LIFESTYLE BEHAVIORS AND CHRONIC DISEASE RISK IN EMERGING ADULTS WITH A HISTORY OF ADVERSE CHILDHOOD EXPERIENCES (ACES)

by

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The growing body of adverse childhood experiences (ACEs) literature over the past two decades provides strong evidence that there is a dose-response relationship between the number of ACEs experienced and the negative downstream effects on one's health. However, due to an abundance of retrospective public health survey data, but absence of data across the lifespan, the mechanisms that contribute to worse health outcomes remain unclear. Recent findings indicate that ACEs are associated with higher chronic disease prevalence even among young adults. Therefore, the present study was designed to examine the relationships between ACEs and various aspects of physical and mental health in a healthy emerging adult population.

Using a college student sample, participants completed an online questionnaire that included the Adverse Childhood Experience Questionnaire (ACE-Q) and measures of physical health, mental health, and lifestyle health behaviors. Correlation analysis revealed strong positive associations between ACEs and symptoms of PTSD, depression, anxiety, ADHD, emotion dysregulation, sleep difficulties, and somatic symptoms; mild positive associations were also observed between ACEs and perceived stress, disordered eating, illicit substance use, and e-cigarette use. Hierarchical linear regression analysis demonstrated that ACE-Q scores significantly predicted these factors when controlling for demographic covariates such as gender, race/ethnicity, and SES.

The findings of this study help to fill an etiological knowledge gap by providing more information about the prevalent risk factors related to individuals with a history of high ACEs that could contribute to the development of chronic diseases. In addition to various mental health symptoms, sleep difficulties and somatic symptoms appear to be prominent health issues for emerging adults with a history of ACEs. Healthcare providers for this age group may want to increase screening, prevention, and intervention efforts to address these symptoms, as this may have a positive influence on reducing chronic disease risk.

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Introduction

Over the last two decades, emerging research on adverse childhood experiences (ACEs) has demonstrated a clear association between early life adversity (e.g. abuse, neglect, household dysfunction) and various states of pathology. Although some recent literature has sought to explore the underlying mechanisms connecting ACEs with subsequent disease states, the pathophysiology remains largely unknown. This is mainly due to the high frequency of ACEs population data and relatively low implementation of experimental approaches to explore disease pathways associated with ACEs. Therefore, specific physiological mechanisms must be explored to inform interventions that target vulnerable youth, adolescents, and young adults with a history of ACEs, as these individuals are at high risk for developing chronic physical and mental health conditions (Felitti et al., 1998).

For individuals with a history of ACEs, the pathway to chronic illness is a process that involves interaction between the body's major systems – namely, the nervous, cardiovascular, endocrine, and immune systems. Cardiovascular disease remains the most prominent cause of death in the US, and is responsible for approximately 25% of all deaths (Benjamin et al., 2019). Accordingly, cardiovascular health is perhaps the most significant target for health interventions, and a large diversity of interventions have been developed to address the presence of biological, behavioral, social, and psychological risk factors at the cardiovascular level (Su, Jimenez, Roberts, & Loucks, 2015). Due to the interactions of these risk factors, the heightened risk for various forms of heart disease is well-documented among individuals with a history of ACEs (Felitti et al., 1998; Dong, et al., 2004; Campbell, Walker, & Egede, 2016; Hughes et al., 2017). In particular, chronic stress has been used to explain the effects of various biological,

psychological, and environmental determinants on both cardiovascular and mental health (Su et al., 2015).

In addition to increased vulnerability for heart disease, individuals with a history of childhood adversity also have a heightened risk for various forms of psychopathology. Those with elevated ACE scores have been consistently found to have higher prevalence of depression, suicide attempts, alcohol and other substance use, interpersonal violence and overall poor mental health (Felitti et al., 1998; Hughes et al., 2017). Combined with this, there is extensive data supporting the increased prevalence mental health conditions among heart disease patients, and the prevalence of depression is about three times higher than in the general population (Chaddha, Robinson, Kline-Rogers, Alexandris-Souphis, & Rubenfire, 2016). While the bidirectionality of this association has been widely accepted – that is, that mental health conditions contribute to heart disease and heart disease contributes to mental health conditions – the exact mechanisms of this relationship are complex, and still largely unknown. Accordingly, it is fitting for this link between psychological and cardiovascular pathology to be simultaneously studied among the ACEs population, a group with heightened vulnerability for both conditions.

Autonomic nervous system (ANS) function is particularly useful in the study of chronic stress due to its sensitivity to both physiological and psychological health influences and the ability to monitor ANS functioning in a laboratory setting. For instance, multiple studies have found that participants with ACE scores ≥ 4 had dysregulated cortisol levels, connecting ACE prevalence to stress sensitization and dysregulation of the HPA axis (Bunea, Szentágotai-Tătar, & Miu, 2017; Kalmakis, Meyer, Chiodo, & Leung, 2015). While autonomic dysfunction is speculated to play a role in the high rates of heart disease among adults with a history of multiple ACEs (Su et al., 2015), the current literature has not yet thoroughly explored this relationship. In

a recent meta-analysis of ACEs and indicators of poor health, only two studies explored autonomic function, and both concluded significant relationships between adversity and physiological dysregulation (Rigterink, Katz, & Hessler, 2010; Shonkoff et al., 2012). This highlights the need for additional research to examine the role of autonomic dysregulation as a potential mechanism in the progression from childhood adversity to subsequent cardiovascular disease.

Through the use of electrocardiogram (ECG) monitoring, heart rate variability (HRV) is one of the most widely used measurements of ANS functioning, and has been conceptualized as a transdiagnostic biomarker of both physical and mental illness (Beauchaine & Thayer, 2015). Among the different frequencies of HRV (e.g. low frequency, very low frequency), high frequency HRV (HF HRV) is often utilized due to the ability to detect vagal control – that is, one's ability to self-regulate heart rate via the vagus nerve. Accordingly, vagal control has been conceptualized as the proper adaptation to the stressors in one's environment, which allows for the conservation of energy, and thereby prevents unnecessary burden on the cardiovascular system (Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012). Favorable health outcomes have been associated with high HF HRV, representing one's ability to engage in adaptive regulatory processes carried out by sympathetic and parasympathetic activity. In comparison, low HF HRV is associated with poorer health outcomes, and indicates problems with self-regulation and cognitive control (Thayer, Hansen, Saus-Rose, & Johnsen, 2009).

In particular, the behavioral inhibition and activation systems (BIS/BAS) have been used as a conceptual framework to interpret HRV analysis (Brenner, Beauchaine, & Sylvers, 2005). These constructs were initially developed to explain how motivational systems can predict the relationship between affect and behavior (Gray, 1981). As such, social engagement is considered

to be an adaptive response to the stressors in one's environment, and would be expected to accompany HRV reactivity that is appropriately responsive to experiences of stress. In contrast, various forms of avoidance are viewed as maladaptive, and would theoretically be found associated with HRV responses that are inadequately lower or excessively higher than what would be expected in relation to the presenting stressor. Accordingly, self-report measures of behavioral inhibition and behavioral activation (Carver & White, 1994) have been used to describe how affective and emotion regulation differences between individuals that may contribute to physiological dysfunction (Brenner et al., 2005).

Significantly, issues of emotion regulation have been identified as a prevalent risk factor among individuals with high ACE scores. The ACEs literature has consistently shown that exposure to early adversity is associated with higher prevalence of mental health issues in adulthood, including depression, suicide, interpersonal violence, and alcohol and other substance use (Felitti et al., 1998; Hughes et al., 2017). A recent assessment of mental health issues in an adolescent sample (ages 13-17) found that elevated ACE scores were associated with heightened symptoms of ADHD (impulsivity, inattention, and combined types), generalized anxiety disorder (GAD), major depressive disorder (MDD), and dysthymic disorder (Horn, Leve, Levitt, & Fisher, 2019). Given the application of HRV analysis to transdiagnostic mental health issues, it is likely to observe an association between ACEs and ANS dysregulation in laboratory samples. Additionally, some statistical models have concluded that emotion and cognitive processes could mediate the relationship between childhood adversity and physiological changes (Jin, Kim, Kim, Hyun, & Lee, 2018).

Given these factors, the ecobiodevelopmental (EBD) theory is the leading framework for understanding the interaction of early life adversity, chronic stress, and subsequent poor health

outcomes (Shonkoff et al., 2012). In a recent policy proposal, the American Academy of Pediatrics voiced their adoption of this framework to discuss the various influences of chronic, or toxic, stress throughout the lifespan. The concept of toxic stress has been used to contrast the experiences of positive stress and tolerable stress, as toxic stress involves prolonged activation of the body's stress response in a way that alters brain development and associated functioning (Franke, 2014). For instance, neurodevelopmental changes to the amygdala, hippocampus, and prefrontal cortex due to toxic stress have been shown to contribute to deficits in learning, memory, executive functioning and anxiety (Shonkoff et al., 2012).

Past research has shown that these neurodevelopmental changes also contribute to autonomic dysregulation. The results of these modified frontolimbic functions have been identified as altered cognition, reduced stress reactivity, and emotion dysregulation, which may lead to impulsivity and a focus on short-term goal attainment, and consequently, may increase one's risk for poor health behaviors (e.g. addiction/substance use, unhealthy eating, physical inactivity) (Lovallo, 2013). Therefore, these changes are likely to influence reactivity and vagal control during a stressor task. In past research, various stress tasks have been implemented during HRV monitoring to generate stress responses that require participants to engage in emotion regulation and cognitive control. This methodology may provide insight into the ways that toxic stress and associated neurodevelopmental alterations can also lead to changes in autonomic dysregulation and cardiovascular reactivity.

While the growing ACEs literature has recently emphasized neurodevelopmental changes within the EBD framework, very few studies have looked at alterations to cardiovascular activity. This is surprising, since research has indicated an increased risk to various forms of heart disease – such as myocardial infarction, coronary artery disease, stroke, and diabetes – for

individuals with a history of ACEs (Gilbert et al., 2015). Additionally, individuals with a higher prevalence of ACEs have been consistently shown to have an increased association with some of the main lifestyle and behavioral contributors to heart disease, such as heavy alcohol use, smoking, risky sex behaviors, physical inactivity, obesity, and problematic drug use (Campbell, Walker, & Egede, 2016; Hughes et al., 2017). These results are consistent with the sample used in the seminal ACE Study (Felitti et al., 1998), which showed a dose-response relationship between ACEs and ischemic heart disease. Further, a follow-up study found that ischemic heart disease was mediated by psychological risk factors commonly associated with childhood adversity (e.g. depression, anger, negative affect) rather than traditional risk factors of ischemia (Dong, Giles et al., 2004). Taken together, this suggests that psychological risk factors may pose a unique risk for developing CVD in individuals in with a history of ACEs, and that this may create vulnerability above and beyond that of unhealthy lifestyle factors (e.g. smoking, physical inactivity, substance misuse, unhealthy eating).

Due to the predominant healthcare burden of heart disease, there remains a pressing need for interventions informed by a deeper understanding of the pathophysiology that contributes to heightened CVD rates in the ACEs population. Since cardiovascular diseases are typically not diagnosed in emerging adulthood, early psychophysiological indicators of CVD may allow for the implementation of early prevention and intervention strategies. Thus, HRV analysis in emerging adults may be able to identify potential mechanisms (e.g. poor vagal control, emotion dysregulation, cognitive dysfunction) for individuals with a history of ACEs. Further, while low HF HRV has been considered a biomarker of disease, self-regulatory vagal control has also been explored as a modifiable risk factor, and improvements may directly reduce one's risk for disease (Thayer & Lane, 2007). Moreover, recent studies have identified that psychotherapeutic

interventions can improve physiological dysfunction during stress tasks, suggesting that adaptive physiological responses can be learned and practiced (Steffen, Fidalgo, Schmuck, Tsui, & Brown, 2014). In summary, HRV analysis in individuals with a history of ACEs may be able to inform effective prevention and intervention strategies related to self-regulation and stress reactivity, and as a result, reduce one's heightened risk for cardiovascular disease.

Literature Review

Adverse Childhood Experiences (ACEs)

Since the publication of the ACEs Study (Felitti et al., 1998), adverse childhood experiences (ACEs) have been generally conceptualized as experiences of abuse and household dysfunction prior to age 18. The construct of ACEs was developed as a way to quantify chronic negative early life experiences and examine the aggregate effects on subsequent physical and mental health. The operationalization of ACEs varies slightly between different measurement tools, but the most commonly assessed items include parental incarceration, domestic violence, household mental illness/suicide, parental separation/divorce, household substance abuse, physical and emotional neglect, and physical, sexual and emotional abuse; less common items include experiences of neighborhood/community violence, economic hardship, serious car accidents, bullying victimization, discrimination, and parental death (Bethell et al., 2017). The Adverse Childhood Experiences Study Questionnaire (ACE-Q) is both the original and most widely used measurement tool, and quantifies an ACE score based on the total number of affirmative questionnaire responses ranging from zero to ten (Felitti et al., 1998).

The prevalence of childhood adversity has been derived from various sources of population data, all of which provide similar estimates. One sample that included the ACE-Q as part of the larger 2010 Behavioral Risk Factor Surveillance Survey administered across 11 regions of the US found that 40.6% of respondents reported zero ACEs, 44.1% reported one to three ACEs, 12.7% reported four to six ACEs, and 2.6% reported seven to nine ACEs (Gilbert et al., 2015). Prevalence data were similar using the ACE questionnaire (Felitti et al., 1998) with undergraduate students at East Carolina University, which found that 40.5% of participants

reported no history of ACEs, 47% reported 1-3 ACEs, and 12.5% reported 4 or more ACEs (N=560, M=1.4, SD=1.7) (Dolbier, 2019; In Prep).

In their seminal work, Felitti and colleagues (1998) found the association between ACEs and poorer health outcomes is most notable for individuals with a history of four or more (≥ 4) types of childhood abuse and family dysfunction. Initial results showed that a history of ≥ 4 ACEs yielded a 4-12 fold increase in health risks for alcohol and other drug use, depression, suicide attempts, and interpersonal violence. Additionally, health risks were at least double for smoking, risky sexual behavior, sexually transmitted diseases and poor self-rated health, and nearly double for physical inactivity and obesity. In a recent systematic review of health outcomes in pediatric samples, ACEs were found to be associated with delays in cognitive development, asthma, infection, somatic complaints, and sleep disruption (Oh et al., 2018). Due to a variety of poor health outcomes, increased mortality rates have been observed in individuals with elevated ACE scores; specifically, those with six or more ACEs were found to die, on average, 20 years earlier than individuals who reported no history of ACEs (Brown et al., 2009).

Poorer physical and mental health for individuals with a history of childhood adversity is seen most clearly through an increased prevalence of chronic health conditions in adulthood. Felitti and colleagues (1998) found that the presence of more types of ACEs was associated with an increased prevalence of ischemic heart disease, chronic lung disease, cancer, and liver disease. Importantly, such findings have been widely replicated. One recent study found that higher ACE scores increased risk for diabetes, coronary artery disease, myocardial infarction, stroke, and depression (Campbell et al., 2017). When controlling for demographic variables in a US sample, there was a dose-response relationship between ACE scores and health outcomes including fair/poor general health, frequent mental distress, asthma, diabetes, coronary heart

disease, stroke, myocardial infarction, and disability (Gilbert et al., 2015). Further, in a nationally representative sample of Irish adults, childhood adversity was associated with earlier onset of cardiovascular and pulmonary diseases, as well as psychiatric disorders (McCrory, Dooley, Layte, & Kenny, 2015). Taken together, this association between early life adversity and worse health outcomes demands a greater understanding of pathways to poor health.

Although population data has been used to describe ACEs prevalence and subsequent health risks, the pathways to disease remain largely unknown. One of the most widely adopted explanations of poorer health is that of toxic stress. As previously noted, the ecobiodevelopmental (EBD) framework is used to examine the various ways that prolonged stress activation at an early age can lead to developmental changes. Thus far, these developmental changes have been primarily assessed at the neurological level. For instance, changes to the amygdala, hippocampus, and prefrontal cortex from toxic stress can reduce learning, memory, and executive functioning (Shonkoff et al., 2012). Further, the EBD framework offers the assumptions that heightened stress at an early age alters the development of adaptive coping responses and provides a basis for altered physiological responses, maladaptive coping, and unhealthy lifestyle behaviors (Garner et al., 2012). Similarly, Lovallo (2013) proposes a model whereby early adversity alters neurodevelopment of the limbic system (one of the brain's major centers of emotion processing). As such, these alterations may contribute to decreased stress reactivity and increased emotion dysregulation, resulting in subsequent impulsive and risky behaviors. This pattern of emotion dysregulation has been widely identified in the literature as a factor that increases the likelihood of addiction and unhealthy coping behaviors (Beauchaine et al., 2018). Thus, both models assume a cascading effect of neurodevelopment on subsequent emotion and behavior. Therefore, ideal prevention and

intervention efforts should aim to identify indicators of stress and physiological dysfunction earlier in the process of neurodevelopment before emotion dysregulation and maladaptive coping behaviors become manifest.

Physiological Stress and Dysfunction

Various biomarkers of stress are used to identify dysfunction in the body's regulatory systems. For the purposes of this study, regulation of the cardiovascular system is of primary interest, since a cardiovascular approach to stress reactivity has been only lightly explored in the ACEs population despite early evidence of unique psychological factors contributing to ischemic heart disease (Dong et al., 2004). Neurodevelopmental changes, as previously noted in the ACEs population, interact with the cardiovascular system via the body's systems of stress reactivity, primarily explained via activity in the autonomic nervous system. More specifically, the hypothalamic-pituitary-adrenocortical (HPA) axis has been extensively identified as a regulatory system that plays a critical role in the body's response to stress, and alterations to this system have been found associated with ACEs (Kalmakis et al., 2015).

The HPA axis operates as one of the primary stress response systems in the body, and chronic activation of this system is associated with various adverse effects. Under normal conditions of stress, corticotropin-releasing hormone (CRH) is produced and released by the hypothalamus to stimulate adrenocorticotrophic hormone (ACTH) production in the pituitary gland. Once ACTH is released into circulation, glucocorticoids (e.g. cortisol) are produced and released by the adrenal gland. When this system is chronically activated, this may lead to increased basal secretion of cortisol, heightened stress reactivity, and/or adrenal fatigue (Herman et al., 2016). Similarly, activation of the sympathetic-adrenal-medullary (SAM) axis during sympathetic arousal stimulates the release of catecholamines such as epinephrine and

norepinephrine. In response to stress, these hormones are released by the adrenal gland to increase neural and cardiovascular activity.

If adequate downregulation via parasympathetic activity does not take place, this can lead to chronic dysregulation, which has more recently been referred to as toxic stress (Shonkoff et al., 2012). In one systematic review of pediatric health outcomes associated with childhood adversity, the authors identified several categories of disease biomarkers associated with ACEs as a result of altered HPA and SAM activity (Oh et al., 2018). The authors identify that studies of endocrine function, such as cortisol levels, found that participants who experienced childhood maltreatment exhibited blunted cortisol responses to stress. In contrast, studies in which participants had ACEs related to parental depression indicated slightly increased cortisol responses. Taken together, this could indicate that maltreatment is associated with a more chronic stress profile, in which cortisol release becomes blunted due to heightened activation and exhaustion. Regarding inflammation and immune function, the authors identify various associations between ACEs and increased cytotoxicity, such as elevated proinflammatory cytokines. Further, the authors discuss biomarkers of autonomic function, and identify a sample in which exposure to domestic violence was associated with lower vagal tone (Rigterink et al., 2010). Given the heterogeneity of adversity experiences among ACE samples, further research is needed to better understand the relationships between disease biomarkers and specific, as well as collective, ACE items. Additionally, it may be important to identify factors of adversity experiences such as frequency and intensity, as this may help distinguish those that are associated with acute versus chronic stress responses.

In recent clinical and research settings, changes to the HPA and SAM systems are most commonly measured through cortisol, a widely used biomarker of physiological stress. Due to

the high accessibility of cortisol in clinical and research settings (measured through sweat, tears, urine, blood and hair), many interventions for children with a history of ACEs use cortisol levels as a primary outcome (Purewal et al., 2018). Overall, the cortisol literature regarding ACEs and stress reactivity has shown that dysregulation is identified by both increases and decreases in cortisol levels. Using a college student sample, one study found that higher ACE scores were associated with lower concentrations of hair cortisol (Kalmakis et al., 2015). Oh and colleagues (2018) found that blunted cortisol levels during childhood were associated with maltreatment, and elevated cortisol levels were associated with maternal mental health issues. Further, a meta-analysis of the association between early-life adversity and cortisol responses to social stress found that the effect sizes of cortisol were higher in studies that focused on maltreatment compared to other types of adversities (Bunea et al., 2017). Thus, specific types of ACEs may have different associations with cortisol and stress reactivity, highlighting the need for additional indicators of stress reactivity other than cortisol.

While there have been multiple reported associations between ACEs and cortisol, this information allows for only speculation regarding the relationship between ACEs and cardiovascular activity. For instance, one study of lifetime adversity (rather than childhood adversity) in adult participants found a similar dose-response rate between experiences of adversity with elevations to both cortisol and heart rate (Lovallo, Farag, Sorocco, Cohoon, & Vincent, 2012). Although cortisol and heart rate responses to stress are both considered measures of ANS activity, studies such as this do not allow for distinctions between heightened cardiac reactivity versus vagal withdrawal, or proper cardiac recovery to rest versus prolonged reactivity. Further, a recent laboratory study identified that increases in cortisol levels during a stress task were preceded by anticipatory decreases in HRV, yet in-task HRV responses were not associated

with cortisol changes or cortisol recovery (Pulopulos, Vanderhasselt, & De Raedt, 2018). This demonstrates how cortisol and HRV are likely related, but provide different information regarding the physiological mechanisms of a stress response. While cortisol research in regard to ACEs has increased in recent years, significantly fewer studies have looked at outcomes related to heart rate variability following a history of childhood adversity; this may address many of the limitations of using cortisol measurements as a way to explain the dynamic process of stress reactivity over time. To better understand the relationship between ACEs and cardiac reactivity, laboratory studies are needed that monitor HRV stress reactivity before, during, and after a stress task.

Heart Rate Variability (HRV)

Due to the limitations of cortisol research in the ACEs population, heart rate variability (HRV) is a more useful measure of cardiovascular stress reactivity, and can provide additional information about the influence of psychological stress and ANS dysregulation. As a measurement of ANS activity, HRV recording is able to capture the heart-brain interaction and provide information about the ANS as an internal regulatory systems (Shaffer & Ginsberg, 2017). Dysregulation of HRV has been associated with various disease states, including hypertension, diabetes, congestive heart failure, renal failure, and subsequent cardiac events after a myocardial infarction (Acharya, Joseph, Kannathal, Lim, & Suri, 2006). Using electrophysiological methods, HRV is measured as the variation of time intervals between heart beats, and is captured during a process of continuous heart rate monitoring. Unlike heart rate, HRV is a measurement of the changes in heartbeats over a period of time; thus, there are smaller time intervals between rapid heartbeats (low HRV) and greater time intervals between slower heartbeats (high HRV). Further, HRV has been described as a non-linear, dynamic measurement

that is subject to considerable fluctuation (Shaffer & Ginsberg, 2017). Within psychophysiology, HRV has been used to examine the function of an individual's regulatory control system as it responds to internal and external stimuli through dynamic processes (Lehrer & Eddie, 2013).

In their discussion of regulatory systems, Lehrer and Eddie (2013) describe several features of health and pathology that can be identified by HRV. Overall, positive functioning is characterized by oscillation, and dysfunction can be identified by a lack of oscillatory patterns. While flexible responses help a system respond to contextual demands, dysfunctional responses may be characterized by simplicity (i.e. dampening), randomness, and heightened reactivity. For example, the baroreflex system implements regulatory fluctuations in blood pressure, heart rate, and vascular tone. These oscillations, which create variability, help to accomplish modulatory control of the baroreflex system in response to demands for blood and oxygen (Lehrer & Eddie, 2013). Accordingly, adaptive HRV regulation is characterized by adequate reactivity and recovery, which signifies proper baroreflex control – this may be contrasted by blunted or heightened reactivity and shortened or prolonged recovery.

The dynamic process of HRV within the baroreflex system is largely influenced by the counteracting effects of the sympathetic and parasympathetic nervous systems. As with the baroreflex system, these two systems within the ANS act as a negative feedback loop: epinephrine and norepinephrine are released by sympathetic fibers to facilitate arousal, while acetylcholine is released from the parasympathetic fibers to promote recovery to baseline (Lehrer & Eddie, 2013). This system is moderated by the sinoatrial node, which has been referred to as a cardiac "pacemaker," as it is subject to various influences that increase and decrease heart rate. Further, the sinoatrial node is innervated by the vagus nerve, therefore an individual's ability to exert control to counteract increases in heart rate is termed vagal control; this action is completed

via activation of the parasympathetic nervous system. During cardiac reactivity monitoring, the high frequency HRV band (HF HRV) is considered a measurement of vagal control (Lehrer & Eddie, 2013). Thus, HF HRV is used to measure how well an individual exerts control over physiological reactivity in order to respond to changes in external and internal stimuli.

One explanation of the adaptive function of HRV, and the utility of HRV measurement, is given by the polyvagal theory (Porges, 2001). Accordingly, ANS regulation is viewed as the result of evolutionary processes, whereby cardiac reactivity functions within a larger system of social engagement. The theory posits that behavior is rooted in biological functioning, and changes in the ANS mediate social functioning. As such, the use of HRV measurement helps to identify stress reactivity in the context of engagement and disengagement with various social demands. Optimal functioning is viewed as a proper application of social behavior to environmental demands, whereas pathological responses reflect inappropriately low or high reactivity. Similar to Lehrer and Eddie's (2013) explanation of the function of negative feedback loops within a control system, Porges (2001) describes how failure to properly respond through escape behavior and vagal control results in compensatory chemical processes (e.g. moderation of corticosteroids and catecholamines) to decrease one's stress response. Utilization of and dependence on these chemical processes – rather than adequate behavioral responses and vagal control – contributes to ANS dysregulation.

In response to this conceptualization of HRV dysregulation as an indicator of maladaptive social responses, HRV has also been regarded as a transdiagnostic biomarker of mental health conditions (Beauchaine & Thayer, 2015). Similar to the polyvagal theory (Porges, 2001), this perspective attempts to identify motivation in the context of evolutionary processes. Specifically, the authors discuss the interaction of HF HRV within the behavioral inhibition and

activation systems (BIS/BAS), whereby autonomic dysregulation and maladaptive behavioral patterns interact to contribute to psychopathology. This takes place, in part, because heart rate is subject to self-regulatory processes within the autonomic nervous system, which can be influenced by various states of psychological distress and well-being. As innate motivational systems, the BAS network deals with appetitive motivation characterized by goal-approach and active avoidance of punishment; this system is commonly associated with more positive health outcomes. In contrast, the BIS network is activated during passive avoidance of punishment, stimulates an anxiety response, and is associated with more negative health outcomes (Gray, 1981).

Of note, while the BIS/BAS systems provide a rationale for ANS dysregulation, as measured by HRV, prior research indicates that self-report measures of BIS/BAS are more closely associated with affective states rather than physiological responses (Brenner et al., 2005). Therefore, the authors conclude that BIS/BAS self-report measures should not be considered a surrogate of HRV measurement, and these two measures are seemingly independent of one another. In other words, a participant's self-evaluation of their response to stress (i.e. inhibition versus approach) may not accurately reflect their physiological functioning, but instead, more closely reflect their affective state. Nonetheless, Beauchaine and Thayer (2015) propose that while HF HRV may be considered a transdiagnostic biomarker of psychopathology, this association is likely strongest when HRV recording is completed in contexts that engage with the BIS/BAS systems (e.g. social-evaluative stressor task), as these contexts require utilization of emotion regulation, cognitive control, and behavioral coping.

Given this relationship between ACEs, toxic stress, emotion regulation difficulties, ANS dysfunction, behavioral inhibition, and psychopathology, each of these factors have been

proposed as underlying mechanisms that contribute to poorer cardiovascular health outcomes. The process by which chronic dysregulation of the autonomic nervous system leads to physiological dysfunction has been referred to as allostatic load. Unlike allostasis, or the body's response to acute stressors – where sympathetic activity is down-regulated by parasympathetic activity so that the body may return to a state of dynamic equilibrium – chronic stressors may result in allostatic load, or a permanent off-setting of homeostasis (McEwen & Stellar, 1993). Pathological responses within allostasis (i.e. the process of adjustment to return to homeostasis) are a result of repeated and continuous stressors, a lack of adaptation, prolonged stress responses, and inadequate stress responses (McEwen, 1998). Consequently, an allostatic load perspective suggests that these stress responses facilitate changes to internal structures and contribute to pathophysiology; namely, those related to the neuroendocrine, immune, metabolic, cardiovascular, and respiratory systems (Juster, McEwen, & Lupien, 2010). Due to the high influence of these systems on cardiovascular stress reactivity, HRV could be considered one of the possible mechanisms by which psychological stress influences physical health via physiological dysregulation, and accordingly, may inform future chronic stress interventions for vulnerable populations.

Proposed Study

Overview

The purpose of the proposed study is to examine the association between ACEs and stress reactivity from a cardiovascular health perspective, and to better understand the connection between childhood adversity and subsequent cardiovascular disease. As previously described, the association between ACEs and poorer health, including increased mortality, is well-documented (Felitti et al., 1998; Oh et al., 2018; Brown et al., 2009). The negative health effects of ACEs are largely due to the prevalence of chronic diseases, and in particular, cardiovascular disease (CVD). Further, the ecobiodevelopmental (EBD) framework, which has been adopted by the American Academy of Pediatrics, proposes that toxic stress and its associated underlying mechanisms are responsible for the progression from elevated ACEs to subsequent poorer health (Shonkoff et al., 2012).

Although research on toxic stress has been increasingly applied to neurological development and alteration, very little research to-date has examined the effects of toxic stress within the cardiovascular system. A relationship between toxic stress and cardiovascular stress reactivity would be expected since other indicators of autonomic dysregulation have already been cited in studies of ACEs and cortisol dysregulation (Bunea et al., 2017; Lovallo et al., 2012). Accordingly, in the proposed study, we seek to identify early signs of cardiovascular dysfunction in individuals with elevated ACE scores through the use of HRV monitoring in emerging adults, a population in which it is typically considered too early to observe indicators of cardiovascular disease.

The use of ECG laboratory monitoring will be used to derive measurements of HRV. Heart rate variability is predominantly associated with dysfunction within domains that involve

avoidance of perceived social stress, and observed differences in measurement are influenced by stress responses within the autonomic nervous system; the habitualization of a stress response across various domains is referred to as autonomic dysregulation. While autonomic dysregulation takes place at the biophysiological level, it is also influenced by cognitive, affective, and behavioral factors. Correspondingly, HRV is influenced by vagal tone – which is highly associated with cognitive control – and the motivational systems of behavioral inhibition and activation (BIS/BAS) – which help to explain the interaction between social stress, emotion regulation, and behavior (Beauchaine & Thayer, 2015). In order to observe this interaction, the present study will obtain HRV measurements for participants at rest, during a laboratory stress task, and during recovery from the stress task. This will serve as a model through which each participant's stress response can be interpreted as a generalized physiological, affective, and behavioral response across various "real-life" contexts.

In order to generate a stress response that reflects autonomic dysregulation, the present study will employ the methodology of the Montreal Imaging Stress Task (MIST) (Dedovic et al., 2005). The MIST is a standardized computer-based math task that has been used in research settings to coincide with HRV monitoring. During task-completion, participants will be asked to perform mental computations under time restraint, and response submission will require rapid operation of a computer mouse. Additionally, the testing platform provides automated stress-inducing feedback, in which participants will be frequently alerted of their low performance compared to peers. The use of the MIST is warranted for various reasons, but primarily because it was designed as a social-evaluative stressor task. This is important, as HRV is conceptualized within the polyvagal theory as a measurement of one's physiological response associated with one's appraisal of social stress and subsequent engagement or disengagement (Porges, 2001). In

a recent study that compared HRV responses during common laboratory stress tasks (e.g. Trier Social Stress Task, Stroop Color-Word Task, speech tasks), the MIST was shown to induce higher cardiovascular responses than other tasks, despite similar participant ratings of perceived stress across all tasks (Brugnera et al., 2018). Further, the authors of this study noted that stress tasks that required participant verbalizations were less able to detect vagal withdrawal, which may have been masked due to the influence of respirations. Interpreted alongside of the EBD framework, exposure to early toxic stress alters neurodevelopment and leads to maladaptive coping, which includes interpreting neutral social stimuli negatively (Iffland, Wiggert, Neuner, & Blechert, 2018). Accordingly, elevated exposure to ACEs would suggest an increased likelihood of autonomic dysregulation during the MIST, identified by cardiovascular dampening, low vagal control, and heightened cardiovascular stress reactivity.

Since HRV has been conceptualized as a transdiagnostic biomarker of psychopathology (Beauchaine & Thayer, 2015), participants will be asked to complete common mental health screening measures of depression (PHQ-9), anxiety (GAD-7), post-traumatic stress (PCL-5), and attention deficit-hyperactivity (ASRS-5). This may help to identify psychological correlates of ACEs and autonomic dysregulation. Additionally, measures of emotion dysregulation will be assessed using measures of behavioral inhibition/avoidance (BIS/BAS), emotion difficulties (DERS-18), perceived stress (PSS), and resilience (BRS). These are an essential component to better understanding the relationship between ACEs and HRV reactivity, as emotion regulation difficulties have been shown to be related to neurodevelopmental changes in response to early adversity (Lovallo, 2013).

Further, common types of adaptive and maladaptive behavioral coping styles will be assessed using measures of various health and lifestyle behaviors. Participants will be asked to

complete measures related to exercise (GLTEQ), sleep difficulties (PSQI), alcohol use (AUDIT-C), tobacco smoking (FTQ), e-cigarette use (EDS), substance misuse (DAST-10), disordered eating (ESP), and sexual risk-taking (SRS). These measures may provide important insight about the relationship between ACEs, emotion regulation difficulties, and subsequent risk-taking, poor health behaviors, and alcohol/substance use risk. To acquire additional information about participant's subjective and objective overall health, the present study will also assess for somatic symptoms (PHQ-15), self-rated health, and healthcare utilization. During the lab portion of data collection, participants will undergo physical measurements of height, weight, and blood pressure.

Taken together, these measures will allow the present study to identify additional ways that psychopathology, cognitive difficulties, emotion dysregulation, and maladaptive behavioral coping may be associated with ACEs, autonomic dysregulation, and poorer general health. This type of comprehensive assessment will contribute to a greater understanding of the progression from childhood adversity to poorer cardiovascular health, which has been well-documented in the existing ACEs literature.

Proposed Aims and Hypotheses

Aim 1

To examine the relationship between early adversity and cardiovascular stress reactivity in a college student sample, which could provide early indication of cardiovascular disease risk among emerging adults with a history of elevated ACEs.

Hypothesis 1

Participants who report a history of ≥ 4 ACEs on the ACE-Q will demonstrate greater autonomic dysregulation during ten minutes of baseline monitoring, measured as significantly lower mean scores of HF HRV, compared to participants who report a history of < 4 ACEs.

Hypothesis 2

Participants who report a history of ≥ 4 ACEs on the ACE-Q will demonstrate greater autonomic dysregulation during the MIST stress task, measured as significantly different HF HRV, compared to participants who report a history of < 4 ACEs. Two patterns of HRV reactivity will be considered maladaptive, as lower cardiovascular reactivity may suggest exhaustion and dampening due to chronic stress, and higher cardiovascular reactivity could indicate low parasympathetic vagal control, as seen in Figure 1.

Hypothesis 3

Compared to participants who report < 4 ACEs on the ACE-Q, participants who report a history of ≥ 4 ACEs will demonstrate greater autonomic dysregulation during a ten-minute recovery period, measured as inadequate return to baseline HRV upon completion of the MIST stress task.

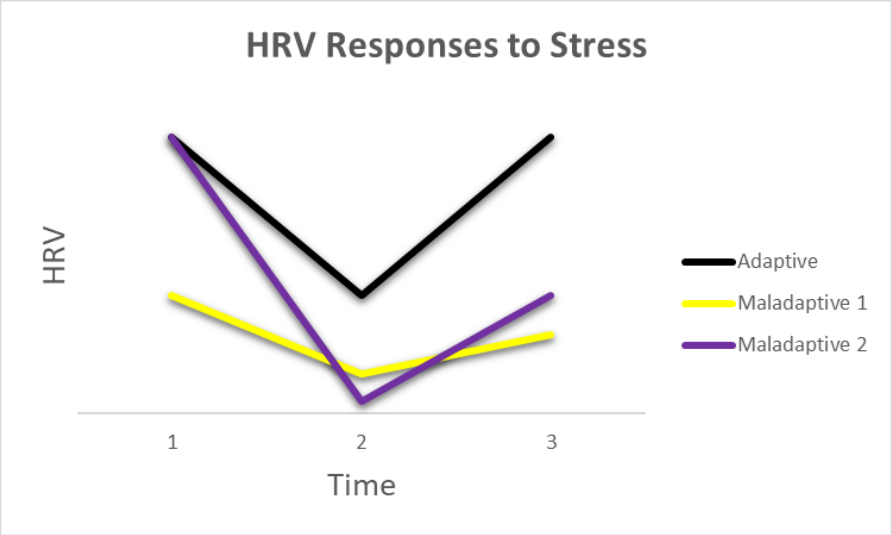

Figure 1. Adaptive versus maladaptive HRV responses. The image demonstrates physiological responses to stress at baseline, task, and recovery periods.

Aim 2

To assess the relationship between ACEs and various forms of psychopathology, as well as other transdiagnostic cognitive and affective factors, that are associated with cardiovascular stress reactivity.

Hypothesis 4

Participants who report a history of ≥ 4 ACEs on the ACE-Q will report a higher prevalence of psychopathology, measured by clinically-significant self-report scales of depression (PHQ-9), anxiety (GAD-7), post-traumatic stress (PCL-5), and attention deficit-hyperactivity disorder (ASRS-5).

Hypothesis 5

Participants who report a history of ≥ 4 ACEs on the ACE-Q will have a higher prevalence of cognitive and affective difficulties, measured as increased scores of perceived

stress (PSS), emotion regulation difficulties (DERS-18), disordered eating (ESP), and behavioral inhibition/avoidance (BIS/BAS).

Aim 3

To evaluate the relationship between ACEs and different lifestyle/health behaviors related to maladaptive behavioral coping and poorer physical health.

Hypothesis 6

Participants who report a history of ≥ 4 ACEs on the ACE-Q will have a higher prevalence of maladaptive behavioral coping, measured as decreased exercise (GLTEQ) and increased scores of sleep difficulties (PSQI), alcohol use (AUDIT-C), smoking (FTQ), nicotine use (EDS), substance misuse (DAST-10), and risky sex behaviors (SRS).

Hypothesis 7

Participants who report a history of ≥ 4 ACEs on the ACE-Q will have a higher prevalence of indicators of poor physical health, such as lower self-rated health (*poor, fair, good, very good, excellent*), and elevated scores of BMI (≥ 25), blood pressure (systole ≥ 120; diastole ≥ 80), somatic symptoms (PHQ-15), and healthcare utilization over the past 12 months.

Proposed Methods

Participants

For the proposed study, we seek to enroll approximately 3,000 participants to complete online survey measures, and a subsample of 158 participants for an in-person laboratory session that includes physical measurements and electrocardiogram (ECG) monitoring. Eligible participants will be undergraduate students (ages 18-25) at East Carolina University enrolled in

the ECU Psychology Department Experimentrak subject pool for their Introductory Psychology course. We aim to enroll a sample that is representative of the ECU student population, which is approximately 59% female, 66% white/Caucasian, 16% black/African American, 7% Hispanic/Latino, 3% Asian, and 8% other/unknown. Additionally, participants will be asked to refrain from exercise for 12 hours prior to laboratory testing, and to withhold from ingesting products containing tobacco, nicotine, or caffeine for at least one hour before testing.

Self-Report Measures

Survey measures will be completed online through Qualtrics, and include a brief demographic and medical history questionnaire, a questionnaire of adversity in childhood (ACE-Q), and measures of psychopathology (PHQ-9, GAD-7, PCL-5, ASRS-5), cognitive and affective difficulties (PSS, DERS-18, ESP, BIS/BAS), and resilience (BRS). In addition, participants will be asked about lifestyle and health behaviors such as exercise (GLTEQ), sleep (PSQI), alcohol use (AUDIT-C), substance misuse (DAST-10), tobacco smoking (FTS), e-cigarette use (EDS), and sexual risk-taking (SRS). The present study will also assess for objective and subjective measures of general physical health by asking participants about somatic symptoms (PHQ-15), self-rated health, and healthcare utilization.

Demographic and Medical History Questionnaire

Participants will be asked to provide basic demographic information about their age, sex, gender, sexual preference, and race/ethnicity. In addition, participants will be screened for a history of a diagnosed heart condition or psychological disorder, and will be asked to provide a list of active prescription medications; responses will be assessed as potential exclusionary criteria by study personnel.

Adverse Childhood Experiences Questionnaire (ACE-Q)

The ACE-Q was initially implemented by Felitti and colleagues (1998), and was developed as an epidemiological measure to better understand the relationship between early psychosocial stress and subsequent health outcomes. The questionnaire includes ten self-report items concerning family dysfunction and abuse/neglect, in which participant total scores are quantified as the cumulative number of affirmative responses to instances of adverse events prior to age 18; accordingly, each participant's ACE-Q score will fall between 0 and 10, with higher scores indicating more adversity experiences. Extensive research has confirmed a positive dose-response relationship between ACE scores and negative health outcomes, and a score of four or higher has consistently yielded the strongest associations with poorer health (Felitti et al., 1998). As such, an ACE-Q score of four has been regarded as a clinical cutoff point.

While the ACE-Q has been widely disseminated as a screening tool to address a major public health concern, the ACEs literature yields a limited understanding of the ACE-Q's psychometric properties. To date, only two studies address the measure's test-retest reliability, and validity statistics have not been well-examined. The earliest study that assessed test-retest reliability found that weighted kappa coefficients for ACE score between Time 1 and Time 2 were acceptable (Cohen's $\kappa = .64$) (Dube, Williamson, Thompson, Felitti, & Anda, 2004). While this questionnaire deviated slightly from the ACE-Q, kappa coefficients for individual ACE-Q items were emotional abuse (.66), physical abuse (.63), sexual abuse (.69), household substance abuse (.75), household mental illness (.51), mother treated violently (.78), parental separation (.86), and household member incarceration (.46). Due to the diversity of adversity experiences under the ACEs construct, internal consistency between items is not a useful metric to assess the measure's psychometric properties. That said, interrelatedness between items have been

identified, such that the endorsement of one ACE-Q item increased the odds for endorsing additional items; and in one large public health sample (n = 8,629), 87% (median) of the respondents who endorsed one ACE-Q item endorsed at least one additional item (Dong et al., 2004). Furthermore, the adjusted odds of endorsing additional ACE-Q items increased significantly as the reported number of ACEs increased, suggesting a coherence within the overall ACEs construct.

Measures of validity and reliability are known obstacles for assessing instances of abuse, neglect, maltreatment, and family dysfunction with retrospective self-report questionnaires. That said, in one recent sample of college athletes, analysis of test-retest reliability was found to be acceptable (r = .71), and a higher stability coefficient was found among items concerning household dysfunction (r = .65) compared to items of abuse and neglect (r = .52) (Zanotti et al., 2018). Nonetheless, issues of validity and test-retest reliability have been found to be a common challenge in the childhood abuse and maltreatment literature, and accordingly, several consideration must be made while interpreting participant responses. For instance, issues such as time lapse since adverse events, subject sensitivity, and memory impairments (potentially associated with significant stressful or traumatic events) may be present, and some literature has suggested both underestimations and underreporting of instances of abuse among respondents (Dube et al., 2004). Additionally, the dichotomous response options on the ACE-Q do not allow for information about frequency and intensity of occurrences. The implications of each of these limitations must be considered during data collection and statistical analysis.

Patient Health Questionnaire 9-item (PHQ-9)

The PHQ-9 (Kroenke, Spitzer, & Williams, 2001) is a brief self-report measure that is commonly used to assess depressive symptoms in clinical and research settings. Respondents are

screened on nine items based on DSM-IV diagnostic criteria for depression over the past two weeks, with scores ranging from 0 (*Not at all*) to 3 (*Nearly every day*). Scores are then totaled, with higher scores reflecting greater levels of depressive symptoms. Total scores of 10 or higher were found to have both a sensitivity and specificity of 88% for major depression, and as such, a total score of 10 or higher has been accepted as a reliable clinical cutoff point (Kroenke et al., 2001).

Generalized Anxiety Disorder 7-item (GAD-7)

The GAD-7 (Spitzer, Kroenke, Williams, & Löwe, 2006) is a seven-item self-report measure that is commonly used to assess anxiety symptoms in clinical and research settings. Respondents are asked to rate items from 0 (*Not at all sure*) to 3 (*Nearly every day*) to indicate the frequency of various anxiety symptoms over the previous two-week period. The questionnaire has been found to have high internal consistency (Cronbach $\alpha = .92$) and test-retest reliability ($r = .83$). During the validation of the GAD-7, the authors identify a total score of 10 or higher as a clinical cutoff with good sensitivity (89%) and specificity (82%).

PTSD Checklist for DSM-5 (PCL-5)

The PCL-5 is a 20-item PTSD symptom screening tool that asks examiners to report issues that have taken place within the past four weeks (Weathers, Litz, Keane, Palmieri, Marx, & Schnurr, 2013). The questionnaire was revised to coincide with the changes in symptom criteria between the DSM fourth and fifth editions, and accordingly, each of the 20 items on the PCL-5 are derived from DSM-5 symptom criteria for PTSD. Participants are asked to think about a stressful experience and rate PTSD-related symptoms over the past month from 0 (*Not at all*) to 5 (*Extremely*). A participant's total score ranges from 0-80, and a cutoff score of 31

indicates the likely presence of sufficient symptoms to meet the DSM-5 criteria for PTSD. The psychometric properties of the revised PCL-5 were evaluated following two studies of trauma-exposed college students, and results demonstrated high internal consistency (Cronbach $\alpha = .94$), test-retest reliability ($r = .82$), convergent validity ($rs = .74$ -.85) and discriminant validity ($crs =$ .31-.60) (Blevins, Weathers, Davis, Witte, & Domino, 2015).

Adult ADHD Self-Report Screening Scale for DSM-5 (ASRS-5)

The ASRS-5 (Ustun et al., 2017) is a six-item self-report measure of ADHD symptoms that asks respondents to rate the frequency of common ADHD symptoms over the past six months using a scale of 0 (*Never*) to 4 (*Very Often*). The questionnaire was developed as a screening tool, and was adapted from a previous version (ASRS-v1.1) to coincide with changes to diagnostic criteria upon release of the DSM-5. Use of the updated ASRS-5 yielded an identification of ADHD cases from non-cases with 91% sensitivity and 96% specificity in a general population sample, and accordingly, a clinical threshold for scores of 14 or higher has been recommended.

Perceived Stress Scale (PSS)

The PSS (Cohen, Kamarck, & Mermelstein, 1983) is a 10-item self-report measure that was developed as a tool to assess how situations and emotions influence levels of perceived stress. Respondents are asked to rate items on a scale from 0 (*Never*) to 4 (*Very often*) as they apply to situations within the past four weeks. After accounting for reverse-scored items (Items 4, 5, 7 and 8), responses are summed, and total scores are categorized as estimations of low stress (0-13), moderate stress (14-26), and high stress (27-40). Initial analysis of internal

consistency appeared moderate-high in predominantly college student samples (Cronbach α = .84-.86), and test-retest reliability was also found to be high (*r* = .85).

Difficulties in Emotion Regulation Scale 18-item (DERS-18)

The DERS-18 is an 18-item short form of the longer 36-item DERS questionnaire (Gratz & Roemer, 2004). This self-report measure asks respondents to respond to items concerning various emotion abilities on a 1 (*Almost never*) to 5 (*Almost always*) scale, which address areas of emotion regulation such as emotional awareness, emotional acceptance, impulse/behavioral control, and use of beneficial emotion regulation strategies. Analysis of the measure's psychometric properties indicated high internal consistency (Cronbach α = .91) of the combined scales, with subscales ranging from .77 to .90 (Victor & Klonsky, 2016). Further, authors reported high concurrent validity (*r* = .98) and significant correlations with difficulty regulating both negative and positive emotions.

Eating Disorder Screen for Primary Care (ESP)

The ESP is a five-item self-report measure that was developed to screen patients for potential eating disorders in a primary care setting (Cotton, Ball, & Robinson, 2003). The questionnaire asks respondents to answer *Yes/No* to common questions related to disordered eating, and two or more affirmative responses indicated high sensitivity (100%) and adequate specificity (71%) in the primary care setting. Subsequently, the ESP was found to be more effective at ruling out disordered eating in a mixed college student and primary care sample (Kagan & Melrose, 2003).

Brief Resilience Scale (BRS)

The BRS (Smith et al., 2008) is a six-item self-report measure of resilience, or one's ability to recover from stress and adversity. Items are labeled from 1 (*Strongly Agree*) through 5 (*Strongly Disagree*), and responses are averaged, with higher scores indicating greater resilience. The authors' analysis of psychometric properties indicated high internal consistency among four groups (Cronbach α = .80-.91).

Behavioral Inhibition/Behavioral Activation Scales (BIS/BAS)

The BIS/BAS scales (Carver & White, 1994) are comprised of 24 self-report items, and were developed to assess the influence of emotion on motivational behaviors. Response options range from 1 (*Very true for me*) to 4 (*Very false for me*), and the questionnaire is subdivided into four sections: BAS Drive (four items), which examines respondents' willingness to pursue goals; BAS Fun-Seeking (four items), which provides insight about respondents' tendency to seek out new and exciting stimuli; BAS Reward-Responsiveness (five items), which assesses motivation for reward; and BIS (seven items), which is used to evaluates one's responses to potentially punishing events. There are also four filler questions that do not factor onto any of the four subscales. Analysis of reliability reflects adequate internal consistency between items (Cronbach α = .66-.76) (Carver & White, 1994) and test-retest reliability across scales (r = .68-.72) (Sutton & Davidson, 1997). These estimates of reliability were replicated in clinical samples, which also yielded appropriate convergent and concurrent validity (Campbell-Sills, Liverant, & Brown, 2004). Additionally, the same authors found a moderate-strong positive correlation for BIS scores with neuroticism (r = .75) and depression (r = .48), and a moderate positive correlation between BAS scores and positive affect (r = .43).

Godin Leisure-Time Exercise Questionnaire (GLTEQ)

The GLTEQ is a self-report measure that asks respondents about exercise frequency over a typical 7-day period (Godin, 1969). Participants are asked to tally the total number of mild, moderate, and strenuous bouts of leisure-time exercise (at least 15-minutes in duration), and are provided with prototypical activities for each category, respectively. To produce a weighted total score, response tallies are multiplied by three (mild), five (moderate) and nine (strenuous), and then categorical scores are summed. This yields a Godin Scale Score, and cutoff values are set at 0-13 (Insufficiently Active/Sedentary), 14-23 (Moderately Active), 24 and above (Active). Recent validation studies have supported the use of the GLTEQ, and conclude that the measure has greater sensitivity to detect moderate-strenuous exercise rather than mild exercise intensity (Motl et al., 2018).

Pittsburgh Sleep Quality Index (PSQI)

The PSQI is a 19-item self-report questionnaire that measures subjective sleep quality throughout the past month (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI assesses several factors related to sleep including subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction over the last month on a scale from 0 (*Not during the past month*) to 3 (*Three or more times a week*). The sum of scores are calculated to derive a global PSQI score ranging from 0-21, with scores greater than 5 reflecting poor sleep quality. While the PSQI has been shown to have good internal consistency (Cronbach $\alpha = .80$) (Carpenter & Andrykowski, 1998) and test-retest reliability ($r = .78$), scores have not been found to reliably predict objective sleep data, such as those derived from polysomnography (Backhaus, Junghanns, Broocks, Riemann, & Hohagen, 2002). Therefore, the PSQI is most appropriately used as to estimate subjective sleep quality.

Alcohol Use Disorders Identification Test - Consumption (AUDIT-C)

The AUDIT was developed by the World Health Organization as a self-report measure of hazardous or harmful alcohol use, and screens for alcohol intake, dependence, and problems related to consumption (WHO, 1982). Since its initial use, the AUDIT has been adapted into several shortened versions, and item reductions have been found to maintain satisfactory psychometric properties (Meneses-Gaya, Zuardi, Loureiro, & Crippa, 2009). One such adaptation is the AUDIT-C, which has been widely validated as a brief screening tool for problem drinking, and performed better than the AUDIT for detecting heaving drinking (Bush, Kivlahan, McDonell, Fihn, & Bradley, 1998). The AUDIT-C is comprised of three self-report items, and responses are marked on a 0-4 scale, yielding potential scores from 0-12. Furthermore, for use among college students, the AUDIT-C has been found to have better detection of at-risk drinking than the AUDIT; and due to the emergence of gender differences in drinking among respondents, the authors recommend that cut-off scores be set at 7 for males and 5 for females (Demartini & Carey, 2012). These gender-based cut-off scores were further supported as an indicator of problematic drinking among college students after evaluation of at-risk drinking in a university primary care setting (Campbell, C. E. & Maisto, 2018).

Drug Abuse Screening Test 10-item (DAST-10)

The DAST-10 is a 10-item self-report measure that was developed as a condensed version of the DAST-28 (Skinner, 1982) to be used as a screening tool to assess both prescription and illicit substance misuse. Review of its psychometric properties indicates high internal consistency (Cronbach $\alpha = .94$) among psychiatric patients (Carey, Carey, & Chandra, 2003) and good test-retest reliability ($r = .78$) when compared to the longer DAST-20 version ($r = .71$) (Cocco & Carey, 1998). Further, the DAST-10 was found to be appropriately sensitive to the

college undergraduate student population, with 85.6% of respondents endorsing at least one DAST-10 item, 49.2% endorsing two or more items, and 25% endorsing three or more items (McCabe, Boyd, Cranford, Morales, & Slayden, 2006).

Fagerstrom Tolerance Questionnaire (FTQ)

The FTQ (Fagerstrom, 1978) was developed as a tool to measure the severity of nicotine dependence among smokers. Accordingly, the FTQ has been found to reliably distinguish between light and heavy smokers when biochemically validated by other indicators of nicotine use, such as cotinine (Pomerleau, O. F., Fertig, & Shanahan, 1983). The FTQ has also been shown to have high test-retest reliability (r = .83) among an American sample (Pomerleau, C. S., Carton, Lutzke, Flessland, & Pomerleau, 1994).

E-Cigarette Dependence Scale 4-item (EDS-4)

The EDS is a self-report measure of e-cigarette dependence (Morean et al., 2018). Upon evaluation of psychometric properties, it performed similarly to the longer 22- and 8-item versions by demonstrating high internal consistency (Cronbach α = .86) and good concurrent validity. Items ask participants to respond on a scale from 0 (*Never*) to 4 (*Almost Always*), with higher scores indicating greater e-cigarette nicotine dependence.

Sexual Risk Survey (SRS)

The SRS (Turchik & Garske, 2009) is a 23-item self-report questionnaire that was developed to assess sexual risk-taking behaviors among college students. A recent meta-analysis of different measurement tools of high-risk sexual behaviors confirmed initial findings that the SRS has good internal consistency (Cronbach α = .61-.93) as well as high test-retest reliability (r = .93) (Mirzaei, Ahmadi, Saadat, & Ramezani, 2016).

Patient Health Questionnaire 15-item (PHQ-15)

The PHQ-15 is a 15-item self-report measure of somatic symptoms that asks respondents to answer on a scale from 0 (*Not bothered at all*) to 2 (*Bothered a lot*) regarding their experience with physical symptoms over the previous four weeks. The questionnaire was normed with the use of a predominantly primary care patient population, and items assess for the most common physical complaints reported in outpatient medical settings. Additionally, increased scores on the PHQ-15 were associated with decreases in symptom-related difficulty, sick days, healthcare utilization, and functional status (Kroenke, Spitzer, & Williams, 2002). As such, somatic symptom severity cutoff points are denoted as minimal (0-4), low (5-9), medium (10-14), and high (15-30).

Self-Rated Health

Self-rated health is a commonly used measure that can be used to estimate participants' subjective understanding of their overall health. Self-rated health measures typically utilize a brief single-item format. For the purposes of the present study, participants will be asked "In general, would you say your health is," with the item responses of "(1) poor," "(2) fair," "(3) good," "(4) very good," and "(5) excellent." This type of informal assessment tool has been widely used across healthcare and research applications, and test-retest reliability has been found to be moderate (r = .75) when participants from ages 20-80 years were asked on two occasions at approximately four weeks apart (Zajacova & Dowd, 2011).

Healthcare Utilization

Brief assessments of one's frequency of engagement with healthcare services can provide additional information about participants' general health status. For the present study,

participants will be asked to indicate the frequency over the last 12 months with which they have received services for both physical and mental health conditions, and responses will be separated categorically. Scores will be calculated as the total number of appointments attended, by category.

Laboratory Measurements and Procedures

Following the completion of online survey measures, participants will be invited into a laboratory setting to complete measures of height, weight, and blood pressure, as well as the MIST stress task during HRV monitoring. It is likely that the sample will yield more responses for the comparison group (< 4 ACEs) than the ACEs group (≥ 4 ACEs), therefore, an oversampling method will be used to capture more participants with clinically significant ACE scores. For respondents who report < 4 ACEs, these participants will be randomly selected for laboratory testing to act as the comparison group. All participants who report ≥ 4 ACEs will be invited to participate in the laboratory portion. The proportion of participants in each group will be monitored and sampling methods increased/decreased, as indicated.

Upon arrival for lab testing, participants will be informed by a lab assistant that they are being asked to take part in a study about how mathematic abilities are related to other psychological factors. Next, participants will also be asked if they have followed requests to refrain from exercise within the 12 hours prior to testing and from ingesting tobacco, nicotine, or caffeine within 60 minutes prior to testing; responses will be recorded and later analyzed for confounding effects. After completing the informed consent process, participants will have their height and weight digitally measured on a DETECTO research scale by a lab assistant. Then, after a five-minute resting period, participants will have their blood pressure measured with the use of a CARESCAPE V100 device, applying the cuff placement and body positioning

procedural guidelines recommended by the American Heart Association (AHA) (Smith, L., 2005). Lab assistants will observe standardized procedures throughout testing.

Next, participants will be asked to complete the Montreal Imaging Stress Task (MIST) during HRV monitoring. Upon entry into the monitoring booth, participants will be connected to an electrocardiogram recording device and respiratory cuff; HRV will be measured with a Lead II ECG using a BIOPAC MP150, and will be processed with the AcqKnowledge software. Prior to starting the MIST, participants will be asked to sit quietly and observe a neutral stimulus for ten minutes while baseline HRV monitoring is performed. The lab assistant will leave the monitoring booth for this portion of psychophysiological monitoring, but upon completion, will re-enter and remain seated behind the participant in the booth for the subsequent Training, Control, and Experimental phases. Of note, the lab assistant will observe and record the times at which the participant begins and ends each task. Following baseline monitoring, a lab assistant will provide a brief orientation to the MIST task during the Training phase (2-5 minutes). This phase will provide examiners with brief practice, and the MIST program will analyze responses and appropriately set the difficulty level and time limit per-item during the Experimental (Stressor) phase.

After the completing the Training phase participants will proceed to a Control and Experimental condition, each approximately five minutes in duration, which both require participants to complete mental arithmetic computations. The first task is considered a Control phase, as it does not apply the same time restrictions and social-evaluative feedback of the Experimental phase. After completing the Control phase, participants will be asked by the lab assistant to provide a verbal response to a brief measure of task engagement (Level of task difficulty: 1-10). After their response is recorded, participants will begin the Experimental phase,

which is designed to generate the most pronounced stress response, and combines arithmetic calculations with an imposed time requirement and a social-evaluative stressor. Throughout this phase of the MIST, the computer monitor will occasionally prompt the examiner to improve their performance due to less than average correct and timely responses. If a participant questions the lab assistant for clarification, they will provide a standardized response such as "Just do your best." Finally, upon completion, the lab assistant will reorient the participant to a neutral stimulus and instruct the participant to sit quietly until the lab assistant returns. This will allow for ten minutes of HRV recording during the recovery period.

Once the MIST task has been completed, participants will be debriefed on the purpose of the study by a lab assistant. They will also be informed about aspects of deception that were utilized throughout data collection, and a lab assistant will explain that the same feedback was provided to all participants during the MIST task, regardless of performance. Participants will be given the opportunity to ask follow-up questions about their participation, and will be provided with a handout that details a list of resources for obtaining healthcare services, if desired.

Data Cleaning and Preparation

Following HRV data collection, laboratory assistants will analyze the data using an automated software by identifying instances of peak frequency of the high frequency (HF) band (0.15–0.4 Hz), known as R peaks. Quality control measures will be taken to correct for incorrect identification of R peaks by the AcqKnowledge software. HF power will be calculated in five-minute increments and then averaged accordingly to create each of the three data collection periods (baseline, stressor task, recovery). This method ensures uniformity in the length of each recording that contributes to the HF power calculation.

Once data has been screened and appropriate corrections have been made, HRV scores will be transformed using the percent deviation method, as is recommended to reduce the potential for Type I and Type II errors (Ellis, Sollers Iii, Edelstein, & Thayer, 2008). Using this transformation method, scores at each time period (baseline, stressor task, recovery) will be calculated as the deviation from the average HRV score across all time periods. When compared to other common transformation methods, a percent deviation transformation has been shown to indicate less false positives than reactivity transformations and less false negatives than log transformations. All of the collected data, including HRV analysis, blood pressure, physical measurements, and self-report survey measures will be compiled in Microsoft Excel and prepared for statistical analysis. Associations between covariates and factors of interest will be explored using correlation analyses, t-tests or chi square, as indicated. Results of these analyses will identify potential covariates.

Proposed Statistical Analysis

The primary outcome under examination is the relationship between ACEs and HF HRV, a measure of cardiovascular stress reactivity. Accordingly, a 2x3 mixed factorial ANCOVA design will be used to explore the interaction of the two levels of the independent variable (ACEs_Low [0-3]; ACEs_High [4-10] with measures of cardiovascular reactivity during three time periods of a stressor task (Baseline; Stressor Task; Recovery). Prior to ANCOVA analysis, covariates will be empirically chosen (e.g., age, sex, gender, BMI, exercise frequency [GLTEQ], depression [PHQ-9], anxiety [GAD-7]). Using the GPower.3.1 software, *a priori* power analysis determined that this study will require a total sample size of 158 participants to detect a medium effect ($f = 0.25$) for fixed effects, main effects, and interactions of the two ACE groups and each of the three time points (numerator $df = 2$, $\alpha = 0.05$, $\beta = 0.80$).

Secondary analysis will examine bivariate correlations between ACEs and participant scores on measures of psychopathology, as well as other common transdiagnostic measures of cognitive and emotion regulation difficulties. Correlation analysis will also examine the relationship between ACEs and lifestyle behaviors, in addition to other indicators of poorer physical health. Significant correlations will also be analyzed with regression analysis to identify potential ways in which ACE-Q scores may predict these various mental and physical health factors.

Current Study

Due to logistical barriers imposed by the COVID-19 pandemic, psychophysiological laboratory data collection was not completed. Accordingly, study aims, hypotheses, and statistical analyses were adjusted to focus on aspects of the research question that could be addressed using online survey data only. Therefore, our former focus on heart rate variability (HRV) analysis has been removed. The following analyses focus on self-reported symptoms of mental health, physical health, and lifestyle health behaviors to better-understand their associations with adverse childhood experiences (ACEs) in emerging adults.

Aims and Hypotheses

Aim 1

To assess the relationship between ACEs and various forms of psychopathology, as well as other transdiagnostic cognitive, behavioral, and affective factors.

Hypothesis 1

Participant scores on the ACE-Q will be positively associated with increased symptoms of depression (PHQ-9), anxiety (GAD-7), post-traumatic stress (PCL-5), attention deficit-hyperactivity disorder (ASRS-5).

Hypothesis 2

Participant scores on the ACE-Q will be positively associated with increased cognitive, behavioral, and emotion regulation difficulties, measured as increased scores of perceived stress (PSS), emotional difficulties (DERS-18), disordered eating (ESP), and behavioral inhibition (BIS); we predict that the ACE-Q will be negatively associated with resilience (BRS) and behavioral approach (BAS) subscales of Drive, Fun Seeking, and Reward Responsiveness.

Aim 2

To evaluate the relationship between ACEs and lifestyle behaviors associated with poorer physical health, as well as other indicators of poorer health.

Hypothesis 3

Participant scores on the ACE-Q will be positively associated with lifestyle behaviors associated with poorer physical health, measured as decreased self-reported exercise (GLTEQ) and increased scores of sleep difficulties (PSQI), alcohol use (AUDIT-C), e-cigarette use (EDS), substance misuse (DAST-10), and risky sex behaviors (SRS).

Hypothesis 4

Participant scores on the ACE-Q will be positively associated with indicators of poor physical health, such as lower self-rated health (poor, fair, good, very good, excellent), and increased self-report of somatic symptoms (PHQ-15) and healthcare utilization over the past 12 months.

Aim 3

Through exploratory analysis based on the results of Aims 1 and 2, to better understand the association between the ACE-Q subscales – categorized as Childhood Maltreatment (items 1-5) and Household Dysfunction (items 6-10) – and measures of mental and physical health that were significantly associated with the ACE-Q.

Hypothesis 5

Participant scores on the Child Maltreatment subscale, compared to scores on the Household Dysfunction subscale, will be more strongly associated with scores on mental health

measures of depression (PHQ-9), anxiety (GAD-7), post-traumatic stress (PCL-5), attention deficit-hyperactivity disorder (ASRS-5), emotion dysregulation (DERS-18), perceived stress (PSS), and disordered eating (ESP).

Hypothesis 6

Participant scores on the Child Maltreatment subscale, compared to scores on the Household Dysfunction subscale, will be more strongly associated with scores on physical health measures of sleep difficulties (PSQI), illicit substance use (DAST-10), e-cigarette use (EDS), and somatic symptoms (PHQ-15).

Methods

Participants and Demographics

For the present study, 236 undergraduate students between the ages of 18 and 23 years completed an online survey. The survey was made accessible via the ECU Psychology Department Experimentrak subject pool, and upon completion, students received participation credit for an Introductory Psychology course. Participant data were collected over the span of the Spring 2020 semester. Upon analysis of demographic information, the sample was found to be 51.8% female sex-at-birth, 48.2% male sex-at-birth, and 0.0% intersex. Additionally, 52.6% of respondents identified as female gender, 47.4% identified as male gender, and 0.0% identified as transgender, gender-nonconforming, or preferred not to answer. The median age of the total sample was 19.0 years. Racial/ethnic characteristics were 66.2% White/Caucasian, 22.1% Black/African American, 4.4% multiracial, 3.9% Hispanic or Latino, 3.1% Asian, 0.9% American Indian or Alaska Native, and 0.4% Other. These findings are consistent with recent student racial/ethnic demographic data provided by the university, which suggests a sample that is representative of the overall student population. Responses on sexual orientation showed that participants were 91.7% heterosexual, 4.4% bisexual, 1.8% questioning, and 0.9% gay/lesbian; 1.3% of participants preferred not to answer. On a proxy measure of SES, 8.6% of participants reported that their families receive some form of government financial assistance, including various food and housing resources.

Data Cleaning

Following the data collection period, the final dataset was exported to SPSS for further data cleaning and analysis. Prior to analysis, participant data was thoroughly checked for validity. Accordingly, participants whose response times fell within the lowest quartile (less than

17.2 minutes) and those with less than 90% survey completion were removed from the sample, reducing the sample from 236 to 170 participants. Of the remaining sample, data was checked for duplicates and invalid responses, such as outliers and nonsensical text. When necessary, invalid responses to individual survey items were deleted. Additional data cleaning was performed so that participant text responses were usable for analysis. When appropriate, text responses were converted to numeric responses (e.g., everyday = 7 days) and means were used to replace number ranges that were entered as text (e.g., 3-5 = 4 days).

Descriptive Statistics

After correcting for unreliable, invalid, and missing data, descriptive statistics were analyzed for each measure of the online survey. To highlight certain measures of greater importance, some measures that did not reach statistical or clinical significance have been removed from this section.

Adverse Childhood Experience Questionnaire (ACE-Q)

Descriptive statistics of ACE-Q scores in the final sample (n=170) yielded a mean score of 1.75 out of 10 (sd=2.08). The frequency of ACE-Q scores were: 0 (63; 37.1%), 1 (35; 20.6%), 2 (27; 15.9%), 3 (17; 10.0%), 4 (7; 4.1%), 5 (9; 5.3%), 6 (5; 2.9%), 7 (3; 1.8%), 8 (3; 1.8%), 9 (0; 0.0%), 10 (1; 0.6%). Using the suggested clinical cutoff score of ≥ 4, 28 participants (16.5%) reached this threshold, and 142 (83.5%) reported an ACE score from 0-3. Additionally, within the final sample, more participants endorsed affirmative responses on items within the Household Dysfunction subcategory (ACE-Q items 6-10: 96; 56.5%) than items within the Childhood Maltreatment subcategory (ACE-Q items 1-5: 64; 37.6%).

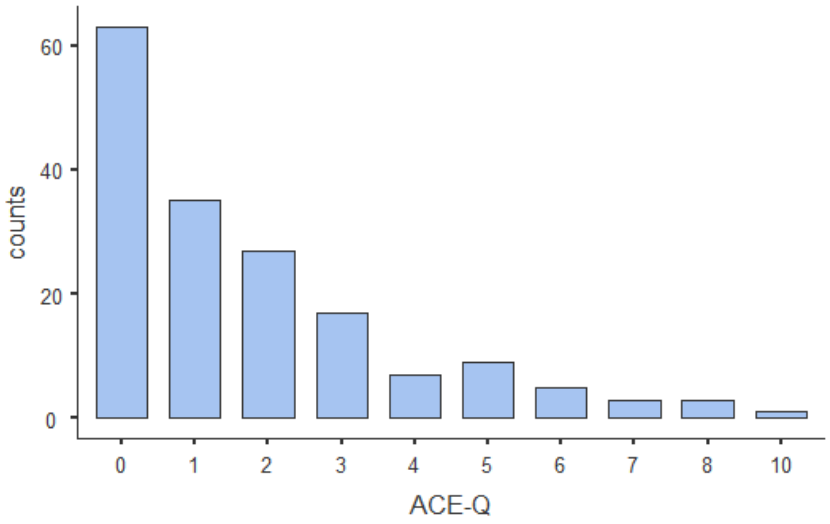

Figure 2. Frequency of Adverse Childhood Experiences.

Patient Health Questionnaire 9-item (PHQ-9)

Upon analysis of depression symptoms endorsed on the PHQ-9, the final sample revealed a mean score of 6.41 out of 27 (*sd*=5.63). Based on the suggested clinical cutoffs, 80 participants (47.1%) did not screen positive for depression, while 47 (27.6%) screened positive for mild depression, 26 (15.3%) screened positive for moderate depression, 9 (5.3%) screened positive for moderately severe depression, and 8 (4.7%) screened positive for severe depression.

Generalized Anxiety Disorder 7-item (GAD-7)

Analysis of participant self-reported anxiety symptoms on the GAD-7 indicated a mean score of 5.25 out of 21 (*sd*=4.92). Using the suggested clinical cutoff scores, 92 participants (54.1%) did not exhibit clinically significant anxiety symptoms, 41 (24.1%) exhibited mild anxiety symptoms, 29 (17.1%) exhibited moderate anxiety, and 8 (4.7%) exhibited severe anxiety.

PTSD Checklist for DSM-5 (PCL-5)

Self-reported post-traumatic stress symptoms on the PCL-5 revealed a mean score of 13.26 out of 80 (*sd*=14.44). Using the clinical cutoff of 33, which has been used to represent a provisional diagnosis of PTSD, 147 participants (86.5%) fell below the cutoff score and 23 (13.5%) met criteria for PTSD.

Adult ADHD Self-Report Screening Scale for DSM-5 (ASRS-5)

Participant responses to items on a measure of ADHD symptoms indicated a mean score of 6.99 out of 24 (*sd*=4.26). Using the clinical cutoff score (≥14), 158 participants (92.9%) fell below, and 12 (7.1%) exceeded, this threshold.

Perceived Stress Scale (PSS)

Responses on items of the PSS revealed a mean score of 17.85 out of 40 (*sd*=6.38). Of these responses, 28 participants (16.5%) recorded scores within the low stress range (0-13), 136 participants (80.0%) recorded scores within the moderate stress range (14-26), and 6 participants (3.5%) recorded scores within the high stress range (27-40).

Difficulties with Emotion Regulation Scale 18-item (DERS-18)

Responses on this measure of emotion dysregulation showed a mean score of 38.62 out of 90 (*sd*=13.63). While there is no suggested clinical cutoff score, 134 participants (78.8%) recorded scores below, and 36 (21.2%) recorded scores above, 50; scores above 50 could be interpreted to indicate concerning levels of emotion regulation difficulties.

Eating Disorder Screen for Primary Care (ESP)

Responses on a measure of disordered eating symptoms indicated a mean score of 1.38 out of 5 (*sd*=0.10). Based on the suggested cutoff score (≥2) used to indicate concern for disordered eating in the primary care setting, the distribution of scores showed that 97 participants (57.0%) endorsed no concerns for disordered eating history, and 73 participants (43.0%) endorsed some concerns for disordered eating history.

Brief Resilience Scale (BRS)

Due to the inclusion of participants with ≥ 90% of survey completion, responses on the BRS were taken from 68 participants in the final sample (*n*=170). Responses to items of resilience showed a mean score of 20.66 out of 30 (*sd*=5.68), with higher scores representing greater participant resilience.

Behavioral Inhibition and Behavioral Activation Scales (BIS/BAS)

Participant responses to items of behavioral inhibition yielded a mean score of 19.79 out of 28 (*sd*=4.03). Responses to items of behavioral activation are broken down by subscale; responses to the BAS-Drive subscale demonstrated a mean score of 11.43 out of 16 (*sd*=2.20), responses to the BAS-Reward Responsiveness subscale revealed a mean score of 17.49 out of 20 (*sd*=1.87), and responses to the BAS-Fun Seeking subscale revealed a mean score of 12.44 out of 16 (*sd*=2.20).

Godin Leisure-Time Exercise Questionnaire (GLTEQ)

Responses on a measure of exercise over the past week were broken down into categories of mild, moderate, and strenuous exercise. After scores were weighted and totaled, results indicated a mean score of 49.69 (*sd*=28.39). Twelve participants (7.1%) fell within the Sedentary

range (0-13), 14 participants (8.2%) fell within the Moderately Active range (14-23), and 144 participants (84.7%) fell within the Active range (24 and above).

Pittsburgh Sleep Quality Index (PSQI)

Due to the inclusion of participants with ≥ 90% of survey completion, responses on the PSQI were taken from 139 participants in the final sample (*n*=170). Responses on a measure of sleep difficulties demonstrated a mean score of 6.98 out of 21 (*sd*=3.78). Using the suggested cutoff score of ≥ 5 as an indicator of poor sleep quality, 94 participants (67.6%) reached this threshold.

Alcohol Use Disorders Identification Test – Consumption (AUDIT-C)

Due to the inclusion of participants with ≥ 90% of survey completion, responses on the AUDIT-C were taken from 138 participants in the final sample (*n*=170). Responses on a measure of alcohol use indicated a mean score of 4.01 out of 12 (*sd*=2.29). Sixty-eight participants (49.3%) scored below the suggested cutoff for hazardous drinking (≥4), and 70 participants (50.7%) met the suggested criteria for hazardous drinking.

Drug Abuse Screening Test 10-item (DAST-10)

Participant responses on this measure of illicit substance use demonstrated a mean score of 1.74 out of 10 (*sd*=0.12). Responses were distributed such that 13 participants (7.6%) scored within the “no problem” range, 123 participants (72.4%) scored in the “low problem” range, 28 participants (16.5%) scored in the “moderate problem” range, 5 participants (3.0%) scored in the “substantial problem” range, and 1 participant (0.6%) scored in the “severe problem” range.

Fagerstrom Tolerance Questionnaire (FTQ)

Only eight participants (4.7%) in the total sample (*n*=170) reported that they smoke cigarettes on most days. Due to a low number of responses this measure was not included in the results.

E-cigarette Dependence Scale 4-item (EDS-4)

Forty-nine participants (28.8%) indicated that they use an e-cigarette on most days. Reponses to items on this measure of e-cigarette dependence yielded a mean score of 2.10 out of 16 (*sd*=0.28). One hundred eleven participants (65.3%) indicated that they "never" experience symptoms of e-cigarette dependence; on average, 21 participants (12.4%) endorsed that they "rarely" experience symptoms of e-cigarette dependence, 17 participants (10.1%) endorsed that they "sometimes" experience symptoms of e-cigarette dependence, and 4 participants (2.4%) endorsed that the "often" experience symptoms of e-cigarette dependence.

Sexual Risk Survey (SRS)

Due to the inclusion of participants with ≥ 90% of survey completion, responses on the SRS were taken from 134 participants in the final sample (*n*=170). Participant responses were separated into four ordinal categories to reduce variability, in which higher scores indicated higher sexual risk-taking behaviors. Fifty-seven participants (42.5%) scored in the lowest risk category, 36 participants (26.9%) scored in the second lowest risk category, 26 participants (19.4%) scored in the second highest risk category, and 15 participants (11.2 %) scored in the highest risk category.

Patient Health Questionnaire 15-item (PHQ-15)

Due to the inclusion of participants with ≥ 90% of survey completion, responses on the PHQ-15 were taken from 140 participants in the final sample (*n*=170). Self-reported somatic

symptoms on this measure yielded a mean score of 6.58 out of 30 (*sd*=4.60). The breakdown of participant responses were such that 49 participants (35.0%) fell in the category of minimal somatic symptom severity (Total Score=0-4), 58 participants (41.4%) fell in the category of low symptom severity (Total Score=5-9), 25 participants (17.9%) fell in the category of medium symptom severity (Total Score=10-14), and 8 participants (5.7%) fell in the category of high symptom severity (Total Score=15-30).

Self-Rated Health

On a subjective measure of health, 28 participants (16.5%) rated their health as "excellent", 15 participants (8.8%) rated their health as "very good", 54 participants (31.8%) rated their health as "good", 72 participants (42.4%) rated their health as "fair", and 1 participant (0.6%) rated their health as "poor".

Healthcare Utilization

On a measure of previous 12-month healthcare utilization, calculated as the total number of physical and mental health visits, 71 participants (42.0%) reported 0 visits, 60 participants (35.5%) reported 1-3 visits, 22 participants (13.0%) reported 4-9 visits, and 16 participants (9.5%) reported 10 or more visits.

Statistical Analysis and Results

Aim 1

The primary aim of this study was to examine the relationship between ACEs and various forms of psychopathology in emerging adults, as well as other transdiagnostic cognitive, behavioral, and affective factors. For Hypothesis 1, Pearson correlations and hierarchical linear regression analyses were used to examine the relationships between ACE scores and self-reported symptoms of depression, anxiety, PTSD, and ADHD. Concerning Hypothesis 2, the same correlation and regression analyses were used to assess the relationship between ACE scores and measures of emotion dysregulation, perceived stress, resilience, and behavioral inhibition/approach.

Hypothesis 1 – Correlation Analysis

Consistent with Hypothesis 1, correlation analysis demonstrated moderate-strong positive correlations between scores on the ACE-Q and symptoms of depression (PHQ-9; r=.42), anxiety (GAD-7; r=.41), PTSD (PCL-5; r=.48), and ADHD (ASRS-5; r=.40); all were significant at the p<.01 level. These associations can be seen in Table 1.

	ACE-Q	PHQ-9	GAD-7	PCL-5	ASRS-5
ACE-Q	—				
PHQ-9	0.421 ***	—			
GAD-7	0.412 ***	0.724 ***	—		
PCL-5	0.479 ***	0.723 ***	0.712 ***	—	
ASRS-5	0.397 ***	0.663 ***	0.652 ***	0.647 ***	—

Table 1. Hypothesis 1 Correlation Matrix. Note: * p < .05, ** p < .01, *** p < .001

Hypothesis 1 – Regression Analysis

Next, hierarchical linear regression analyses were completed to further examine the relationship between the ACE-Q and screening measures for depression, anxiety, PTSD, and ADHD. In the first step of each regression model, we controlled for demographic covariates – including gender, race/ethnicity, and SES variables – to see if they significantly accounted for variance on scores for each screening measure. In the second step of each model, the ACE-Q was added as a predictor variable to assess for additional variance in the scores for each screening measure, respectively. The second step was also assessed for significant predictor variables in the total model. The results of these hierarchical linear regression analyses are reported below.

Results for the PHQ-9 demonstrated that demographic covariates in the first step accounted for 7.7% of variance in depression scores, which was significantly different than zero ($F(3, 166) = 4.63$, $p<.01$). When the ACE-Q was added in the second step, this accounted for an additional 15.3% of variance ($F\Delta(1,165) = 32.87$, $p<.01$). In the total model, only ACE-Q scores ($B = 1.10$, $p<.01$) and race/ethnicity ($B = 2.23$, $p<.01$) were significant predictors of scores on the PHQ-9, such that being White (vs non-White) and higher ACE-Q scores predicted higher self-reported depressive symptoms over the past two weeks. See Appendix A for the effects of each predictor variable.

For the GAD-7, results showed that demographic covariates in the first step accounted for 7.6% of variance in anxiety scores, which was significantly different than zero ($F(3, 166) = 4.58$, $p<.01$). When the ACE-Q was added in the second step, this accounted for an additional 15.4% of variance ($F\Delta(1,165) = 33.10$, $p<.01$). In the overall model, ACE-Q scores ($B = 0.97$, $p<.01$) and race/ethnicity ($B = 2.23$, $p<.01$) significantly predicted participant scores on the GAD-7, such that higher ACE-Q scores and being White (vs non-White) predicted higher self-reported

anxiety symptoms over the past two weeks. See Appendix A for the effects of each predictor variable.

Regression analysis for the PCL-5 indicated that demographic covariates accounted for 2.8% of variance in PTSD scores, but this first step of the model was not statistically significant ($F(1,166) = 1.61$, $p = .19$). When the ACE-Q was added in the second step, this accounted for an additional 22.8% of variance, which was significantly different than zero ($F\Delta(1,165) = 50.69$, $p<.01$). Only ACE-Q scores were a significant predictor of PCL-5 scores in the overall model ($B = 3.44$, $p<.01$), such that higher ACE-Q scores predicted higher self-reported distress from PTSD symptoms over the past month. See Appendix A for the effects of each predictor variable.

Results for the ASRS-5 demonstrated that the demographic covariates accounted for 5.3% of variance in ADHD scores, and this was significantly different from zero $F(1,166) = 3.07$, $p=.03$). When ACE-Q scores were added in the second step, this accounted for an additional 14.0% of total variance, and this step was also statistically significant ($F(4, 165) = 28.51$, $p<.01$). In the total model, ACE-Q scores ($B = 0.79$, $p<.01$) and race/ethnicity ($B = 1.43$, $p=.03$) significantly predicted scores on the ASRS-5, such that higher ACE-Q scores and being White (vs non-White) predicted higher self-reported ADHD symptoms within the last 6 months. See Appendix A for the effects of each predictor variable.

Hypothesis 2 – Correlation Analysis

Correlation analysis for Hypothesis 2 yielded a moderate-strong positive correlation between scores on the ACE-Q and emotion dysregulation (DERS-18; $r=.42$). Additionally, results indicated a moderate positive association between the ACE-Q and disordered eating (ESP; $r=.26$) and perceived stress (PSS; $r=.24$). We observed a moderate negative association

between the ACE-Q and scores of behavioral approach-reward responsiveness (BAS-Reward Responsiveness; r=-.23). All were significant at the p<.01 level. Correlations between the ACE-Q and scores of behavioral inhibition, behavioral approach-drive, and behavioral approach-fun seeking did not reach significance. Further details concerning these associations can be seen in Figure 4.

	ACE-Q	DERS18	PSS	ESP	BRS	BIS	BAS_ Reward	BAS_ Drive	BAS_ Fun
ACE-Q	—								
DERS18	0.420 ***	—							
PSS	0.241 **	0.406 ***	—						
ESP	0.292 ***	0.458 ***	0.338 ***	—					
BRS	-0.232	-0.752 ***	-0.450 ***	-0.453 ***	—				
BIS	0.052	0.385 ***	0.272 ***	0.340 ***	-0.613 ***	—			
BAS_Reward	-0.234 **	-0.187 *	0.017	-0.050	0.228	0.209 **	—		
BAS_Drive	-0.062	-0.190 *	-0.011	-0.104	0.268 *	-0.070	0.449 ***	—	
BAS_Fun	0.024	0.056	0.036	-0.013	0.097	-0.131	0.364 ***	0.512 ***	—

Table 2. Hypothesis 2 Correlation Matrix. Note: * p < .05, ** p < .01, *** p < .001

Hypothesis 2 – Regression Analysis

Next, hierarchical linear regression analyses were completed to examine the relationship between the ACE-Q and emotion dysregulation, perceived stress, resilience, and behavioral inhibition/approach. In the first step of each regression model, we controlled for demographic covariates (gender, race/ethnicity, and SES) to see if they significantly accounted for variance on scores for each measure. In the second step of each model, the ACE-Q was added as a predictor variable to assess for additional variance in the scores for each measure, respectively. The second step was also assessed for significant predictor variables in the total model. The results of these hierarchical linear regression analyses are reported below.

Results for the DERS-18 demonstrated that demographic covariates in the first step accounted for 4.6% of variance in depression scores, but this did not reach statistical significance ($F(3,166) = 2.64$, $p=.51$). When the ACE-Q was added in the second step, this accounted for an additional 15.5% of variance, which was significantly different than zero ($F\Delta(1,165) = 31.99$, $p<.01$). In the total model, only the ACE-Q ($B = 2.68$, $p<.01$) was a significant predictor of scores on the DERS-18, such that higher ACE-Q scores predicted higher self-reported emotion dysregulation. See Appendix A for the effects of each predictor variable.

Regression analysis for the ESP showed that demographic covariates in the first step accounted for 12.3% of variance in scores of self-reported disordered eating, and this was significantly greater than zero ($F(3, 166) = 7.76$, $p<.01$). When the ACE-Q was added in the second step, this accounted for an additional 6.9% of variance, which was also statistically significant ($F\Delta(1,165) = 14.05$, $p<.01$). In the total model, ACE-Q scores ($B = 0.17$, $p<.01$) and gender ($B = 0.69$, $p<.01$) were significant predictors of ESP scores, such that higher ACE-Q scores and being female (vs male) predicted more self-reported symptoms of disordered eating history. See Appendix B for the effects of each predictor variable.

Regression analysis for the PSS indicated that demographic covariates in the first step accounted for 6.5% of variance in perceived stress scores, which was significantly different than zero ($F(3,166) = 3.85$, $p=.01$). When the ACE-Q was added in the second step, this accounted for an additional 5.2% of variance, and this was also significant ($F\Delta(1,165) = 9.70$, $p<.01$). In the total model, ACE-Q scores ($B = 0.73$, $p<.01$) and race/ethnicity ($B = 2.48$, $p<.01$) were significant predictors of scores on the PSS, such that being White (vs non-White) and higher ACE-Q scores predicted higher self-reported perceived stress during the past month. See Appendix A for the effects of each predictor variable.

For the BRS, demographic covariates in the first step accounted for 21.3% of variance in resilience scores, which was significantly different than zero ($F(3,64) = 5.78$, $p<.01$). When the ACE-Q was added in the second step, this accounted for an additional 3.6% of variance, though this step was not statistically significant ($F\Delta(1,63) = 3.03$, $p=.09$). In the total model, only race/ethnicity significantly predicted BRS scores ($B = 4.70$, $p<.01$), such that being non-White (vs White) predicted higher scores of resilience. See Appendix A for the effects of each predictor variable.

Results for the BIS showed that demographic covariates in the first step accounted for 14.6% of variance in scores of behavioral inhibition, which was significantly different than zero ($F(3,166) = 9.44$, $p<.01$). When the ACE-Q was added in the second step, this accounted for an additional 0.1% of variance, and this step did not reach statistical significance ($F\Delta(1,165) = 0.14$, $p=.71$). In the total model, gender was the only significant predictor of BIS scores ($B = 2.85$, $p<.01$), such that being female (vs male) predicted higher behavioral inhibition scores. See Appendix A for the effects of each predictor variable.

For the BAS-Reward Responsiveness subscale, demographic covariates in the first step accounted for 2.1% of variance in scores of behavioral approach, but this fell short of statistical significance ($F(3,166) = 1.79$, $p=.17$). When the ACE-Q was added in the second step, this accounted for an additional 7.4% of variance, which was significantly different than zero ($F\Delta(1,165) = 13.54$, $p<.01$). In the total model, both ACE-Q scores ($B = 0.25$, $p<.01$) and gender ($B = 0.69$, $p=.02$) significantly predicted BAS-Reward Responsiveness scores, such that lower ACE-Q scores and being female (vs male) predicted higher behavioral approach scores. See Appendix A for the effects of each predictor variable.

Regression analysis for the BAS-Drive subscale revealed that demographic covariates in the first step accounted for 1.8% of variance in scores of behavioral approach, which was not significantly different than zero ($F(3, 166) = 0.99$, $p=.40$). When the ACE-Q was added in the second step, this did not account for any additional variance, falling short of statistical significance ($F\Delta(1,165) = 0.04$, $p=.85$). In the total model, none of the predictor variables significantly predicted BAS-Drive scores. See Appendix A for the effects of each predictor variable.

For the BAS-Fun Seeking subscale, demographic covariates in the first step accounted for 2.6% of variance in scores of behavioral approach, which was not significantly different than zero ($F(3, 166) = 1.47$, $p=.22$). When the ACE-Q was added in the second step, this accounted for an additional 0.2% of variance, but this step also fell short of significance ($F\Delta(1,165) = 0.41$, $p=.52$). In the total model, none of the predictor variables significantly predicted BAS-Fun Seeking scores. See Appendix A for the effects of each predictor variable.

Aim 2

The secondary aim of this study was to examine the relationship between ACEs and physical health in emerging adults. For Hypothesis 3, Pearson correlations and hierarchical linear regression analyses were used to examine the relationships between ACE scores and lifestyle health behaviors such as sleep, exercise, alcohol use, substance use, e-cigarette use, disordered eating, and risky sexual behaviors. Regarding Hypothesis 4, the same correlation and regression analyses were used to assess the relationship between ACE scores and other indicators of poorer physical health.

Hypothesis 3 – Correlation Analysis

Correlation analysis for Hypothesis 3 yielded mixed results. We observed a strong positive correlation between scores on the ACE-Q and self-reported sleep difficulties on the PSQI (r=.50, p<.01). Additionally, results indicated mild-moderate correlations between the ACE-Q and scores of illicit substance use (DAST-10; r=.20) and e-cigarette use (EDS; r=.22); both were significant at the p<.05 level. Correlations between the ACE-Q and exercise, alcohol use, and risky sex behaviors did not reach significance. Further details regarding these associations can be seen in Figure 5.

	ACE-Q	GLTEQ	PSQI	AUDIT-C	DAST-10	EDS	SRS
ACE-Q	—						
GLTEQ	-0.124	—					
PSQI	0.485 ***	-0.027	—				
AUDIT-C	-0.093	0.027	0.024	—			
DAST-10	0.231 **	-0.041	0.117	0.304 ***	—		
EDS	0.211 **	0.037	0.081	0.369 ***	0.517 ***	—	
SRS	0.151	-0.058	0.008	0.310 ***	0.253 **	0.405 ***	—

Table 3. Hypothesis 3 Correlation Matrix. Note: * p < .05, ** p < .01, *** p < .001

Hypothesis 3 – Regression Analysis

Next, hierarchical linear regression analyses were completed to further examine the relationship between the ACE-Q and lifestyle behaviors. In the first step of each regression model, we controlled for demographic covariates (gender, race/ethnicity, and SES) to see if they significantly accounted for variance on scores for each measure. In the second step of each model, the ACE-Q was added as a predictor variable to assess for additional variance in the scores for each lifestyle measure, respectively. The second step was also assessed for significant predictor variables in the total model. The results of these hierarchical linear regression analyses are reported below.

Results for the PSQI demonstrated that demographic covariates in the first step accounted for 3.9% of variance in scores of sleep difficulties, but this was not statistically significant ($F(3, 135) = 1.81$, $p=.15$). When the ACE-Q was added in the second step, this accounted for an additional 22.7% of variance, which was significantly different than zero ($F\Delta(1,134) = 41.48$, $p<.01$). In the total model, ACE-Q scores were the only significant predictor of PSQI scores ($B = .89$, $p<.01$), such that higher ACE-Q scores predicted increased sleep difficulties during the past month. See Appendix B for the effects of each predictor variable.

Regression analysis for the GLTEQ revealed that demographic covariates in the first step accounted for 8.1% of variance in scores of self-reported exercise, which was significantly greater than zero ($F(3, 166) = 4.90$, $p<.01$). When the ACE-Q was added in the second step, this was not statistically significant, and only accounted for an additional 0.3% of variance ($F\Delta(1,165) = 0.58$, $p=.45$). In the total model, race/ethnicity ($B = 10.25$, $p=.03$) and SES ($B = 17.72$, $p=.03$) were the only significant predictors of GLTEQ scores, such that being non-White (vs White) and receiving government financial assistance (vs no assistance) predicted lower self-reported exercise over a seven-day period. See Appendix B for the effects of each predictor variable.

For the AUDIT-C, regression analysis revealed that demographic covariates in the first step accounted for 16.3% of variance in scores of self-reported alcohol use, which was significantly greater than zero ($F(3, 134) = 8.72$, $p<.01$). When the ACE-Q was added in the second step, this did not account for any additional variance ($F\Delta(1,133) = 0.00$, $p=.95$). In the total model, gender ($B = 1.54$, $p<.01$) and race/ethnicity ($B = 0.87$, $p=.03$) were the only significant predictors of alcohol use, such that being male (vs female) and White (vs non-White)

predicted higher self-reported alcohol use within the past year. See Appendix B for the effects of each predictor variable.

Regression analysis for the DAST-10 showed that demographic covariates in the first step accounted for 2.3% of variance in scores of self-reported illicit substance use, which was not significantly greater than zero ($F(3, 166) = 1.32$, $p=.27$). When the ACE-Q was added in the second step, this accounted for an additional 7.1% of variance, which was statistically significant ($F\Delta(1,165) = 13.03$, $p<.01$). In the total model, ACE-Q scores ($B = 0.20$, $p<.01$) and SES ($B = 0.94$, $p=.03$) were significant predictors of DAST-10 scores, such that higher ACE-Q scores and not receiving government financial assistance (vs receiving assistance) predicted higher self-reported illicit substance use within the past year. See Appendix B for the effects of each predictor variable.

For the EDS-4, regression analysis showed that demographic covariates in the first step accounted for 2.5% of variance in scores of self-reported e-cigarette dependence, which was not significantly greater than zero ($F(3, 166) = 1.42$, $p=.24$). When the ACE-Q was added in the second step, this accounted for an additional 6.9% of variance, which was statistically significant ($F\Delta(1,165) = 12.56$, $p<.01$). In the total model, ACE-Q scores ($B = 0.47$, $p<.01$) and gender ($B = 1.23$, $p=.03$) were significant predictors of EDS-4 scores, such that higher ACE-Q scores and being male (vs female) predicted higher self-reported e-cigarette dependence. See Appendix B for the effects of each predictor variable.

For the SRS, regression analysis showed that demographic covariates in the first step accounted for 1.5% of variance in scores of risky sex behaviors, which was not significantly greater than zero ($F(3, 130) = 0.67$, $p=.57$). When the ACE-Q was added in the second step, this accounted for an additional 3.8% of variance, which was statistically significant ($F\Delta(1,129) =$

5.23, *p*=.02). In the total model, only ACE-Q scores were a significant predictor of SRS scores (*B* = 0.12, *p*=.02), such that higher ACE-Q scores predicted higher self-reported history of risky sex behaviors. See Appendix B for the effects of each predictor variable.

Hypothesis 4 – Correlation Analysis

Correlation analysis for Hypothesis 4 indicated a strong positive association between ACE-Q scores and self-reported somatic symptoms on the PHQ-15 (*r*=.50, *p*<.01). Correlations between the ACE-Q, self-rated health, and healthcare utilization over the past 12 months were not statistically significant. Further details regarding these associations can be seen in Figure 6.

	ACE-Q	Self-Rated Health	Healthcare Utilization	PHQ-15
ACE-Q	—			
Self-Rated Health	-0.097	—		
Healthcare Utilization	0.039	-0.134	—	
PHQ-15	0.492 ***	-0.146	0.138	—

Table 4. Hypothesis 4 Correlation Matrix. Note: * p < .05, ** p < .01, *** p < .001

Hypothesis 4 – Regression Analysis

Next, hierarchical linear regression analyses were completed to examine the relationship between the ACE-Q and indicators of physical health, including somatic symptoms, self-rated health, and healthcare utilization. In the first step of each regression model, we controlled for demographic covariates – including gender, race/ethnicity, and SES variables – to see if they significantly accounted for variance on scores for each measure. In the second step of each model, the ACE-Q was added as a predictor variable to assess for additional variance in the scores for each physical health measure, respectively. The second step was also assessed for

significant predictor variables in the total model. The results of these hierarchical linear regression analyses are reported below.

For the PHQ-15, regression analysis showed that demographic covariates in the first step accounted for 14.7% of variance in scores of self-reported somatic symptoms, which was significantly greater than zero ($F(3, 136) = 7.84$, $p<.01$). When the ACE-Q was added in the second step, this accounted for an additional 17.7% of variance, which was also statistically significant ($F\Delta(1,135) = 35.35$, $p<.01$). In the total model, ACE-Q scores ($B = 1.00$, $p<.01$) and gender ($B = 2.61$, $p<.01$) were significant predictors of PHQ-15 scores, such that higher ACE-Q scores and being female (vs male) predicted higher self-reported somatic symptoms over the past month. See Appendix B for the effects of each predictor variable.

Regression analysis of a self-rated health measure showed that demographic covariates in the first step accounted for 5.2% of variance in scores of subjective health, which was significantly greater than zero ($F(3, 166) = 3.03$, $p=.03$). When the ACE-Q was added in the second step, this accounted for an additional 0.7% of variance, but this step was not statistically significant ($F\Delta(1,165) = 1.18$, $p=.28$). In the total model, only race/ethnicity was a significant predictor of self-rated health ($B = 0.38$, $p=.04$), such that being White (vs non-White) predicted lower subjective ratings of personal health. See Appendix B for the effects of each predictor variable.

Regression analysis of the healthcare utilization questionnaire demonstrated that demographic covariates in the first step accounted for 7.2% of variance in 12-month healthcare utilization, which was significantly greater than zero ($F(3, 165) = 4.28$, $p<.01$). When the ACE-Q was added in the second step, this did not account for any additional variance, and this step was not statistically significant ($F\Delta(1,164) = 0.03$, $p=.87$). In the total model, only gender was a

significant predictor of healthcare utilization (B = 2.74, p<.01), such that being female (vs male) predicted increased healthcare utilization over the previous 12 months. See Appendix B for the effects of each predictor variable.

Aim 3

The final aim of the current study was to better understand the respective influence of childhood maltreatment and household dysfunction items of the ACE-Q on mental and physical health. In Hypothesis 5, Pearson correlations and hierarchical linear regression analyses were used to explore the associations between the Childhood Maltreatment subscale (ACE-Q items 1-5), Household Dysfunction subscale (ACE-Q items 6-10), and mental health measures that were clinically and statistically significant in Aim 1. The same correlation and regression analyses were also used in Hypothesis 6 to examine the relationships between the ACE-Q subscales and physical health measures that were clinically and statistically significant in Aim 2.

Hypothesis 5 – Correlation Analysis

For the mental health measures defined in Hypothesis 5, correlation analysis revealed that scores on the Childhood Maltreatment (CM) and Household Dysfunction (HD) subscales were both significantly associated with scores on the PHQ-9 (CM: r=.35; HD: r=.40), GAD-7 (CM: r=.40; HD: r=.34), PCL-5 (CM: r=.44; HD: r=.41), ASRS-5 (CM: r=.32; HD: r=.38), DERS-18 (CM: r=.38; HD: r=.37), and ESP (CM: r=.31, p<.01; HD: r=.21, p<.01). All of these associations were significant at the p<.01 level, and can be seen in Figure 7.

	ACE-Q	ACE-Q (CM)	ACE-Q (HD)	PHQ-9	GAD-7	PCL-5	ASRS-5	DERS-18	PSS	ESP
ACE-Q	—									
ACE-Q (CM)	0.871 ***	—								
ACE-Q (HD)	0.900 ***	0.569 ***	—							
PHQ-9	0.421 ***	0.346 ***	0.398 ***	—						
GAD-7	0.412 ***	0.400 ***	0.335 ***	0.724 ***	—					
PCL-5	0.479 ***	0.437 ***	0.414 ***	0.723 ***	0.712 ***	—				
ASRS-5	0.397 ***	0.323 ***	0.377 ***	0.663 ***	0.652 ***	0.647 ***	—			
DERS-18	0.420 ***	0.375 ***	0.370 ***	0.704 ***	0.681 ***	0.674 ***	0.657 ***	—		
PSS	0.241 **	0.226 **	0.202 **	0.454 ***	0.534 ***	0.506 ***	0.476 ***	0.406 ***	—	
ESP	0.292 ***	0.314 ***	0.210 **	0.502 ***	0.497 ***	0.452 ***	0.370 ***	0.458 ***	0.338 ***	—

Table 5. Hypothesis 5 Correlation Matrix. Note: * p < .05, ** p < .01, *** p < .001

Hypothesis 5 – Regression Analysis

Next, hierarchical linear regression analyses were used to further examine these relationships. In the first step of each regression model, we controlled for demographic covariates (gender, race/ethnicity, and SES variables) to see if they significantly accounted for variance on scores for each mental health measure. In the second step of each model, scores on the Childhood Maltreatment and Household Dysfunction subscales were added to evaluate whether both subscales were significant predictors of each mental health measure, or if the significant relationship between the ACE-Q and each mental health measure was more strongly influenced by one subscale. For this analysis, results concerning demographic covariates were not reported, as these were previously discussed in the results for Aims 1 and 2. Further, our objective was to explore the influence of each ACE-Q subscale, and reporting on demographic covariates is peripheral to this aim.

Regression analyses confirmed that step 2 (CM and HD subscales) significantly accounted for a proportion of variance in scores on the PHQ-9, GAD-7, PCL-5, ASRS-5, DERS-18, and ESP, as demonstrated in Aim 1. In the overall model, both ACE-Q subscales

significantly predicted scores on the PHQ-9 (CM: β=0.18, p=.04; HD: β=0.28, p<.01), PCL-5 (CM: β=0.29, p<.01; HD: β=0.27, p<.01) and DERS-18 (CM: β=0.26, p<.01; HD: β=0.20, p=.02); only the Childhood Maltreatment subscale significantly predicted scores on the GAD-7 (CM: β=0.31, p<.01; HD: β=0.15, p=.07) and ESP (CM: β=0.23, p<.01; HD: β=0.08, p=.39); only the Household Dysfunction subscale significantly predicted scores on the ASRS-5 (CM: β=0.17, p=.05; HD: β=0.27, p<.01). See Appendix C for the effects of each predictor variable.

Hypothesis 6 – Correlation Analysis

For the physical health measures defined in Hypothesis 6, correlation analysis revealed that scores on the Childhood Maltreatment (CM) and Household Dysfunction (HD) subscales were both significantly associated with scores on the PSQI (CM: r=.51, p<.01; HD: r=.36, p<.01), and PHQ-15 (CM: r=.46, p<.01; HD: r=.41, p<.01); only the Household Dysfunction subscale was significantly associated with scores on the DAST-10 (CM: r=.13, p=.19; HD: r=.23, p<.01) and EDS (CM: r=.14, p=.06; HD: r=.23, p<.01). These associations can be seen in Figure 8.

	ACE-Q	ACE-Q (CM)	ACE-Q (HD)	PSQI	DAST10	EDS	PHQ-15
ACE-Q	—						
ACE-Q (CM)	0.871 ***	—					
ACE-Q (HD)	0.900 ***	0.569 ***	—				
PSQI	0.485 ***	0.509 ***	0.361 ***	—			
DAST10	0.231 **	0.131	0.270 ***	0.117	—		
EDS	0.211 **	0.139	0.230 **	0.081	0.517 ***	—	
PHQ-15	0.492 ***	0.459 ***	0.407 ***	0.646 ***	0.330 ***	0.168 *	—

Table 6. Hypothesis 6 Correlation Matrix. Note: * p < .05, ** p < .01, *** p < .001

Hypothesis 6 – Regression Analysis

Next, hierarchical linear regression analyses were used to further examine these relationships. In the first step of each regression model, we controlled for demographic covariates – including gender, race/ethnicity, and SES variables – to see if they significantly accounted for variance on scores for each physical health measure. In the second step of each model, scores on the Childhood Maltreatment and Household Dysfunction subscales were added to examine whether both subscales, or only one subscale, significantly predicted scores on physical health measures. Once again, results concerning demographic covariates were not reported.

Regression analyses confirmed that step 2 (CM and HD subscales) significantly accounted for a proportion of variance in scores on the PSQI, DAST-10, EDS, and PHQ-15, as demonstrated in Aim 2. In the overall model, both ACE-Q subscales significantly predicted scores on the PHQ-15 (CM: β=0.31, p<.01; HD: β=0.20, p=.03); only the Childhood Maltreatment subscale significantly predicted scores on the PSQI (CM: β=0.45, p<.01; HD: β=0.12, p=.21); only the Household Dysfunction subscale significantly predicted scores on the DAST-10 (CM: β=0.04, p=.65; HD: β=0.35, p<.01) and EDS (CM: β=0.03, p=.72; HD: β=0.27, p<.01). See Appendix C for the effects of each predictor variable.

Discussion

Adverse childhood experiences (ACEs) are an important public health concern, and the past two decades of research have continued to highlight the need for early prevention and intervention strategies that address the heightened chronic disease risks associated with ACEs in adulthood. Presently, there is a need for further research that utilizes an etiological framework to help identify the mechanisms responsible for poorer health outcomes. Accordingly, the current study sought to better understand physical and mental health characteristics associated with ACEs in the emerging adult population – by targeting this age group, results may help elucidate clinically significant health issues within this stratum of the population. Previous research has largely focused on the aggregated adult population and the pediatric population, with comparatively much less being known about the health issues in emerging adults with a history of ACEs.

Mental Health Factors

Our results provide important information about the mental health of emerging adults with a history of ACEs, and we believe it is essential to compare these results with previous research on pediatric, emerging adult, and aggregated adult samples. The pediatric literature has most consistently demonstrated a relationship between ACEs and depression, anxiety, and ADHD (Horn et al., 2019; Bright et al., 2016). In addition to depression, anxiety, and ADHD, the aggregated adult literature has also demonstrated associations between ACEs and PTSD (Kalmakis & Chandler, 2015; Felitti et al., 1998). Recent findings within a young adult sample suggest a higher prevalence of worsening stress and mental health over the course of the semester for college students with high (versus low) ACE scores, though these results were only limited to measurement with the PHQ-9 (Karatekin, 2018).

In comparison, results within our emerging adult sample demonstrated associations between ACEs and depression, anxiety, ADHD, PTSD, disordered eating, emotion dysregulation, and perceived stress. Thus, our findings suggest a wider array of mental health concerns than what has been previously reported. This is likely due, in part, to our decision to analyze our mental health measures continuously rather than using a suggested clinical cutoff score to categorize participants as screening "positive" or "negative" for each respective mental health concern. That said, our results suggest numerous mental health concerns for emerging adults with multiple ACEs, even if those concerns are screened as subclinical. Taken together, we contend that this helps to identify aspects of pathology and potential mechanisms related to increased mental health symptomology.

Demographic Differences in Mental Health Factors

Interestingly, our analysis of demographic covariates revealed important information about potential cultural differences in the presentation of mental health symptoms among emerging adults. For instance, when looking at race/ethnicity, being White predicted higher self-reported symptoms of depression, anxiety, ADHD, and perceived stress. Additionally, membership of a non-White racial/ethnic group predicted higher resilience scores. This trend could indicate that membership of a minority racial/ethnic group is related to greater resilience, a protective factor against psychopathology and mental distress. However, this may also be partially explained by well-documented mental health stigma among certain minority racial/ethnic minority groups (Miranda et al., 2015), potentially contributing to the under-reporting of mental health symptoms. Of note, demographic covariates such as gender and socioeconomic status did not appear to significantly predict psychopathology or transdiagnostic mental health factors.

Physical Health Factors

Concerning physical health factors, it is also important to distinguish the differences in ACE-related symptoms among pediatric, emerging adult, and aggregated adult populations. Research with pediatric samples has identified various health complaints associated with ACEs, including asthma, infection, somatic complaints, and sleep disruption (Oh et al., 2018). Additionally, others have documented an increased prevalence of weight gain/loss, enuresis, constipation, and poorer diabetes control (Purewal et al., 2016). Evidence for physical health symptoms is much more robust among aggregated adult samples, suggesting that ACEs are associated with cardiovascular issues such as ischemic heart disease, coronary artery disease, diabetes, myocardial infarction, and stroke (Campbell et al., 2017), in addition to chronic lung disease, cancer, and liver disease (Felitti et al., 1998). Concerning younger adult samples, recent findings have identified higher rates of chronic disease onset for individuals between ages 18 and 34 (Sonu et al., 2019). Within our emerging adult sample, our findings demonstrated that ACEs had a strong positive relationship with somatic complaints, although many of these other physical health concerns were not explored.

To our knowledge, these results provide new evidence that ACEs are associated with somatic complaints in a healthy emerging adult population. Although somatic complaints were previously found in a systematic review of pediatric studies (Oh et al., 2018), we contend that these symptoms are important to note in an emerging adult sample, since they have also not yet been identified in ACEs studies with aggregated adult samples. Further, there are key differences in the types of somatic complaints made by pediatric participants and emerging adults. For instance, research with children and early adolescents are more likely to focus on stomachaches, vomiting, headaches, dizziness, and nightmares, and are largely dependent on parental report

(Flaherty et al., 2009). Based on our results, the somatic symptoms most frequently endorsed included racing heart/pounding chest, dizziness, trouble sleeping, low energy/fatigue, headaches, back pain, and shortness of breath. Importantly, these somatic symptoms are also associated with psychological distress (i.e., symptoms of depression, anxiety, panic, PTSD), suggesting an area in which physical complaints could benefit from mental health interventions. Future research could further illuminate differences in somatic symptoms between pediatric, emerging adult, and adult populations.

Lifestyle Health Behaviors

Within our emerging adult sample, we identified a strong association between ACEs and sleep difficulties, and more modest associations between ACEs and measures of illicit substance use and e-cigarette dependence. Our results surrounding ACEs and health behaviors in emerging adults build on a very limitted literature, and draw several distinctions from previous research findings. In the past, health behaviors like cigarette smoking, over-eating, and alcohol/drug use have been theorized as mechanisms by which childhood adversity contributes to downstream chronic health conditions. From this perspective, individuals with more childhood adversity disproportionately engage in unhealthy coping behaviors to compensate for chronic stress (Felitti, 2009). Of note, these lifestyle health behaviors have been mostly studied with older adults, and we continue to highlight the need for disaggregated adult samples that are stratified by narrower age-groups to better understand the disproportionate development of chronic disease in later adulthood.

Interestingly, we did not observe significant relationships between ACEs and indicators of poorer physical health highlighted elsewhere in the adult literature, such as cigarette smoking, low physical activity, heavy alcohol use, risky sex behaviors, and lower self-rated health. In

contrast, our findings suggest that sleep difficulties and somatic complaints may play a larger role than previously understood, and particularly for emerging adults. Concerning this trend, we suggest that emerging adulthood may act as a transitional period, and that engagement in unhealthy lifestyle behaviors (e.g., over-eating, low physical activity, cigarette smoking, heavy alcohol use, risky sex behaviors) may become more prominent over time, if unaddressed. Additionally, evidence suggests that unhealthy coping behaviors like sexual risk-taking, heavy alcohol use, and illicit substance use are more normative among college students than the general adult population (Krieger et al., 2018), which could mask the significance of problematic engagement in these behaviors for emerging adults with a higher ACE history.

Compared to the results of other emerging adult samples, our results also demonstrate differences concerning ACEs and lifestyle behaviors. In a recent study on college students (Windle et al., 2018), the authors found that ACE scores predicted higher cigarette use, alcohol use, marijuana use, and BMI, and lower self-reported hours of sleep and fruit and vegetable consumption. In contrast, we did not observe a significant relationship between ACEs and cigarette use or alcohol use; though, this may be due to survey differences and our smaller sample size. Additionally, we did not assess for BMI and fruit/vegetable consumption, which limits the ability to compare these findings. While the two studies were designed similarly (e.g., online survey method among college students), key methodological differences could have also contributed to this difference in findings. For instance, the questionnaires used in the current study were much more robust, and data was collected using common measures of lifestyle health behaviors (e.g., PSQI, AUDIT-C, DAST-10) rather than single-item questionnaires.

Key Differences by ACE-Q Subscale

The current study also provides further insight into the respective influences of the ACE-Q subscales (childhood maltreatment and household dysfunction) on measures of mental and physical health; this approach was adapted from a recent study of mental health issues based on latent ACE classes (child maltreatment, household dysfunction, community violence, low adversity) by Lee et al. (2020). When comparing scores on the child maltreatment and household dysfunction subscales, the authors reported that only the child maltreatment subscale was associated with a greater odds ratio of depression, anxiety, and PTSD. In comparison, the results of the present study showed that both subscales significantly predicted scores of depression, PTSD, and emotion dysregulation; however, only the childhood maltreatment subscale predicted scores of anxiety and disordered eating, and only the household dysfunction subscale predicted scores of ADHD. This finding could indicate etiological differences between specific mental health issues in the ACEs population, but more research is needed.

The aforementioned authors (Lee et al., 2020) did not assess the relationships between ACE-Q subscales and measures of physical health in their sample of young adults – therefore, this became the focus of our third study aim. In the present study, both the childhood maltreatment and household dysfunction subscales predicted self-reported somatic symptoms. Only the childhood maltreatment subscale predicted sleep difficulties, and this relationship seemed to be particularly strong. On measures of illicit substance use and e-cigarette dependence, analysis of the ACE-Q subscales revealed that household dysfunction was more predictive of substance use and dependence. This finding provides further explanation about substance use risk in the ACEs population, and suggests that environmental factors (e.g., household substance use) could pose a greater risk for subsequent substance use compared to

childhood abuse and neglect. Taken together, analysis of these subscales suggests that participants with a history of childhood maltreatment may demonstrate different physical health concerns related to trauma and chronic stress (e.g., sleep difficulties) compared to participants with a history of household dysfunction (e.g., e-cigarette and illicit substance use). However, further research is needed to better understand these relationships.

Conclusion

Altogether, our results suggest that even among healthy emerging adults, ACEs are associated with both mental and physical health difficulties. These findings provide important information about the pathway from early adversity to chronic disease onset. Previously, the ACEs literature thoroughly addressed the increased risk of chronic diseases in older adulthood for individuals with a history of higher childhood adversity; in particular, those with ≥ 4 ACEs have been consistently found to have an increased risk of chronic conditions like depression, cardiovascular disease, and cancer, among others (Felitti et al., 1998). Of note, our results support recent findings that these same chronic health difficulties are disproportionately present among younger adults (18-34 years) with a higher ACEs history (Sonu et al., 2019), and not just among individuals in later adulthood. That is, our findings identify numerous mental and physical chronic health concerns associated with ACEs even among healthy emerging adults (18-25 years old). To our knowledge, this is the most comprehensive study to concurrently assess for both mental and physical health concerns related to ACEs in the emerging adult population.

After consideration of the current ACEs literature, we believe that the most important contribution of the present study is the growing understanding that ACEs are associated with sleep difficulties and somatic complaints in emerging adults. These findings are particularly relevant for healthcare providers that oversee the care of this age group (e.g., primary care and

mental health clinicians), since sleep and somatic symptoms are likely to be overlooked in healthy emerging adults. Adverse childhood experiences have already been identified as a widespread public health concern, contributing to increased risk for chronic physical and mental health concerns throughout the lifespan. From a population health perspective, there are vast benefits to identifying a patient population with a higher prevalence of these symptoms, such as the cost-effectiveness of early treatments that could potentially reduce chronic disease onset and healthcare utilization in later adulthood. Integrated primary care clinics could be an ideal setting to address sleep difficulties and pain symptoms in emerging adults, as behavioral health interventions are already being implemented to target these issues. Through an increased understanding of the health risks related to childhood adversity, providers can more effectively screen for potential health concerns and implement prevention and early intervention strategies.

Future directions within this line of research could expand on our etiological focus, and we support further attempts to connect ACE-related health risks with specific biological mechanisms, physical symptoms, and lifestyle health behaviors. Felitti (2009) famously distinguished that the two most likely pathways from ACEs to chronic illness are compensatory coping through poor health behaviors and inflammatory biological responses due to chronic stress. Our results suggest both of these to be accurate, with e-cigarette and illicit substance use pertaining to the former, and somatic complaints relating to the latter. Further, we believe that sleep difficulties could be more accurately understood as a health issue related to psychological distress due to the common presentation of sleep difficulties as a PTSD symptom. Regarding sleep difficulties, researchers may want to further examine the relationship between ACEs and individual PSQI components (e.g., subjective sleep quality, latency, duration, efficiency, disturbances, sleep medication use, daytime dysfunction), as well as other sleep-related factors

(e.g., sleep hygiene, unhelpful beliefs about sleep). Given the strong associations between ACEs and sleep and pain symptomology, future research may want to evaluate these health concerns from a chronic stress perspective.

Limitations

Of note, the present study was met with several limitations. We encountered substantial logistical obstacles due to restrictions related to the COVID-19 pandemic, which prevented us from collecting in-person laboratory participant data to explore our original aim – the relationship between ACEs and heart rate variability (HRV). Due to autonomic dysregulation caused by chronic/toxic stress, we identified HRV as a possible correlate of childhood adversity, and sought to understand HRV dysregulation as a potential mechanism of downstream chronic disease onset. In the absence of laboratory data, our study aims were adjusted accordingly, and we directed our attention to other physical and mental health factors assessed via online survey that could influence chronic disease risk in later adulthood. Although unintended, this deviation allowed us to look deeper at the relationships between ACEs and other physical and mental health factors. In addition to HRV monitoring, we were also unable to measure BMI and blood pressure, and recommend that these measurements be collected in future research on ACEs and physical health in emerging adults.

While our results concerning various physical and mental health concerns align with the existing ACEs literature, we also recognize the potential influence of factors related to the COVID-19 pandemic. For instance, the COVID-19 pandemic has been widely recognized as a substantial stressor, with the potential for increases in psychological distress (e.g., depression, anxiety, perceived stress) and maladaptive coping (e.g., increased sleep difficulties and substance misuse; decreased physical activity). Accordingly, due to the limitations of self-report survey

data, participant responses may have been unduly impacted by current events. To address this, future analyses with this dataset could include a COVID-19 moderator variable to explore differences in participant responses from before and after the start of the pandemic. However, due to the high effect sizes of many of our statistically significant findings, we contend that our conclusions remain valid.

We also note some of the limitations of using certain survey measures and screening tools in an online questionnaire format. For instance, we were surprised that our results did not endorse gender as a significant predictor of PTSD symptoms within our sample, as this relationship has been widely acknowledged in the literature (Tolin & Foa, 2006). Since the PCL-5 was completed as part of an online questionnaire, we were unable to provide participants with detailed instructions, though they were provided with a prompt to answer questions with regard to a specific stressor. This degree of ambiguity may have contributed to responses that more generally referenced a significant stressor rather than a previous DSM-5 traumatic event. Additionally, due to the layout of the Sexual Risk Survey within our online Qualtrics survey (e.g., use of string, rather than numeric, variables), this seemingly contributed to a higher rate of invalid responses. Thus, we interpret our results with caution, such that other methods may have identified an association between ACEs and risky sex behaviors in emerging adults.

Finally, some of our results may have been limited by our modest sample size (n=236). Due to the low prevalence of self-reported daily tobacco smoking in our sample, we did not have sufficient power to assess for associations between ACEs and cigarette smoking. However, a recent study of ACEs and smoking behaviors reported increasing rates of cigarette smoking among youths with higher ACE scores, despite decreasing rates of cigarette smoking in participants with no ACEs (Parks et al., 2018). Therefore, in addition to risky sex behaviors, we

contend that this lifestyle health behavior should be further explored in the emerging adult ACEs population.

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Appendix A: Aim 1 Hierarchical Linear Regression Results (SPSS)

PHQ-9

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.278[a]	.077	.061	5.45814	.077	4.630	3	166	.004
2	.480[b]	.231	.212	4.99929	.153	32.870	1	165	.000

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	413.820	3	137.940	4.630	.004[b]
	Residual	4945.357	166	29.791		
	Total	5359.176	169			
2	Regression	1235.344	4	308.836	12.357	.000[c]
	Residual	4123.832	165	24.993		
	Total	5359.176	169			

a. Dependent Variable: PHQ9_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	5.555	.703		7.901	.000
	Gender_r	2.231	.843	.198	2.646	.009
	Race_Eth_r	-1.760	.896	-.147	-1.965	.051
	SES_r	2.518	1.577	.119	1.596	.112
2	(Constant)	4.376	.676		6.474	.000
	Gender_r	1.316	.789	.117	1.669	.097
	Race_Eth_r	-2.230	.825	-.186	-2.704	.008
	SES_r	1.308	1.460	.062	.896	.372
	ACE_Total	1.101	.192	.406	5.733	.000

a. Dependent Variable: PHQ9_Total

GAD-7

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.276[a]	.076	.060	4.77252	.076	4.576	3	166	.004
2	.480[b]	.231	.212	4.36873	.154	33.104	1	165	.000

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	312.653	3	104.218	4.576	.004[b]
	Residual	3780.970	166	22.777		
	Total	4093.624	169			
2	Regression	944.471	4	236.118	12.371	.000[c]
	Residual	3149.152	165	19.086		
	Total	4093.624	169			

a. Dependent Variable: GAD7_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	4.662	.615		7.583	.000
	Gender_r	1.956	.737	.198	2.654	.009
	Race_Eth_r	-1.822	.784	-.174	-2.326	.021
	SES_r	1.220	1.379	.066	.884	.378
2	(Constant)	3.628	.591		6.141	.000
	Gender_r	1.154	.689	.117	1.675	.096
	Race_Eth_r	-2.234	.721	-.213	-3.100	.002
	SES_r	.159	1.276	.009	.125	.901
	ACE_Total	.965	.168	.408	5.754	.000

a. Dependent Variable: GAD7_Total

PCL-5

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.168[a]	.028	.011	14.35988	.028	1.614	3	166	.188
2	.507[b]	.257	.239	12.59758	.228	50.693	1	165	.000

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	998.381	3	332.794	1.614	.188[b]
	Residual	34230.231	166	206.206		
	Total	35228.612	169			
2	Regression	9043.254	4	2260.813	14.246	.000[c]
	Residual	26185.358	165	158.699		
	Total	35228.612	169			

a. Dependent Variable: PCL5_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	11.956	1.850		6.464	.000
	Gender_r	4.021	2.218	.139	1.813	.072
	Race_Eth_r	-2.777	2.357	-.090	-1.178	.240
	SES_r	-.285	4.150	-.005	-.069	.945
2	(Constant)	8.266	1.703		4.853	.000
	Gender_r	1.158	1.987	.040	.583	.561
	Race_Eth_r	-4.248	2.078	-.138	-2.044	.043
	SES_r	-4.072	3.679	-.075	-1.107	.270
	ACE_Total	3.444	.484	.496	7.120	.000

a. Dependent Variable: PCL5_Total

ASRS-5

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.229[a]	.053	.035	4.18316	.053	3.070	3	166	.029
2	.438[b]	.192	.173	3.87444	.140	28.508	1	165	.000

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	161.173	3	53.724	3.070	.029[b]
	Residual	2904.803	166	17.499		
	Total	3065.976	169			
2	Regression	589.113	4	147.278	9.811	.000[c]
	Residual	2476.864	165	15.011		
	Total	3065.976	169			

a. Dependent Variable: ASRS5_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	6.532	.539		12.122	.000
	Gender_r	1.161	.646	.136	1.797	.074
	Race_Eth_r	-1.087	.687	-.120	-1.583	.115
	SES_r	2.172	1.209	.136	1.796	.074
2	(Constant)	5.681	.524		10.844	.000
	Gender_r	.501	.611	.059	.820	.413
	Race_Eth_r	-1.426	.639	-.157	-2.231	.027
	SES_r	1.298	1.132	.081	1.147	.253
	ACE_Total	.794	.149	.388	5.339	.000

a. Dependent Variable: ASRS5_Total

DERS-18

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.214[a]	.046	.028	13.43837	.046	2.644	3	166	.051
2	.448[b]	.201	.181	12.33605	.155	31.992	1	165	.000

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	1432.260	3	477.420	2.644	.051[b]
	Residual	29977.887	166	180.590		
	Total	31410.147	169			
2	Regression	6300.740	4	1575.185	10.351	.000[c]
	Residual	25109.407	165	152.178		
	Total	31410.147	169			

a. Dependent Variable: DERS18_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	36.963	1.731		21.355	.000
	Gender_r	3.002	2.076	.110	1.446	.150
	Race_Eth_r	-2.029	2.206	-.070	-.920	.359
	SES_r	8.508	3.884	.166	2.191	.030
2	(Constant)	34.093	1.668		20.440	.000
	Gender_r	.775	1.946	.028	.398	.691
	Race_Eth_r	-3.173	2.035	-.109	-1.559	.121
	SES_r	5.562	3.603	.109	1.544	.125
	ACE_Total	2.680	.474	.408	5.656	.000

a. Dependent Variable: DERS18_Total

PSS

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.255[a]	.065	.048	6.22112	.065	3.854	3	166	.011
2	.342[b]	.117	.096	6.06433	.052	9.695	1	165	.002

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	447.435	3	149.145	3.854	.011[b]
	Residual	6424.588	166	38.702		
	Total	6872.024	169			
2	Regression	803.964	4	200.991	5.465	.000[c]
	Residual	6068.059	165	36.776		
	Total	6872.024	169			

a. Dependent Variable: PSS_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	17.214	.801		21.482	.000
	Gender_r	2.465	.961	.193	2.565	.011
	Race_Eth_r	-2.172	1.021	-.160	-2.127	.035
	SES_r	-.357	1.798	-.015	-.199	.843
2	(Constant)	16.437	.820		20.046	.000
	Gender_r	1.862	.957	.146	1.947	.053
	Race_Eth_r	-2.482	1.001	-.183	-2.481	.014
	SES_r	-1.154	1.771	-.048	-.652	.516
	ACE_Total	.725	.233	.236	3.114	.002

a. Dependent Variable: PSS_Total

ESP

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.351[a]	.123	.107	1.23380	.123	7.763	3	166	.000
2	.438[b]	.192	.172	1.18800	.069	14.047	1	165	.000

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	35.451	3	11.817	7.763	.000[b]
	Residual	252.696	166	1.522		
	Total	288.147	169			
2	Regression	55.277	4	13.819	9.792	.000[c]
	Residual	232.870	165	1.411		
	Total	288.147	169			

a. Dependent Variable: ESP_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	1.056	.159		6.647	.000
	Gender_r	.833	.191	.318	4.369	.000
	Race_Eth_r	-.309	.203	-.111	-1.526	.129
	SES_r	-.450	.357	-.092	-1.262	.209
2	(Constant)	.873	.161		5.436	.000
	Gender_r	.690	.187	.264	3.685	.000
	Race_Eth_r	-.382	.196	-.137	-1.949	.053
	SES_r	-.638	.347	-.130	-1.838	.068
	ACE_Total	.171	.046	.272	3.748	.000

a. Dependent Variable: ESP_Total

BRS

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.462[a]	.213	.176	5.15220	.213	5.781	3	64	.001
2	.499[b]	.249	.202	5.07242	.036	3.029	1	63	.087

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	460.333	3	153.444	5.781	.001[b]
	Residual	1698.888	64	26.545		
	Total	2159.221	67			
2	Regression	538.268	4	134.567	5.230	.001[c]
	Residual	1620.952	63	25.729		
	Total	2159.221	67			

a. Dependent Variable: BRS_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	20.513	1.013		20.255	.000
	Gender_r	-2.016	1.260	-.179	-1.600	.115
	Race_Eth_r	4.535	1.348	.381	3.365	.001
	SES_r	-4.241	2.085	-.229	-2.033	.046
2	(Constant)	21.039	1.042		20.194	.000
	Gender_r	-1.371	1.295	-.121	-1.059	.294
	Race_Eth_r	4.703	1.330	.395	3.535	.001
	SES_r	-3.900	2.062	-.210	-1.891	.063
	ACE_Total	-.482	.277	-.199	-1.740	.087

a. Dependent Variable: BRS_Total

BIS

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.382[a]	.146	.130	3.75907	.146	9.438	3	166	.000
2	.383[b]	.146	.126	3.76886	.001	.139	1	165	.710

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	400.107	3	133.369	9.438	.000[b]
	Residual	2345.687	166	14.131		
	Total	2745.794	169			
2	Regression	402.080	4	100.520	7.077	.000[c]
	Residual	2343.714	165	14.204		
	Total	2745.794	169			

a. Dependent Variable: BIS_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	18.377	.484		37.954	.000
	Gender_r	2.805	.581	.347	4.830	.000
	Race_Eth_r	-.807	.617	-.094	-1.308	.193
	SES_r	1.669	1.086	.110	1.536	.126
2	(Constant)	18.435	.510		36.175	.000
	Gender_r	2.849	.594	.353	4.793	.000
	Race_Eth_r	-.784	.622	-.091	-1.262	.209
	SES_r	1.729	1.101	.114	1.570	.118
	ACE_Total	-.054	.145	-.028	-.373	.710

a. Dependent Variable: BIS_Total

BAS–Reward Responsiveness

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.145[a]	.021	.009	1.86251	.021	1.787	2	166	.171
2	.309[b]	.095	.079	1.79591	.074	13.540	1	165	.000

a. Predictors: (Constant), Race_Eth_r, Gender_r

b. Predictors: (Constant), Race_Eth_r, Gender_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	12.394	2	6.197	1.787	.171[b]
	Residual	575.842	166	3.469		
	Total	588.237	168			
2	Regression	56.065	3	18.688	5.794	.001[c]
	Residual	532.171	165	3.225		
	Total	588.237	168			

a. Dependent Variable: BAS_Reward_Total

b. Predictors: (Constant), Race_Eth_r, Gender_r

c. Predictors: (Constant), Race_Eth_r, Gender_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	17.132	.238		71.938	.000
	Gender_r	.475	.288	.127	1.647	.102
	Race_Eth_r	.307	.308	.077	.999	.319
2	(Constant)	17.418	.242		71.847	.000
	Gender_r	.687	.284	.183	2.421	.017
	Race_Eth_r	.418	.298	.104	1.402	.163
	ACE_Total	-.251	.068	-.279	-3.680	.000

a. Dependent Variable: BAS_Reward_Total

BAS–Drive

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.162[a]	.026	.008	2.18577	.026	1.477	3	165	.223
2	.169[b]	.029	.005	2.18969	.002	.410	1	164	.523

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	21.165	3	7.055	1.477	.223[b]
	Residual	788.302	165	4.778		
	Total	809.467	168			
2	Regression	23.132	4	5.783	1.206	.310[c]
	Residual	786.336	164	4.795		
	Total	809.467	168			

a. Dependent Variable: BAS_Drive_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	11.447	.282		40.616	.000
	Gender_r	-.228	.339	-.052	-.674	.501
	Race_Eth_r	.544	.361	.116	1.505	.134
	SES_r	-.824	.632	-.100	-1.303	.194
2	(Constant)	11.505	.296		38.826	.000
	Gender_r	-.183	.346	-.042	-.529	.598
	Race_Eth_r	.567	.364	.121	1.559	.121
	SES_r	-.764	.640	-.093	-1.195	.234
	ACE_Total	-.054	.084	-.051	-.640	.523

a. Dependent Variable: BAS_Drive_Total

BAS–Fun Seeking

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.133[a]	.018	.000	2.19832	.018	.989	3	165	.399
2	.134[b]	.018	-.006	2.20477	.000	.036	1	164	.851

a. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r

b. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	14.338	3	4.779	.989	.399[b]
	Residual	797.378	165	4.833		
	Total	811.716	168			
2	Regression	14.511	4	3.628	.746	.562[c]
	Residual	797.205	164	4.861		
	Total	811.716	168			

a. Dependent Variable: BAS_Fun_Total

b. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r

c. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	12.199	.283		43.070	.000
	Gender_r	.211	.340	.048	.621	.536
	Race_Eth_r	.519	.361	.111	1.437	.153
	SES_r	-.529	.636	-.064	-.832	.406
2	(Constant)	12.182	.298		40.820	.000
	Gender_r	.198	.349	.045	.566	.572
	Race_Eth_r	.513	.364	.110	1.408	.161
	SES_r	-.546	.644	-.066	-.848	.397
	ACE_Total	.016	.085	.015	.189	.851

a. Dependent Variable: BAS_Fun_Total

Appendix B: Hierarchical Linear Regression Results (SPSS)

PSQI

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.197[a]	.039	.017	3.74437	.039	1.811	3	135	.148
2	.516[b]	.266	.244	3.28421	.227	41.481	1	134	.000

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	76.191	3	25.397	1.811	.148[b]
	Residual	1892.745	135	14.020		
	Total	1968.935	138			
2	Regression	523.603	4	130.901	12.136	.000[c]
	Residual	1445.332	134	10.786		
	Total	1968.935	138			

a. Dependent Variable: PSQI_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	6.511	.513		12.685	.000
	Gender_r	1.314	.636	.174	2.065	.041
	Race_Eth_r	-.738	.686	-.091	-1.076	.284
	SES_r	.202	1.370	.012	.147	.883
2	(Constant)	5.658	.469		12.058	.000
	Gender_r	.538	.571	.071	.943	.347
	Race_Eth_r	-1.166	.605	-.144	-1.927	.056
	SES_r	-.913	1.214	-.057	-.752	.453
	ACE_Total	.894	.139	.496	6.441	.000

a. Dependent Variable: PSQI_Total

GLTEQ

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.285[a]	.081	.065	27.45946	.081	4.901	3	166	.003
2	.291[b]	.085	.062	27.49461	.003	.576	1	165	.449

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	11085.768	3	3695.256	4.901	.003[b]
	Residual	125167.645	166	754.022		
	Total	136253.413	169			
2	Regression	11521.054	4	2880.264	3.810	.005[c]
	Residual	124732.359	165	755.954		
	Total	136253.413	169			

a. Dependent Variable: GLTEQ_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	58.749	3.537		16.610	.000
	Gender_r	-7.621	4.242	-.134	-1.797	.074
	Race_Eth_r	-10.595	4.508	-.175	-2.350	.020
	SES_r	-18.600	7.936	-.175	-2.344	.020
2	(Constant)	59.608	3.718		16.034	.000
	Gender_r	-6.955	4.337	-.122	-1.604	.111
	Race_Eth_r	-10.252	4.536	-.169	-2.260	.025
	SES_r	-17.719	8.031	-.166	-2.206	.029
	ACE_Total	-.801	1.056	-.059	-.759	.449

a. Dependent Variable: GLTEQ_Total

AUDIT-C

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.404[a]	.163	.145	2.11719	.163	8.724	3	134	.000
2	.404[b]	.163	.138	2.12510	.000	.004	1	133	.950

a. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r

b. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	117.316	3	39.105	8.724	.000[b]
	Residual	600.655	134	4.483		
	Total	717.971	137			
2	Regression	117.334	4	29.333	6.495	.000[c]
	Residual	600.637	133	4.516		
	Total	717.971	137			

a. Dependent Variable: AUDITC_Total

b. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r

c. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	5.165	.295		17.489	.000
	Gender_r	-1.537	.366	-.336	-4.199	.000
	Race_Eth_r	-.872	.401	-.173	-2.176	.031
	SES_r	-1.138	.740	-.123	-1.539	.126
2	(Constant)	5.158	.316		16.321	.000
	Gender_r	-1.541	.373	-.337	-4.132	.000
	Race_Eth_r	-.873	.402	-.174	-2.169	.032
	SES_r	-1.147	.756	-.124	-1.518	.131
	ACE_Total	.006	.089	.005	.063	.950

a. Dependent Variable: AUDITC_Total

DAST-10

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.153[a]	.023	.006	1.50039	.023	1.320	3	166	.270
2	.308[b]	.095	.073	1.44883	.071	13.027	1	165	.000

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	8.916	3	2.972	1.320	.270[b]
	Residual	373.695	166	2.251		
	Total	382.612	169			
2	Regression	36.261	4	9.065	4.319	.002[c]
	Residual	346.351	165	2.099		
	Total	382.612	169			

a. Dependent Variable: DAST10_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	1.842	.193		9.533	.000
	Gender_r	-.187	.232	-.062	-.805	.422
	Race_Eth_r	.176	.246	.055	.714	.476
	SES_r	-.718	.434	-.127	-1.656	.100
2	(Constant)	1.627	.196		8.307	.000
	Gender_r	-.353	.229	-.117	-1.547	.124
	Race_Eth_r	.090	.239	.028	.377	.707
	SES_r	-.939	.423	-.166	-2.219	.028
	ACE_Total	.201	.056	.277	3.609	.000

a. Dependent Variable: DAST10_Total

EDS-4

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.158[a]	.025	.007	3.58925	.025	1.417	3	166	.240
2	.306[b]	.094	.072	3.47047	.069	12.558	1	165	.001

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	54.765	3	18.255	1.417	.240[b]
	Residual	2138.535	166	12.883		
	Total	2193.300	169			
2	Regression	206.018	4	51.504	4.276	.003[c]
	Residual	1987.282	165	12.044		
	Total	2193.300	169			

a. Dependent Variable: EDS_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	2.811	.462		6.081	.000
	Gender_r	-.838	.554	-.116	-1.512	.133
	Race_Eth_r	-.506	.589	-.066	-.858	.392
	SES_r	-1.101	1.037	-.081	-1.061	.290
2	(Constant)	2.305	.469		4.913	.000
	Gender_r	-1.231	.547	-.170	-2.248	.026
	Race_Eth_r	-.707	.573	-.092	-1.235	.219
	SES_r	-1.620	1.014	-.120	-1.598	.112
	ACE_Total	.472	.133	.272	3.544	.001

a. Dependent Variable: EDS_Total

SRS

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.124[a]	.015	-.007	1.04068	.015	.677	3	130	.568
2	.232[b]	.054	.024	1.02416	.038	5.229	1	129	.024

a. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r

b. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	2.200	3	.733	.677	.568[b]
	Residual	140.793	130	1.083		
	Total	142.993	133			
2	Regression	7.684	4	1.921	1.831	.127[c]
	Residual	135.309	129	1.049		
	Total	142.993	133			

a. Dependent Variable: SRS_Total

b. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r

c. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	2.134	.148		14.431	.000
	Gender_r	-.253	.182	-.122	-1.394	.166
	Race_Eth_r	.016	.197	.007	.082	.934
	SES_r	-.062	.343	-.016	-.180	.858
2	(Constant)	2.030	.153		13.305	.000
	Gender_r	-.363	.185	-.175	-1.962	.052
	Race_Eth_r	.003	.193	.001	.015	.988
	SES_r	-.175	.341	-.045	-.513	.609
	ACE_Total	.107	.047	.206	2.287	.024

a. Dependent Variable: SRS_Total

PHQ-15

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.384[a]	.147	.129	4.29181	.147	7.837	3	136	.000
2	.570[b]	.324	.304	3.83474	.177	35.352	1	135	.000

a. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r

b. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	433.063	3	144.354	7.837	.000[b]
	Residual	2505.073	136	18.420		
	Total	2938.136	139			
2	Regression	952.927	4	238.232	16.200	.000[c]
	Residual	1985.209	135	14.705		
	Total	2938.136	139			

a. Dependent Variable: PHQ15_Total

b. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r

c. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	4.346	.675		6.442	.000
	Gender_r	3.403	.762	.356	4.468	.000
	Race_Eth_r	-.271	.766	-.028	-.354	.724
	SES_r	1.941	1.415	.109	1.371	.173
2	(Constant)	3.435	.622		5.523	.000
	Gender_r	2.605	.694	.272	3.755	.000
	Race_Eth_r	-.781	.690	-.081	-1.133	.259
	SES_r	.373	1.292	.021	.289	.773
	ACE_Total	1.006	.169	.442	5.946	.000

a. Dependent Variable: PHQ15_Total

Self-Rated Health

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.228[a]	.052	.035	1.07665	.052	3.026	3	166	.031
2	.242[b]	.059	.036	1.07607	.007	1.180	1	165	.279

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	10.524	3	3.508	3.026	.031[b]
	Residual	192.423	166	1.159		
	Total	202.947	169			
2	Regression	11.890	4	2.973	2.567	.040[c]
	Residual	191.057	165	1.158		
	Total	202.947	169			

a. Dependent Variable: SRH_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	3.232	.139		23.309	.000
	Gender_r	-.229	.166	-.104	-1.379	.170
	Race_Eth_r	-.395	.177	-.169	-2.237	.027
	SES_r	.522	.311	.127	1.679	.095
2	(Constant)	3.281	.145		22.547	.000
	Gender_r	-.192	.170	-.087	-1.132	.259
	Race_Eth_r	-.376	.178	-.161	-2.119	.036
	SES_r	.572	.314	.139	1.819	.071
	ACE_Total	-.045	.041	-.085	-1.086	.279

a. Dependent Variable: SRH_r

Healthcare Utilization

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.269[a]	.072	.055	6.280	.072	4.280	3	165	.006
2	.269[b]	.072	.050	6.299	.000	.027	1	164	.870

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	506.364	3	168.788	4.280	.006[b]
	Residual	6507.304	165	39.438		
	Total	7013.669	168			
2	Regression	507.430	4	126.858	3.198	.015[c]
	Residual	6506.239	164	39.672		
	Total	7013.669	168			

a. Dependent Variable: Hlth_Utl_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	2.296	.810		2.834	.005
	Gender_r	2.780	.973	.214	2.856	.005
	Race_Eth_r	-1.945	1.037	-.141	-1.875	.063
	SES_r	-1.721	1.815	-.071	-.948	.345
2	(Constant)	2.254	.852		2.647	.009
	Gender_r	2.742	1.003	.212	2.735	.007
	Race_Eth_r	-1.957	1.043	-.142	-1.876	.062
	SES_r	-1.767	1.843	-.073	-.959	.339
	ACE_Total	.041	.250	.013	.164	.870

a. Dependent Variable: Hlth_Utl_r

Appendix C: Hierarchical Linear Regression Results (SPSS)

PHQ-9

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.278[a]	.077	.061	5.45814	.077	4.630	3	166	.004
2	.482[b]	.232	.208	5.01012	.155	16.508	2	164	.000

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_ChildhoodMaltreatment, ACE_HouseholdDysfunction

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	413.820	3	137.940	4.630	.004[b]
	Residual	4945.357	166	29.791		
	Total	5359.176	169			
2	Regression	1242.570	5	248.514	9.900	.000[c]
	Residual	4116.607	164	25.101		
	Total	5359.176	169			

a. Dependent Variable: PHQ9_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_ChildhoodMaltreatment, ACE_HouseholdDysfunction

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	5.555	.703		7.901	.000
	Gender_r	2.231	.843	.198	2.646	.009
	Race_Eth_r	-1.760	.896	-.147	-1.965	.051
	SES_r	2.518	1.577	.119	1.596	.112
2	(Constant)	4.316	.687		6.285	.000
	Gender_r	1.356	.794	.120	1.708	.090
	Race_Eth_r	-2.212	.827	-.184	-2.674	.008
	SES_r	1.151	1.492	.055	.772	.441
	ACE_ChildhoodMaltreatment	.891	.435	.175	2.050	.042
	ACE_HouseholdDysfunction	1.282	.389	.283	3.296	.001

a. Dependent Variable: PHQ9_Total

GAD-7

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.276[a]	.076	.060	4.77252	.076	4.576	3	166	.004
2	.487[b]	.238	.214	4.36263	.161	17.329	2	164	.000

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_ChildhoodMaltreatment, ACE_HouseholdDysfunction

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	312.653	3	104.218	4.576	.004[b]
	Residual	3780.970	166	22.777		
	Total	4093.624	169			
2	Regression	972.286	5	194.457	10.217	.000[c]
	Residual	3121.337	164	19.033		
	Total	4093.624	169			

a. Dependent Variable: GAD7_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_ChildhoodMaltreatment, ACE_HouseholdDysfunction

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	4.662	.615		7.583	.000
	Gender_r	1.956	.737	.198	2.654	.009
	Race_Eth_r	-1.822	.784	-.174	-2.326	.021
	SES_r	1.220	1.379	.066	.884	.378
2	(Constant)	3.746	.598		6.265	.000
	Gender_r	1.077	.691	.109	1.558	.121
	Race_Eth_r	-2.270	.720	-.216	-3.151	.002
	SES_r	.466	1.299	.025	.359	.720
	ACE_ChildhoodMaltreatment	1.376	.379	.308	3.633	.000
	ACE_HouseholdDysfunction	.609	.339	.154	1.799	.074

a. Dependent Variable: GAD7_Total

PCL-5

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.168[a]	.028	.011	14.35988	.028	1.614	3	166	.188
2	.507[b]	.257	.235	12.63022	.229	25.290	2	164	.000

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_ChildhoodMaltreatment, ACE_HouseholdDysfunction

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	998.381	3	332.794	1.614	.188[b]
	Residual	34230.231	166	206.206		
	Total	35228.612	169			
2	Regression	9066.943	5	1813.389	11.368	.000[c]
	Residual	26161.669	164	159.522		
	Total	35228.612	169			

a. Dependent Variable: PCL5_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_ChildhoodMaltreatment, ACE_HouseholdDysfunction

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	11.956	1.850		6.464	.000
	Gender_r	4.021	2.218	.139	1.813	.072
	Race_Eth_r	-2.777	2.357	-.090	-1.178	.240
	SES_r	-.285	4.150	-.005	-.069	.945
2	(Constant)	8.375	1.731		4.838	.000
	Gender_r	1.086	2.001	.038	.543	.588
	Race_Eth_r	-4.281	2.086	-.139	-2.053	.042
	SES_r	-3.788	3.762	-.070	-1.007	.315
	ACE_ChildhoodMaltreatment	3.823	1.096	.292	3.488	.001
	ACE_HouseholdDysfunction	3.116	.981	.268	3.177	.002

a. Dependent Variable: PCL5_Total

ASRS-5

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.229[a]	.053	.035	4.18316	.053	3.070	3	166	.029
2	.439[b]	.193	.169	3.88381	.141	14.288	2	164	.000

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_ChildhoodMaltreatment, ACE_HouseholdDysfunction

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	161.173	3	53.724	3.070	.029[b]
	Residual	2904.803	166	17.499		
	Total	3065.976	169			
2	Regression	592.208	5	118.442	7.852	.000[c]
	Residual	2473.768	164	15.084		
	Total	3065.976	169			

a. Dependent Variable: ASRS5_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_ChildhoodMaltreatment, ACE_HouseholdDysfunction

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	6.532	.539		12.122	.000
	Gender_r	1.161	.646	.136	1.797	.074
	Race_Eth_r	-1.087	.687	-.120	-1.583	.115
	SES_r	2.172	1.209	.136	1.796	.074
2	(Constant)	5.641	.532		10.598	.000
	Gender_r	.527	.615	.062	.856	.393
	Race_Eth_r	-1.414	.641	-.156	-2.205	.029
	SES_r	1.196	1.157	.075	1.034	.303
	ACE_ChildhoodMaltreatment	.657	.337	.170	1.950	.053
	ACE_HouseholdDysfunction	.913	.302	.266	3.028	.003

a. Dependent Variable: ASRS5_Total

DERS-18

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.214[a]	.046	.028	13.43837	.046	2.644	3	166	.051
2	.450[b]	.202	.178	12.36102	.157	16.099	2	164	.000

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_ChildhoodMaltreatment, ACE_HouseholdDysfunction

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	1432.260	3	477.420	2.644	.051[b]
	Residual	29977.887	166	180.590		
	Total	31410.147	169			
2	Regression	6351.807	5	1270.361	8.314	.000[c]
	Residual	25058.340	164	152.795		
	Total	31410.147	169			

a. Dependent Variable: DERS18_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_ChildhoodMaltreatment, ACE_HouseholdDysfunction

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	36.963	1.731		21.355	.000
	Gender_r	3.002	2.076	.110	1.446	.150
	Race_Eth_r	-2.029	2.206	-.070	-.920	.359
	SES_r	8.508	3.884	.166	2.191	.030
2	(Constant)	34.253	1.694		20.219	.000
	Gender_r	.670	1.958	.025	.342	.733
	Race_Eth_r	-3.221	2.041	-.111	-1.578	.116
	SES_r	5.979	3.682	.117	1.624	.106
	ACE_ChildhoodMaltreatment	3.236	1.073	.262	3.016	.003
	ACE_HouseholdDysfunction	2.197	.960	.200	2.289	.023

a. Dependent Variable: DERS18_Total

ESP

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.351[a]	.123	.107	1.23380	.123	7.763	3	166	.000
2	.445[b]	.198	.174	1.18691	.075	7.688	2	164	.001

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_ChildhoodMaltreatment, ACE_HouseholdDysfunction

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	35.451	3	11.817	7.763	.000[b]
	Residual	252.696	166	1.522		
	Total	288.147	169			
2	Regression	57.111	5	11.422	8.108	.000[c]
	Residual	231.036	164	1.409		
	Total	288.147	169			

a. Dependent Variable: ESP_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_ChildhoodMaltreatment, ACE_HouseholdDysfunction

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	1.056	.159		6.647	.000
	Gender_r	.833	.191	.318	4.369	.000
	Race_Eth_r	-.309	.203	-.111	-1.526	.129
	SES_r	-.450	.357	-.092	-1.262	.209
2	(Constant)	.904	.163		5.554	.000
	Gender_r	.671	.188	.256	3.567	.000
	Race_Eth_r	-.391	.196	-.141	-1.996	.048
	SES_r	-.559	.354	-.114	-1.581	.116
	ACE_ChildhoodMaltreatment	.276	.103	.234	2.683	.008
	ACE_HouseholdDysfunction	.080	.092	.076	.863	.389

a. Dependent Variable: ESP_Total

PSQI

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.197[a]	.039	.017	3.74437	.039	1.811	3	135	.148
2	.540[b]	.292	.265	3.23849	.253	23.735	2	133	.000

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_HouseholdDysfunction, ACE_ChildhoodMaltreatment

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	76.191	3	25.397	1.811	.148[b]
	Residual	1892.745	135	14.020		
	Total	1968.935	138			
2	Regression	574.055	5	114.811	10.947	.000[c]
	Residual	1394.881	133	10.488		
	Total	1968.935	138			

a. Dependent Variable: PSQI_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_HouseholdDysfunction, ACE_ChildhoodMaltreatment

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	6.511	.513		12.685	.000
	Gender_r	1.314	.636	.174	2.065	.041
	Race_Eth_r	-.738	.686	-.091	-1.076	.284
	SES_r	.202	1.370	.012	.147	.883
2	(Constant)	5.868	.472		12.420	.000
	Gender_r	.349	.569	.046	.614	.540
	Race_Eth_r	-1.194	.597	-.148	-1.999	.048
	SES_r	-.615	1.205	-.038	-.510	.611
	ACE_ChildhoodMaltreatment	1.506	.311	.449	4.845	.000
	ACE_HouseholdDysfunction	.354	.281	.115	1.258	.211

a. Dependent Variable: PSQI_Total

DAST-10

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.153[a]	.023	.006	1.50039	.023	1.320	3	166	.270
2	.351[b]	.123	.097	1.43002	.100	9.370	2	164	.000

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_ChildhoodMaltreatment, ACE_HouseholdDysfunction

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	8.916	3	2.972	1.320	.270[b]
	Residual	373.695	166	2.251		
	Total	382.612	169			
2	Regression	47.238	5	9.448	4.620	.001[c]
	Residual	335.374	164	2.045		
	Total	382.612	169			

a. Dependent Variable: DAST10_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_ChildhoodMaltreatment, ACE_HouseholdDysfunction

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	1.842	.193		9.533	.000
	Gender_r	-.187	.232	-.062	-.805	.422
	Race_Eth_r	.176	.246	.055	.714	.476
	SES_r	-.718	.434	-.127	-1.656	.100
2	(Constant)	1.553	.196		7.924	.000
	Gender_r	-.305	.227	-.101	-1.346	.180
	Race_Eth_r	.112	.236	.035	.476	.635
	SES_r	-1.132	.426	-.201	-2.658	.009
	ACE_ChildhoodMaltreatment	-.057	.124	-.042	-.460	.646
	ACE_HouseholdDysfunction	.424	.111	.350	3.822	.000

a. Dependent Variable: DAST10_Total

EDS-4

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.158[a]	.025	.007	3.58925	.025	1.417	3	166	.240
2	.322[b]	.104	.077	3.46167	.079	7.231	2	164	.001

a. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_ChildhoodMaltreatment, ACE_HouseholdDysfunction

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	54.765	3	18.255	1.417	.240[b]
	Residual	2138.535	166	12.883		
	Total	2193.300	169			
2	Regression	228.057	5	45.611	3.806	.003[c]
	Residual	1965.243	164	11.983		
	Total	2193.300	169			

a. Dependent Variable: EDS_Total

b. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r

c. Predictors: (Constant), SES_r, Gender_r, Race_Eth_r, ACE_ChildhoodMaltreatment, ACE_HouseholdDysfunction

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	2.811	.462		6.081	.000
	Gender_r	-.838	.554	-.116	-1.512	.133
	Race_Eth_r	-.506	.589	-.066	-.858	.392
	SES_r	-1.101	1.037	-.081	-1.061	.290
2	(Constant)	2.200	.474		4.638	.000
	Gender_r	-1.162	.548	-.161	-2.119	.036
	Race_Eth_r	-.676	.572	-.088	-1.182	.239
	SES_r	-1.894	1.031	-.140	-1.837	.068
	ACE_ChildhoodMaltreatment	.107	.300	.033	.356	.723
	ACE_HouseholdDysfunction	.789	.269	.272	2.936	.004

a. Dependent Variable: EDS_Total

PHQ-15

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.384[a]	.147	.129	4.29181	.147	7.837	3	136	.000
2	.573[b]	.328	.303	3.83763	.181	18.048	2	134	.000

a. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r

b. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r, ACE_ChildhoodMaltreatment, ACE_HouseholdDysfunction

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	433.063	3	144.354	7.837	.000[b]
	Residual	2505.073	136	18.420		
	Total	2938.136	139			
2	Regression	964.665	5	192.933	13.100	.000[c]
	Residual	1973.471	134	14.727		
	Total	2938.136	139			

a. Dependent Variable: PHQ15_Total

b. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r

c. Predictors: (Constant), SES_r, Race_Eth_r, Gender_r, ACE_ChildhoodMaltreatment, ACE_HouseholdDysfunction

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	4.346	.675		6.442	.000
	Gender_r	3.403	.762	.356	4.468	.000
	Race_Eth_r	-.271	.766	-.028	-.354	.724
	SES_r	1.941	1.415	.109	1.371	.173
2	(Constant)	3.493	.626		5.582	.000
	Gender_r	2.573	.695	.269	3.702	.000
	Race_Eth_r	-.772	.690	-.080	-1.118	.266
	SES_r	.650	1.329	.037	.489	.626
	ACE_ChildhoodMaltreatment	1.290	.360	.306	3.585	.000
	ACE_HouseholdDysfunction	.747	.336	.197	2.220	.028

a. Dependent Variable: PHQ15_Total

Appendix D: IRB 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 Initial Approval: Expedited

From: Biomedical IRB
To: Alexander Capiaghi
CC: Matthew Whited
Date: 2/17/2020
Re: UMCIRB 19-003332
Adverse Childhood Experiences and Heart Rate Variability

I am pleased to inform you that your Expedited Application was approved. Approval of the study and any consent form(s) occurred on 2/14/2020. The research study is eligible for review under expedited category # 4,7. The Chairperson (or designee) deemed this study no more than minimal risk.

Changes to this approved research may not be initiated without UMCIRB review except when necessary to eliminate an apparent immediate hazard to the participant. All unanticipated problems involving risks to participants and others must be promptly reported to the UMCIRB. The investigator must submit a Final Report application to the UMCIRB prior to the Expected End Date provided in the IRB application. If the study is not completed by this date, an Amendment will need to be submitted to extend the Expected End Date. The Investigator must adhere to all reporting requirements for this study.

Approved consent documents with the IRB approval date stamped on the document should be used to consent participants (consent documents with the IRB approval date stamp are found under the Documents tab in the study workspace).

The approval includes the following items:

Name	Description
Capiaghi - Thesis Proposal	Study Protocol or Grant Application
Debriefing Statement	Debriefing Statement
Laboratory Consent Form	Consent Forms
List of Self-Report Measures	Surveys and Questionnaires
Online Consent Form	Consent Forms
Qualtrics Survey	Surveys and Questionnaires
Recruitment Script	Recruitment Documents/Scripts

For research studies where a waiver of HIPAA Authorization has been approved, each of the waiver criteria in 45 CFR 164.512(i)(2)(ii) has 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.

Appendix E: Informed Consent for Online Portion of Study

Dear Participant,

I am a student at East Carolina University in the Department of Psychology. I am asking you to take part in my research study entitled, “Overcoming Adverse Childhood Experiences.”

The purpose of this research is to better understand the relationship between early life experiences and health. By doing this research, I hope to explore ways that health interventions may be improved for emerging adults**.** Your participation is completely voluntary.

You are being invited to take part in this research because you are a student between 18 and 25 years of age in an ECU introductory psychology course. The amount of time it will take you to complete this survey is approximately 45 to 60 minutes.

If you agree to take part in this survey, you will be asked questions that relate to childhood experiences and various components of mental and physical health. Once you complete the online survey portion of this study, you may also opt to sign up and take part in the in-person laboratory portion.

This research is overseen by the University and Medical Center Institutional Review Board(UMCIRB) at ECU. Therefore, some of the UMCIRB members or the UMCIRB staff may need to review your research data. Your identity will be evident to those individuals who see this information. However, I will take precautions to ensure that anyone not authorized to see your identity will not be given that information

Identifiers might be removed from the identifiable private information and, after such removal, the information could be used for future research studies or distributed to another investigator for future research studies without additional informed consent from you or your Legally Authorized Representative (LAR). However, there still may be a chance that someone could figure out the information is about you.

Please contact Alex Capiaghi at 845-216-8751 or capiaghia18@students.ecu.edu for any research related questions. If you have questions about your rights when taking part in this research, call the University and Medical Center Institutional Review Board (UMCIRB) at252-744-2914 (Monday-Friday, 8:00AM-5:00PM). If you would like to report a complaint or concern about this research study, call the Director of Human Research Protections at 252-744-2914.

You do not have to take part in this research, and you can stop at any time. If you decide you are willing to take part in this study, check the AGREE box below and the research questions will appear.

Thank you for taking the time to participate in my research.

Sincerely, Alex Capiaghi, Principal Investigator

By clicking "I agree," I certify that:

1) I have read all of the above information
2) I am between 18 and 25 years of age
3) I am currently enrolled in an Introductory Psychology course
4) I know that I can stop taking part in this study at any time