EARLY FAMILY ENVIRONMENTS AND MEMORY: THE ROLE OF PHYSIOLOGICAL AND PSYCHOLOGICAL RESPONSES TO ACUTE STRESS

By

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A thesis submitted in partial fulfillment
of the requirements for the degree

of

Master of Science

in

Psychological Science

MONTANA STATE UNIVERSITY
Bozeman, Montana

December 2020

DEDICATION

I dedicate the following work to my Mother who has shown me unwavering support through the best and worst times. Someone who is very strong and makes me feel very valuable. To my Father who taught me the importance of hard work and a job well done, who passed away from complications of multiple sclerosis in 2013. To my brother who was a symbol of unconditional love and the guru of enjoying the present moment. Someone who was always proud of me and taught me the importance of treating people with respect even when it is not reciprocated. You lost your battle with depression in 2017, yet I still feel your love for me. I hope someday all the rules will bend, and you and I will meet again. I dedicate this to my childhood and current best friend, who has taught me so much about unconditional love and how the darkest nights produce the brightest stars. Thank you for standing by me as I learned how to grow. I dedicate this to my group of friends that bring me out the best in me. Thank you for choosing me. To my girlfriend who loves to see me thrive and finds value in places of me that I never knew existed. Lastly, I dedicate this work to the children and adults who have experienced adversity, neglect, and/or abuse at any point in the lifespan. To be born into an environment outside of your control can be very challenging. For that environment to harm you and impede your growth and success is one of the greatest treasons I can imagine. I hope that life gives you the capability to turn past agony into opportunity as it has done for me.

ACKNOWLEDGEMENTS

With my deepest gratitude, I would like to thank my mentor Dr. Neha John-Henderson for choosing me as a beneficiary of her expertise and unwavering support. Thank you for giving me the opportunity to grow into an individual that has the ability and resources to contribute to an important body of work. Thank you for giving me the chance to become a small reflection of the large body of impactful work you have created thus far. Thank you to Dr. Michelle Meade for unwavering support and expert guidance in translating a concept into a sound empirical study. Thank you to Dr. Cara Palmer and Dr. Kalli Decker for accepting my invitation to be a part of my thesis committee and providing me feedback on how to improve the study.

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ABSTRACT

Childhood family environments have important implications for outcomes in adulthood. Specifically, the experience of adversity in childhood is related to numerous maladaptive outcomes later in life. It is currently unknown how early adversity affects memory consolidation and processing. Previous research has established an association with anxiety and depression possessing a negative memory bias. A negative memory bias is defined as attentional and perceptual favor towards information that is contextually negative or threatening. Research has not examined the relationship between negative memory bias and childhood adversity after the induction of stress. Stress has previously shown to be disruptive to memory outcomes. Further, a growing body of research has shown that early childhood adversity associates with blunted physiological responses to stress. It is possible that through the pathway of blunted reactivity, early childhood adversity associates with negative memory bias. To test these hypotheses, a sample of college students (N=64) studied a 50-word list that included 25 emotionally negative words and 25 emotionally neutral words. Participants then completed the Trier Social Stress Test, an evaluative stressor well known for inducing stress. After the stress task, participants were asked to freely recall words they previously studied. Results showed that higher ratings or risk and emotional abuse in childhood associated with increased negative word recall. The relationship was partially mediated through blunted heart rate reactivity to the stress task. Implications and future directions are discussed.

INTRODUCTION

Childhood family environments have important implications for outcomes into adulthood. Through family dynamics, an individual learns about their surroundings and how to navigate their current environment. In many ways, successful navigation is contingent upon adaptive and successful modeling that caters to a child's developmental and emotional needs. As a child grows older and gains autonomy, previous modeling and experience often dictates the nature in which they engage with and respond to their environment on a psychological and physiological level. In a healthy model, a child has their emotional needs met through active and engaging parenting where warmth and understanding are facilitators. When these demands are met, a child is likely to gain the capacity to see the world as a place filled with challenges. However, the guidance and support they received during their early years helps them to navigate obstacles in an adaptive manner.

Unfortunately, not all childhood environments support normative development. Individuals who are raised in childhood environments characterized by traumatic experiences, abuse, neglect, and low levels of warmth are more likely to experience several adverse mental and physical health outcomes into adulthood. Memory is an important component to adult success and health which has been shown to be affected by early upbringing (de Neubourg et al., 2018; Evans & Schamberg, 2009). The consequences of early life adversity for specific memory processes later in life are not well understood. The current research examined word recall for a delayed memory test in college students to test differences of memory. Specifically, the goal was to understand how memory is affected after a controlled stressor for individuals who experienced

adversity or abuse during childhood. The study investigated the role that physiological reactivity to induced stress influenced memory recall.

Childhood Abuse: Implications into Adulthood

A large body of literature documents consequences of being raised in a family environment with the presence of adversity and/or abuse, with implications across several domains including brain morphology, physiological processes, cognitive functioning, and psychosocial outcomes (Gallo et al., 2018; Norman et al., 2012; Petrucelli, Davis, & Berman, 2019; Purewal et al., 2018;). Abuse and neglect are formally recognized as separate and distinct outcomes. Independent of reports of neglect, previous research indicates that subtypes of childhood abuse (i.e. emotional, physical and sexual), may differently affect markers of mental and physical health (Infurna et al., 2016; Witt et al., 2016). Physical and sexual abuse are strong predictors of adverse mental and physical health outcomes into adulthood (Coles et al., 2015; Mullen et al., 1993; Springer et al., 2007). Additonal recent research suggests that emotional abuse in childhood may have unique implications for future health.

Emotional abuse, or the exposure to behavior from a perpetrator that may result in psychological trauma is independently predictive of distinct health outcomes in adulthood (Cecil et al., 2017; Neacsiu, Bohus, & Linehan, 2014). Self-reported childhood emotional abuse is associated with increased illicit drug use, depressive symptoms, psychological distress, suicidality (Jina et al., 2012), and maladaptive behavioral outcomes such as binge eating (Feinson & Hornik-Lurie, 2016). Research has shown poor health and behavioral outcomes may be partially explained by maladaptation to new stressors. In a sample of young adults, greater reports of childhood emotional abuse were correlated with increased levels of depressive

symptoms when confronted with current individual dependent stressors when controlling for childhood physical and sexual abuse (Shapero et al., 2014). Further, maladaptive health behaviors may appear in response to stress (John-Henderson et al., 2018). Additionally, emotional abuse has also related to memory outcomes in previous research. Reports of emotional abuse appeared to dampen autobiographical memory specificity (Raes et al., 2005) and create lapses in childhood memories (Melchert & Parker, 1997). Recent research found that reports of childhood maltreatment associated with lower reports of working memory capacity (Dodaj et al., 2017). It is currently unknown how childhood emotional abuse may impact memory in a delayed non-referential memory recall test, following completion of a stress inducing task. Alterations in normative memory processes is a potential pathway in which childhood emotional abuse may further impact well-being and health.

Risk, Affection, and Warmth in Family Environment and Adult Outcomes

Measures of childhood adversity typically focus solely on the experiences of trauma and abuse, and fail to focus on the influence of the presence or absence of protective factors such as warmth and affection in the family environment. Warmth and/or affection may promote resilience even in the context of trauma and adversity in childhood. For example, in a large sample of middle-aged adults, those who reported childhood abuse and parental warmth displayed lower levels of chronic stress in comparison to those who reported similar levels of childhood abuse and low levels of parental warmth (Carroll et al., 2013). This work suggests that parental warmth may serve as a buffer against the effects of toxic childhood stress, and is a factor which should be considered when investigating the relationships between early life adversity and outcomes later in life. *The Risky Families Questionnaire* (Taylor et al., 2004; Felitti et al., 1998),

is unique in its consideration of both the presence of trauma and the absence of warmth and affection when calculating a measure of overall risk. It is hypothesized that risky family environments early in life create a developmental deficit in a child's control of their emotions and lower levels of social competence which may increase risk for dysregulated stress responses and psychosocial disturbances (Repetti, Taylor, & Seeman, 2002). Specific to psychosocial outcomes, individuals who are raised in risky family environments are at greater risk of experiencing depression into adulthood (Poole, Dobson, & Pusch, 2017), and exhibit lower social competence (Luecken, Roubinov, & Tanaka, 2013). Furthermore, individuals from higher risk family environments display a heightened and prolonged physiological reactivity to stress (Repetti et al., 2002). Repeated stress activation is associated with numerous chronic health conditions including hypertension, heart disease, and early death (Schnurr & Green, 2004; Esler, 2017). There is not current research examining the relationship between risky family environment and memory outcomes. Chronic stress has previously shown to affect memory (Huang et al., 2015; Jeong et al., 2006) and the ability to recall information. It is possible being raised in a childhood family environment characterized by higher levels of adversity and low levels of warmth and affection affects memory through pathways of dysregulated stress response and disruptions to normative brain processes.

Brain Structure and Childhood Adversity

A large body of work indicates that the amygdala, a region of the brain which is involved in emotional and memory processing, is affected by the experience of adversity and abuse early in life through increased sensitivity of the amygdala (Jedd et al., 2015; Calem et al., 2017), which is thought to sensitize an individual's threat perception. A recent study in a sample of

adults showed childhood maltreatment associated with an increased amygdala response to emotional faces viewed as threatening or salient compared to non-maltreated controls (van Harmelan et al., 2013), suggesting a perceptual prioritization of threatening stimulus may be present for those who experienced childhood maltreatment. Structural MRI scans have shown an association between childhood abuse and reduced cortical thickness in brain regions involved in emotional processing (Gold et al., 2016). Specifically, Gold and colleagues found abuse predicted reduced thickness in lateral prefrontal cortex (PFC) and both medial and lateral temporal cortex which are involved in explicit and effortful emotion regulation processes. Interestingly, childhood abuse related to both reduced volume of the PFC and altered patterns of PFC engagement during attempted emotion regulation. Differences in brain structure and activity may be a pathway that influences differences in social and emotional processing commonly observed in those who experience abuse (Riggs, Cusimano, & Benson, 2011; Tyler, 2002). The observed differences in brain structures involved in social, emotional, and perceptual outcomes in those who experienced childhood adversity may contribute to an increased tendency to interpret information as threatening and difficulty disengaging from threatening stimulus. This bias towards threatening and negative stimulus may serve as a protective factor during the period where risk and abuse is present, however this bias may affect performance on tasks involving memory processes where recall outside of the context or perception of threat is required.

Physiological Processes and Childhood Adversity

Previously mentioned is the important role that physiological responses to stress may have for well-being and health. When a threat to well-being (either real or perceived) is registered, a cascade of events begins with sensory systems. When an impending danger is

perceived through visual and auditory processes, information is sent to the amygdala for interpretation. When danger is registered, the amygdala rapidly sends a signal to the hippocampus. The hippocampus communicates with the body through the autonomic nervous system. The two pathways of the autonomic nervous system are the sympathetic and parasympathetic responses. The sympathetic system orchestrates changes in physiology which are commonly referred to as the fight-or-flight response (Bolis et al., 2003). Signals are sent to the adrenal glands which in turn begin circulating epinephrine throughout the body. Epinephrine influences an increase in heart rate, blood pressure, respiration, and a release of stored energy into the blood stream in order to mobilize action to escape from the impending danger. As initial epinephrine levels begin to decrease, the hypothalamus-pituitary-adrenal (HPA) axis begins to activate. The HPA axis is reliant upon signals received from the sympathetic nervous system. If the brain continues to perceive danger, a sequence of events begins with the hypothalamic release of corticotropin-releasing hormones which travel to the pituitary gland. The pituitary gland responds by releasing adrenocorticotropic hormone. The hormone travels to the adrenal glands prompting a secretion of cortisol. Cortisol keeps the system at a heightened alert as well as works to reduce circulating inflammation (Yeager, Pioli, & Guyre, 2011). When a danger is no longer registered, the parasympathetic or “rest and digest” response engages and discontinues the stress response.

Chronic levels of stress are problematic in part because of persistent output of the hormones associated with the response to perceived stress. Persistent epinephrine surges can damage the blood vessels and arteries which in turn increase blood pressure and risk of heart attack and stroke (World Health Organization, 2005). Chronic high levels of cortisol are also

problematic. Cortisol levels typically follow a diurnal rhythm in which cortisol levels are high upon waking, increase approximately 30 minutes after initial awakening, and begin to drop rapidly after the 30-minute surge. Throughout the day, cortisol levels typically drop at a slower rate until hitting their expected lowest point around bedtime. Chronic stress can disrupt the slope by flattening the decrease of cortisol throughout the day which may pose major consequences. An insufficient output of cortisol may affect our ability to respond to success efficiently. For example, prior studies have found an association between flatter cortisol slopes and depression (Doane et al., 2013), fatigue (Bower et al., 2005), and cardiovascular disease (Matthews et al., 2006). In a meta-analysis of early life adversity on cortisol response to social stress, it was found that childhood adversity more strongly related to a blunted cortisol response (Bunea, Szentágotai-Tătar, & Miu, 2017). Studies looking at specific measures have found that childhood emotional abuse associated with significantly diminished cortisol response when tested by DEX-CRH test, a reliable tool used to evaluate neuroendocrine course and autonomic dysregulation in psychiatric disorders (Carpenter et al., 2009). Individuals from risky families have shown to have lower cortisol awakening response and higher bedtime cortisol levels (Willner et al., 2014). Blunted cortisol responses may be problematic by diminishing an individual's physiological ability to reduce inflammation associated with stress. As shown, individuals raised in risky or abusive family environments may be at risk for disruption of normative cortisol responses. Dysregulated cortisol levels at baseline and in response to stress may be particularly important for memory outcomes. Previous research has shown that too little or too much circulating cortisol in the brain may disrupt memory consolidation and retrieval (de Quervian, Roozendaal,

& McGaugh, 1998; Roozendaal et al., 2003). It is possible that lower levels of cortisol associated with childhood emotional abuse and childhood risk may be a pathway that affects memory.

Biological Embedding of Adversity Model

Childhood adversity is thought to get under the skin and affect physiological systems and processes. Previously mentioned is research that may provide insight into how childhood adversity affects molecular mechanisms and alter physiology. The biological embedding of childhood adversity theory posits that early life adversity might not affect endocrine systems alone. Childhood adversity may influence neural, immune, metabolic, and gut microbial outcomes as well. Early life adversity has shown to pervasively change brain structure, dysregulate HPA axis responding, influence chronic inflammation, and influence gut dysbiosis (Berens, Jensen, & Nelson, 2017). The theory suggests that events early in life may have a higher prevalence of becoming embedded in physiology due to a greater responsiveness to experience (i.e. plasticity). Sensitive periods refer to timeframes during the lifespan in which the effects of environmental stimuli on the developing individual are particularly strong. Early childhood is viewed as a sensitive period for the programming of a wide range of systems and processes including metabolic processes (Miller, Chen, & Parker, 2011) and the immune system (Avitsur et al., 2015). According to the biological embedding of early life adversity model, increased threat responsive brain programming may be adaptive and protective in the context of high threat or adversity but may be maladaptive if it persists into adulthood in spite of changes in the environment (Berens, Jensen, & Nelson, 2017). Prior work has shown increased amygdala activity in response to emotionally negative faces for those who experienced childhood maltreatment (Elzinga, 2013). It is possible that individuals who experience childhood adversity

develop altered pathways that respond and engage to negative stimulus above and beyond other forms of stimulus such as neutral or positive. This perceptual bias could be detrimental to adult memory outcomes.

Pathways Connecting Early Life Adversity to Later Health

Three pathways have been identified which may in part explain the relationship between childhood abuse and poor mental and physical health in adulthood. First, abuse is commonly viewed as an experience which contributes to previously mentioned dysregulated stress response. Second, abuse is thought to promote poor health behaviors (e.g. addictive behaviors), which increased risk of downstream adverse mental and physical health outcomes. (Phillips, Ginty, & Highes, 2013; Pitzer & Fingerman, 2010). Third, it is posited that early life adversity may affect health in adulthood by affecting appraisals of stress. (Ellis & Boyce, 2008; Liu et al., 2013).

In consideration of differences in physiological stress responding, there is mixed findings regarding the manner in which early life abuse and adversity shapes the nature of the stress response. Some research indicates that abuse associates with exaggerated reactivity (Jovanovic et al., 2010; Elzinga et al., 2010), while others find that abuse associates with blunted reactivity. Blunted reactivity refers to diminished or substantially lower physiological increase to a stressor. Some research suggests blunted reactivity is associated with motivational dysregulation in the brain. Specifically, Ginty and colleagues (2013) found that blunted reactivity associated with deactivation in the amygdala and frontal cortex independent of self-reported task engagement, difficulty, and stressfulness. In line with this, it is possible that blunted reactivity may bias memory through dysregulated pathways within brain systems responsible for processing stimulus. In addition to physiological responses to stress, early life adversity is thought to affect

individual appraisal of threat. The experience of early life adversity has shown to influence interpretation of ambiguous information as more threatening compared to low adversity counterparts (Chen et al., 2004) Living in an environment that is undesirable has shown to increase individual sense of danger and uncertainty (Taylor & Shumaker, 1990) which in turn may leave individuals more likely to create interpretations and/or recognize threat. It is possible that pathways of blunted stress reactivity and dysregulated brain processes which has been previously shown to associate with childhood emotional abuse and levels of risk affects memory through recognize and perception of threat.

Stress and Memory: Consideration of the Role of Early Life Adversity

Cortisol is well documented as a critical influence of inflammation and stress. Cortisol has also associated with differences in memory processes and outcomes. For example, Increased cortisol has been shown to impair episodic memory retrieval (Wolf, 2009), reduce autobiographic memory retrieval at high doses (Young et al., 2011), and lower verbal memory performance (Domes et al., 2005). In a small sample of adult females, researchers investigated the role of cortisol on memory retrieval when examining mental disorders. The healthy control group showed increased levels of cortisol associated with compromised memory retrieval. Interestingly, an inverse relationship existed for those with borderline personality disorder where higher cortisol seemingly had an enhancing effect on memory resulting in increased recall (Wingenfeld, 2013). Less is known about the effect of cortisol on memory for those who experienced risk or abuse in childhood. Previously mentioned work showed low levels of circulating cortisol, which has been found in adults who experienced childhood abuse and/or

risk, may impair memory (Roozendaal et al., 2003). A similar relationship may persist for those who experienced abuse or risk in childhood.

Prior research has shown those who experience childhood adversity experience greater stress (Bunea et al., 2017). Research has found associations between heightened stress and poorer performance on memory recall tests for both adults and children. In one research investigation, children participated in the Trier Social Stress Test, a well validated in-lab stressor in which participants are asked to give a speech in front of evaluators. The participants then completed a delayed memory recall test in which overall word recall appeared to be negatively affected by stress (Quesada et al., 2012). Separate research work provided mechanistic evidence that cortisol affects memory. Individuals who were given an intravenous glucocorticoid injection (i.e. raising cortisol levels) had significantly lower word recall (Wolf et al., 2001; Coluccia et al., 2008). While this work indicates relationships between stress and memory, recent research has not investigated the relationship between early life adversity and memory processing in college students following a stressful experience. It is possible that differences in physiological and psychological responses to stress related to childhood abuse and risk, may contribute to differences in memory recollection and memory consolidation when comparing to low risk/no abuse counterparts. Previous research has shown that early life adversity impedes important brain structure functioning involved in memory such as the hippocampus and prefrontal cortex while increasing amygdala activity and sensitivity to stressors and emotional responses later in life (Krugers et al., 2017). However, the research did not examine how these differences may influence memory. Memory differences related to early life adversity could have important academic and social implications for college students. For example, research has identified an

association between childhood abuse and neglect and higher dropout rates in comparison to those reporting low levels of abuse and neglect (Duncan, 2000). Memory disruptions influenced by early family environment may contribute to observed dropout rates and impair overall academic performance.

While prior work has considered how psychosocial traits may affect memory biases, the work in this domain largely focuses on symptoms of anxiety and depression as predictors of negative memory bias (Eden et al., 2014; Herrera, Montorio, & Cabrera, 2017; Krans, de Bree, & Bryant, 2014; Pzyszczynski et al., 1989; Hamilton & Gotlib, 2007). A negative memory bias is defined as increased recollection and attention to emotionally negative stimulus in comparison to neutral stimulus (Joorman, Teachman, & Gotlib, 2009). Research on individuals with depressive symptomology has shown that compared to those without depressive symptoms, individuals with depressive symptomology have greater recollection of negative words, lower overall word recall, and these individuals are more likely to falsely recall negative words in a word memory test (Blaut et al., 2013; Joormann, Teachman, & Gotlib, 2009). Individuals with anxiety display similar outcomes. For example, negative valence words were better recognized by individuals with anxiety compared to non-anxious individuals (Ho et al., 2018) and anxious individuals had a higher recall of negative autobiographical memories (Krans, De Bree, & Bryant, 2014). This body of work focuses on current symptoms of depression and anxiety and does not consider the impact of early life adversity on memory. Childhood risk and/or abuse may be additional outcomes predictive of memory recall that is enhanced for information that is negatively valenced.

Pathways Connecting Early Life Adversity to Memory

Previously mentioned research highlighted pathways that connect early adversity to health. These included differences in physiological responses to stress, differences in health behaviors, and differences in psychological appraisals of stress. Based on this body of work, it is possible that that early life risk and abuse may affect recall and memory biases through two of these pathways. First, it is possible that early life risk or abuse may affect these outcomes through differences in stress appraisals. In a rodent model, a controlled post-natal maternal separation associated with impairments of the fear processing parts on the brain into adulthood (Chocyk et al., 2014). In humans, individuals from more risky family environments during childhood are more likely to appraise ambiguous situations as threatening (Chen & Matthews, 2001) Secondly, it is possible that risk in early life family environments may affect memory processes by shaping the pattern of physiological responses to stress. For example, research indicates that individuals from high risk family environments display a dysregulated stress response characterized by accelerated reactivity to potential stressors, prolonged responding, and difficulties disengaging from a stressor after it has been eliminated (Repetti, Taylor, & Seeman, 2002). As noted previously, separate research also indicates that abuse early in life associates with blunted patterns of reactivity (Lovallo et al., 2012; Hengesch et al., 2018).

Overview and Hypotheses

Based on previous literature, I hypothesize that if a relationship exists between early life abuse or risk, biased memory recall, this relationship will be partially explained by differences in psychological appraisal and physiological reactivity in response to a stressor. See Appendix A

for conceptual model. While it is also possible that early life adversity may affect memory recall through health behaviors, this is outside the scope of the current work. I predict that greater early life adversity will associate with increased threat appraisals of the stressor as well as blunted heart rate and blood pressure reactivity responses to the stressor. Further, I predict that these patterns will shape memory recall and bias. With regards to memory, I predict that individuals who experienced more abuse or risk during their childhood will have lower overall recall of words, higher recall of negative words, and greater false recall of negative items in a memory recall test. The following outcomes are hypothesized to be partially explained by diminished physiological reactivity and heightened threat appraisals of the stressor. Theses hypotheses are outlined below:

Hypothesis 1: Increased risk or abuse during childhood will associate with lower word recall, increased negative word recall, and increased negative word intrusions.

Hypothesis 2: The relationship between childhood abuse or risk and word recall will be partially mediated by physiological response to stress.

Hypothesis 3: The relationship between childhood abuse or risk and word recall will be partially mediated by psychological appraisal of stress.

See appendix for statistical models

STUDY ONE

METHOD

Participants

Participants were drawn from a convenience sample at Montana State University comprised of introduction to psychology students. As part of their coursework, students participate in research studies for class credit. Participants were recruited via SONA systems online data base where they can locate study participation time slots. Exclusion criteria for this study included individuals with pre-existing chronic health conditions and/or taking medications that affect inflammation or cardiovascular outcomes including blood pressure and heart rate.

Measures

Background and demographics

Participants were asked to self-report basic demographic information including age, gender, race, and parental occupation, income, and education.

Childhood Family Environment

The *Risky Families Questionnaire* (Taylor et al, 2004; Felitti et al., 1998) was administered to assess levels of abuse and neglect as well as levels of warmth in childhood family environments from the ages of 5-15 years old. Questions are rated on a 5-point Likert scale ranging from 1= not at all to 5= very often. To assess levels of risk in familial environment, all items related to abuse and chaos are summed to capture an overall total rating of risk. Example questions include, “how often did a parent or other adult in the household swear at you,

insult you, put you down, or act in a way that made you feel threatened?" and "how often did a parent or other adult in the household make you feel that you were loved, supported, or cared for?" The *Risky Families Questionnaire* is unique in which measures of total risk can be calculated to include reports of familial warmth. Example questions of warmth include, "how often did a parent or other adult in the household make you feel that you were loved, supported, and cared for?" and "how often did a parent or adult in the household express physical affection for you, such as hugging, or other physical gestures of warmth and affection?". Questions targeting warmth are reverse scored and added to the total measure of risk to create a separate variable that examines total risk with the consideration of the absence of warmth and affection as an additional risk factor. The *Risky Families Questionnaire* was internally consistent in our sample (α=.78).

The *Childhood Trauma Questionnaire* (Bernstein et al., 1994) is a 28-item test that measures severity of abuse and neglect in childhood. Responses are rated on a 5-point Likert scale ranging from 1= never true to 5= very often true. Responses from the questionnaire can be separated by type of abuse and neglect in order to explore differences amongst categories. Example questions include, "I thought my parents wished I was never born", "I believe I was sexually abused", and "I felt loved". The childhood trauma questionnaire includes 5 subscales of abuse and neglect. The subscales include: emotional abuse, emotional neglect, physical abuse, physical neglect, and sexual abuse. In this work, the subscales demonstrated good internal consistency (α=.72).

Psychosocial outcomes

The *Hospital Anxiety and Depression Scale* (Zigmond & Snaith, 1983) determines the levels of anxiety and depression that an individual is currently feeling. Participants respond to statements on a 4-point Likert scale ranging from 0= not at all to 3= most of the time. Example statements include, "I still enjoy things like I used to." and "I feel tense or wound up". There are two subscales in the questionnaire assessing depression and anxiety. Each subscale consists of seven questions in which questions within are summed to create a total score where higher scores reflect greater current symptoms. Many studies have confirmed the validity of the HADS as a useful instrument in multiple settings (Snaith, 2003) including the use in college samples (Brar & Moneta, 2009; Andrews & Wilding, 2004) when examining downstream adverse outcomes. The anxiety and depression subscale in this sample demonstrated good internal consistency (α>.75).

Perceived socioeconomic status

The *MacArthur Scale of Subjective Social Status* (Adler, 2004) is a single item measure that determines an individual's perception of their social rank in comparison to others around them. Participants are presented with a 10-rung ladder and told to consider the image as a representation of where their family stands relative to the rest of society. The bottom of the ladder represents those with the lowest income, education, and least respected jobs in society. Participants were shown the following prompts**:** "When you were a child, where would you place your family relative to the rest of the society with regards to socioeconomic status (e.g. income, education, occupation**).** The bottom of the ladder represents families with the lowest income, lowest level of education, and the least respected jobs or no jobs. The top of the ladder represents families with the highest income, highest education and most respected jobs." and

"think of the ladder with 10 rungs representing where people stand IN OUR SOCIETY**.** At the top of the ladder are the people who are the best off, those who have the MOST MONEY, MOST EDUCATION, and BEST JOBS. At the bottom are the people who are the worst off, those who have the least money, least education, and worst jobs or no job. Between 1 (bottom) and 10 (top), where do you think you stand on the ladder?". These two measures capture subjective assessments of current socioeconomic status and childhood socioeconomic status, respectively. Both scales of the measure have been previously used in college students (Counts, Grubin, & John-Henderson, 2018, Zorotovich, Johnson, & Linn, 2016).

Physiological measures

Electrocardiogram (ECG) were recorded to determine heart rate. Six electrodes (circular stickers with a place in the center to attach wires) were placed on the participants. One electrode was placed on the right hip and one electrode is placed on the left hip. One electrode was placed below the left collarbone and one electrode is placed on the lower part of the sternum. One electrode was placed on the back of the neck and the final electrode was placed on the spine, 2 inches above the adjacent location of the electrode located on the sternum. Participants had the option to place the electrodes on themselves or have a researcher assist them. Six wires (one to each electrode) were then attached and participants were given a small box to clip on their pants (MindWare Mobile Impedance Cardiograph, Model 50-2303-00). The ECG data was transmitted to a computer in which the trace of heart rate activity was recorded.

Salivary cortisol. Saliva samples were collected using a double tube salivary cortisol collection device for future assessment of circulating levels of the hormone cortisol. In the device is a piece of cotton that participants are asked to chew on for 60 seconds before replacing

the cotton back into collection device. Cortisol was collected before the preparation period and 5 minutes after the recovery period. The collected samples were not utilized in the analyses for the results of the study.

Blood spot samples. Dried blood samples were collected to measure levels of the inflammatory immune system protein C-reactive protein (CRP). Participants are asked to administer a small insertion into the tip of their finger with a 21-gauge pressure activated single-use safety lancet. After using the lancet, participants are asked to manually apply 5 separate blood spot sample to a Whatman protein saver card for later analysis. Samples were collected before the start of the preparation period and 5 minutes after the recovery period. Samples were not processed and included in the analyses of the study.

Blood pressure and heart rate measures. Participants wore a blood pressure cuff on an arm that was chosen by the participant for comfort. Research assistants collected a reading from the machine every 2 minutes during the 10-minute anticipation period, totaling 5 readings. During the stress task, a reading was collected at the 1-minute, 3-minutes, and 5-minutes totaling 3 readings. Finally, during the recovery period, a reading was collected every 2 minutes during the 10-minute period totaling 5 readings. Data was cleaned and analyzed by collecting a single mean score of blood pressure and heart rate for each of the three periods.

In Lab Measures

Cognitive Appraisal survey. The cognitive appraisal survey was a three-part survey aimed at assessing levels of anxiety to the stress induction task, perceptions of the demand as challenging or threatening, and feelings of stress to the upcoming task. Questions were rated on a 7-point Likert Scale. Example questions include, “I am cognitively anxious”, “I view the task as

a threat", and "how stressed do you feel about the upcoming task". Items are summed based upon category in which higher numbers equates to increase of target outcome.

The *In-Lab Memory Test* was a 50-item (25 pair) word list was utilized for the study delayed free recall test. Word pairs were selected from Macleod (2007) 140-word pair list that connected words based upon length and frequency. The word pairs included an emotionally neutral and emotionally negative word rated by a panel of psychology students. Word pairs were targeted to include words relevant to early childhood adversity (i.e trauma, assault, distress).

Post-Memory Recall Survey. A 9-question survey was administered after the free recall memory test to assess levels of confidence in ability to recall the target words, perceptions of physiological arousal to words, and subjective reports of the influence of physiological reactivity on memory influence. The questions were rated on a 5-point Likert scale in which 1 = not very confident, not very strong or not very much to 5 = very confident, very strong, or very much.

Procedure

Participants (N=64) were recruited from the SONA pool at Montana State University consisting of introduction to psychology students. As part of their coursework, students are asked to participate in research timeslots. Research timeslot were scheduled in 1 hour and 30 minute blocks. Participants were given a consent form and encouraged to ask the research assistant any questions or concerns they may have. After the consent form was completed, participants were seated at a desk with a computer and read the following memory recall test instructions by a research assistant: "You will see a series of words presented on the computer screen. Your job is to simply pay attention to each of these items because your memory for them will be tested later. No response on the computer is necessary. Simply pay careful attention to each word

because I am going to test your memory for these words. Do you have any questions before we start? Go ahead and click the mouse key to begin." A total of 50 words were presented individually on the screen. Each word was presented for a duration of 3 seconds. Participants were shown the word list at the beginning in order to reduce potential physiological arousal that may occur after providing biomarkers including blood and saliva, as well as equipping the ECG device.

Next, participants were returned to a medical chair in which they provided a baseline cortisol and blood spot sample. Research assistants guided the process, and helped if a participant was unclear of any of the procedure. Once the samples were properly stored, research assistants began the process of setting up the ECG device on the participant. A total of six electrodes were placed on both hips, at the bottom of the sternum, the upper middle chest below the jugular notch, mid back, and upper back/neck area in order to collect readings of heart rate variability and impedance. Once the participant was seated with the ECG device, the participant began the preparation period for the Trier Social Stress Test. The following script was read to the participant: "You will be participating in a speech task today. This is the preparation portion of the task. You are being asked to present a 5-minute oral speech describing why you would be a good candidate for your ideal job. Your speech will be videotaped, and recorded by a panel of judges trained in public speaking. You will be given 10 minutes to mentally prepare and I will be collecting blood pressure measurements throughout the 10-minute period". The research assistant asked the participant if they had any questions and begin once any possible concerns were addressed. During the 10-minute period a research assistant recorded blood pressure and heart rate measures every 2 minutes, collecting 5 measurements during the period. At the end of

the 10-minute period, a research assistant read the following script: “this is the speech portion of the task. You have 5 minutes to describe why you would be a good candidate for your ideal job. You should speak the entire 5-minute period. Your time begins now”. During the speech task, blood pressure readings were collected at 1, 3, and 5-minute mark. When the 5-minute period was over, a research assistant provided the participant with an IPad which had the cognitive appraisal survey loaded from a Qualtrics database. After completion of the online survey, a research assistant provided the participant with a blank word recall sheet and read the following instructions: “Now you’re going to complete a memory test. I need you to recall as many items as you can from the words previously presented on the computer screen. I ask that you don’t guess, but please try to recall as many items as you can from the list. I’ll give you a sheet of paper, and you are to write down as many words as you can remember from the computer presentation. You can recall the words in any order. You will have five minutes. If at the end of the five minutes you need more time to finish recalling the items, please let us know and you will be allowed extra time.” None of the participants asked for extra time and the average time it took to recall was approximately two minutes. Once the participant declared they were done with the recall, a research assistant filed away the recall sheet for later analysis.

A 9-question survey created by Dr. Michelle Meade was administered to the participant after the memory recall test. The survey included questions asking about confidence in memory recall for each type of word, and the influence of physiological arousal on the recall of studied words. Next, the participant began a 10-minute recovery phase. During the 10-minute recovery period, blood pressure was recorded in 2-minute intervals, totaling 5 readings. An additional 5-minute waiting period proceeded the 10 minutes. A post-task salivary cortisol sample and blood

spot sample was collected after the 5 minutes. Before ending the in-lab portion of the study, research assistants asked the participant to provide a valid email address in which a group of surveys were sent 24 hours after the lab visit. Surveys were scheduled to send automatically after 24 hours containing the survey link and participant ID. An in-lab debriefing form was given to the participant and a research assistant explained the true nature of the stress test. Research assistants were trained to tell the participants that the stress induction task was designed to be stressful and does not reflect their true abilities as well as the deception used in which they were not actually being recorded.

Credit was granted to the participant after the completion of the 24-hour post survey. The survey included a 24-hour post lab memory recall test in which participants were asked to think back to the words they studied for the memory test during their lab visit and report as many as they could remember. Other surveys included were demographics, the *MacArthur Ladder of Subjective Social Status, Risky Families Questionnaire* and *Childhood Trauma Questionnaire*.

Statistical Analyses

In order to test the hypotheses of the study we used IBM SPSS software version 26 (IBM Corp., 2019). To test Hypothesis 1, we utilized hierarchal multiple regression with the covariates of age, sex, depression, anxiety, childhood socioeconomic status, and current socioeconomic status. Inclusion of these covariates was based on documented associations between these factors and our outcomes of interest. Previous research suggests there are differences in negative and positive word recall based upon age (Kensinger, 2008) and sex differences in the brain during memory formation (Mizuno & Giese, 2010). As previously mentioned, depression and anxiety are included as covariates based on previous work indicating their relationship with negative

memory bias in previous research (Pyszczynski et al., 1989; Hamilton & Gotlib, 2007; Herrera, et al., 2017). We included subjective measures of SES in childhood and adulthood collected from the Macarthur Ladder of Subjective Social Status, based on previous research indicating these factors associate with physiological and psychological reactivity to stress (Chen, Miller, & Matthews, 2007; Evans et al., 2013).

The first series of hierarchal regression analysis tested whether early life abuse and risk predicted total word recall. In block 1, we added all of the previously described covariates. In block 2, we added the following measures in three separate regression models: risky family total, risky family total with warmth, and childhood emotional abuse.

The second series of hierarchal regression analysis tested whether early life abuse and risk predicted total negative word recall. Covariates were added into block 1. In block 2, we added risky family total, risky family total with warmth, and childhood emotional abuse in three separate regression models.

The final series of hierarchal regression analysis for Hypothesis 1, tested whether early life abuse and risk predicted negative word intrusion (false recall of negative words). Covariates were added into block 1. In block 2, we added risky family total, risky family total with warmth, and childhood emotional abuse in three separates regression models.

Next, we conducted analyses to test Hypothesis 2, which posited that physiological reactivity would mediate the observed relationships between early adversity and negative word recall. We utilized the SPSS version 26 Hayes process macro mediation model 4 (Hayes, 2018) to test this hypothesis. Specifically, we examined whether the relationship between reported childhood emotional abuse and negative word recall was mediated by average heart rate during

the stressful task. In this model, we included the previously described covariates, and also added average heart rate levels during the period preceding the task.

Hypothesis 3 was not able to be tested due to significant missing data (n=17). Bivariate correlations are reported for psychological appraisal of threat and interest variables.

RESULTS

Descriptive Statistics

Descriptive statistics for main variables of interest are listed in **Table 1** located in the Tables section below. The average age of participants was approximately 20 years old and the sample was relatively balanced on reported biological sex (59% female).

Next, bivariate correlations were calculated in order to investigate relationships between childhood adversity measures and interest items including model covariates and outcome variables. The results from these analyses are displayed in **Table 2** located in the Tables section below.

Bivariate Correlations

In line with the Hypothesis 1, there was a positive correlation between total negative word recall and risk in early family environments (r = .41, n = 62, *p* = .001), risky family environment with warmth (r = .42, n = 61, *p* = .001), and childhood emotional abuse (r = .41, n = 62, *p* = .001). The results were not significant for childhood physical abuse or sexual abuse.

Post 24-hour word recall did not associate with any of the measures of childhood adversity (rs>.23, *p*s>. 08)

Average heart rate during the stressful task was significantly related to childhood emotional abuse (r = -.73, n = 61, *p* = <. 001), and risky family environment with warmth items (r = -.28, n = 60, *p* = .03). Specifically, greater reports of emotional abuse during childhood and more risky family environments associated with lower average heart rate during the task. Baseline systolic blood pressure did not significantly relate to any of the measures of childhood adversity. Baseline diastolic blood pressure was negatively related to childhood emotional abuse

(r = -.53, n = 63, p = <.001), with greater reports of childhood emotional abuse associating with lower baseline diastolic blood pressure. While task systolic pressure did not significantly relate to childhood adversity, there was a negative correlation between task diastolic blood pressure and childhood emotional abuse (r = -.4, n = 61, p = <.001), with greater reports of childhood emotional abuse associating with lower task diastolic blood pressure.

Childhood Adversity, Word Recall and Intrusions

Next, hierarchal multiple regression analyses were performed to further understand the observed relationships between main variables of interest. The results of the first regression analyses focused on the relationship between childhood adversity and total word recall are displayed in Figure 3. Total word recall was calculated by summing all correct response from the list of 50 total items including the neutral and negative target words. Measures of early life risk or abuse did not predict lower rates of total word recall (β = <.26, p = > .08).

The risky family total measure associated with higher negative word recall rate (β = .42, t(48) = 2.96, p = < .01). Risky family total explained a significant proportion of variance in negative word recall R^2 = .13, F(7, 48) = 2.72, p = .02. Risky family total with warmth measure associated with higher negative word recall rate (β = .39, t(48) = 2.73, p = < .01) and explained a significant proportion of variance in negative word recall R^2 = .11, F(7, 48) = 2.50 p = .03. Childhood emotional abuse was associated with higher negative word recall (β = .43, t(48) = 3.18, p = < .01) Childhood emotional abuse explained a significant proportion of variance in negative word recall R^2 = .15, F(7, 48) = 2.95, p = .01.

Other measures of childhood adversity did not predict increased negative word intrusions (all βs = < .62 p = > .33).

Other measures of childhood adversity did not significantly predict post 24-hour memory recall βs = < .23 p = > .08).

Childhood Adversity and Physiological Reactivity

Next, we examined the relationship between childhood abuse or risk and physiological reactivity to the in-lab stressor. First, we tested the relationship between childhood adversity and heart rate reactivity. In separate hierarchal regression models controlling for age, sex, depression, anxiety, childhood SES, and current SES, childhood emotional abuse predicted blunted heart rate reactivity to the stress task (β = -.67, t(48) = -6.27, p = < .01). Childhood emotional abuse explained a significant proportion of variance in task heart rate average R^2 = .13, F(7, 47) = 9.23, p = <.01 The risky family total, risky family total with warmth, childhood physical and sexual abuse did not significantly predict heart rate reactivity to the stressor task (all βs = < .25 p = > .22).

Next, we examined the relationship between childhood abuse or risk and diastolic blood pressure. In separate hierarchal regression models controlling for age, sex, depression, anxiety, current and childhood SES, childhood emotional abuse significantly predicted lower diastolic pressure reactivity to the stress task (β = -.15, t(47) = -3.76, p = < .01). Childhood emotional abuse explained a significant proportion of variance in diastolic pressure reactivity R^2 = .22, F(7, 47) = 2.71, p = .02. Risky family total, risky family total with warmth, childhood physical and sexual abuse did not significantly predict diastolic blood pressure reactivity to the stress task (all βs = < -.83 p = > .30).

Next, we examined if childhood adversity predicted systolic blood pressure reactivity to the stress task. In separate hierarchal multiple regression analyses controlling for age, sex,

depression, anxiety, current SES, and childhood SES we examined each measure of childhood adversity. Measures of childhood adversity did not predict systolic blood pressure (all βs = < .21 p = > .12)

Childhood Adversity, Physiological Reactivity and Negative Word Recall

To test Hypothesis 2 which posited that heart rate reactivity would mediate observed relationships between early life adversity and memory outcomes, we utilized multiple regression analyses and Andrew Hayes process model on SPSS version 26. (Hayes, 2018). As with our previous analyses, we included anxiety, depression, age, sex, current socioeconomic status, and childhood socioeconomic status as covariates and added baseline average heart rate levels as an additional covariate. The relationship between childhood emotional abuse and total negative word recall was partially mediated by task heart rate. **Figure 1** shows the standardized regression coefficient between childhood emotional abuse and task heart rate was statistically significant as well as the standardized regression coefficient between task heart rate and total negative word recall. We tested the significance of the observed indirect effect using Hayes process mediation model 4 in IBM SPSS version 26. Unstandardized indirect effects were computed for each of the 5,000 bootstrapped samples within the model. The bootstrapped unstandardized indirect effect was -.09, and the 95% confidence interval ranged from -.22 to -.02 indicating the indirect effect was statistically significant. We did not analyze the mediation model for risky family total, risky family total with warmth, childhood physical or sexual abuse due to lack of significant relationship in hierarchal regression model.

Due to missing data, the proposed mediation model of the relationship between childhood adversity and negative word recall mediated by psychological appraisal was not tested.

GENERAL DISCUSSION

In a sample of college students, higher reported risk and adversity in childhood associated with negative word recall on a short-delayed free recall test. To date, past research focused on the relationship between psychosocial factors and memory outcomes has largely focused on anxiety and depression. Further, previous work on negative memory bias has predominantly utilized clinical populations (Bradley, Mogg, & Miller, 1996; Docteur et al., 2013). The current research investigated these relationships in a sample of college students. We found that after accounting for anxiety and depression within the models, higher risk in childhood family environments and greater reports of childhood emotional abuse predicted increased recall of negative words. As mentioned, previous investigations have failed to consider the role of childhood family environments and childhood abuse in shaping performance on free recall memory tasks. A study conducted by Vogel and colleagues (2014) found an association between negative memory bias and early life adversity when using a self-referent encoding task. The researchers had a 2.5-minute delay between encoding and free recall. The study used an adapted version of the List of Threatening Events questionnaire which captures an overall sum of general adverse events. Our study appears to be unique to previous research on early adversity as we were able to show differences in memory outcomes by specific abuse or risk experienced in childhood. Emotional abuse and higher risk in childhood were found to predict negative memory bias while physical and sexual abuse as well as emotional neglect did not. The outcome highlights potential differences that may exist between type of abuse experienced in childhood and later life outcomes, adding to the limited literature related to how different subtypes of childhood abuse may influence overall health (Infurna et al., 2016; Witt et al., 2016).

Furthermore, the memory test used in this research did not use a self-reference condition when memorizing negative or neutral words. Some research argues that self-reference has one of the most powerful effects on memory (Dewhurst et al., 2017). We were able to find a similar memory outcome in a word recall test that used intentional encoding (i.e., "you will be asked to recall the words you see later") as well a much longer delay between encoding and free recall (approximately 25-30 minutes).

When compared to similar free recall memory tests, our study produced a profoundly low rate of recall regardless of early family environment (avg. ≅ 10% total word recall). Research which presented words for recall in a similar manner and featured a delayed recall observed average recall rates of approximately 50% (Roediger, Meade, Gallo, & Olson, 2014). The results highlight the potentially debilitating effects that immediate stress exposure has on memory. This outcome has important implications for college students. As mentioned, college is characterized by increased exposure to stressors. An inability to manage stress efficiently may be detrimental to academic outcomes such as testing performance. Further, stress may be affecting memory in an increasingly complex way for college students who experienced risk or abuse during childhood. Recollection of material studied may be biased towards information that is emotionally negative and degraded for neutral or positive information which would affect long term success through failure to recall total information. We also collected data on memory recall 24 hours following the in-lab visit. We found that childhood adversity did not associate with word recall 24 hours after the in-lab visit. This may indicate that early life adversity may affect the relationship between stress and memory in acute situations, but these effects may not persist long-term.

In line with a growing body of research, we found that greater familial risk and emotional abuse in childhood associated with blunted physiological reactivity to a validated stressor (Carpenter et al., 2007). These findings potentially reflect differential patterns of responses to stress at the brain level. Structures of the brain which are involved in orchestration of the stress response show significant plasticity during early childhood, as well as some plasticity during later childhood and adolescence (Teicher et al., 2006; Fumagalli et al., 2007). As such, it is possible that adaptation of structures in response to exposure of risk and adversity during childhood may translate to differences in cardiovascular and mental responses to stress, which may persist into adulthood. Duration, timing, and intensity of adverse childhood events may play a role. Environments that demand excessive and prolonged stress response during critical periods of development may be linked to abnormal neurodevelopment which inform alterations in stress reactivity (Schoedl et al., 2010). Future research should focus on timing of abuse or risk to determine if differences exist based upon age of occurrence.

Previous research has shown an association between blunted reactivity and maladaptive outcomes (Brindle et al., 2013; Ginty et al., 2012; Ginty et al., 2016; John-Henderson et al., 2019). It is possible that blunted cardiovascular reactivity associated with the experience of childhood abuse or adversity may direct attention to negative words by way of heightened vigilance for threat through altered brain and perceptual pathways. If blunted reactivity is due to limited or dysregulated physiological capacity to respond, limited mental processes may be prioritized to respond and encode perceived threat stimulus in order to protect well-being. This possibility would make sense when considering animal and human species are hardwired toward survival.

Risky family environments predicted higher negative word recall rate, and this was true with the risky family environment measure that included only measures focused on adversity or trauma, as well as for the risk family environment measure that included measurement of the absence of warmth and affection. The measure which focused exclusively on risk had a somewhat stronger relationship with recall of negative words, compared to the measure which considered the absence of warmth and affection. This may slightly reflect the potential for warmth and affection to buffer against the negative effects of early life adversity on outcomes later in life. As noted earlier, research conducted by Carroll and colleagues (2013) suggested that parental warmth may reduce the degree to which childhood abuse affects biological stress responses. In a similar manner, it is possible that warmth may dampen the association between early life adversity and memory biases. Future research is needed to better understand the potential for warmth and affection to buffer the degree to which early life risk may affect memory outcomes into adulthood.

The use of the *Childhood Trauma Questionnaire* resulted in an association similar to previous research showing emotional abuse predicted outcomes above other abuse or neglect outcomes. Here, childhood emotional abuse predicted increased negative word recall while childhood emotional neglect did not affect negative word recall in a similar manner. While abuse is referred to as an active involving a voluntary behavior, neglect reflects the absence of provision of emotional support and emotional resources including warmth and affection. In the present study, emotional abuse was measured with items such as “I thought my parents wished I was never born,” while neglect was measured by agreement with the statements of items such as “I felt loved” which were negatively scored. In a sample of high risk youth, emotional abuse was

found to be the main independent predictor of psychiatric symptomology above all other forms of abuse and neglect (Cecil et al., 2017). Furthermore, research found that childhood emotional maltreatment in college students significantly associated with increased psychological distress which in turn negatively affected sleep quality, while reports of physical and sexual abuse did not (John-Henderson et al., 2018). We found that sexual and physical abuse did not significantly relate to increased word recall. It is possible that this data did not include sufficient variability in the measures to find a significant effect. As such, it is possible that in a sample of individuals who have experienced these forms of abuse in childhood, we would find that the degree or severity of abuse would predict differences in memory recall or biases. On the other hand, the possibility exists that emotional abuse may be more related to negative word recall because of increased exposure to negative and threatening dialogue during childhood. This increased exposure could make the negative words presented during the word recall more salient or relevant to individuals who experienced high levels of emotional abuse.

Biases in memory processing could have major implications for academic performance, performance in the workplace, social relationships and overall well-being. If certain types of information are prioritized in memory such as negative information, an individual may experience limitations in everyday life that demands sufficiency of memory. For college students, memory and recall of information is an important component to academic success. An individual who has biased memory may experience compromised academic success. It is also well documented that college is a time of increased stress and demand (Weinstein & Lavarghetta, 2009; Jones, Park, & Lefevor, 2018). In this research, we found a measure of physiological reactivity (e.g heart rate) in response to an acute stressor partially mediated the

relationship between childhood adversity and word recall. In the context of college, it is possible then, that individuals who have experienced high levels of adversity in childhood may experience similar physiological responses in response to academic stressors, which may negatively impact their performance on tests and other evaluations. A better understanding about the factors which may inform memory bias in this population is needed in order to inform future interventions aiming to improve memory, academic success, and well-being.

LIMITATIONS

There are important limitations of the research to note. First, the observed relationship was correlational, thus causation cannot be established. Future work should utilize longitudinal research designs in order to better understand the prospective relationship between childhood adversity and negative memory bias. It is important to note that modeling mediation analyses with cross-sectional data could provide biased and misleading results (Maxwell & Cole, 2007). Self-report measures of abuse and risk during childhood are retrospective and driven by subjectivity and interpretation. It is possible that higher reports of childhood abuse or risk reflect a general negativity bias. Due to access restrictions on campus, analyses of other physiological measures including cortisol, interleukin-6, and ECG readings were not able to be completed. When mandates are eradicated in response to the COVID-19 outbreak, further physiological measures will be added to the data to understand these relationships across different physiological systems. In spite of these limitations, the findings provide initial support of relationships between early life adversity, heart rate and diastolic blood pressure responses to an acute stressor, and subsequent memory recall. Finally, we were unable to investigate whether differences psychological appraisal of the task accounted for relationships between measures of early life adversity and memory recall. The data for these analyses are missing because of an error in the survey that the participants completed during the in-lab session. The mistake was discovered and resolved after 17 research participants. In order to overcome the limitation, additional research participants will be added to the sample in order to explore a potential influence of appraisal on the relationship between childhood adversity and negative recall. It should also be known that the goal of the research study was to collect data on at least 100

participants. Due to the COVID-19 pandemic, we were forced to seize data collection before we were able to meet the goal sample. We were able to find significant associations for two of the three hypotheses despite the small sample.

FUTURE DIRECTIONS

Future research will utilize the endocrine, immune system inflammation, heart rate variability and cardiac impedance measures which were collected as part of this project. This data will allow us to investigate whether early life adversity may influence memory recall following a stressful task by affecting patterns of activity across multiple physiological systems.

Within our data set, we observed a profoundly low rate of correct total word recall (avg. ≅ 10%) when considering the relatively brief time delay (Approx. 25-30 min.) between word study and word recall. To better understand the low recall we observed, we will be conducting a subsequent research study which will largely resemble the design of this research with one notable exception. This new research will include a new control group, in which in lieu of a stress induction task, participants will complete non-arousing filler tasks. The follow up experimental design will help us further understand the influence of acute psychological stress on memory recall, and we will be able to investigate whether the observed relationships are specific to the context of stress, or whether early life adversity would have similar implications for memory recall, even in the absence of psychological stress.

CONCLUSION

In a sample of college students, an association between childhood adversity and negative word recall was observed. Specifically, those who experienced higher rates of childhood emotional abuse recalled a significantly higher rate of emotionally negative words in a short-delayed memory test. This relationship was also found true for individuals reporting greater risk in childhood family environment. We did not observe the same relationship for emotionally neutral word recall or total recall. The results remained significant after controlling for age, sex, childhood and current SES, anxiety, and depression. These outcomes are consistent with those observed in previous work focused on the relationship between physiological reactivity to stress and memory. (Wang et al., 2011; Walker et al., 2011; Daughters et al., 2013; Barry et al., 2015).

To our knowledge, this is the first research to investigate the relationships between childhood adversity, physiological reactivity, and memory recall following the experience of acute psychological stress. The findings are novel and have important implications, and provide impetus for more research in memory outcomes for those who face childhood adversity.

LIST OF TABLES

***Table 1*. Descriptive Statistics of Interest Variables**

Variable	N	Mean (%)	Std. Deviation	Range
Age	59	19.97	3.47	18-37
Sex	61	59% (Female)	.496	1-2
Current SES	60	6.35	1.62	2-10
Childhood SES	60	6.5	1.8	2-10
Anxiety	61	15.31	4.36	7-27
Depression	61	11.69	3.42	7-21
RF total	63	21.94	8.56	10-45
RF total with affection	63	26.75	8.95	12-49
CTQ Sexual Abuse	60	6.08	3.2	5-18
CTQ Physical Abuse	61	6.56	3.3	5-23
CTQ Emotional Abuse	64	13.52	5.52	5-25
Total Recall	62	5.8	2.64	2-14
Total Neutral Recall	62	2.63	1.74	0-10
Total Negative Recall	62	3.18	1.82	1-10
Baseline Heart Rate Avg.	63	84.29	12.86	43.6-110.2
Task Heart Rate Avg.	61	89.54	11.64	62-113

Table 2. Bivariate Correlations of Interest Variables

Variables	Age	Sex	SES	Childhood SES	Anxiety	Depression	Total Recall	Neutral Recall	Negative Recall	24-hr total recall	Pre HR	Task HR	Threat Appraisal
Physical Abuse	.03	-.08	.01	-.29*	.31*	.32*	-.11	-.1	-.07	-.03	-.22	-.22	.08
Sexual Abuse	.0	.01	-.09	-.41**	.4**	.11	-.19	-.14	-.15	-.06	-.23	-.19	.14
Emotional Abuse	.07	-.25	-.29*	-.08	.3*	.11	.18	-.15	.41**	.23	-.54**	-.73**	-.21
Risky Family	-.04	.006	-.432**	-.27*	.1	.1	.24	-.14	.48**	-.11	-.29*	-.24	.05
RF with warmth	-.02	.05	-.447**	-.3*	.16	.2	.14	-.22	.42**	-.15	-.32*	-.28*	-.05

**. Correlation is significant at the 0.01 level (two-tailed)
*. Correlation is significant at the 0.05 level (two-tailed)

Table 3. Summary of hierarchal regression analysis for risky family total predicting negative word recall rate and heart rate average during stress task

	Negative Word Recall			**Heart Rate during Stress**		
Model 1(no covariates)	**β**	**S.E.**	***p***	**β**	**S.E.**	***p***
Risky Family Total	.48	.03	<.01	-.24	.17	.06
Model 2(with covariates)						
Risky Family Total	.42	.03	<.01	-.16	.22	.29
Age	-.09	.06	.93	.01	.47	.92
Sex	-.15	.44	.23	.23	3.04	.08
Depression	-.23	.08	.10	-.28	.53	.06
Anxiety	.75	.06	.46	-.16	.40	.27
Childhood SES	.33	.15	.04	-.16	1.05	.32
Current SES	-.21	.18	.23	.14	1.26	.42
Pre-Heart Rate Avg.	-	-	-	.86	.08	<.01

Table 4. Summary of hierarchal regression analysis for risky family total with affection predicting negative word recall rate and heart rate average during stress task

	Negative Word Recall			**Heart Rate during Stress**		
Model 1(no covariates)	**β**	**S.E.**	***p***	**β**	**S.E.**	***p***
Risky Family Total with Warmth	.42	.03	<.01	-.28	.16	.03
Model 2(with covariates)						
Risky Family Total with Affection	.39	.03	<.01	-.18	.20	.23
Age	-.03	.07	.83	.02	.46	.92
Sex	-.17	.44	.19	.24	3.05	.07
Depression	-.26	.08	.07	-.27	.52	.07
Anxiety	.10	.08	.50	-.16	.40	.29
Childhood SES	.33	.15	.04	-.16	1.05	.31
Current SES	-.24	.15	.16	.14	1.24	.42
Pre-Heart Rate Avg.	-	-	-			

Table 5. Summary of hierarchal regression analysis for childhood emotional abuse predicting negative word recall rate and heart rate average during stress task

	Negative Word Recall			**Heart Rate during Stress**		
Model 1(no covariates)	**β**	**S.E.**	***p***	**β**	**S.E.**	***p***
Childhood Emotional Abuse	.41	.04	<.01	-.73	.19	<.01
Model 2(with covariates)						
Childhood Emotional Abuse	.43	.05	<.01	-.67	.24	<.01
Age	-.07	.07	.61	-.01	.34	.92
Sex	-.02	.45	.91	.03	2.39	.79
Depression	-.34	.08	.02	-.12	.40	.06
Anxiety	.08	.06	.55	-.14	.30	.27
Childhood SES	.23	.15	.15	-.04	.79	.74
Current SES	-.27	.17	.09	.03	.89	.84
Pre-task Heart Rate Avg.	-	-	-	.69	.08	<.01

Table 6. Summary of hierarchal regression analysis for childhood abuse predicted blood pressure reactivity during stress task

	Task DBP			**Task SBP**		
Model 1(no covariates)	**β**	**S.E.**	***p***	**β**	**S.E.**	***p***
Childhood Emotional Abuse	-.40	.23	<.01	-.11	.48	.38
Model 2(with covariates)						
Childhood Emotional Abuse	-.15	.23	.24	.11	.47	.40
Age	-.09	.38	.49	-.46	.71	<.01
Sex	-.16	2.64	.22	-.01	5.01	.43
Depression	-.07	.45	.61	.02	.85	.91
Anxiety	-.09	.33	.50	-.19	.63	.15
Childhood SES	-.08	.87	.63	-.17	1.65	.25
Current SES	-.08	.99.	.63	.31	1.88	.04
Pre DPB/SPB	.68	.16	<.01	.38	.24	<.01

LIST OF FIGURES

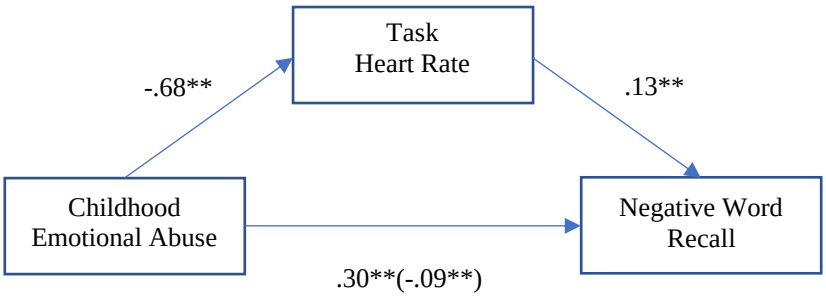

Figure 1. Standardized regression coefficients for the relationship between childhood emotional abuse and total negative word recall as mediated by task heart rate reactivity. The standardized regression coefficient between childhood emotional abuse and total negative word recall controlling for task heart rate reactivity, is in parentheses. *$p < .05$, **$p < .01$

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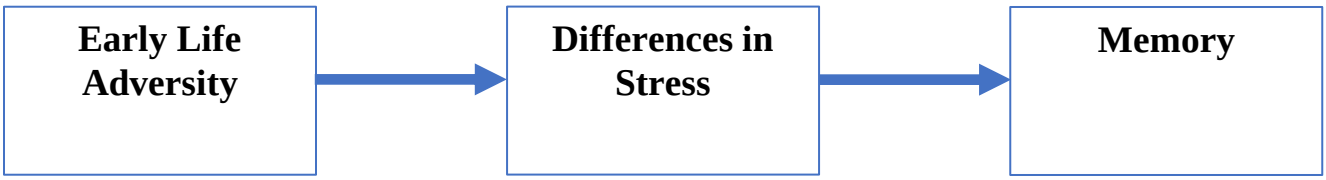
APPENDIX A

THEORETICAL MODEL


Early Life Adversity
Differences in Stress
Memory

Conceptual Mediation Models