4.24%).

The examination of the interaction of stress using the PA change score by time also indicated moderate evidence in favor of the null (B_{10} = 0.25 ±4.4%), suggesting no effect of the interaction on frontal theta. When examining all possible 2-way interactions, weak to moderate evidence in favor of the model (B_{10} = 2.45 ±7.72%) suggests no effect of 2-way interactions on frontal theta. Finally, the examination of the three-way interaction indicated strong evidence in favor of the null (B_{10} = 0.002 ±12.79%) suggesting no effect of the 3-way interaction using PA change scores on theta frontal theta.

A Bayes factor was also calculated for the model examining the main effect of stress using the NA change score on frontal theta indicated weak evidence for the null (B_{10} = 0.82 ±11.03%). Thus, there is not enough evidence to suggest that theta changes at frontal theta from WM task 1 to WM task 2 are due to the main effect of stress using NA change scores. Examining the joint main effects of time, subject, stress, and ACEs also indicated weak evidence in favor of the model (B_{10} = 1.00 ±4.24%).

The examination of the interaction of stress using the NA change score by time also indicated moderate evidence in favor of the null (B_{10} = 0.11±19.8%) suggesting no effect of the interaction on frontal theta. When examining all possible 2-way interactions, strong evidence in favor of the null (B_{10} = 0.05 ±7.91%) suggests no effect of 2-way interactions on frontal theta. Finally, the examination of the three-way interaction indicated strong evidence in favor of the null (B_{10} = 0.11 ±14.36%) suggesting no effect of the 3-way interaction on theta frontal theta.

PANAS-Parietal Alpha

A Bayes factor was also calculated for the model examine the main effect of stress using the PA change score on parietal alpha with moderate evidence for the model ($B_{10} = 0.56 \pm 7.39\%$). Thus, there is not enough evidence to suggest that parietal alpha changes from WM task 1 to WM task 2 are due to the main effect of stress using the PA change score. Examining the joint main effects of time, subject, stress, and ACEs also indicated evidence for the null ($B_{10} = 0.19 \pm 3.15\%$).

The examination of the interaction of stress using the PA change scores by time also indicated moderate evidence ($B_{10} = 0.19 \pm 6.34\%$) suggesting no effect of the interaction parietal alpha. When examining all possible 2-way interactions, extremely strong evidence in favor of the null ($B_{10} = 0.27 \pm 7.52\%$) suggests no effect of 2-way interactions on parietal alpha. Finally, the examination of the three-way interaction indicated extremely strong evidence for the null ($B_{10} = 0.001 \pm 24.69\%$) suggesting no effect of the 3-way interaction on parietal alpha.

A Bayes factor was also calculated for the model examine the main effect of stress using the NA change score on parietal alpha with weak evidence for in favor of the null ($B_{10} = 0.68 \pm 18.67\%$). Thus, there is not enough evidence to suggest that parietal alpha changes from WM task 1 to WM task 2 are due to the main effect of stress. Examining the joint main effects of time, subject, stress, and ACEs also indicated strong evidence in favor of the null ($B_{10} = 0.12 \pm 3.44\%$).

The examination of the interaction of stress using the NA change score by time also indicated weak evidence in favor of the null ($B_{10} = 0.36 \pm 6.16\%$), suggesting no effect of the interaction parietal alpha. When examining all possible 2-way interactions, strong evidence in

favor of the null (B_{10} = 0.86 ±7.12%) suggests no effect of 2-way interactions on parietal alpha. Finally, the examination of the three-way interaction indicated strong evidence in favor of the null (B_{10} = 0.05 ±19.99%), suggesting there is not enough evidence to suggest an effect of the 3-way interaction on parietal alpha.

Condition of Set Size

In the initial analysis, working memory (WM) capacity was calculated for each participant, and comparisons of accuracy, frontal theta, and parietal alpha between each group were made based on each participants WM capacity. Each participant was presented set sizes of different digits, ranging from 4 digits to 9 digits to be held in WM at one time. Each participant's WM capacity could be reached at a different set size; for example, one participant may have reached capacity at set size 5, while another may have reached capacity at set size 6. Differences may exist not only at the set size at which participants' WM capacity was reached. It is possible that effects of acute stress and ACEs may exist on WM performance before, or after, WM capacity is reached. Furthermore, the epoch windows used in the initial analysis was 2000ms, which may have been too long capture the potentially subtle differences in frequency change because the latency at which changes in frequency occurred may have been more localized to a shorter window (see section Set Size- EEG data processing: Window 1 for a description of the procedure).

To examine these possible effects, additional exploratory analyses were conducted. These analyses were conducted by adding the condition of set size to each of the models that were previously tested. By adding the condition of set size to each of the models, the effects of stress and ACEs on differences between WM performance, theta, and alpha at varying levels of task difficulty could be examined. For example, WM related theta may be different across groups at

the easiest set size (i.e. set size 4). This could provide insight into whether participants who experienced acute stress and had high ACEs scores would have lower WM performance, higher theta, and higher alpha when the task is easier compared to those who experienced no stress and have lower ACEs scores.

Set Size- Working Memory Performance

Exploratory analyses examining whether WM performance at each set size of digits, set 4 through set 9, varied from WM task 1 and 2 were conducted (means and standard deviations provided in Table 4). A Bayes factor was calculated for the model examining the main effect of set size, with extremely strong evidence in favor of the null ($B_{10} < 0.01 \pm 0.69\%$). Thus, there is no evidence to suggest that WM performance changes from WM task 1 to WM task 2 are due to the main effect of set size. Examining the joint main effects of time, subject, set size, stress, and ACEs also indicated extremely strong evidence ($B_{10} < 0.01 \pm 2.81\%$) for the null.

The examination of the interaction of set size by time also indicated extremely strong evidence ($B_{10} = 0.01 \pm 3.24\%$) in favor of the null, suggesting no effect of the interaction on WM performance. When examining all possible 2-way interactions, strong evidence in favor of the null ($B_{10} = 0.06 \pm 18.$) suggests no effect of 2-way interactions on WM performance. Finally, the examination of the four-way interaction indicated extremely strong evidence in favor of the null ($B_{10} < 0.01 \pm 48.93\%$) suggesting no effect of the 4-way interaction on WM performance.

Set Size- EEG data processing: Window 1

No effects on the EEG data were observed during the onset of the first digit to the offset of the second digit for each trial in the WM capacity set size. The EEG data was first extracted in epochs of 2000 ms in duration from the onset of the first digit presentation for each trial for

every condition of set size. It is possible that the size of the epochs of EEG data used in the analyses of theta and alpha at WM capacity were too large, preventing the detection of changes in theta and alpha related to WM performance. Therefore, the first series of exploratory analyses of the EEG data examined changes across set size. Event-related spectral perturbation (ERSP) plots for each set size across all participants were then created in EEGLAB (see Figures 5-9, Appendix C). Visual inspection of ERSP plots was used to determine the time window of interest in which the greatest amount of change in frontal theta and parietal alpha may occur (See Figure 3).

The EEG data for each set size was then re-epoched using the new time windows that were determined from the visual inspection of ERSP plots. Spectral analysis was conducted using EEGLAB and spectral power was calculated in 1Hz bins for each frequency in the range of 8-12Hz at electrode locations Fz, and FCz to examine theta activity. In addition to examining power at the two individual electrodes, mean power for the two electrodes was collapsed to create one Frontal Theta variable. To examine theta activity, power was calculated in 1Hz bins ranging from 4-8Hz at electrode locations Cz, Pz, and POz. In addition to examining power at the three individual electrodes, mean power for the three electrodes was collapsed to create one Parietal Alpha variable (means and standard deviations provided in Table 4).

Figure 3: Time course of stimulus presentation and epoch window. Data was first extracted from beginning from the onset of the first digit to the offset of the second digit (2000ms). Visual inspection was then used to determine the time window within the epoched data in which the greatest changes in theta and alpha were likely to occur (for this example, 500ms).

Set Size- Frontal Theta: Window 1

Exploratory analyses examining whether frontal theta for each set size of digits varied from WM task 1 and 2 were conducted. A Bayes factor was calculated for the model examine the main effect of set size, which ranged from 4 to 9 digits, with strong evidence for the null (B_{10} = 0.006 ±0.63%). Thus, there is not enough evidence to suggest that frontal theta from WM task 1 to WM task 2 are due to the main effect of set size. Examining the joint main effects of time, subject, set size, stress, and ACEs also indicated strong evidence for the null (B_{10} < 0.01 ±10.75%).

The examination of the interaction of set size by time also indicated strong evidence in favor of the null (B_{10} = 0.15 ±5.79%) suggesting no effect of the interaction on frontal theta. When examining all possible 2-way interactions, extremely strong evidence in favor of the null

(B_{10} < 0.01 ±3), suggesting no effect of 2-way interactions on frontal theta. Finally, the examination of the four-way interaction indicated extremely strong evidence in favor of the null (B_{10} < 0.01 ±90.53%) suggesting no effect of the 4-way interaction on frontal theta.

Set Size - Parietal Alpha: Window 1

Exploratory analyses examining whether parietal alpha for each set size of digits varied from WM task 1 and 2 were conducted. Therefore, a Bayes factor was calculated for the model examine the main effect of set size, which ranged from 4 to 9 digits, with very strong evidence for the null (B_{10} = 0.01±0.43%). Thus, there is not enough evidence to suggest that parietal alpha changes from WM task 1 to WM task 2 are due to the main effect of set size. Examining the joint main effects of time, subject, set size, stress, and ACEs also indicated extremely strong evidence in favor of the null (B_{10} < 0.01 ±9.95%).

The examination of the interaction of set size by time also indicated moderate evidence in favor of the null (B_{10} = 0.12 ±5.06%), suggesting no effect of the interaction on changes in parietal alpha. When examining all possible 2-way interactions, weak evidence (B_{10} < 0.01 ± 24.33%) suggests no effect of 2-way interactions on changes in parietal alpha. Finally, the examination of the four-way interaction indicated extremely strong evidence for the null (B_{10} < 0.01 ±62.95%) suggesting no effect of the 4-way interaction on changes in parietal alpha.

Table 4.
Working Memory by Set Size: Means and Standard Deviations

Accuracy						
Stress WM1	**4**	**5**	**6**	**7**	**8**	**9**
M	92.56	85.12	73.21	43.15	28.87	10.42
SD	9.71	12.08	23.61	26.55	31.06	17.07
N	28	28	28	28	28	28
Stress WM2						
M	96.13	83.04	69.35	44.35	29.17	13.99
SD	6.20	15.63	24.43	29.84	29.35	21.88
N	28	28	28	28	28	28
Neutral WM1						
M	90.28	82.29	59.03	33.33	14.24	4.51
SD	10.03	16.36	27.13	26.47	20.78	10.98
N	24	24	24	24	24	24
Neutral WM2						
M	92.71	82.64	59.38	31.94	20.14	7.64
SD	10.22	14.52	29.42	27.87	24.07	15.72
N	24	24	24	24	24	24
Fz Theta						
Stress WM1	**4**	**5**	**6**	**7**	**8**	**9**
M	11.81	13.80	14.41	13.84	13.58	13.78
SD	9.17	15.24	10.67	12.90	8.52	12.29
N	26	26	26	26	26	26
Stress WM2						
M	22.97	14.85	12.26	13.08	14.00	20.69
SD	32.70	12.10	8.77	8.52	12.14	37.20
N	26	26	26	26	26	26
Neutral WM1						
M	9.54	8.34	16.86	10.71	14.41	14.35
SD	6.98	7.27	23.20	8.12	23.82	30.68
N	24	24	24	24	24	24
Neutral WM2						
M	16.16	14.68	16.85	18.66	27.09	17.56
SD	16.67	11.51	17.70	24.67	46.63	19.86
N	24	24	24	24	24	24
FCz Theta						
Stress WM1	**4**	**5**	**6**	**7**	**8**	**9**
M	3.38	4.91	3.28	3.58	3.42	4.01
SD	3.80	10.61	4.17	3.16	3.98	5.29
N	26	26	26	26	26	26
Stress WM2						
M	10.11	3.21	2.79	2.75	2.69	13.81

SD	27.81	4.29	2.54	2.26	2.47	57.17
N	26	26	26	26	26	26
Neutral WM1						
M	3.60	2.11	4.96	2.65	3.50	4.82
SD	5.80	1.84	10.77	3.91	4.81	12.62
N	24	24	24	24	24	24
Neutral WM2						
M	3.32	3.23	2.77	7.48	9.75	4.26
SD	5.05	3.67	2.29	18.30	19.27	6.68
N	24	24	24	24	24	24
Cz Alpha						
Stress WM1	**4**	**5**	**6**	**7**	**8**	**9**
M	10.71	10.65	11.15	8.52	8.58	10.43
SD	8.31	8.41	8.10	4.42	3.64	6.26
N	26	26	26	26	26	26
Stress WM2						
M	9.70	10.34	9.59	9.75	9.89	12.15
SD	7.48	5.67	7.99	6.49	8.30	15.22
N	26	26	26	26	26	26
Neutral WM1						
M	8.09	9.89	11.92	12.84	15.73	16.96
SD	5.62	6.73	12.55	15.20	24.88	35.47
N	24	24	24	24	24	24
Neutral WM2						
M	10.47	12.63	11.30	13.06	18.71	12.33
SD	8.17	8.69	8.05	9.83	38.13	7.50
N	24	24	24	24	24	24
Pz Alpha						
Stress WM1	**4**	**5**	**6**	**7**	**8**	**9**
M	7.03	7.84	9.61	7.00	8.11	7.30
SD	5.55	10.50	14.31	9.03	9.95	8.26
N	26	26	26	26	26	26
Stress WM2						
M	8.09	8.58	7.94	6.71	6.74	9.08
SD	9.04	9.23	8.30	6.69	6.31	11.10
N	26	26	26	26	26	26
Neutral WM1						
M	8.50	9.44	10.79	9.55	8.72	11.03
SD	11.24	12.06	12.45	12.28	8.07	15.93
N	24	24	24	24	24	24
Neutral WM2						
M	7.60	9.69	12.58	10.18	12.28	9.16
SD	8.55	11.00	16.59	11.27	16.19	11.37
N	24	24	24	24	24	24
POz Alpha						
Stress WM1	**4**	**5**	**6**	**7**	**8**	**9**

M	4.00	3.89	3.70	3.96	3.82	3.48
SD	3.25	4.59	3.44	4.15	3.10	2.42
N	26	26	26	26	26	26
Stress WM2						
M	3.63	4.06	4.08	3.57	4.02	4.46
SD	2.82	3.09	3.34	2.04	2.68	4.96
N	26	26	26	26	26	26
Neutral WM1						
M	4.08	4.26	5.28	5.23	3.81	6.48
SD	3.99	3.42	6.49	6.01	3.45	13.14
N	24	24	24	24	24	24
Neutral WM2						
M	4.82	4.75	4.64	5.01	6.39	4.35
SD	5.46	5.50	5.24	7.11	9.55	5.59
N	24	24	24	24	24	24
Frontal Theta						
Stress WM1	**4**	**5**	**6**	**7**	**8**	**9**
M	7.59	9.35	8.84	8.71	10.54	8.89
SD	6.00	12.12	7.04	6.84	12.13	8.47
N	26	26	26	26	26	26
Stress WM2						
M	16.53	14.97	10.49	7.91	8.34	17.24
SD	29.30	30.91	16.20	4.78	6.94	46.84
N	26	26	26	26	26	26
Neutral WM1						
M	6.56	5.22	9.80	6.68	8.95	9.58
SD	5.90	4.12	9.38	5.07	14.17	21.59
N	24	24	24	24	24	24
Neutral WM2						
M	9.73	8.89	9.80	13.06	18.63	10.90
SD	9.58	6.50	9.38	20.84	31.33	11.96
N	24	24	24	24	24	24
Parietal Alpha						
Stress WM1	**4**	**5**	**6**	**7**	**8**	**9**
M	7.24	7.46	8.15	6.67	9.6	7.07
SD	4.68	6.28	7.55	3.8	16.42	4.19
N	26	26	26	26	26	26
Stress WM2						
M	7.14	8.81	10.39	11.60	6.88	8.56
SD	5.04	7.46	18.60	7.67	4.16	9.07
N	26	26	26	26	26	26
Neutral WM1						
M	6.88	7.86	9.50	9.20	9.42	11.48
SD	5.71	5.88	8.98	9.05	10.25	21.01
N	24	24	24	24	24	24

Neutral WM2						
M	7.63	9.02	9.50	9.41	12.45	8.61
SD	6.64	7.56	8.98	8.33	17.86	6.98
N	24	24	24	24	24	24

Set Size- Window 2

When examining a smaller epoch windows, no effects on the EEG data were observed during the onset of the first digit to the offset of the second digit for each trial within each set size. The epoch window selected for both the initial analysis and the exploratory analysis occurred during the first 2000ms of digit presentation, during which encoding of the first two digits is expected to occur for each set size. It is possible that for each set size, that as the cognitive load increases (i.e. encoding the fifth digit after encoding the first four digits) differences in theta and alpha may be detectable after several digits have been encoded. During a trial of set size 9 (i.e. 9 digits presentations) for example, once a participants' WM capacity has been reached after encoding 6 digits, the encoding process for the final 3 digits may involve recruitment of cognitive resources that were not necessary for encoding the first two digits of the trial.

Thus, the epoch window during the presentation of the first two digits may not capture the differences in theta and alpha that may occur as a result of having had to encode several digits during the course of the trial. Therefore, a second series of exploratory analyses of the EEG data were used to examine differences across set size during the presentation of the last two digits of every trial. Following the same procedure as the first series of exploratory analyses examining the condition of set size, epochs were extracted from the final 2000 ms of stimulus presentation. The condition of set size was the added to each model examining potential differences during this second time window of interest.

Set Size- EEG data processing: Window 2

The EEG data was first extracted in epochs of 2000 ms in duration from the onset of the second to last digit presentation to the end of each trial for every condition of set size. This produced epochs containing EEG data from the last 2000 ms of the stimulus presentation. Event-related spectral perturbation (ERSP) plots for each set size across all participants were then created in EEGLAB (see Figures 5-9, Apendix C).Visual inspection of the plots was used to determine the time window of interest in which the greatest amount of change in frontal theta and parietal alpha may occur (See Figure 4).

The EEG data for each set size was then re-epoched using the new time windows that were determined from the visual inspection of ERSP plots. Spectral analysis was conducted using EEGLAB and spectral power was calculated in 1Hz bins for each frequency in the range of 8-12Hz at electrode locations Fz, and FCz to examine theta activity. In addition to examining power at the two individual electrodes, mean power for the two electrodes was collapsed to create one Frontal Theta variable. To examine theta activity, power was calculated in 1Hz bins ranging from 4-8Hz at electrode locations Cz, Pz, and POz. In addition to examining power at the three individual electrodes, mean power for the three electrodes was collapsed to create one Parietal Alpha variable (means and standard deviations provided in Table 5).

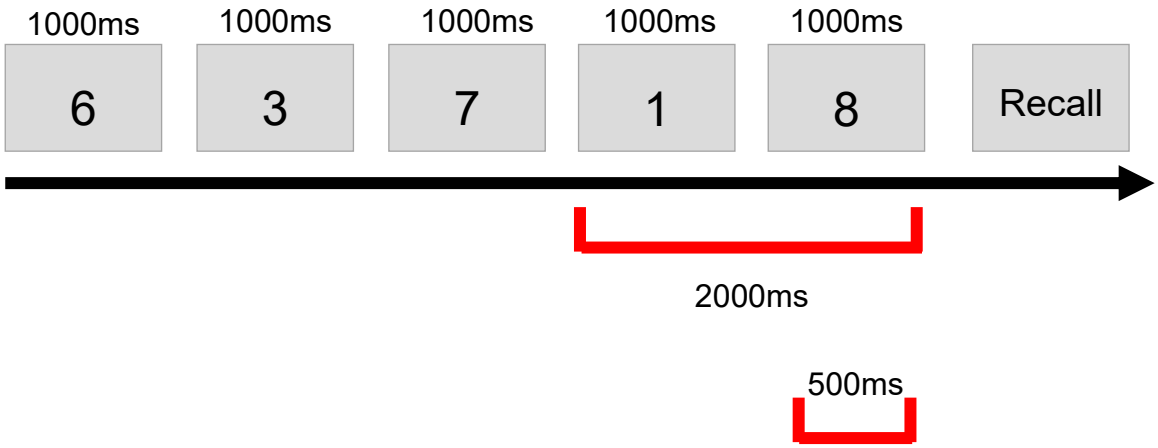

Figure 4: Time course of stimulus presentation and epoch window. Data was first extracted from beginning from the onset of the second to last digit to the offset of the last digit (2000ms). Visual inspection was then used to determine the time window within the epoched data in which the greatest changes in theta and alpha were likely to occur (for this example, 500ms).

Set Size- Frontal Theta: Window 2

Exploratory analyses examining whether frontal theta for each set size during the presentation of the last two digits within each trial varied from WM task 1 and 2 were conducted (means and standard deviations provided in Table 5). A Bayes factor was calculated for the model examine the main effect of set size, with moderate to strong evidence for the null (B_{10} = 0.21 ±6.03%). Thus, there is not enough evidence to suggest that frontal theta from WM task 1 to WM task 2 are due to the main effect of set size. Examining the joint main effects of time, subject, set size, stress, and ACEs also indicated extremely strong evidence in favor of the null (B_{10} = 0.03 ±2.46%).

The examination of the interaction of set size by time also indicated moderate evidence in favor of the null (B_{10} = 0.40 ±8.67%), suggesting no effect of the interaction on frontal theta. When examining all possible 2-way interactions, extremely strong evidence in favor of the null

(B_{10} = 0.02 ±11.45%), suggesting no effect of 2-way interactions on frontal theta. Finally, the examination of the four-way interaction indicated weak evidence (B_{10} < 0.01±3.73%) suggesting no effect of the 4-way interaction on frontal theta.

Set Size - Parietal Alpha: Window 2

Exploratory analyses examining whether parietal alpha for each which ranged from 4 to 9 digits varied from WM task 1 and 2 were conducted. Therefore, a Bayes factor was calculated for the model examine the main effect of set size, with extremely strong evidence in favor of the null (B_{10} = 0.35 ±7.36%). Thus, there is not enough evidence to suggest that from WM task 1 to WM task 2 are due to the main effect of set size. Examining the joint main effects of time, subject, set size, stress, and ACEs also indicated extremely strong evidence in favor of the null (B_{10} = 0.03 ±5.93%).

The examination of the interaction of set size by time also indicated moderate evidence in favor of the null (B_{10} = 0.36 ±6.89%), suggesting no effect of the interaction on parietal alpha. When examining all possible 2-way interactions, extremely strong evidence in favor of the null (B_{10} = 0.07 ±7.77%) suggests no effect of 2-way interactions on parietal alpha. Finally, the examination of the four-way interaction indicated extremely strong evidence in favor of the null (B_{10} < 0.01 ±8.9%) suggesting no effect of the 4-way interaction on changes in parietal alpha.

Table 5.
Working Memory by Set Size and Standard Deviations Last Two Digits

Accuracy						
Stress WM1	**4**	**5**	**6**	**7**	**8**	**9**
M	92.56	85.12	73.21	43.15	28.87	10.42
SD	9.71	12.08	23.61	26.55	31.06	17.07
N	28	28	28	28	28	28
Stress WM2						
M	96.131	83.0357	69.35	44.35	29.17	13.99
SD	6.2039	15.6285	24.43	29.84	29.35	21.88
N	28	28	28	28	28	28
Neutral WM1						
M	90.28	82.29	59.03	33.33	14.24	4.51
SD	10.03	16.36	27.13	26.47	20.78	10.98
N	24	24	24	24	24	24
Neutral WM2						
M	92.71	82.6389	59.38	31.94	20.14	7.64
SD	10.22	14.5207	29.42	27.87	24.07	15.72
N	24	24	24	24	24	24
Fz Theta						
Stress WM1	**4**	**5**	**6**	**7**	**8**	**9**
M	10.90	10.02	10.78	12.98	12.19	9.57
SD	7.03	9.39	9.88	9.55	9.60	7.60
N	26	26	26	26	26	26
Stress WM2						
M	12.51	15.21	13.40	16.01	12.15	11.72
SD	16.67	17.51	13.01	24.70	12.09	9.08
N	26	26	26	26	26	26
Neutral WM1						
M	10.44	12.87	10.54	10.83	10.02	13.82
SD	8.09	11.90	5.98	7.37	7.45	20.94
N	24	24	24	24	24	24
Neutral WM2						
M	12.21	13.52	19.46	13.45	17.39	13.01
SD	7.52	11.10	18.34	11.43	21.35	8.60
N	24	24	24	24	24	24
FCz Theta						
Stress WM1	**4**	**5**	**6**	**7**	**8**	**9**
M	3.16	2.12	2.23	2.97	2.14	3.48

SD	4.11	1.74	1.81	3.51	2.42	4.53
N	26	26	26	26	26	26
Stress WM2						
M	7.78	8.10	2.23	2.45	2.27	2.16
SD	21.23	29.29	1.95	2.74	2.20	1.67
N	26	26	26	26	26	26
Neutral WM1						
M	3.23	3.00	3.59008	2.38	2.87	5.53
SD	4.97	4.41	6.46939	3.82	4.81	17.07
N	24	24	24	24	24	24
Neutral WM2						
M	4.09	3.71	6.26	4.54	4.43	2.93
SD	8.25	6.44	13.06	6.84	7.25	4.48
N	24	24	24	24	24	24
Cz Alpha						
Stress WM1	**4**	**5**	**6**	**7**	**8**	**9**
M	11.74	11.82	11.03	13.05	10.85	15.64
SD	7.40	6.67	9.47	7.97	6.11	19.58
N	26	26	26	26	26	26
Stress WM2						
M	12.91	15.40	10.08	11.90	11.22	12.45
SD	11.16	20.97	7.62	8.13	7.58	8.50
N	26	26	26	26	26	26
Neutral WM1						
M	12.00	13.07	12.37	11.80	11.17	14.03
SD	5.10	9.12	8.91	8.85	8.84	15.06
N	24	24	24	24	24	24
Neutral WM2						
M	11.96	9.87	10.44	10.34	13.65	12.98
SD	8.25	5.24	6.19	5.78	14.09	9.34
N	24	24	24	24	24	24
Pz Alpha						
Stress WM1	**4**	**5**	**6**	**7**	**8**	**9**
M	5.76	4.86	5.33	5.12	6.34	5.44
SD	5.11	3.79	5.66	3.45	5.07	4.19
N	26	26	26	26	26	26
Stress WM2						
M	7.63	6.46	5.92	5.71	6.45	6.42
SD	12.17	5.53	5.61	4.43	5.97	7.19
N	26	26	26	26	26	26

Neutral WM1						
M	6.45	7.47	12.37	8.52	6.68	5.35
SD	7.03	8.84	8.91	9.23	6.70	4.43
N	24	24	24	24	24	24
Neutral WM2						
M	6.06	7.07	6.07	5.55	6.80	6.88
SD	5.66	8.58	5.23	5.66	6.27	5.58
N	24	24	24	24	24	24
Poz Alpha						
Stress WM1	**4**	**5**	**6**	**7**	**8**	**9**
M	3.49	3.09	2.74	2.91	3.01	3.50
SD	4.45	2.33	1.77	2.04	1.38	4.06
N	26	26	26	26	26	26
Stress WM2						
M	3.56	3.60	3.14	2.91	3.01	3.50
SD	3.42	3.07	1.76	2.04	1.38	4.06
N	26	26	26	26	26	26
Neutral WM1						
M	2.97	3.68	3.51	4.47	3.67	2.76
SD	2.88	4.19	3.20	5.17	3.29	2.16
N	24	24	24	24	24	24
Neutral WM2						
M	2.94	3.24	2.66	3.23	3.78	3.27
SD	2.39	3.48	1.76	3.11	3.99	2.87
N	24	24	24	24	24	24
Frontal Theta						
Stress WM1	**4**	**5**	**6**	**7**	**8**	**9**
M	7.03	11.67	6.50	7.97	7.16	6.52
SD	4.55	30.43	5.37	5.50	5.68	5.47
N	26	26	26	26	26	26
Stress WM2						
M	8.15	11.65	6.38	9.23	7.20	6.94
SD	6.97	33.87	4.02	12.81	6.45	4.96
N	26	26	26	26	26	26
Neutral WM1						
M	6.83	7.93	7.06	6.60	6.44	9.67
SD	5.59	7.60	4.93	4.60	5.73	18.83
N	24	24	24	24	24	24
Neutral WM2						
M	8.15	8.61	12.86	8.99	10.91	7.97

SD	6.97	7.41	14.19	8.77	13.03	5.83
N	24	24	24	24	24	24
Parietal Alpha						
Stress WM1	**4**	**5**	**6**	**7**	**8**	**9**
M	6.99	30.9	6.39	6.91	7.04	8.18
SD	4.46	119.3	4.41	3.25	3.93	8.40
N	26	26	26	26	26	26
Stress WM2						
M	6.98	8.48	10.45	6.84	6.89	7.47
SD	4.60	8.41	7.79	4.14	3.73	5.00
N	26	26	26	26	26	26
Neutral WM1						
M	7.14	8.07	7.81	8.26	7.17	7.37
SD	3.77	6.08	6.95	7.07	4.78	6.79
N	24	24	24	24	24	24
Neutral WM2						
M	6.98	6.72	6.39	6.37	8.07	7.71
SD	4.60	4.45	3.75	4.10	7.37	4.46
N	24	24	24	24	24	24

Chapter 6. Discussion

The current study sought to examine the potential relationship between stress and working memory performance in an area that has yet to be investigated. Previous research has examined the influence of acute stress on working memory, with inconsistent findings. Some research has indicated a negative relationship, while others have found little to no relationship. The current study is the first to examine whether a relationship exists between adverse childhood experiences, acute stress, and working memory. As the findings on the impact of acute stress on working memory have been mixed (Porcelli, 2008), with demonstrations of negative effects on working memory (Porcelli et al., 2008; Kirschbaum et al., 1996; Kuhlmann et al., 2005; Oei et al., 2006; Patil et al., 1995), and no effects on WM performance (Domes et al., 2002; McMorris et al., 2006; Smeets et al., 2006), the relationship between acute stress and working memory remains somewhat unclear. Thus, the examination of factors that may impact the relationship between acute stress and working memory may provide clarity into the inconsistent research findings.

Working memory performance has been shown to vary across individuals and has been implicated as an important component of problem solving and day to day functioning (Baddeley, 1992; Baddeley et al., 1986; Baddeley, 2003; Dong et al., 2015; Duncan et al., 2000; Gevins, 1997; Goel & Grafman, 1995; Levy & Goldman-Rakic, 2000; Prabhakaran et al., 2001). This variation across individuals may reflect differences in communication between brain structures, and efficiency of communication is dependent upon experiences that influence brain development.

The suggestion that exposure to adverse childhood experiences is associated with increased risk for cognitive delays (Anda et al., 2006; Levy & Goldman-Rakic, 2000) has been

further supported by research indicating negative impacts on brain development associated with adverse childhood experiences (Bellis & Zisk, 2014; Dunlop, Archer, Quinlivan, Beazley, & Newnham, 1997; Martin H Teicher et al., 2002). As working memory is an important component of higher order cognitive functions (Baddeley, 1992; Baddeley et al., 1986; Baddeley, 2003; Dong et al., 2015; Duncan et al., 2000; Goel & Grafman, 1995; Levy & Goldman-Rakic, 2000; Prabhakaran, Rypma, & Gabrieli, 2001), which are necessary for academic and occupational performance (Alloway, 2009; Alloway & Alloway, 2010; Dong et al., 2015), understanding stress related factors that impact working memory could aid in identifying strategies and development of intervention to improve in these areas. Therefore, the current study sought to use participants' scores on the ACEs questionnaire to examine potential individual differences in WM performance, the relationship between experience of acute stress and WM performance, and WM related changes in frequency power.

Presently, the findings of the current study do not provide conclusive evidence to suggest that there is a relationship between ACEs, the experience of acute stress, WM performance, and WM related changes in frequency power. Using a Bayes Factor for model comparisons to examine the main effects of stress and ACEs, as well as interactions of stress by ACEs indicated weak to moderate evidence in favor of the null for each model examining WM performance (Kass & Raftery, 1995). Thus, results indicated that neither the stress induction nor previous exposure to stressful events in the form of ACEs seem to be related to WM performance on the digit span task. Model comparison also demonstrated weak to strong evidence in favor of the null for models examining frontal theta and parietal, also indicating no effects of acute stress and ACEs.

Self-report scores on the PANAS measure were used to ensure that the stress induction was effective in producing changes in affect for participants in the stress condition. It was hypothesized that if the stress induction were successful, positive affect for the group who experienced an acute stressor would be lower than participants who were did not experience the stressor. It was also hypothesized that negative affect would be increased for the stress group compared to the neutral group. According to the Bayes Factors calculated, it appears there is strong evidence to suggest that participants who experienced the acute stressor had a decrease in positive affect, as well as strong evidence for an increase in negative affect compared to the neutral group. Thus, results suggest that the acute stressor used for the current study may have produced the desired stress response. Therefore, it can be concluded from these findings that the successful stress induction was not related to changes in WM performance.

The PANAS was used to measure the effectiveness of the stress induction, which indicated that the acute stressor increased negative affect and decreased positive affect for participants in the stress group, compared to the increase in positive affect for the neutral group. This suggests that changes in scores on the PANAS could be used as a measure of stress in the models rather than dichotomously coding participants as either being in the stress or neutral condition. Therefore, exploratory analyses were conducted to investigate whether observed changes in PANAS scores could be a more sensitive measure of stress that could be used to detect effects of stress that the categorical variable of group may not have been sensitive enough to reveal. These exploratory analyses were conducted by replacing the variable of stress within the models with PA change scores and NA change scores. When examining the main effect of PA change score, the Bayes factor indicated strong evidence for a difference in PA between the two groups. However, all other Bayes factors demonstrated weak to strong evidence for the null

for each of the models. Thus, results indicate that the evidence does not reveal effects of stress as measured by the PANAS on WM performance, frontal theta, and parietal alpha.

While the initial hypotheses and analyses examined differences between the set size at which each participant reached their WM capacity, further differences could potentially be found at other levels of difficulty, i.e. either before or after WM capacity is reached. It is possible that effects of stress or ACEs may be associated with WM performance, WM-related theta, and WM-related alpha as the number of digits that each participant must hold in WM increases. At the beginning of the task, participants were shown a set size of 4, which required them to maintain 4 digits in WM. After every 4 trials, another digit was added to the set size, with the largest set size presenting 9 digits. Differences in WM performance, WM-related theta, and WM-related alpha could also occur at each set size. For example, when comparing differences in WM capacity for each participant, comparisons were made between theta and alpha for a participant whose WM capacity set size was 6 digits, while another participant's WM capacity set size was 4 digits. Furthermore, a potential limitation of the initial analyses may lie in the length of the epoch window used for examining differences in theta and alpha frequencies. Therefore, the examination of shorter windows may better localize changes in frequency.

Thus, comparisons could be also be made between these participants at the same set size, i.e., what are the differences between groups at set size 4. Therefore, additional analyses were conducted by adding the condition of set size to each of the models that were previously tested. To address the limitation of the epoch window length, these analyses included measures of theta and alpha that were calculated by extracting shorter epoch windows. Bayes factors for each model comparison indicated weak to strong evidence in favor of the null. Thus, with the addition of the variable of set size in the models, the analyses of the effect of set size also indicated there

is no evidence that the acute stressor nor ACEs scores were associated with differences in WM performance, frontal theta, or parietal alpha from WM task 1 to WM task 2.

The final series of exploratory analyses considered the condition set size and the more localized and shorter time windows, but specifically were used to examine whether effects could be detected later during the encoding process for each trial. It is possible that by examining the changes in frontal theta and parietal alpha during the presentation of the first two digits of each set size that differences were not present, as encoding the first two digits for each set size results in the same amount of information being maintained across trials. Whereas by examining the final 2000 ms for each set size, participants have had to maintain more information by the end of the trial for large set sizes than for smaller set sizes.

For example, when presented a set size of five digits, participants were required to encode and maintain three digits prior to the final two digits; when presented a size of nine digits, participants were required to encode and maintain seven digits prior to the presentation of the final two digits. As it is suggested the frontal theta and parietal alpha should increase as the task gets more difficult, it may be that differences across set size would be more likely toward the end of each trial. Therefore, the time window for the epochs was shifted to examine the changes in power during the presentation of the last two digits for each set size. The condition of set size was added to each of the models, and for each model the Bayes factor indicated weak to strong evidence in favor of the null. These results indicate that there is no evidence for the effects of acute stress and ACEs on frequency at the end of each trial.

As measurement of the PANAS was used to determine whether stress was induced, the conclusion that may be drawn is that the experience of the acute stressor for the current study may have produced a stress response, but that stress response did not have an impact on WM

performance or changes in frequency. These findings are consistent with Domes and colleagues (2002), who examined the relationship between a psychosocial stressor and cortisol levels and found no impact on working memory performance in women. Also evaluating the effect of a psychosocial stressor on WM performance, Smeets and colleagues (2006) found no effect of their acute stressor on participants' performance on the digit span task, like the task used in the current study. These findings are further supported by McMorris and colleagues (2006), who reported heat stress did not impact performance on a spatial or verbal recall task.

The current study did not use measure of cortisol to determine the impact of the stress induction. However, the rationale for predicting a decline in WM performance due to stress induction was rooted in previous research demonstrating the acute stress is associated with increases in cortisol. It is possible that the stress induction method used for the current study, while sufficient for altering self-report changes in affect, may have produced too little cortisol to have an impact on WM. As Domes et al. 2002 noted in the discussion of their findings, memory that involves retrieval that is associated with hippocampal functioning appears to be the most impacted by increases in cortisol. As for the current study, examining the time in which participants were instructed to recall and respond may have revealed retrieval-related frequency differences that were associated with an acute stressor or ACEs. Therefore, future studies using a task such as the digit span may observe effects during retrieval that were not observed during the encoding trials.

Another potential limitation of the design may have been the use of the ACEs measure to examine the effects of early life stressors. While the ACEs measure has become widely used to inform research and interventions, the measure itself is not without its limitations. The measure has faced several criticisms, one of which being a limitation in the composite nature of the score

that is given. It has been suggested that the variables examined with the ACEs items are not precise. The items on the measure do not reflect the severity, duration, or timing of the types of stressors experienced. Furthermore, critiques of the psychometric properties of the measure have highlighted the failure of the ACEs measure from ensuring that respondents answer only one question at a time or containing "double-barreled" questions (Asmundson & Afifi, 2019). As previously noted, both the severity and type of stress experienced is associated with detriments in brain development (Bick & Nelson, 2016). Therefore, it is possible that one specific type of stressor may be associated with detriments to WM functioning. The ACEs questionnaire provides a composite score of several types of stressors. In understanding the relationship between WM and early life stressors, research may benefit from using a measure that examines a specific type of stressor that may be specifically related to detriments to WM functioning. future studies examining the relationship between exposure to acute stress and early life stressors/adverse childhood experience may benefit from focusing on specific domain of adverse experiences instead of a whole range,

Furthermore, a limitation noted of ACEs is that they may be indirectly associated with negative physical and mental health outcomes through other pathways (Finkelhor, 2018). It has been noted that outcomes may be associated with ACEs through risk taking behaviors or poor health practices (Finkelhor, 2018), and researchers caution the conclusions that can be drawn about retrospective reports of child maltreatment and health outcomes (e.g., Raphael, Chandler, & Ciccone, 2004; Widom, Raphael, & DuMont, 2004; White, Widom, & Chen, 2007). As the current study was cross sectional and used retrospective reporting, it may not have captured the time in which working memory functioning may be most susceptible to the experience of an acute stressor. Future studies using a longitudinal design to assess working memory functioning

during potentially critical periods of development into adulthood may further the understanding of how stress may impact memory functioning during different periods of development.

A potential factor that was not considered in the current study is the impact of alcohol and substance use during adolescence. Maltreatment in the form of emotional abuse, sexual abuse, and physical abuse experienced during adolescence has been shown to be associated with increased risk for substance use (Ireland et al., 2002; Moran et al., 2004; Tonmyr et al., 2010). As previously discussed, there may be critical periods of brain development that occur during adolescence in which stressors may have the most impact. It is suggested that detriments to brain development may be associated with neurotoxicity of substance and alcohol use during adolescence (Monti et al., 2005). Both adolescent alcohol use and marijuana use has been shown to be associated with reduced hippocampal volumes (Medina et al., 2007).

It is possible that participants within our sample who had higher ACEs scores may have engaged in less alcohol and substance use than what has been observed in samples in previous studies. Thus, participants in our sample with high ACEs scores may not reflect differences in working memory functioning as they may not have engaged in risk-taking behaviors and poor health practices (Finkelhor, 2018) that may be related to exposure to ACEs and outcomes later in life. The sample of the current study came what may be considered a rural, Southern town. It is possible that rates of adolescent alcohol and substance use may be below the national average. It is possible that an influence of local cultural norms are associated with a decrease in use during adolescence. In future, studies that seek to understand the relationship between acute stress, adverse childhood experiences, and memory functioning may benefit from including measures of alcohol and substance use during adolescence, as well as measures of current use.

Exposure to ACEs and outcomes for college students is still an area of research that needs to be explored (Khrapatina & Berman, 2017). Research examining the relationship between ACEs and socioeconomic status has indicated that those with higher ACEs scores are less likely to complete a high school degree (Metzler et al., 2017). Therefore, those with higher ACEs scores may be less likely to attend college. A strong relationship between academic performance and working memory functioning has been demonstrated (Alloway, 2009; Simmering & Perone, 2013). As the sample for the current study consists of college students, perhaps the participants within our sample with high ACEs scores have experienced less detriments to working memory functioning than non-college students with high ACEs scores. Research into the differences in working memory functioning among those with high ACEs scores across education level may assist in assessing whether a relationship between ACEs, working memory, and academic performance exists.

The topic of the role of resilience in mental health has grown in attention over the past several years, with research examining the protective factors that promote better health outcomes for those who have been exposed to ACEs (Poole et al., 2017). Resilience seems to be a multidimensional construct that reflect an ability to adapt and cope in the midst of adversity (Bonanno, 2004; Poole et al., 2017). The students within our sample with higher ACEs scores may be a subset of those who are higher in resilience and have more protective factors that may serve as a buffer for the negative mental, physical, and socioeconomic outcomes. Therefore, future research may better understand the relationship between working memory and previous exposure to stressful events during childhood by also measuring protective factors that promote better outcomes for those with high ACEs scores.

It is also possible that the effects of acute stress on working memory performance may reflect a complex relationship between the stress-response system and memory functioning. While the scores on the PANAS indicated that a stress response was produced, it is possible the acute stressor created a hypervigilant processing state (Aston-Jones & Cohen, 2005), which may actually be associated with memory-improvements (Henckens et al., 2009). Furthermore, studies have shown that stress has different effects on different types of memory, such as spatial or verbal memory (Gärtner et al., 2014; Shackman et al., 2006; Vytal et al., 2012).

Therefore, another potential avenue to explore in future studies examining the relationship between an acute stressor, adverse childhood experiences, and working memory may be the use of another working memory task instead of the digit span to assess effects on working memory functioning. As working memory is a complex mechanism, different working memory tasks are thought to measure different aspects of working memory functioning, and low correlations between these tasks are often reported (Engle et al., 1999; Wilhelm et al., 2013). For example, Gärtner et al. (2014) found an effect of an acute stressor on working memory performance and frontal theta using the n-back task. Distinctions between tasks can be made based on the processes the specific task seems to measure, and can be broken down into what is known as passive or active storage (Vecchi et al., 2005; Vecchi & Girelli, 1998). The digit span task involves the recall of information which may be considered passive storage, as participants were asked to simply recall the information that was presented. Perhaps a working memory task involving more manipulation of information would be more sensitive to the effects of an acute stressor.

In conclusion, the present findings do not support the hypothesized relationship between the experience of an acute stressor, adverse childhood experiences, and working memory.

Further research should explore whether this relationship exists using other types of working memory tasks and modalities. Future directions for research should potentially take into account the complexity of the current study's design. Perhaps establishing an effect of stress on a working memory task that has been shown to be sensitive to effects of acute stress should be the first step. Then, once the effect of acute stress has been established, introducing the exposure to ACEs as a variable of interest in the relationship may provide more insight.

References

Alloway, T. P. (2009). Working Memory, but Not IQ, Predicts Subsequent Learning in Children with Learning Difficulties. *European Journal of Psychological Assessment*, *25*(2), 92–98. https://doi.org/10.1027/1015-5759.25.2.92

Alloway, T. P., & Alloway, R. G. (2010). Investigating the predictive roles of working memory and IQ in academic attainment. *Journal of Experimental Child Psychology*, *106*(1), 20–29. https://doi.org/10.1016/j.jecp.2009.11.003

Anda, R. F., Felitti, V. J., Bremner, J. D., Walker, J. D., Whitfield, C., Perry, B. D., … Giles, W. H. (2006). The enduring effects of abuse and related adverse experiences in childhood. *European Archives of Psychiatry and Clinical Neuroscience*, *256*(3), 174–186.

Anderson, K. L., Rajagovindan, R., Ghacibeh, G. A., Meador, K. J., & Ding, M. (2010). Theta Oscillations Mediate Interaction between Prefrontal Cortex and Medial Temporal Lobe in Human Memory. *Cerebral Cortex*, *20*(7), 1604–1612. https://doi.org/10.1093/cercor/bhp223

Anderson, K. L., Rajagovindan, R., Ghacibeh, G. A., Meador, K. J., & Ding, M. (2010). Theta Oscillations Mediate Interaction between Prefrontal Cortex and Medial Temporal Lobe in Human Memory. *Cerebral Cortex*, *20*(7), 1604–1612. https://doi.org/10.1093/cercor/bhp223

Arnolds, D. E. A. T., Lopes da Silva, F. H., Aitink, J. W., Kamp, A., & Boeijinga, P. (1980). The Spectral Properties of Hippocampal EEG Related to Behavior in Man. *Electroencephalography and Clinical Neurophiosology*, *50*, 324–328.

Arnsten, A. F. T. (2007). Catecholamine and Second Messenger Influences on Prefrontal Cortical Networks of "Representational Knowledge": A Rational Bridge between

Genetics and the Symptoms of Mental Illness. *Cerebral Cortex*, *17*(suppl_1), i6–i15. https://doi.org/10.1093/cercor/bhm033

Arnsten, A. F. T. (2007). Catecholamine and Second Messenger Influences on Prefrontal Cortical Networks of "Representational Knowledge": A Rational Bridge between Genetics and the Symptoms of Mental Illness. *Cerebral Cortex*, *17*(suppl_1), i6–i15. https://doi.org/10.1093/cercor/bhm033

Arnsten, A. F. T. (2009). Stress signalling pathways that impair prefrontal cortex structure and function. *Nature Reviews. Neuroscience*, *10*(6), 410–422. https://doi.org/10.1038/nrn2648

Arnsten, A. F. T. (2009). Stress signalling pathways that impair prefrontal cortex structure and function. *Nature Reviews. Neuroscience*, *10*(6), 410–422. https://doi.org/10.1038/nrn2648

Arnsten, A. F. T., & Li, B.-M. (2005). Neurobiology of Executive Functions: Catecholamine Influences on Prefrontal Cortical Functions. *Biological Psychiatry*, *57*(11), 1377–1384. https://doi.org/10.1016/j.biopsych.2004.08.019

Arnsten, A. F. T., & Li, B.-M. (2005). Neurobiology of Executive Functions: Catecholamine Influences on Prefrontal Cortical Functions. *Biological Psychiatry*, *57*(11), 1377–1384. https://doi.org/10.1016/j.biopsych.2004.08.019

Arnsten, A. F. T., Mathew, R., Ubriani, R., Taylor, J. R., & Li, B.-M. (1999). α-1 noradrenergic receptor stimulation impairs prefrontal cortical cognitive function. *Biological Psychiatry*, *45*(1), 26–31. https://doi.org/10.1016/S0006-3223(98)00296-0

Arnsten, A. F. T., Mathew, R., Ubriani, R., Taylor, J. R., & Li, B.-M. (1999). α-1 noradrenergic receptor stimulation impairs prefrontal cortical cognitive function. *Biological Psychiatry*, *45*(1), 26–31. https://doi.org/10.1016/S0006-3223(98)00296-0

Asada, H., Fukuda, Y., Tsunoda, S., Yamaguchi, M., & Tonoike, M. (1999). Frontal midline theta rhythms reflect alternative activation of prefrontal cortex and anterior cingulate cortex in humans. *Neuroscience Letters*, *274*(1), 29–32. https://doi.org/10.1016/S0304-3940(99)00679-5

Asmundson, G. J. G., & Afifi, T. O. (2019). *Adverse Childhood Experiences: Using Evidence to Advance Research, Practice, Policy, and Prevention*. Academic Press.

Aston-Jones, G., & Cohen, J. D. (2005). An Integrative Theory of Locus Coeruleus-Norepinephrine Function: Adaptive Gain and Optimal Performance. *Annual Review of Neuroscience*, *28*(1), 403–450. https://doi.org/10.1146/annurev.neuro.28.061604.135709

Baddeley, A. (1992). Working memory. *Science (Washington)*, *255*(5044), 556–559.

Baddeley, A., Logie, R., Bressi, S., Sala, S. D., & Spinnler, H. (1986). Dementia and Working Memory. *The Quarterly Journal of Experimental Psychology Section A*, *38*(4), 603–618. https://doi.org/10.1080/14640748608401616

Baddeley, Alan. (2003). Working memory: looking back and looking forward. *Nature Reviews Neuroscience*, *4*(10), 829–839. https://doi.org/10.1038/nrn1201

Baddeley, Alan. (2010). Working memory. *Current Biology*, *20*(4), R136–R140. https://doi.org/10.1016/j.cub.2009.12.014

Bastiaansen, M., & Hagoort, P. (2003). Event-Induced Theta Responses as a Window on the Dynamics of Memory. *Cortex*, *39*(4), 967–992. https://doi.org/10.1016/S0010-9452(08)70873-6

Bellis, M. D. D., & Zisk, A. (2014). The Biological Effects of Childhood Trauma. *Child and Adolescent Psychiatric Clinics*, *23*(2), 185–222. https://doi.org/10.1016/j.chc.2014.01.002

Bellis, M. D. D., & Zisk, A. (2014). The Biological Effects of Childhood Trauma. *Child and Adolescent Psychiatric Clinics*, *23*(2), 185–222. https://doi.org/10.1016/j.chc.2014.01.002

Bick, J., & Nelson, C. A. (2016). Early Adverse Experiences and the Developing Brain. *Neuropsychopharmacology*, *41*, 177–196. https://doi.org/10.1038/npp.2015.252

Bick, J., & Nelson, C. A. (2016). Early Adverse Experiences and the Developing Brain. *Neuropsychopharmacology*, *41*, 177–196. https://doi.org/10.1038/npp.2015.252

Bonanno, G. A. (2004). Loss, Trauma, and Human Resilience: Have We Underestimated the Human Capacity to Thrive After Extremely Aversive Events? *American Psychologist*, *59*(1), 20–28. https://doi.org/10.1037/0003-066X.59.1.20

Bremner, J. D., Randall, P., Vermetten, E., Staib, L., Bronen, R. A., Mazure, C., … Charney, D. S. (1997). Magnetic resonance imaging-based measurement of hippocampal volume in posttraumatic stress disorder related to childhood physical and sexual abuse—a preliminary report. *Biological Psychiatry*, *41*(1), 23–32. https://doi.org/10.1016/S0006-3223(96)00162-X

Brody, B. A., Kinney, H. C., Kloman, A. S., & Gilles, F. H. (1987). Sequence of central nervous system myelination in human infancy. I. An autopsy study of myelination. *Journal of Neuropathology and Experimental Neurology*, *46*(3), 283–301.

Buckner, R. L., & Wheeler, M. E. (2001). The cognitive neuroscience og remembering. *Nature Reviews Neuroscience*, *2*(9), 624–634. https://doi.org/10.1038/35090048

Carpenter, L. L., Carvalho, J. P., Tyrka, A. R., Wier, L. M., Mello, A. F., Mello, M. F., … 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. https://doi.org/10.1016/j.biopsych.2007.05.002

Carrey, N. J., Butter, H. J., Persinger, M. A., & Bialik, R. J. (1995). Physiological and Cognitive Correlates of Child Abuse. *Journal of the American Academy of Child & Adolescent Psychiatry*, *34*(8), 1067–1075. https://doi.org/10.1097/00004583-199508000-00017

Carrion, V. G., Weems, C. F., Eliez, S., Patwardhan, A., Brown, W., Ray, R. D., & Reiss, A. L. (2001). Attenuation of frontal asymmetry in pediatric posttraumatic stress disorder. *Biological Psychiatry*, *50*(12), 943–951. https://doi.org/10.1016/S0006-3223(01)01218-5

Cavanagh, J. F., & Frank, M. J. (2014). Frontal theta as a mechanism for cognitive control. *Trends in Cognitive Sciences*, *18*(8), 414–421. https://doi.org/10.1016/j.tics.2014.04.012

Chrousos, G. P., & Gold, P. W. (1992). The Concepts of Stress and Stress System Disorders: Overview of Physical and Behavioral Homeostasis. *JAMA*, *267*(9), 1244–1252. https://doi.org/10.1001/jama.1992.03480090092034

Clayton, M. S., Yeung, N., & Cohen Kadosh, R. (2015). The roles of cortical oscillations in sustained attention. *Trends in Cognitive Sciences*, *19*(4), 188–195. https://doi.org/10.1016/j.tics.2015.02.004

Cohen, M. X. (2011). Error-related medial frontal theta activity predicts cingulate-related structural connectivity. *NeuroImage*, *55*(3), 1373–1383. https://doi.org/10.1016/j.neuroimage.2010.12.072

Cohen, M. X. (2011). Error-related medial frontal theta activity predicts cingulate-related structural connectivity. *NeuroImage*, *55*(3), 1373–1383. https://doi.org/10.1016/j.neuroimage.2010.12.072

Conway, A., R. A., Kane, M. J., Bunting, M. F., Hambrick, D. Z., Wilhelm, O., & Engle, R. W. (2005). Working memory span tasks: A methodological review and user's guide. *Psychonomic Bulletin & Review*, *12*(5), 769–786. https://doi.org/10.3758/BF03196772

Cooper, N. R., Croft, R. J., Dominey, S. J. J., Burgess, A. P., & Gruzelier, J. H. (2003). Paradox lost? Exploring the role of alpha oscillations during externally vs. internally directed attention and the implications for idling and inhibition hypotheses. *International Journal of Psychophysiology*, *47*(1), 65–74. https://doi.org/10.1016/S0167-8760(02)00107-1

Cowan, N. (2001). The magical number 4 in short-term memory: A reconsideration of mental storage capacity. *Behavioral and Brain Sciences*, *24*(1), 87–114. https://doi.org/10.1017/S0140525X01003922

Cowan, N. (2001). The magical number 4 in short-term memory: A reconsideration of mental storage capacity. *Behavioral and Brain Sciences*, *24*(1), 87–114. https://doi.org/10.1017/S0140525X01003922

Cowell, R. A., Bussey, T. J., & Saksida, L. M. (2010). Functional dissociations within the ventral object processing pathway: Cognitive modules or a hierarchical continuum? *Journal of Cognitive Neuroscience*, *22*(11), 2460–2479. https://doi.org/10.1162/jocn.2009.21373

Culp, R. E., Little, V., Letts, D., & Lawrence, H. (1991). Maltreated children's self-concept: Effects of a comprehensive treatment program. *American Journal of Orthopsychiatry*, *61*(1), 114–121. https://doi.org/10.1037/h0079233

De Bellis, M. D. (2001). Developmental traumatology: the psychobiological development of maltreated children and its implications for research, treatment, and policy. *Development and Psychopathology*, *13*(3), 539–564.

De Bellis, Michael 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∗∗See accompanying Editorial, in this issue. *Biological Psychiatry*, *45*(10), 1259–1270. https://doi.org/10.1016/S0006-3223(99)00044-X

De Bellis, Michael D., Keshavan, M. S., Shifflett, H., Iyengar, S., Beers, S. R., Hall, J., & Moritz, G. (2002). Brain structures in pediatric maltreatment-related posttraumatic stress disorder: a sociodemographically matched study. *Biological Psychiatry*, *52*(11), 1066–1078. https://doi.org/10.1016/S0006-3223(02)01459-2

Delorme, A., & Makeig, S. (2004). EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *Journal of Neuroscience Methods*, *134*(1), 9–21. https://doi.org/10.1016/j.jneumeth.2003.10.009

Dempster, F. N., & Cooney, J. B. (1982). Individual differences in digit span, susceptibility to proactive interference, and aptitude/achievement test scores. *Intelligence*, *6*(4), 399–416. https://doi.org/10.1016/0160-2896(82)90026-5

Diamond, D. M., Park, C. R., Heman, K. L., & Rose, G. M. (1999). Exposing rats to a predator impairs spatial working memory in the radial arm water maze. *Hippocampus*, *9*(5), 542–552. https://doi.org/10.1002/(SICI)1098-1063(1999)9:5<542::AID-HIPO8>3.0.CO;2-N

Diamond, David M., Fleshner, M., Ingersoll, N., & Rose, G. (1996). Psychological stress impairs spatial working memory: Relevance to electrophysiological studies of hippocampal

function. *Behavioral Neuroscience*, *110*(4), 661–672. https://doi.org/10.1037/0735-7044.110.4.661

Domes, G., Heinrichs, M., Reichwald, U., & Hautzinger, M. (2002). Hypothalamic–pituitary–adrenal axis reactivity to psychological stress and memory in middle-aged women: High responders exhibit enhanced declarative memory performance. *Psychoneuroendocrinology*, *27*(7), 843–853. https://doi.org/10.1016/S0306-4530(01)00085-3

Domes, G., Heinrichs, M., Reichwald, U., & Hautzinger, M. (2002). Hypothalamic–pituitary–adrenal axis reactivity to psychological stress and memory in middle-aged women: high responders exhibit enhanced declarative memory performance. *Psychoneuroendocrinology*, *27*(7), 843–853. https://doi.org/10.1016/S0306-4530(01)00085-3

Dong, S., Reder, L. M., Yao, Y., Liu, Y., & Chen, F. (2015). Individual differences in working memory capacity are reflected in different ERP and EEG patterns to task difficulty. *Brain Research*, *1616*, 146–156. https://doi.org/10.1016/j.brainres.2015.05.003

Drachman, D. A., & Arbit, J. (1966). Memory and the Hippocampal Complex: II. Is Memory a Multiple Process? *Archives of Neurology*, *15*(1), 52–61. https://doi.org/10.1001/archneur.1966.00470130056005

Duncan, J., Seitz, R. J., Kolodny, J., Bor, D., Herzog, H., Ahmed, A., … Emslie, H. (2000). A Neural Basis for General Intelligence. *Science*, *289*(5478), 457–460. https://doi.org/10.1126/science.289.5478.457

Dunlop, S. A., Archer, M. A., Quinlivan, J. A., Beazley, L. D., & Newnham, J. P. (1997). Repeated prenatal corticosteroids delay myelination in the ovine central nervous system.

The Journal of Maternal-Fetal Medicine, *6*(6), 309–313. https://doi.org/10.1002/(SICI)1520-6661(199711/12)6:6<309::AID-MFM1>3.0.CO;2-S

Eckenrode, J., Laird, M., & Doris, J. (1993). School performance and disciplinary problems among abused and neglected children. *Developmental Psychology*, *29*(1), 53–62. https://doi.org/10.1037/0012-1649.29.1.53

Engle, R. W., Laughlin, J. E., Tuholski, S. W., & Conway, A. R. A. (1999). *Working Memory, Short-Term Memory, and General Fluid Intelligence: A Latent-Variable Approach*. 23.

Felitti, V., J., Anda, Robert F., Nordernberg, Dale, Williamson, David F., Spitz, Alison M., Edwards, V., … Marks, James S. (1998). Relationship of Childhood Abuse and Household Dysfunction to Many of the Leading Causes of Death in Adults The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicin*, *14*(4), 245–250. http://dx.doi.org/10.1016/S0749-3797(98)00017-8

Finkelhor, D. (2018). Screening for adverse childhood experiences (ACEs): Cautions and suggestions. *Child Abuse & Neglect*, *85*, 174–179. https://doi.org/10.1016/j.chiabu.2017.07.016

Foxe, J. J., & Snyder, A. C. (2011). The Role of Alpha-Band Brain Oscillations as a Sensory Suppression Mechanism during Selective Attention. *Frontiers in Psychology*, *2*. https://doi.org/10.3389/fpsyg.2011.00154

Frodl, T., & O'Keane, V. (2013). How does the brain deal with cumulative stress? A review with focus on developmental stress, HPA axis function and hippocampal structure in humans. *Neurobiology of Disease*, *52*, 24–37. https://doi.org/10.1016/j.nbd.2012.03.012

Fuller-Thomson, E., Baird, S. L., Dhrodia, R., & Brennenstuhl, S. (2016). The association between adverse childhood experiences (ACEs) and suicide attempts in a population-

based study. *Child: Care, Health & Development*, *42*(5), 725–734. https://doi.org/10.1111/cch.12351

Gärtner, M., Rohde-Liebenau, L., Grimm, S., & Bajbouj, M. (2014). Working memory-related frontal theta activity is decreased under acute stress. *Psychoneuroendocrinology*, *43*, 105–113. https://doi.org/10.1016/j.psyneuen.2014.02.009

Gärtner, M., Rohde-Liebenau, L., Grimm, S., & Bajbouj, M. (2014). Working memory-related frontal theta activity is decreased under acute stress. *Psychoneuroendocrinology*, *43*, 105–113. https://doi.org/10.1016/j.psyneuen.2014.02.009

Gevins, A. (1997). High-resolution EEG mapping of cortical activation related to working memory: effects of task difficulty, type of processing, and practice. *Cerebral Cortex*, *7*(4), 374–385. https://doi.org/10.1093/cercor/7.4.374

Goel, V., & Grafman, J. (1995). Are the frontal lobes implicated in "planning" functions? Interpreting data from the Tower of Hanoi. *Neuropsychologia*, *33*(5), 623–642. https://doi.org/10.1016/0028-3932(95)90866-P

Hanslmayr, S., Gross, J., Klimesch, W., & Shapiro, K. L. (2011). The role of alpha oscillations in temporal attention. *Brain Research Reviews*, *67*(1), 331–343. https://doi.org/10.1016/j.brainresrev.2011.04.002

Hanson, J. L., Nacewicz, B. M., Sutterer, M. J., Cayo, A. A., Schaefer, S. M., Rudolph, K. D., … Davidson, R. J. (2015). Behavioral problems after early life stress: contributions of the hippocampus and amygdala. *Biological Psychiatry*, *77*(4), 314–323. https://doi.org/10.1016/j.biopsych.2014.04.020

Henckens, M. J. A. G., Hermans, E. J., Pu, Z., Joels, M., & Fernandez, G. (2009). Stressed Memories: How Acute Stress Affects Memory Formation in Humans. *Journal of Neuroscience*, *29*(32), 10111–10119. https://doi.org/10.1523/JNEUROSCI.1184-09.2009

Henckens, M. J. A. G., Hermans, E. J., Pu, Z., Joels, M., & Fernandez, G. (2009). Stressed Memories: How Acute Stress Affects Memory Formation in Humans. *Journal of Neuroscience*, *29*(32), 10111–10119. https://doi.org/10.1523/JNEUROSCI.1184-09.2009

Hermans, E. J., Marle, H. J. F. van, Ossewaarde, L., Henckens, M. J. A. G., Qin, S., Kesteren, M. T. R. van, … Fernández, G. (2011). Stress-Related Noradrenergic Activity Prompts Large-Scale Neural Network Reconfiguration. *Science*, *334*(6059), 1151–1153. https://doi.org/10.1126/science.1209603

Herrmann, C. S., & Knight, R. T. (2001). Mechanisms of human attention: event-related potentials and oscillations. *Neuroscience & Biobehavioral Reviews*, *25*(6), 465–476. https://doi.org/10.1016/S0149-7634(01)00027-6

Hsieh, L.-T., & Ranganath, C. (2014). Frontal midline theta oscillations during working memory maintenance and episodic encoding and retrieval. *NeuroImage*, *85*, 721–729. https://doi.org/10.1016/j.neuroimage.2013.08.003

Hughes, Karen, Bellis, M., A., Hardcastle, K. A., Sethi, D., Butchart, A., Mikton, C., … Dunne, M. P. (2017). The effect of multiple adverse childhood experiences on health: a systematic review and meta-analysis. *Lancet Public Health*, *2*, 356–366. https://doi.org/10.1016/S2468-2667(17)30118-4

Ireland, T. O., Smith, C. A., & Thornberry, T. P. (2002). Developmental Issues in the Impact of Child Maltreatment on Later Delinquency and Drug Use*. *Criminology*, *40*(2), 359–400. https://doi.org/10.1111/j.1745-9125.2002.tb00960.x

Jabès, A., & Nelson, C. A. (2015). 20 years after "The ontogeny of human memory: A cognitive neuroscience perspective," where are we? *International Journal of Behavioral Development*, *39*(4), 293–303. https://doi.org/10.1177/0165025415575766

Jeneson, A., & Squire, L. R. (2012). Working memory, long-term memory, and medial temporal lobe function. *Learning & Memory*, *19*(1), 15–25. https://doi.org/10.1101/lm.024018.111

Juster, R.-P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience and Biobehavioral Reviews*, *35*(1), 2–16. https://doi.org/10.1016/j.neubiorev.2009.10.002

Kass, R. E., & Raftery, A. E. (1995). Bayes Factors. *Journal of the American Statistical Association*, *90*(430), 773–795. JSTOR. https://doi.org/10.2307/2291091

Kelly, P. A., Viding, E., Wallace, G. L., Schaer, M., De Brito, S. A., Robustelli, B., & McCrory, E. J. (2013). Cortical thickness, surface area, and gyrification abnormalities in children exposed to maltreatment: neural markers of vulnerability? *Biological Psychiatry*, *74*(11), 845–852. https://doi.org/10.1016/j.biopsych.2013.06.020

Khrapatina, I., & Berman, P. (2017). The Impact of Adverse Childhood Experiences on Health in College Students. *Journal of Child & Adolescent Trauma*, *10*(3), 275–287. https://doi.org/10.1007/s40653-016-0093-0

Kim, D.-J., Bolbecker, A. R., Howell, J., Rass, O., Sporns, O., Hetrick, W. P., … O'Donnell, B. F. (2013). Disturbed resting state EEG synchronization in bipolar disorder: A graph-theoretic analysis. *NeuroImage : Clinical*, *2*, 414–423. https://doi.org/10.1016/j.nicl.2013.03.007

Kirschbaum, C., Wolf, O. T., May, M., Wippich, W., & Hellhammer, D. H. (1996). Stress- and treatment-induced elevations of cortisol levels associated with impaired declarative

memory in healthy adults. *Life Sciences*, *58*(17), 1475–1483. https://doi.org/10.1016/0024-3205(96)00118-X

Klimesch, W, Doppelmayr, M., Russegger, H., Pachinger, T., & Schwaiger, J. (1998). Induced alpha band power changes in the human EEG and attention. *Neuroscience Letters*, *244*(2), 73–76. https://doi.org/10.1016/S0304-3940(98)00122-0

Klimesch, Wolfgang. (1996). Memory processes, brain oscillations and EEG synchronization. *International Journal of Psychophysiology*, *24*(1), 61–100. https://doi.org/10.1016/S0167-8760(96)00057-8

Klimesch, Wolfgang. (2012). Alpha-band oscillations, attention, and controlled access to stored information. *Trends in Cognitive Sciences*, *16*(12), 606–617. https://doi.org/10.1016/j.tics.2012.10.007

Koss, Mary P., Yuan, Nicole P., Dightman, Douglas, Prince, Ronald J., Polacca, Mona, Sanderson, Byron, & Goldman, David. (2003). Adverse Childhood Exposures and AlcoholDependence Among Seven Native American Tribes. *American Journal of Preventive Medicine*, *25*(3), 238–244. https://doi.org/doi:10.1016/S0749-3797(03)00195-8

Kuhlmann, S., Piel, M., & Wolf, O. T. (2005). Impaired memory retrieval after psychosocial stress in healthy young men. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, *25*(11), 2977–2982. https://doi.org/10.1523/JNEUROSCI.5139-04.2005

Lagerlund, T. D. (1982). EEG Source Localization (Model-Dependent and Model-Independent Methods). In *) Electroencephalography: Basic Principles, Clinical Applications, and Related Fields* (pp. 809–822). Williams & Wilkins.

Lee, A. C. H., Yeung, L.-K., & Barense, M. D. (2012). The hippocampus and visual perception. *Frontiers in Human Neuroscience*, *6*. https://doi.org/10.3389/fnhum.2012.00091

Levy, B. A., & Murdock, B. B. (1968). The effects of delayed auditory feedback and intralist similarity in short-term memory. *Journal of Verbal Learning and Verbal Behavior*, *7*(5), 887–894. https://doi.org/10.1016/S0022-5371(68)80092-1

Levy, R., & Goldman-Rakic, P. S. (2000). Segregation of working memory functions within the dorsolateral prefrontal cortex. *Experimental Brain Research*, *133*(1), 23–32. https://doi.org/10.1007/s002210000397

Loo, S. K., & Barkley, R. A. (2005). Clinical utility of EEG in attention deficit hyperactivity disorder. *Applied Neuropsychology*, *12*(2), 64–76. https://doi.org/10.1207/s15324826an1202_2

Luck, S. J., & Vogel, E. K. (1997). The capacity of visualworking memory for features and conjunctions. *Nature*, *390*(20), 279–281. https://doi.org/10.1038/36846

Luck, S. J., & Vogel, E. K. (1997). The capacity of visualworking memory for features and conjunctions. *Nature*, *390*(20), 279–281. https://doi.org/10.1038/36846

Luck, S. J., & Vogel, E. K. (2013). Visual working memory capacity: from psychophysics and neurobiology to individual differences. *Trends in Cognitive Sciences*, *17*(8), 391–400. https://doi.org/10.1016/j.tics.2013.06.006

Luethi, M., Meier, B., & Sandi, C. (2009). Stress effects on working memory, explicit memory, and implicit memory for neutral and emotional stimuli in healthy men. *Frontiers in Behavioral Neuroscience*, *2*. https://doi.org/10.3389/neuro.08.005.2008

Lupien, S. J., Maheu, F., Tu, M., Fiocco, A., & Schramek, T. E. (2007). The effects of stress and stress hormones on human cognition: Implications for the field of brain and cognition. *Brain and Cognition*, *65*(3), 209–237. https://doi.org/10.1016/j.bandc.2007.02.007

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews. Neuroscience*, *10*(6), 434–445. https://doi.org/10.1038/nrn2639

Lupien, Sonia 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*(3), 420–430. https://doi.org/10.1037/0735-7044.113.3.420

Lupien, Sonia J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews. Neuroscience*, *10*(6), 434–445. https://doi.org/10.1038/nrn2639

McEwen, B. S. (2000). The neurobiology of stress: From serendipity to clinical relevance11Published on the World Wide Web on 22 November 2000. *Brain Research*, *886*(1), 172–189. https://doi.org/10.1016/S0006-8993(00)02950-4

McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews*, *87*(3), 873–904. https://doi.org/10.1152/physrev.00041.2006

McEwen, B. S., & Morrison, J. H. (2013). The Brain on Stress: Vulnerability and Plasticity of the Prefrontal Cortex over the Life Course. *Neuron*, *79*(1), 16–29. https://doi.org/10.1016/j.neuron.2013.06.028

McFadyen, R. G., & Kitson, W. J. H. (1996). Language Comprehension and Expression Among Adolescents Who Have Experienced Childhood Physical Abuse. *Journal of Child Psychology and Psychiatry*, *37*(5), 551–562. https://doi.org/10.1111/j.1469-7610.1996.tb01441.x

McMorris, T., Swain, J., Smith, M., Corbett, J., Delves, S., Sale, C., Harris, R. C., & Potter, J. (2006). Heat stress, plasma concentrations of adrenaline, noradrenaline, 5-hydroxytryptamine and cortisol, mood state and cognitive performance. *International Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology*, *61*(2), 204–215. https://doi.org/10.1016/j.ijpsycho.2005.10.002

Meaney, M. J. (2001). Maternal Care, Gene Expression, and the Transmission of Individual Differences in Stress Reactivity Across Generations. *Annual Review of Neuroscience*, *24*(1), 1161–1192. https://doi.org/10.1146/annurev.neuro.24.1.1161

Medina, K. L., Schweinsburg, A. D., Cohen-Zion, M., Nagel, B. J., & Tapert, S. F. (2007). Effects of Alcohol and Combined Marijuana and Alcohol Use During Adolescence on Hippocampal Volume and Asymmetry. *Neurotoxicology and Teratology*, *29*(1), 141–152. https://doi.org/10.1016/j.ntt.2006.10.010

Mersky, J.P., Topitzes, J., & Reynolds, A.J. (2013). Impacts of adverse childhood experiences on health, mental health, and substance use in early adulthood: A cohort study of an urban, minority sample. *Child Abuse & Neglect*, *37*, 917–925. http://dx.doi.org/10.1016/j.chiabu.2013.07.011

Metzler, M., Merrick, M. T., Klevens, J., Ports, K. A., & Ford, D. C. (2017). Adverse childhood experiences and life opportunities: Shifting the narrative. *Children and Youth Services Review*, *72*, 141–149. https://doi.org/10.1016/j.childyouth.2016.10.021

Miller, E. K. (2000). The prefontral cortex and cognitive control. *Nature Reviews Neuroscience*, *1*(1), 59–65. https://doi.org/10.1038/35036228

Mitchell, D. J., McNaughton, N., Flanagan, D., & Kirk, I. J. (2008). Frontal-midline theta from the perspective of hippocampal "theta." *Progress in Neurobiology*, *86*(3), 156–185. https://doi.org/10.1016/j.pneurobio.2008.09.005

Mizoguchi, K., Yuzurihara, M., Ishige, A., Sasaki, H., Chui, D. H., & Tabira, T. (2000). Chronic stress induces impairment of spatial working memory because of prefrontal dopaminergic dysfunction. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, *20*(4), 1568–1574.

Monti, P. M., Miranda, R., Nixon, K., Sher, K. J., Swartzwelder, H. S., Tapert, S. F., White, A., & Crews, F. T. (2005). Adolescence: Booze, Brains, and Behavior. *Alcoholism: Clinical and Experimental Research*, *29*(2), 207–220. https://doi.org/10.1097/01.ALC.0000153551.11000.F3

Moran, P. B., Vuchinich, S., & Hall, N. K. (2004). Associations between types of maltreatment and substance use during adolescence. *Child Abuse & Neglect*, *28*(5), 565–574. https://doi.org/10.1016/j.chiabu.2003.12.002

Nunez, P. L., & Srinivasan, R. (2006). Spatial-Temporal Properties of EEG. In *Electric Fields of the Brain* (pp. 244–247). Oxford University Press.

Oei, N. Y. L., Everaerd, W. T. A. M., Elzinga, B. M., van Well, S., & Bermond, B. (2006). Psychosocial stress impairs working memory at high loads: An association with cortisol levels and memory retrieval. *Stress*, *9*(3), 133–141. https://doi.org/10.1080/10253890600965773

Onton, J., Delorme, A., & Makeig, S. (2005). Frontal midline EEG dynamics during working memory. *NeuroImage*, 341–356.

Ossewaarde, L., Hermans, E. J., van Wingen, G. A., Kooijman, S. C., Johansson, I.-M., Bäckström, T., & Fernández, G. (2010). Neural mechanisms underlying changes in stress-sensitivity across the menstrual cycle. *Psychoneuroendocrinology*, *35*(1), 47–55. https://doi.org/10.1016/j.psyneuen.2009.08.011

Patil, P. G., Apfelbaum, J. L., & Zacny, J. P. (1995). Effects of a cold-water stressor on psychomotor and cognitive functioning in humans. *Physiology & Behavior*, *58*(6), 1281–1286. https://doi.org/10.1016/0031-9384(95)02071-3

Pfurtscheller, G., & Lopes da Silva, F. H. (1999). Event-related EEG/MEG synchronization and desynchronization: basic principles. *Clinical Neurophysiology*, *110*(11), 1842–1857. https://doi.org/10.1016/S1388-2457(99)00141-8

Pfurtscheller, Gert, Neuper, C., Pichler-Zalaudek, K., Edlinger, G., & Lopes da Silva, F. H. (2000). Do brain oscillations of different frequencies indicate interaction between cortical areas in humans? *Neuroscience Letters*, *286*(1), 66–68. https://doi.org/10.1016/S0304-3940(00)01055-7

Philip, N. S., Sweet, L. H., Tyrka, A. R., Carpenter, S. L., Albright, S. E., Price, L. H., & Carpenter, L. L. (2016). Exposure to childhood trauma is associated with altered n-back activation and performance in healthy adults: implications for a commonly used working memory task. *Brain Imaging and Behavior*, *10*(1), 124–135. https://doi.org/10.1007/s11682-015-9373-9

Plotsky, P. M., & Meaney, M. J. (1993). Early, postnatal experience alters hypothalamic corticotropin-releasing factor (CRF) mRNA, median eminence CRF content and stress-induced release in adult rats. *Brain Research. Molecular Brain Research*, *18*(3), 195–200.

Poole, J. C., Dobson, K. S., & Pusch, D. (2017). Childhood adversity and adult depression: The protective role of psychological resilience. *Child Abuse & Neglect*, *64*, 89–100. https://doi.org/10.1016/j.chiabu.2016.12.012

Porcelli, A. J., Cruz, D., Wenberg, K., Patterson, M. D., Biswal, B. B., & Rypma, B. (2008). The effects of acute stress on human prefrontal working memory systems. *Physiology & Behavior*, *95*(3), 282–289. https://doi.org/10.1016/j.physbeh.2008.04.027

Posner, M. I. (1994). Attention: the mechanisms of consciousness. *Proceedings of the National Academy of Sciences of the United States of America*, *91*(16), 7398–7403.

Prabhakaran, V., Rypma, B., & Gabrieli, J. D. (2001). Neural substrates of mathematical reasoning: a functional magnetic resonance imaging study of neocortical activation during performance of the necessary arithmetic operations test. *Neuropsychology*, *15*(1), 115–127.

Qin, S., Cousijn, H., Rijpkema, M., Luo, J., Franke, B., Hermans, E., & Fernández, G. (2012). The effect of moderate acute psychological stress on working memory-related neural activity is modulated by a genetic variation in catecholaminergic function in humans. *Frontiers in Integrative Neuroscience*, *6*. https://doi.org/10.3389/fnint.2012.00016

Qin, S., Hermans, E. J., van Marle, H. J. F., Luo, J., & Fernández, G. (2009). Acute psychological stress reduces working memory-related activity in the dorsolateral prefrontal cortex. *Biological Psychiatry*, *66*(1), 25–32. https://doi.org/10.1016/j.biopsych.2009.03.006

R Development Core Team., 2019. R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. ISBN 3-900051-07-0, URL http://www.R-project.org

Raghavachari, S., Kahana, M. J., Rizzuto, D. S., Caplan, J. B., Kirschen, M. P., Bourgeois, B., … Lisman, J. E. (2001). Gating of Human Theta Oscillations by a Working Memory Task. *The Journal of Neuroscience*, *21*(9), 3175–3183. https://doi.org/10.1523/JNEUROSCI.21-09-03175.2001

Ranganath, C., & Blumenfeld, R. S. (2005). Doubts about double dissociations between short- and long-term memory. *Trends in Cognitive Sciences*, *9*(8), 374–380. https://doi.org/10.1016/j.tics.2005.06.009

Ranganath, C., Johnson, M. K., & D'Esposito, M. (2003). Prefrontal activity associated with working memory and episodic long-term memory. *Neuropsychologia*, *41*(3), 378–389. https://doi.org/10.1016/S0028-3932(02)00169-0

Roberts, R. J., & Pennington, B. F. (1996). An interactive framework for examining prefrontal cognitive processes. *Developmental Neuropsychology*, *12*(1), 105–126. https://doi.org/10.1080/87565649609540642

Rouder, J. N., Speckman, P. L., Sun, D., Morey, R. D., & Iverson, G. (2009). Bayesian t tests for accepting and rejecting the null hypothesis. *Psychonomic Bulletin & Review*, *16*(2), 225–237. https://doi.org/10.3758/PBR.16.2.225

Roux, F., & Uhlhaas, P. J. (2014). Working memory and neural oscillations: alpha–gamma versus theta–gamma codes for distinct WM information? *Trends in Cognitive Sciences*, *18*(1), 16–25. https://doi.org/10.1016/j.tics.2013.10.010

Sánchez, M. M., Ladd, C. O., & Plotsky, P. M. (2001). Early adverse experience as a developmental risk factor for later psychopathology: evidence from rodent and primate models. *Development and Psychopathology*, *13*(3), 419–449.

Sapolsky, R. M., & Meaney, M. J. (1986). Maturation of the adrenocortical stress response: Neuroendocrine control mechanisms and the stress hyporesponsive period. *Brain Research Reviews*, *11*(1), 65–76. https://doi.org/10.1016/0165-0173(86)90010-X

Sauseng, P., Griesmayr, B., Freunberger, R., & Klimesch, W. (2010). Control mechanisms in working memory: A possible function of EEG theta oscillations. *Neuroscience and Biobehavioral Reviews*, *34*, 1015–1022. https://doi.org/doi:10.1016/j.neubiorev.2009.12.006

Scheeringa, R., Bastiaansen, M. C. M., Petersson, K. M., Oostenveld, R., Norris, D. G., & Hagoort, P. (2008). Frontal theta EEG activity correlates negatively with the default mode network in resting state. *International Journal of Psychophysiology*, *67*(3), 242–251. https://doi.org/10.1016/j.ijpsycho.2007.05.017

Schoofs, D., Preuß, D., & Wolf, O. T. (2008). Psychosocial stress induces working memory impairments in an n-back paradigm. *Psychoneuroendocrinology*, *33*(5), 643–653. https://doi.org/10.1016/j.psyneuen.2008.02.004

Scoville, W. B., & Milner, B. (2000). Loss of recent memory after bilateral hippocampal lesions. 1957. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *12*(1), 103–113. https://doi.org/10.1176/jnp.12.1.103

Shackman, A. J., Sarinopoulos, I., Maxwell, J. S., Pizzagalli, D. A., Lavric, A., & Davidson, R. J. (2006). Anxiety selectively disrupts visuospatial working memory. *Emotion (Washington, D.C.)*, *6*(1), 40–61. https://doi.org/10.1037/1528-3542.6.1.40

Simmering, V. R., & Perone, S. (2013). Working memory capacity as a dynamic process. *Frontiers in Psychology*, *3*(567), 1–26. https://doi.org/10.3389/fpsyg.2012.00567

Simons, J. S., & Spiers, H. J. (2003). Prefrontal and medial temporal lobe interactions in long-term memory. *Nature Reviews Neuroscience*, *4*(8), 637-. Retrieved from Academic OneFile.

Smeets, T., Jelicic, M., & Merckelbach, H. (2006). The effect of acute stress on memory depends on word valence. *International Journal of Psychophysiology*, *62*(1), 30–37. https://doi.org/10.1016/j.ijpsycho.2005.11.007

Smeets, T., Jelicic, M., & Merckelbach, H. (2006). The effect of acute stress on memory depends on word valence. *International Journal of Psychophysiology*, *62*(1), 30–37. https://doi.org/10.1016/j.ijpsycho.2005.11.007

Stein, M. B., Koverola, C., Hanna, C., Torchia, M. G., & McClarty, B. (1997). Hippocampal volume in women victimized by childhood sexual abuse. *Psychological Medicine*, *27*(4), 951–959. https://doi.org/10.1017/S0033291797005242

Stephane, M., Ince, N. F., Kuskowski, M., Leuthold, A., Tewfik, A. H., Nelson, K., McClannahan, K., Fletcher, C. R., & Tadipatri, V. A. (2010). Neural oscillations associated with the primacy and recency effects of verbal working memory. *Neuroscience Letters*, *473*(3), 172–177. https://doi.org/10.1016/j.neulet.2010.02.025

Teicher, M. H. (2006). Neurobiological Consequences of Early Stress and Childhood Maltreatment: Are Results from Human and Animal Studies Comparable? *Annals of the New York Academy of Sciences*, *1071*(1), 313–323. https://doi.org/10.1196/annals.1364.024

Teicher, Martin H, Andersen, S. L., Polcari, A., Anderson, C. M., & Navalta, C. P. (2002). Developmental neurobiology of childhood stress and trauma. *Psychiatric Clinics of North America*, *25*(2), 397–426. https://doi.org/10.1016/S0193-953X(01)00003-X

Teicher, Martin H., & Samson, J. A. (2013). Childhood Maltreatment and Psychopathology: A Case for Ecophenotypic Variants as Clinically and Neurobiologically Distinct Subtypes. *American Journal of Psychiatry*, *170*(10), 1114–1133. https://doi.org/10.1176/appi.ajp.2013.12070957

Tonmyr, L., Thornton, T., Draca, J., & Wekerle, C. (2010). A Review of Childhood Maltreatment and Adolescent Substance Use Relationship. *Current Psychiatry Reviews*, *6*(3), 223–234. https://doi.org/10.2174/157340010791792581

Trickett, P. K., & McBride-Chang, C. (1995). The Developmental Impact of Different Forms of Child Abuse and Neglect. *Developmental Review*, *15*(3), 311–337. https://doi.org/10.1006/drev.1995.1012

Tsigos, C., & Chrousos, G. P. (2002). Hypothalamic–pituitary–adrenal axis, neuroendocrine factors and stress. *Journal of Psychosomatic Research*, *53*(4), 865–871. https://doi.org/10.1016/S0022-3999(02)00429-4

Vecchi, T., & Girelli, L. (1998). Gender differences in visuo-spatial processing: The importance of distinguishing between passive storage and active manipulation. *Acta Psychologica*, *99*(1), 1–16. https://doi.org/10.1016/S0001-6918(97)00052-8

Vecchi, T., Richardson, J., & Cavallini, E. (2005). Passive storage versus active processing in working memory: Evidence from age-related variations in performance. *European Journal of Cognitive Psychology*, *17*(4), 521–539. https://doi.org/10.1080/09541440440000140

Vytal, K., Cornwell, B., Arkin, N., & Grillon, C. (2012). Describing the interplay between anxiety and cognition: From impaired performance under low cognitive load to reduced anxiety under high load. *Psychophysiology*, *49*(6), 842–852. https://doi.org/10.1111/j.1469-8986.2012.01358.x

Watson, D., Clark, L. A., & Carey, G. (1988). Positive and negative affectivity and their relation to anxiety and depressive disorders. *Journal of Abnormal Psychology*, 346353.

Whittle, S., Vijayakumar, N., Simmons, J., Yucel, M., & Lubman, D. (2016). Childhood maltreatment, psychopathology, and the development of hippocampal subregions during adolescence. *Brain and Behavior*, *7*(2), 1–9. https://doi.org/10.1002/brb3.607

Wilhelm, O., Hildebrandt, A., & Oberauer, K. (2013). What is working memory capacity, and how can we measure it? *Frontiers in Psychology*, *4*. https://doi.org/10.3389/fpsyg.2013.00433

Yakovlev, P., & Lecours, A. R. (1967). *The Myelogenetic Cycles of Regional Maturation of the Brain. Regional Development of the Brain in early Life*. Retrieved from https://www.scienceopen.com/document?vid=1c16c21a-8793-4f48-bfcc-87c6f65e1bf9

Yerkes, R. M., & Dodson, J. D. (1908). The relation of strength of stimulus to rapidity of habit-formation. *Journal of Comparative Neurology and Psychology*, *18*(5), 459–482. https://doi.org/10.1002/cne.920180503

Yonelinas, A. P. (2013). The hippocampus supports high-resolution binding in the service of perception, working memory and long-term memory. *Behavioural Brain Research*, *254*, 34–44. https://doi.org/10.1016/j.bbr.2013.05.030

APPENDICES

Appendix A

Adverse Childhood Experience (ACE) Questionnaire Finding your ACE Score

While you were growing up, during your first 18 years of life:

1. Did a parent or other adult in the household **often** …
   Swear at you, insult you, put you down, or humiliate you?
   or
   Act in a way that made you afraid that you might be physically hurt?

   Yes No If yes enter 1 _____

2. Did a parent or other adult in the household **often** …
   Push, grab, slap, or throw something at you?
   or
   Ever hit you so hard that you had marks or were injured?

   Yes No If yes enter 1 _____

3. Did an adult or person at least 5 years older than you **ever**…
   Touch or fondle you or have you touch their body in a sexual way?
   or
   Try to or actually have oral, anal, or vaginal sex with you?

   Yes No If yes enter 1 _____

4. Did you **often** feel that …
   No one in your family loved you or thought you were important or special?
   or
   Your family didn't look out for each other, feel close to each other, or support each other?

   Yes No If yes enter 1 _____

5. Did you **often** feel that …
   You didn't have enough to eat, had to wear dirty clothes, and had no one to protect you?
   or
   Your parents were too drunk or high to take care of you or take you to the doctor if you needed it?

   Yes No If yes enter 1 _____

6. Were your parents **ever** separated or divorced?

Yes No If yes enter 1 _____

7. Was your mother or stepmother:

Often pushed, grabbed, slapped, or had something thrown at her?

or

Sometimes or often kicked, bitten, hit with a fist, or hit with something hard?

or

Ever repeatedly hit over at least a few minutes or threatened with a gun or knife?

Yes No If yes enter 1 _____

8. Did you live with anyone who was a problem drinker or alcoholic or who used street drugs?

Yes No If yes enter 1 _____

9. Was a household member depressed or mentally ill or did a household member attempt suicide?

Yes No If yes enter 1 _____

10. Did a household member go to prison?

Yes No If yes enter 1 _____

Now add up your "Yes" answers: _____ This is your ACE Score

Appendix B

The Positive and Negative Affect Schedule (PANAS; Watson et al., 1988)

PANAS Questionnaire

This scale consists of a number of words that describe different feelings and emotions. Read each item and then list the number from the scale below next to each word. **Indicate to what extent you feel this way right now, that is, at the present moment (circle the instructions you followed when taking this measure)**

1	2	3 4 5
Very Slightly or Not at All	A Little	Moderately Quite a Bit Extremely

_____ 1. Interested	_____ 11. Irritable
_____ 2. Distressed	_____ 12. Alert
_____ 3. Excited	_____ 13. Ashamed
_____ 4. Upset	_____ 14. Inspired
_____ 5. Strong	_____ 15. Nervous
_____ 6. Guilty	_____ 16. Determined
_____ 7. Scared	_____ 17. Attentive
_____ 8. Hostile	_____ 18. Jittery
_____ 9. Enthusiastic	_____ 19. Active
_____ 10. Proud	_____ 20. Afraid

Scoring Instructions:

Positive Affect Score: Add the scores on items 1, 3, 5, 9, 10, 12, 14, 16, 17, and 19. Scores can range from 10 – 50, with higher scores represent- ing higher levels of positive affect. Mean Scores: Momentary = 29.7 (*SD* = 7.9); Weekly = 33.3 (*SD = 7.2)*

Negative Affect Score: Add the scores on items 2, 4, 6, 7, 8, 11, 13, 15, 18, and 20. Scores can range from 10 – 50, with lower scores represent- ing lower levels of negative affect. Mean Score: Momentary = 14.8 (*SD* = 5.4); Weekly = 17.4 (*SD* = 6.2)

Appendix C: Supplemental Figures

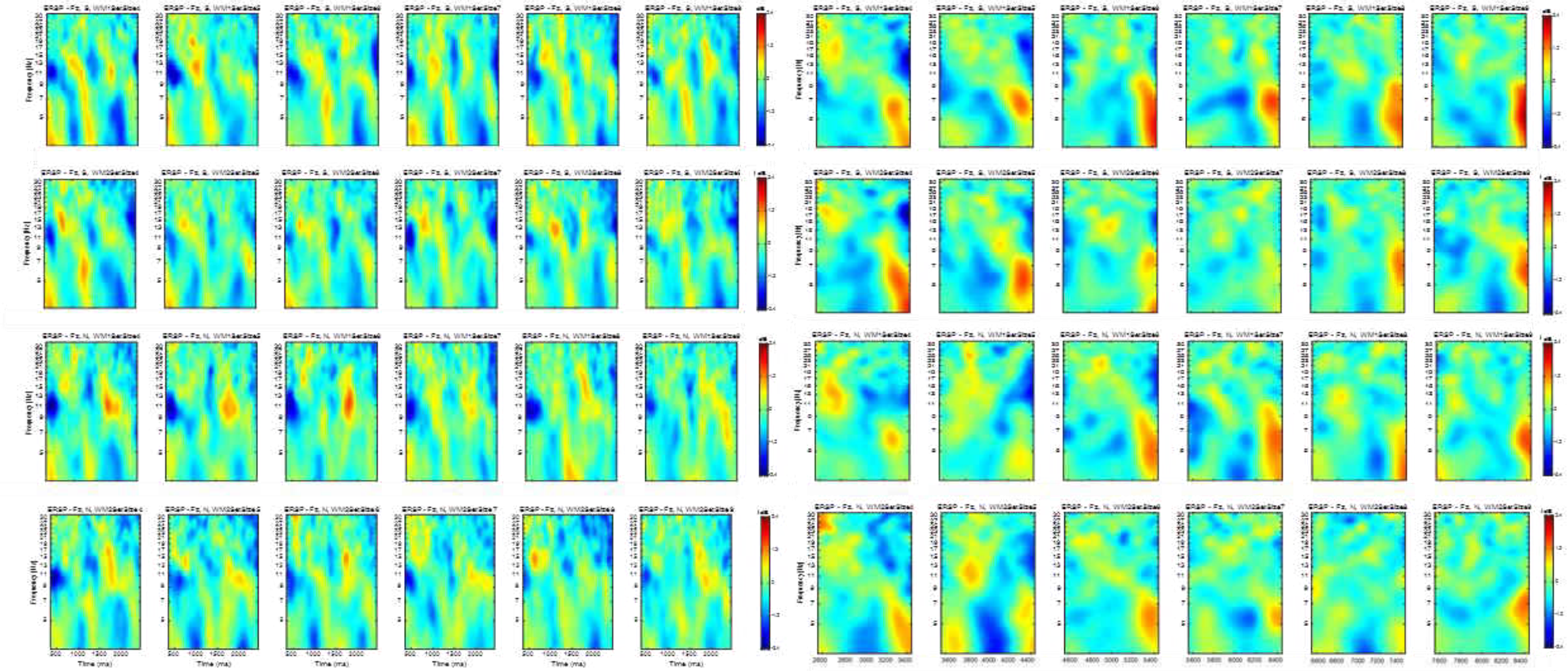

Figure 5): ERSP plots examining for electrode Fz for the presentation of the first two digits (left) and last two digits (right) across set sizes.

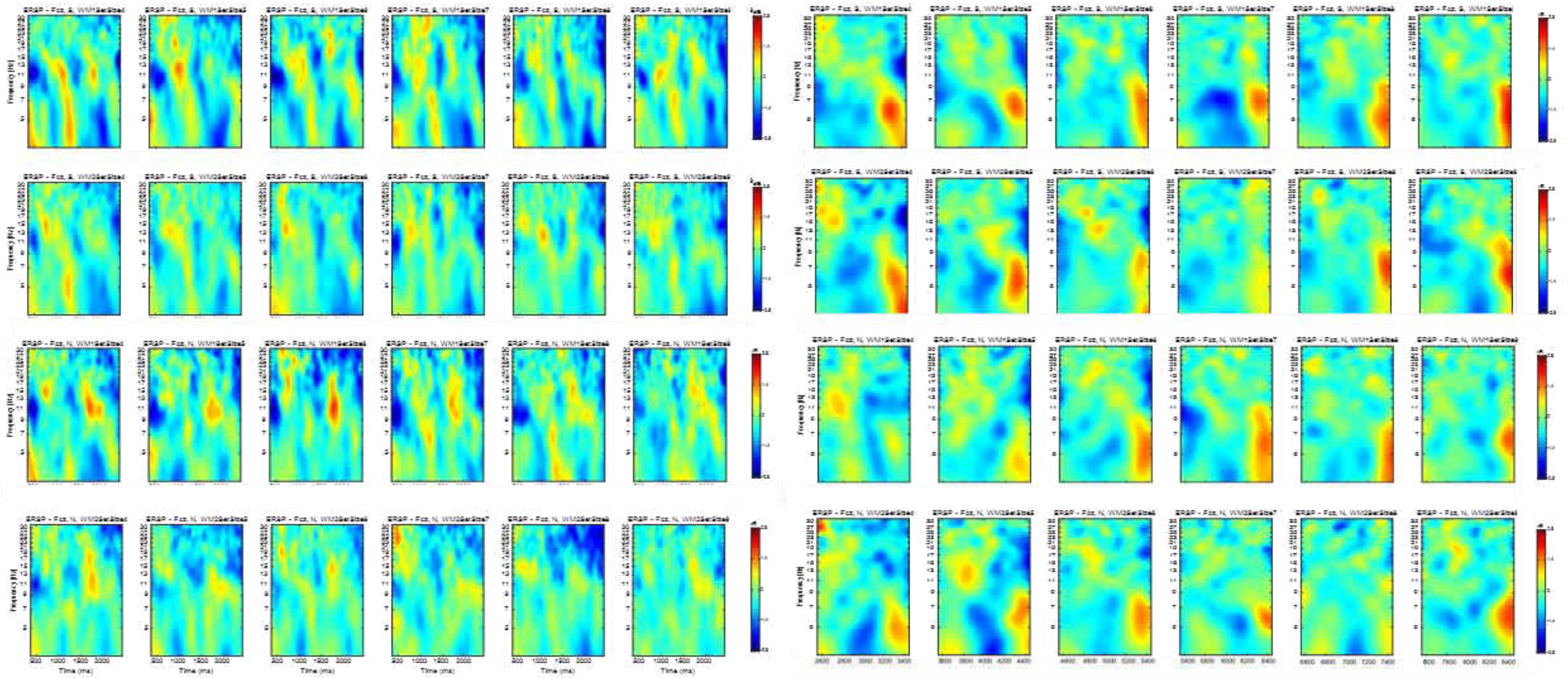

Figure 6): ERSP plots examining for electrode FCz for the presentation of the first two digits (left) and last two digits (right) across set sizes.

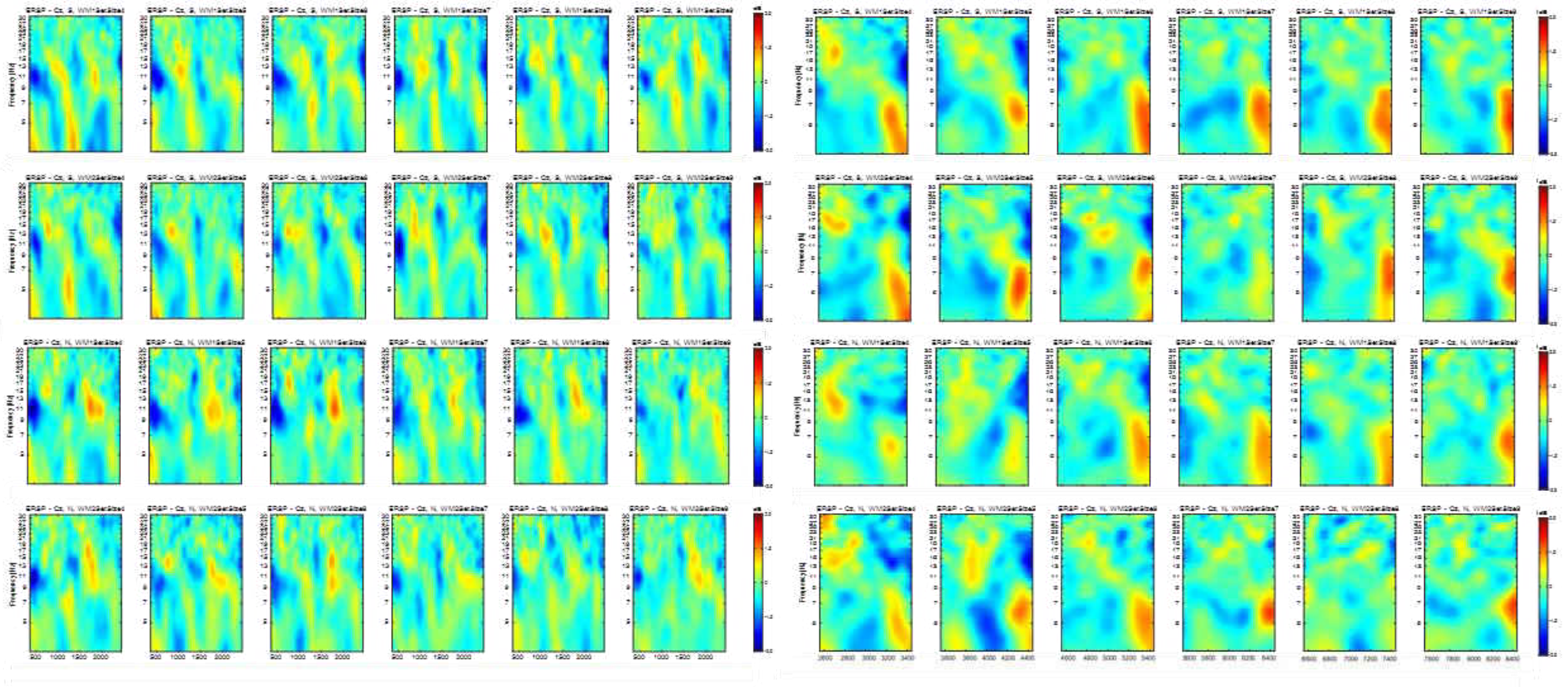

Figure 7): ERSP plots examining for electrode Cz for the presentation of the first two digits (left) and last two digits (right) across set sizes.

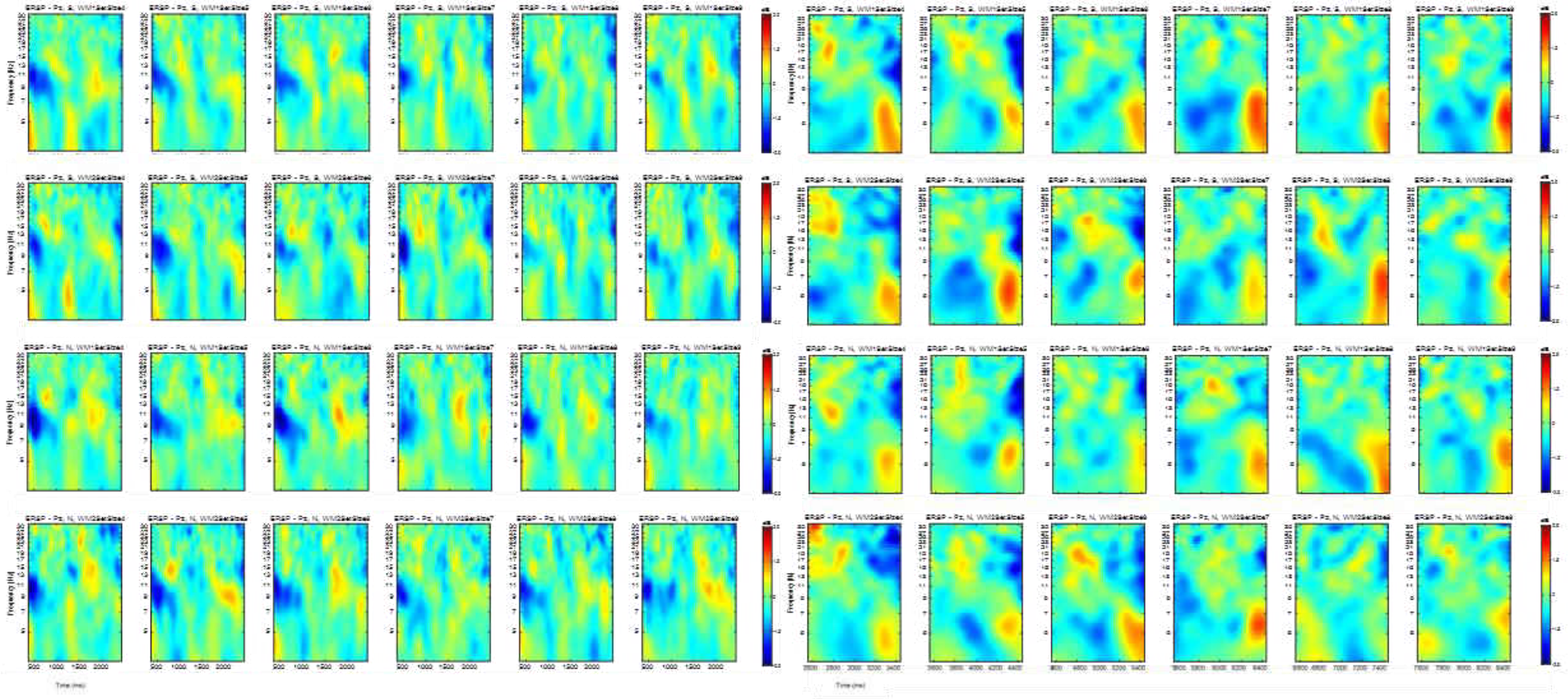

Figure 8): ERSP plots examining for electrode Pz for the presentation of the first two digits (left) and last two digits (right) across set sizes.

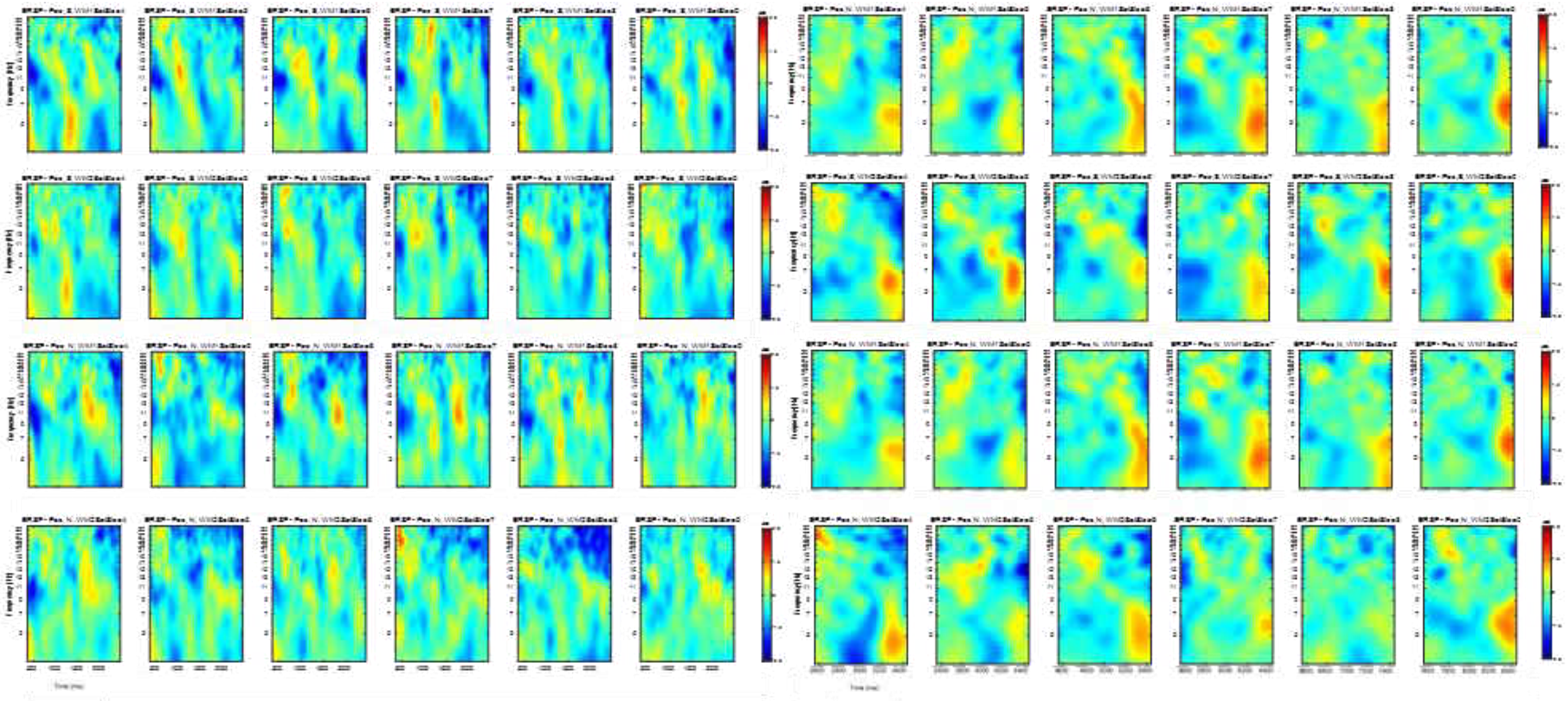

Figure 9): ERSP plots examining for electrode Pz for the presentation of the first two digits (left) and last two digits (right) across set sizes.

VITA

MARISSA JONES

Education: Ph.D. Psychology, concentration in Experimental Psychology, East Tennessee State University, Johnson City, Tennessee, 2020

M.A. Psychology, concentration in Experimental Psychology, East Tennessee State University, Johnson City, Tennessee, 2017

B.S. Clinical Psychology, East Tennessee State University, Johnson City, Tennessee, 2015

Public Schools, Elizabethton, Tennessee

Professional Experience: Graduate Assistant, East Tennessee State University, College of Arts and Sciences, 2015-2020

Publications: Jones, M.R., & Sellers, E.W. (2019) Faces, Locations, and Tools: A Proposed Two-Stimulus P300 Brain Computer Interface. Journal of Neural Engineering, 16(3) DOI:10.1088/1741-2552/aaff22

Kellicut-Jones, M.R., & Sellers, E.W. (2018) P300 Brain-Computer Interface: Comparing Faces to Size Matched Non-Face Stimuli. Brain Computer Interfaces, 5(1), 30-39. DOI: 10.1080/2326263X.2018.1433776

Palmatier, M. I., Kellicut, M.R., Sheppard, A. B., Brown, R. W., &

Robinson, D. L. (2014) The incentive amplifying effects of nicotine are attenuated by selective and nonselective dopamine antagonists in rats. Pharmacology, Biochemistry, and Behavior, 126, 50-62. DOI:10.1016/j.pbb.2014.08.012