Examining the Association Between Adverse Childhood Experiences and Sleep Quality

by

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ABSTRACT

How early life is experienced and perceived can greatly affect mental and physical health outcomes. An individual is greatly influenced by their first models of what social relationships look and feel like, and with time also learn how to survive when less favorable social experiences occur. The lessons learned may lead to healthy problem solving and resilience, or it may lead to unhealthy problem-solving habits that hinder well-being. Anxious thoughts and other mental health symptoms may accompany an individual long-term and hinder an essential need for a healthy life. The first main purpose of this thesis is to examine the impact of Adverse Childhood Experiences (ACEs) on mental health (anxiety symptoms), and on sleep quality (an essential need). The second purpose of my thesis is to investigate the impact of genetics on resilience, specifically, the mu-opioid receptor gene. The first hypothesis proposed ACEs that were perceived as more traumatic and occurred more frequently would be associated with more poor sleep quality symptoms. The second hypothesis predicted that anxiety symptoms would mediate the association. The third hypothesis (exploratory) suggested that an individual's alleles for the mu-opioid receptor gene would moderate the mediation pathway. The study was conducted with 318 participants between the ages of 18 and 35 years old. The study demonstrated a direct effect for ACEs and sleep. Anxiety mediated the association between ACEs (exposure and severity) and sleep (insomnia, quality, sleepiness), suggesting that ACEs possibly increase feelings of anxiety which, in turn, lead to worse sleep outcomes. Finally, the moderated-mediation model with OPRM1 as the moderator, was not significant for the mediation pathway A; however, there was a significant interaction with anxiety and sleep symptoms.

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TABLE OF CONTENTS

Page

Page

APPENDIX

LIST OF TABLES

LIST OF FIGURES

CHAPTER 1

INTRODUCTION

Childhood is a time of rapid development where children soak in every fragment of the world around them. It does not take long for children to learn basic survival skills for avoiding negative stimuli. A child will touch a hot stove-top once and be cautious going forward. Outside of physical environmental threats, children also learn how to survive on a social level by modeling those within their close proximity. Home, a term that can bring forth feelings of happiness, sadness, warmth, anger, security, stress, regret, grief, confusion, etc., is usually the first place that children will pick up on social cues and behaviors. Due to the fact that no two homes are exactly alike, what a child experiences in the place they call "home" can have advantageous benefits or it can increase risk for disadvantageous outcomes to one's physical and mental health.

My thesis investigated Adverse Childhood Experiences (ACEs), which can bring forth those disadvantageous outcomes due to the stress and trauma that often accompany the trauma. ACEs are not a rarity but quite the opposite, it is estimated that 58% of youth in the United States (as of 2015) had experienced at least one ACE in their childhood (Kajeepeta et al., 2015). The impact from ACEs has continued to be a popular topic in research; however, there are still many gaps in the literature and in practice that require further insight into the potential impact of ACEs lasting throughout an individual's lifespan, and how best to identify those at risk before too much damage has been done. In my thesis, I explored the negative impact on mental health and sleep quality during pivotal times in development, late adolescence and young adulthood. Adolescent development has been heavily examined as an important developmental milestone for

puberty and neural plasticity. However, many developmental milestones would not thrive to their fullest without good mental health, support, nutrition, and sleep. Sleep studies have only skimmed the surface when investigating the impact of ACEs, adolescent development, and sleep quality. My thesis aimed to fill some of the literature gaps by investigating possible mechanisms on an individual level that can influence how our bodies and minds respond to negative social stimuli. Just as no two homes are alike, no two humans are exactly alike either. To ensure future practice is providing the best resources for psychiatric treatment when working with individuals who experienced adversity early in life, my thesis introduced a possible piece to the puzzle by examining a gene that is associated with social resilience and mental health outcomes, the mu-opioid receptor gene (OPRM1).

Adverse Childhood Experiences and Lasting Impact

Society has come a long way toward understanding and addressing ACEs and their lasting impact. ACEs are defined as early stressful and traumatic experiences that occur before an individual is 18 years old (Kajeepeta et al., 2015). ACEs include physical, sexual, and emotional abuse, neglect (emotional and physical), having a loved one incarcerated, and witnessing domestic violence, the death of a loved one, substance use, and mental illness within the household (Kajeepeta et al., 2015; Kural & Kovacs, 2022). According to the Centers for Disease Control and Prevention [CDC] (2019), 61% of adults across 25 states in the United States had experienced at least one ACE, and 16% had experienced four or more ACEs. Exposure to ACEs can negatively impact children's mental and physical development, and childhood includes many critical developmental periods (such as infancy and adolescence) that would benefit from stable and healthy

environments. Research on early development and attachment styles have emphasized the importance of stability and healthy socialization with primary caretakers to aid in the formation of secure attachment styles during adolescence and adulthood (Nelson, 2017). If a child experiences toxic or negative social interactions repeatedly during their childhood, future social interactions might be approached with hesitancy and caution, limiting the full potential for healthy and positive social interactions and relationships. Experiencing ACEs can lead to insecure attachment styles, which includes anxious attachment and avoidant attachment styles (Barnett & Howe, 2021). Individuals with anxious attachment styles desire to feel secure and loved; however, these individuals tend to have low self-esteem, extreme anxiety, and fear of abandonment (Kural & Kovac, 2022; Simpson & Rholes, 2017). The fear of abandonment can present itself in a number of behaviors, such as clingy and codependent behavior, making new best friends frequently, experiencing anxiety if a friend does not return a call, and attempting to "mindread" a romantic partner for any sign indicating the relationship will end. Unfortunately, due to the fear of abandonment, anxious attachment individuals are more likely to stay in an abusive relationship than leave one (Kural & Kovac, 2022). Anxious attachment styles can develop when a child is abandoned or neglected from a loved one that leaves the child feeling confused, hurt, worthless, and insecure (Simpson & Rholes, 2017). Individuals with avoidant attachment styles do not carry the anxiety and fear that is prevalent with anxious attachment, instead they are highly independent and self-assured. Avoidant individuals do not seek emotional closeness with others, and when faced with a problem, they are less likely to seek out help. Avoidant attachment styles can be formed when a child's needs (emotional and physical) are not being met by their

caretaker, which as a result, leaves the child no choice but to fend for themselves (Simpson & Rholes, 2017). Unfortunately, as the child's needs continue to be neglected during these critical parental bonding years, which nourish the child's emotional-social development, the lack of emotional security can lead the child to grow into an adult that avoids opportunities for emotional closeness, social support, and lasting bonds.

Moreover, ACEs can affect cognition, neural development, structures, and pathways (Danese & McEwen, 2012; Teicher & Samson, 2016). A study in Romania examined neglect and its effect on children's developmental outcomes when raised in an orphanage versus when not (Nelson, 2017). The study noted significant neurological deficits, and social and emotional behavior differences. The children who were raised in the orphanage had more internalizing and externalizing behaviors, poorer executive functioning skills, heightened stress responses, lower IQs, less gray matter and white matter volume in the brain, and less neural activity compared to healthy controls (Nelson, 2017). A lack of stimulation from caregivers can result in the deprivation of brain stimulation that is beneficial for cognition and mental processes. Other abnormalities in the brain can occur within the prefrontal cortex (decision making, impulse control), amygdala (emotion and fear processing), hypothalamus (mood, hunger, sleep, etc), hippocampus (learning and memory), and the striatum (reward) (Danese & McEwen, 2012; Teicher & Samson, 2016). Consequently, physical health and mental health are greatly at risk. In the CDC-Kaiser Permanente Adverse Childhood Experiences Study (1998), adults with ACEs had an increased risk of ischemic heart disease, cancer, stroke, depression, anxiety, substance use disorder, and diabetes (Felitti et al., 1998). Whether an individual develops poor mental and physical health will depend on their ability to adapt

to adversity. Therefore, it is important to examine ineffective adaptation by disruptive human systems (biological, social, emotional, etc.,) prior to risk estimation (Masten, 2001; Monroe et al., 2009).

Sleep Quality

Western medicine has been working toward uncovering and fully understanding sleep in humans. Sleep studies have been going on for centuries from dreaming and interpretation, neuroimaging and the discovery of REM sleep, and the continued linkage between sleep and mental health (Winkelman & Plante, 2010). Many specialists today have adopted a more holistic view toward diagnosis and treatment by acknowledging the "seen" and "unseen" symptoms before addressing the best treatment plan for optimal care. However, there is still room for improvement when it comes to addressing trauma and sleep quality concerns. Individuals who experience ACEs have an increased risk of developing poor sleep quality symptoms during adolescence and adulthood (Kajeepeta et al., 2015). Due to adolescence being a critical phase for neural and hormonal developments, examining sleep quality symptoms and ACEs could provide valuable information into poor sleep quality development and maintenance into adulthood. Many factors can contribute to poor sleep temporarily, but when chronic and unyielding, physical and mental health declines.

A common factor of poor sleep quality is experiencing sleep deprivation. During times of stress or after experiencing a traumatic event, sleep deprivation can occur and negatively impact physical and mental health, such as healthy digestion and gut health, reflexes, processing of emotions and rewards, social behavior, learning, memory, attention, and impulse control (de Zambotti et al., 2018; Tarokh et al., 2016; Winkelman

& Plante, 2010). Experiencing an ACE one time may or may not have lasting effects on sleep depending on other environmental and biological factors; however, adults that experienced more than one incident of maltreatment in childhood were two times more likely to have difficulty sleeping than adults who did not experience maltreatment in childhood (Baiden et al., 2015).

The type of maltreatment could also be associated with other sleep quality concerns. For example, women who disclosed that they experienced sexual abuse 10 years prior (when they were still children or adolescents), reported more frequent sleep disturbances than females who did not experience sexual abuse as children (Noll et al., 2006). Sexual abuse victims can often have fear associated with sleep because of similarities in the nighttime setting that remind them of the abuse (Sadeh et al., 2001). The fear and anxiety experienced around bedtime can affect sleep efficacy and overall sleep quality. For situations that are not daytime specific, such as neglect, research has attempted to find a component within sleep efficiency that could explain poor sleep quality symptoms, such as sleep fragmentation. Sleep fragmentation is when an individual wakes frequently from sleep (Semsar et al., 2021). Individuals that experienced severe emotional neglect in childhood were more likely to have frequent awakenings throughout the night than those who did not experience neglect (Semsar et al., 2021). Waking frequently from sleep could also cause symptoms of sleep deprivation depending on the severity.

Furthermore, difficulty falling and staying asleep (frequent awakenings) are also common symptoms of insomnia (de Zambotti et al., 2018). According to the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM–5; American Psychiatric

Association, 2013), it is estimated that about 33% of individuals in the United States have had insomnia and that females receive more diagnoses of insomnia than males (American Psychiatric Association, 2013). In regard to ACEs and insomnia, risk of developing symptoms of insomnia is high (Wang et al., 2016). This risk increased when considering the frequency, duration, and type of adversity experienced. When considering the type of ACE, physically abused children have lower sleep efficiency and spend a significantly greater amount of time moving and waking during sleep than non-abused children, and sexually abused children (Sadeh et al., 1995). In addition, the more frequent an ACE occurs, the more likely an individual will exhibit insomnia symptoms of frequent night awakenings, movement, and nocturnal activity during night (Bader et al., 2013). When sleep continues to be hindered during times when sleep is appropriate the sleep-wake cycle can continue to be thrown off and seeking professional help is always advised.

In addition, some individuals develop symptoms of excessive daytime sleepiness, Daytime sleepiness when related to ACEs often develops during late adolescence into young adulthood. Symptoms include recurrent periods of sleep within the same day, prolonged sleep episodes of more than 9 hours that are unrefreshing, and/or having difficulty being fully awake (American Psychiatric Association 2013). Individuals that experienced ACEs experienced more symptoms of chronic fatigue, anxiety, and depression (Heim et al., 2006). The development of excessive daytime sleepiness could be due to prolonged stress, head injuries, viral infections, and genetics (American Psychiatric Association, 2013; Lazarus et al., 2013). ACEs and excessive daytime sleepiness may be connected through prolonged stress responses. As a result, the repeated stress response leads to increased allostatic load (wear and tear in the body), which

affects multiple regulatory systems in the body, such as sleep-wake patterns that depend on chemical secretions (e.g., melatonin) and effective neural communication pathways (Danese & McEwen, 2012; McEwen, 2000). It is also possible that an unregulated sleep-wake pattern was established in childhood as a means to avoid negative and stressful events (Wang et al., 2021). If that was the case, treatment to undo all the established neural programming around sleep would prove challenging. All in all, ACEs have the potential to dysregulate a healthy sleep-wake cycle that could greatly affect the ability to live a fulfilling conscious life.

ACEs have also been mentioned in a few studies involving sleep paralysis, narcolepsy, and parasomnias (nightmares, night terrors, and sleepwalking). Sleep paralysis can be a very frightening experience and occurs when an individual awakens from REM (rapid eye movement); however, the motor paralysis that is normal during REM sleep, remains after waking (American Psychiatric Association, 2013). Women who experienced sexual abuse in childhood were more at risk of experiencing sleep paralysis upon awakening than women with no history of sexual abuse in childhood (McNally & Clancy, 2005). Abrams et al. (2008) reached the same conclusion. They also found that individuals who were sexually abused as children had more heightened emotional responses (e.g., more anger, sadness, and fear) than individuals without history of sexual abuse. It is worth mentioning that other contributing factors that increase the risk of sleep paralysis can stem from other poor sleep quality symptoms, such as the inability to fall asleep and sleep deprivation (Denis, 2018). As a result, if an individual is already experiencing poor sleep quality, the risk of experiencing sleep paralysis is higher

for that individual than someone without poor sleep quality symptoms, which further emphasizes the importance of examining sleep and ACEs.

Narcolepsy is also characterized by sleep paralysis but includes excessive sleepiness which leads to falling asleep at inappropriate periods of time (e.g., while driving a car). Individuals with narcolepsy may experience hallucinations right before falling asleep, vivid dreams, and quicker onset of REM sleep. Another symptom is cataplectic attacks during which an individual loses muscle control, resulting in falls and slumped postures, after experiencing a strong emotional response (American Psychiatric Association, 2013; Schiappa et al., 2018). Symptoms of narcolepsy may be due to low concentrations of hypocretin in the cerebrospinal fluid, dysfunctional limbic system response, and the relation between the hypocretin system and stress response (Schiappa et al., 2018). The development of narcolepsy with cataplexy is thought to be influenced by environmental components that strongly affect emotion regulation due to the emotional component of cataplexy (Schiappa et al., 2018). Also, according to Berridge et al. (2010), high arousal aversive situations activate the hypocretin/orexin system which plays a role in stress-related behavioral and physiological actions. As a result, stressful aversive experiences such as ACEs can activate this system and influence an individual/s biological systems and sleep patterns.

Stressful events can also bring about poor sleep quality symptoms very early in life. Poor sleep quality can manifest itself differently during childhood than in adulthood. Children will often display more disruptive and aggressive behaviors such as hitting or kicking in their sleep, frequent nightmares, and experiencing night terrors (Cecil et al., 2015; Okada et al; 2018). It is estimated that about 2% of adults, 37% of children at 18

months of age, and about 20% of children at 30 months of age have experienced a night terror episode (American Psychiatric Association, 2013). However, due to the higher prevalence among young children in general, not all occurrences are related to ACE exposure. Children also experience sleepwalking episodes with an estimated prevalence of 10-30% (American Psychiatric Association, 2013). Children that are more prone to sleepwalking might have more emotional and conduct problems, which may be explained by disruptive sleeping patterns (Stallman et al., 2017). However, sudden onset of sleepwalking has been reported in some children who experienced maltreatment (Cecil et al., 2015. Even so there is not enough research on early psychological trauma and sleepwalking to make a direct and concrete conclusion at this time. Nevertheless, to truly comprehend the substantial effect that ACEs can have on sleep quality and health, it is important to consider factors that can aid or hinder sleep quality, such as mental health symptoms.

Mental Health

A common theme for children that experience ACEs is the development of poor mental health symptoms, such as anxiety and depression symptoms (Kajeepeta et al., 2015; Nielsen et al., 2020; Sousa et al., 2018; Wadman et al., 2020). The development of these symptoms has been theorized by a number of gene and environmental stress models, such as the Diathesis Stress Model, and the Differential Susceptibility Model (Gould et al., 2022; Morgan et al., 2012; Mosley-Johnson et al., 2021; Nielsen et al., 2020; Rende & Plomin, 1992).The Diathesis Stress Model proposes that genetics interact with adverse stressful environments and increase the risk of developing poor mental health symptoms and disorders (Mosley-Johnson, 2021; Nielsen et al., 2020; Rende &

Plomin, 1992). The Differential Susceptibility Model purports that genetic variation is related to both supportive and stressful environments, which can buffer or hinder mental health accordingly (Gould et al., 2022; Morgan et al., 2012). For example, experiencing a traumatic ACE can be stressful and wear on the body; however, if an individual has supportive relationships and the means to seek professional help, the risk to mental health decreases, which then decreases the risk to physical health. On the other hand, if an individual does not have a support network or the means to seek out professional help, the lack of a supportive environment could increase risk to mental and physical health.

When chronic stress from a traumatic event occurs, it can negatively impact the body and mind by disrupting healthy thinking patterns and development. A symptom for many children that experience ACEs is the development of anxious ruminating thoughts (Elmore & Crouch, 2020; Kajeepeta et al., 2015). Areas in the brain involved with emotion and stress regulation can undergo structural and chemical changes in response to ACEs (Cohen et al., 2012; Herringa et al., 2013). For example, maltreatment can affect connectivity between the amygdala and hippocampus, which can increase the risk of anxiety and depression symptoms (Herringa et al., 2013). The anxious and depressive thoughts can affect daily functioning (e.g., sleep) if not treated. However, when considering the impact of ACEs on mental health and sleep quality, the model would not be complete without examining the interaction of genes on mental health outcomes and risk to daily functioning.

Genetic Components

Researchers investigating ACEs and genetic components have become increasingly interested in single nucleotide polymorphisms that are associated with

specific receptor genes. A number of genetic variants of receptor genes are linked to differences in mental and physical health outcomes when early life stress is present (Sheikh et al., 2013; Tyrka et al., 2009). The moderating effect for many of these studies is the genotype inherited for a gene (Meier et al., 2021; Sheikh et al., 2013; Slavich et al., 2014; Tyrka et al., 2009).

The mu-opioid receptor gene (OPRM1) is associated with social reward and sensitivity. OPRM1 has a single nucleotide polymorphism A118G/rs1799971 where the variant, guanine (G), replaces adenine (A) in the DNA sequence (Meier et al., 2021; Slavich et al., 2014). Research unveiled social sensitivity differences in individuals who inherit the two G alleles (greater sensitivity to social threat) versus the two A alleles, or one of each (Meier et al., 2021). Individuals with the GG genotype who experience negative social experiences over an extended period of time might have a blunted response to social rewards and an increased sensitivity to social threats as evidenced by increased activation in brain regions associated with pain and threat (Meier et al., 2021; Nelson, 2017; Slavich et al., 2014; Way et al., 2009). Therefore, frequent unpleasant mental health symptoms, such as anxiety, can decrease the motivation to connect with others, due to the threat response ingrained in the brain (Meier et al., 2021; Nelson, 2017). For example, if an individual experiences positive social experiences during childhood and adolescent development, the likelihood that an individual seeks out new social experiences increases. However, if an individual experiences negative social experiences, such as verbal abuse or physical abuse, the likelihood that an individual seeks out new social experiences decreases. As a result, individuals with the GG

genotype may have a heightened sensitivity to early social experiences, which can either benefit (if positive) or hinder (if negative) social motivation and mental health later in life.

Thesis Study

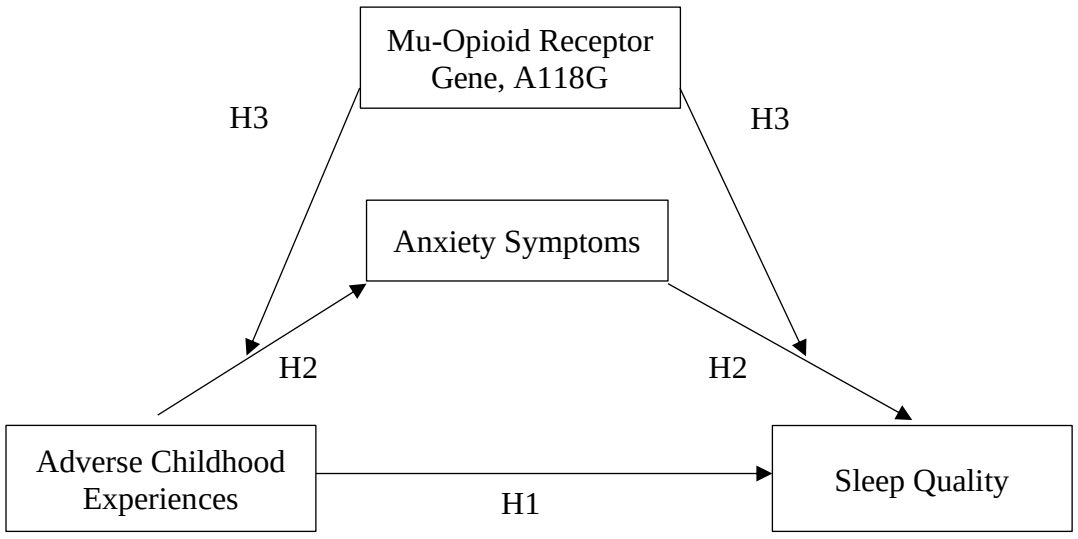
Critical developmental periods are sensitive to negative stimuli, especially if the hindering stimulus is frequent and causes constant stress. Sleep, an important daily need, is often negatively affected (e.g., frequent sleep disturbances). Sleep is important for personal wellbeing including physical health, neural development, social interactions, and when facing stress. Previous research that looked at ACEs and poor sleep quality symptoms discussed depression and anxiety as factors influencing sleep; however, studies have not assessed this association during critical developmental periods such as late adolescence into young adulthood. Therefore, based on the research and literature reviewed in the sections above, I further explored the association between ACEs and sleep quality symptoms by taking into consideration mental health and genetic makeup. My thesis has three main aims. First, I evaluated the direct pathway from ACEs to sleep quality symptoms. Next, I examined whether anxiety symptoms mediated the pathway from ACEs to sleep quality symptoms. Finally, I investigated whether the genotype for OPRM1 moderated the mediated pathways.

I proposed that ACEs and sleep quality would be positively associated. Thereby, the greater number of ACEs, greater perceived trauma scores, and greater occurrence of ACEs would be associated with more poor sleep quality symptoms. I next proposed that anxiety symptoms would mediate the pathway between ACEs and sleep quality symptoms. Specifically, higher scores on the ACE measure would be associated with

more anxiety symptoms, and, subsequently, more poor sleep quality symptoms. Finally, I proposed that the genotypes for OPRM1 would moderate the mediational pathways. Specifically, I proposed that individuals with the GG genotype for the mu-opioid receptor gene would show a stronger positive association from ACEs to anxiety symptoms, and from anxiety symptoms to sleep quality symptoms.

In order to test the hypotheses, I collected retrospective and current history data via self-report. Participants answered questions regarding, sleep habits, sleep history from adolescence to now, mental health measures, and ACE measures. Additionally, a small subsample of participants were asked to provide a saliva sample to explore the genetic analysis portion of the study.

Figure 1.
Proposed Moderated-Mediation Model



CHAPTER 2

METHODS

Participants and Procedures

Participants were recruited from the online survey recruitment platform, Prolific, as well as from the ASU SONA student research participation system. Participants were prescreened and were eligible if they were residents of the United States, English literate, and between the age of 18 and 35 years old. A total of 328 participants were recruited for the study, 152 participants from Prolific and 176 from SONA. The final sample after removing participants that failed the attention checks, included 318 participants, 150 participants from Prolific and 168 participants from SONA. Of the 168 participants from SONA, 21 of the participants completed the study in-person at the ASU West campus. Prior to participating in the study, all participants were shown and signed a physical informed consent form. All participants completed the self-report measures on a computer, either remotely or on the ASU West campus. The 21 in-person participants completed the self-report measures online on a research lab computer or on their own personal computer within the lab. In-person participants also provided a saliva sample for the DNA analysis portion of the study after signing a second informed consent form that explained the saliva sample process and how data would be used. One participant did not sign the informed consent form and opted out of the saliva sample. A total of 20 samples were collected and stored at or below 4°Celsius. The sample was delivered to the ASU Tempe Biotechnology Genomics Laboratory for DNA extraction and analysis of the OPRM1 gene's SNP A118G. Prolific participants received monetary compensation of $3.17 following survey completion, while SONA participants received one course credit

following survey completion. The in-person SONA participants that also provided a saliva sample were compensated $10 in cash and entered into a raffle for a chance to win a $50 gift card. Participants ranged from 18- 35 years old (M = 23.57, SD = 4.68). A majority of the participants were female (79%), white (58%), and had at least some college education (48%).

Measures

Sociodemographics. To gather descriptive information for the sample, I collected the participant's residential location, age, sex, gender, income level, relationship status, sexual orientation, household composition, employment status, education level, race, and ethnicity. *Residential location* included any of the 50 States of America. *Age* was between 18 to 35 years of age. *Biological sex* included, male, female, and prefer not to say. *Gender* included three response variables: male, female, and non-binary/other. *Sexual orientation* included heterosexual, homosexual, bisexual, and other. *Race/Ethnicity* included, American Indian or Alaska Native, Asian, Black or African American, Hispanic or Latino or Spanish Origin, Native Hawaiian or Pacific Islander, and White. *Relationship status* included: single, dating, in a Relationship (not cohabiting), in a relationship (cohabitating), married, divorced, and widowed. Education level included five response categories: some high school, high school/GED, some college (no degree), undergraduate degree (bachelor or associates), and graduate (masters/PhD). Employment status was dichotomized into eight response variables and then rank ordered into one main variable: full time (40 or more hours per week), part time (less than 40 hours per week), undergraduate student (associates and/or bachelors), graduate student (masters and/or doctorate), self-employed, retired, and unemployed.

Household composition included questions about living arrangements and housing status. The living arrangements included seven response variables: living alone, living with spouse/partner, living with roommate(s)/friends, living with siblings, living with parent(s)/in-laws, living with children, and other. Housing status was separated into four response options within one main variable: rent an apartment/condo/home, own an apartment/condo/home, live with family, friends, partner, or other (don't financially contribute toward rent/mortgage), and other housing status (e.g., living in a dorm). Income level included $10,000 increments from less than $10,000 per year to more than $150,000 per year.

Adverse Childhood Experiences Scale. To measure for adverse childhood experiences, participants completed an adapted version of the *Adverse Childhood Experiences Questionnaire* (ACE-Q; Felitti et al., 1998), which is a 10-item retrospective measure that identifies specific types of childhood adversity that occurred before the age of 18. The measure included statements in which the participant confirms or denies experiencing psychological abuse, physical abuse, sexual abuse, neglect, parental separation/divorce, exposure to substance abuse, mental illness, physical harm to mother, and criminal behavior. An example statement for psychological abuse would include, "Did a parent or other adult in the household often or very often swear at you, insult you, put you down, or humiliate you? *or* Act in a way that made you afraid that you might be physically hurt?" Participants answered *yes* or *no* to each statement. The numerical value 1 was assigned to each *yes* response, while a numerical value of 0 was assigned to each *no* response. If a participant confirmed exposure to an adversity, then follow up questions (adapted by researcher and not included in original ACE-Q) were shown that asked the

participants to provide their estimated age range in which the adversity occurred, how often the ACE occurred, and if they perceived the ACE that they experienced as traumatic. Age range was scored based on the number of years of exposure to ACEs and then recoded from 0 to 4 (0 = zero years/no ACE, 1 = one year of ACEs, 2 = two to three years of ACEs, 3 = four to nine years of ACEs, and 4 = ten or more years of exposure). All numerical values were totaled and higher values were associated with more years of ACE exposure. The frequency of ACEs was scored on a Likert scale from 0 (never) to 5 (daily). Values were totaled and higher scores indicated experiencing ACEs more often. Perceived trauma from ACEs was scored on a 5-point Likert scale from 0 to 4 with 0 = not at all traumatic to 4 = severely traumatic. Values were summed and higher scores were associated with more perceived trauma.

I decided to analyze ACEs in the following two ways: ACE exposure and ACE severity. For ACE exposure I added the total number of ACEs experienced to get an exposure score. There were ten ACE categories and if an ACE was experienced within a category, it was coded as 1. All the values were added for the ACE exposure sum total. Cronbach alpha indicated good reliability ($\alpha = .77$.). For ACE severity, I summed the recoded values (0 to 4) for the total number of ACE years, the ACE frequency (0 = never to 4 = daily), and the perceived trauma from ACEs (0 = not traumatic to 4 = very traumatic). Cronbach alpha had good internal consistency ($\alpha = .79$).

Anxiety Symptoms and History. To measure participants' general anxiety symptoms, participants completed a portion of the *State-Trait Anxiety Inventory for Adults* (STAI-AD; Spielberger et al., 1983), which included 20 items measuring trait anxiety and 20 items measuring state anxiety. Participants completed the trait anxiety

portion to capture how they *generally* feel. Positive statements (9) were not included in the analysis and one negative statement was omitted from the measure to ensure anxiety was being captured in the measure only. Participants rated each statement from 0 = not at all to 4 = always. An example of a trait anxiety statement would include, "I feel like a failure." Numerical values for each response were paired with each statement to determine the weight of the response. Higher scores were associated with more anxiety symptoms. Cronbach's alpha indicated good reliability ($\alpha = .89$).

Sleep Quality. To measure sleep quality history, participants completed three sleep measures. First, participants completed the *Epworth Sleepiness Scale* (ESS), which assessed daytime sleepiness (Johns, 1991; 1992). The ESS included 8 items that asked the participant to rate their chances of dozing off from 0= not at all likely to 4= highly likely of dozing off, for each statement. Example items included, "Sitting and reading," and "Sitting and talking to someone." Total scores range from 0 to 24 with higher scores associated with more daytime sleepiness. Current reflection for sleepiness had shown a fair reliability ($\alpha = .68$).

Next, participants completed the *Insomnia Severity Index* (Morin et al, 2011), which included 7 items that asked participants to reflect over the past two weeks regarding their ability to stay asleep, fall asleep, and their overall sleep quality. To capture more sleep data, participants were asked to reflect from the past month, instead of from the past two weeks. Participants rated each statement regarding their sleep from 0 to 4 with 0= none or very satisfied to 4= very dissatisfied/worried/noticeable. One question was omitted from the measure, the remaining 6 items were sum totaled with higher

scores associated with more symptoms of insomnia or poor sleep quality. Cronbach's alpha indicated good reliably for current symptoms ($\alpha = .82$).

Finally, participants completed an adapted version of the *Pittsburgh Sleep Quality Index* (PSQI; Buysse et al., 1989), in which participants reflected on their sleep quality from within the past month. The PSQI used for this thesis study had 18 individual items that asked about subjective sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. For example, "During the past month how often had you taken medicine (prescribed or "over the counter") to help you sleep?" Participants rated each item from 0= not during the past month/NA to 3= three or more times per week. Based on a systematic review across different countries, PSQI had a Cronbach's alpha ranging from .64 to .83 (Zhang et al., 2020). In the current study, the Cronbach alpha indicated good reliability ($\alpha = .71$).

Mu-Opioid Receptor Gene. Genetic components were assessed by analyzing one single nucleotide polymorphism of the mu-opioid receptor gene, rs1799971/A118G. The genetic material was gathered via saliva collection that was self-collected by participants. The samples were delivered to the ASU Genomics Lab in Tempe, AZ for DNA library preparation, DNA extraction, PCR, and Sanger Sequencing to record the alleles of the SNP, rs1799971/A118G of the mu-opioid receptor gene. The DNA sequencing results can include the following alleles (A or G) which make up the following genotypes: AA, AG, GG.

Overview of Analyses

Prior to testing the main hypotheses, I tested whether the data met the linear regression assumptions (e.g., linearity, homoscedasticity, etc.,). In order to test the hypothesis, I conducted PROCESS Model 4 in SPSS, with anxiety as the mediator (Hayes, 2022). ACE exposure and ACE severity were entered as the independent variables in separate analyses. Next, I tested the third hypothesis (exploratory) via PROCESS Model 58 in SPSS, that utilized a moderated-mediation model with the OPRM1 genotypes entered as the moderator. See Figure 1 for the proposed model.

Table 1.
Pearson Correlations for Main Study Variables

	ACE[1]	ACE[2]	ANX	SLQ	IN	SEF
ACE[1]						
ACE[2]	**.894****					
ANX	**.283****	**.216****	--			
SLQ	.072	.083	**.255****	--		
IN	**.266****	**.176****	**.493****	**.239****	--	
SEF	**.348****	**.260****	**.529****	**.302****	**.726****	--
OP	-.174	-.239	.116	-.208	-.304	-.209

Correlation is significant at the 0.01 level (2-tailed)**

Correlation is significant at the 0.05 level (2-tailed)*

ACE[1] (Adverse Childhood Experience Severity), ACE[2] (Adverse Childhood Experience Exposure), ANX (Anxiety), SLE (Sleepiness Now), IN (Insomnia), SLQ (Sleep Quality), and OP (OPRM1).

CHAPTER 3

RESULTS

Sample Descriptive

A total of 258 participants (81% of the sample) experienced at least one ACE during this childhood (i.e., prior to 18 years of age). When looking at ACE exposure volume, 68% experienced two or more ACEs, 51% experienced three or more ACEs, and 38% experienced four or more ACEs. Out of the ten ACE categories, males experienced 2.3 ACEs on average, females experienced 3.3 ACEs on average, and other (non-binary, third gender, or transgender) experienced 4.3 ACEs on average (See *Table 2* for the descriptive statistics). Verbal abuse was the most prevalent ACE within the sample (57%), followed by emotional neglect (51%) physical abuse (37%), and parental loss (34%). Females experienced more verbal abuse (59%), emotional neglect (50%), physical abuse (39%) and parental loss (45%) than males. See *Appendix* for ACE Exposure Descriptive Statistics (Table 3). However, non-binary participants experienced more emotional neglect (85%), verbal abuse (80%), physical abuse (39%), and sexual abuse (29%) than males and females in the sample. Non-binary/other participants also had more anxiety symptoms (24%) than males (19%) and females (20%) in the sample. In addition, males scored the lowest on the sleep quality measures, while non-binary/other individuals scored the highest. See *Appendix* for the main study variables by gender descriptive statistics (Table 4). In regard to the exploratory DNA analysis subsample, 13 participants (65% of the subsample) had the AA genotype and 7 (35% of the subsample) had the AG genotype. See *Appendix* for subsample descriptive statistics for main study variables (Table 5).

Table 2.

Main Study Variable Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
ACE Exposure	318	.00	10.00	3.15	2.56
ACE Severity	318	.00	151.00	29.60	30.70
Anxiety	318	.00	40.00	20.00	7.90
Sleepiness	318	.00	20.00	5.14	3.35
Insomnia	318	.00	24.00	9.35	5.11
Sleep Quality	318	.00	32.00	13.06	6.13

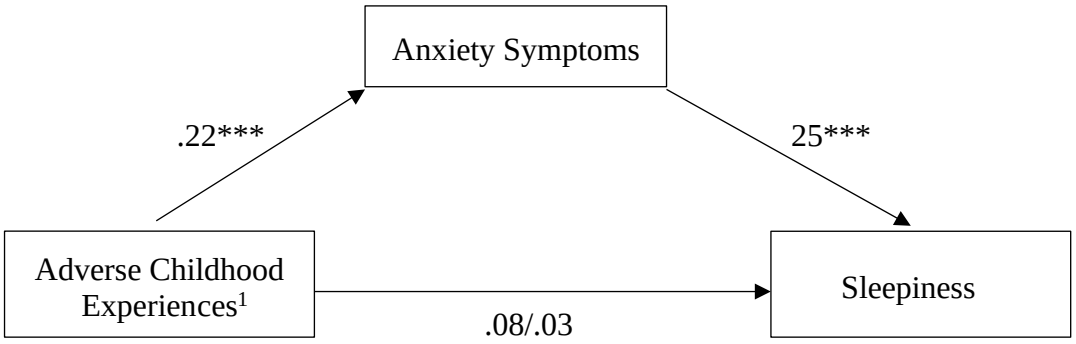
Sleep Quality

Sleepiness A simple mediation through SPSS PROCESS Model 4 resulted in a significant association (path A) when examining ACE exposure and anxiety symptoms (b = .22, SE = .17, B = .67, p < .001, 95% CI = .34, 1.00). In addition, there was a significant association (path B) for anxiety symptoms and sleepiness symptoms (b = .25, SE = .02, B = .11, p < .001, 95% CI = .06, .15). Furthermore, the results indicated no direct association (path C) for ACE exposure and current sleepiness, and no significant association for ACE exposure on current sleepiness when controlling for anxiety (path C'). However, the model indicated a significant indirect effect for ACE exposure on sleepiness (b = .05, SE = .02, p < .05, 95% CI = .02, .09). The significant indirect effect confirmed full mediation in the model such that, the more ACES an individual experiences is associated with more anxiety symptoms, which is associated with more sleepiness. See Figure 2 for the mediation model and coefficients.

In addition, a simple mediation model examined the association between ACE severity and sleepiness symptoms. The results indicated a significant association (path A) for ACE severity and anxiety symptoms (b = .28, SE = .01, B = .07, p < .001, 95% CI =

.05, .10), and a significant association (path B) for anxiety symptoms and sleepiness (*b* = .26 *SE* = .02, B = .11, *p* < .001, 95% CI = .06, .16). The results indicated that there was not a direct association (path C) for ACE severity on sleepiness, and no significant association for ACE severity and sleepiness when controlling for anxiety (path C'). However, there was a significant indirect effect for ACE severity and sleepiness (*b* = .07, *SE* = .02, *p* < .05, 95% CI = .03, .12), which indicated mediation in the model, such that higher levels of ACE severity (more traumatic/frequent occurrences of ACEs), were associated with more anxiety symptoms, which were associated with more sleepiness symptoms. See *Appendix* for Mediation Figure 3.

Figure 2.
Adverse Childhood Experiences (Exposure) and Sleepiness Mediation Model



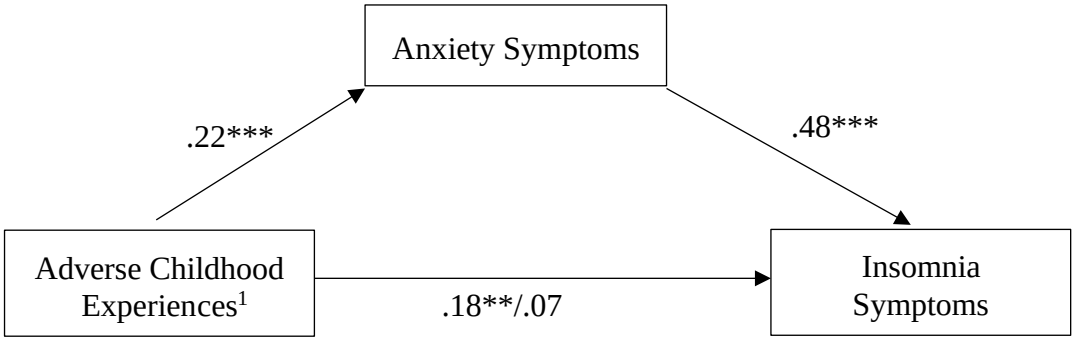
p is significant at the .001 level. ***

[1] Exposure

Coefficients are standardized.

Insomnia Symptoms A simple mediation model examining ACE exposure and insomnia symptoms resulted in a significant association (path A) from ACE exposure to anxiety symptoms as mentioned is the previous model, and it resulted in a significant association (path B) from anxiety symptoms to insomnia symptoms (*b* = .48, *SE* = .03, B = .31, *p* < .001, 95% CI = .25, .37). There was also a significant direct effect (path C) for ACE exposure and insomnia symptoms (*b* = .18, *SE* = .11, B = .35, *p* < .01, 95% CI = .13, .57). The pathway (path C') for ACE exposure and insomnia symptoms while controlling for anxiety symptoms was not significant. However, there was a significant indirect effect for ACE exposure on insomnia symptoms, which confirms that mediation is present in the model (*b* = .10, *SE* = .03, B = .21, *p* < .05, 95% CI = .05, .16). The presence of mediation in the model would indicate that experiencing a higher number of ACEs is associated with experiencing more anxiety symptoms, and that more symptoms of anxiety are associated with more symptoms of insomnia. See Figure 4.

Figure 4.
Adverse Childhood Experiences (Exposure) and Insomnia Mediation Model



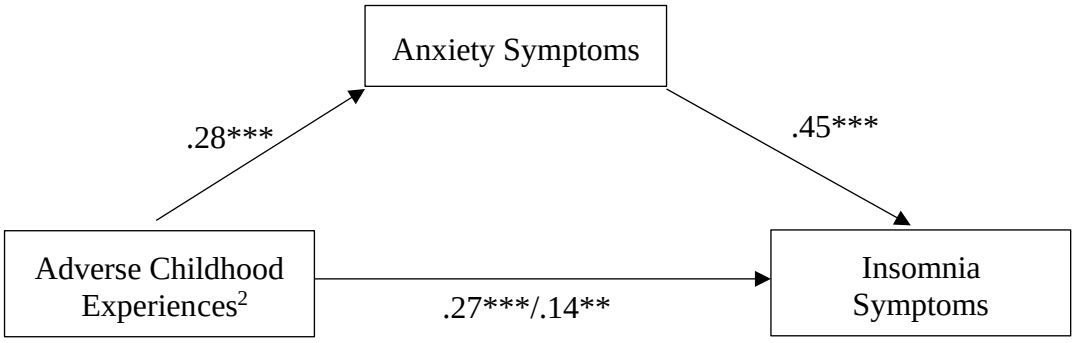
p is significant at the .01 level. ** *p* is significant at the .001 level. ***

[1] Exposure

Coefficients are standardized.

A simple mediation model was also utilized to examine the pathway between ACE severity and insomnia symptoms. The model indicated a significant association (path B) for anxiety symptoms and insomnia symptoms (b = .45 SE = .03, B = .29, p < .001, 95% CI = .23, .36). The results produced a significant direct association (path C) for ACE severity and insomnia symptoms (b = .27, SE = .01, B = .04, p < .001, 95% CI = .03, .06), and when controlling for anxiety (path C') in the model (b = .14, SE = .01, B = .02, p < .01, 95% CI = .01, .04). Lastly, there was a significant indirect effect for ACE severity and insomnia symptoms (b = .13, SE = .3, p < .05, 95% CI = .07, .19), which indicated mediation was occurring in the model, and that higher levels of ACE severity (more traumatic/frequent occurrences of ACEs), were associated with more anxiety symptoms, which was associated with more symptoms of insomnia. See Figure 5.

Figure 5.
Adverse Childhood Experiences (Severity) and Insomnia Mediation Model



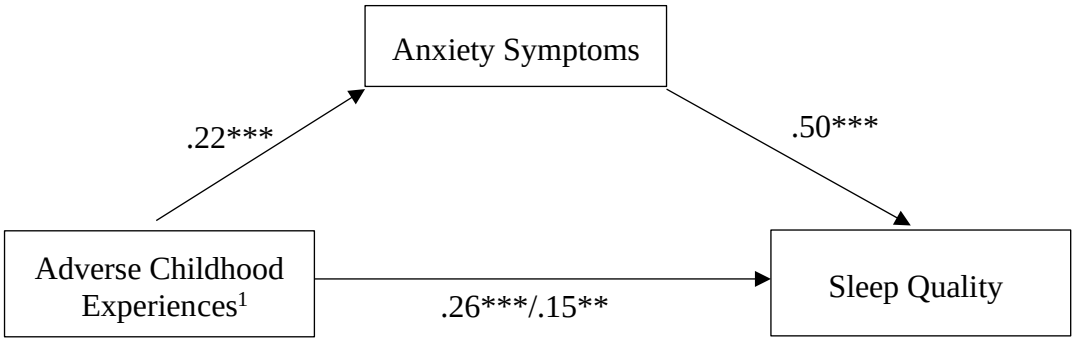
p is significant at the .01 level. ** *p* is significant at the .001 level. ***

[2] Severity

Coefficients are standardized.

Next, a simple mediation examined ACE exposure and sleep quality symptoms. There was a significant direct effect (path C) for ACE exposure and sleep quality symptoms (*b* = .26, *SE* = .13, B = .62, *p* < .001, 95% CI = .37, .88), and a significant effect for ACE exposure and sleep quality (path C') when controlling for anxiety (*b* = .15, *SE* = .12, B = .37, *p* < .01, 95% CI = .14, .59). Furthermore, there was a significant indirect effect for the model, indicating mediation is occurring in the model (*b* = .11, *SE* = .03, B = .26, *p* < .05, 95% CI = .05, .17), such that experiencing more types of ACEs was positively associated with experiencing more anxiety symptoms, and more anxiety symptoms were positively associated with more poor sleep quality symptoms. See Figure 6.

Figure 6.
Adverse Childhood Experiences (Exposure) and Sleep Quality Mediation Model



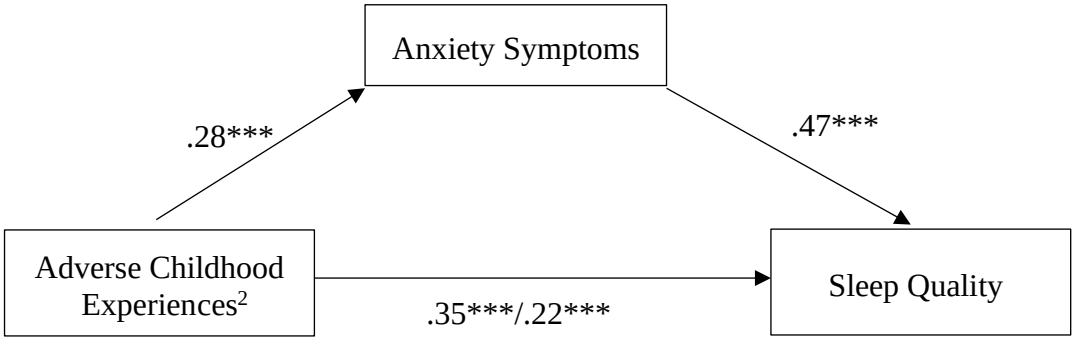
p is significant at the .01 level. ** *p* is significant at the .001 level. ***

[1]Exposure

Coefficients are standardized.

Lastly, I conducted a s simple mediation model to examine ACE severity and sleep quality symptoms. The results indicated that there was a significant direct effect (path C) for ACE severity and sleep quality symptoms (b = .35, SE = .01, B = .07, p < .001, 95% CI = .05, .09), and a significant effect for ACE severity and sleep quality (path C') when controlling for anxiety (b = .22, SE = .01, B = .04, p < .001, 95% CI = .02, .06), Furthermore, there was a significant indirect effect for the model, indicating mediation is occurring in the model (b = .13, SE = .03, B = .03, p < .05, 95% CI = .07, .19). Specifically, that experiencing more ACE severity (more trauma/more frequent ACEs) was positively associated with experiencing more anxiety symptoms, and amore anxiety symptoms were positively associated with more poor sleep quality symptoms. See Figure 7.

Figure 7.
Adverse Childhood Experiences (Severity) and Sleep Quality Mediation Model



p is significant at the .001 level. ***

[2] Severity

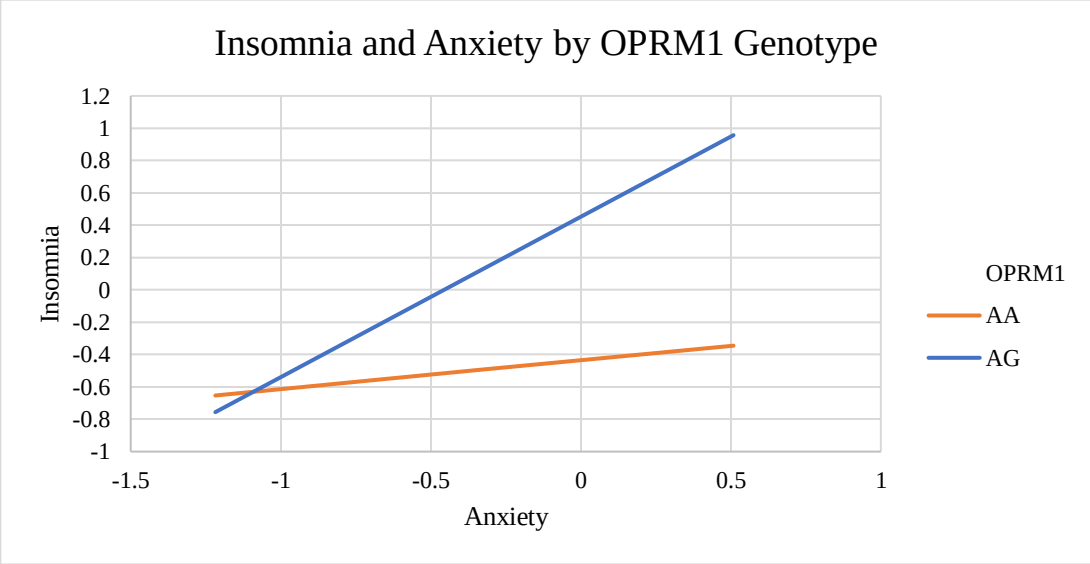
Coefficients are standardized.

Exploratory Moderated-Mediation Model

In my subsample of 20 individuals, none of the participants had the GG genotype. Consistent with previous research and literature on social rejection and resilience, I defined the groups by the absence vs presence of the G allele (Slavich et al., 2014; Way et al., 2009). I conducted the moderated-mediation model using PROCESS Model 58 with the OPRM1 genotypes (AA vs AG) input as the moderator. I also conducted the models with ACE exposure as the predictor and then repeated the analysis with ACE severity as the predictor. In regard to the results, there were no significant interactions or pathways when sleepiness was input as the outcome variable in the model. Indicating that OPRM1 (the genotype one carries for the OPRM1 gene) does not moderate the mediation pathway from ACE exposure/ACE severity to anxiety symptoms and does not moderate the mediation pathway from anxiety symptoms to sleepiness. Next, I tested if OPRM1 would moderate the mediation model when insomnia was entered as the outcome variable. The model demonstrated a significant interaction for OPRM1, anxiety symptoms, and insomnia symptoms when ACE exposure was input as the predictor (B = .53, *SE* = .22, *p* = .03, 95% CI = .07, .99), and when ACE severity was input as the predictor (B = .48, *SE* = .21, *p* = .04, 95% CI = .03, .94). The statistically significant interaction indicates that OPRM1 genotypes moderated the mediation pathway for anxiety and insomnia symptoms. Specifically, individuals with the AG genotype who have more anxiety symptoms were associated with experiencing more insomnia symptoms than individuals with the AA genotype. See Figure 8. No other significant results were reported. Lastly, sleep quality was input as the outcome variable in the moderated-mediation model. The results did not show a significant

interaction for OPRM1, anxiety symptoms, and sleep quality; however, anxiety did decrease in significance to marginally significant $p < .10$. This could indicate some variance was absorbed by the moderator, OPRM1.

Figure 8.
Moderated-Mediation Interaction with Insomnia as Outcome



CHAPTER 4

DISCUSSION

The thesis aimed to acquire greater perspective and insight into the impact from ACEs by investigating sleep, mental health, and genetics. Examining the model through a biopsychosocial lens provided more opportunity for subjective and objective data that could aid research and treatment toward optimal outcomes for individuals with early life trauma. The thesis also discussed the importance of sleep and how sleep quality can decline when considering the impact of one's ACE history.

The mediation model results supported previous research that had examined ACE impact and increased risk to mental and physical health. ACEs also had a significant impact on sleep quality, experiencing insomnia symptoms, and experiencing sleepiness, which all greatly impact daily functioning and health. Without proper sleep, vital regulatory functions within the body and mind can become impeded, such as digestion, memory, attention, emotion regulation, and problem-solving ability (de Zambotti et al., 2018; Tarokh et al., 2016). ACEs, a known public health risk in itself, has similar overlapping risks to physical and mental health, such as the increased risk of hypertension, diabetes, anxiety, and depression (Felitti et al., 1998; de Zambotti et al., 2018; Tarokh et al., 2016). Although, the mediation model confirmed the ACE, anxiety, and poor sleep quality association, it is important to note that not all contributing factors to poor sleep were collected in the self-report measure or controlled for in the analyses. Specifically, participants were not asked about drug or alcohol use, which could greatly affect sleep duration and quality. If an individual gets two hours of sleep most evenings because of stimulant use, the sleep data may not accurately reflect natural sleep patterns

and should be controlled for when running the analyses. In addition, if an individual frequently consumes central nervous system depressants such as alcohol on a frequent basis, the sleep data may show increased sleepiness or longer durations of sleep, which would also affect the data and would need to be controlled for to ensure valid interpretations of the data can be made. Future studies would greatly benefit from participants' drug and alcohol use data in conjunction with sleep data, and when examining any post-ACE habits, behaviors, and possible coping mechanisms.

Furthermore, the study had a lengthy self-report which asked about sensitive life experiences such as retrospective and current ACE history, perceived trauma, and statements regarding what an individual may think to themselves when anxious, depressed, and stressed. Many participants completed the self-report measures remotely in their own private space, which could be beneficial for their assurance of anonymity; however, due to the nature of the measures, there is also concern of not fully knowing how each participant felt during and after completing the measure. Future studies would benefit from gathering information on participants wellbeing during and post participation to ensure accurate data is being collected and burnout is not a risk.

In regard to the moderated-mediation model, the exploratory analysis showed a statistically significant interaction for OPRM1, anxiety, and insomnia symptoms. The model demonstrated that individuals with the AG genotype experience more insomnia symptoms when anxiety is high than individuals with the AA genotype. As the research literature has previously predicted and tested, mental health and sleep are very intertwined and perhaps the OPRM1 gene does have an effect on that association. Due to the significant result, further investigation into the OPRM1 gene, sleep, and anxiety could be

promising; however, no other significant pathways were present in the model. The lacking association for OPRM1, anxiety, and ACEs was unexpected and did not support the literature. In response, specific factors should be considered when interpreting these results. First, the sample for the DNA analysis was small and was limited to individuals that lived within 20 miles of ASU West campus. Due to the smaller sample size, results did not collect a representative sample for the GG genotype. The majority of the results included individuals with the AA genotype (65% of the DNA sample), followed by the AG genotype (35% of DNA sample). Therefore, due to the lack of GG genotypes in the sample, the study was not able to test the exploratory moderation with all genotypes present (as originally planned). Future studies would benefit from recruiting a larger sample size to allow more opportunity for acquiring more variant samples.

Furthermore, the DNA samples may differ in overall collection quality. Participants were able to self-collect with guidance from the researcher; however, participants were not pre-screened prior to saliva collection in terms of ensuring they did not eat or drink any substance that could hinder the quality of the sample and affect the ability to extract usable DNA from the samples. Also, recent health history was not collected. If an individual was sick or had the start of a throat infection, the saliva quality would greatly differ in overall bacterial composition and viscosity. The lab's ability to extract and interpret DNA from the saliva sample, could be greatly decreased. Although, all DNA sequencing samples in the exploratory portion of this study were able to be analyzed for the genotypes, future studies would benefit from pre-screening participants with a valid and reliable tool to ensure the best quality sample is supplied.

In addition, the in-person participants for the saliva collection were all ASU undergraduate students which could have been a limitation by not providing the most diverse sample in terms of age distribution among the young adult sample. Most of the participants that provided a saliva sample were within the late teens and early twenties age range, which created a lost opportunity to explore any major differences when investigating differences across the cohort in terms of ACE perception, overall well-being, and if there are any genotype differences in terms of overall resilience. Also, due to the younger age range within the saliva collection sample, the younger participants might not have fully addressed their own mental health and adverse childhood experiences yet, which could affect how they self-report. Additionally, many of the young adults could have been adapting to college life for the first time, which could affect sleep quality as well. Future studies would benefit from recruiting participants from all professions and life milestones within the young adult population to gather the full transitional period into adulthood.

Lastly, the utilization of a cross sectional design provides convenience and access to a large amount of data within a short period of time; however, it lacks the ability to collect data over a longer period of time to investigate changes in mental and physical health throughout young adulthood. The age-range of the young adult population provides a great opportunity for exploring the transition from adolescence to adulthood due to the still developing frontal lobe in the early half of young adulthood versus the fully developed frontal lobe by the second half of the young adulthood. Future studies would benefit from using a longitudinal research design to collect trend data for these important developmental periods.

CHAPTER 5

CONCLUSION

Human development relies on optimal environmental conditions for healthy development and outcomes. ACEs can hinder development and result in life long negative consequences. Throughout this thesis, I have outlined the impact from ACEs within a biopsychosocial scope to emphasize all interacting components of human development. Childhood paves the path for social and behavioral learning, which is shaped by our experiences and social survival instincts. For children who do not get the support they need to create more healthy bonds and relationships, may be at an increased risk for developing negative behavior patterns, coping mechanisms, and anxious or avoidant attachment styles (Barnett & Howe, 2021, Kural & Kovac, 2022; Simpson & Rholes, 2017). My thesis aimed to take a step further into how these negative patterns in childhood could manifest in psychological and biological processes during adolescent development and young adulthood, which introduced anxiety, sleep, and genetics into the model design.

I proposed that ACEs and sleep quality would be positively associated and that higher scores on the ACE measures (more ACE exposure and/or more ACE severity) would be positively associated with more poor sleep symptoms (insomnia, sleepiness, and poor sleep quality symptoms). The direct effect (path C) was not significant for the outcome of sleepiness; however, insomnia symptoms was significant when ACE severity was the predictor, and general sleep quality symptoms was a significant outcome for both predictors (ACE severity and ACE exposure). In addition, there was significant indirect effects for all mediation models, indicating mediation is occurring in the model and that

anxiety is mediating the pathway between ACEs and sleep. The significant results supported the prior research on ACE impact and the increased risk of poor mental and physical health (Danese & McEwen, 2012; Elmore & Crouch, 2020; Kajeepeta et al., 2015; Nelson, 2017; Sousa et al., 2018; Wadman et al., 2020). The significant mediation also supported prior research that discussed the impact of poor mental health resulting in an increased risk of experiencing poor sleep quality symptoms, such as the inability to fall asleep, and frequent awakenings (Abrams et al., 2008; Bader et al, 2013; Baiden et al., 2015; Cecil et al., 2015; Kajeepeta et al., 2015; McNally & Clancy, 2005; Noll et al., 2006; Stallman et al., 2018; Tarokh et al., 2016). Finally, I proposed that the OPRM1 genotypes for the SNP rs1799971/A118G would moderate the mediational pathways. The analyses for the moderated-mediation model did not provide enough support for my exploratory hypothesis. Instead, the majority of the model resulted in nonsignificant results. The only interaction that was significant included insomnia as the outcome variable, which did support the anxiety and insomnia literature. However, due to the majority of the paths resulting in no significance and due to a small sample size, the results should be interpreted carefully. A larger sample size is warranted to gather a more conclusive result.

In conclusion, this thesis demonstrated support for the association between ACEs, anxiety, and poor sleep symptoms, and for the moderated effect of OPRM1 on anxiety and insomnia symptoms. Although ACEs are well known to society, their presence and impact continue to remain. While professionals work to prevent ACEs in at-risk populations, there are still those that have already been affected. By continuing to

discover and understand individual differences in processing ACEs, future research and treatment can be tailored with the individual in mind, and as a result, introduce new paths for healing.

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APPENDIX A

STUDY MEASURES

Appendix A1: Adverse Childhood Experiences (Felitti et al., 1998)

Each statement is scored either Yes (1) or No (0). Add all values to get the ACE score.

Reflect on your childhood:

1. Did a parent or other adult in the household often or very often...

Swear at you, insult you, put you down, or humiliate you?

Act in a way that made you afraid that you might be physically hurt?

2. Did a parent or other adult in the household often or very often...

Push, grab, slap, or throw something at you?

Ever hit you so hard that you had marks or were injured?

3. Did an adult or person ever...

Touch or fondle you or have you touch their body in a sexual way?

Attempt or actually have oral, anal, or vaginal intercourse with you?

4. Did you often or very often feel that ...

No one in your family loved you or thought you were important or special?

Your family didn't look out for each other, feel close to each other, or support each other?

5. Did you often or very often feel that ...

You didn't have enough to eat, had to wear dirty clothes, and had no one to protect you?

Your parents were too drunk or high to take care of you or take you to the doctor if you needed

6. Was a biological parent ever lost to you through..

Divorce, abandonment, or other reason?

7. Was your mother or stepmother…

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

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

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

8. Did you live with anyone who…

Was a problem drinker or alcoholic who used street drugs?

9. Was a household member…

Depressed or mentally ill or did a household member attempt suicide?

10. Did a household member ever…

Go to prison?

Perceived Trauma *(Added to measure)*
How traumatic was [ACE]?

0 = not traumatic 1= slightly 2 = moderately 3 = quite 4= severely

ACE Frequency *(Added to measure)*
How often did [ACE] occur?

0 = never 1 = less than once a year 2 = once a year

3 = a couple times a year 4 = monthly 5 = daily or weekly

ACE Years *(Added to measure)*
Provide the age range in which you experienced [ACE]

Starting Age_____ __ Ending Age_____

Appendix A2 Trait Anxiety (Spielberger, 1983)

Read each statement and select the statement that indicates ***how you*** *g*enerally feel. There are no right or wrong answers. Do not spend too much time on one statement but give the answer which seems to describe your present feelings best.

Never (0) Rarely (1) Sometimes (2) Often (3) Always (4)

1. **I feel pleasant**
2. **I feel nervous and restless**
3. **I wish I could be a happy as others seems to be**
4. **I feel like a failure**
5. **I feel rested**
6. **I am "calm, cool, and collected"**
7. **I feel difficulties are piling up so that I cannot overcome them**
8. **I worry too much over something that doesn't really matter**
9. **I am happy**
10. **I have disturbing thoughts**
11. **I lack self-confidence**
12. **I feel secure**
13. **I make decisions easily**
14. **I feel inadequate**
15. **I am content**
16. **I take disappointment so keenly that I can't put them out of my mind**
17. **I am a steady person**

18. I get in a state of tension or turmoil as I think over my recent concerns and interests.

19. have you felt nervous and “stressed”?

Appendix A3: Epworth Sleepiness Scale (Johns, 1991)

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

0 = would **never** doze
1 = **slight chance** of dozing
2 = **moderate chance** of dozing
3 = **high chance** of dozing

1. Sitting and reading

2. Watching TV

3. Sitting, inactive in a public place (e.g., a theatre or a meeting)

4. As a passenger in a car for an hour without a break

5. Lying down to rest in the afternoon when circumstances permit

6. Sitting and talking to someone

7. Sitting quietly after a lunch without alcohol

8. In a car, while stopped for a few minutes in the traffic

Appendix A4: Insomnia Severity Index (Morin et al., 2011)

The Insomnia Severity Index has seven questions. The seven answers are added up to get a total score. Rate each statement based on your sleep quality **within the past month.**

None (0) Mild (1) Moderate (2) Severe (3) Very Severe (4)

1. Difficulty falling asleep

2. Difficulty staying asleep

3. Problems waking up too early

Very Satisfied (0) Satisfied (1)
Moderately Satisfied (2) Dissatisfied (3) Very Dissatisfied (4)

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

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

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

Total score categories:
0–7 = No clinically significant insomnia
8–14 = Subthreshold insomnia
15–21 = Clinical insomnia (moderate severity)
22–28 = Clinical insomnia (severe)

Appendix A5: Pittsburgh Sleep Quality Index (Buysse et al., 1989)

The following questions relate to your usual sleep habits during the past month only.

1. **During the past month, when have you usually gone to bed at night?**
2. **During the past month, how long (in minutes) does it usually take you to fall asleep each night?**
3. **During the past month, when have you usually gotten up in the morning?**
4. **During the past month, how many hours of actual sleep did you get at night?**

For each of the following questions, select the one best response.

Not during the past month (0)
Less than once a week (1)
Once or twice a week (2)
Three of more times a week (3)

5. During the past month, how often have you had trouble sleeping because you….

a. Cannot get to sleep within 30 minutes
b. Wake up in the middle of the night or early morning
c. Have to get up to use the bathroom
d. Cannot breathe comfortably
e. Cough or snore loudly
f. Feel too cold
g. Feel too hot
h. Had bad dreams
i. Have pain
j. Other, please describe

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

Very good (0) Fairly good (1) Fairly Bad (2) Very Bed (3)

---

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

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

Not during the past month(0) Less than once a week (1)
Once or twice a week (2) Three or more times a week (3)

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

No problem at all (0) Only a very slight problem (1)
Somewhat of a problem (2) A very big problem (3)

APPENDIX B

TABLES AND FIGURES

B1: Descriptive Statistics and Correlation Tables

Table 3.
Descriptive Statistics for ACE Exposure by Gender

Gender		VA	PA	SA	EN	PN	DV	PL	SUB	MH	PRI
Male	Frequency	30	20	9	28	7	13	18	13	14	5
	%	43	29	13	41	10	19	26	19	20	7
	N	69	69	69	69	69	69	69	69	69	69
Female	Frequency	131	86	65	111	30	42	81	56	83	25
	%	59	39	29	50	13	19	36	25	37	11
	N	223	223	222	223	223	223	223	223	223	223
Non-Binary	Frequency	21	12	10	22	7	5	8	15	9	1
	%	81	46	38	85	27	19	31	58	35	4
	N	26	26	26	26	26	26	26	26	26	26

Note. Frequency = total number of participants with ACE history within gender, % = Percent of participants with ACE history within gender, N = total number of responses, VA = verbal abuse, PA = physical abuse, SA = sexual abuse, EN = emotional neglect, PN = physical neglect, DV = domestic violence, PL = parental loss, SUB = substance abuse within household, MH = mental illness within household, PRI = household member incarcerated

Table 4.

Main Variable Descriptive Statistics by Gender

Gender		ACE Exposure	ACE Severity	Anxiety	Sleepiness	Insomnia	Sleep Quality
Male	Mean	2.32	21.28	18.59	4.58	8.14	10.72
	N	69	69	69	69	69	69
	Std. Deviation	2.59	29.72	8.06	3.08	5.04	5.70
Female	Mean	3.28	30.15	20.00	5.30	9.42	13.35
	N	223	223	223	223	223	223
	Std. Deviation	2.50	29.71	7.57	3.46	4.95	5.87
Non-	Mean	4.31	46.96	23.73	5.31	11.92	16.77
Binary	N	26	26	26	26	26	26
	Std. Deviation	2.41	34.68	9.22	3.03	5.73	7.23

Table 5.

Main Variable Descriptive Statistics by OPRM1 Genotype

OPRM1 Genotype		ACE Exposure	ACE Severity	Anxiety	Sleepiness	Insomnia	Sleep Quality
AA	Mean	4.00	35.62	18.00	5.46	6.85	10.31
	N	13	13	13	13	13	13
	Std. Deviation	3.39	35.12	6.98	4.07	2.94	4.01
AG	Mean	4.71	44.57	15.71	3.86	9.00	14.29
	N	7	7	7	7	7	7
	Std. Deviation	3.59	43.63	6.78	2.61	4.83	5.88

Appendix B2: Mediation Coefficients Tables

Table 6.

Simple Mediation Models for Sleepiness

	Outcome variables							
	ACE[1] on Sleepiness				ACE[2] on Sleepiness			
	Coeff	*SE*	*p*	CI [LL, UL]	*Coeff*	*SE*	*p*	CI [LL, UL]
Effect of ACE on Anxiety (Path A)	**.67**	**.17**	**< .001**	**.34, 1.00**	**.07**	**.01**	**< .001**	**.05, .10**
Effect of Anxiety on DV (Path B)	**.11**	**.02**	**< .001**	**.06, .15**	**.11**	**.02**	**< .001**	**.06, .16**
Direct effect of ACE on DV (Path C)	.11	.07	.14	-.04, .25	.01	.01	.20	-.00, .02
Effect of ACE on DV (Path C')	.04	.07	.61	-.11, .18	.00	.01	.99	-.01, .01
Indirect effect of ACE on DV	**.07**	**.02**	**< .05**	**.03, .12**	**.01**	**.00**	**< .05**	**.00, .01**

Note. Significant effects are shown in **bold** font. Coefficients are unstandardized. *N* = 318

Coeff = coefficient; *SE* = standard error, CI = confidence intervals; LL = lower limit, UL = upper limit; ACE[1] = Adverse Childhood Experience Exposure; ACE[2] = Adverse Childhood Experience Severity. 55

Table 7.

Simple Mediation Models for Insomnia Symptoms

	Outcome variables							
	ACE[1] on Insomnia				ACE[2] on Insomnia			
	Coeff	*SE*	*p*	CI [LL, UL]	*Coeff*	*SE*	*p*	CI [LL, UL]
Effect of ACE on Anxiety (Path A)	**.67**	**.17**	**< .001**	**.34, 1.00**	**.07**	**.01**	**< .001**	**.05, .10**
Effect of Anxiety on DV (Path B)	**.31**	**.03**	**< .001**	**.25, .37**	**.45**	**.03**	**< .001**	**.23, .36**
Direct effect of ACE on DV (Path C)	**.35**	**.11**	**<.01**	**.13, .57**	**.04**	**.01**	**< .001**	**.03, .06**
Effect of ACE on DV (Path C')	.15	.10	.15	-.05, .34	**.02**	**.01**	**< .01**	**.01, .04**
Indirect effect of ACE on DV	**.21**	**.06**	**< .05**	**.10, .34**	**.02**	**.01**	**< .05**	**.00, .03**

Note. Significant effects are shown in **bold** font. Coefficients are unstandardized. $N = 318$

Coeff = coefficient; *SE* = standard error, CI = confidence intervals; LL = lower limit, UL = upper limit; ACE[1] = Adverse Childhood Experience Exposure; ACE[2] = Adverse Childhood Experience Severity.

Table 8.

Simple Mediation Models for General Sleep Quality Symptoms

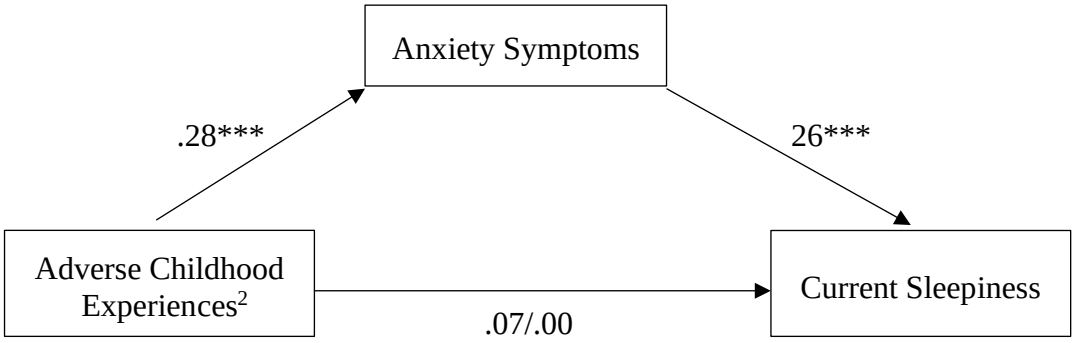
	Outcome variables							
	ACE[1] on Sleep Quality				ACE[2] on Sleep Quality			
	Coeff	*SE*	*p*	CI [LL, UL]	*Coeff*	*SE*	*p*	CI [LL, UL]
Effect of ACE on Anxiety (Path A)	**.67**	**.17**	**< .001**	**.34, 1.00**	**.07**	**.01**	**< .001**	**.05, .10**
Effect of Anxiety on DV (Path B)	**.39**	**.04**	**< .001**	**.31, .46**	**.36**	**.04**	**< .001**	**.29, .44**
Direct effect of ACE on DV (Path C)	**.62**	**.13**	**<.001**	**.37, .88**	**.07**	**.01**	**< .001**	**.05, .09**
Effect of ACE on DV (Path C')	**.37**	**.12**	**<.01**	**.14, .59**	**.04**	**.01**	**< .001**	**.02, .06**
Indirect effect of ACE on DV	**.26**	**.07**	**< .05**	**.13, .41**	**.03**	**.01**	**< .05**	**.02, .04**

Note. Significant effects are shown in **bold** font. Coefficients are unstandardized. $N = 318$

Coeff = coefficient; *SE* = standard error, CI = confidence intervals; LL = lower limit, UL = upper limit; ACE[1] = Adverse Childhood Experience Exposure; ACE[2] = Adverse Childhood Experience Severity.

B3: Mediation Pathway Models

Figure 3.
Adverse Childhood Experiences (Severity) and Sleepiness Mediation Model



p is significant at the .001 level. ***

[2] Severity

Coefficients are standardized.

APPENDIX C

IRB APPROVAL LETTERS

C1: IRB Approval

APPROVAL: EXPEDITED REVIEW

Kristin Mickelson
NCIAS: Social and Behavioral Sciences, School of (SSBS)
602/543-1632
Kristin.Mickelson@asu.edu

Dear Kristin Mickelson:

On 2/3/2022 the ASU IRB reviewed the following protocol:

Type of Review:	Initial Study
Title:	Examining the Association Between Adverse Childhood Experiences and Sleep Quality
Investigator:	Kristin Mickelson
IRB ID:	STUDY00015357
Category of review:	
Funding:	Name: Arizona State University (ASU)
Grant Title:	
Grant ID:	
Documents Reviewed:	• Consent Forms.pdf, Category: Consent Form; • IRB: Social and Behavioral, Category: IRB Protocol; • Recruitment Script.pdf, Category: Recruitment Materials; • Self-Report Measures, Category: Measures (Survey questions/Interview questions /interview guides/focus group questions);

The IRB approved the protocol from 2/3/2022 to 2/2/2023 inclusive. Three weeks before 2/2/2023 you are to submit a completed Continuing Review application and required attachments to request continuing approval or closure.

If continuing review approval is not granted before the expiration date of 2/2/2023 approval of this protocol expires on that date. When consent is appropriate, you must use final, watermarked versions available under the "Documents" tab in ERA-IRB.

In conducting this protocol you are required to follow the requirements listed in the INVESTIGATOR MANUAL (HRP-103).

REMINDER - - Effective January 12, 2022, in-person interactions with human subjects require adherence to all current policies for ASU faculty, staff, students and visitors. Up-to-date information regarding ASU's COVID-19 Management Strategy can be found here. IRB approval is related to the research activity involving human subjects, all other protocols related to COVID-19 management including face coverings, health checks, facility access, etc. are governed by current ASU policy.

Sincerely,

IRB Administrator

cc: Elise Bailey

C2: IRB Modification Approval

APPROVAL: MODIFICATION

Kristin Mickelson
NCIAS: Social and Behavioral Sciences, School of (SSBS)
602/543-1632
Kristin.Mickelson@asu.edu

Dear Kristin Mickelson:

On 4/30/2022 the ASU IRB reviewed the following protocol:

Type of Review:	Modification / Update
Title:	Examining the Association Between Adverse Childhood Experiences and Sleep Quality
Investigator:	Kristin Mickelson
IRB ID:	STUDY00015357
Funding:	Name: Arizona State University (ASU)
Grant Title:	None
Grant ID:	None
Documents Reviewed:	• IRB Social Behavioral Form_Phase2.pdf, Category: IRB Protocol; • On Campus Directions to Lab, Category: Participant materials (specific directions for them); • Phase Two Consent.pdf, Category: Consent Form; • Phase Two Recruitment Confirmation and Availability , Category: Recruitment Materials; • Saliva Kit Info and Instructions.pdf, Category: Participant materials (specific directions for them);

The IRB approved the modification.

When consent is appropriate, you must use final, watermarked versions available under the "Documents" tab in ERA-IRB.

In conducting this protocol you are required to follow the requirements listed in the INVESTIGATOR MANUAL (HRP-103).

REMINDER - Effective January 12, 2022, in-person interactions with human subjects require adherence to all current policies for ASU faculty, staff, students and visitors. Up-to-date information regarding ASU's COVID-19 Management Strategy can be found here. IRB approval is related to the research activity involving human subjects, all other protocols related to COVID-19 management including face coverings, health checks, facility access, etc. are governed by current ASU policy.

Sincerely,

IRB Administrator

cc: Elise Bailey
Elise Bailey
Kristin Mickelson