ASSOCIATION BETWEEN CUMULATIVE LIFETIME STRESS, COGNITIVE FUNCTION, AND SLEEP: THE MODERATING EFFECTS OF RESILIENCE

By

Anya Savransky, BA

July 2023

Director of Thesis: D. Erik Everhart, PhD, ABPP

Major Department: Psychology

Stress is multidimensional and can be labeled as acute, chronic, or cumulative. *Cumulative lifetime stress* encompasses the exposure to stressors occurring over the course of the lifetime, instead of focusing on a specific time point. Not surprisingly, exposure to stress over the lifetime can pose significant negative effects on one's physical, mental, and emotional wellbeing. It has further been suggested that exposure to cumulative lifetime stress can have varying effects on cognitive function and sleep. While some studies have previously examined these relationships, more research is needed. As such, the goal of the current study was to examine the relationship between cumulative lifetime stress and cognitive function, the relationship between cumulative lifetime stress and sleep function, and the role of resilience as a moderator in the stress-cognitive function and stress-sleep relationships in a college student sample (n = 153). Linear regressions and moderation analyses were used to ascertain those relationships. Results demonstrated that cumulative lifetime stress did not predict variables of cognitive function, except for working memory, where greater cumulative lifetime stress was associated with better working memory task performance. In contrast, cumulative lifetime stress predicted sleep outcomes, with greater cumulative lifetime stress associated with more sleep quality

disturbances, and greater insomnia severity, and daytime sleepiness. In both analyses, resilience as a moderator did not influence those relationships. Findings from the current study underscore the detrimental effects of lifetime stress exposure on health outcomes, further adding to existing literature.

Keywords (#)*: cumulative lifetime stress, STRAIN, cognitive function, sleep function, resilience*

ASSOCIATION BETWEEN CUMULATIVE LIFETIME STRESS, COGNITIVE FUNCTION, AND SLEEP: THE MODERATING EFFECTS OF RESILIENCE

A Thesis

Presented to the Faculty of the Department of Psychology

East Carolina University

In Partial Fulfillment of the Requirement for the Degree

Master of Arts in Psychology

By

Anya Savransky, BA

July, 2023

Director of Thesis: D. Erik Everhart, PhD, ABPP
Degree Thesis Committee Members:
Christyn Dolbier, PhD
Matthew Whited, PhD

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I: INTRODUCTION

Stress is an integral part of life that is experienced throughout one's lifespan. The construct of stress is a complex phenomenon that can be categorized as acute, chronic, or cumulative. Acute stress refers to the experience of events that occur and subside suddenly, such as getting into a car accident or getting fired from a job, whereas chronic stress refers to adversities that persist over a prolonged period, such as living with a chronic health condition or experiencing ongoing financial instability (Shields & Slavich, 2017; Slavich & Shields, 2018). Cumulative stress exposure is conceptualized as the sum of both acute and chronic stressors occurring throughout one's lifetime (Lupien et al., 2009; Slavich & Shields, 2018). Depending on the type, frequency, duration, and intensity of a particular stressor, there may be a variation in neural, neuroendocrine, and inflammatory processes, which in turn may negatively impact one's health (e.g., by increasing the risk for heart disease, metabolic syndrome, anxiety, depression, sleep disruption, and/or cognitive impairment; Cazassa et al., 2020; Slavich & Shields, 2018).

The experience of both acute and chronic stress may have unique and variable effects on individuals' mental and physical health. For instance, although exposure to acute stress in healthy individuals may be adaptive and serve an essential function such as a "fight or flight" response, exposure to chronic stress can have damaging effects on one's health and wellbeing (Muscatell et al., 2009; Schneiderman et al., 2005; Slavich, 2016). Exposure to repeated stressors has been linked to structural, functional, and chemical changes in the brain, cardiovascular disease, metabolic syndrome, as well as several other psychiatric and medical conditions (Cohen et al., 1998; Linsky et al., 1985; Lucassen et al., 2014; Mariotti, 2015; Rohleder et al., 2019).

Given its importance in clinical health outcomes, to date, there are several existent questionnaires that aim to measure the experience of lifetime stress. However, these assessments

are often criticized in the literature for their relatively narrow focus on specific types of stressors (e.g., major life events, daily stressors) occurring at a specific time point in an individual's life (e.g., during childhood, in the past month). As such, these measures may not comprehensively capture the range of responses consistent with cumulative lifetime stress exposure (Shields & Slavich 2017). To address this gap in stress measurement, the Adult Stress and Adversity Inventory (STRAIN) was developed to assess stress accumulation over the lifespan (Shields & Slavich, 2017; Slavich & Shields, 2018). The STRAIN is an online, self-report instrument that assesses both acute and chronic stressors experienced over a lifetime including the assessment of severity, frequency, timing, and duration of those stressors (Slavich & Epel, 2010; Slavich & Shields, 2018).

Not surprisingly, prolonged exposure to stress exerts negative effects on cognitive function and sleep in both healthy individuals and those living with mental illness (Marin et al., 2011; McEwen & Sapolsky, 1995). Existing studies utilizing the STRAIN suggest that cumulative lifetime stress is associated with disrupted memory (Goldfarb et al., 2017) and poor sleep quality (Cazassa et al., 2020; Slavich & Shields, 2018). Although some aspects of cognition in relation to the STRAIN have already been examined (e.g., episodic memory, stimulus-response memory; Senft Miller et al., 2021; Shields et al., 2019), the effects of cumulative lifetime stress and other abilities contributing to cognitive function (e.g., processing speed, verbal memory, visual memory) need further exploration. Similarly, while sleep quality has already been investigated in relation to the STRAIN, other aspects of sleep impairment (e.g., subcomponents of sleep quality, insomnia severity, and daytime sleepiness) require further investigation (Cazassa et al., 2020; Slavich & Shields, 2018). Furthermore, to our knowledge, no studies have explored how resilience (i.e., the process of successfully adapting to stressors)

moderates these relationships between cumulative lifetime stress and cognitive function, and cumulative lifetime stress and sleep.

Given the relative lack of existing literature on the relationships between cumulative lifetime stress measured by the STRAIN, cognitive function, sleep, and resilience, the current study has several goals. The first goal is to examine the relationship between cumulative lifetime stress and four domains of cognitive function (i.e., working memory, processing speed, attention, verbal and visual episodic memory). The second goal is to explore the relationships between cumulative lifetime stress and three domains of sleep: sleep quality, insomnia severity, and daytime sleepiness. The final goal is to examine the moderating role of resilience in both of these relationships. To date, little is known about the influence of resilience on cumulative lifetime stress exposure and its effects on individuals' mental and physical health.

CHAPTER II: LITERATURE REVIEW

Part 1: Stress

Overview of Stress, Allostasis, and Allostatic Load

Stress is vital for survival and is experienced by humans across the lifespan. In the literature, the concept of stress is often associated with Hans Selye's descriptions of the "fight or flight" response to a threat (Selye, 1976). Earlier theories have attributed biological responses to stress as adaptive mechanisms leading to survival rather than categorizing them as chronic states. However, in the past 50 years, coinciding with the emergence of neuroscience, it has been suggested that stress response is not purely an “emergency system,” but instead an evolving mechanism, in which the brain and the body adapt and persevere in response to adversities. These biological adaptations are further thought to be highly dependent on the individual’s context, environment, and social support (McEwen & Akil, 2020).

Although acute responses to stress are essential for survival and functioning (e.g., "fight or flight" response; alertness), chronic and frequent stress occurrences can become toxic, ultimately harming both physical and mental health. Therefore, stress can further be differentiated and conceptualized as either "good” (eustress) or "bad" (distress; McEwen, 2019; Spencer-Segal & Akil, 2019). The body's adaptive responses that promote successful coping are referred to as *allostasis* (McEwen, 2000; McEwen, 2004). With repeated maladaptive responses to adversity, the body may begin experiencing "wear and tear," referred to as *allostatic load*, leading to negative physical and mental health consequences (McEwen 2000; McEwen 2004). The concepts of allostasis and allostatic load highlight the protective factors of multiple variables associated with adaptation and represent the damage that occurs if the same mediators are dysregulated (McEwen & Akil, 2020).

The brain plays a central role in the biological stress response as it distinguishes between threatening and non-threatening stimuli and subsequently determines its physiological and behavioral responses (McEwen, 1998). The brain is a vulnerable organ that can change and adapt to acute and chronic stressors by involving and directing several body systems, including cardiovascular, metabolic, immune, and neuroendocrine markers. As a result, these systems contribute to short- or long-term consequences of undergoing stressful experiences. The adaptive plasticity and resilience of the brain evolve over the course of the lifespan; however, during certain periods such as early childhood and adolescence, the brain may be particularly susceptible to stress. Although the brain can be resilient and adaptable, repeated stress may impair these abilities leading to damaging effects throughout the body (Lupien et al., 2007; McEwen, 2020).

Understanding the neurobiology of stress is important given its clinical implications and role in effective interventions and treatment planning. A complex set of mediators participate in allostasis, resulting in adaptive plasticity and activation of maladaptive processes. Acute or unexpected stimuli, either internal or environmental, trigger neural responses in the brain. Stimuli perceived as threatening, more specifically, activate a complex set of brain-body processes known as a stress response. A key stress response system is the hypothalamic-pituitary-adrenal (HPA) axis, which consists of a complex mechanism that captures positive and negative feedback between respective glands. The hormones of the HPA-system consist of corticotropin, corticotropin-releasing hormone, and cortisol, and the receptors of those hormones (Hosseinichimeh et al., 2015; Lupien et al., 2007; Lupien et al., 2009).

After an organism perceives a stimulus, the corticotropic-releasing hormone (CRH) is released from the hippocampus to the anterior pituitary, where CRF stimulates the secretion of

adrenocorticotropin (ACTH). In turn, ACTH stimulates the synthesis and release of glucocorticoids (GCs) from the adrenal cortex (Lupien et al., 2009). While a healthy response to an acute stressor requires a rapid activation of the synthesis and release of CGs, it also requires an appropriate and timely termination of those processes to avoid the overload of stress hormones in the body (McEwen, 1998). More specifically, after the system is activated, and eventually after the response to perceived stressors has lessened in intensity, feedback loops are activated within several systems, including the adrenal gland, hypothalamus, hippocampus, and frontal cortex, in order to shut down the HPA axis activity and return to homeostasis (Lupien et al., 2009). However, many factors can interfere with the appropriate activation and deactivation of the HPA axis, such as repeated stress exposure, which may further lead to adverse effects on cognitive, emotional, behavioral, and physical outcomes throughout the lifespan (Lupien et al., 2009).

Chronic exposure to stress has a lasting impact on the brain and the body due to the activation of the HPA axis and the subsequent release of CGs. It has been further suggested that stress affects different brain regions depending on the developmental stage (Lupien, 2009). For instance, exposure to stress during the prenatal period negatively affects several regions associated with the HPA axis, such as the frontal cortex, amygdala, and hippocampus. During childhood, the hippocampus may be the most affected by stress exposure due to increased CRH within the region. By adolescence, the development is focused on the frontal cortex, indicating a particular vulnerability to stress. In adulthood, brain regions are particularly influenced by the adverse effects of repeated stress (Lupien et al., 2009). Animal studies with rodents demonstrate variable effects of acute versus chronic stress on the brain and behavior. The effects of acute stressors depend on the level of glucocorticoids; it has been suggested that even small increases

in glucocorticoids result in improved hippocampus-dependent memory and learning functions as these hormones may serve an adaptive function by increasing attention and concertation during acute stressors (Lupien et al., 2009).

Chronic administration of glucocorticoids in rodents causes dendritic atrophy in hippocampal neurons, with these changes taking several weeks to develop. Chronic exposure to stress in rats can also cause hippocampal volume loss and has been related to the impairment of spatial learning and episodic memory (Kim et al., 2015). The findings from human studies follow similar trends as it has been suggested that there is an inverted-U-shaped relationship between glucocorticoid levels and cognitive performance, meaning that at moderate levels, glucocorticoids influence optimal performance on tasks. At the same time, too much release of glucocorticoids, or no release at all, leads to worsened or impaired performance (Raison & Miller, 2003). Taken together, these findings underscore the importance of studying the deletions effects of chronic stress accumulation across the lifespan.

Cumulative Lifetime Stress

Similar to the idea of allostatic load, the concept of cumulative stress is defined by repeated stress exposure, both acute and chronic, throughout the lifespan. Several theories have proposed that the accumulation of acute and chronic challenges across the lifespan can have a cumulative effect on health. More specifically, cumulative stress exposure has been associated with physical and mental health outcomes, including negative effects on memory and sleep function, increased inflammation, biological aging, and even premature death (Graham et al., 2006; Lupien et al., 2009; McEwen, 1998; Slavich & Shields, 2018). Additionally, as discussed above, research suggests that acute and chronic stress and the timing of those stressors (e.g.,

during prenatal development, adolescence, or adulthood) may exert variable effects on individuals' health and well-being (Lupien et al., 2009).

The idea of biological stress accumulation and the wear and tear on the body is not a new concept. However, the ability to measure the presence of both acute and chronic stressors over a lifetime remains relatively recent (Slavich, 2016). Therefore, it is essential to use measurement methods that comprehensively capture diverse effects of cumulative stress while accounting for a wealth of different types of stressors, the timing of the exposure to the stressors, and the severity. The instrument to measure cumulative lifetime stress and related studies will be discussed below.

Measuring Cumulative Stress

Although many studies have assessed the effects of different types of stressors on health, the brain, and behavior, very few studies have examined the effects of cumulative stress throughout a lifespan, often due to the challenge of assessing cumulative stress in a time- and cost-efficient manner (Slavich, 2016). Rather, most assessments are relatively narrow, focusing on specific types of stressors (e.g., major life events, daily stressors) at specific time points (e.g., childhood trauma, the perceived experience of stress in the past month), thus, they may not accurately capture cumulative lifetime stress exposure (Shields & Slavich, 2017).

To address these issues, the Stress and Adversity Inventory for Adults (Adult STRAIN; Slavich & Shields, 2018) was developed to access stress over the course of a lifetime. The STRAIN is an online, self-report instrument that assesses stressors that a person has experienced over a lifetime. Another unique feature of the STRAIN is that it also assesses the severity, frequency, timing, and duration of each of the stressors endorsed by the respondent. The inventory has shown good concurrent validity (r= .147 - .552), and test-retest reliability (rs = .904 –.919; Slavich & Shields, 2018). The STRAIN has also demonstrated validity in relation to several stress-related outcome measures and biological markers, including executive function,

memory, working memory, decision making, diurnal cortisol levels, biological responses to acute stress measured by cortisol and dehydroepiandrosterone (DHEA), aging, depression, and self-reported physical and mental health complaints (Cazassa et al., 2020; Goldfarb et al., 2017; Kim et al., 2021; Lam et al., 2019; McLoughlin et al., 2022; Senft Miller et al., 2021; Slavich & Shields, 2018).

Part 2: Cognitive Function

Cumulative Lifetime Stress and Cognitive Function

There is consensus within the literature that exposure to stress over a lifetime (e.g., environmental stressors) is associated with an increased risk of mild cognitive impairment (MCI), Alzheimer's disease (AD), and overall cognitive decline, likely due to excessive secretion of stress hormones such as GCs in patients with AD (Elgh et al., 2006; Sotiropoulos et al., 2011; Wilson et al., 2003; Wilson et al., 2008).

Animal and human studies have shown a relationship between increased glucocorticoids (GCs) and impairment in cognitive functioning. Neuroimaging studies have demonstrated the effects of stress, as indicated by GC secretion levels, affect the volume of the hippocampus, the region of the brain known to be implicated in learning and memory (i.e., cognitive function; Lupien et al., 2009; Vyas et al., 2016). Sotiropoulos and colleagues (2011) demonstrated that exposure to stress and GCs act cumulatively, suggesting that repeated or prolonged exposure to adverse events over a lifetime increases the likelihood of developing AD. Similarly, it has been reported that exposure to significant cumulative lifetime stressors (e.g., trauma) may lead to accelerated epigenetic aging (i.e., age-associated molecular changes) in African-Americans, leading to higher disease risk (Zannas et al., 2015) and the potential for changes in cognition. In the post-traumatic stress disorder (PTSD) literature, evidence suggests that the relationship

between PTSD and cognitive impairment is likely to be bidirectional. That is, having low intelligence quotient (IQ) scores before traumatic experiences are associated with an increased risk of developing PTSD (Brewin et al., 2000; Macklin et al., 1998; Parslow & Jorm, 2007; Sumner et al., 2017). Conversely, PTSD has been associated with the onset of cognitive impairment following exposure to traumatic events. For example, in a sample of male Vietnam War veterans, the severity of PTSD was negatively correlated with the performance on cognitive tasks, even after adjusting for estimated IQ before serving in the military and level of combat exposure during, suggesting that the exposure to trauma affected baseline cognitive functioning (Vasterning et al., 2002).

Sumner and colleagues (2016) investigated the relationship between lifetime traumatic stress exposure and domains of cognitive functioning, including psychomotor speed, attention, learning, and working memory. Their findings revealed that elevated symptoms of PTSD were associated with diminished scores on all the cognitive function measures of interest, and this relationship remained significant even after accounting for depression and medical conditions that could contribute to cognitive decline. Given that PTSD is associated with obesity, lack of physical activity, sleep disturbances, and dysregulation of the inflammatory, neuroendocrine, and HPA axis systems, it is not surprising that the experience of trauma may result in cognitive function impairment (Bartoli et al., 2015; Maher et al., 2016; Sherin et al., 2011). Additionally, the elevated sum of high-impact stressors (e.g., the experience of rape, attempted murder, and being kidnapped) has also been associated with impaired performance on memory-related tasks such as Logical Memory (Weschler Memory Scale-Revised; Wechsler, 1987), both immediate and delayed recall conditions, even when controlling for estimated IQ and psychopathology severity. This suggests that the degree of prior traumatic experience predicts performance on

tasks of immediate and delayed verbal memory (Nixon et al., 2004). The lifetime traumatic stress load has further been associated with poorer performance on tasks of executive functioning and complex reasoning; however, the relationship did not survive after covarying out for age, sex, and socioeconomic status (SES; Barzilay et al., 2018).

Given the chronic course of PTSD, the current study will utilize cognitive measures based on the cognitive profile of patients with PTSD (Chopra et al., 2014; Suliman et al., 2009). Current research suggests that individuals with PTSD exhibit impairment in multiple cognitive domains including attention, working memory, processing speed, episodic learning, and memory (Schuitevoerder et al., 2013; Scott et al., 2015). Considering the variable effects of stressful life experiences on several domains of cognition, the relationship between cumulative stress and different aspects of cognitive function (e.g., working memory, processing speed, and verbal and visual memory episodic memory) will be the focus of this current study, and therefore, will be reviewed in more detail below.

Working Memory. Working memory (WM) is a prefrontal cortex (PFC)-dependent ability that involves the temporary retention of memory and has a limited capacity for storing information (Baddeley, 2003; Markowitz et al., 2015). The function of WM is essential for managing ongoing activities, and assisting with decision-making abilities, reasoning, and comprehension given its facilitation in comprehension, planning, reasoning, and problem-solving (Cowan, 2014). Although the literature remains relatively sparse, several studies have suggested a relationship between cumulative stress exposure and WM impairments. Exposure to high levels of chronic stress has been linked to the disruption of PFC functions such as WM and attention (Arnsten, 2011; Lupien et al., 2009). These consequences are thought to result from the interplay between perceived stress and the chronic activation of the neurobiological system responsive to

stress regulation (Liston et al., 2009). Findings from behavioral research have demonstrated significant impairment in WM function in individuals under chronic stressors, such as caregiver stress (Evans & Schamberg, 2009; Mackenzie et al., 2009). In their longitudinal study, Evans and Schamberg (2009) examined the relationship between prolonged childhood poverty, stress exposure as indexed by allostatic load, and WM and found worsened WM performance in young adulthood. Notably, longer periods of childhood poverty were associated with higher levels of allostatic load throughout childhood and were subsequently associated with a more significant impairment in WM as the children transitioned to young adulthood.

Processing Speed. Processing speed is a cognitive ability defined as the time it takes to process and react to information received from the environment (e.g., visual and verbal information; Horning & David, 2012). Individuals with a slower processing speed may experience difficulties within various domains including learning and reading. To date, the literature on cumulative stress and processing speed remains limited; however, it has been suggested that prolonged exposure to stress is associated with impaired performance on processing speed tasks. For example, a study examining the effects of trauma-induced stress on information processing speed has found that individuals diagnosed with PTSD as a result of childhood sexual abuse performed slower on a modified emotional Stroop task when responding to sexual/victimization words (de Leeuw et al., 2015; Field et al., 2001) Another study reported that reduced perceptual speed performance had been associated with an increased number of stressful life events in a sample of older adults. Interestingly, the same study has found that an illness of a loved one such as a parent or a friend was associated with improved perceptual speed performance, thought to be attributed to increased motivation necessary to maintain one's health through exercise and activity to help care for a loved one (Rosnick et al., 2007).

Verbal and Visual Episodic Memory. Episodic memory is a type of memory that involves conscious recollection of past events (e.g., what was eaten for breakfast, where the car is parked). It can be categorized as verbal (i.e., remembering a list of words) or visual (i.e., remembering a figure; Roediger et al., 2016). Animal and human studies have shown impairment in both forms of episodic memory after prolonged stress exposure (McComick & Green, 2013; Shields, 2017). More specifically, one study demonstrated that participants who reported more significant stressful life events exhibited a reduction in word recall (i.e., verbal episodic memory) as compared to those who reported less (Lupien et al., 1997).

Allostatic Load and Cognitive Function

In regard to the studies incorporating allostatic load, results have been mixed. A meta-analysis examining the relationship between allostatic load and cognitive function revealed that allostatic load was associated with diminished executive function, although the effect size was relatively small ($r = 0.20$). In terms of overall memory function, several studies reported a negative correlation between allostatic load and memory, with two studies reporting null findings (D'Amico et al., 2020). In reference to WM, one study reported a significant negative correlation between allostatic load and WM, but not between allostatic load and perceptual and visuomotor speed (Kobrosly et al., 2012). Ottino-Gonzalez et al. (2019) found a significant relationship between allostatic load and WM, it was limited to participants who were overweight. The lack of significant findings may be confounded by the average age of the participants, as they ranged between the ages of 21-40 years old. Different findings utilizing the allostatic load construct must be interpreted with caution given the variability of allostatic load measurement strategies and concept operationalization. Although the results are mixed, there are consistent findings suggesting the negative effects of elevated cumulative stress (measured by allostatic load) and

cognitive function. Future studies examining the association between cumulative lifetime stress and different components of cognitive function are warranted.

STRAIN and Cognitive Function Studies. To date, only six studies have examined the relationship between lifetime stress exposure using the STRAIN and measures of cognitive function. Slavich and Shields (2018) conducted the initial validation study of the STRAIN, assessing the association between lifetime stress and a measure of executive function. Their results revealed the number of total lifetime stressors was a significant predictor of poorer executive function outcomes. In contrast, another initial validation study of the STRAIN administered in the Brazilian Portuguese language did not yield significant results in the relationship between the STRAIN and the same measure of executive functioning (Cazassa et al., 2020). Moreover, Senft Miller and colleagues (2021) examined the effects of acute and chronic stressors occurring throughout a lifespan on cognition, as well as psychiatric symptomology in a sample of older adult women. In this study, objective cognition was measured using the Letter Sequencing Task (WM measure from the Weschler Memory Scale; Wechsler, 1955), and the Buschke Selective Reminding Test (episodic memory measure; Buschke & Fuld, 1974). Contradictory to the hypothesis, the number of total lifetime stressors and their severity was not associated with immediate and delayed recall or WM performance. Meanwhile, more lifetime stress exposure was associated with increased symptoms of subjective memory function, as well as elevated rates of self-reported anxiety and depression. Overall, increased lifetime stress and its severity were associated with subjective, but not objective, memory decline. To explain this finding, the authors state that in normal aging, both objective and subjective decline appear to occur simultaneously (Senft Miller et al., 2021). However, in abnormal aging, subjective reports of cognitive decline may precede objective measures of decline given perceived subjective

change in cognition is considered to be one of the earliest signs of a neurocognitive disorder, such as in AD and MCI (Gatchel et al., 2020; Studart & Nitrini, 2016). Finally, a study conducted by Goldfarb et al. (2020) found that lower levels of lifetime stress accumulation were associated with the decreased use of stimulus-response association. Specifically, low levels of stress were associated with diminished memory for stimulus-response relative to those with moderate stress levels. These results indicate that some stress exposure may have adaptive mechanisms for improving memory function.

In summary, while cumulative lifetime stress has been associated with impairment in cognitive function, including reduced WM, episodic memory, and processing speed, other studies have reported mixed or non-significant findings. Given the mixed and limited literature on cumulative stress and cognition, more research is needed to elucidate these findings. As such, the current study will explore the relationships between cumulative lifetime stress and WM, processing speed, verbal episodic memory, and visual episodic memory.

Part 3: Sleep

Overview of Sleep and Health

Sleep is a neurobiological process that is essential for health and survival. Sleep plays a critical role in different types of physiological functioning, such as the cardiovascular, metabolic, hormone, and immune systems (Medic et al., 2017). A wealth of literature suggests that adults require an average of seven hours of sleep per night. Healthy sleep is characterized by sufficient duration, quality, and regularity, as well as the absence of sleep disorders that may interfere with optimal sleep quality (Watson et al., 2015). Sleep abnormalities can be further delineated into separate domains; the current edition of the International Classification of Sleep Disorders system identified seven major categories of sleep disorders: insomnia, hypersomnolence (e.g.,

daytime sleepiness, narcolepsy), sleep-breathing disorders (e.g., sleep apnea), circadian rhythm sleep-wake disorders, parasomnias (e.g., sleep walking, sleep terrors), and sleep-related movement disorders (e.g., restless leg syndrome; Sateia, 2014).

In addition to the presence of sleep disorders, numerous other factors can contribute to disruptions in sleep including stress, lifestyle habits (e.g., being a college student, engaging in shift work), and certain medical conditions. Both sleep deprivation and sleep excess have been linked to cardiovascular disease and Type II diabetes, which can be attributed to atherosclerosis and worsening glucose metabolism, respectively (Shan et al., 2015). Inadequate sleep has been found to have additional detrimental short- and long-term health consequences. For example, sleep deprivation (e.g., underpinning chronic insomnia) has been associated with prolonged activation of the HPA-axis, resulting in increased secretion of epinephrine, norepinephrine, cortisol, and ACTH, hormones that play a role in both stress and the 24-hour sleep-wake cycle (Tiermeir et al., 2002; Vgontzas et al., 2001).

In healthy adults, the short-term consequences of fragmented and interrupted sleep have resulted in increased stress responsivity associated with the activation of the sympathetic nervous system. The consequential activation is believed to be related to the premature disruption of sleep. Short-term consequences of sleep disruption have also been associated with increased levels of mental health concerns, somatic pain, emotional distress, memory deficits, and reduced quality of life (Medic et al., 2017).

In summary, sleep is a complex concept to study in relation to health outcomes as it consists of numerous constructs (e.g., sleep latency, duration, and disturbances) that can lead to myriad sleep-related conditions (Buysse, 2014). In the current study, the operational definition of sleep quality includes objective aspects of sleep such as sleep duration and sleep latency, as well

as more subjective aspects of sleep such as the extent of feeling restful. To assist with the focus of this current literature review, the current study will examine sleep quality specifically, as well as related consequences of poor sleep quality such as insomnia and daytime sleepiness.

Sleep in a College Student Population

Given that the proposed study will consist of a college student sample, it is important to note the prevalence of sleep disturbance in this population. College is a unique developmental period in that students are often met with increased demands and new challenges such as moving away from home, forming new relationships with peers, adjusting to demanding and variable college schedules, and for some, increased consumption of alcohol and drugs (Taylor et al., 2013). As such, it is not surprising that these young adults may experience problems with sleep. Although the recommended sleep duration for young adults is between seven and nine hours, one survey conducted with undergraduate students in the United States suggested that approximately 25% of students receive less than 6.5 hours of sleep per night. Furthermore, up to 60% of college students self-report poor sleep quality as assessed by the Pittsburg Sleep Quality Index (PSQI; Buysse et al., 1989), and approximately 27% of students meet the criteria for at least one sleep disorder (Gaultney, 2010; Hirshkowitz et al., 2015; Lund et al., 2010). Other literature supports this finding, suggesting that about 31% of college students report feeling tired in the morning and, as a result, report reduced overall daytime functioning (Alapin et al., 2000; Buboltz et al., 2001). Research also indicates that the lack of sleep is widely associated with learning problems, particularly for tasks that rely on procedural and declarative memory systems. This, in turn, contributes to diminished cognitive function and, subsequently, may result in lower grades in coursework (Curcio et al., 2006). In summary, undergraduate college students appear to exhibit unique sleep characteristics as indicated by their decreased overall sleep quality, sleep duration,

and increased rate of sleep disturbances as compared to the general public. Given the significant implications of sleep on cognitive and academic performance, the use of a college student sample adds to the particular importance of this study.

Overview of Stress and Sleep

Stress is a well-known risk factor for sleep disturbances (Han et al., 2012). The literature indicates a complex bidirectional relationship between stress and sleep in animal and human studies, suggesting that stress-related factors negatively impact the sleep-wake cycle. The level of this impact is likely to be dependent on several factors including the type of stressor (i.e., acute versus chronic), the duration of the stressor, and other individual differences (Lo Martire et al., 2020). Research suggests that sleep-related disorders can also significantly impact one's stress responses, consequently influencing perceived quality of life. Even after several days of sleep disruption or deprivation (e.g., due to acute stress), individuals may experience increases in appetite, blood pressure, and blood glucose levels, as well as increases in pro-inflammatory cytokines, an indicator of the body's inflammatory response. Additionally, sleep deprivation has been associated with increased sympathetic nervous system response and elevated cortisol levels. Studies examining the long-term effects of stress have reported that adverse experiences in childhood can greatly influence sleep quality in adulthood (Palagini et al., 2014).

The experience of stressful events contributes to numerous bodily responses, including hormonal, molecular, and behavioral changes, in effort to help one's body cope with environmental challenges and maintain homeostasis. Considering the influence of the HPA-axis in managing and maintaining the stress response, various HPA-axis variables may also influence sleep function. Importantly, sleep deprivation can itself be categorized as a stressor, leading to negative consequences for both the brain and the body (Medic et al., 2017). Considering that

stress and sleep are linked to the function of the HPA axis, this relationship warrants careful consideration (Steiger, 2003; Van Reeth et al., 2000).

Cumulative Stress and Sleep

In the past several decades, there has been a significant shift in emphasizing the effects of cumulative stress on health and disease. Studies on sleep often encompass several dimensions of the construct, including sleep quality, duration, continuity, and depth (Hall et al., 2015) measured both objectively (e.g., actigraphy and polysomnography data) and subjectively (i.e., via self-report; Morin et al., 2003; Kim & Dimsdale, 2007). Stress, on the other hand, is often measured with a focus on a specific type of stressor or stress duration (e.g., focusing on either acute or chronic stress). Given the variability of both sleep and stress concept operationalization and the measures used to ascertain related data, the relationship between lifetime stress and sleep often yields mixed results.

Unsurprisingly, prolonged exposure to stress significantly impacts sleep architecture in humans. For instance, individuals undergoing a marital separation, working rotating shift jobs, and those without adequate social support in the workplace are particularly prone to sleep disruptions (e.g., waking up in the middle of the night; Lo Martire et al., 2020). Changes are also seen in allostatic load. For instance, prolonged sleep disruption can cause high blood pressure, increased oxidative stress, increased cortisol and insulin levels, and diminished parasympathetic nervous system response. Even several days of sleep deprivation can lead to measurable physiological changes within the body (e.g., blood pressure, pro-inflammatory cytokines), as stated above (McEwen & Karatsoreos, 2015). Chronic disruption of circadian rhythms can increase the risk of developing cardiovascular disease and metabolic syndromes such as diabetes and obesity (Tobaldini et al., 2017).

Chronic and acute stressors have been shown to contribute to reduced sleep quality (Bruce et al., 2021; Hanson & Chen, 2010). Hall et al. (2015) examined the relationship between lifetime stress, objective and subjective sleep quality, and insomnia among middle-aged women. Their results indicated that women with higher levels of lifetime stress exposure were more likely to report lower sleep quality, experience anxiety, and show increased wakefulness during sleep as measured by polysomnography compared to women with lower reports of lifetime stress. Another study revealed similar findings, demonstrating that increased levels of PTSD symptomology have been associated with higher scores of reported sleep disturbance, supporting the notion that greater lifetime exposure to traumatic events leads to more sleep problems (Follette et al., 1996). Finally, Hux et al. (2017) found that higher levels of allostatic load in early pregnancy are associated with poor sleep quality as indexed by the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989), further suggesting a link between cumulative stress exposure and diminished sleep quality.

Given the links between stress and cognitive function and stress and sleep, it is unsurprising that the literature demonstrates a relationship between stress and cognitive function. While evidence suggests sleep difficulties associated with acute stress exposure can have short-term adaptive functions, exposure to prolonged chronic stressors – and, therefore, prolonged sleep difficulties - can lead to negative health outcomes, particularly as it relates to everyday function (Juster et al., 2010). Specifically, adequate amounts of sleep are associated with improved physical and cognitive functioning and performance, while sleep deprivation is linked to worsened performance in daily activities. Studies indicate that those who sleep less than six hours per night on average have a significant reduction in tasks of memory and other cognitive performance (Karatsoreos et al., 2011; Kwon et al., 2015; Tsapanou et al., 2017). Shorter sleep

duration has been associated with a greater incidence of major neurocognitive disorders (e.g., AD) in older adulthood, which may act in an U-shaped association, where greater risk is associated with reduced and increased sleep duration, and lower risk being in individuals who sleep approximately seven hours per night (Chen et al., 2016).

Given the known negative effects of prolonged stress exposure on sleep and their interactive consequences on health, it is essential to continue investigating the relationship between cumulative lifetime stress exposure using comprehensive stress measures such as the STRAIN and various sleep domains.

STRAIN and Sleep Studies. To date, only three studies have explored the relationship between lifetime STRAIN and sleep quality as assessed by the PSQI (see Byrne et al., 2021; Cazassa et al., 2020; Slavich & Shields, 2018). Results from these studies suggest that elevated lifetime stressor count is associated with worsened sleep quality over the previous month, and greater lifetime stressor severity is associated with poorer sleep quality. Moreover, while existing studies have examined the association between the STRAIN and sleep quality measured by the PSQI, we are unaware of studies that have investigated the association between cumulative lifetime stress and the subcomponents of the PSQI, including the duration of sleep, sleep disturbance, sleep latency, daytime dysfunction due to sleepiness and sleep efficiency. Additionally, no existing studies have examined the relationship between cumulative lifetime stress as measured by the STRAIN and insomnia and daytime sleepiness. Therefore, a central goal of this study is to further explore the relationship between cumulative lifetime stress and other sleep-related domains.

Part 4: Resilience

Resilience Definition

Different approaches to studying resilience have led to inconsistencies within the literature regarding its definition and method of assessment. Broadly, “resilience” is a term often used to explain positive adaptation to stressful events (Bonanno, 2012). The American Psychological Association (APA; 2014) defines it as "the process of adapting well in the face of adversity, trauma, tragedy, threats or even significant sources of stress" (para. #4). While this definition encompasses core components of the concept, it does not capture the complexity of resilience phenomena. Decades of research have shown that resilience is impacted by individual genetic, biological, psychological, social, and cultural factors. These factors, experienced either separately or in combination, determine how individuals respond to stressful events or experiences (Southwick et al., 2014). The resilience model developed by Richardson and colleagues suggest that individuals' bodies and minds adapt to life circumstances, which they label “biopsychospiritual homeostasis” (Richardson et al., 1990; Richardson, 2002). In the model, the unavoidability of experiencing internal and external stressors is highlighted, along with the notion that an individual’s ability to cope with inevitable life stressors is likely influenced by successful or unsuccessful adaptations to past adversities. In instances where adaptations or protective factors are ineffective, biopsychospiritual homeostasis may become dysregulated. The disruption of this homeostatic process can result in a range of outcomes: an opportunity for increased resilience, a return to a baseline level of homeostasis to get past or beyond the disruption, achievement of recovery but with some loss (resulting in a lower level of baseline homeostasis), or establishment of a dysfunctional state by use of maladaptive strategies to cope with stress (Richardson et al., 1990; Richardson, 2002).

Resilience can be viewed as a trait, a process, or an outcome; it can also be categorized dichotomously in that it is either present or absent. While it is helpful to consider the different conceptualizations of resilience, it likely exists on a continuum where it presents in various strengths across multiple domains of life (Southwick et al., 2014). For example, individuals who adapt well to personal changes such as a romantic break-up or a divorce may not demonstrate the same adaptability as it relates to workplace adversity. Research suggests characteristics of resilient people include viewing stressors as a challenge and/or opportunity, possessing high levels of self-efficacy, having patience, being tolerant of negative affect, having a history of past successes, and having religious faith (Kobasa, 1979; Lyons, 1991; Rutter 1985).

Although the concept of resilience could play a crucial role in explaining resistance to the negative influences of adversity over a lifetime, different approaches to studying resilience across studies have contributed to inconsistencies in operational definitions, prevalence, and measurement of the construct (Luthar et al., 2000; Windle et al., 2011). One review reported that individuals' resiliency status across publications ranged from 25% to 85%, highlighting the many different ways in which researchers conceptualize resiliency (Vanderbilt-Adriance & Shaw, 2008). Considering the variability in assessing the broad concept of resiliency, the operationalization of resiliency in the present study is of particular importance.

To operationalize resilience for the current study, it was decided to select a definition that has been composed based on the literature review of existing resilience-related studies. More specifically, Windle and colleagues (2011) proposed an operational definition of resilience derived from examining 270 studies that will be used for the current project: "Resilience is the process of negotiating, managing, and adapting to significant sources of stress or trauma. Assets and resources within the individual, their life, and environment facilitate this capacity for

adaptation and bouncing back in the face of adversity. Across the life course, the experience of resilience will vary" (p. 163).

Cumulative Lifetime Stress and Resilience

The ability to recover from adverse or stressful events is characteristic of resilient individuals. Although resilient individuals may experience negative emotions as it relates to the event, they are likely to use adaptive coping strategies. When maladaptive coping strategies are used and/or when one is unable to recover from negative events, it may result in the prolonged activation of several neurobiological systems (Southwick et al., 2014).

Overall, the relationship between resilience and prolonged stressors has been examined in the literature, however, the examination of resiliency as it relates to cognitive functioning and cumulative lifetime stress is particularly sparse, and therefore requires further attention. Although most research focuses on undesirable aspects of stress, some studies have demonstrated that moderate levels of stress may indeed improve psychological resilience (Seery & Holman, 2010). For example, studies using young monkeys undergoing stressors showed greater resilience to future stressors compared to monkeys who were never exposed to any stressors (Parker et al., 2006). Moderate perceived threat predicts improved self-reported psychological benefits in veterans, compared to decreased or increased threat, suggesting an U-shaped relationship (Fontana & Rosenheck, 1998). Similarly, children who experienced moderate levels of early life adversity had a lower cortisol response during a laboratory stress experiment compared to children with severe early life adversity (Gunnar et al., 2009).

Cognitive Function and Resilience

In recent years, several studies have examined the role resilience plays in mitigating cognitive function. For instance, one study reported that individuals identified as having less

resilience had more amyloid-related cognitive impairment (Wolf et al., 2019). Similarly, Wingo and colleagues (2010) reported that resilient individuals with a history of trauma performed better on non-verbal memory tasks compared to their peers who were classified as "non-resilient." Castor and Massioui (2018) reported that higher levels of self-reported resilience were associated with better performance on cognitive tasks in individuals with brain damage, further suggesting a positive relationship between resiliency and cognitive functioning (Connor & Davidson, 2003). Although the literature on resilience and cognitive function remains limited, existing evidence underscores the importance of further understanding the role resilience plays in the relationship between stress and cognitive function.

Sleep and Resilience

Although it is well-recognized that sleep is essential for good health, limited studies have investigated the link between sleep and psychological resiliency. Some studies suggest that good sleep quality is essential for achieving resilience (Cai et al., 2021; Palagini et al., 2018). In the military literature, sleep is conceptualized as an essential requirement for developing resilience, similar to Maslow's Hierarchy of Needs, which states that sleep is the basic requirement for resilience similar to other survival essentials such as food, water, and oxygen (Maslow, 1943; Young-McCaughan et al., 2018). The relationship between resilience and sleep is likely to be bidirectional, in that increased resilience and better coping skills may contribute to better objective and subjective reports of sleep, while poorer sleep may contribute to a lack of coping skills use and consequently lead to reduced resilience, especially if repeatedly encountered over time. It has been suggested that healthy sleep may enhance resilience to both existing and future stressors (Gargiulo et al., 2021). Given that sleep and resilience appear to have a significant

overlap, it is imperative to examine resilience as a potential moderating variable in the interaction between stress and sleep.

The Present Study

Considering the novelty of the STRAIN measure and its comprehensive method of measuring lifetime accumulation of stress, the goal of the current study is to examine the relationships between cumulative stress measured by the STRAIN and cognitive function and sleep outcomes in a sample of college students. To our knowledge, this is the first study to explore the role resilience plays in the relationship between cumulative lifetime stress, as assessed by the STRAIN, and cognitive function and sleep.

Definitions of Terms

Listed below are the definitions of the variables included in this study:

Cumulative lifetime stress refers to the accumulation of prolonged and repeated stress exposure, both acute and chronic throughout the lifespan.

Acute stress refers to the experience of events that occur and subside suddenly, such as getting into a car accident or getting fired from a job.

Chronic stress refers to adversities that persist over a prolonged period of time, such as living with a chronic health condition or not having a stable source of income.

Resilience, as described above, will be defined as "the process of negotiating, managing, and adapting to significant stress or trauma sources. Assets and resources within the individual, their life, and environment facilitate this capacity for adaptation and 'bouncing back' in the face of adversity. Across the life course, the experience of resilience will vary" (Windle, 2011; p. 163).

The term *sleep function* refers to subjective sleep quality. In this study, insomnia severity and daytime sleepiness will also be assessed.

The term *cognitive function* will encompass the measures of WM, processing speed, and verbal and visual episodic memory.

Covariate Variables

Several variables have been selected to ensure that the relationships between cumulative lifetime stress and cognitive function, as well as cumulative lifetime stress and sleep, are not confounded by extraneous variables. The variables selected include participants' sex, parents' education, quantity and frequency of alcohol and stimulant intake and given the historical context in which this study is being conducted, the extent of their ongoing worry about the coronavirus (i.e., COVID-19) pandemic. These variables were selected based on their known association with stress, cognitive function, and sleep-related variables (Anders et al., 2014; Anthenelli & Grandison, 2012; Bangasser et al., 2018; Bale & Epperson, 2015; Baum et al., 1999; Gutwinski et al., 2018; Handa et al., 2002; He et al., 2019; Keyes et al., 2012; Moore et al., 2002; Yang et al., 2021).

Proposed Study Hypotheses

Hypothesis one: It was hypothesized that cumulative lifetime stress would relate to scores on cognitive function measures (i.e., WM, processing speed, and verbal and visual episodic memory)**.**

a. It was hypothesized that cumulative stress would be negatively associated with the global cognitive function score. The global cognitive function was assessed by averaging the scores of the four cognitive function variables (i.e., WM, processing speed, and verbal and verbal episodic memory)**.**

b. It was hypothesized that cumulative stress would be negatively associated with WM scores.
c. It was hypothesized that cumulative stress would be negatively associated with processing speed scores.
d. It was hypothesized that cumulative stress would be negatively associated with verbal and episodic memory scores.

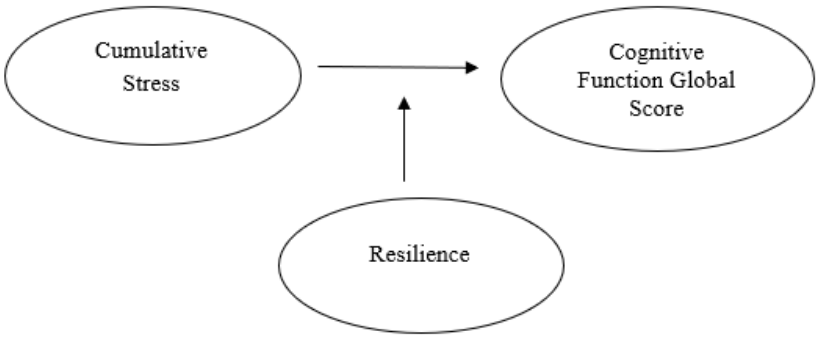
Hypothesis two: It was hypothesized that cumulative stress would relate to sleep-related outcomes.

a. It was hypothesized that cumulative stress would be positively associated with sleep quality disturbances.
b. It was hypothesized that cumulative stress would be positively associated with insomnia severity.
c. It was hypothesized that cumulative stress would be positively associated daytime sleepiness.

Hypothesis three: It was hypothesized that resilience would moderate the cumulative lifetime stress and cognitive function relationship. More specifically, it was assumed that higher levels of resilience would weaken the negative relationship between cumulative lifetime stress and cognitive function global score (See Figure 1).

Figure 1

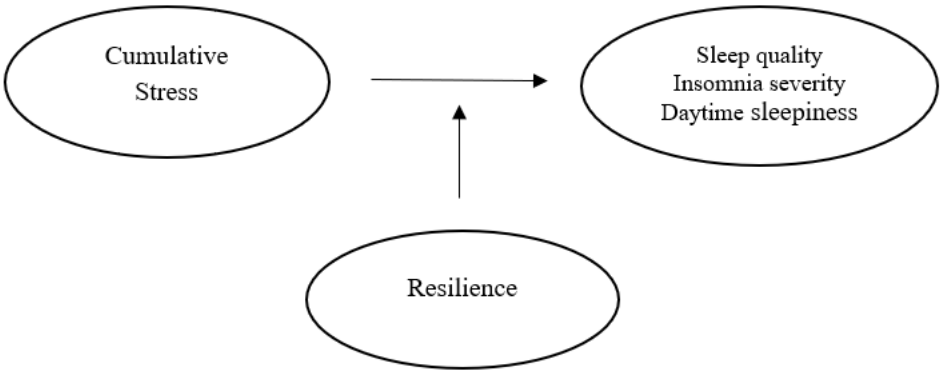
Resilience as a Moderator in Stress-Cognitive Function Relationship



Hypothesis four: It was hypothesized that resilience would moderate the cumulative stress and sleep relationships. More specifically, it was hypothesized that higher levels of resilience would weaken the positive relationship between cumulative lifetime stress and sleep quality disturbance, insomnia severity and daytime sleepiness (See Figure 2).

Figure 2

Resilience as a Moderator in Stress-Sleep Relationship



CHAPTER III: RESEARCH METHODS

Participants and Procedures

A statistical power analysis indicated that a sample of 150 participants would be sufficient to detect medium- sized effects (see additional details regarding power analysis below). All testing was completed in the Department of Psychology at East Carolina University in Greenville, North Carolina. Participants were recruited from the university's psychology participant pool using the SONA online system and included undergraduate students primarily enrolled in introductory psychology courses. The exclusion criteria included being under the age of 18 years and over the age of 25 years, reported history of recent head trauma, and not having proficiency in the English language. All study data were collected through Qualtrics and the software provided by the STRAIN and TestMyBrain (TMB) developers.

Upon signing up for a study slot online, each participant completed an approximately two-hour-long study visit in a private laboratory setting led by the principal investigator (PI) or a trained research assistant. After completing the informed consent form, participants were directed to Qualtrics where they began completing a series of surveys. The first survey gathered personal health and demographic-related information (e.g., age, sex, gender, race, ethnicity, parental education, and significant medical history). Next, the participants were asked to complete three sleep-related questionnaires followed by the resilience questionnaire (see Appendix A). Participants were then directed to complete the STRAIN questionnaire followed by the link to the TestMyBrain (TMB) website to complete cognition-related measures (See Measures and Materials below). Survey navigation was overseen by the study PI or trained research personnel. Additional information about the specific measures used is provided below.

Power Analysis

An *a priori* statistical power analysis was performed using G*Power v. 3.1.9.2 to assess the necessary sample size needed for linear regression. This study was powered to answer one of the main study hypotheses (i.e., hypothesis 2). To date, two studies have reported the effect sizes for the relationship between cumulative lifetime stress (measured by the STRAIN) and sleep quality (measured by PSQI); medium effect sizes were reported (Cazassa et al., 2020; Slavich & Shields, 2017). More specifically, Cazassa et al. (2020) reported a medium-size correlation coefficient of (r= .402, N= 330), and Slavich and Shields (2017) reported a medium-size correlation coefficient of (r= .493, N= 205). Given that previous studies indicated a relationship of moderate magnitude between cumulative stress and sleep, an *a priori* power analysis was conducted using the medium effect size (f^2= .15). The power analysis included one primary predictor (i.e., sleep quality) and four potential covariates (e.g., demographic variables). Finally, using the power of .80, a Type I error rate of .05, the power analysis indicated that 92 participants would be needed to detect a medium effect size in the relationship between cumulative lifetime stress and sleep while controlling for potential covariates. In regard to moderation analysis, another *a priori* power analysis was conducted to determine the sample size needed to achieve the medium effect size (f^2= .15). This analysis included three predictor variables (i.e., sleep quality, resilience, and stressXresilience, which is a computed moderation variable). Using the power of .80, a Type I error rate of .05, the power analysis indicated that 77 participants would be needed to detect a medium effect size to examine the role resilience plays in the cumulative sleep/sleep relationship. Overall, given the potential attrition, issues with validity, and incompletions, the current goal was to double that of the power analysis and recruit approximately 150 participants.

Validity Checks

Embedded within the Qualtrics survey between questionnaires were two validity items to ensure participants were paying attention and responding appropriately. The two items were "Are you an alien from outer space?" and "What is 5 x 3?" The participants were excluded if they answered less than 50% of the validity questions accurately.

Measures and Questionnaires

Demographics Questions

Participant demographic (e.g., age, sex), health history (e.g., presence of physical and/or mental health conditions; question about one's overall physical health and wellbeing), and lifestyle (e.g., past and present alcohol use) information was obtained through items developed specifically for this study (See Appendix A). See Table 8 for the summary of all measures.

Cumulative Stress Measure

The *Stress and Adversity Inventory (STRAIN)* is an online self-report interviewing assessment that provides a reliable cumulative stress measure over the course of the lifespan. It takes, on average, 18-25 minutes to complete (Slavich & Epel, 2010; Slavich & Shields, 2018). To administer the STRAIN, participants are presented with items, one at a time, on a computer screen and are asked to select a response prior to moving on to the next item. For every endorsed stressor, participants are asked follow-up questions pertaining to the specific stressor's frequency, severity, timing, and duration. If every item is endorsed, approximately 220 items can be administered throughout the assessment (Slavich & Shields., 2018).

The STRAIN assesses a total of 55 major lifetime stressors. Out of all the stressors, 26 questions inquire about exposure to acute stressors (e.g., divorce, death of a loved one, and job loss), and 29 questions inquire about chronic stressors (e.g., ongoing relationship, work, and

financial problems). Given that each item is followed up with additional information (e.g., severity, frequency, timing, duration) for each stressor endorsed, it allows for the data to be separated into time-limited categories such as prenatal stress, early adversity, adulthood adversity, and recent (i.e., past six months) stressors (Slavich & Shields, 2018). Of note, in this study, only the total count of stressors was examined.

The STRAIN has shown good concurrent validity (*r*= .147 - .552) and test-retest reliability (*rs* = .904 –.919; Slavich & Shields, 2018). Additionally, the STRAIN has demonstrated validity in relation to several stress-related outcome measures and biological markers, including executive function, memory, WM, decision making, diurnal cortisol levels, biological responses to acute stress measured by cortisol and DHEA, aging, depression, and self-reported physical and mental health complaints (Cazassa et al., 2020; Goldfarb et al., 2017; Kim et al., 2021; Lam et al., 2019; McLoughlin et al., 2022; Senft Miller et al., 2021; Slavich & Shields, 2018).

Cognitive Assessment Measures

The Test My Brain (TMB) Toolkit (TestMyBrain.org) was used to assess several domains of cognitive functioning. The online toolkit offers access to a set of traditional neuropsychological assessments delivered via the web. The TMB toolkit was selected for the current study for its custom set of neuropsychological batteries, which provide the option to retrieve raw data, along with the summary and normalization of data based on age, gender, and years of education. To maintain participant confidentiality, the data is provided for each participant using a unique ID number.

The overarching goal of the TMB organization is to provide a stable and secure platform for developing web-based cognitive assessment protocols for commonly used tests. Since 2008, the

TMB organization has conducted studies with over 2.5 million individuals worldwide to facilitate the development, validation, and normalization of cognitive measures TMB framework has received support and collaborative input from many reputable organizations including the National Institute of Mental Health, the National Institute on Aging, the National Institute of Diabetes and Digestive and Kidney Diseases, and the National Cancer Institute (The Many Brains Project, n.d.).The TMB assessments used in the current study are described below:

1. **TMB Digit Span (Forward/Backward).** This test, adapted from Wechsler Adult Intelligence Scales (WAIS; Wechsler, 2008), measures short-term memory, attention, and WM. Numbers are presented to participants visually on the monitor, as opposed to verbally. Both conditions begin with two practice trials, in which participants are given feedback on the accuracy of their performance. The trials are presented up to 11 digits or until the participant gets both sequences incorrect on a given trial. In the Forward condition, respondents are shown a sequence of numbers on the computer screen (one-second per number). In the Backward condition, the participants are asked to recall the numbers in reverse order of that presented on the screen. After being shown the sequence, participants are asked the type in the sequence that they saw. In terms of validity, both TMB Forward Digit Span and Backward Digit Span have shown to be significantly correlated with WAIS-IV Digit Span Forward and Backward tests (r= .53 and r= .54, N= 49 respectively). Additionally, both tests have shown good test-retest reliability (r= .73 and r=.68, N=7,457 respectively; Chaytor et al., 2020; Fortenbaugh et al., 2015; Germine et al., 2012; Hartshorne & Germine, 2015).
2. **TMB Digit Symbol Matching.** This TMB Digit Symbol Matching Test, adapted from the Digit Symbol Coding subtest on the WAIS (2008), was administered to participants

as a measure of processing speed (Joy et al., 2004). This test takes approximately 3 minutes to complete. Here, participants are asked to match as many symbols and numbers as possible to the symbol-number key in 90 seconds. The score for this test is based on the number of trials completed correctly in 90 seconds. The TMB Digit Symbol Matching test exhibits good test-retest reliability (r= .72, N= 1,026). In terms of validity, this test has been correlated with the response speed in a simple reaction time test (r= .32, N= 21,023), and a choice reaction test (r= .39, N= 12,441). The mean raw score of the TMB Digit Symbol Matching test is 48.22 (Chayton et al., 2020; Hartshorne & Germine, 2015; Passell et al., 2019; Rutter et al., 2020).

3. **TMB Verbal Paired Associates.** This test measures verbal episodic memory, as well as sustained attention, response inhibition, and cognitive control. The TMB Verbal Paired Associates test is adapted based on paradigms that assess context-dependent encoding and verbal memory retrieval, as opposed to recognition. It takes approximately five minutes to complete (2.4 minutes memorization, 2.4 minutes recall). Respondents are asked to learn and then recall a set of word pairs. During a practice trial, respondents are presented with 25 pairs of words, one pair at a time for six seconds each. After a brief delay, participants are shown the words originally presented and are asked to choose the second word from the list of four words (Germine et al., 2012; Wilmer et al., 2010). The score of the TMB Verbal Paired Associates is based on the number of pairs identified accurately out of 25 words presented. This test has shown good internal (split-half) reliability, (r= .82, N= 2,073). In regard to validity, it has been shown that TMB Verbal Paired Associates is correlated with Forward Digit Span (r= .23, N=494), and Digit

Symbol Matching (*r*= .36, *N*=517), as well as vocabulary performance (*r*= .37, *N*= 521; Germine et al., 2012; Passell et al., 2019).

4. **TMB Visual Paired Associates.** This test measures visual memory and episodic memory. The test takes approximately five minutes to complete (2.4 memorization, 2.4 minutes recall). Similar to the TMB Verbal Paired Associates test, this test is adapted from paradigms that assess context-dependent encoding and memory retrieval, as opposed to recognition (Vellutino et al., 1975). During this test, the participants are asked to learn and then recall a set of image pairs. During the memorization phase, participants are presented with 24 pairs of images, one pair at a time, six seconds per pair. After a brief delay, participants are then shown the images originally presented and are asked to choose the second image from the set of six images. The score on the TMB Visual Paired Associates is based on the number of pairs matched accurately out of 24 words presented. This test has shown high internal reliability (split-half) (*r*= .79, *N*= 6,380). In regard to validation, TMB Visual Paired Associates has been correlated with multiple object tracking (a test of visual perception and attention) (*r*= .30, *N*= 5288). The mean raw score of this test is 15.74 (Germine et al., 2012; Passell et al., 2019).

Sleep Measures

1. **Pittsburg Sleep Quality Index (PSQI)** is a 19-item self-report measure assessing respondents' sleep quality over the previous month (Bastien et al., 2001). The 19 items are divided into seven subcomponent scores, with the scores ranging from 0 to 3. The seven-subcomponent scores include the following variables: duration of sleep, sleep latency, sleep disturbance, daytime sleepiness, sleep efficiency, subjective sleep quality, and the use of medications. A global PSQI score is calculated by summing the seven subcomponent scores, ranging from 0 to 21, with the higher scores indicating worse sleep

quality (Buysse et al., 1989). The analysis of the psychometric properties of PSQI show good validity and internal consistency (Cronbach's alpha = .80; Buysse et al., 1989; Carpenter & Andykowski, 1998).

2. **The Insomnia Severity Index (ISI)** is a brief self-report questionnaire, which measures an individual's perception of both nocturnal and diurnal symptoms of insomnia (Bastien et al., 2001). The ISI consists of seven items that assess the perceived severity of sleep-related challenges over the past two weeks. Scores for all seven items are summed, with the total score ranging between 0-28, with the greater score indicating the severity of insomnia. The analysis of the psychometric properties of ISI reveals good concurrent validity and internal consistency (Cronbach's alpha = .85; Bastien et al., 2001; Morin, 1993).

3. **The Epworth Sleepiness Scale (ESS)** is an eight-item brief self-report measure that assesses individuals' general levels of daytime sleepiness (Johns et al., 1991; Johns, 1992). Respondents are asked to rate their usual chances of falling asleep in eight commonly experienced daytime situations (e.g., when sitting and reading) on a scale of 0 ("would never dose") to 3 ("high chance of dozing"). The total score is calculated by adding the sum of all eight items, yielding a range of 0-24, with higher scores indicating increased daytime sleepiness (Johns, 1991). The analysis of the psychometric properties of ESS indicates good validity and internal consistency (Cronbach alpha = .88; Johns et al., 1991; Johns, 1992).

Resilience questionnaire

The Connor-Davidson Resilience Scale (CD-RISC-10; Connor & Davidson, 2003) is an abbreviated and a widely used self-report measure that quantifies the levels of perceived resilience, such as the ability to cope with stressful events. In general, the scale aims to address the following concepts: flexibility, self-efficacy, optimism, ability to regulate emotion, and maintenance of cognitive focus and attention under stress. The total score is obtained by summing all the scores, which can range from 0 to 40, with higher total scores indicating greater resilience and lower scores indicating less resilience The analysis of the psychometric properties of CD-RISC-10 indicates good internal consistency (Cronbach's Alpha = .85) and construct validity (Campbell-Sills & Stein, 2007).

Mental and Physical Health Complaints

To assess physical and mental health complains experienced within the last month, the Physical Health Questionnaire (PHQ) and the Kessler 6-Item Psychological Distress Inventory (K-6) and the were used (Kessler et al., 2002; Schat et al., 2005). The total scores are obtained by summing the responses separately for each of the two questionnaires, with the higher scores indicating more physical and mental health complaints over the last month. These measures have shown to have good internal consistency (Cronbach's Alpha = .91 and .89, respectively; Kessler et al., 2002; Schat et al., 2005). Of note, both questionnaires are already embedded in the STRAIN questionnaire software (Slavich & Shields, 2018).

Data Analysis Plan

All data analyses were performed using the computer software SPSS (Version 26; IBM, 2020). Before running analyses, descriptive statistics (i.e., mean, median, mode, standard deviation, skewness, and kurtosis) were obtained for demographic background (i.e., sex, gender,

SES, alcohol intake quantity and frequency, the use of ADHD medications and caffeine, and COVID-19 related distress questions) and primary study variables (i.e., cumulative lifetime stress, WM, processing speed, verbal and visual episodic memory, sleep quality, insomnia severity, daytime sleepiness, resilience) to ensure statistical modeling assumptions (e.g., normality, homoscedasticity, independence) were met. For instance, log and square root transformations were conducted to normalize the data for the cognitive measures; however, it did not reduce the skew. As a result, it was decided to use the original values for the cognitive measures that did not include transformations. Multicollinearity analyses revealed that neither of the key variables met both criteria for multicollinearity. Finally, a number of outliers were removed from key variables; the outliers were determined by examining histograms and identifying values that fell outside of the interquartile (IQR) range "fences" (e.g., Q3 ± (1.5 * IQR)).

Hypothesis one. It was hypothesized that cumulative lifetime stress would relate to cognitive function (i.e., WM, processing speed, and verbal and visual episodic memory)**.**

Analysis of hypothesis one. Linear regression analyses were performed to examine whether cumulative lifetime stress would predict cognitive function variables.

Cognitive function measures were the criterion variables and cumulative lifetime stress was the predictor variable. More specially, four separate regression analyses were performed for each of the criterion variables (e.g., WM, processing speed, visual and verbal episodic memory). Next, additional variables such as sex, parental education, alcohol intake, and COVID-19-related variables were included as predictors in the regression model to examine the role of those covariates in the stress-cognitive function relationship. Additionally, as exploratory analyses of

the covariates, the role of the use of stimulants (e.g., ADHD medications and caffeine) was also examined.

Hypothesis two. It was hypothesized that cumulative stress would relate to sleep function (i.e., sleep quality, insomnia severity, daytime sleepiness).

Analysis of hypothesis two. Linear regression analyses were performed to examine whether cumulative lifetime stress would predict sleep function variables. Sleep-related measures were the criterion variables and cumulative lifetime stress was the predictor variable. More specially, three separate regression analyses were performed for each of the predictors (e.g., sleep quality, insomnia severity, and daytime sleepiness). Next, additional variables such as sex, parental education, alcohol intake, and COVID-19-related variables were included as predictors in the regression model to examine the role of those covariates in the stress-sleep relationship. Additionally, as exploratory analyses of the covariates, the role of the use of stimulants (e.g., ADHD medications and caffeine) was also examined.

Finally, exploratory analysis using linear regression was conducted to examine the relationship between cumulative lifetime stress and the subcomponents of PSQI (i.e., the duration of sleep, sleep disturbance, sleep latency, daytime dysfunction due to sleepiness, and sleep efficiency).

Hypothesis three. It was hypothesized that resilience would moderate the cumulative lifetime stress and cognitive function relationship. More specifically, it was hypothesized that higher levels of resilience would weaken the negative relationship between cumulative lifetime stress and global cognitive function outcome.

Analysis of hypothesis three. To determine if resilience was a significant moderator of the relationship between cumulative lifetime stress and the global cognitive function score, a

moderation analysis was conducted using the Hayes PROCESS macro in SPSS (Hayes, 2018). More specifically, cognitive function global score was the criterion variable, and cumulative lifetime stress was the predictor variable. Finally, stressXresilience (stress variable multiplied by resilience variable) was included as a computed moderator variable.

Hypothesis four. It was hypothesized that resilience would moderate the cumulative stress and sleep relationship. More specifically, it was hypothesized that higher levels of resilience would weaken the positive relationship between cumulative lifetime stress and sleep quality disturbance, insomnia severity and daytime sleepiness.

Analysis of hypothesis four. To determine if resilience was a significant moderator of the relationship between cumulative lifetime stress and each of the sleep measures of interest (sleep quality, insomnia severity, and daytime sleepiness), three moderation analyses were conducted using the Hayes PROCESS macro in SPSS (Hayes, 2018). Sleep function measures were the criterion variables and cumulative lifetime stress was the predictor variable. More specially, three separate regression analyses were performed for each of the criterion variables (e.g., sleep quality, insomnia severity, daytime sleepiness); cumulative lifetime stress was the predictor variable. Finally, stressXresilience (stress variable multiplied by resilience variable) was included as a computed moderator variable. Overall, three separate moderation analyses were conducted, given that there are several criterion variables in this hypothesis.

CHAPTER IV: RESULTS

Participant Demographics

A total of 153 individuals participated in the study. The ages of the participants ranged from 18 to 25 years old (*M*= 18.65, *SD*=1.23) as indicated by the inclusion criteria. The sample consisted of 100 women and 52 men, and one student who identified as transgender/gender queer/non-binary. In terms of race, 0.7% of participants identified as American Indian/Alaska Native, 3% identified as Asian, 20% identified as Black/African American, 66% as European American/White, and 10% of participants identified as multi-racial or other, which is representative of University's demographics (*ECU*, n.d). Additional demographic characteristics are reported in Table 1. Eleven participants were removed from the study due to various data collection errors. One participant was removed as they did not pass a validity check; as a result, the final sample included 153 participants.

Information about participants' self-reported wellbeing was also collected. Psychological distress and physical health complaints in the past month were assessed using K-6 (*M*= 39.62, *SD*= 13.49, range: 16-79) and PHQ (*M*= 15.15, *SD*= 5.83, range: 6-30) measures, respectively. Furthermore, participants were asked to subjectively rate their overall physical health and wellbeing on a scale from 0 to 4, with 0 being "excellent health," and 4 being "poor health" (*M* = 1.19, *SD* = 0.89). On average, participants reported their physical health and wellbeing to be within the "good" range, with commonly reported health-related problems including the following: a history of allergies, mild visual problems (e.g., astigmatism), asthma, migraines or chronic headaches, and sickle cell anemia. Common mental health self-reported histories included anxiety, learning disorders, ADHD, and depression (See Table 3).

Descriptive Statistics of the Main Study Variables

The descriptive statistics of continuous main study variables included the following: the STRAIN total score; cognitive measures: WM (Forward and Backward Digit Span), processing speed (Digit Symbol Matching), verbal and visual episodic memory (Verbal and Visual Paired Associates); sleep measures: insomnia severity, daytime sleepiness, sleep quality and finally the measure of resilience (See Table 2 for details regarding means and standard deviations). The skew and kurtosis information, as well as the internal validity of main measures, are also reported in Table 2.

Cumulative Lifetime Stress and Cognitive Function

Hypothesis one. Hypothesis one posited that elevated levels of cumulative lifetime stress as measured by the STRAIN would relate to worse global cognitive function. The goal was to then separately examine each of the cognitive function measures (i.e., WM, processing speed, verbal episodic memory, visual episodic memory), and linear regression analyses were performed to examine these relationships. Of note, for the global cognitive function scores, both raw and scaled scores were explored. Scaled scores were adjusted for age, gender, and educational background. Using regression, participants' cumulative lifetime stress did not predict global cognitive function raw, $R^2 = 0.00$, $F(1, 135) = 0.03$, $p = .864$, or scaled scores, $R^2 = 0.01$, $F(1, 134) = 1.01$, $p = .317$. Similarly, after controlling for sex, parental education, quantity and frequency of alcohol intake, and the extent of their ongoing worry about the COVID-19 pandemic, cumulative lifetime stress did not significantly predict raw, $R^2 = 0.04$, $F(1, 134) = 1.11$, $p = .982$, or scaled global cognitive function scores, $R^2 = 0.03$, $F(1, 133) = 0.77$, $p = .221$. None of the individual covariates included in the model as predictors were significant (see details in Table 4).

Next, each cognitive function domains including WM (measured by Forward and Backward Digit Span), processing speed (measured by Digit Symbol Matching), and verbal and visual episodic memory (measured by Verbal and Visual Paired Associates, respectively) were examined separately. Of note, given the similarity of the results between raw and scaled scores of individual cognitive function measures, only scaled scores were reported. As mentioned earlier, scaled scores have been adjusted for age, gender, and educational background and therefore allowed for clear interpretation and direct comparison to other cognitive measures (See details in Table 4).

Cumulative lifetime stress did not significantly predict WM (Forward Digit Span), R^2 = 0.01, $F(1, 143) = 2.56$, $p = .112$. However, after controlling for covariates, the regression model became significant, $R^2 = 0.02$, $F(1, 142) = 1.57$, $p = .027$. This suggests that higher cumulative lifetime stress was associated with better performance on a WM task. Conversely, cumulative lifetime stress did not significantly predict WM scores as measured by Backward Digit Span, R^2 = 0.00, $F(1, 141) = 0.27$, $p = .608$; after controlling for covariates, the model remained non-significant, $R^2 = 0.02$, $F(1, 140) = 0.64$, $p = .652$. None of the individual predictors included as covariates were significant.

The model predicting processing speed score did not yield significant results, $R^2 = 0.00$, $F(1, 137) = 0.01$, $p = .918$; after controlling for covariates, the model remained non-significant, $R^2 = 0.03$, $F(1, 136) = 0.87$, $p = .869$. None of the individual predictors included as covariates were significant.

Finally, cumulative lifetime stress did not significantly predict verbal episodic memory performance: $R^2 = 0.00$, $F(1, 143) = 0.46$, $p = .498$; after controlling for covariates, the model remained non-significant, $R^2 = 0.02$, $F(1, 142) = 0.58$, $p = .349$. None of the individual predictors

included as covariates were significant. Similarly, cumulative lifetime stress did not significantly predict visual episodic memory scores: R^2 = 0.00, F(1, 142) = 0.01, p = .907; after controlling for covariates, the model remained non-significant, R^2 = 0.05, F(1, 141) = 1.35, p = .971. After including covariates into the model, COVID-19-related distress was the only significant predicting covariate of visual episodic memory (B= 1.57, SE= 0.67, β = 0.19, p = .028).

For all analyses described above, the use of stimulants (e.g., ADHD medications and caffeine) did not significantly predict cognitive function. Of note, in order to limit power-related concerns associated with having a large number of covariates, those variables were not included in the overall model. Overall, the results showed that cumulative lifetime stress was not associated with any of the cognitive function measures, except for WM as measured by TMB Forward Digit Span, specifically.

Cumulative Lifetime Stress and Sleep

Hypothesis two. Hypothesis two posited that cumulative lifetime stress as indicated by the STRAIN would relate to worse sleep quality, greater insomnia severity, and greater daytime sleepiness; linear regression analyses were performed to examine these relationships. As hypothesized, cumulative lifetime stress significantly predicted sleep quality, R^2 = 0.08, F(1, 144) =12.41, p <.001, suggesting that greater cumulative lifetime stress was associated with greater sleep disturbance. After controlling for covariates, the regression model remained significant, R^2 = 0.16, F(1, 143) = 5.31, p=.002. Next, each individual covariate as a predictor was examined; the only variable that emerged as a significant predictor was COVID-19-related worries (B= 1.10, SE= 0.41, β = 0.22, p= .009), suggesting that poor sleep quality was associated with more worries related to the pandemic (See Table 5 for details). Additionally, the use of stimulants (e.g., ADHD medication and caffeine) did not significantly predict sleep quality. As

mentioned earlier, those variables were not included in the overall model to limit power-related concerns associated with having a number of covariates.

Furthermore, exploratory analyses were conducted examining the seven subcomponents of the PSQI. Cumulative lifetime stress significantly predicted the following subcomponents: subjective sleep quality, R^2 = .04, F(1, 146) = 6.72, p = .011; sleep duration, R^2 = 0.05, F(1, 146) = 7.24, p= .008; sleep disturbances, R^2 = 0.05, F(1, 141) = 7.62, p= .007; and daytime dysfunction, R^2 = 0.12, F(1, 146) = 20.53, p< .001. The remaining subcomponents of the PSQI (i.e., sleep latency, habitual sleep efficiency, and the use of sleeping medications) were not significantly predicted by cumulative lifetime stress.

In another model, cumulative lifetime stress significantly predicted insomnia severity, R^2 = 0.14, F(1, 146)= 23.04, p <.001, suggesting that greater cumulative lifetime stress was associated with greater insomnia severity. After controlling for covariates listed above, the regression model remained significant, R^2 = 0.22, F(1, 145) = 7.83, p <.001. After including covariates into the model, COVID-19-related distress and parental education were the only significant predicting covariates of insomnia severity (B= 1.57, SE= 0.67, β = 0.19, p = .020 and B= -0.73, SE= 0.37, β = -0.15, p = .049, respectively; See Table 5 for details). The use of stimulants (e.g., ADHD medication or caffeine) did not significantly predict insomnia severity.

Finally, as hypothesized, cumulative lifetime stress also significantly predicted daytime sleepiness, R^2 = 0.06, F(1, 144) = 8.67, p = .004, suggesting that greater cumulative lifetime stress was associated with more daytime sleepiness. After including covariates, the model remained significant, R^2 = 0.12, F(1, 143) = 3.66, p = .010. Only sex emerged as a marginally significant predictor of daytime sleepiness, B= 1.16, SE= 0.60, β = 0.16, p = .056, where women were more likely to experience greater daytime sleepiness. None of the other covariates were

significant predictors (See Table 5 for details). Similar to the findings above, the use of stimulants (e.g., ADHD medication or caffeine) did not significantly predict daytime sleepiness.

Cumulative Lifetime Stress, Cognitive Function, and Resilience

Before exploring resilience as a moderator of stress and cognitive function, the relationships between cumulative lifetime stress, global cognitive function, and resilience were examined. First, cumulative lifetime stress did not significantly predict resilience, $R^2 = 0.01$, $F(1, 146) = 1.45$, $p = .231$. After accounting for covariates, the relationship remained non-significant, $R^2 = 0.10$, $F(1, 145) = 3.24$, $p = .640$. When examining each of the individual covariates, only COVID-19-related worries emerged as a significant predictor (B= -2.40, SE= 0.84, $\beta = -0.24$, $p = .005$). Sex emerged as a marginally significant predictor (B= -1.94, SE= 1.01, $\beta = -0.16$, $p = .056$), where men were more likely to report greater resilience. Next, we examined resilience as a predictor of cognitive function; however, it did not emerge as a significant predictor for any of the cognitive function domains.

Hypothesis three. It was hypothesized that resilience would moderate the relationship between cumulative lifetime stress and cognitive function (see Figure 1). More specifically, it was hypothesized that higher levels of resilience would weaken the relationship between cumulative lifetime stress and global cognitive function score. However, given that cumulative lifetime stress did not significantly predict any of the cognitive function domains, the moderation was not tested.

Cumulative Lifetime Stress, Sleep, and Resilience

Before exploring resilience as a moderator of cumulative lifetime stress and sleep, the relationship between sleep and resilience was explored. Self-reported resilience significantly predicted sleep quality, $R^2 = 0.09$, $F(1, 150) = 13.91$, $p < .001$, suggesting that increased resilience

was associated with less sleep quality disturbance (B= -0.16, SE= 0.04, β = -0.29). Resilience significantly predicted insomnia severity, R^2 = 0.08, F(1, 151) = 13.24, p <.001, suggesting that higher levels of resilience were associated with reduced reported insomnia severity (B= -0.25, SE= 0.07, β = -0.29). Finally, resilience significantly predicted daytime sleepiness R^2 = 0.04, F(1, 150) = 5.41, p= .021, suggesting that increased resilience was associated with reduced reports of daytime sleepiness (B= -0.12, SE= 0.05, β = -0.19).

Hypothesis four. It was hypothesized that resilience would moderate cumulative stress and sleep relationships. More specifically, it was hypothesized that higher levels of resilience would weaken the relationship between cumulative lifetime stress and sleep disturbance (see Figure 2). The interaction effects of cumulative lifetime stress and resilience were examined using the Hayes PROCESS macro in SPSS (Hayes, 2018). None of the interaction terms with cumulative lifetime stress were significant. The slopes, standard errors, and p-values can be found in Table 5.

CHAPTER V: DISCUSSION

The objective of the current study was to examine the relationships of cumulative stress with cognitive function, and sleep outcomes, and examine resilience as a moderator in a sample of college students. Specifically, the study had the following goals: 1) to examine the relationship between cumulative lifetime stress and domains of cognitive function; 2) to explore the relationships between cumulative lifetime stress and several domains of sleep; and 3) to examine the moderating role of resilience in cumulative stress and cognitive function, as well as cumulative lifetime stress and sleep relationships.

It is important to note that the college student sample used in the current study adds a unique contribution to the literature, as many young adults experience problems with sleep given the increased demands and challenges associated with pursuing a college degree (Taylor et al., 2013). Further, existing research suggests that college education contributes to improved domain-general abilities such as cognitive control, reasoning, WM, and processing speed (Cattell, 1971; Deary et al., 2007; Guerra-Carrillo et al., 2017; Lövdén et al., 2020). Higher education has predicted better performance on verbal and perceptual reasoning subcomponents of the FSIQ test, suggesting that education has varying positive effects on certain domains of cognitive function contributing to general intelligence estimates (Kaufman et al., 2009). The amount of formal education one has received has also been positively correlated with cognitive ability throughout the lifespan and is associated with a lower risk of dementia later in life, potentially due to the contribution of cognitive reserve (Lövdén et al., 2020). Overall, considering the unique effects of stress on sleep and cognitive function in a college student population, it is essential to consider these individual differences while interpreting the study's findings.

Cumulative Lifetime Stress and Cognitive Function

The initial hypothesis was based on theories that propose that the exposure to various stressors over the course of one's lifetime would contribute to negative effects on physical and mental health outcomes, including but not limited to cognitive function (Senft Miller et al., 2021; Slavich & Shields, 2018; Toussaint et al., 2016). Specifically, it was hypothesized that greater cumulative lifetime stress would be associated with greater cognitive impairment, such as reduced WM, processing speed, and verbal and visual episodic memory. Contrary to the hypothesis, the results did not yield significant outcomes, except for with scores from the WM measure (i.e., TMB Forward Digit Span). Instead, it was found that greater cumulative lifetime stress was associated with better performance on a particular WM task after controlling for sex, parental education, alcohol intake, and COVID-19-related distress. This finding contradicts the hypothesis that posited an inverse relationship. Previous studies have consistently reported that greater chronic stress exposure is usually associated with WM impairment (Evans & Schamberg 2009; Lee & Goto, 2015). However, studies examining the effects of acute stress on WM have suggested that elevated cortisol secretions were associated with better performance on a WM task in terms of encoding and maintenance. Based on the present findings, it is proposed that elevated cortisol may work as a facilitating mechanism assisting subsequent long-term memory consolidation (Cornelisse et al., 2011; Stauble et al., 2013). Finally, another study also found that moderate stress exposure may improve one's reaction time during a WM task without decreasing the accuracy (Lai et al., 2014). It is possible that while exposure to acute stress under experimental conditions may improve WM in a unique way, it does not necessarily parallel with the cumulative lifetime stress effects. Future studies should further explore the effects of acute versus chronic stress on WM, as well as examine stress levels using biomarkers (e.g., cortisol,

norepinephrine) to see whether self-report cumulative stress and its effects on cognitive function are congruent with physiological markers of stress.

Contrary to the proposed hypothesis, there were no significant findings with any other measures of cognition, essentially suggesting no statistically significant relationship between cumulative lifetime stress and cognitive function as measured by TMB. Previous studies utilizing the STRAIN have reported that greater cumulative lifetime stress was associated with lower processing speed and executive functioning. It has also been found that low levels of stress were associated with diminished memory for stimulus-response relative to those with moderate stress levels (Goldfarb et al., 2020; Slavich & Shields, 2018). However, one validation study of the STRAIN conducted with a Brazilian cohort did not yield significant results in the relationship between the STRAIN and executive function (Stroop task; Cazassa et al., 2020). In another study using a sample of older adult women, the number of total lifetime stressors and/or their severity was also not significantly associated with immediate and delayed recall or WM (Senft Miller et al., 2021). However, greater lifetime stress exposure was associated with increased symptoms of subjective memory function. This finding parallels the current study's findings as the statistical results between the STRAIN and most cognitive measures (except for Forward Digit Span) were not significant. Perhaps, future studies should focus on further understanding the role of lifetime stress exposure in subjective memory, as well as using gold-standard measures of cognition.

Given mixed findings within the STRAIN literature regarding cognitive function, as well as mostly null findings in this study, a few possible explanations are provided. For instance, it appears that most of the previous TMB validation studies utilized community, college undergraduate, and clinical samples, where participants' average age ranged from 27-60 years old and had varying levels of education (Chaytor et al., 2021; Fortenbaugh et al., 2015; Germine

et al., 2012; Rutter et al., 2020; Vogel et al., 2020; Wilmer et al., 2012). In contrast, the sample in this study strictly consisted of college students, with the average age being much lower than in the studies mentioned above (age: *M*= 18.65, *SD*= 1.23) as most students were within their first year of college. However, it is important to note that when comparing the raw cognitive function scores of this study with scores in TMB sample validation study, the scores were comparable (Singh et al., 2021; see Table 7 for comparison scores). The similarity between test scores suggests that the validity of TMB in a college student population is not likely to be of concern.

It is also possible that TMB might not be sensitive enough to detect subtle cognitive impairment as it relates to cumulative lifetime stress, when examined together. For instance, while cognitive function scores of TMB in this sample were comparable with previous validation studies, the cumulative lifetime stress total count score was lower compared to what has been reported in the original STRAIN validation study (Singh et al., 2021; Slavich & Shields, 2018). Specifically, there were two main differences between the current sample and the original validation study sample: 1) age (*M*[*SD*]= 18.65 [1.23] vs. 37.82 [11.72], respectively) and 2) cumulative lifetime stress total count (*M*[*SD*]= 15.91 [9.68] vs. 25.77 [16.85]), respectively; Slavich & Shields, 2018). It is possible that given the age and the stage in life of the current sample, college students have yet to fully experience the negative and pervasive effects of cumulative lifetime stress exposure. Therefore, cumulative life stress did not predict performance on cognitive functioning tasks due to restricted range of scores. This is not to say that younger adults do not experience significant stressors and trauma; instead, it is argued that the current sample demographics may limit the ability to fully capture the relationship.

Additionally, based on the general intelligence theories of Horn and Cattell (1966), it has been posited that the two overarching categories of intelligence (i.e., fluid abilities and

crystallized abilities) peak at different ages (Schneider & McGrew, 2012), suggesting potential variations in cognitive function outcomes depending on age. For instance, using the TMB neuropsychological test battery, it has been shown that performance on measures of WM (i.e., fluid intelligence) tasks may peak at around 30 years old, which is significantly later than the optimal peak of processing speed performance (i.e., fluid intelligence), and a significantly earlier peak for optimal vocabulary performance; however, the age-of-peak performance for verbal and visual memory was not significant (Hartshorne & Germine, 2015). Overall, these findings provide further insight into the idea that the current sample, in addition to having limited exposure to significant lifetime stressors, may have yet to reach their age-of-peak performance on certain cognitive tasks, therefore limiting the significant findings. Future studies may wish to explore a more diverse sample including older and non-traditional college students.

Finally, it has been argued that traditional neuropsychological tests have not always been successful at detecting impairment in cognitive functioning in college students as evidenced by ample ADHD literature. This lack of a specific relationship between ADHD diagnosis and executive dysfunction as measured by standard neuropsychological batteries creates further challenges in understanding the usefulness of neuropsychological assessment in a college student sample. While it is widely understood that ADHD is a disorder of executive dysfunction, even in persons with an ADHD diagnosis, this is not always evident on neuropsychological tests of the same construct. As such, seeing impairment on any single or even multiple executive functioning tests does not necessarily help to reliability differentiate between a person with and without ADHD diagnosis (Koziol & Stevens, 2012). Some arguments for this discrepancy are related to the ecological validity of tests of executive function, in that laboratory-based measures of cognitive functioning do not fully capture the nuances of the same constructs in everyday life

(Barkley, 2015). It is possible, then, in the current study that the same concerns of ecological validity are present. It is also necessary to consider that students who pursue college education may generally exhibit higher cognitive functioning (e.g., due to adaptive strategies) which serves as a protective factor against the deleterious effects of cumulative lifetime stress (Meng & D'Arcy, 2012). Overall, it is possible in the context of the present study whereby college students participated in the 1.5-hour research study required for course credit, impairments in cognitive functioning thought to be related to cumulative lifetime stress as assessed by the TMB software were not as prominent or easily detectable as compared to other methods of assessment (e.g., self-report measures).

Cumulative Lifetime Stress and Sleep

Hypothesis two examined cumulative lifetime stress and sleep with a specific focus on three different sleep-related factors: sleep quality, insomnia severity, and daytime sleepiness. Regarding sleep quality, which was assessed by the PSQI, it was found that cumulative lifetime stress measured by the STRAIN was positively associated with poor sleep quality, suggesting that the more cumulative stress one has experienced, the more reported sleep quality concerns there are. This finding was expected, as previous studies have examined the relationship between cumulative lifetime stress measured by the STRAIN and sleep quality measured by the PSQI (Cazassa et al., 2020; Slavich & Shields, 2018). The same findings are present even in varying samples (e.g., U.S. community sample, Brazilian cohort) underscoring the strong predictive validity of STRAIN in relation to sleep quality measured by the PSQI, despite potential cultural and background differences of participants (Cazassa et al., 2020; Slavich & Shields, 2018). This study adds to the literature on these relationships by revealing the same findings are present in college students attending a large, southeastern U.S. university.

While many studies using the PSQI and STRAIN exist, to our knowledge, this is the first study to further explore cumulative lifetime stress and its relation to all seven components of PSQI. As indicated above, results from the current study show that cumulative lifetime stress is significantly related to the PSQI components of subjective sleep quality, sleep duration, sleep disturbance, and daytime dysfunction. That is, greater cumulative lifetime stress was associated with diminished subjective sleep quality, increased sleep duration, greater sleep disturbance, and greater daytime dysfunction.

Initially, one unexpected finding was a positive relationship between stress and greater sleep duration, suggesting the more stress one has experienced the more hours of sleep one may experience each night. This finding appears inconsistent with previous sleep literature in which studies have shown either a non-significant or negative relationship between these two variables (i.e., suggesting that individuals with shorter sleep duration have higher odds of reporting perceived stress; Bassett et al., 2016; Kim et al., 2019). However, the positive relationship between greater stress and greater sleep duration is consistent with depression literature (Zhai et al., 2015). Given that cumulative lifetime stress is also positively associated with symptoms of depression, it can be argued that the relationship between stress and longer sleep duration may have similar or overlapping mechanisms; that is, individuals experiencing a significant amount of chronic stressors over a prolonged period of time, may experience chronic exhaustion and an increased risk for health concerns, therefore requiring more sleep (Jike et al., 2018; Zhai et al., 2015).

This was also the first study to examine the relationship between cumulative lifetime stress measured by the STRAIN and insomnia severity and daytime sleepiness. As hypothesized, cumulative lifetime stress predicted insomnia severity and daytime sleepiness, suggesting that

more cumulative stress was associated with greater insomnia severity and daytime sleepiness. These findings are not surprising given that several studies ascertained a relationship between the above-mentioned variables. For instance, Pillai et al. (2014) found that the relationship between stress and the risk of insomnia was moderated by the chronicity of the stressors, suggesting that individuals who experience chronic stress were more likely to develop insomnia; additionally, higher levels of stress were also associated with higher chances of developing insomnia. Burnout, which represents chronic exposure to lifetime stressors and chronic fatigue, and cognitive depletion, has also been significantly associated with overall sleep disturbances and more specifically, chronic insomnia (Lo Martire et al., 2020). This relationship is likely to be bi-directional; that is, chronic stressors and burnout may lead to insomnia, and experiencing insomnia may also lead to fatigue and depletion of resources and motivation, consequently contributing to chronic stress and burnout. Similarly, it has long been suggested that insomnia can be initiated by stressful life events, where hyperarousal can negatively affect sleep and influence the onset of insomnia (Kalmbach et al., 2018). The biobehavioral model of insomnia, also known as the "3P" or diathesis-stress model, can also help explain the findings. This model considers three major factors and their contribution to insomnia: predisposing factors (e.g., age, genetics, family history), precipitating factors (i.e., specific events that precipitate acute insomnia symptoms such as studying for a test), and perpetuating factors (i.e., coping styles and compensatory behaviors; Spielman et al., 1987; Wright et al., 2019). Based on this model, it can be further argued that all three factors evolve and negatively influence sleep as a result of cumulative stress exposure. For instance, chronic accumulation of stress (e.g., prolonged financial difficulties) may act both as predisposing and precipitating factors of insomnia; at the same time, prolonged financial difficulties can lead to the development of negative style thinking

(i.e., perpetuating factors), further continuing to the maintenance of insomnia (Wright et al., 2019). Finally, in this study, greater sleep disturbance and insomnia severity were associated with greater COVID-19 distress. This finding is not surprising, given that a number of studies have shown such a link, further suggesting the detrimental effects of the pandemic on one's overall health and wellbeing (Bhat & Chokroverty, 2022).

Resilience as a Moderator of Cumulative Lifetime Stress and Cognitive Function and Cumulative Lifetime Stress and Sleep Relationships

The third and fourth hypotheses posited that resilience would moderate the stress-cognitive function relationship and stress-sleep relationship, respectively. Specifically for hypothesis three, it was predicted that resilience would lessen the negative interaction of stress and cognitive function. Prior to conducting moderation analyses, the relationships between cumulative lifetime stress and resilience, and sleep and resilience, were explored.

Regarding the relationship between stress and resilience, although the association was negative, cumulative lifetime stress did not significantly predict resilience. This finding is consistent with previous work in which one study found no significant difference in resilience in students who were experiencing different stress levels (i.e., low, moderate, high) during a prolonged stressful experience (i.e., three years during a college entrance examination in China; Sun et al., 2021) for which the authors argued could be due to resilience being a stable or a trait characteristic that does not differ depending on the situation (Sun et al., 2021; Leipold & Greve, 2009). In contrast, a number of studies have reported an U-shaped relationship between stress and resilience. For example, moderate exposure to adverse life events has been associated with lower global distress, greater life satisfaction, fewer PSTD symptoms, and more positive psychophysiological responses when exposed to laboratory stressors compared to having no or

high adversity (Seery et al., 2010; 2013). Similarly, under low, but not high, family and neighborhood stress, personal characteristics and individual differences distinguished resilience and non-resilience in children (Jaffee, 2007).

With this discrepancy in findings, it is important to note as such differences may be due to several factors such as the type of stress measures used (e.g., early childhood trauma, perceived stress in the past month, chronic stress), the type of resilience measures used, and varying ways of categorizing stressors (high vs. low; or high vs. moderate vs. low). Regarding this study's findings, it is important to note that we only focused on exploring the relationship between cumulative lifetime stress and resilience assessed by the STRAIN. To date, only one previous study examined the relationship between cumulative lifetime stress measured by the STRAIN and resilience. The authors found that moderate stress exposure was associated with increased resilience in breast cancer survivors (Dooley et al. 2017). Future studies should further examine whether there would be a different interaction if exploring the effects of acute versus chronic stress exposures. Different timelines of those stressors (e.g., childhood stress versus adulthood stress) and their relationship to resilience should also be investigated. While further exploration of different types of stress experienced during a specific time period of life was not the focus of this project, the STRAIN gives as a unique opportunity to explore this further. Finally, to examine if previous studies can be replicated in this sample, future analyses should also include separating the severity of stressors by low, moderate, and high to further understand how stress severity may be related to resilience.

The relationship between sleep and resilience was also explored. In this study, resilience predicted sleep, suggesting that higher levels of resilience were associated with more sleep disruption. This finding was not surprising given a plethora of studies have suggested an inverse

relationship between resilience, poor sleep quality, and insomnia symptoms (Cai et al., 2021; Lenzo et al., 2022; Palagini et al., 2022; Seelig et al., 2016; Zhang et al., 2022; Zhou et al., 2022). This relationship is likely due to several factors including emotion dysregulation, high sleep reactivity to stress, and cognitive pre-sleep hyperarousal. It has also been suggested that individuals with sleep-related disturbances such as insomnia, may have a low capacity to adapt to stress, further affecting arousal and emotion regulation, which as a result, further contributes to the development and maintenance of insomnia (Palagini et al., 2018). Given the link between sleep and resilience, interventions targeting resilience to improve sleep and overall wellbeing need to be considered and tailored to an individual depending on presentation.

Finally, based on the proposed hypotheses, the moderation analysis was conducted. Contrary to the initial hypotheses, the interaction terms were not significant, suggesting that resilience as a moderator did not influence either the stress-cognitive function relationship or the stress-sleep relationship. After examining the moderations, it is evident that there was no interaction effect, meaning that the relationship between cumulative lifetime stress and cognitive function global score, as well as the relationship between cumulative lifetime stress and sleep (e.g., sleep quality, insomnia severity, daytime sleepiness) remained the same regardless of the resilience level (i.e., low vs. moderate vs. high).

As discussed earlier, one potential explanation for the lack of findings is the relatively limited exposure to cumulative lifetime stress as a whole due to the average age of this sample's participants. It is also possible due to age and relatively limited exposure to lifetime stressors, the participants in this sample have not had enough experiences to utilize the benefits of resilience to the full extent. Of note, the average scores on the CDRS-10 initial validation study (*M*= 27.21, *SD*= 5.84) which included a sample of undergraduate students with an average age of 18.8 years,

were comparable with the average CDRS-10 scores in this study (Campbell-Sills & Stein, 2007). However, when examining resilience levels in an older adult sample, as expected, those participants exhibited higher average resilience scores (*M*= 31.11, *SD*= 15.19) compared to this study's sample (*M*= 28.46, *SD*= 7.93), further suggesting that relatively limited experience with resilience and cumulative lifetime stress may have influenced the current study's finding (Tourunen et al., 2021).

It is also important to consider that younger adults experience psychological resilience differently compared to older cohorts. That is, depending on age, individuals may differ in the way they react to or evaluate hopelessness, depression, and illness perception. One study found that older adults are likely to exhibit higher resilience levels compared to younger adults, especially in regard to problem-solving and emotion regulation abilities. In younger adults, greater resilience was associated with more social support. Low energy and poor perceptions of health were associated with lower resilience, and low hopelessness was associated with greater resilience in both groups. Overall, these results suggest that depending on age, individuals may have varying levels of resilience, with different features of resilience being more prominent than others at certain timepoints (Gooding et al., 2012). Based on this finding, it is possible that the lack of significant moderation findings was due to the younger age of the participants and the features of resilience pertinent to this population. Future studies should further examine the role of resilience as a moderator in the abovementioned relationship using a sample of different ages. Additionally, the resilience measure used in this study (i.e., CD-RISC-10) contains diverse items pertaining to the following domains: flexibility, self-efficacy, ability to regular emotion, optimism, and cognitive focus/maintaining attention under stress (Campbell-Sills & Stein, 2007).

Given the potential variability in the type of resilience expressed depending on age, future studies should further examine different aspects of resilience as moderators.

Limitations

Despite the important findings discussed earlier and their clinical implications, this study has several limitations. First, this is a cross-sectional design, which prevents us from making causal statements about the relationship between cumulative lifetime stress, cognitive function, and sleep. Second, the population of college students has unique challenges that could influence their cognitive function and sleep, therefore limiting the generalizability of our findings. It is also important to note that the relatively young age of the current sample may have contributed to the lack of findings, particularly pertaining to the relationship between cumulative lifetime and cognitive function. It is likely that because of their age, the participants in the current sample (as a whole) did not have as much exposure to lifetime stressors as individuals in other life stages, limiting the negative effects on cognition. Additionally, it is possible that given that the participants in this sample are pursuing higher education, on average, they are likely to experience better general functioning and related adaptability, which further prevents the deleterious effects of cumulative lifetime stress on cognition. Future studies should include a sample consisting of individuals with diverse demographics and varying physical and mental health diagnoses and symptomology. Therefore, to assist the generalizability of the findings, future studies should include a community sample of older individuals with varying life experiences. Additionally, it has also been noted that current TMB tests lack demographically adjusted normative data. Future studies are needed to assess performance and criterion performance validity using diverse clinical samples, which again, would improve the generalizability, sensitivity, and specificity of test results (Singh et al., 2021).

Third, while online neuropsychological assessment has great advantages, limitations, and challenges associated with online-based assessment must be considered. For instance, a common limitation of online-based assessment is the variability of devices used to complete assessments (e.g., computer, tablet, smartphone; Germine et al., 2019). However, the advantage of this study is that participants completed the TMB battery in person at the laboratory under the supervision of a researcher and used the same devices across participants. Given that the TMB battery was completed in the laboratory, participants had the opportunity to ask the PI study-related questions, further limiting the opportunity for technological errors. However, while the assessments were completed in the laboratory, behavioral observations were not recorded for the study, as might typically be the case with traditional neuropsychological assessment, making it challenging to determine if the participants were fully engaged (Feenstra et al., 2017). Future work should focus on developing the ability to have item-level data of digital tests to better understand different levels of engagement throughout testing (Singh et al., 2021).

Future Considerations

Given several limitations described earlier, directions for future studies will be considered. First, while this study solely focused on examining the effects of cumulative lifetime stress exposures, different types of stress (e.g., acute vs. chronic and the severity of stress) may have varying effects on cognitive function and sleep (Lo Martire et al., 2020; Sandi, 2013). As a result, future studies should focus on examining and delineating the unique effects of different types of stress and the severity of stress. The advantage of the STRAIN is that the measure output provides a convenient breakdown by different types of stressors (e.g., acute, chronic, recent and the severity of stress), which will assist future studies.

Second, given that TMB battery has not been validated in the college student population, future studies should include traditional neuropsychological batteries for comparison of findings. A previous study using the STRAIN found a relationship between cumulative lifetime stress and subjective cognitive functioning measures, but not objective (Senft Miller et al., 2021). Therefore, given the lack of significant findings using objective neuropsychological measures in this study, future projects should include a combination of gold-standard neuropsychological measures, online-based measures such as TMB, as well as subjective reports of cognitive functioning. Additionally, this study did not include a comprehensive neuropsychological battery and instead only assessed four individual cognitive domains (i.e., processing speed, WM, verbal, and visual episodic memory); future studies should include additional neuropsychological assessments to fully capture a profile of the relationship between cumulative lifetime stress and cognitive function. To further understand the cumulative effects of lifetime stress on cognitive function, future studies might also wish to focus on assessing cognitive function at several time points (i.e., one year versus five years versus 10 years) to fully capture the cumulative effects.

Third, the current study only included subjective measures of sleep. In addition to subjective assessment of sleep, future studies should include objective measures such as actigraphy or sleep electroencephalogram recordings to further understand the biological underpinnings of the relationship between cumulative lifetime stress and sleep. While this study only assessed aspects of sleep function such as sleep quality, insomnia severity, and daytime sleepiness, future studies should also include a more comprehensive assessment of different sleep conditions (i.e., sleep apnea, narcolepsy, restless leg syndrome) or utilize different sleep-related measures (i.e., Dysfunctional Beliefs and Attitudes About Sleep Scale, the

Perceptual Vigilance Task) that have yet to be explored in relation to the STRAIN (Dinges & Powell, 1985; Morin et al., 1993).

Finally, as discussed earlier, the relatively young age, as well as the relatively healthy status of the current sample may have contributed to the lack of some of significant findings in this sample. Therefore, future studies should focus on including a diverse sample consisting of participants of diverse demographics, cultural backgrounds, and varying clinical presentations (i.e., patients with ADHD diagnosis, severe mental illness, older adults, dementia patients, caregivers).

Conclusions and Study Summary

In summary, the current study consisted of two parts. First, the goal was to examine the relationship between cumulative lifetime stress and cognitive function, and the potential moderating role of resilience. Despite the proposed hypotheses, stress did not significantly predict cognitive function, except for WM (Forward Digit Span), where greater cumulative lifetime stress was associated with better WM task performance; resilience as a moderator did not influence the relationship. The second goal was to examine the relationship between cumulative lifetime stress and sleep, and the potential moderating role of resilience. As hypothesized, stress predicted sleep outcomes, suggesting that greater cumulative lifetime stress was associated with worsened sleep quality, increased insomnia severity, and daytime sleepiness; resilience as a moderator did not influence the relationship. This was the first study to examine the relationship between cumulative lifetime stress using the STRAIN and online-based neuropsychological assessment battery, TMB. Additionally, this was also the first study to examine the relationship between the STRAIN and subcomponents of PSQI, as well as other sleep-related measures such as ISI and ESS. Overall, the current study underlines the deleterious

effects of lifetime stress exposure on sleep, further adding to existing literature. To improve the ecological validity of the current findings, future studies should expand beyond a college student sample and focus on examining the abovementioned relationships using demographically, culturally, and clinically diverse samples.

REFERENCES

Alapin, I., Fichten, C. S., Libman, E., Creti, L., Bailes, S., & Wright, J. (2000). How is good and poor sleep in older adults and college students related to daytime sleepiness, fatigue, and ability to concentrate? *Journal of Psychosomatic Research*, *49*(5), 381–390. https://doi.org/10.1016/s0022-3999(00)00194-x

American Psychological Association (2014). The road to resilience. Washington, DC: American Psychological Association. http://www.apa.org/helpcenter/road-resilience.aspx

Anders, M. P., Breckenkamp, J., Blettner, M., Schlehofer, B., & Berg-Beckhoff, G. (2014). Association between socioeconomic factors and sleep quality in an urban population-based sample in Germany. *European Journal of Public Health, 24*(6), 968-973. https://doi.org/10.1093/eurpub/ckt175

Anthenelli, R. M., & Maxwell, R. A. (2002). Independent alcohol and tobacco effects on stress axis function. *Alcoholism, Clinical and Experimental Research, 26*(12), 1932-1933. https://doi.org/10.1111/j.1530-0277.2002.tb02508.x

Arnsten A. F. (2011). Catecholamine influences on dorsolateral prefrontal cortical networks. *Biological Psychiatry*, *69*(12), e89–e99. https://doi.org/10.1016/j.biopsych.2011.01.027

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

Bale, T. L., & Epperson, C. N. (2015). Sex differences and stress across the lifespan. *Nature Neuroscience*, *18*(10), 1413–1420. https://doi.org/10.1038/nn.4112

Bangasser, D. A., Eck, S. R., & Ordoñes Sanchez, E. (2019). Sex differences in stress reactivity in arousal and attention systems. *Neuropsychopharmacology: Official Publication of the*

American College of Neuropsychopharmacology, *44*(1), 129–139. https://doi.org/10.1038/s41386-018-0137-2

Barkley, R.A. (2015). Concentration deficit disorder (sluggish cognitive tempo). In R.A. Barkley's Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment (4th ed.) (pp. 435-452). New York, NY: Guilford.

Bartoli, F., Crocamo, C., Alamia, A., Amidani, F., Paggi, E., Pini, E., Clerici, M., & Carrà, G. (2015). Posttraumatic stress disorder and risk of obesity: systematic review and meta-analysis. *The Journal of Clinical Psychiatry*, *76*(10), e1253–e1261. https://doi.org/10.4088/JCP.14r09199

Barzilay, R., Calkins, M. E., Moore, T. M., Wolf, D. H., Satterthwaite, T. D., Cobb Scott, J., Jones, J. D., Benton, T. D., Gur, R. C., & Gur, R. E. (2019). Association between traumatic stress load, psychopathology, and cognition in the Philadelphia Neurodevelopmental Cohort. *Psychological Medicine*, *49*(2), 325–334. https://doi.org/10.1017/S0033291718000880

Bassett, S. M., Lupis, S. B., Gianferante, D., Rohleder, N., & Wolf, J. M. (2015). Sleep quality but not sleep quantity effects on cortisol responses to acute psychosocial stress. *Stress (Amsterdam, Netherlands)*, *18*(6), 638–644. https://doi.org/10.3109/10253890.2015.1087503

Bastien, C. H., Vallières, A., & Morin, C. M. (2001). Validation of the insomnia severity index as an outcome measure for insomnia research. *Sleep Medicine*, *2*(4), 297–307. https://doi.org/10.1016/s1389-9457(00)00065-4

Baum, A., Garofalo, J. P., & Yali, A. M. (1999). Socioeconomic status and chronic stress. Does stress account for SES effects on health? *Annals of the New York Academy of Sciences, 896*, 131–144. https://doi.org/10.1111/j.1749-6632.1999.tb08111.x

Bhat, S., & Chokroverty, S. (2022). Sleep disorders and COVID-19. *Sleep Medicine, 91*, 253–261. https://doi.org/10.1016/j.sleep.2021.07.021

Bonanno, G. A. (2012). Uses and abuses of the resilience construct: Loss, trauma, and health-related adversities. *Social Science Medicine (1982), 74*(5), 753-756. https://doi.org/10.1016/j.socscimed.2011.11.022

Brewin, C. R., Andrews, B., & Valentine, J. D. (2000). Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *Journal of Consulting and Clinical Psychology*, *68*(5), 748–766. https://doi.org/10.1037//0022-006x.68.5.748

Bruce, H. A., Kochunov, P., Chiappelli, J., Savransky, A., Carino, K., Sewell, J., Marshall, W., Kvarta, M., McMahon, F. J., Ament, S. A., Postolache, T. T., O'Connell, J., Shuldiner, A., Mitchell, B., & Hong, L. E. (2021). Genetic versus stress and mood determinants of sleep in the Amish. *American journal of medical genetics. Part B, Neuropsychiatric genetics: the official publication of the International Society of Psychiatric Genetics*, *186*(2), 113–121. https://doi.org/10.1002/ajmg.b.32840

Buboltz, W. C., Brown, F., & Soper, B. (2001). Sleep habits and patterns of college students: A preliminary study. *Journal of American College Health, 50*(3), 131-135. https://doi.org/10.1080/07448480109596017

Buschke, H., & Fuld, P. A. (1974). Evaluating storage, retention, and retrieval in disordered memory and learning. *Neurology*, *24*(11), 1019–1025. https://doi.org/10.1212/wnl.24.11.1019

Buysse, D. J. (2014). Sleep health: can we define it? Does it matter? *Sleep (New York, N.Y.), 37*(1), 9-17. 10.5665/sleep.3298

Buysse, D. J., Reynolds, C. F., 3rd, Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Research, 28*(2), 193–213. https://doi.org/10.1016/0165-1781(89)90047-4

Byrne, M. L., Lind, M. N., Horn, S. R., Mills, K. L., Nelson, B. W., Barnes, M. L., Slavich, G. M., & Allen, N. B. (2021c). Using mobile sensing data to assess stress: Associations with perceived and lifetime stress, mental health, sleep, and inflammation. *Digital Health, 7,* https://doi.org/205520762110372.10.1177/20552076211037227

Cai, Y., Wang, J., & Hou, L. (2021). Resilience improves the sleep quality in disabled elders: The role of perceived stress. *Frontiers in Psychology, 12,* 585816. https://doi.org/10.3389/fpsyg.2021.585816

Campbell-Sills, L., & Stein, M. B. (2007). Psychometric analysis and refinement of the Connor-Davidson resilience scale (CD-RISC): Validation of a 10-item measure of resilience. *Journal of Traumatic Stress, 20*(6), https://doi.org/1019-1028. 10.1002/jts.20271

Carpenter, J., & Andrykowski, M. (1998). Psychometric evaluation of the Pittsburgh Sleep Quality Index. *Journal of Psychosomatic Research*, 45(1), 5-13. https://doi.org/10.1016/S0022-3999(97)00298-5

Castor, N., & El Massioui, F. (2020). Resilience after a neurological pathology: What impact on the cognitive abilities of patients with brain damage? *Neuropsychological Rehabilitation, 30*(5), 853-871. https://doi.org/10.1080/09602011.2018.1512873

Cattell, R. B. (1971). *Abilities: Their structure, growth, and action.* Boston, MA: Houghton Mifflin.

Cazassa, M. J., Oliveira, M. d. S., Spahr, C. M., Shields, G. S., & Slavich, G. M. (2019). The Stress and Adversity Inventory for Adults (Adult STRAIN) in Brazilian Portuguese: Initial validation and links with executive Function, sleep, and mental and physical health. *Frontiers in Psychology*, *10*, 3083. https://doi.org/10.3389/fpsyg.2019.03083

Charles, S. T., Piazza, J. R., Mogle, J., Sliwinski, M. J., & Almeida, D. M. (2013). The wear and tear of daily stressors on mental health. *Psychological Science*, *24*(5), 733-741. https://doi.org/10.1177/0956797612462222

Chaytor, N. S., Barbosa-Leiker, C., Germine, L. T., Fonseca, L. M., Mcpherson, S. M., & Tuttle, K. R. (2020). Construct validity, ecological validity and acceptance of self-administered online neuropsychological assessment in adults. *The Clinical Neuropsychologist*, *35*(1), 148-164. https://doi.org/10.1080/13854046.2020.1811893

Chaytor, N. S., Barbosa-Leiker, C., Germine, L. T., Fonseca, L. M., McPherson, S. M., & Tuttle, K. R. (2021). Construct validity, ecological validity and acceptance of self-administered online neuropsychological assessment in adults. *The Clinical Neuropsychologist*, *35*(1), 148–164. https://doi.org/10.1080/13854046.2020.1811893

Chen, J. C., Espeland, M. A., Brunner, R. L., Lovato, L. C., Wallace, R. B., Leng, X., Phillips, L. S., Robinson, J. G., Kotchen, J. M., Johnson, K. C., Manson, J. E., Stefanick, M. L., Sarto, G. E., & Mysiw, W. J. (2016). Sleep duration, cognitive decline, and dementia risk in older women. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, *12*(1), 21–33. https://doi.org/10.1016/j.jalz.2015.03.004

Chopra, K., Misra, S., & Kuhad, A. (2011). Neurobiological aspects of Alzheimer's disease. *Expert Opinion on Therapeutic Targets, 15*(5), 535–555. https://doi.org/10.1517/14728222.2011.557363

Cohen, S., Frank, E., Doyle, W. J., Skoner, D. P., Rabin, B. S., & Gwatney, J. M. (1998). Types of stressors that increase susceptibility to the common cold in healthy adults. *Health Psychology, 17*(3), 214-223. https://doi.org/10.1037/0278-6133.17.3.214

Connor, K. M., & Davidson, J. R. (2003). Development of a new resilience scale: the Connor-Davidson Resilience Scale (CD-RISC). *Depression and Anxiety*, *18*(2), 76–82. https://doi.org/10.1002/da.10113

Cornelisse, S., van Stegeren, A. H., & Joëls, M. (2011). Implications of psychosocial stress on memory formation in a typical male versus female student sample. *Psychoneuroendocrinology*, *36*(4), 569–578. https://doi.org/10.1016/j.psyneuen.201

Cowan, N. (2014). Working memory underpins cognitive development, learning, and education. *Educational Psychology Review, 26*(2), 197-223. https://doi.org/10.1007/s10648-013-9246-y

Curcio, G., Ferrara, M., & De Gennaro, L. (2006). Sleep loss, learning capacity and academic performance. *Sleep Medicine Reviews*, *10*(5), 323–337. https://doi.org/10.1016/j.smrv.2005.11.001

D'Amico, D., Amestoy, M. E., & Fiocco, A. J. (2020). The association between allostatic load and cognitive function: A systematic and meta-analytic review. *Psychoneuroendocrinology, 121*, 104849. https://doi.org/10.1016/j.psyneuen.2020.104849

Davidson, J.R.T. (n.d). *Connor-Davidson Resilience Scale (CD-RISC) Manual. Unpublished. 08-19-2018, accessible at www.cd-risc.com.*

de Leeuw J. R. (2015). jsPsych: a JavaScript library for creating behavioral experiments in a Web browser. *Behavior Research Methods*, *47*(1), 1–12. https://doi.org/10.3758/s13428-014-0458-y

Deary, I. J., Strand, S., Smith, P., & Fernandes, C. (2007). Intelligence and educational achievement. *Intelligence, 35*(1), 13–21. https://doi.org/10.1016/j.intell.2006.02.001

Dinges, D. F., & Powell, J. W. (1985). Microcomputer analyses of performance on a portable, simple visual RT task during sustained operations. *Behavior Research Methods, Instruments & Computers, 17*(6), 652–655. https://doi.org/10.3758/BF03200977

Du, C., Zan, M. C. H., Cho, M. J., Fenton, J. I., Hsiao, P. Y., Hsiao, R., Keaver, L., Lai, C. C., Lee, H., Ludy, M. J., Shen, W., Swee, W. C. S., Thrivikraman, J., Tseng, K. W., Tseng, W. C., & Tucker, R. M. (2020). Increased resilience weakens the relationship between perceived stress and anxiety on sleep quality: A moderated mediation analysis of higher education students from 7 countries. *Clocks & Sleep, 2*(3), 334–353. https://doi.org/10.3390/clockssleep2030025

Eaves, J. L., & Payne, N. (2019). Resilience, stress and burnout in student midwives. *Nurse Education Today, 79, 188*–193. https://doi.org/10.1016/j.nedt.2019.05.012

ECU by the numbers | facts.ecu.edu | ECU. (n.d.). https://facts.ecu.edu/

Elgh, E., Lindqvist Astot, A., Fagerlund, M., Eriksson, S., Olsson, T., & Näsman, B. (2006). Cognitive dysfunction, hippocampal atrophy and glucocorticoid feedback in Alzheimer's disease. *Biological Psychiatry*, *59*(2), 155–161. https://doi.org/10.1016/j.biopsych.2005.06.017

Evans, G. W., & Schamberg, M. A. (2009). Childhood poverty, chronic stress, and adult working memory. *Proceedings of the National Academy of Sciences - PNAS, 106*(16), 6545-6549. https://doi.org/10.1073/pnas.0811910106

Feenstra, H. E., Vermeulen, I. E., Murre, J. M., & Schagen, S. B. (2017). Online cognition: factors facilitating reliable online neuropsychological test results. *The Clinical Neuropsychologist, 31*(1), 59–84. https://doi.org/10.1080/13854046.2016.1190405

Field, N. P., Classen, C., Butler, L. D., Koopman, C., Zarcone, J., & Spiegel, D. (2001). Revictimization and information processing in women survivors of childhood sexual abuse. *Journal of Anxiety Disorders, 15*(5), 459–469. https://doi.org/10.1016/s0887-6185(01)00076-7.

Follette, V. M., Polusny, M. A., Bechtle, A. E., & Naugle, A. E. (1996). Cumulative trauma: The impact of child sexual abuse, adult sexual assault, and spouse abuse. *Journal of Traumatic Stress, 9*(1), 25-35. https://doi.org/10.1002/jts.2490090104

Fontana, A., & Rosenheck, R. (1998). Psychological benefits and liabilities of traumatic exposure in the war zone. *Journal of Traumatic Stress, 11*(3), 485-503. https://doi.org/.1023/A:1024452612412

Fortenbaugh, F. C., DeGutis, J., Germine, L., Wilmer, J. B., Grosso, M., Russo, K., & Esterman, M. (2015). Sustained attention across the life span in a sample of 10,000: Dissociating ability and strategy. *Psychological Science, 26*(9), 1497-1510. https://doi.org/10.1177/0956797615594896

Gargiulo, A. T., Peterson, L. M., & Grafe, L. A. (2021). Stress, coping, resilience, and sleep during the COVID-19 pandemic: A representative survey study of US adults. *Brain and Behavior, 11*(11), e2384. https://doi.org/10.1002/brb3.2384

Gatchel, J. R., Lopera, F., Norton, D. J., Baena, A., Guzman-Velez, E., Sanchez, J. S., D'oleire Uquillas, F., Schultz, A., Vannini, P., Artola, A., Amariglio, R. E., Rentz, D. M., Tariot, P. N., Reiman, E. M., Johnson, K. A., Sperling, R. A., Marshall, G. A., & Quiroz, Y. T. (2019). Association of subjective cognitive decline with markers of brain pathology in preclinical autosomal dominant Alzheimer's disease. *British Medical Journal* (*BMJ)*. https://doi.org/10.1136/jnnp-2019-321205

Gaultney, J. F. (2010). The prevalence of sleep disorders in college students: Impact on academic performance. *Null*, *59*(2), 91-97. https://doi.org/10.1080/07448481.2010.483708

Germine, L., Nakayama, K., Duchaine, B. C., Chabris, C. F., Chatterjee, G., & Wilmer, J. B. (2012). Is the Web as good as the lab? Comparable performance from Web and lab in cognitive/perceptual experiments. *Psychonomic Bulletin & Review*, *19*(5), 847-857. https://doi.org/10.3758/s13423-012-0296-9

Germine, L., Reinecke, K., & Chaytor, N. S. (2019). Digital neuropsychology: Challenges and opportunities at the intersection of science and software. *The Clinical Neuropsychologist*, *33*(2), 271–286. https://doi.org/10.1080/13854046.2018.1535662

Goldfarb, E. V., Shields, G. S., Daw, N. D., Slavich, G. M., & Phelps, E. A. (2017). Low lifetime stress exposure is associated with reduced stimulus-response memory. *Learning & memory (Cold Spring Harbor, N.Y.)*, *24*(4), 162–168. https://doi.org/10.1101/lm.045179.117

Gooding, P. A., Hurst, A., Johnson, J., & Tarrier, N. (2012). Psychological resilience in young and older adults. *International Journal of Geriatric Psychiatry*, *27*(3), 262–270. https://doi.org/10.1002/gps.2712

Graham, J. E., Christian, L. M., & Kiecolt-Glaser, J. K. (2006). Stress, age, and immune function: Toward a lifespan approach. *Journal of Behavioral Medicine, 29*(4), 389–400. https://doi.org/10.1007/s10865-006-9057-4

Guerra-Carrillo, B., Katovich, K., & Bunge, S. A. (2017). Does higher education hone cognitive functioning and learning efficacy? Findings from a large and diverse sample. *PloS one*, *12*(8), e0182276. https://doi.org/10.1371/journal.pone.0182276

Gunnar, M. R., Frenn, K., Wewerka, S. S., & Van Ryzin, M. J. (2009). Moderate versus severe early life stress: Associations with stress reactivity and regulation in 10–12-year-old children. *Psychoneuroendocrinology, 34*(1), 62-75. https://doi.org/10.1016/j.psyneuen.2008.08.013

Gutwinski, S., Schreiter, S., Priller, J., Henssler, J., Wiers, C. E., & Heinz, A. (2018). Drink and think: Impact of alcohol on cognitive functions and dementia - Evidence of dose-related effects. *Pharmacopsychiatry*, *51*(4), 136–143. https://doi.org/10.1055/s-0043-118664

Hall, M. H., Casement, M. D., Troxel, W. M., Matthews, K. A., Bromberger, J. T., Kravitz, H. M., Krafty, R. T., & Buysse, D. J. (2015). Chronic stress is prospectively associated with sleep in midlife women: The SWAN sleep study. *Sleep*, *38*(10), 1645-54. https://doi.org/10.5665/sleep.5066

Han, K. S., Kim, L., & Shim, I. (2012). Stress and sleep disorder. *Experimental Neurobiology, 21*(4), 141-150. https://doi.org/10.5607/en.2012.21.4.141

Handa, R. J., & McGivern, R. F. (2016). Stress response: Sex differences. In *The Curated Reference Collection in Neuroscience and Biobehavioral Psychology* (pp. 511-517). Elsevier Science Ltd.. https://doi.org/10.1016/B978-0-12-809324-5.02865-0

Hanson, M. D., & Chen, E. (2010). Daily Stress, Cortisol, and Sleep. *Health Psychology*, *29*(4), 394-402. https://doi.org/10.1037/a0019879

Hartshorne, J. K., & Germine, L. T. (2015). When does cognitive functioning peak? The asynchronous rise and fall of different cognitive abilities across the life span. *Psychological Science, 26*(4), 433-443. https://doi.org/10.1177/0956797614567339

Hayes, A. F. (2018). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach* (2nd edition). New York: The Guilford Press.

He, S., Hasler, B. P., & Chakravorty, S. (2019). Alcohol and sleep-related problems. *Current Opinion in Psychology, 30,* 117-122. https://doi.org/10.1016/j.copsyc.2019.03.007

Hirshkowitz, M., Whiton, K., Albert, S. M., Alessi, C., Bruni, O., DonCarlos, L., Hazen, N., Herman, J., Katz, E. S., Kheirandish-Gozal, L., Neubauer, D. N., O'Donnell, A. E., Ohayon, M., Peever, J., Rawding, R., Sachdeva, R. C., Setters, B., Vitiello, M. V., Ware, J. C., & Adams Hillard, P. J. (2015). National Sleep Foundation's sleep time duration recommendations: methodology and results summary. *Sleep Health, 1*(1), 40–43. https://doi.org/10.1016/j.sleh.2014.12.010

Horn, J. L., & Cattell, R. B. (1966). Refinement and test of the theory of fluid and crystallized general intelligences. *Journal of Educational Psychology, 57*(5), 253–270. https://doi.org/10.1037/h0023816

Horning, S., & Davis, H. P. (2012). Aging and cognition. In V. S. Ramachandran (Ed.), *Encyclopedia of Human Behavior (Second Edition)* (pp. 44-52). Academic Press. https://doi.org/10.1016/B978-0-12-375000-6.00007-0

Hosseinichimeh, N., Rahmandad, H., & Wittenborn, A. K. (2015). Modeling the hypothalamus–pituitary–adrenal axis: A review and extension. *Mathematical Biosciences, 268*, 52-65. https://doi.org/10.1016/j.mbs.2015.08.004

Hux, V. J., Roberts, J. M., & Okun, M. L. (2017). Allostatic load in early pregnancy is associated with poor sleep quality. *Sleep Medicine*, *33*, 85–90. https://doi.org/10.1016/j.sleep.2016.09.001

Jike, M., Itani, O., Watanabe, N., Buysse, D. J., & Kaneita, Y. (2018). Long sleep duration and health outcomes: A systematic review, meta-analysis and meta-regression. *Sleep Medicine Reviews*, *39*, 25–36. https://doi.org/10.1016/j.smrv.2017.06.011

Johns M. W. (1991). A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep*, *14*(6), 540–545. https://doi.org/10.1093/sleep/14.6.540

Johns M. W. (1992). Reliability and factor analysis of the Epworth Sleepiness Scale. *Sleep*, *15*(4), 376–381. https://doi.org/10.1093/sleep/15.4.376

Joy, S., Kaplan, E., & Fein, D. (2004). Speed and memory in the WAIS-III Digit Symbol? Coding subtest across the adult lifespan. *Archives of Clinical Neuropsychology*, *19*, 759-767. https://doi.org/10.1016/j.acn.2003.09.009

Juster, R., 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

Kalmbach, D. A., Cuamatzi-Castelan, A. S., Tonnu, C. V., Tran, K. M., Anderson, J. R., Roth, T., & Drake, C. L. (2018). Hyperarousal and sleep reactivity in insomnia: current insights. *Nature and Science of Sleep*, *10*, 193–201. https://doi.org/10.2147/NSS.S138823

Karatsoreos, I. N., Bhagat, S., Bloss, E. B., Morrison, J. H., & McEwen, B. S. (2011). Disruption of circadian clocks has ramifications for metabolism, brain, and behavior. *Proceedings of the National Academy of Sciences - PNAS, 108*(4), 1657-1662. https://doi.org/10.1073/pnas.1018375108

Kaufman, A. S., Kaufman, J. C., Liu, X., & Johnson, C. K. (2009). How do educational attainment and gender relate to fluid intelligence, crystallized intelligence, and academic skills at ages 22–90 years? *Archives of Clinical Neuropsychology, 24*(2), 153–163. https://doi.org/10.1093/arclin/acp015

Kessler, R. C., Andrews, G., Colpe, L. J., Hiripi, E., Mroczek, D. K., Normand, S. L., Walters, E. E., & Zaslavsky, A. M. (2002). Short screening scales to monitor population prevalences and trends in non-specific psychological distress. *Psychological Medicine*, *32*(6), 959–976. https://doi.org/10.1017/s0033291702006074

Keyes, K. M., Hatzenbuehler, M. L., Grant, B. F., & Hasin, D. S. (2012). Stress and alcohol: epidemiologic evidence. *Alcohol Research: Current Reviews*, *34*(4), 391–400.

Kim, E. J., Pellman, B., & Kim, J. J. (2015). Stress effects on the hippocampus: a critical review. *Learning & Memory*, *22*(9), 411–416. https://doi.org/10.1101/lm.037291.114

Kim, E., & Dimsdale, J. E. (2007). The effect of psychosocial stress on sleep: A review of polysomnographic evidence. *Behavioral Sleep Medicine, 5*(4), 256-278. https://doi.org/10.1080/15402000701557383

Kim, H. J., Oh, S. Y., Joo, J. H., Choi, D. W., & Park, E. C. (2019). The relationship between sleep duration and perceived stress: Findings from the 2017 Community Health Survey in Korea. *International Journal of Environmental Research and Public Health, 16*(17), 3208. https://doi.org/10.3390/ijerph16173208

Kim, S., Zhang, W., Pak, V., Aqua, J. K., Hertzberg, V. S., Spahr, C. M., Slavich, G. M., & Bai, J. (2021). How stress, discrimination, acculturation and the gut microbiome affect depression, anxiety and sleep among Chinese and Korean immigrants in the USA: A cross-sectional pilot study protocol. *BMJ Open, 11*(7):e047281. doi: 10.1136/bmjopen-2020-047281

Kobasa, S. C. (1979). Stressful life events, personality, and health: An inquiry into hardiness. *Journal of Personality and Social Psychology, 37*(1), 1-11. 10.1037/0022-3514.37.1.1

Kobrosly, R. W., Seplaki, C. L., Jones, C. M., & van Wijngaarden, E. (2012). Physiologic dysfunction scores and cognitive function test performance in U.S. adults. *Psychosomatic Medicine, 74*(1), 81-88. https://doi.org/10.1097/PSY.0b013e3182385b1e

Koziol, L. F., & Stevens, M. C. (2012). Neuropsychological assessment and the paradox of ADHD. *Applied Neuropsychology. Child, 1*(2), 79–89. https://doi.org/10.1080/2162

Kwon, K. J., Lee, E. J., Kim, M. K., Jeon, S. J., Choi, Y. Y., Shin, C. Y., & Han, S. (2015). The potential role of melatonin on sleep deprivation-induced cognitive impairments: Implication of FMRP on cognitive function. *Neuroscience, 301,* 403-414. https://doi.org/10.1016/j.neuroscience.2015.05.079

Lai, V.D., Theppitak, C., Makizuka, T., Higuchi, Y., Movahed, M., Kumudini, G., Izumi, H., & Kumashiro, M. (2014). A normal intensity level of psycho-physiological stress can benefit working memory performance at high load. *International Journal of Industrial Ergonomics, 44,* 362-367.

Lam, J. C. W., Shields, G. S., Trainor, B. C., Slavich, G. M., & Yonelinas, A. P. (2019). Greater lifetime stress exposure predicts blunted cortisol but heightened DHEA responses to acute stress. *Stress and Health, 35*(1), 15-26. https://doi.org/10.1002/smi.2835

Lee, Y. A., & Goto, Y. (2015). Chronic stress effects on working memory: association with prefrontal cortical tyrosine hydroxylase. *Behavioural Brain Research, 286,* 122–127. https://doi.org/10.1016/j.bbr.2015.03.007

Leipold, B., & Greve, W. (2009). Resilience: A conceptual bridge between coping and development. *European Psychologist, 14*(1), 40–50. https://doi.org/10.1027/1016-9040.14.1.40

Lenzo, V., Sardella, A., Musetti, A., Freda, M. F., Lemmo, D., Vegni, E., Borghi, L., Plazzi, G., Palagini, L., Castelnuovo, G., Cattivelli, R., Mariani, R., Michelini, G., Manari, T., Saita, E., Quattropani, M. C., & Franceschini, C. (2022). The Relationship between resilience and sleep quality during the second wave of the COVID-19 pandemic: A longitudinal study. *Nature and Science of Sleep, 14,* 41–51. https://doi.org/10.2147/NSS.S344042

Linsky, A. S., Straus, M. A., & Colby, J., J P. (1985). Stressful events, stressful conditions and alcohol problems in the United States: a partial test of Bales's theory. *Journal of Studies on Alcohol, 46*(1), 72-80. https://doi.org/10.15288/jsa.1985.46.72

Liston, C., Mcewen, B. S., & Casey, B. J. (2009). Psychosocial stress reversibly disrupts prefrontal processing and attentional control. *Proceedings of the National Academy of Sciences - PNAS, 106*(3), 912-917. https://doi.org/10.1073/pnas.0807041106

Lo Martire, V., Caruso, D., Palagini, L., Zoccoli, G., & Bastianini, S. (2020). Stress & sleep: A relationship lasting a lifetime. *Neuroscience and Biobehavioral Reviews, 117,* 65-77. https://doi.org/10.1016/j.neubiorev.2019.08.024

Lövdén, M., Fratiglioni, L., Glymour, M. M., Lindenberger, U., & Tucker-Drob, E. M. (2020). Education and cognitive functioning across the life span. *Psychological Science in the Public Interest: A Journal of the American Psychological Society, 21*(1), 6–41. https://doi.org/10.1177/1529100620920576

Lucassen, P. J., Pruessner, J., Sousa, N., Almeida, O. F., Van Dam, A. M., Rajkowska, G., Swaab, D. F., & Czéh, B. (2014). Neuropathology of stress. *Acta Neuropathologica*, *127*(1), 109–135. https://doi.org/10.1007/s00401-013-1223-5

Lund, H. G., Reider, B. D., Whiting, A. B., & Prichard, J. R. (2010). Sleep patterns and predictors of disturbed sleep in a large population of college students. *The Journal of adolescent health: official publication of the Society for Adolescent Medicine*, *46*(2), 124–132. https://doi.org/10.1016/j.jadohealth.2009.06.016

Lupien, S. J., Gaudreau, S., Tchiteya, B. M., Maheu, F., Sharma, S., Nair, N. P. V., Hauger, R. L., McEwen, B. S., & Meaney, M. J. (1997). Stress-induced declarative memory impairment in healthy elderly subjects: Relationship to cortisol reactivity. *The Journal of Clinical Endocrinology and Metabolism, 82*(7), 2070-2075. https://doi.org/10.1210/jc.82.7.2070

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

Luthar, S. S., Cicchetti, D., & Becker, B. (2000). The construct of resilience: a critical evaluation and guidelines for future work. *Child Development, 71*(3), 543–562. https://doi.org/10.1111/1467-8624.00164

Lyons, J. A. (1991). Strategies for assessing the potential for positive adjustment following trauma. *Journal of Traumatic Stress, 4*(1), 93-111. https://doi.org/10.1002/jts.2490040108

Lyu, J., & Burr, J. A. (2016). Socioeconomic status across the life course and cognitive function among older adults. *Journal of Aging and Health, 28*(1), 40-67. https://doi.org/10.1177/0898264315585504

Mackenzie, C. S., Wiprzycka, U. J., Hasher, L., & Goldstein, D. (2009). Associations between psychological distress, learning, and memory in spouse caregivers of older adults. *The Journals of Gerontology. Series B, Psychological Sciences and Social Sciences*, *64*(6), 742–746. https://doi.org/10.1093/geronb/gbp076

Macklin, M. L., Metzger, L. J., Litz, B. T., McNally, R. J., Lasko, N. B., Orr, S. P., & Pitman, R. K. (1998). Lower precombat intelligence is a risk factor for posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology, 66*(2), 323-326. https://doi.org/10.1037/0022-006X.66.2.323

Maher, M. J., Rego, S. A., & Asnis, G. M. (2006). Sleep disturbances in patients with post-traumatic stress disorder: epidemiology, impact and approaches to management. *CNS drugs*, *20*(7), 567–590. https://doi.org/10.2165/00023210-200620070-00003

Marin, M., Lord, C., Andrews, J., Juster, R., Sindi, S., Arsenault-Lapierre, G., Fiocco, A. J., & Lupien, S. J. (2011). Chronic stress, cognitive functioning and mental

health. *Neurobiology of Learning and Memory, 96*(4), 583-595. https://doi.org/10.1016/j.nlm.2011.02.016

Mariotti, A. (2015). The effects of chronic stress on health: new insights into the molecular mechanisms of brain–body communication. *Future Science OA, 1*(3), FSO23. https://doi.org/10.4155/fso.15.21

Markowitz, D. A., Curtis, C. E., & Pesaran, B. (2015). Multiple component networks support working memory in prefrontal cortex. *Proceedings of the National Academy of Sciences - PNAS, 112*(35), 11084-11089. https://doi.org/10.1073/pnas.1504172112

Maslow A.H. (1943) A theory of human motivation, *Psychol Rev*, *50*(4), 370–96.

McCormick, C. M., & Green, M. R. (2013). From the stressed adolescent to the anxious and depressed adult: Investigations in rodent models. *Neuroscience, 249*, 242-257. https://doi.org/10.1016/j.neuroscience.2012.08.063

McCormick, C. M., & Green, M. R. (2013). From the stressed adolescent to the anxious and depressed adult: Investigations in rodent models. *Neuroscience, 249*, 242-257. https://10.1016/j.neuroscience.2012.08.063

McEwen B. S. (1998). Protective and damaging effects of stress mediators. *The New England journal of Medicine, 338*(3), 171–179. https://doi.org/10.1056/NEJM199801153380307

McEwen B. S. (2000). Allostasis and allostatic load: implications for neuropsychopharmacology. *Neuropsychopharmacology: Official Publication of the American College of Neuropsychopharmacology*, *22*(2), 108–124. https://doi.org/10.1016/S0893-133X(99)00129-3

McEwen, B. S. (2004). Protection and damage from acute and chronic stress: Allostasis and allostatic overload and relevance to the pathophysiology of psychiatric disorders. *Annals*

of the New York Academy of Sciences, 1032(1), 1-7. https://doi.org/10.1196/annals.1314.001

McEwen, B. S. (2019). The good side of stress. *Stress (Amsterdam, Netherlands), 22*(5), 524-525. https://doi.org/10.1080/10253890.2019.1631794

McEwen, B. S., & Akil, H. (2020). Revisiting the stress concept: Implications for affective disorders. *The Journal of Neuroscience, 40*(1), 12-21. https://doi.org/10.1523/JNEUROSCI.0733-19.2019

McEwen, B. S., & Sapolsky, R. M. (1995). Stress and cognitive function. *Current Opinion in Neurobiology*, *5*(2), 205–216. https://doi.org/10.1016/0959-4388(95)80028-x

McEwen, B. S., PhD, & Karatsoreos, I. N., PhD. (2015). Sleep deprivation and circadian disruption. *Sleep Medicine Clinics, 10*(1), https://doi.org/1-10. 10.1016/j.jsmc.2014.11.007

McLoughlin, E., Arnold, R., Fletcher, D., Spahr, C. M., Slavich, G. M., & Moore, L. J. (2022). Assessing lifetime stressor exposure in sport performers: Associations with trait stress appraisals, health, well-being, and performance. *Psychology of Sport and Exercise*, *58*, 102078. https://doi.org/10.1016/j.psychsport.2021.102078

Medic, G., Wille, M., & Hemels, M. E. (2017). Short- and long-term health consequences of sleep disruption. *Nature and Science of Sleep*, *9*, 151–161. https://doi.org/10.2147/NSS.S134864

Memory: A Comprehensive Reference, 2nd edition, Byrne, J.H. (ed.), 7–19.

Meng, X., & D'Arcy, C. (2012). Education and dementia in the context of the cognitive reserve hypothesis: a systematic review with meta-analyses and qualitative analyses. *PloS one*, *7*(6), e38268. https://doi.org/10.1371/journal.pone.0038268

Metz, S., Aust, S., Fan, Y., Bönke, L., Harki, Z., Gärtner, M., Bajbouj, M., & Grimm, S. (2018). The influence of early life stress on the integration of emotion and working memory. *Behavioural Brain Research, 339,* 179–185. https://doi.org/10.1016/j.bbr.2017.11.022 model. *Health Education, 21*, 33-39.

Moore, P. J., Adler, N. E., Williams, D. R., & Jackson, J. S. (2002). Socioeconomic status and health: The role of sleep. *Psychosomatic Medicine, 64*(2), 337-344. https://doi.org/10.1097/00006842-200203000-00018

Morin, C. M., Rodrigue, S., & Ivers, H. (2003). Role of Stress, Arousal, and Coping Skills in Primary Insomnia. *Psychosomatic Medicine, 65*(2), 259-267. https://doi.org/10.1097/01.PSY.0000030391.09558.A3

Muscatell, K., Slavich, G., Monroe, S., & Gotlib, I. (2009). Stressful life events, chronic difficulties, and the symptoms of clinical depression. *The Journal of Nervous and Mental Disease, 197*(3), 154-160. https://doi.org/10.1097/NMD.0b013e318199f77b

Nixon, R.D., Nishith, P. and Resick, P.A. (2004) The accumulative effect of trauma exposure on short-term and delayed verbal memory in a treatment-seeking sample of female rape victims. *Journal of Traumatic Stress, 17,* 31-35. https://doi.org/10.1023/B:JOTS.0000014673.02925.db

Ottino-González, J., Jurado, M. A., García-García, I., Caldú, X., Prats-Soteras, X., Tor, E., Sender-Palacios, M. J., & Garolera, M. (2019). Allostatic load and executive functions in overweight adults. *Psychoneuroendocrinology, 106,* 165–170. https://doi.org/10.1016/j.psyneuen.2019.04.009

Ottino-González, J., Jurado, M. A., García-García, I., Segura, B., Marqués-Iturria, I., Sender-Palacios, M. J., Tor, E., Prats-Soteras, X., Caldú, X., Junqué, C., Pasternak, O., &

Garolera, M. (2018). Allostatic load and disordered white matter microstructure in overweight adults. *Scientific Reports, 8*(1), 15898. https://doi.org/10.1038/s41598-018-34219-8

Palagini, L., Biber, K., & Riemann, D. (2014). The genetics of insomnia--evidence for epigenetic mechanisms? *Sleep Medicine Reviews, 18*(3), 225–235. https://doi.org/10.1016/j.smrv.2013.05.002

Palagini, L., Miniati, M., Marazziti, D., Franceschini, C., Zerbinati, L., Grassi, L., Sharma, V., & Riemann, D. (2022). Insomnia symptoms are associated with impaired resilience in bipolar disorder: Potential links with early life stressors may affect mood features and suicidal risk. *Journal of Affective Disorders, 299, 596*–603.

Palagini, L., Moretto, U., Novi, M., Masci, I., Caruso, D., Drake, C. L., & Riemann, D. (2018). Lack of resilience is related to stress-related sleep reactivity, hyperarousal, and emotion dysregulation in insomnia disorder. *Journal of Clinical Sleep Medicine (JCSM): Official Publication of the American Academy of Sleep Medicine, 14*(5), 759–766.

Parker, K. J., Buckmaster, C. L., Sundlass, K., Schatzberg, A. F., & Lyons, D. M. (2006). Maternal mediation, Stress inoculation, and the development of neuroendocrine stress resistance in primates. *Proceedings of the National Academy of Sciences - PNAS, 103*(8), 3000-3005. https://doi.org/10.1073/pnas.0506571103

Parslow, R. A., & Jorm, A. F. (2007). Pretrauma and posttrauma neurocognitive functioning and PTSD symptoms in a community sample of young adults. *The American Journal of Psychiatry, 164*(3), 509-515. https://doi.org/10.1176/ajp.2007.164.3.509

Passell, E., Dillon, D. G., Baker, J. T., Vogel, S. C., Scheuer, L. S., Mirin, N. L., ... & Germine, L. (2019). Digital cognitive assessment: Results from the TestMyBrain NIMH Research Domain Criteria (RDoC) field test battery report.

Pillai, V., Roth, T., Mullins, H. M., & Drake, C. L. (2014). Moderators and mediators of the relationship between stress and insomnia: stressor chronicity, cognitive intrusion, and coping. *Sleep*, *37*(7), 1199-1208A.

Raison, C. L., & Miller, A. H. (2003). When not enough is too much: The role of insufficient glucocorticoid signaling in the pathophysiology of stress-related disorders. *The American Journal of Psychiatry*, *160*(9), 1554-1565. https://doi.org/10.1176/appi.ajp.160.9.1554

Richardson, G. E. (2002). The metatheory of resilience and resiliency. *Journal of Clinical Psychology*, *4*(4), 307-321. https://doi.org/10.1002/jclp.10020

Richardson, G. E., Neiger, B., Jensen, S., & Kumpfer, K. (1990). The resiliency

Roediger III, H.L., Zaromb, F.M., Lin, W. (2017) A Typology of memory terms. In:

Rohleder, N. (2019). Stress and inflammation – The need to address the gap in the transition between acute and chronic stress effects. *Psychoneuroendocrinology, 105*, 164-171. https://doi.org/10.1016/j.psyneuen.2019.02.021

Rosnick, C. B., Small, B. J., McEvoy, C. L., Borenstein, A. R., & Mortimer, J. A. (2007). Negative life events and cognitive performance in a population of older adults. *Journal of Aging and Health*, *19*(4), 612-629. https://doi.org/10.1177/0898264307300975

Rutter M. (1985). Resilience in the face of adversity. Protective factors and resistance to psychiatric disorder. *The British Journal of Psychiatry: The Journal of Mental Science*, *147*, 598–611. https://doi.org/10.1192/bjp.147.6.598

Rutter, L. A., Vahia, I. V., Forester, B. P., Ressler, K. J., & Germine, L. (2020). Heterogeneous indicators of cognitive performance and performance variability across the lifespan. *Frontiers in Aging Neuroscience*, *12*, 62. https://doi.org/10.3389/fnagi

Sandi C. (2013). Stress and cognition. *Wiley Interdisciplinary Reviews. Cognitive Science*, *4*(3), 245–261. https://doi.org/10.1002/wcs.1222

Sateia M. J. (2014). International classification of sleep disorders-third edition: highlights and modifications. *Chest*, *146*(5), 1387–1394. https://doi.org/10.1378/chest.14-097

Schat, A. C. H., Kelloway, E. K., & Desmarais, S. (2005). The Physical Health Questionnaire (PHQ): construct validation of a self-report scale of somatic symptoms. *Journal of Occupational Health Psychology*, *10*(4), 363–381. https://doi.org/10.1037/1076-8998.10.4.363

Schneider, W. J., & McGrew, K. S. (2012). The Cattell-Horn-Carroll model of intelligence. In D. P. Flanagan & P. L. Harrison (Eds.), *Contemporary Intellectual Assessment: Theories, Tests, and Issues* (pp. 99–144). The Guilford Press.

Schneiderman, N., Ironson, G., & Siegel, S. D. (2005). Stress and health: Psychological, behavioral, and biological determinants. *Annual Review of Clinical Psychology*, *1*(1), 607-628. https://doi.org/ 10.1146/annurev.clinpsy.1.102803.144141

Schuitevoerder, S., Rosen, J. W., Twamley, E. W., Ayers, C. R., Sones, H., Lohr, J. B., Goetter, E. M., Fonzo, G. A., Holloway, K. J., & Thorp, S. R. (2013). A meta-analysis of cognitive functioning in older adults with PTSD. *Journal of Anxiety Disorders, 27*(6), 550-558. https://doi.org/10.1016/j.janxdis.2013.01.001

Scott, J. C., Matt, G. E., Wrocklage, K. M., Crnich, C., Jordan, J., Southwick, S. M., Krystal, J. H., & Schweinsburg, B. C. (2015). A Quantitative meta-analysis of neurocognitive

functioning in posttraumatic stress disorder. *Psychological Bulletin, 141*(1), 105-140. https://doi.org/10.1037/a0038039

Seelig, A. D., Jacobson, I. G., Donoho, C. J., Trone, D. W., Crum-Cianflone, N. F., & Balkin, T. J. (2016). Sleep and health resilience metrics in a large military cohort. *Sleep, 39*(5), 1111–1120. https://doi.org/10.5665/sleep.5766

Seery, M. D., Holman, E. A., & Silver, R. C. (2010). Whatever does not kill us: Cumulative lifetime adversity, vulnerability, and resilience. *Journal of Personality and Social Psychology, 99*(6), https://doi.org/1025-1041. 10.1037/a0021344

Seery, M. D., Leo, R. J., Lupien, S. P., Kondrak, C. L., & Almonte, J. L. (2013). An upside to adversity?: moderate cumulative lifetime adversity is associated with resilient responses in the face of controlled stressors. *Psychological Science, 24*(7), 1181–1189. https://doi.org/10.1177/0956797612469210

Selye, H. (1976). Stress without distress. In *Psychopathology of human adaptation* (pp. 137-146). Springer, Boston, MA.

Senft Miller, A., Nop, O., Slavich, G. M., & Dumas, J. A. (2022). Lifetime stress exposure, cognition, and psychiatric wellbeing in women. *Aging & mental health, 26*(9), 1765–1770. https://doi.org/10.1080/13607863.2021.1958144

Shan, Z., Ma, H., Xie, M., Yan, P., Guo, Y., Bao, W., Rong, Y., Jackson, C. L., Hu, F. B., & Liu, L. (2015). Sleep duration and risk of type 2 diabetes: a meta-analysis of prospective studies. *Diabetes Care, 38*(3), 529-537. https://doi.org/10.2337/dc14-2073

Sherin, J. E., & Nemeroff, C. B. (2011). Post-traumatic stress disorder: the neurobiological impact of psychological trauma. *Dialogues in Clinical Neuroscience, 13*(3), 263–278. https://doi.org/10.31887/DCNS.2011.13.2/jsherin

Shields, G. S., & Slavich, G. M. (2017). Lifetime stress exposure and health: A review of contemporary assessment methods and biological mechanisms. *Social and Personality Psychology Compass, 11*(8), https://doi.org/e12335-n/a. 10.1111/spc3.12335

Shields, G. S., Ramey, M. M., Slavich, G. M., & Yonelinas, A. P. (2019). Determining the mechanisms through which recent life stress predicts working memory impairments: precision or capacity? *Stress (Amsterdam, Netherlands), 22*(2), 280–285. https://doi.org/10.1080/10253890.2018.1556635

Shields, G. S., Sazma, M. A., McCullough, A. M., & Yonelinas, A. P. (2017). The effects of acute stress on episodic memory: A meta-analysis and integrative review. *Psychological Bulletin, 143*(6), 636-675. https://doi.org/10.1037/bul0000100

Singh, S., Strong, R. W., Jung, L., Li, F. H., Grinspoon, L., Scheuer, L. S., Passell, E. J., Martini, P., Chaytor, N., Soble, J. R., & Germine, L. (2021). The TestMyBrain Digital Neuropsychology Toolkit: Development and psychometric characteristics. *Journal of Clinical and Experimental Neuropsychology, 43*(8), 786–795. https://doi.org/10.1080/13803395.2021.2002269

Slavich, G. M. (2016). Life stress and health. *Teaching of Psychology, 43*(4), 346-355. https://doi.org/10.1177/0098628316662768

Slavich, G. M., & Epel, E. S. (2010). The Stress and Adversity Inventory (STRAIN): An automated system for assessing cumulative stress exposure. *Los Angeles: University of California, Los Angeles*.

Slavich, G. M., & Shields, G. S. (2018). Assessing lifetime stress exposure using the Stress and Adversity Inventory for Adults (Adult STRAIN): An overview and initial

validation. *Psychosomatic Medicine, 80*(1), 17-27. https://doi.org 10.1097/PSY.0000000000000534

Sotiropoulos, I., Catania, C., Pinto, L. G., Silva, R., Pollerberg, G. E., Takashima, A., Sousa, N., & Almeida, O. F. (2011). Stress acts cumulatively to precipitate Alzheimer's disease-like tau pathology and cognitive deficits. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, *31*(21), 7840–7847. https://doi.org/10.1523/JNEUROSCI.0730-11.2011

Sotiropoulos, I., Catania, C., Pinto, L. G., Silva, R., Pollerberg, G. E., Takashima, A., Sousa, N., & Almeida, O. F. X. (2011a). Stress acts cumulatively to precipitate Alzheimer's disease-like tau pathology and cognitive deficits. *The Journal of Neuroscience, 31*(21), 7840-7847. https://doi.org/10.1523/JNEUROSCI.0730-11.2011

Southwick, S. M., Bonanno, G. A., Masten, A. S., Panter-Brick, C., & Yehuda, R. (2014). Resilience definitions, theory, and challenges: interdisciplinary perspectives. *Null, 5*(1), 25338. https://doi.org/10.3402/ejpt.v5.25338

Spencer-Segal, J. L., & Akil, H. (2019). Glucocorticoids and resilience. *Hormones and Behavior*, *111*, 131–134. https://doi.org/10.1016/j.yhbeh.2018.11.005

Spielman, A. J., Caruso, L. S., & Glovinsky, P. B. (1987). A behavioral perspective on insomnia treatment. *Psychiatric Clinics of North America, 10*(4), 541–553.

Stauble, M. R., Thompson, L. A., & Morgan, G. (2013). Increases in cortisol are positively associated with gains in encoding and maintenance working memory performance in young men. *Stress (Amsterdam, Netherlands), 16*(4), 402–410. https://doi.org/10.3109/10253890.2013.780236

Steiger, A. (2003), Sleep and endocrinology. *Journal of Internal Medicine, 254*, 13-22. https://doi.org/10.1046/j.1365-2796.2003.01175.x

Stephen Joy, Edith Kaplan, Deborah Fein, Speed and memory in the WAIS-III Digit Symbol—Coding subtest across the adult lifespan, *Archives of Clinical Neuropsychology*, Volume 19, Issue 6, September 2004, Pages 759–767, https://doi.org/10.1016/j.acn.2003.09.009

Studart, A., Neto, & Nitrini, R. (2016). Subjective cognitive decline: The first clinical manifestation of Alzheimer's disease? *Dementia & Neuropsychologia*, *10*(3), 170–177. https://doi.org/10.1590/S1980-5764-2016DN1003002

Suliman, S., Mkabile, S. G., Fincham, D. S., Ahmed, R., Stein, D. J., & Seedat, S. (2009a). Cumulative effect of multiple trauma on symptoms of posttraumatic stress disorder, anxiety, and depression in adolescents. *Comprehensive Psychiatry, 50*(2), 121-127. https://doi.org/10.1016/j.comppsych.2008.06.006

Sumner, J. A., Hagan, K., Grodstein, F., Roberts, A. L., Harel, B., & Koenen, K. C. (2017). Posttraumatic stress disorder symptoms and cognitive function in a large cohort of middle-aged women. *Depression and Anxiety*, *34*(4), 356–366. https://doi.org/10.1002/da.22600

Sun, Y., Pan, W., Zhang, Y., Xu, G., Xi, J., Bao, Q., & Bian, X. (2021). The relationship between stress, resilience, and quality of life in Chinese high school students. *Annals of Palliative Medicine*, *10*(5), 5483–5493. https://doi.org/10.21037/apm-21-929

Taylor, D. J., Vatthauer, K. E., Bramoweth, A. D., Ruggero, C., & Roane, B. (2013). The role of sleep in predicting college academic performance: is it a unique predictor? *Behavioral Sleep Medicine*, *11*(3), 159–172. https://doi.org/10.1080/15402002.2011.602776

The Many Brains Project. (n.d.). The Many Brains Project. Retrieved June 24, 2023, from https://www.manybrains.net/

Tiemeier, H., Pelzer, E., Jönck, L., Möller, H., & Rao, M. L. (2002). Plasma catecholamines and selective slow wave sleep deprivation. *Neuropsychobiology, 45*(2), 81-86. https://doi.org/10.1159/000048681

Tobaldini, E., Costantino, G., Solbiati, M., Cogliati, C., Kara, T., Nobili, L., & Montano, N. (2017). Sleep, sleep deprivation, autonomic nervous system and cardiovascular diseases. *Neuroscience and Biobehavioral Reviews, 74*(Pt B), 321-329. https://doi.org/10.1016/j.neubiorev.2016.07.004

Tourunen, A., Siltanen, S., Saajanaho, M., Koivunen, K., Kokko, K., & Rantanen, T. (2021). Psychometric properties of the 10-item Connor-Davidson resilience scale among Finnish older adults. *Aging & Mental Health, 25*(1), 99–106. https://doi.org/10.1080/13607863.2019.1683812

Toussaint, L., Shields, G. S., Dorn, G., & Slavich, G. M. (2016). Effects of lifetime stress exposure on mental and physical health in young adulthood: How stress degrades and forgiveness protects health. *Journal of Health Psychology, 21*(6), 1004–1014. https://doi.org/10.1177/1359105314544132

Tsapanou, A., Vlachos, G. S., Cosentino, S., Gu, Y., Manly, J. J., Brickman, A. M., Schupf, N., Zimmerman, M. E., Yannakoulia, M., Kosmidis, M. H., Dardiotis, E., Hadjigeorgiou, G., Sakka, P., Stern, Y., Scarmeas, N., & Mayeux, R. (2019). Sleep and subjective cognitive decline in cognitively healthy elderly: Results from two cohorts. *Journal of Sleep Research*, *28*(5), e12759. https://doi.org/10.1111/jsr.12759

Van Reeth, O., Weibel, L., Spiegel, K., Leproult, R., Dugovic, C., & Maccari, S. (2000). Physiology sleep (Review) - Interactions between stress and sleep: from basic research to clinical situations. *Sleep Medicine Reviews, 4*(2), 201-219. https://doi.org/10.1053/smrv.1999.0097

Vanderbilt-Adriance, E., & Shaw, D. S. (2008). Conceptualizing and re-evaluating resilience across levels of risk, time, and domains of competence. *Clinical Child and Family Psychology Review, 11*(1-2), 30-58. https://doi.org/10.1007/s10567-008-0031-2

Vasterling, J. J., Duke, L. M., Brailey, K., Constans, J. I., Allain, A. N., Jr., & Sutker, P. B. (2002). Attention, learning, and memory performances and intellectual resources in Vietnam veterans: PTSD and no disorder comparisons. *Neuropsychology, 16*(1), 5–14. https://doi.org/10.1037/0894-4105.16.1.5

Vellutino, F. R., Steger, J. A., DeSetto, L., & Phillips, F. (1975). Immediate and delayed recognition of visual stimuli in poor and normal readers. *Journal of Experimental Child Psychology, 19*(2), 223-232. https://doi.org/https://doi.org/10.1016/0022-0965(75)90086-7

Vgontzas, A. N., Bixler, E. O., Lin, H. M., Prolo, P., Mastorakos, G., Vela-Bueno, A., Kales, A., & Chrousos, G. P. (2001). Chronic insomnia is associated with nyctohemeral activation of the hypothalamic-pituitary-adrenal axis: clinical implications. *The Journal of Clinical Endocrinology and Metabolism*, *86*(8), 3787–3794. https://doi.org/10.1210/jcem.86.8.7778

Vogel, S. C., Esterman, M., DeGutis, J., Wilmer, J. B., Ressler, K. J., & Germine, L. T. (2020). Childhood adversity and dimensional variations in adult sustained attention. *Frontiers in Psychology*, *11*, 691. https://doi.org/10.3389/fpsyg.2020.00691

Vyas, S., Rodrigues, A. J., Silva, J. M., Tronche, F., Almeida, O. F. X., Sousa, N., & Sotiropoulos, I. (2016). Chronic stress and glucocorticoids: From neuronal plasticity to neurodegeneration. *Neural Plasticity, 2016*, 6391686-15. https://doi.org/10.1155/2016/6391686

Watson, N. F., Badr, M. S., Belenky, G., Bliwise, D. L., Buxton, O. M., Buysse, D., Dinges, D. F., Gangwisch, J., Grandner, M. A., Kushida, C., Malhotra, R. K., Martin, J. L., Patel, S. R., Quan, S. F., & Tasali, E. (2015). Recommended amount of sleep for a healthy adult: A Joint consensus statement of the American Academy of Sleep Medicine and Sleep Research Society. *Sleep (New York, N.Y.), 38*(6), 843-844. https://doi.org/10.5665/sleep.4716

Wechsler, D. (1955). *Manual for the Wechsler Adult Intelligence Scale.* Psychological Corp. San Antonio, TX: Pearson.

Wechsler, D. (1987). *Manual for the Wechsler Memory Scale-Revised*, The Psychological Corporation, San Antonio, TX.

Wechsler, D. (2008). *Wechsler adult intelligence scale–Fourth Edition (WAIS–IV).* San Antonio, TX: Pearson.

Weiner, M. F., Vobach, S., Olsson, K., Svetlik, D., & Risser, R. C. (1997). Cortisol secretion and Alzheimer's disease progression. *Biological Psychiatry*, *42*(11), 1030–1038. https://doi.org/10.1016/s0006-3223(97)00165-0

Wilmer, J. B., Germine, L., Chabris, C. F., Chatterjee, G., Gerbasi, M., & Nakayama, K. (2012). Capturing specific abilities as a window into human individuality: the example of face recognition. *Cognitive Neuropsychology*, *29*(5-6), 360–392. https://doi.org/10.1080/02643294.2012.753433

Wilson, R. S. (2008). Neurological factors in cognitive aging. In S. M. Hofer & D. F. Alwin (Eds.), *Handbook of Cognitive Aging: Interdisciplinary Perspectives* (pp. 298–307). Sage Publications, Inc. https://doi.org/10.4135/9781412976589.n18

Wilson, R. S., Evans, D. A., Bienias, J. L., Mendes de Leon, C. F, Schneider J. A., & Bennet, D. A. (2003). Proneness to psychological distress is associated with risk of Alzheimer's disease. *Neurology, 61*(11), 1479-1485. https://doi.org/10.1212/01.WNL.0000096167.56734.59

Windle, G. (2011). What is resilience? A review and concept analysis. *Reviews in Clinical Gerontology, 21*(2), 152-169. https://doi.org/10.1017/S0959259810000420

Wingo, A. P., Fani, N., Bradley, B., & Ressler, K. J. (2010). Psychological resilience and neurocognitive performance in a traumatized community sample. *Depression and Anxiety, 4*(8), 768-774. https://doi.org/10.1002/da.20675

Wolf, D., Fischer, F. U., Fellgiebel, A., & Alzheimer's Disease Neuroimaging Initiative (2019). Impact of resilience on the association between Amyloid-β and longitudinal cognitive decline in cognitively healthy older adults. *Journal of Alzheimer's Disease: JAD*, *70*(2), 361–370. https://doi.org/10.3233/JAD-190370

Wright, C. D., Tiani, A. G., Billingsley, A. L., Steinman, S. A., Larkin, K. T., & McNeil, D. W. (2019). A Framework for understanding the role of psychological processes in disease development, maintenance, and treatment: The 3P-Disease Model. *Frontiers in Psychology*, *10*, 2498. https://doi.org/10.3389/fpsyg.2019.02498

Yang, C., Chen, A., & Chen, Y. (2021). College students' stress and health in the COVID-19 pandemic: The role of academic workload, separation from school, and fears of contagion. *PloS One*, *16*(2), e0246676. https://doi.org/10.1371/journal.pone.0246676

Yap, Y., Slavish, D. C., Taylor, D. J., Bei, B., & Wiley, J. F. (2020). Bi-directional relations between stress and self-reported and actigraphy-assessed sleep: A daily intensive longitudinal study. *Sleep (New York, N.Y.), 43*(3), 1. https://doi.org/10.1093/sleep/zsz250

Young-McCaughan S., Peterson A.L., Bingham M.O. (2018) The role of sleep in the health and resiliency of military personnel. In: Vermetten E., Germain A., Neylan T. (eds) *Sleep and Combat-Related Post Traumatic Stress Disorder*. Springer, New York, NY. https://doi.org/10.1007/978-1-4939-7148-0_5

Yu, J. T., Xu, W., Tan, C. C., Andrieu, S., Suckling, J., Evangelou, E., Pan, A., Zhang, C., Jia, J., Feng, L., Kua, E. H., Wang, Y. J., Wang, H. F., Tan, M. S., Li, J. Q., Hou, X. H., Wan, Y., Tan, L., Mok, V., Tan, L., … Vellas, B. (2020). Evidence-based prevention of Alzheimer's disease: systematic review and meta-analysis of 243 observational prospective studies and 153 randomized controlled trials. *Journal of Neurology, Neurosurgery, and Psychiatry, 91*(11), 1201–1209. https://doi.org/10.1136/jnnp-2019-321913

Yu, M., Hazelton, W. D., Luebeck, G. E., & Grady, W. M. (2019). Epigenetic aging: More than just a clock when it comes to cancer. *American Association for Cancer Research, 80*(3), 367-374. https://doi.org/10.1158/0008-5472.can-19-0924

Zannas, A. S., Arloth, J., Carrillo-Roa, T., Iurato, S., Röh, S., Ressler, K. J., Nemeroff, C. B., Smith, A. K., Bradley, B., Heim, C., Menke, A., Lange, J. F., Brückl, T., Ising, M., Wray, N. R., Erhardt, A., Binder, E. B., & Mehta, D. (2015). Lifetime stress accelerates epigenetic aging in an urban, African American cohort: Relevance of glucocorticoid signaling. *Genome Biology, 16*(1), 266. https://doi.org/10.1186/s13059-015-0828-5

Zhai, L., Zhang, H., & Zhang, D. (2015). Sleep duration and depression among adults: A meta-analysis of prospective studies. *Depression and Anxiety, 32*(9), 664–670. https://doi.org/10.1002/da.22386

Zhang, Q., Song, D., Liu, Y., Chang, L., Li, C., & Li, Y. (2022). Sleep quality, caregiver burden, and individual resilience among parents of children with epilepsy. *Epilepsy & Behavior: E&B, 135*, 108873. https://doi.org/10.1016/j.yebeh.2022.108873

Zhou, S., Chen, J., Lin, H., Ye, Y., Xiao, Y., Ouyang, N., Pan, S., Feng, S., Xie, M., & Li, B. (2022). Associations among workplace bullying, resilience, insomnia severity, and subjective wellbeing in Chinese resident doctors. *Frontiers in Psychiatry, 13*, 840945. https://doi.org/10.3389/fpsyt.2022.840945

APPENDIX A: TABLES

Table 1

Demographics of Study Participants (N= 153)

Variable	*n*	%
Race		
American Indian/Alaska Native	1	0.7
Asian	5	3.3
Black/African American	31	20.3
European American/White	101	66.0
Multi-racial	10	6.5
Other	5	3.3
Ethnicity		
Hispanic/LatinX	24	15.7
Not Hispanic/LatinX	129	84.3
Sex/Gender		
Female/Woman	100	65.4
Male/Man	52	34.0
Transgender/Gender queer/Non-binary	1	0.7
Mothers' Education		
12 Years or Less	10	6.5
High School Graduate/GED	17	11.1
Some College/AA degree	21	13.7
College Graduate (Bachelor's)	63	41.2
Master's Degree or Beyond	42	27.5
Fathers' Education		
12 Years or Less	14	9.2
High School Graduate/GED	32	20.9
Some College/AA degree	31	20.3
College Graduate (Bachelor's)	53	34.6
Master's Degree or Beyond	22	14.4

Table 2

Main Study Variables Descriptive Statistics

Variable	*n*	*M*	*SD*	Min	Max	A	Skew	Kurtosis
Cumulative Lifetime Stress	147	15.91	9.68	0.00	42.00	-	0.74	-0.11
Cognitive Measures								
Forward Digit Span	149	6.13	1.25	4.00	10.00	-	0.65	0.07
Backward Digit Span	150	5.00	1.47	1.00	9.00	-	0.19	-0.25
Digit Symbol Matching	147	52.73	9.70	33.00	79.00	-	0.68	0.14
Verbal Paired Associates	145	16.75	4.90	5.00	25.00	-	-0.54	-0.38
Visual Paired Associates	149	15.79	5.06	3.00	24.00	-	-0.15	-0.86
Sleep measures								
Sleep Quality	151	6.54	3.21	1.00	19.00	0.73	0.67	0.07
Insomnia Severity	151	15.19	5.18	8.00	28.00	0.86	0.84	0.11
Daytime Sleepiness	151	7.77	3.67	0.00	19.00	0.68	0.29	-0.36
Resilience	153	27.70	5.79	14.00	40.00	0.85	0.00	0.21

Table 3

Physical and Mental Health History Frequencies

Variable	*n*	%
Physical Health History		
Allergies	51	33
Asthma	23	16
Autoimmune disease	6	4
Cancer	1	0.7
Developmental disorder	3	2
Diabetes	1	0.7
Hearing loss	2	1.3
High blood pressure	5	3
Migraine or chronic headaches	18	12
Musculoskeletal problems	2	1.3
Obesity	6	4
Seizures or epilepsy	3	2
Sickle-cell anemia	14	9.2
Thyroid disease	1	0.7
Visual problems	37	24
Other	17	11
Mental Health and Other History		
ADHD	14	9.2
Anxiety-related disorders	34	22
Bipolar disorder	4	2.6
Developmental disorder	3	2
Depression	19	12.4
Learning disorder	22	14.4
Sleep disorder	2	1.3
Other psychological/psychiatric disorder	3	2

Table 4

Cumulative Lifetime Stress as a Predictor of Cognitive Variables

Variable	B	SE	*β*	*p*
Global Cognitive Function				
Constant	-0.47	0.25	-	.062
Cumulative lifetime stress	0.01	0.01	0.11	.221
Sex	0.07	0.10	0.06	.516
Parental educ.	0.00	0.01	0.00	.975
Alcohol intake	0.00	0.00	-0.04	.691
COVID-19 distress	0.07	0.08	0.08	.405
Forward Digit Span				
Constant	-0.11	0.33	-	.737
Cumulative lifetime stress	0.16	0.01	0.19	.027
Sex	0.03	0.13	0.02	.816
Parental educ.	0.04	0.06	0.05	.538
Alcohol intake	-0.001	0.00	-0.08	.349
COVID-19 distress	-0.21	0.11	-0.17	.060
Backward Digit Span				
Constant	-0.60	0.35	-	.085
Cumulative Lifetime Stress	0.00	0.01	0.04	.652
Sex	0.22	0.14	0.14	.110
Parental educ.	-0.01	0.06	-0.02	.852
Alcohol intake	0.00	0.00	0.03	.698
COVID-19 distress	-0.01	0.16	-0.01	.922
Digit Symbol Matching				
Constant	-0.39	0.31	-	.216
Cumulative Lifetime Stress	0.00	0.01	0.02	.869
Sex	-0.54	0.13	-0.04	.671
Parental education	-0.01	0.06	-0.02	.849
Alcohol intake	-0.001	0.00	-0.04	.665
COVID-19 distress	0.20	0.10	0.17	.062
Verbal Paired Associates				
Constant	-0.70	0.40	-	.081
Cumulative Lifetime Stress	0.01	0.01	0.08	.349
Sex	-0.05	0.16	-0.03	.746
Parental educ.	0.03	0.07	0.04	.685
Alcohol intake	0.001	0.002	0.03	.757
COVID-19 distress	0.15	0.13	0.10	.248
Visual Paired Associates				
Constant	-0.24	0.48	-	.613
Cumulative Lifetime Stress	0.00	0.01	0.003	.971
Sex	-0.02	0.19	-0.01	.906
Parental educ.	-0.03	0.09	-0.03	.730
Alcohol intake	-0.002	0.002	-0.07	.409
COVID-19 distress	0.35	0.16	0.20	.028

Table 5

Cumulative Lifetime Stress as a Predictor as Sleep Variables

Variable	B	SE	*B*	*P*
PSQI (Sleep Quality)				
Constant	2.30	1.26	-	.070
Cumulative lifetime stress	0.08	0.03	0.25	.002
Sex	0.84	0.50	0.25	.093
Parental educ.	-0.06	0.23	-0.02	.788
Alcohol intake	0.01	0.01	0.06	.448
COVID-19 distress	1.10	0.41	0.22	.009
ISI (Insomnia Severity)				
Constant	10.23	2.04	-	<.001
Cumulative lifetime stress	0.18	0.04	0.34	<.001
Sex	1.51	0.80	0.14	.062
Parental educ.	-0.73	0.37	-0.15	.049
Alcohol intake	0.00	0.01	0.02	.772
COVID-19 distress	1.57	0.67	0.19	.020
ESS (Daytime Sleepiness)				
Constant	4.27	1.53	-	.006
Cumulative Lifetime Stress	0.08	0.03	0.21	.010
Sex	1.16	0.60	0.16	.056
Parental educ.	-0.40	0.28	-0.12	.148
Alcohol intake	0.01	0.01	0.14	.090
COVID-19 distress	0.44	0.50	0.07	.384

Note. PSQI= Pittsburgh Sleep Quality Index; ISI= Insomnia Severity Index; ESS= Epworth Sleepiness Scale.

Table 6

Resilience as a Moderator in the Relationship Between Cumulative Lifetime Stress and Sleep

Variable	B	SE	*p*
PSQI (Sleep Quality)			
Interaction of Stress X Resilience	0.00	0.00	.720
ISI (Insomnia Severity)			
Interaction of Stress X Resilience	-0.002	0.01	.764
ESS (Daytime Sleepiness)			
Interaction of Stress X Resilience	-0.01	0.01	.185

Note. PSQI= Pittsburgh Sleep Quality Index; ISI= Insomnia Severity Index; ESS= Epworth Sleepiness Scale.

Table 7

Descriptive Statistics of Selected TMB Scores of this Study Compared to Selected TMB Normative Sample

	Current Study		**TMB Normative Sample**	
Test	*n*	*M* (*SD*)	*n*	*M* (*SD*)
Forward Digit Span	149	6.13 (1.25)	9858	6.43 (1.58)
Backward Digit Span	150	5.00 (1.47)	6269	5.33 (1.58)
Verbal Paired Associates	145	16.75 (4.90)	8496	18.21 (5.63)
Visual Paired Associates	149	15.79 (5.06)	9280	15.97 (4.53)

Note. TMB Normative Sample scores were provided in the Singh et al. (2021) article.

Table 8

Summary of Measures

Questionnaire	Administration Time	# of Items	Construct(s) Measured	Scale Interpretation
1. Adult STRAIN	~18-25	Adaptive Questionnaire Approx. 220 questions	Cumulative lifetime stress	The developers of the STRAIN will use 115 different cumulative life stress summary variables to summarize a person's stress exposure.
2. TMB Digit Symbol Matching	~5 min	N/A	Processing speed	The results output provided by TMB will include raw scores (total score and total number of errors), normative z-scores based on gender, age and education level.
3. TMB Digit Span	~5 min	N/A	Short-term memory, attention, and working memory (backward version)	Refer to item #2 description
4. TMB Verbal Paired Associates	~5 min	N/A	Verbal memory and episodic memory: measures sustained attention, response inhibition, and cognitive control	Refer to item #2 description
5. TMB Visual Paired Associates	~5 min	N/A	Visual memory and episodic memory	Refer to item #2 description

6.	Pittsburg Sleep Quality Index (PSQI)	~5-10 min	19	Sleep quality	0-21; score of >5 is considered as a significant sleep disturbance.
7.	Epworth Sleepiness Scale (ESS)	~3	8	Daytime sleepiness	0-24; Higher scores suggest excessive daytime sleepiness
8.	Insomnia Severity Scale (ISI)	~3	7	Severity of sleep initiation, maintenance, and awakening; sleep satisfaction; daily consequences; attributed impairment to sleep; concern for sleep	0-28; higher scores suggest clinical insomnia
9.	The Connor-Davidson Resilience Scale (CD-RISC 10)	~5 min	10	Resilience	0-40; higher scores suggest higher resilience.

APPENDIX B: STUDY MEASURES

DEMOGRAPHIC QUESTIONS

1. **Birth Date (MM/DD/YYYY): _____**
Age: _____ years

2. **What is your biological sex?**
Male
Female
Intersex
Other (please specify): _____
I'd prefer not to say

3. **What is your gender?**
Female/Woman
Male/Man
Transgender/ Gender queer/non-binary
Unspecified

4. **What is your Race**
American Indian/Alaska Native
Asian
Black or African American
Native Hawaiian or Other Pacific Islander
White or European American
Multi-racial
Unknown or other: _____

5. **Ethnicity**
Hispanic/Latinx
NOT Hispanic/Latinx

Medical History

1. **How would you describe your general health?**

- Excellent
- Very good
- Good
- Fair
- Poor

2. **Have you ever been diagnosed with any of the following?**

Medical condition	No	Yes	If yes, what year?	If yes, please specify the name of the disease (e.g., Anxiety-related disorders: Generalized Anxiety Disorder)
Allergies				
Asthma				
Anxiety-related disorders				
Autoimmune disease (e.g., MS, lupus)				
Bipolar disorder				
Cancer (e.g., breast cancer, brain tumor)				
Developmental disorder (e.g., autism)				
Depression				
Diabetes				
Drug or alcohol abuse/dependence				
Head injury or concussion				
Hearing loss				
High blood pressure				
Learning disorder (reading, writing, math, other)				
Migraine or chronic headaches				
Musculoskeletal problems				
Obesity				
Other psychological/psychiatric disorder				
Schizophrenia				
Seizures or epilepsy				

Sick-cell anemia				
Sleep disorder (e.g., insomnia, sleep apnea)				
Stroke				
Thyroid disease				
Vision Problems				
Other:				

3. **Below, please list all prescription medications, over-the-counter medication, herbal and/or vitamin supplements that you have been taking over the last 12 months:**

Medication Name	**Dose**	**Frequency**	**Route**	**Date Started**	**Purpose**
e.g., Medication	20 mg	2x/day	Oral	09/10/2017	Diabetes

Alcohol and Substance Use-Related Questions

Now, we would like to learn more about your drinking and substance-use habits. Please keep in mind that your responses will be kept **strictly confidential.**

Please answer the following questions about your use of alcohol and other substances **over the past month**.

One drink equals:
12 oz. Beer
5 oz. Wine
1.5 oz Liquor (one shot)

1. Over the past month, how often did you consume drinks containing alcohol?
- Never
- Monthly or less
- 2-4 times a month
- 2-3 times a week
- 4 or more times a week

2. Over the past month, how many alcoholic drinks have you had on a typical day when you were drinking alcoholic beverages?

- 0
- 1
- 2
- 3
- 4
- 5
- 6
- 7
- 8
- 9
- 10+
- I prefer not to answer

3. Over the past month, on average how often did you consume caffeinated drinks (i.e., coffee, caffeinated tea, energy drinks, caffeinated soda)?

- Never
- Monthly or less
- 2-4 times a month
- 2-3 times a week
- 4 or more times a week

4. Over the past month, how many cups or cans of caffeinated drinks (i.e., coffee, caffeinated tea, energy drinks, caffeinated soda) have you had <u>on a typical day</u> when you were drinking caffeinated beverages?
    - 0
    - 1
    - 2
    - 3
    - 4
    - 5
    - 6
    - 7
    - 8
    - 9
    - 10+
    - I prefer not to answer

5. Over the past month, how often have you used marijuana (e.g., a joint, pot, weed, hash, or hash oil) or cannabis?
    - 0
    - 1
    - 2
    - 3

- 4
- 5
- 6
- 7
- 8
- 9
- 10
- 11
- 12
- 13
- 14
- 15
- 16
- 17
- 18
- 19
- 20
- 21
- 22
- 23
- 24
- 25
- 26
- 27
- 28
- 29
- 30
- 31+
- I prefer not to answer

6. Over the past month, how often have you used a medication for anxiety or sleep (e.g., Xanax, Ativan or Klonopin)? This could be medication prescribed for you or not prescribed for you.
    - 0
    - 1
    - 2
    - 3
    - 4
    - 5
    - 6
    - 7
    - 8
    - 9
    - 10

- 11
- 12
- 13
- 14
- 15
- 16
- 17
- 18
- 19
- 20
- 21
- 22
- 23
- 24
- 25
- 26
- 27
- 28
- 29
- 30
- 31+
- I prefer not to answer

7. Over the past month, how often have you used medication for ADHD (e.g., Adderall, Ritalin)? This could be medication prescribed for you or not prescribed for you.
   - 0
   - 1
   - 2
   - 3
   - 4
   - 5
   - 6
   - 7
   - 8
   - 9
   - 10
   - 11
   - 12
   - 13
   - 14
   - 15
   - 16
   - 17
   - 18

- 19
- 20
- 21
- 22
- 23
- 24
- 25
- 26
- 27
- 28
- 29
- 30
- 31+
- I prefer not to answer

8. Over the past month, how often have you used nicotine (e.g., cigarette smoking, chewing tobacco, vaping, patches, gum, etc.).

- Never
- Monthly or less
- 2-4 times a month
- 2-3 times a week
- 4 or more times a week

9. Over the past month, how often have you used nicotine (e.g., cigarette smoking, chewing tobacco, vaping, patches, gum, etc.) <u>on a typical day</u>?
    - 0
    - 1
    - 2
    - 3
    - 4
    - 5
    - 6
    - 7
    - 8
    - 9
    - 10+
    - I prefer not to answer

Socioeconomic Status (SES)-Related Questions

1. **What is the highest level of education completed by your parent or a guardian (e.g, mother, father, grandmother, grandfather, legal guardian, adoptive/foster parents)?**

- Less than 12 years
- High school graduate/GED
- Some college/AA degree
- College graduate (Bachelor's degree)
- Graduate or professional degree: Master's or Doctorate degree (MD, PhD, JD)

2. **What is the highest level of education completed by your other parent or a guardian (e.g, mother, father, grandmother, grandfather, legal guardian, adoptive/foster parents)?**

- Less than 12 years
- High school graduate /GED
- Some college/AA degree
- College graduate (Bachelor's degree)
- Graduate school degree: Master's or Doctorate degree (MD, PhD, JD)

COVID-19 Related Questionnaires

1. Please rate your overall degree of stress and worry over the COVID-19 pandemic in the last 6 months?

0	1	2	3	4
Not worried at all		**Neutral**		**Very worried**

2. How often have you worried about COVID-19 in the past week?

0- Not worried at all
1- Several days
2- More than half the days
3- Nearly every day

SLEEP MEASURES

1. *Pittsburg Sleep Quality Index (PSQI)*

INSTRUCTIONS

The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

1. During the past month, what time have you usually gone to bed at night?
   BED TIME _____
2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night?
   NUMBER OF MINUTES _____
3. During the past month, what time have you usually gotten up in the morning?
   GETTING UP TIME _____
4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed.)
   HOURS OF SLEEP PER NIGHT _____

For each of the remaining questions, check the one best response. Please answer all questions.

5. During the past month, how often have you had trouble sleeping because you . . .
   a) Cannot get to sleep within 30 minutes

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

   b) Wake up in the middle of the night or early morning

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

   c) Have to get up to use the bathroom

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

   d) Cannot breathe comfortably

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

   e) Cough or snore loudly

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

f) Feel too cold

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

g) Feel too hot

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

h) Had bad dreams

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

i) Have pain

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

j) Other reason(s), please describe_____

How often during the past month have you had trouble sleeping because of this?

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

6. During the past month, how would you rate your sleep quality overall?

Very good
Fairly good
Fairly bad
Very bad

7. During the past month, how often have you taken medicine to help you sleep (prescribed or "over the counter")?

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

6. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

7. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?

No problem at all _____
Only a very slight problem _____
Somewhat of a problem _____
A very big problem _____

8. Do you have a bed partner or roommate?

No bed partner or room mate _____
Partner/room mate in other room _____
Partner in same room, but not same bed _____
Partner in same bed _____

2. Insomnia Severity Questionnaire (ISI)

The Insomnia Severity Index has seven questions. For each question, please CIRCLE the number that best describes your answer.

Please rate the CURRENT (i.e. LAST 2 WEEKS) SEVERITY of your insomnia problem(s).

Insomnia Problem	None	Mild	Moderate	Severe	Very Severe
1. Difficulty falling asleep	0	1	2	3	4

2. Difficulty staying asleep	0	1	2	3	4
3. Problems waking up too early	0	1	2	3	4

4. How SATISFIED/DISSATISFIED are you with your CURRENT sleep pattern?

Very Satisfied	Satisfied	Moderately Satisfied	Dissatisfied	Very Dissatisfied

5. How NOTICEABLE to others do you think your sleep problem is in terms of impairing the quality of your life?

Not at all Noticeable	A Little	Somewhat	Much	Very Much Noticeable

6. How WORRIED/DISTRESSED are you about your current sleep problem?

Not at all Worried	A Little	Somewhat	Much	Very Much Worried

7. To what extent do you consider your sleep problem to INTERFERE with your daily functioning (e.g. daytime fatigue, mood, ability to function at work/daily chores, concentration, memory, mood, etc.) CURRENTLY?

Not at all Interfering	A Little	Somewhat	Much	Very Much Interfering

3. Epworth Sleepiness Scale

How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired?

This refers to your usual way of life in recent times.

Even if you haven't done some of these things recently try to work out how they would have affected you.

Use the following scale to choose the **most appropriate number** for each situation:

0 = would **never** doze

1= **slight chance** of dozing

2= **moderate chance** of dozing

3= **high chance** of dozing

It is important that you answer each question as best as you can.

Situation	**Change of Dozing**
Sitting and reading	0 1 2 3
Watching TV	0 1 2 3
Sitting, inactive in a public place (e.g., theater or a meeting)	0 1 2 3
As a passenger in a car for an hour without a break	0 1 2 3
Lying down to rest in the afternoon when circumstances permit	0 1 2 3
Sitting and talking to someone	0 1 2 3
Sitting quietly after a lunch without alcohol	0 1 2 3
In a car, while stopped for a few minutes in the traffic	0 1 2 3

RESILIENCE MEASURE

Connor- Davidson Resilience Scale 10 (CD-RISC-10)

Please indicate how much you agree with the following statements as they apply to you over ***the last month***. If a particular situation has not occurred recently, answer according to how you think you would have felt.

	not true at all (0)	**rarely true (1)**	**sometimes true (2)**	**often true (3)**	**true nearly all the time (4)**
1. I am able to adapt when changes occur.					
2. I can deal with whatever comes my way.					
3. I try to see the humorous side of things when I am.					
4. Having to cope with stress can make me stronger.					
5. I tend to bounce back after illness, injury, or other hardships.					
6. I believe I can achieve my goals, even if there are obstacles.					
7. Under pressure, I stay focused and think clearly.					
8. I am not easily discouraged by failure.					
9. I think of myself as a strong person when dealing with life's challenges and difficulties.					
10. I am able to handle unpleasant or painful feelings like sadness, fear, and anger.					

Appendix C: IRB Study Approval Letter

EAST CAROLINA UNIVERSITY
University & Medical Center Institutional Review Board
4N-64 Brody Medical Sciences Building · Mail Stop 682
600 Moye Boulevard · Greenville, NC 27834
Office 252-744-2914 · Fax 252-744-2284 ·
rede.ecu.edu/umcirb/

Notification of Exempt Certification

From: Social/Behavioral IRB
To: Anya Savransky
CC: Daniel Everhart
Date: 2/9/2022
Re: UMCIRB 21-002227
Association Between Cumulative Lifetime Stress, Cognitive Function and Sleep

I am pleased to inform you that your research submission has been certified as exempt on 2/9/2022. This study is eligible for Exempt Certification under category # 2c.

It is your responsibility to ensure that this research is conducted in the manner reported in your application and/or protocol, as well as being consistent with the ethical principles of the Belmont Report and your profession.

This research study does not require any additional interaction with the UMCIRB unless there are proposed changes to this study. Any change, prior to implementing that change, must be submitted to the UMCIRB for review and approval. The UMCIRB will determine if the change impacts the eligibility of the research for exempt status. If more substantive review is required, you will be notified within five business days.

Document	Description
Connor-Davidson Resilience Scale 10 (CD-RISC-10).pdf(0.01)	Surveys and Questionnaires
Debriefing and list of resources .docx(0.01)	Additional Items
Demographic Measures_IRB1.docx(0.01)	Surveys and Questionnaires
Epworth Sleepiness Scale (ESS).pdf(0.01)	Surveys and Questionnaires
Informed Consent_exempt_final1.doc(0.01)	Consent Forms
InsomniaSeverityIndex (ISI).pdf(0.01)	Surveys and Questionnaires
Medical and Medication History questions.docx(0.01)	Surveys and Questionnaires
PQ-16.pdf(0.01)	Surveys and Questionnaires
PSQI-Instrument.pdf(0.01)	Surveys and Questionnaires
Recruitment script for SONA.docx(0.01)	Recruitment Documents/Scripts
Savransky Thesis Proposal_IRB.docx(0.01)	Study Protocol or Grant Application
Survey link to Qualtrics1.docx(0.01)	Surveys and Questionnaires
The Stress and Adversity Inventory (STRAIN)_updated.docx(0.02)	Surveys and Questionnaires

For research studies where a waiver or alteration of HIPAA Authorization has been approved, the IRB states that each of the waiver criteria in 45 CFR 164.512(i)(1)(i)(A) and (2)(i) through (v) have been met. Additionally, the

elements of PHI to be collected as described in items 1 and 2 of the Application for Waiver of Authorization have been determined to be the minimal necessary for the specified research.

The Chairperson (or designee) does not have a potential for conflict of interest on this study.

IRB00000705 East Carolina U IRB #1 (Biomedical) IORG0000418
IRB00003781 East Carolina U IRB #2 (Behavioral/SS) IORG0000418