CALIFORNIA STATE UNIVERSITY SAN MARCOS

THESIS SIGNATURE PAGE

THESIS SUBMITTED IN PARTIAL FULFILLMENT
OF THE REQUIREMENTS FOR THE DEGREE

MASTER OF ARTS

IN

PSYCHOLOGICAL SCIENCE

THESIS TITLE: The Role Ethnicity Plays in the Relationship Between Sleep and Stress Response

AUTHOR: Ruby Cuellar

DATE OF SUCCESSFUL DEFENSE: April 30, 2018

THE THESIS HAS BEEN ACCEPTED BY THE THESIS COMMITTEE IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF ARTS IN PYCHOLOGICAL SCIENCE.

Kim Pulvers THESIS COMMITTEE CHAIR	Kim Pulvers SIGNATURE	5/2/18 DATE
Kim D'Anna Hernandez THESIS COMMITTEE MEMBER	SIGNATURE	5/1/18 DATE
Kim Tsai THESIS COMMITTEE MEMBER	KTsai SIGNATURE	5/1/18 DATE

The Role Ethnicity Plays in the Relationship Between Sleep and Stress Response

Ruby Cuellar

California State University San Marcos

Table of Contents

Abstract

Sleep disturbances are widely prevalent and represent a significant health problem in the general population. Minorities sleep a significantly less amount of time than Caucasians. Given that Latinos are the largest ethnic minority in the U.S., it is important to understand patterns and correlations of sleep characteristics in this population. The overall objective of this study was to examine the association between self-reported sleep characteristics and stress response between Mexican American and Caucasian college students. For this purpose, a study was designed where stress response would be measured by changes in heart rate and changes in cortisol levels following a stressful lab task with 133 undergraduates. Sleep was measured using ten consecutive days of sleep diaries preceding the lab stress test. It was hypothesized that poor sleep would be associated with a greater stress response. It was also hypothesized that Mexican Americans would report less sleep duration and efficiency; more sleep variability, as well as worse quality of sleep compared to Caucasians. Given these predictions, it was expected that Mexican Americans would exhibit higher levels of stress response, compared to Caucasians. Results indicated no significant sleep differences between Mexican Americans and Caucasians. However, a non-significant trend was found indicating ethnicity as a potential moderator for the relationship between sleep duration and stress reactivity when measured by heart rate. No other relationships was significant. Ultimately, the present study was able to fill a gap in the literature and determine whether any sleep differences exist in a sample of equally represented Mexican American and Caucasian. This study in particular, addressed the low representation of Latinos in previous studies and the college population, as well as possible negative health outcomes related to sleep problems.

The Role Ethnicity Plays in the Relationship Between Sleep and Stress Response

Sleep disturbances are widely prevalent and represent a significant health problem in the general population. Minorities sleep significantly less than Caucasians (Jean-Louis et al., 2000; Loredo et al., 2010), however little sleep research has been conducted with Latinos. Given that Latinos are the largest growing minority in the U.S., with Mexicans representing the largest group, it is essential to learn more about patterns of sleep in this population. In addition to determining whether there is a difference in sleep between Mexican Americans and Caucasians, it is important to examine the link between sleep and stress response given that it is a potential risk factor for subsequent health problems. This is especially necessary among young adults given that college students face unique stressors and have higher risks of being poor sleepers (Doane, Gress-Smith, & Breitenstien, 2015; Sladek & Doane, 2015). Research has recently identified a bi-directional relationship between sleep and stress; however, most previous research has studied stress as a predictor of sleep (Garde et al., 2012). The present study examined the less-studied relationship of sleep as a predictor of stress response. The reason for this is emerging evidence indicating a self-reinforcing cycle between sleep and stress. In other words, poor sleep is associated with more stress the following day, and greater stress in the day is associated with poor sleep during the night. Stress response was measured via changes in salivary cortisol and heart rate following a lab-based stress test among a sample of Mexican Americans and Caucasians college students.

This study aimed to explain the health risks associated with sleep disturbances as well as provide details on the link between psychological stress response and sleep. The background of this paper describes how lack of sleep or sleep disturbance can affect stress health outcomes and how stress can contribute in the development of psychological disorders such as insomnia, depression, and anxiety. This paper informs readers of the negative impacts that can arise from insufficient and poor quality sleep. The overall objective of this research was to examine the association between self-reported sleep characteristics and stress response between Mexican Americans and Caucasians, specifically, the role that sleep plays in how one responds and recovers from stress. Furthermore, this paper outlines the research questions, hypotheses, and methods designed to answer these questions. Finally, results are reported and future directions are discussed.

Background

Sleep is essential for daily functioning, health, and optimal development. Sleep problems such as trouble falling asleep and staying asleep are common in the general population, with prevalence estimates ranging from 13% to as high as 52% (Hamilton, Nelson, Stevens, Kitzman, & Heather, 2007). Many have speculated that sleep patterns have changed over the past several decades in the U.S. In particular, evidence suggests that the percentage of short sleepers — individuals who on average sleep less than eight hours per night —has increased over the past 30-40 years (Knutson, Van Cauter, Rathouz, Deleire & Lauderdale, 2010). The National Sleep Foundation (2006) recommends that adults obtain eight hours of sleep per night; however, the time adults in the United States spend asleep has steadily fallen. On average, the sleep duration for American adults is only 6.9 hours per night (Patel et al., 2004). These numbers are even worst among young adults, especially college students. Specifically, evidence suggests that adolescents do not obtain sufficient sleep as they transition into college (Doane et al., 2015; Sladek & Doane, 2015) and that younger adults go to bed later and receive less sleep than teenagers (Tsai & Li, 2004). In fact, many university students meet the requirements for partial sleep deprivation (e.g. less than 5 hours of sleep in a 24 hour period) and delayed sleep phase syndromes (difficulty falling asleep and waking up).

Minority Sleep

Minorities, specifically Latinos, sleep significantly less than Caucasians (Jean-Louis et al., 2000; Loredo et al., 2010). Latinos are the largest ethnic minority group in the U.S and are expected to comprise approximately 30% of the population by 2050, but little research has been done to understand the sleep patterns and sleep characteristics of this population. Of the Latino population living in the United States, Mexicans and Mexican Americans are the largest subgroup; therefore, they were be the focal sample in this investigation. Most studies on human sleep physiology and epidemiology have been obtained from Caucasian populations; therefore, results cannot be generalized to minority populations (Pedraza et al., 2011). There is evidence that suggests the prevalence of sleep problems and predictors of sleep duration are different in Latinos versus Caucasians (Loredo et al., 2010).

First, past studies have reported differences in sleep architecture — percentage of time spent in rapid eye movement (REM) and non-REM sleep— between different racial groups, with Latino children experiencing less deep sleep than Caucasian children (Loredo et al., 2010). Similarly, compared to Caucasian youth, Mexican Americans had a higher risk for insomnia and hypersomnia after controlling for age, gender, and socioeconomic status (Roberts, Roberts, & Chen, 2000). Finally, there is evidence suggesting ethnic differences in environmental determinants of sleep quality (e.g. number of sleepers per room, proximity of room noise, inner city living), such that Latino children are more likely to live in these types of environmental conditions compared to Caucasians (Loredo et al., 2010) and thus potentially suffer from adverse sleep quality.

There are also conflicting findings that report Latinos are less likely to be short sleepers compared to Caucasians (Knutson et al., 2010). Similarly, Krueger and Friedman (2009) found that Mexican Americans have the same odds as Caucasians —of sleeping both long and short hours. A different study found that relative to Caucasians respondents, non-Mexican Latinos were at an increased risk of reporting short sleep, however, Mexican Americans are more likely to be long sleepers (sleep more than 9 hours, Hale & Do, 2007). One possible reason for the inconsistency in the literature can be the sample sizes. Most studies that look at ethnic differences have a sample of predominantly Caucasian individuals and the rest are of various ethnic minorities, with Latinos comprising less than 15% of the total sample (Knutson et al., 2010, Krueger & Friedman, 2009, Jean-Louis, 2000). The current study filled a gap in the literature by recruiting an equal number of Latino and Caucasian participants to compare sleep characteristics of these groups.

More research is required to examine sleep health and stress among different ethnic and racial groups. Research suggests that racial/ethnic discrimination may be associated with poor sleep, as it is perceived as a chronic stressor to minority groups (Loredo et al., 2010). There is literature that demonstrates low sleep duration may be a risk factor for developing physical health conditions (i.e. cardiovascular disease, obesity, and diabetes), as well as impact mental health and well-being (depression or depressive symptoms, anxiety, and psychological distress) (Fuligni & Hardway, 2006). However, little is known about sleep characteristics and sleep related health issues among Latinos. Considering the rapid growth of the Latinos population in the United States and the gaps in the literature focusing on this population, examining factors associated with sleep disturbances is crucial for the development of culturally sensitive prevention and intervention programs. This study aimed to examine whether ethnicity plays as a role in the relationship between sleep and stress. Because research implies that minorities sleep less than Caucasians, examining this relationship will provide clarification on whether being Latino influences the strength of the relationship between sleep and stress response.

Sleep characteristics and disturbances

Sleep characteristics have been broadly addressed in terms of sleep duration, sleep quality, and sleep variability (Kachikis & Breitkopf, 2012). Based on existing literature, sleep duration and quality have been shown to be strongly related to health and well-being (Galambos, Dalton, & Maggs, 2009). Additionally, in the research that links stress to poor sleep, researchers tend to focus on sleep duration and quality more than any other sleep related variables. Sleep duration is described as the quantity or amount of sleep an individual gets. More specifically, sleep duration can be further broken down into sleep efficiency. Sleep efficiency can be described as the proportion of the time that an individual spends actually asleep from the moment they fall asleep to the moment they wake up (Alapin et al., 2000). Thus, sleep efficiency takes into consideration periods when an individual may wake up during the night. On the other hand, sleep quality is described as the overall satisfaction of an individual's sleep and is typically self-reported by the individual. Correlational studies indicate that poor sleep quality is related to physical and psychological health complaints, such as anxiety, depression, and fatigue (Galambos et al., 2009).

Sleep variability is another important component of sleep research, however only a small number of studies have focused on the natural patterns of sleep. Sleep variability represents how much participants' sleep varies across days. This is calculated by taking the mean of the absolute differences between participants' average nightly sleep time and each individual night's sleep time (Fuligni & Hardway, 2006). Most of this literature has studied sleep variability by using a single survey and estimating general patterns and sleep correlates. These studies possess limitations that the present study addresses with the use of daily diaries. It is more difficult to obtain valid estimates of daily variability by asking participants to recall their sleep behaviors from the prior week. The use of daily diaries allowed estimations of students' general sleep patterns that were more accurate.

Sleep disturbances or problems include various challenges such as trouble falling asleep, trouble maintaining sleep, shorter sleep than average sleep times, higher activity during sleep, several extended night awakenings, or sleep schedules that vary from night to night (Yip, 2015). The sleep disturbances can be quantified into sleep characteristics. For example, sleep duration can be measured as number of awakenings at night and sleep latency, where sleep quality includes largely subjective indices of sleep, such as depth of sleep, how rested one feels upon awakening, and general satisfaction with sleep (Pilcher, Ginter, & Sadowsky, 1997). Additionally, sleep disturbances, as a risk factor, have been observed to be associated with compromised physical and mental health, such fatigue, daytime sleepiness, and depression (Hamilton et al., 2007; Yip, 2015)

Research Methodologies to Assess Sleep

Sleep can be measured in a variety of ways. To measure sleep in a more objective manner, equipment such as actigraphy watches and polysomnography (PSG) can be used. Sleep actigraphy can be used to asses sleep duration; efficiency and latency by having individuals wear watch-like devices that monitor their physical movements throughout the day and/or night. Another form of measuring sleep objectively is by PSG. PSG is used to record brain waves, the oxygen level in the blood, heart rate, breathing, as well as eye and leg movement during sleep. PSG can be used to measure sleep duration, sleep efficiency, sleep architecture, and disordered breathing (Beatty et al., 2011). Sleep can also be measured subjectively with self-reports. There are two forms of self-report: retrospective measures and daily diaries. An example of a retrospective measure is The Pittsburg Sleep Quality Index (PSQI) that measures sleep quality over the previous month. There are limitations to retrospective measures, such that asking participants to recall sleep patterns and behaviors from the past month can produce inaccurate estimations. The use of daily diaries provides a more accurate representation of sleep patterns across multiple nights as individuals are asked to report on their sleep behaviors, such as the time they went to bed and woke up, each day. Additionally, daily self-reports are particularly valuable in capturing the day-to-day variability of sleep.

Health Risks Associated with Sleep Disturbances

Sleep disturbances are associated with compromised physical and psychological health outcomes. Sleep deprivation may increase the risks for developing cardiovascular disease, obesity, and diabetes (Kachikis & Breitkopf, 2012; Krueger & Friedman, 2009; Patel et al. 2004). In addition, poor sleep can impair mental health and well-being, such as contributing to depression or depressive symptoms, anxiety (Hamilton et al., 2007), psychological distress (Beatty et al, 2011; Yip, 2015), learning and memory problems, (Krueger & Friedman, 2009) and an increased risk for morbidity and mortality (Hale & Do, 2007; Hamilton et al., 2007; Kachikis & Breitkopf, 2012; Knutson et al., 2010; Patel, Gottlieb, White, & Hu, 2006).

Stress affects sleep

Various psychological factors, such as stress, daily hassles, rumination, and hyper-arousal are known to play essential roles in the development of sleep disturbances. Stress is one of the most common and well-known predictors of sleep disturbances (Winzeler, Voellmin, Schafer, Meyer, Cajochen, Wilhelm, & Bader, 2014) and for that reason, it was the primary psychological factor that this research study focused on. Minor and major stressful events are correlated with more sleep disturbances (Kashani, Eliasson, & Vernalis, 2012). Major stressors, such as a severe illness or significant loss, are associated with insomnia and an increased risk for the development of sleep problems. Minor stressors, such as work or school pressure, have also been associated with disturbed sleep (Winzeler et al., 2014). A study on the relationship between stress and sleep over a six-week period in 50 healthy adults showed that bedtime stress and worry were the two main predictors of poor subjective sleep quality (Akerstedt et al., 2012). Evidence for a bi-directional association between stress and sleep is emerging, indicating a self-reinforcing cycle (see Figure 1).

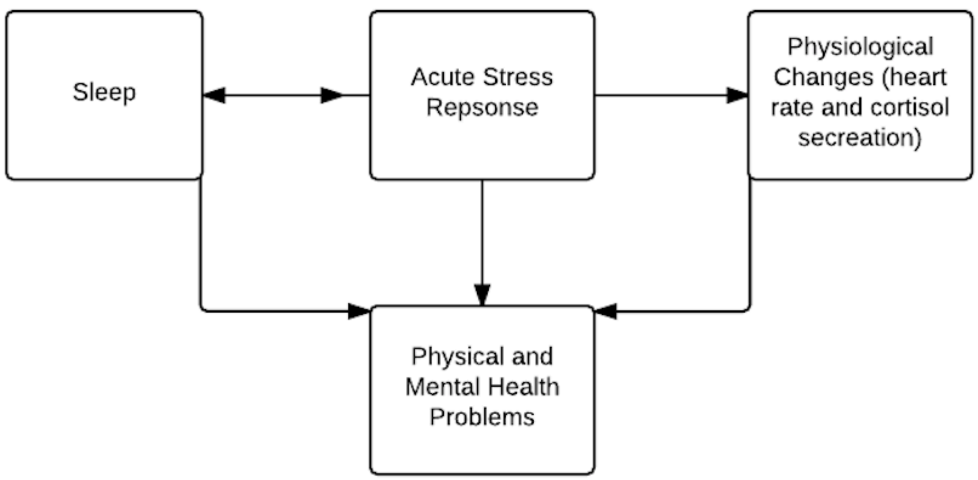


Figure 1. Conceptual Model of Sleep and Stress Response. Where sleep and stress are associated with physiological changes and physical and mental health problems.

Sleep affects stress response

A majority of research focuses on the effects that stress or daily hassles have on sleep. Garde et al. (2012) found evidence for a bi-directional relationship between stress and sleep. In this study, higher ratings of stress at bedtime were associated with ratings of poor sleep the following night. Additionally, higher ratings of poor sleep in the morning were associated with higher ratings of stress during the following day. Similarly, Galambos et al., (2010) found that better sleep was an indicator of positive affect and decreased negative affect in college students. When focusing on healthy students, results showed that moderately impaired sleep was linked to higher psychological arousal, such as increase stress and worry at bedtime. Likewise, researchers found that sleep duration and sleep quality protected patients with chronic pain from emotional disruptions related to physical pain and stress (Hamilton et al., 2007). Zohar and colleagues (2005) suggest that sleep ensures adequate cognitive energy to meet the demands of both challenging and stressful situations. In particular, longer sleep durations tend to reduce stress response —the capacity to respond to a stressor (Schlotz, Yim, Zoccola, Jansen, & Schulz, 2011). Furthermore, Hamilton and colleagues (2007) conducted a study examining the relationship between sleep habits and dimensions of psychological wellbeing, such as appraisal. They found that optimal sleepers reported fewer symptoms of stress, anxiety, and depression. Similarly, Ryff et al. (2004) showed that sleep duration was positively correlated with psychological well-being, such as self-development and purposeful engagement. Therefore, there is evidence indicating the crucial role that sleep plays in one’s response to stress and consequentially, one’s psychological well-being.

Findings such as these suggest that adequate sleep plays an important role in how individuals respond to stress. Of the few studies that have focused on how sleep affects one’s stress response, there is still more that needs to be learned. In a search of the literature to understand how daily sleep affect stress, no study was found to have examined sleep patterns across multiple days and followed it up by exposing participants to a stress task. The closest study included an experimental condition in which participants were sleep deprived and a control condition. After a 3-day sleep manipulation, participants completed the Trier Social Stressor in order to assess the effects of sleep deprivation on physiological stress response (Minkel et al., 2014). Whereas the Minkel et al., (2014) study manipulated participants' sleep, the present study aims to examine young adults' natural sleep patterns. Specifically, how sleep influences their stress response. Although it is undoubtedly true that disturbed sleep plays an important role in the etiology and/or maintenance of some forms of psychopathology and physical illness, optimal sleep may be as important to psychological wellbeing. In other words, the absence of disturbed sleep does not equate with the presence of restorative sleep necessary for the body. It is therefore important that sleep research utilizes broad assessment to characterize both normal/restorative and disturbed sleep.

Sleep and Psychopathology

Sleep deprivation or poor sleep quality have significant implications for mental health, as sleep disturbances are common among individuals with anxiety disorders and depressive symptoms. In fact, the majority of sleep research has focused on the relationship between sleep pathology (e.g. insomnia) and psychopathology, specifically depression and anxiety. Sleep disturbances are one of the most common symptoms of depression (Patel et al., 2006) with 60%-90% of depressed individuals reporting disturbed sleep. Several studies have found a relationship between sleep disturbances, especially insomnia, and anxiety (Breslau et al., 1996; Krueger & Friedman, 2009; Kachikis & Breitkopf, 2012). In anxiety disorders, there is increased arousal that makes it difficult to fall or stay asleep. It is also likely that those who suffer from anxiety also experience a general tendency to worry, especially when they are stressed, which then results in the inability to sleep (Kelly, 2003). Breslau, Roth, Rosenthal, and Andreski (2005) found that people who sleep five hours or less per night were 2.1 times more likely to meet criteria for an anxiety disorder.

The stress-diathesis model of psychopathology suggests that individuals have, to varying degrees, vulnerabilities or “diatheses” that predispose them toward potentially developing mental health problems. Once these vulnerabilities interact with stressful life events, they increase the chances of triggering the onset of mental health problems, such as insomnia (Drake, Pillai, & Roth, 2014). In contrast to sleep disturbances, insomnia is a clinical disorder characterized by chronic difficulties to initiate or maintain sleep; these difficulties are then associated with significant daytime impairment or distress. A longitudinal study by Morin et al. (2009) found that 10%-14% of individuals who reported sleep disturbances at baseline developed insomnia disorder a year later at their follow-up assessment. Sleep reactivity is the tendency to exhibit pronounced sleep disturbances in response to daytime stressors; thus, sleep reactivity may then constitute a premorbid vulnerability for future insomnia. This is especially important because individuals, who respond to chronic stress with more sleep reactivity, may harbor a predisposition for insomnia (Drake, Pillai, & Roth, 2014). Because past literature has found evidence for a bi-directional association between stress and sleep that indicate a self-reinforcing cycle, it is important to expand the model in the opposite direction by studying how sleep characteristics affect stress response. It is important to identify the impact of disturbed sleep as a stressor, given the negative cascade of mental health problems that can occur.

Sleep and Physiological Stress Response

The stress response consists of two mechanisms: the autonomic nervous system (ANS) and the hypothalamic pituitary adrenal (HPA) axis. Assessing beats per minute (BPM) is a commonly used marker of the ANS. Changes in heart rate indicate stress responses more consistently and reliably that other psychophysiological indicators (Croissamt, Demmel, Rist, Olbrich, 2011).

Another biomarker used to measure stress is through hormonal secretion. During the activation of the HPA axis, acute stress stimulates elevated cortisol levels via the secretion of corticotropin- releasing hormone (CRH) from the hypothalamus and adrenocorticotropic hormone of the pituitary. Similar to the physiological stress response, stress can also trigger a psychological response. Exposure to acute stress may lead to adaptive processes including emotion regulation and the display of behavioral responses (Hellhammer & Schubert, 2011). These types of adaptations are important in coping with stressors and may possibly work as mediators for positive or negative emotional outcomes.

There are factors that affect the HPA axis; one of those factors could be sleep. The HPA axis is the main neuroendocrine mediator of the stress response (McEwen, 2000; Maruyama et al., 2012). Hormones of the HPA axis, such as CRH and cortisol, play a specific role in sleep regulation (Balbo, Leproult, & Van Cauter, 2010). Sleep onset exerts an inhibitory effect on cortisol secretion, whereas awakening and sleep offset are accompanied by cortisol stimulation. In other words, during sleep, the stress response is shut down, thus impeding the secretion of cortisol. Upon awakening, the stress response becomes activated and the production of cortisol is once again stimulated.

Some authors have reported no change or a slight decrease in cortisol levels after one or several nights of total deprivation (Balbo et al., 2010). The findings supporting decreased cortisol levels after sleep deprivation have been interpreted as related to an increase in fatigue and sleepiness; if the body is too tired because it has been exposed to prolonged stress, the body experiences blunted cortisol levels. However, conflicting findings have also been reported. Many studies have reported an elevation of cortisol levels during both the night of sleep deprivation and the following day, particularly in the afternoon (Balbo et al., 2010, McEwen, 2006). There are also studies that find high glucocorticoid levels in conditions of acute sleep loss (Balbo et al., 2010). These finding are interpreted as reflecting the stress put on the body to maintain wakefulness. It is important to note that having either high or low levels of stress is not relevant; any deviation from the norm is what can cause detrimental outcomes. Because the focus of this study involved using an acute stressor, and not a chronic stressor, it was predicted that participants who report poor sleep (e.g., less sleep duration and efficiency, more sleep variability, and poor sleep quality) and who are exposed to the TSST would experience higher levels of cortisol than those who report normal sleep.

Although there is sufficient evidence that suggests that stress can affect the outcome of sleep, there is less evidence that focuses on the effects that sleep can have on stress response. The few articles that do look at reverse effects report conflicting findings. It is important to investigate how sleep affects the stress response given that it is a potential risk factor for subsequent health problems. A goal of this study was to examine the role various sleep characteristics have on stress response. Additionally, this study was interested in examining the relationship between baseline cortisol levels and heart rate and post-stressor cortisol levels and heart rate depending on these sleep characteristics. It was predicted that individuals who report less sleep duration, quality, efficiency, and more variability would have higher cortisol levels and increased heart rate after exposure to the stressor compared to individuals who report better sleep.

Laboratory Social Stress Task

Psychological stress is known to play an important role in the etiology of human psychopathologies such as anxiety and depression (Bryne, Davenport, & Mazanov, 2007). Therefore, examining the changes in physiological stress response requires the application of valid repeatable stress provocation tools. Acute stress responses can be induced experimentally by exposing participants to uncontrollable situations that constitute social-evaluative threats, such as the Trier Social Stressor Test (TSST) (Kirschbaum, 1993). The TSST consists of a mock interview and a serial subtraction task.

The TSST induces a physiological response through an increase in heart rate (ANS) and cortisol levels (HPA axis), as well as psychological measure such as stress, emotional insecurity, and anxiety in subjects (Hellhammer & Schubert. 2011). Reliable laboratory stressor paradigms are essential tools for studies that aim to investigate neurobiological mechanisms that link stress response to health outcomes (Chrousos & Gold, 1993; Yim, Quas, Rush, Granger, & Skoluda, 2015). The TSST has become one of the most widely used lab stressors to systematically manipulate the degree of arousal, which is why it was chosen for this study. Extensive research suggests the effectiveness of the TSST in inducing a multidimensional stress response (Boesh et al., 2014; Taylor et al., 2010, Wiemers, Schoofs, & Woolf, 2013). Measuring both the HPA axis and ANS together – the two main physiological stress systems – demonstrates that the TSST is a powerful psychosocial stress task inducing an acute stress response of all main physiologic stress systems.

Stress Health Outcomes

Stress is a threat, real or implied, to homeostasis; homeostasis refers to the maintenance of vital physiological parameters necessary for survival (McEwen, 2000). The body responds to stress by activating physiological and behavioral central nervous systems and peripheral adaptive responses. If these responses become excessive and or prolonged, they may affect personality development and behavior; they may also have adverse consequences on physiological function (Charmandari, Tsigos, & Chrousos, 2005). Past research suggests that stress in moderation can be seen as beneficial, however, consequences of prolonged stress include adverse psychological and physical health effects as well as an increased risk of premature mortality (Keller et al., 2012; Lantz, House, Mero, & Williams, 2005; Dinan, 2005). Importantly, the effects of stress on well-being are so well recognized that U.S. Public Health officials have been calling for a reduction of stress since the 1970s (PSPHS, 1979).

Stressful life events in adolescents and young adulthood are associated with negative outcomes such as decreased well-being and impaired mental health (Laurent et al., 2015; Frederick, 2016). Stress is one of the most common and well-known factors associated with the development and maintenance of symptoms in major psychiatric disorders such as depression and anxiety. Similarly, there is a clear relationship between the HPA axis (indicated by high response levels of cortisol), stress exposure, and increased risk for internalizing psychopathology, such as depression and anxiety (Laurent et al., 2015).

Psychological stress can affect cognitive function in the short-term and long term. In the short-term, minor daily stressors can produce temporary effects on cognition by reducing the amount of attention resources available for information processing (Sliwinski, Smyth, Hofer, & Stawski, 2006). Daily stress has also been associated with short-term increases in inflammation as well as in negative mood (Scott et al., 2015). Similarly, stressful experiences can trigger the release of hormones and other chemicals that can damage the immune systems and other vital organs in the long-term (Braveman, Egerter, & Mockenhaupt, 2011). This physiological chain of events can trigger a more rapid onset and progression of chronic illnesses, including cardiovascular disease. Over the long-term, chronic stress has also been associated with poorer cognitive functions, accelerated cognitive decline, and increased incidence of dementia. A possible explanation for these long-term effects is that individuals who experience chronic stress have an increased risk for biological “wear and tear” (i.e., allostatic load), that results in both dysregulated endocrine function and pro-inflammatory effects that can impair the neural structure and function underlying cognitive performance (Scott et al., 2015; Korten, Sliwinski, Comijis, & Smith, 2014). The notion of allostatic load implies that stressful experiences must result in chronic or prolonged responses to stressors in order to have a negative long-term effect on cognitive function.

Evidence suggests that the accumulated strain from trying to cope with daily stressors may, over time, contribute to far more physiological damage than a single stressful event (McEwen, 2006). The perception that stress affects one’s health is conceptually distinct from the amount of stress the individual experiences; for example, one could report experiencing very little stress but still believe it has a great impact on health. This can be especially true among groups that face more psychosocial stressors and health disparities.

The Current Study

The overall objective of the present study was to examine the association between self-reported sleep characteristics and stress response between Mexican Americans and Caucasian college students. For this purpose, a study was designed where sleep characteristics were measured using ten consecutive days of sleep diaries. Stress response was measured by change in heart rate and change in cortisol levels from exposure to the TSST. The study was designed to answer the following questions:

RQ1: Is there a difference in sleep between Mexican American and Caucasian college students?

RQ2: How does sleep impact stress response and recovery, and are there ethnic differences in this association?

Specifically, the following hypotheses were made:

H1: There will be a significant difference between Mexican Americans and Caucasians on the duration, quality, efficiency, and variability of their sleep. Specifically, Mexican Americans will report less sleep duration and efficiency; more sleep variability, as well as worse quality of sleep compared to Caucasians.

H2: Sleep efficiency, quality, duration, and variability will be significant predictors of stress response and recovery. Specifically, less sleep duration and efficiency, more sleep variability, and poorer sleep quality will be associated with greater stress response.

H3: Ethnic differences will play a significant role in the relationship between sleep and stress response and recovery. Specifically, Mexican Americans will experience greater stress response compared to Caucasians.

Preliminary work

In preparation for this study, a pilot study consisting of 18 participants (67% female, 41% Latinos, mean age = 22.26, *SD* = 2.5) was conducted. The goals of the pilot were to 1) gain experience with Trier Social Stress Task (TSST), and 2) test the sensitivity of procedures for measuring changes in cortisol of participants exposed to the TSST. The pilot study was designed to observe patterns in the data rather than being powered to detect statistically significant differences. This study provided preliminary evidence of an effective laboratory induced stressor and changes in physiological response.

In the pilot study, participants were recruited through summer classes at California State University San Marcos. Participants who qualified during the intake were then given instructions on how to complete the daily diaries (see procedure for more details) and were scheduled to come in for the second session. During the second session, participants were asked to abstain from eating or drinking anything an hour prior to their appointment. This was done because certain foods increase cortisol levels and food particles can contaminate the saliva samples. Participants were asked to give a saliva sample as soon as they walked in. Following the saliva sample, participants completed a packet of questionnaires. Once participants were done, they were connected to the Electrocardiogram (ECG) equipment in order to measure their heart rate. Participants were then read the speech prompt and given three minutes to prepare before they were sent into the TSST room. Following the stressor, participants were asked to give a second saliva sample (S2). A twenty-minute rest period began immediately after receiving S2. At the completion of the twenty-minutes, saliva sample 3 (S3) was taken; participants were unhooked from the ECG equipment, debriefed, and thanked for their participation.

All of the participants (100%) provided at least eight (M = 9.3, SD = 1.8) out of ten daily diaries; 72% provided 100% of daily diaries. This provides preliminary evidence of the feasibility of the daily diary method. In addition, the majority of participants (94%) who completed Session 1 also completed Session 2. Therefore, no more than 5% attrition is expected.

All session two testing were conducted between the hours of 10:00 am and 4:00 pm because cortisol activity follows a diurnal profile pattern. Previous studies have found a rise in cortisol following the exposure of a laboratory stressor such as the TSST (Hellhammer & Schubert, 2011). However, patterns in the pilot study revealed high levels in cortisol at S1 compared to S2 or S3 (see Table 1 for means). This could be due to pre-study nerves.

Additionally, other patterns showed that cortisol levels in S3 were higher than levels in S2. This could mean that the stressor was stronger than expected, and that participants need more time to recover and return to normal. Two one-way ANOVAs were conducted to detect differences in mean cortisol levels between S1, S2 and S3 for Latinos, F (2, 20) = 1.72, p = .207 and Caucasians, F (2, 26) = 1.29, p = .292. The results indicated no difference between the two groups and the time their saliva was taken. These abnormal cortisol patterns prompted us to modify the procedure in which saliva samples were taken. In order to give the participants more time to acclimate to the testing room, S1 was taken after participants were connected to the ECG equipment and a baseline heart rate established. Additionally, to account for the rise in cortisol from S2 to S3, participants were given 35 minutes to recover rather than 20. After consulting with a faculty member experienced with the TSST, these changes were believed to better capture the changes in cortisol levels from pre-TSST to post-TSST.

Methods

Participants

A power analysis was conducted to establish the appropriate size for the current study using the only study (Minkel et al., 2014) examining the effect of sleep deprivation on HPA axis stress response. The sleep-deprived participants reported significantly higher cortisol levels at baseline (d = 1.39) compared to control (d = .89). A moderate sized effect of .46 was present 40 minutes after the stressor. Because the present study did not sleep deprive participants, but rather examined normal sleep patterns, it seemed appropriate to use the lowest effect size in the literature. Therefore, 100 participants were required to detect a medium effect size of .46 with .80 power.

Participants were over-sampled by 10% to account for attrition, which resulted in a total sample of 133 undergraduate students (77% female, M_{age} = 20 years, 53% Mexican, 47% Caucasians, 90% 2nd generation or post 2nd generation) were recruited from the Human Participant Pool (HPP) in the Psychology Department at California State University San Marcos. Participants recruited through HPP were compensated with HPP credit. When recruitment extended into the summer, those recruited outside of HPP were offered course extra credit or were entered in a raffle to win a $25 Amazon gift card.

Inclusion criteria required participants to be enrolled college students, be 18-24 years old, be proficient in English and identify themselves as being Mexican American or Caucasian. Participants who reported any medical condition that could be exacerbated from the stress task were excluded from the study. Additionally, anyone who reported having an extreme fear of public speaking was also excluded from the study. Similarly, participants with a current psychiatric diagnosis related to stress or an inability to cope with stress were also excluded from the study.

Measures

Descriptive Measures.

Demographics. Participants were asked demographic questions about race/ethnicity, generation status, gender, and age.

Predictor Variable.

Daily Diary. Sleep patterns were monitored with an online sleep diary. Students were provided with a web link each day to complete a series of daily dairies privately and independently each night before going to bed. Participants were required to fill out the daily dairy for ten consecutive nights. A reminder email was automatically sent to every participant the following morning to ensure the completion of every diary. Diaries that were submitted after noon the following day were considered late and not used in final data. The daily diaries consisted of questions that asked about sleep duration, quality, naps, and caffeine (adapted from Fuligni, Tsai, Krull & Gonzales, 2015, see Appendix A for copy of diary). Upon completion, participants were scheduled for session two of the study where the TSST was completed. An aim of this study was to examine sleep patterns across the ten days, rather than at one time point in order to capture variability in individuals' sleep characteristics. A mean for sleep duration, quality, efficiency, and variability was calculated from the daily diary survey.

Stress Induction.

Trier Social Stress Task. The Trier Social Stress Test (TSST), adapted by Yim, Quas, Rush, Granger, & Skoluda (2015) was used to induce stress. Reliable laboratory stressor paradigms are essential tools for studies examining stress response. During the TSST, the participant entered a room where a male and female judge were waiting; they were wearing lab coats and already seated. The lead researcher explained to the participant that he/she would complete a speech and math tasks while being videotaped, and judges would analyze that video recording. The participants were given a prompt to talk about and were allowed three minutes to prepare. The lead researcher then walked the participant into the testing room, leaving the participant with the two judges. The participant was then instructed to begin his/her speech. The judges were trained to maintain a neutral stance throughout the TSST. The participants were prompted to speak freely for five minutes; if at any moment they stopped, they were instructed to continue speaking until the five minutes were up (only two reminders were allowed). If they stopped again or remained silent for 15 seconds, the judge would instruct them to continue standing for the remainder of the time. Following the speech tasks, the mental arithmetic task consisted of a 5-minute serial subtraction task (subtracting 17 from 2023). When participants made a mistake, they were instructed to start over from 2023. The whole TSST, including instructions and preparation period, was approximately 15 minutes. After the TSST was complete, the judges instructed the participant to exit the room and meet the lead researcher in the other room.

Outcome Variables.

HPA Activation. To measure the stress response to the TSST, participants' salivary cortisol levels were assessed. Three salivary cortisol samples were collected for this study. Stress reactivity was measured by calculating the change score in salivary cortisol, or BPM, from baseline (S1) and salivary cortisol, or BPM post stressor (S2). Similarly, stress recovery was measured by calculating the change score in salivary cortisol, or BPM, from post stressor (S2) and recovery (S3). Participants were instructed not to eat or drink anything an hour before their appointment. Salivary cortisol samples are a reliable and noninvasive marker for measuring stress. In order to avoid peak cortisol levels, testing was conducted between 10:00am and 4:00pm. Samples were stored in a -80 Celsius freezer until assayed. Samples were assayed using the High Sensitivity Cortisol Enzyme Immunoassay Kit (Salimetrics, State College, PA).

Autonomic stress response. The present study assessed the participants' autonomic response to stress by measuring heart rate using Biopac wireless Electrocardiogram (ECG) equipment and Bionomadix software. Participants were connected to the Biopac system using wireless Electrocardiogram (ECG) equipment five minutes before the initiation of the TSST in order to get a baseline measure and remained connected until the completion of the study.

Procedure

Session 1: Consent and Instruction. Participants were provided a consent form, which research assistants reviewed with them. Participants were given as much time as they needed to consider their consent. If a participant did not provide consent, they were still compensated one credit for their time, thanked, and dismissed. Participants recruited outside of HPP were given the opportunity to complete an alternate assignment in order to earn the same extra credit or were entered in a raffle a $25 Amazon gift card. Participants were screened out for fear of public speaking, any existing psychiatric diagnosis, or medical illness related to hormone excretion during a stress response (such as hyper- or hypothyroid), and any cardiac related diagnosis (see Appendix B for full list of exclusion criteria). If participants answered “yes” to questions pertaining to any exclusion criteria, they were thanked for their time, provided one credit, and dismissed from the study. Qualified participants were then asked to fill out their demographics and were instructed in the use of daily diaries. Lastly, research assistants scheduled participants for session 2 and answered any study related questions. Session 2 was scheduled about two weeks after the initial visit.

At Home Portion: Daily Dairies. Participants were sent a link to complete a daily diary around 6 pm for ten days. Participants were instructed to complete the daily dairies every night before bed. Participants automatically received an email at 6 am the following morning reminding them to complete the diary in case they had forgotten. Participants were told to ignore the second email if the diary had been completed the night before. The diary assessed the previous night’s sleep and current day’s naps and caffeine use.

Session 2: Psychological battery and TSST.

Pre-Task. Participants were asked to abstain from food or liquids an hour prior to testing. Compliance to this was ascertained at the beginning of the testing session. Upon arrival, participants were introduced to the task environment, and hooked up to the wireless ECG equipment. Participants were asked to sit down and breathe normally for five minutes while baseline heart rate was measured. At the end of the 5-minute acclimation period, saliva sample 1(S1) was obtained by having participants passively drool in a saliva storage tube. Participants were permitted to drink water only after the saliva sample has been taken, but they were instructed to abstain from drinking after entering the next part of the experiment.

TSST. Participants were briefed on the speech task. They were given three minutes to prepare a 5-minute speech to give in front of a panel that they believed were judging their performance. Following the speech, participants were instructed to perform some basic mental arithmetic – serial subtraction – for 5 minutes. At the conclusion of both tasks, the second saliva sample (S2) was collected.

Post-Task. Following the second saliva sample (S2), the thirty-five minute rest period began. Participants were given a questionnaire packet to complete and an eleventh daily diary as a measure to control for the prior night's sleep. Upon finishing, participants were invited to read neutral material and relax in the pre-task room for the remainder of the time. At the conclusion of the 35 minutes, the final saliva sample (S3) was obtained and participants were unhooked from the Biopac equipment. At the end, participants were debriefed on the study and supplied a referral for counseling services at the university (see Figure 2 for study flowchart).

Other Design Considerations

Additional to all the measures listed above, the study included exploratory measures such as the Depression, Anxiety and Stress Scale (Lovibond & Lovibond, 1995), the Societal, Attitudinal, Environmental, and Familial Acculturative Stress Scale (Mena, Padilla, & Maldonado, 1987), and the Everyday Discrimination Scale (Williams, Yu, Jackson, & Anderson, 1997). These measures were not included in the hypotheses because they are not the focus of the study, yet it was believed that including these measures would add value to better understanding ethnic difference in sleep related health issues.

The Depression, Anxiety, and Stress Scale (DASS-21). The Depression, Anxiety, Stress Scale-21 items (DASS-21) is a set of three self-report scales designed to measure the emotional states of depression (e.g., loss of self- esteem/incentives and depressed mood), anxiety (e.g., fear and anticipation of negative events), and stress (e.g., persistent state of arousal and low frustration tolerance) (Lovibond & Lovibond, 1995). A maximum score of 42 on each scale indicated high levels of depression, anxiety, and stress. Items included, “I experience trembling in the hands” (anxiety), “I felt that I have nothing to look forward to” (depression), and “I found it hard to wind down” (stress). Participants were asked, on a four-point scale, to rate how much each of the items applied to them over the past week, with 0 = “*did not apple to me at all*” to 3= “*applied to me much, or most of the time.*” The DASS-21 demonstrates good overall reliability (α = .88) and good reliability within each subscale (Depression scale $\alpha = .82$; Anxiety scale $\alpha = .90$; and the Stress scale $\alpha = .93$)

Acculturative Stress. Acculturative stress was measured using the Societal, Attitudinal, Familial, and Environmental Acculturative Stress Scale (SAFE) (Mena, Padilla, & Maldonado, 1987). This scale consists of 24 items and answers range from 1 (*Not Stressful*) to 5 (*Extremely Stressful*). Questions include: “I feel uncomfortable when others make jokes about or put down people of my ethnic background,” “I have more barriers to overcome than most people," and "Close family members and people look down upon me if I practice customs of my culture." This scale is shown to have good reliability ($\alpha = .89$).

Everyday Discrimination Scale. The Everyday Discrimination Scale (EDS) was used to measure chronic and daily unfair treatment (Williams, Yu, Jackson, & Anderson, 1997).

Participants were asked to report, on a 6-point Likert scale, how often they experienced unfair treatment in their everyday life. Responses range from 1 (never) to 6 (almost every day), with higher scores signifying greater perceived discrimination. Questions include “You are treated with less respect than other people are” and “People act as if they are afraid of you.” The EDS revealed good overall internal consistency (Cronbach’s alpha = .88).

Analytic Plan

Hypothesis 1: There will be a significant difference between Mexican Americans and Caucasians on the duration, quality, efficiency, and variability of their sleep. Specifically, Mexican Americans will report less sleep duration and efficiency; more sleep variability, as well as worse quality of sleep compared to Caucasians. A mean for sleep duration, efficiency, variability and quality was calculated from the daily diary survey, and these values were compared between Mexican Americans and Caucasian using independent samples t-tests. *Hypothesis 2: Sleep efficiency, quality, duration, and variability will be significant predictors of stress response and recovery. Specially, less sleep duration and efficiency, more sleep variability, and poorer sleep quality will be associated with greater stress response.* Stress reactivity, measured by cortisol and heart rate was regressed on sleep efficiency, quality, duration, and variability. Similarly, stress response, measured by cortisol and heart rate was regressed on the sleep variables mentioned above. Therefore, four multiple regressions were used to analyze hypothesis 2. *Hypothesis 3: Ethnic differences will play a significant role in the relationship between sleep and stress response and recovery. Specifically, Mexican Americans will experience greater stress response compared to Caucasians.* Four multiple regressions were tested to investigate whether the association between sleep (sleep efficiency, quality, duration, and variability) and stress reactivity (measured by both cortisol and heart rate) varied by ethnicity. Additionally, four more multiple regressions were tested to investigate whether the association between sleep (sleep efficiency, quality, duration, and variability) and stress recovery (measured by both cortisol and heart rate) varied by ethnicity

Missing Data.

Participants were only allowed to miss two out of the ten daily diaries in order for their data to be useable. This was done because sleep measures were averaged across the ten days, and an accurate representation of their sleep patterns would not be captured with more than two days of missing diaries. Based on pilot data, 25% of participants missed only one diary entry. No one missed more than one day. Additionally, only one person failed to complete all components of the study. In order to account for attrition, the study oversampled by 10%. One hundred and thirty-three participants were used in the final analytic sample.

Results

Following data collection and data entry, all data was checked in order to assess for normality, skewness, kurtosis, missing data, and outliers. Winsorizing was used to correct for normality and to address outliers with z scores greater than 2.58 SD above and below the mean for sleep efficiency (three participants), sleep variability (four participants), cortisol stress response (four participants), cortisol stress recovery (two participants), BPM stress response (one participant), and BPM stress recovery (two participants). The Winsorizing technique was used in order to retain all participant data even it is was not following the normal curve. Given the high attrition rate of this study, 133 participants completed the daily diaries; however, not all participants completed the second session. Due to this and uninterruptable salivary cortisol and heart rate data, the final sample was reduced.

After analyzing the salivary cortisol, the samples revealed the timing of the collection did not represent the pattern of a normal stress response (see Figure 3). The mean of sample 1 (baseline) was 0.26, the mean of sample 2 (response) was 0.28 and the mean of sample 3 (recovery) was also 0.28. These results reveal no difference between time 1 to time 2, or between time 2 to time 3. Additionally, this study also measured stress response using heart rate, specifically, average beats per minute (BPM). Three mean heart rate values were extracted for each participant from a 5-minute sample. In other words, average BPM were calculated from 5 minutes at baseline, 5 minutes during the Trier, and 5 minutes during the recovery period. When analyzing heart rate, average BPM demonstrates a normal stress response (see Figure 4).

H1: There will be a significant difference between Mexican Americans and Caucasians on the duration, quality, efficiency, and variability of their sleep. Specifically, Mexican Americans will report less sleep duration and efficiency; more sleep variability, as well as worse quality of sleep compared to Caucasians.

Four independent sample t-tests was conducted on a sample of college students, 71 Mexican Americans and 62 Caucasians to determine whether there was a difference in average self- reported sleep measures across ten days. As shown in Figure 5, results show no difference in the average amount of sleep duration reported between Mexican Americans (*M* = 7:13, *SD* = 1:05) and Caucasian students (*M* = 7:33, SD = 1:08), *t* (131) = 1.1, *p* = .27, 95% CI [-0:010:07, 0:34:27]. A second independent sample t-test was conducted to determine whether there was a difference in average self-reported sleep quality across ten days. As shown in Figure 6, results indicated no difference in reported sleep quality across ten days between Mexican Americans (*M* = 3.1, *SD* = .58) and Caucasian students (M = 3.2, SD = .50), *t* (134) = 1.4, *p* = .17, 95% CI [-.06, .32]. When testing whether there was a difference in mean sleep variability, Figure 7 shows no significant difference between Mexican Americans (*M* = 78.8, *SD* = 34.3) and Caucasians (*M* = 74.1, *SD* = 32.1), *t* (131)= .81, *p* = .42, 95% CI [-5.8, 16.1]. Similarly, Figure 8 showed no significant results when testing mean sleep efficiency between Mexican American (*M* = .89, *SD* = .06) and Caucasian college students (*M* = .91, *SD* = .06), *t* (131) = -1.4, *p* = .60, 95% CI [-.04, 005].

H2: Sleep efficiency, quality, duration, and variability will be significant predictors of stress response and recovery. Specifically, less sleep duration and efficiency, more sleep variability, and poorer sleep quality will be associated with greater stress response.

One hundred and five college students were surveyed about their sleep patterns and reaction to a social stressor. Four separate multiple regressions were conducted to test the predictive power of each sleep dimension (sleep duration, quality, efficiency, and variability) on stress response and recovery. All assumptions were met and two multivariate outliers were detected and removed. At first, the planned analyses controlled for day 11 (the day they came in for S2) sleep efficiency, duration, and quality. However, after seeing that model 1 did not contribute significantly to the overall model in all four multiple regression (with values less than 3%) and seeing that these additional variables violated the assumption of multi-collinearity, they were not included in the final model. In order to see if sleep predicted stress reactivity, the change score from baseline salivary cortisol to immediate post stress task salivary cortisol was regressed on the four sleep variables. Results indicated that the model as a whole contributed significantly and explained 12% of the variance, $R^2 = .12$ *F* (4,105) = 3.4, *p* = .011. Two variables emerged as significant unique predictors of stress reactivity: sleep variability and sleep duration. Specifically, less sleep duration and less sleep variability were associated with greater stress reactivity. Sleep quality and efficiency did not significantly predict stress reactivity among college students (see Table 2 for item wording and regression coefficients).

Similarly, to test the predictive power of sleep on stress recovery, change score from the saliva sample taken immediately after the stressor to the 35-minute post stressor saliva sample was regressed on the four self-reported sleep variables (sleep duration, quality, efficiency, and variability). Results indicated that the four sleep variables of interest did not contribute significantly to the model, $R^2 = .05, F$ (4, 104) = 1.2, *p* = .32. None of the variables emerged as significant unique predictors for stress recovery (see Table 3 for item wording and regression coefficients). These results indicate the sleep variability and duration were good predictors of stress reactivity but not recovery.

In addition to measuring the stress response and recovery with the use of salivary cortisol, this study also used average BPM. Stress reactivity was calculated by the change score in BPM from baseline and time 2, and stress recovery was calculated by change score from time 2 and time 3. Results indicated that the four sleep variables of interest did not contribute significantly to the regression model and only explained 2% of the variance, $R^2 = .03, F$ (4,107) = .73, *p* = .58 (see Table 4 for item wording and regression coefficients). Likewise, this model was also tested with stress recovery as the outcome. Again, the four predictor variables did not contribute significantly to the model and explained only 2% of the variance, $R^2 = .02, F$ (4,106) = .50, *p* = .74 (see Table 5 for item wording and regression coefficients).

H3: Ethnic differences will play a significant role in the relationship between sleep and stress response and recovery. Specifically, Mexican Americans will experience greater stress response compared to Caucasians.

Cortisol

In order to conduct these analyzes without inflating type I error, only two sleep variables were used in the regressions. Sleep duration was selected because it was most highly correlated with sleep variability and efficiency. Sleep quality was also selected because previous literature has found it to be the most significant sleep predictor for stress, and it was a primary variable of interest. This study examined the effect of ethnicity on the relationship between sleep duration and stress response in 107 college students. The Hayes PROCESS macro (Hayes, 2012) was used to conduct the multiple regression analyses. Participants' stress reactivity (changes in cortisol levels) was regressed onto their self-reported measures of sleep duration and ethnicity and the interaction between them. Ethnicity was a not significant moderator of the relationship between sleep duration and stress reactivity (b = -.0003, SE = .0002, p = .23, CI [-.0007, .0002]). The effect of ethnicity on the relationship between sleep duration and stress recovery was also examined. This time the participants' stress recovery was regressed onto their self-reported measures of sleep duration and ethnicity and the interaction between them. Ethnicity was not a significant moderator of the relationship between sleep duration and stress recovery (b = .00, SE = .0004, p = .92, CI [-.0008, .0008). Overall, these results suggest that being of Mexican American or Caucasian ethnicity does not influence the relationship between sleep duration and stress response or recovery.

Similarly, the effect of ethnicity on the relationship between sleep quality and stress recovery in 107 college students was examined. The same method above was used to conduct the multiple regression analyses. Results demonstrated ethnicity was not a significant moderator of the relationship between sleep quality and stress reactivity (b = .02, SE = .03, p = .36, CI [-.03, .08]). Next, this effect was examined with stress recovery as the outcome. Ethnicity was also not a significant moderator of the relationship between sleep quality and stress recovery (*b* = -.05, SE = .05, *p* = .33, CI [-.14, .05]). These results indicate that ethnicity did not influence the relationship between sleep quality and stress response and recovery.

Heart Rate

This study also measured it using heart rate, specifically, average beats per minute (BPM). As mentioned before, the change score in BPM from the stressor and baseline calculated stress response, and the change score in BPM from the recovery period and the Trier calculated stress recovery. When examining the effect of ethnicity on the relationship between sleep duration and average BPM during stress reactivity, results indicated a non-significant trend (*b* = -.08, SE = .04, *p* = .053, CI [-.15, .001). These patterns indicate that the more Caucasians sleep, the less changes in their average BPM, or less physiological activity is noted during the stressor. Mexicans on the other hand, showed a unique pattern of results. Although not significant, their pattern shows that the more sleep they reported, the higher their changes in their average BPM. In order words, more sleep resulted in a greater stress reactivity (see Figure 9). When examining stress recovery, results revealed the overall model was significant, R^2 = .10, F(3, 108) = 3.91, *p* = .01, but, the interaction between sleep duration and ethnicity was not significant (*b* = .04, SE = .03, *p* = .26, CI [-.03, .11]). These results indicate that no variable contributed to predicting variances in the overall model. Overall, there were no significant findings of ethnicity on the relationship between sleep duration and stress reactivity or recovery.

Similarly, this relationship was also examined with sleep quality as a predictor. Results show that ethnicity was not a moderator of the relationship between sleep quality and stress reactivity (*b* = -1.83, SE = 4.47, *p* = .68, CI [-10.68, 7.03]). When this relationship was examined looking at sleep quality as a predictor of stress recovery, results revealed the overall model was significant, $R^2 = .08$, $F(3, 108) = 3.23$, $p = .03$, but, the interaction between sleep quality and ethnicity was not significant ($b = -.97$, SE = 3.97, $p = .81$, CI [-.8.84, 6.90]). Overall, there were no significant findings of ethnicity on the relationship between sleep and stress response or recovery.

Discussion

The first aim of this study was to test the effects of ethnicity on the relationship between sleep and stress reactivity and recovery. In order to do that, the study first examined differences in sleep duration, efficiency, variability, and quality between Mexican Americans and Caucasians. Contrary to hypothesis 1, there was no significant difference between mean sleep duration, efficiency, variability, and quality reported by the two groups. The second goal of this study was to examine whether various sleep variables (sleep duration, quality, variability and efficiency), were significant predictors of stress response and stress recovery. The results revealed that two sleep variables contributed significantly to predicting stress response (measured by salivary cortisol). Specifically, sleep duration and variability emerged as significant unique predictors of ANS stress response, where less sleep duration and variability were associated with greater stress response. When examining this same model with heart rate measuring stress response, these findings were not significant. Similarly, when using these four variables to predict stress recovery (both cortisol and heart rate), the results were again not significant.

Finally, when testing whether ethnicity played a significant role in the relationship between sleep and stress none of the moderations were significant. Results, however, did reveal a non-significant trend when examining the effect of ethnicity on the relationship between sleep duration and average BPM during stress reactivity. These patterns demonstrated that the more

Caucasians sleep, the less changes in the average BPM, or the less reactivity noted during the stressor. However, Mexican Americans displayed a different pattern. Their pattern showed that the more sleep duration they reported, the more changes in their average BPM. An in-depth analysis of the results of the study's main hypothesis and research questions follows.

Sleep Differences Based on Ethnicity

When examining sleep differences between two ethnic groups, Mexican Americans and Caucasian, this study was unable to replicate the findings that sleep problems (e.g. less sleep time, less sleep efficiency) are more common among nonwhite minorities (Bixler et al., 2002; Jean-Louis et al., 2000). However, it should be noted that other studies have found the opposite, that minorities sleep more, to be true (Hale & Do, 2007). The two studies that found differences in sleep based on ethnicity had a sample size comprised of multiple ethnic minority backgrounds and some that did not include Latinos at all (Bixler et al., 2002; Jean-Louis et al., 2000). Additionally, the studies that looked at Latinos, specifically Mexican Americans have found conflicting results. Hale and Do (2007) found that Mexican Americans were more likely to be long sleepers (sleeping more than nine hours) compared to Caucasians; however Mexican Americans comprised only 4% of the entire sample, as opposed to their Caucasian counterparts who accounted for 79% of the entire sample. Another study found that compared to Caucasian youth, Mexican Americans had a higher risk for insomnia and hypersomnia after controlling for age, gender, and socioeconomic status (Roberts et al., 2000). These studies have yielded conflicting results that serve as evidence for the importance of equally represented minority groups.

The present study was able to fill a gap in the literature and determine whether any sleep differences exist in a sample of equally represented Mexican American and Caucasian. This study in particular, addressed the low representation of Latinos in previous studies and the college population, as college students are known to experience lifestyle changes that create precipitating factors that can affect both sleep and stress (e.g. erratic schedules, all-nighters, high-stress period like final exams; Lund, Reider, Whiting, & Prichard, 2010). In fact, many college students meet criteria for partial sleep deprivation (e.g., sleeping < 5 hr. in 24 hrs; Galambos et al., 2009). This transition process can take a toll on college students' overall health, and most importantly, it can be detrimental to their sleep behaviors. Results of this study indicated no significant differences between Mexican American and Caucasian college students in the amount of mean sleep duration, efficiency, variability, or the quality of sleep. Mexican Americans slept on average, 20 minutes less than their Caucasian counterparts did, but this relationship was not significant. One reason behind these findings could be the fact that college students in general report routinely getting insufficient sleep (Sladek & Doane, 2015). Any possible ethnic differences could have been overpowered due to the common characteristic of being a college student and the overall poor sleep of college students.

Sleep Predictors of Stress Response and Recovery

It is well established that stress throughout the day is associated with sleep disturbances at night (Akerstedt et al., 2012). Specifically, stress is one of the most common causes of sleep disturbances and insomnia (Winzeler et al., 2014). More recently, studies began to examine the reverse relationship, how poor sleep is linked to stress, poor daytime function, and overall well-being (Garde et al., 2010). When examining the predictive power of four sleep variables (sleep duration, quality, efficiency and variability) on stress reactivity and recovery (measuring both salivary cortisol and BMP), only one model was found significant. The results demonstrated that the overall model contributed significantly to predicting stress reactivity measured by salivary cortisol. Sleep variability and duration emerged as unique predictors of stress reactivity. The results indicate that less sleep duration across the ten days, and less sleep variability throughout the ten days, are associated with an increase in stress reactivity.

As expected from previous literature, less sleep duration was associated with a higher response to stress. Past studies have found that insufficient sleep contributes to poor mental and physical health over time, as poor sleep causes dysregulation in the HPA axis. (Minkel et al., 2014; Mrug, Tyson, Turan, & Granger, 2016). However, it was expected that more sleep variability to associate with an increased stress response, instead the opposite effect was found. Less sleep variability was associated with an increase in stress response. Similar to previous findings, the average daily variability in sleep of the entire sample was about an hour and fifteen min (*SD* = 33 min, Galambos et al., 2009; Lund et al., 2010). These studies have also found that college students are known to experience bedtime delay (later weekend than weeknight bedtime) and oversleep (sleeping longer on weekends than weeknights). These results can be due to that fact that less sleep variability was also associated with less sleep duration. In addition, most participants tended to have the weekend to compensate loss of sleep during the week, although those days were not tracked, this could have caused their average variability to go up. On the other hand, those who were unable to compensate sleep could be at risk for high stress response. Furthermore, this study was unable to replicate the findings that poor sleep quality was associated with higher levels of stress (Garde et al., 2012; Lund et al., 2010) or that poor sleep efficiency was associated with stress.

Ethnicity as a Moderator for Sleep

When examining the relationship between ethnicity and psychological distress or disorders, including sleep disorders, two competing hypotheses exist. One argues that observed ethnic differences are due mainly to social class effects (minority-status perspective); the other argues that ethnic effects are due primarily to mental health (ethnic-culture perspective) (Mirowsky & Ross, 1980). Roberts et al. (2006) examined ethnic differences in symptoms of insomnia among adolescents and found no significant ethnic differences, supporting the hypothesis that observed ethnic difference are primarily related to the effects of status differences (e.g. age, gender, SES, etc.). The present findings were unable to find significant differences in sleep based on ethnicity. Again, these findings may be related to the sample of college students that was used.

Similarly, no significant findings were reported using ethnicity as a moderator of sleep and stress. However, a non-significant trend was found when examining the effect of ethnicity on the relationship between sleep duration and average BPM during stress reactivity. These patterns demonstrate that the more Caucasians sleep, the less changes in the average BPM, or the less reactivity is noted during the stressor. These findings are consistent with the literature suggesting that more sleep is associated with less stress and overall well-being, as sleep is seen as a protective factor that reduce the response to stress (Schlotz et al., 2011) In fact, researchers suggest that sleep may be thought of as a resource for managing stress (Hamilton et al., 2007). Mexican Americans, on the other hand, displayed a different pattern. Their pattern suggest that the more sleep they had, the more they responded to the stressor. Although these patterns were not significant, they are consistent with the findings that more sleep leads to an enhanced stress response (Mrug et al., 2016). Mrug and colleagues found more sleep was associated to higher stress and interpreted these results as a possible compensatory mechanism for lack of sleep quality. In order words, when sleep quality is poor, one might compensate by sleeping longer.

This interpretation did not hold for this study because, both Mexican Americans and

Caucasians reported a normal sleep satisfaction. However, it should also be noted that although the overall model was a non-significant trend, the relationship of sleep and stress was stronger in the Caucasian group compared to the Mexican American group. While, Mexican Americans look like they are experiencing more stress the more they sleep, this relationship is not significant. These results serve as evidence for the difference in stress response associated with poor sleep between Mexican Americans and Caucasians. More research is needed on Mexican Americans and Caucasians in order to establish differences in sleeping behaviors and how they are linked to stress. More specifically, what factors are contributing to differences in stress response between the two groups, while getting the same sleep?

Strengths and Limitations

This study was the first to examine ethnicity as a potential moderator for the relationship between sleep and stress and is one of the few studies that focus on sleep in Mexican Americans. A strength of this study was the use of two physiological biomarkers to measure stress reactivity and recovery. This study used both salivary cortisol and heart rate to accurately capture stress response. The data for cortisol did not seem to significantly change across the three time points, but the data for heart rate nicely demonstrated the peak in stress and the recovery. Although this study was unable to find significant results for these measures when using a change score, other methods of analyses may yield different results.

The use of daily diaries can be seen as both a strength and limitation. Daily diaries strengthened the study because they record subjects' sleep patterns across several days and do not introduce errors of estimation related to retrospective recall as compared to a questionnaire that has participants recall their sleep in the past two weeks. Additionally, the present study was able to measure four types of sleep characteristics such as variability that is difficult to obtain using a single survey. Collecting these four measures broadened the scope of knowledge of the participants' overall sleep behaviors and patterns. However, they are still a subjective method of collecting sleep behaviors. The use of objective sleep measures (i.e. polysomnograms or actigraphy watches) is more reliable and yields more usable data.

There are also limitations to the study. First, weekdays and weekends were not tracked. Daily diaries were limited to include only one weekend within the ten days, but those days were not distinguished. This was done because weekends are usually used to recover from school and most often, to catch up on sleep. Previous studies found that on average, students sleep 1-2 hours more on the weekends, than they do on weekdays (Galambos et al., 2009; Lund et al., 2010). When examining the data, similar patterns were noted for all participants. On some days, which can only be assumed as weekdays, students were sleeping anywhere from 4-7 hours. However, on two days, sometimes three, patterns were noticed of these same participants sleeping anywhere from 7-12 hours. These sleep behaviors caused their average to go up, leaving each group with about seven to seven and a half hours of sleep.

Another limitation is the sample used. Although this study aimed to find ethnic difference in sleep and stress, these effects may have been missed due to focusing on college students. Sleep problems are very prominent in the general population, however, college students recognized as a population particularly affected by them. In fact, about 70% of college students report regular sleep difficulties (Buboltz, Brown, & Soper, 2001). These problems have often been associated with academic pressure, stress, and social experiences (Galambos et al., 2011). One study examined the effects of sleep deprivation on stress and found that in a sample of 22-50 year olds, sleep deprivation was linked to higher levels of stress when exposed to the TSST (Minkle et al., 2014). As this analysis focused on college students, a population known to experience the most sleep disturbances and stress, possible sleep differences between these two ethnicities may not have been captured.

A third limitation to the study was the timing of collection of salivary cortisol. The inconsistency in pattern between cortisol and heart rate leads us to believe that the window to capture the stress response for cortisol was missed. Because there was not a large difference between time 1 and time 2 samples, participants may not have had enough time to acclimate the testing room before starting the study. An extra 5-10 minutes could have allowed them to relax and provided a more accurate baseline. Additionally, there was no difference in cortisol levels from time 2 to time 3. These time points are measured 35 minutes apart, which should have been enough time for the participants to recover from the stressor. Given that time 2 and 3 cortisol levels were identical (M = .28), questions about whether the stressor may have been too stressful or the recovery period taking longer than expected arise.

Future Directions

Future studies should seek to extend the literature regarding sleep and stress in minorities and college students. Both of these populations are at risk for poor sleep and poor mental health. More sleep research with minorities will clarify any discrepancies in previous literature, and allow for more significant interpretation of sleep patterns across all ethnicities. Moreover, additional research on the sleep behaviors and patterns of college students can help elucidate what factors are contributing to or exacerbating sleep disturbance in this population. The hope for future studies is to examine these effects in non-college students in order to capture any ethnic differences that are unrelated to the stress and lack of sleep associated with school.

Secondly, examining sleep patterns and stress response using both objective and subjective measures will increase the validity of the study and leave less room for error. Both measures are needed in order to correlate the participants' physiological responses and behaviors to their perceived responses and behaviors. Future research should also consider cortisol timing. As seen here, participants needed more time to acclimate before taking saliva sample 1. Additionally, adding a forth saliva sample, or extending the timing of sample 3, could have allowed enough time for participants to recover from the stressor and return to baseline.

Lastly, future research should consider distinguishing between weekdays and weekends. Weekend oversleep reflects an attempt to recover accumulated sleep loss that is needed to maintain a sense of feeling rested (Jarrin, Mcgrath, Silverstein, & Drake, 2003). In order to fully examine sleep, weekdays and weekend should be examined separately. Weekend oversleep is considered an indicator of irregular sleep schedules, and "catching up" on sleep can be seen as indirect evidence of sleep deprivation (Jarrin et al., 2003).

Conclusion

The current study found that sleep variability and duration were negatively related to stress reactivity, whereas sleep quality and efficiency were unrelated. These findings are evidence of the importance of getting good sleep and can serve as important information used to educate the public about sleep health. This study was unable to find ethnicity as a moderator for the relationship between sleep and stress. However, the findings in this study suggest that stress reactivity in Mexican Americans is different from that of Caucasians. This study serves as preliminary evidence for possible differences in stress response across different ethnic minorities. While the present study was unable to find ethnic difference between sleep and stress, this may be an artifact of the sample.

It is important to note that sleep related health problems are important and must be recognized as an important public health concern. Obesity, hypertension, heart disease, depression, among others, are all associated with poor sleep and sleep disorders (Loredo et al., 2010). Latino health specifically, deserves significant concern as it continues to expand and affect millions of people. Currently, there are many racial and ethnic disparities as it pertains to health care and health status (Loredo et al., 2010). Seeing how small and inconsistent the literature is on sleep among Latinos not much is known about sleep and sleep related health concerns in this population. Critical information on sleep habits, attitudes toward sleep, knowledge about sleep, risk factors for sleep disorders, and objective sleep measures is lacking from this population (Loredo et al., 2010) and should be a focus of future work.

References

Åkerstedt, T., Orsini, N., Petersen, H., Axelsson, J., Lekander, M., & Kecklund, G. (2012). Predicting sleep quality from stress and prior sleep: A study of day-to-day covariation across six weeks. *Sleep Medicine, 13*(6), 674-679. doi: 10.1016/j.sleep.2011.12.013

Alapin, I., Fichten, C., Libman, E., Creti, L., Bailes, S., & Wright, J. (2000). How is good and poor sleep in older adults and college students related to daytime sleepiness, fatigue, and ability to concentrate? *Journal of Psychosomatic Research, 49*(5), 381-390. doi**:** 10.1016/S0022-3999(00)00194-X

Balbo, M., Leproult, R., & Van Cauter, E. (2010). Impact of sleep and its disturbances on hypothalamo-pituitary-adrenal axis activity. *International Journal of Endocrinology, 16*. doi**:** 10.1155/2010/759234

Beatty, D. L., Hall, M. H., Kamarck, T. A., Buysse, D. J., Owens, J. F., Reis, S. E., . . . Kazak, A.E. (2011). Unfair treatment is associated with poor sleep in African American and Caucasian adults: Pittsburgh SleepSCORE project. *Health Psychology, 30*(3), 351–359. doi:10.1037/a0022976

Bixler, E. O., Vgontzas, A. N., Lin, H. M., Vela-Bueno, A., & Kales, A. (2002). Insomnia in central Pennsylvania. *Journal of Psychosomatic Research, 53*(1), 589-592. doi**:** 10.1016/S0022-3999(02)00450-6

Braveman, P. A., Egerter, S. A., & Mockenhaupt, R. E. (2011). Broadening the focus: The need to address the social determinants of health. *American Journal of Preventive Medicine, 40,* S4–S18. doi:10.1016/ j.amepre.2010.10.002

Breslau, N., Roth, T., Rosenthal, L., & Andreski, P. (1996). Sleep disturbance and psychiatric disorders: A longitudinal epidemiological study of young adults.

(Clinical report). *Biological Psychiatry, 39*(6), 411-418. doi: 10.1016/0006-3223(95)00188-3

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

Byrne, D. G., Davenport, S. C. & Mazanov, J., (2007). Profiles of adolescent stress: The development of the adolescent stress questionnaire (ASQ). J*ournal of Adolescence*, *30*(3), 393-416. doi**:** 10.1016/j.adolescence.2006.04.004

Charmandari, E., Tsigos, C., & Chrousos, G. (2005). Endocrinology of the stress response. *Annual Review of Physiology, 67,* 259-284. doi:10.1146/annurev.physiol.67.040403.120816

Chrousos, G.P. & Gold, P.W. (1992). The concepts of stress and stress system disorders: Overview of physical and behavioral homeostasis. *JAMA, 267*(9), 1244-1252. doi**:** 10.1001/jama.1992.03480090092034.

Croissant, B., Demmel, R., Rist, F., & Olbrich. R. (2011). Heart rate stress response dampening: The impact of alcohol, family history, and gender on at risk children and siblings. *International Journal of Psychophysiology, 80*(1), 11-18. doi**:** 10.1016/j.ijpsycho.2011.01.009

Doane, L., Gress-Smith, D., & Breitenstein, J. (2015). Multi-method assessments of sleep over the transition to college and the associations with depression and anxiety symptoms. *Journal of Youth and Adolescence, 44*(2), 389-404. doi**:** 10.1007/s10964-014-0150-7

Drake, C., Pillai, V., & Roth, T. (2014). Stress and sleep reactivity: A prospective investigation of the stress-diathesis model of insomnia. *Sleep, 37*(8), 1295-1304. doi: 10.5665/sleep.3916

Anyan, F. & Hjemdal, O. (2016). Adolescent stress and symptoms of anxiety and depression: Resilience explains and differentiates the relationships. *Journal of Affective Disorders, 203*, 213-220. doi: 10.1016/j.jad.2016.05.031

Fuligni, A., & Hardway, C. (2006). Daily variation in adolescents' sleep, activities, and psychological well-being. *Journal of Research on Adolescence, 16*(3), 353-378. doi: 10.1111/j.1532-7795.2006.00498.x

Fuligni, A., Tsai, K., Krull, J.L., & Gonzales, N. (2015). Daily concordance between parent and adolescent sleep habits. *Journal of Adolescent Health*, *56*(2), 244-250. doi: 10.1016/j.jadohealth.2014.09.013

Galambos, N. L, Dalton, A., & Maggs, J. L (2009). Losing sleep over it: Daily variation in sleep duration and quality in Canadian students' first semester of university. *Journal of Research on Adolescence, 19*(4), 741-761. doi: 10.1111/j.1532-7795.2009.00618.x

Galambos, N. L., Howard, A. L., & Maggs, J. L. (2011). Rise and fall of sleep quantity and quality with student experiences across the first year of university. *Journal of Research on Adolescence*, *21(*2), 342-349. doi: 10.1111/j.1532-7795.2010.00679.x

Garde, A., Albertsen, K., Persson, R., Hansen, & Rugulies, R. (2012). Bi-directional associations between psychological arousal, cortisol, and sleep. *Behavioral Sleep Medicine, 10*(1), 28-40. doi: 10.1080/15402002.2012.636272

Hale, L., & Do, D. P. (2007). Racial differences in self-reports of sleep duration in a population-based study. *Sleep: Journal of Sleep and Sleep Disorders Research, 30*(9), 1096-1103.

Hamilton, N. A., Nelson, C. A., Stevens, N., & Kitzman, H. (2007). Sleep and psychological well-Being. *Social Indicators Research, 82*(1), 147-163. doi: 10.1007/s11205-006-9030-1

Hayes, A. F. (2012). PROCESS: A versatile computational tool for observed variable mediation, moderation, and conditional process modeling [White paper]. Retrieved from http://www.afhayes.com/public/process2012.pdf

Hellhammer, J., & Schubert, M. (2012). The physiological response to Trier Social Stress Test relates to subjective measures of stress during but not before or after the test. *Psychoneuroendocrinology, 37*(1), 119-124. doi**:** 10.1016/j.psyneuen.2011.05.012

Jarrin, D., Mcgrath, J., Silverstein, J., & Drake, C. (2013). Objective and subjective socioeconomic gradients exist for sleep quality, sleep latency, sleep duration, weekend oversleep, and daytime sleepiness in adults. *Behavioral Sleep Medicine, 11*(2), 144-158. doi: 10.1080/15402002.2011.636112

Jean-Louis, G., Kripke, D. F., Ancoli-Israel, S., Klauber, M. R., & Sepulveda, R. S. (2000). Sleep duration, illumination, and activity patterns in a population sample: Effects of gender and ethnicity. *Biological Psychiatry, 47*(10), 921-927. doi: 10.1016/S00063223(99)00169-9

Kachikis, A.B. & Breitkopf, C.R. (2012). Predictors of sleep characteristics among women in southeast Texas. *Women's Health Issues, 22*(1), e99-e109. doi:10.1016/j.whi.2011.07.004

Kashani, M., Eliasson, A., & Vernalis, M. (2012). Perceived stress correlates with disturbed sleep: A link connecting stress and cardiovascular disease. *Stress*, *15*(1), 45-51. doi: 10.3109/10253890.2011.578266

Keller, A., Litzelman, K., Wisk, L. E., Maddox, T., Cheng, E. R., Creswell, P. D., & Witt, W. P. (2012). Does the perception that stress affects health matter? The association with health and mortality. *Health Psychology, 31*(5), 677-684. doi**:** 10.1037/a0026743

Kelly, W. E. (2003). Some correlates of sleep disturbance ascribed to worry. *Individual differences research, 1*(2), 137-146. doi: 10.1080/00221320209598685

Kirschbaum, C., Pirke, K. M. & Hellhammer, D.H., 1993. The ‘Trier Social Stress Test’: A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, *28*(1–2), 76–81. doi**:** 10.1159/000119004

Korten, N., Sliwinski, M., Comijs, H., & Smyth, J. (2014). Mediators of the relationship between life events and memory functioning in a community sample of adults. *Applied Cognitive Psychology*, *28*(5), 626-633. doi: 10.1002/acp.3043

Knutson, K., Van Cauter, E., Rathouz, P., Deleire, T., & Lauderdale, D. (2010). Trends in the prevalence of short sleepers in the USA: 1975-2006. *Sleep, 33*(1), 37-45.

Krueger, P. M. & Friedman, E. M. (2009). Sleep duration in the United States: A cross-sectional population based study. *American Journal of Epidemiology, 169*(9), 1052-1063. doi: 10.1093/aje/kwp023

Laurent, H., Gilliam, K., Wright, D., & Fisher, P. (2015). Child anxiety symptoms related to longitudinal cortisol trajectories and acute stress responses: Evidence of developmental stress sensitization. *Journal of Abnormal Psychology, 124*(1), 68- 79. doi**:** 10.1037/abn0000009

Lantz, P., House, J., Mero, R., & Williams, D. (2005). Stress, life events, and socioeconomic disparities in health: Results from the Americans' changing lives study. *Journal of Health and Social Behavior, 46*(3), 274-288. doi**:** 10.1177/002214650504600305

Loredo, J., Soler, X., Bardwell, W., Ancoli-Israel, S., Dimsdale, J., & Palinkas, L. (2010). Sleep health in U.S. Hispanic population. *Sleep, 33*(7), 962-967.

Lovibond, S. H & Lovibond, Peter F & Psychology Foundation of Australia (1995). Manual for the depression anxiety stress scales (2nd ed). Psychology Foundation of Australia, Sydney, N.S.W

Lund, H., Reider, B., Whiting, A. B., & Prichard, R. J. (2010). Sleep patterns and predictors of disturbed sleep in a large population of college students. *Journal of Adolescent Health, 46*(2), 124-132. doi: 10.1016/j.jadohealth.2009.06.016

Maruyama, Y., Kawano, A., Okamoto, S., Ando, T., Ishitobi, Y., Tanaka, Y., . . . Akiyoshi, J. (2012). Differences in salivary alpha-amylase and cortisol responsiveness following exposure to electrical stimulation versus the Trier Social Stress Tests. *PLos ONE*, *7*(7), E39375. doi:10.1371/journal.pone.0039375.g001

Mena, F. J., Padilla, A. M., & Maldonado, M. (1987). Acculturative stress and specific coping strategies among immigrant and later generation college students. *Hispanic Journal of Behavioral Sciences*, *9*, 207–225.

doi: 10.1177/07399863870092006

McEwen, B. (2000). The neurobiology of stress: From serendipity to clinical relevance. *Brain Research, 886*(1), 172-189. doi: 10.1016/S0006-8993(00)02950- 4

McEwen, B. (2006). Protective and damaging effects of stress mediators: Central role of the brain. *Dialogues in Clinical Neuroscience, 8*(4), 367-81.

Minkel, J., Moreta, M., Muto, J., Htaik, O., Jones, C., Basner, M., . . . Kazak, Anne E. (2014). Sleep deprivation potentiates HPA axis stress reactivity in healthy adults. *Health Psychology, 33*(11), 1430-1434. doi: 10.1037/a0034219

Mirowsky, J., & Ross, C. (1980). Minority status, ethnic culture, and distress: A comparison of blacks, whites, Mexicans, and Mexican Americans. *American Journal of Sociology, 86*(3), 479-495. doi**:** 10.1086/227277

Morin, C., Bélanger, L., Leblanc, M., Ivers, H., Savard, J., Espie, C., . . . Grégoire, J. (2009). The natural history of insomnia: A population-based 3-year longitudinal study. *Archives of Internal Medicine, 169*(5), 447-453. doi 10.1001/archinternmed.2008.610

Mrug, S., Tyson, A., Turan, B., & Granger, D.A. (2016). Sleep problems predict cortisol reactivity to stress in urban adolescents. *Physiology & Behavior, 155*, 95-101. doi: 10.1016/j.physbeh.2015.12.003

National Sleep Foundation. (2006). Summary of findings: 2006 sleep in America poll. Retrieved from http://sleepfoundation.org/sites/default/files/2006_summary_of_findings.pdf.

Patel, S. R., Ayas, N. T., Malhotra, M. R., White, D. P., Schernhammer, E. S., Speizer, F. E., Stampfer M. J., & Hu, F. B. (2004). A prospective study of sleep duration and mortality risk in women. *Sleep: Journal of Sleep and Sleep Disorders Research, 27*(3), 440-444.

Patel, S. R., Malhotra, A., Gottlieb, D., White, D. P, & Hu, F. (2006). Correlates of long sleep duration. *Sleep, 29*(7), 881-889.

Pedraza, S., Al Snih, S., Ottenbacher, K., Markides, K., & Raji, M. A. (2011). Sleep quality and sleep problems in Mexican Americans aged 75 and older. *Aging Clinical and Experimental Research, 24*(4), 391-397. doi: 10.3275/8106

Pilcher, J.J., Ginter, D.R., & Sadowsky, B. (1997). Sleep quality versus sleep duration: Relationships between sleep and measures of health, well-being and sleepiness in college students. *Journal of Psychosomatic Research, 42*(6), 583-596. doi: 10.1016/S0022-3999(97)00004-4

Roberts, R. E., Roberts, C. R., & Chen, I. G. (2001). Functioning of adolescents with symptoms of disturbed sleep. *Journal of Youth and Adolescence, 30*(1), 1-18. doi: 10.1023/A:1005230820074

Roberts, R. E, Roberts, C. R, & Chan, W. (2006). Ethnic differences in symptoms of insomnia among adolescents. *Sleep, 29*(3), 359-365.

Ryff, C., Huppert, F., Baylis, N., Keverne, B., Singer, B., & Dienberg Love, G. (2004). Positive health: Connecting wellbeing with biology. *Philosophical Transactions of the Royal Society B: Biological Sciences, 359*(1449), 1383-1394. doi: 10.1098/rstb.2004.1521

Schlotz, W., Yim, I. S., Zoccola, P. M., Jansen, L., & Schulz, P. (2011). The perceived stress reactivity scale: Measurement invariance, stability, and validity in three countries. *Psychological Assessment, 23*(1), 80-94. doi-10.1037/a002114.

Scott, S. B., Graham-Engeland, J. E., Engeland, C. G., Smyth, J. M., Almeida, D. M., Katz, M. J., . . . Sliwinski, M. J. (2015). The effects of stress on cognitive aging, physiology and emotion (ESCAPE) project. *BMC Psychiatry, 15*, 146. doi: 10.1186/s12888-015-0497-7

Sladek, M., & Doane, R. (2015). Daily diary reports of social connection, objective sleep, and the cortisol awakening response during adolescents' first year of college. *Journal of Youth and Adolescence, 44*(2), 298-316. doi: 10.1007/s10964-014-0244-2

Sliwinski, M. J., Smyth, J. M., Hofer, S. M., & Stawski, R. S. (2006). Intraindividual coupling of daily stress and cognition. *Psychology and Aging, 21*(3), 545-557. doi:10.1037/0882-7974.21.3.545

Taylor, S., Seeman, T., Eisenberger, N., Kozanian, T., Moore, A., Moons, W., & Wesley, G. (2010). Effects of a supportive or an unsupportive audience on biological and psychological responses to stress. *Journal of Personality and Social Psychology, 98*(1), 47-56. doi**:** 10.1037/a0016563

Tsai, L. L., & Li, S. P. (2004). Sleep patterns in college students: Gender and grade differences. *Journal of Psychosomatic Research*, *56*(2), 231-237. doi: 10.1016/S0022-3999(03)00507-5

Wiemers, U. S., Schoofs, D. & Wolf, O.T. (2013) A friendly version of the Trier Social Stress Test does not activate the HPA axis in healthy men and women. *Stress, 16*(2), 254-260. doi**:** 10.3109/10253890.2012.714427

Williams, D. R., Yu, Y., Jackson, J., & Anderson, N. B. (1997). Racial differences in physical and mental health: Socio-economic status, stress, and discrimination. *Journal of Health Psychology, 2,* 335–351. doi: 10.1177/135910539700200305

Winzeler, K., Voellmin, A., Schäfer, V., Meyer, A. H., Cajochen, C., Wilhelm, F. H., & Bader, K. (2014). Daily stress, pressleep arousal, and sleep in healthy young women: A daily life computerized sleep diary and actigraphy study. *Sleep Medicine, 15*(3), 359-366. doi: 10.1016/j.sleep.2013.09.027

Yim, L., Quas, J. A., Rush, E. B., Granger, D. A., & Skoluda, N. (2015). Experimental manipulation of the Trier Social Stress Test-Modified (TSST-M) to vary arousal across development. *Psychoneuroendocrinology, 57*, 61-71. doi: 10.1016/j.psyneuen.2015.03.021

Yip, T. (2015). The effects of ethnic/racial discrimination and sleep quality on depressive symptoms and self-esteem trajectories among diverse adolescents. *Journal of Youth and Adolescence, 44*(2), 419-430. doi:10.1007/s10964-014-0123-x

Zohar, D., Tzischinsky, O., Epstein, R., & Lavie, P. (2005). The effects of sleep loss on medical residents' emotional reactions to work events: A cognitive-energy model. *Sleep, 28*(1), 47-54.

Table 1. *Mean saliva samples from participants at times 1, 2, and 3 for pilot study.*

Participant ID	Saliva Sample 1	Saliva Sample 2	Saliva Sample 3
4010	.044	.059	.132
4020	.099	.384	.81
4030	.467	.188	.277
4040	.089	.164	.055
4050	.019	.03	.027
4060	.115	.215	.002
4070	.097	.109	.122
4090	.276	.216	.222
4100	.145	.112	.538
4110	.235	.221	.216
4120	.092	.231	.357
4130	.005	.091	.14
4140	.042	.04	.048
4160	.215	.127	.175
4170	.159	.318	.865
4180	.153	.295	.789

Table 2. *Multiple Linear Regressions Predicting Stress Response with Salivary Cortisol.*

Predictor Variables	β	*T-test*	F	R^2
Mean Sleep Duration	-.28*	-2.2	3.5	.12
Mean Sleep Quality	.05	.50		
Mean Sleep Efficiency	.17	1.4		
Mean Sleep Variability	-.32*	-2.5		

Note: β represents the standardized regression coefficients, * $p < .05$, ** $p < .001$.

Table 3. *Multiple Linear Regressions Predicting Stress Recovery with Salivary Cortisol*

Predictor Variables	β	*T-test*	F	R^2
Mean Sleep Duration	-.09	-.68	1.2	.05
Mean Sleep Quality	-.13	-1.3		
Mean Sleep Efficiency	-.10	-.73		
Mean Sleep Variability	-.20	-1.6		

Note: β represents the unstandardized regression coefficients, * $p < .05$, ** $p < .001$.

Table 4. *Multiple Linear Regressions Predicting Stress Response with Heart Rate*

Predictor Variables	β	*T-test*	*F*	R^2
Mean Sleep Duration	-.001	-.004	.73	.03
Mean Sleep Quality	.10	1.0		
Mean Sleep Efficiency	-.16	-1.3		
Mean Sleep Variability	-.09	-.75		

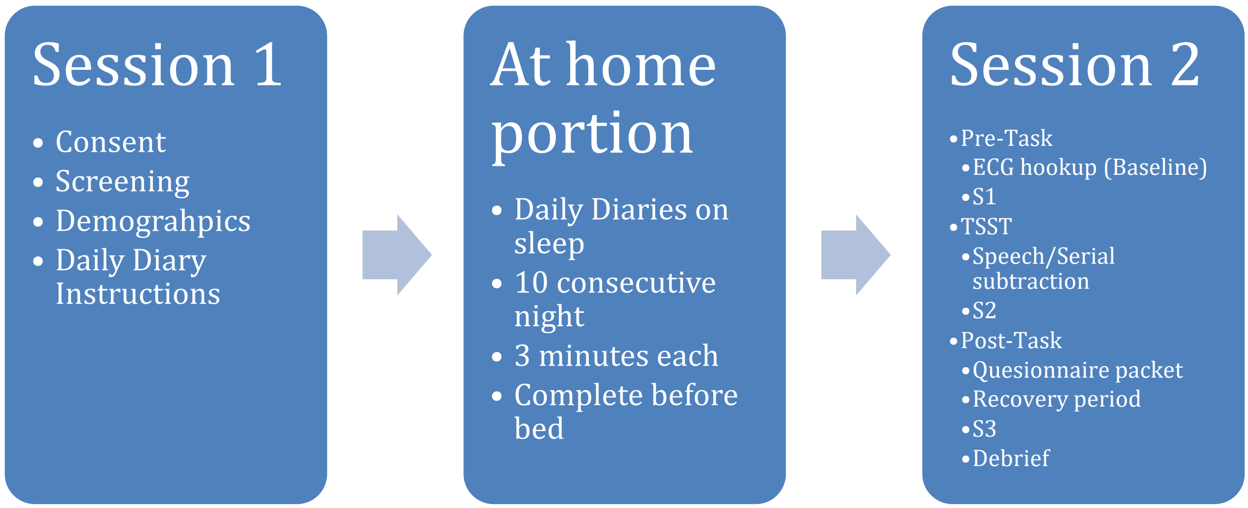
Note: β represents the unstandardized regression coefficients, * p < .05, ** p < .001.

Table 5. *Multiple Linear Regressions Predicting Stress Recovery with Heart Rate*

Predictor Variables	β	*T-test*	*F*	R^2
Mean Sleep Duration	-.10	-.82	.50	.02
Mean Sleep Quality	-.07	-.69		
Mean Sleep Efficiency	.10	.83		
Mean Sleep Variability	-.04	-.34		

Note: β represents the unstandardized regression coefficients, * p < .05, ** p < .001.

Figure 2. The Study Flowchart



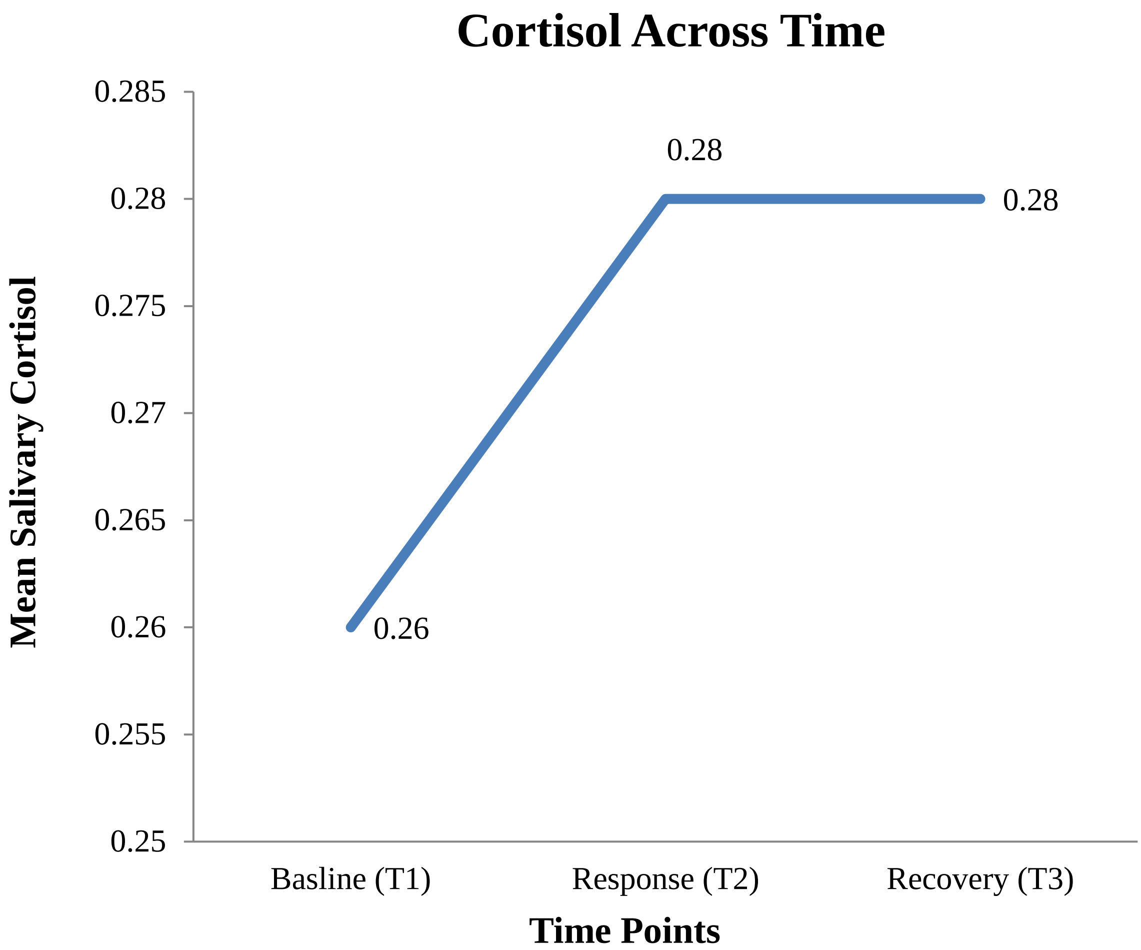


Figure 3. Mean salivary cortisol during baseline, stress response, and stress recovery.

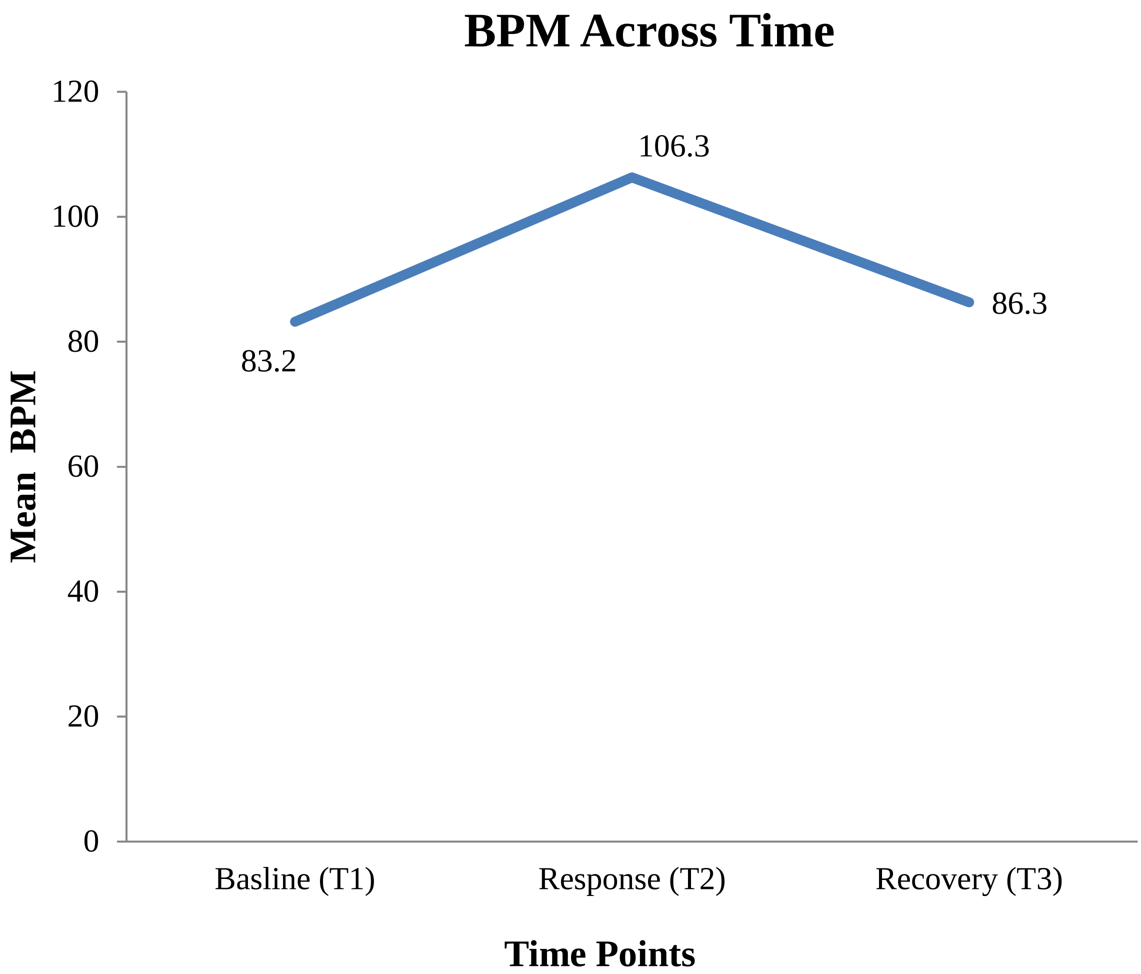


Figure 4. Mean heart beats per minute (BPM) during baseline, stress response, and stress recovery.

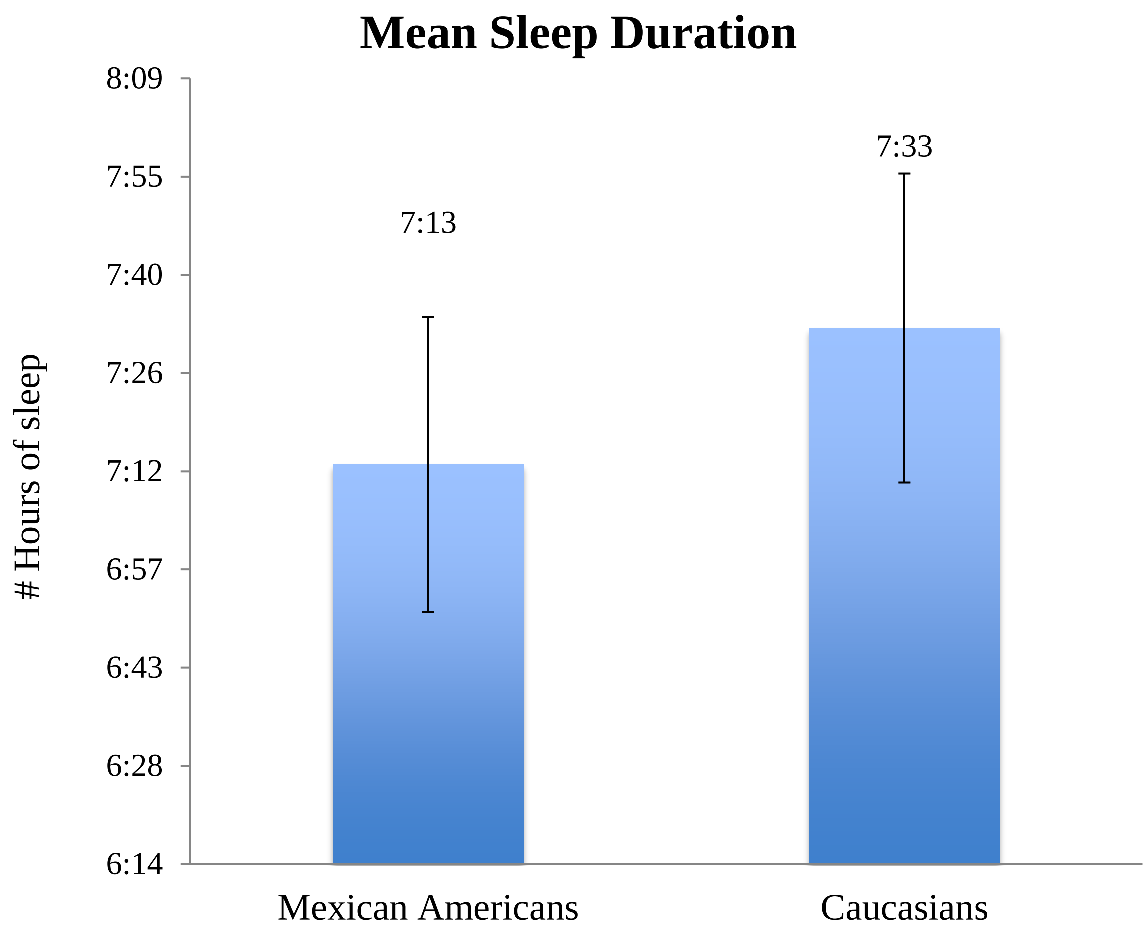


Figure 5. Mean sleep duration (in hours) reported by Mexican American and Caucasian college students.

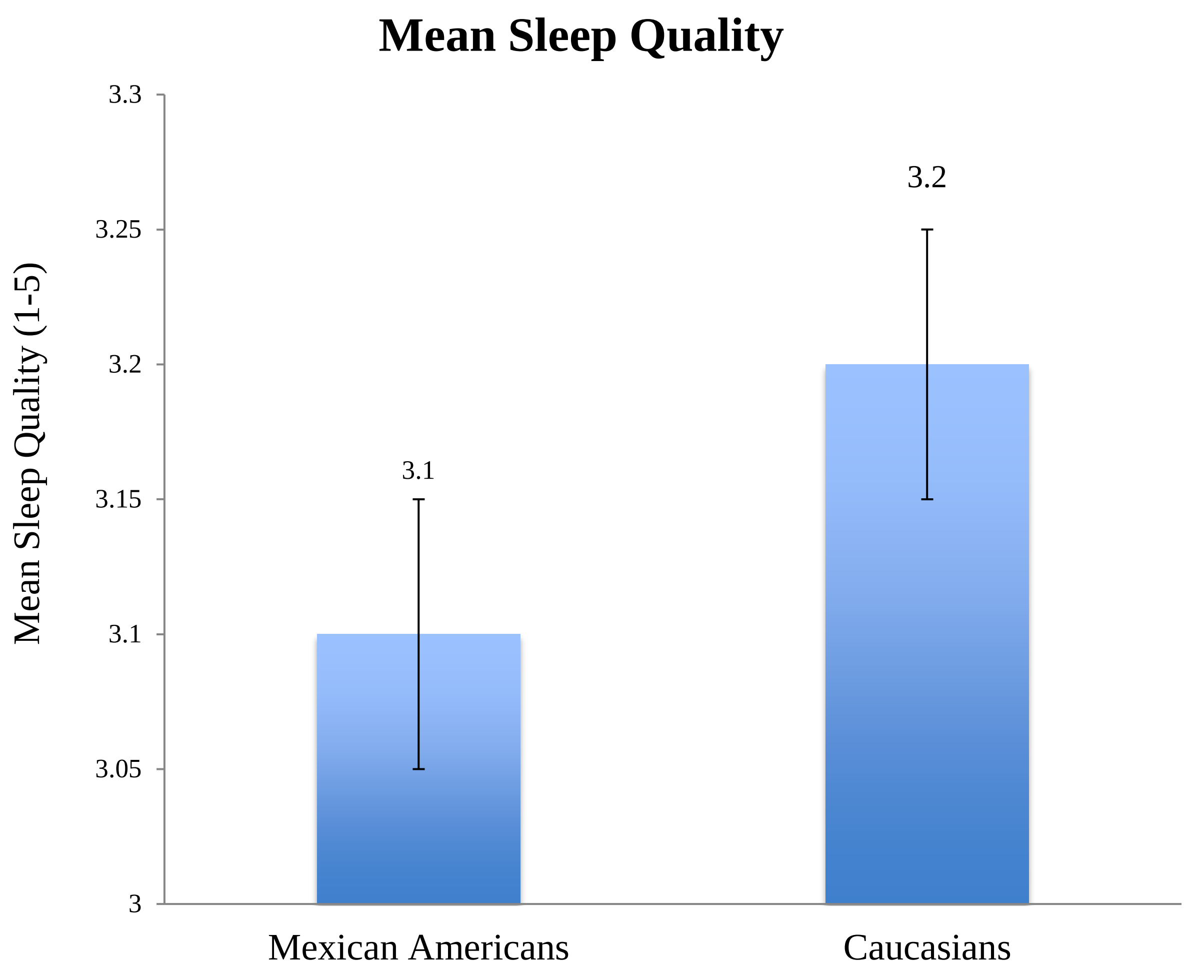


Figure 6. Mean sleep quality (on a Likert scale form 1-5, with 1 being poor quality) reported by Mexican American and Caucasian college students.

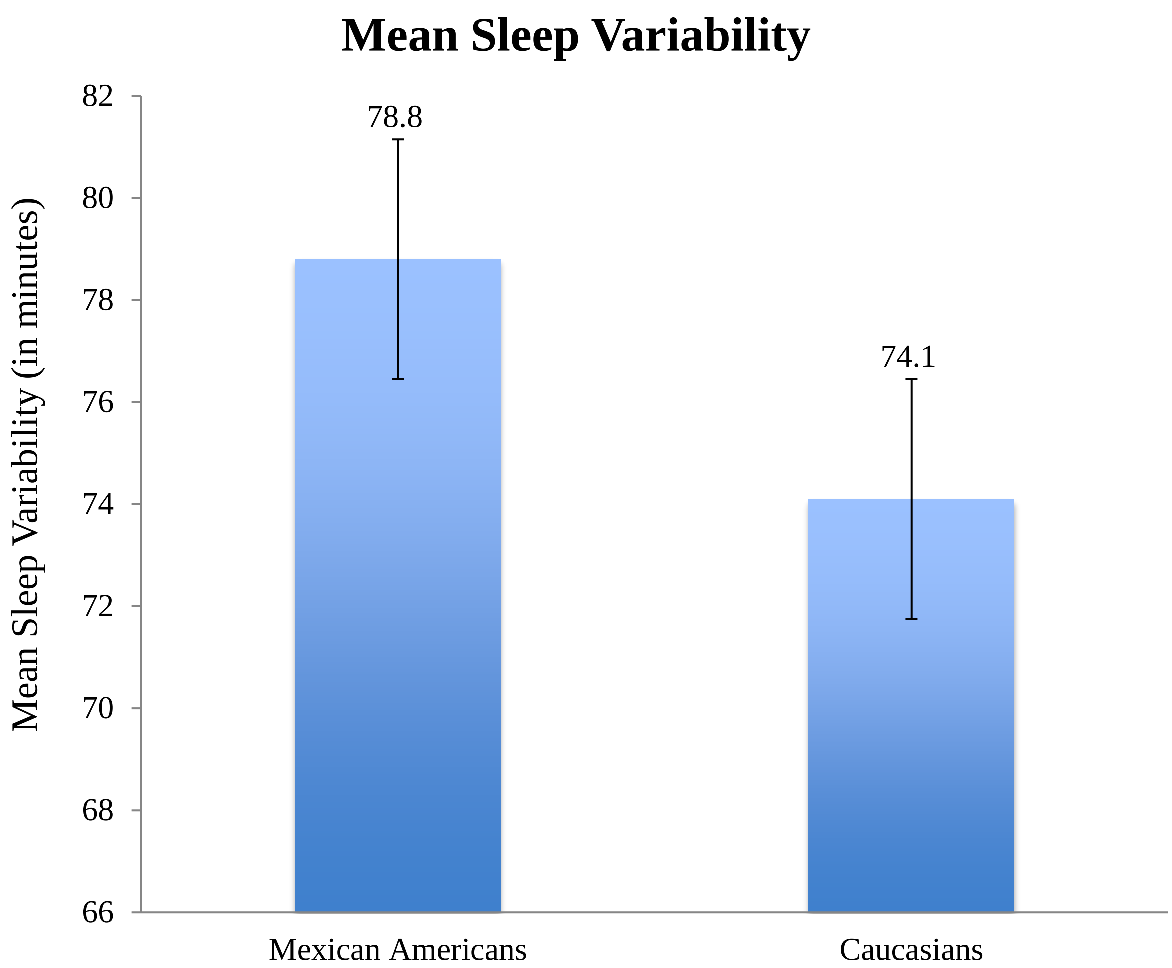


Figure 7. Mean sleep variability (in minutes) reported by Mexican American and Caucasian college students.

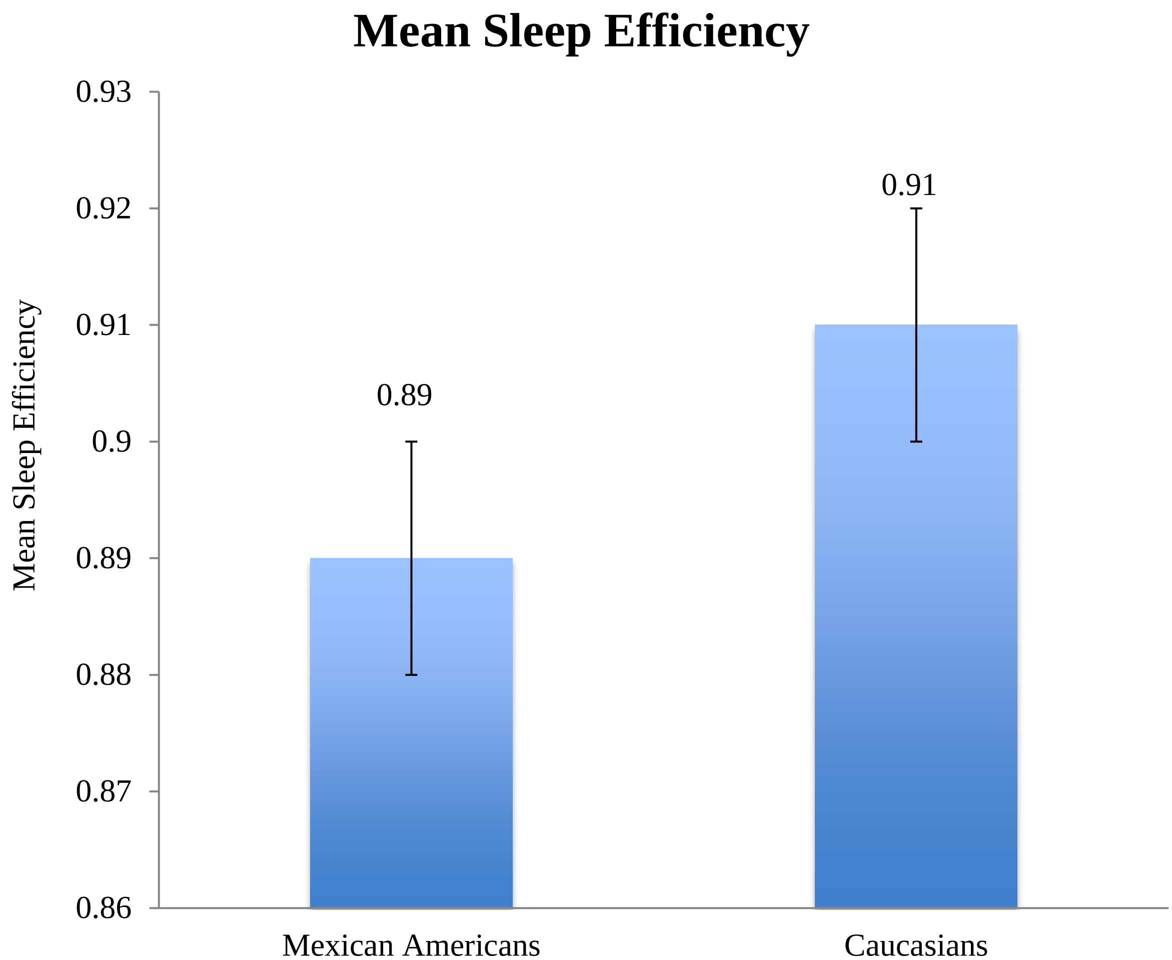


Figure 8. Mean sleep efficiency reported by Mexican American and Caucasian college students.

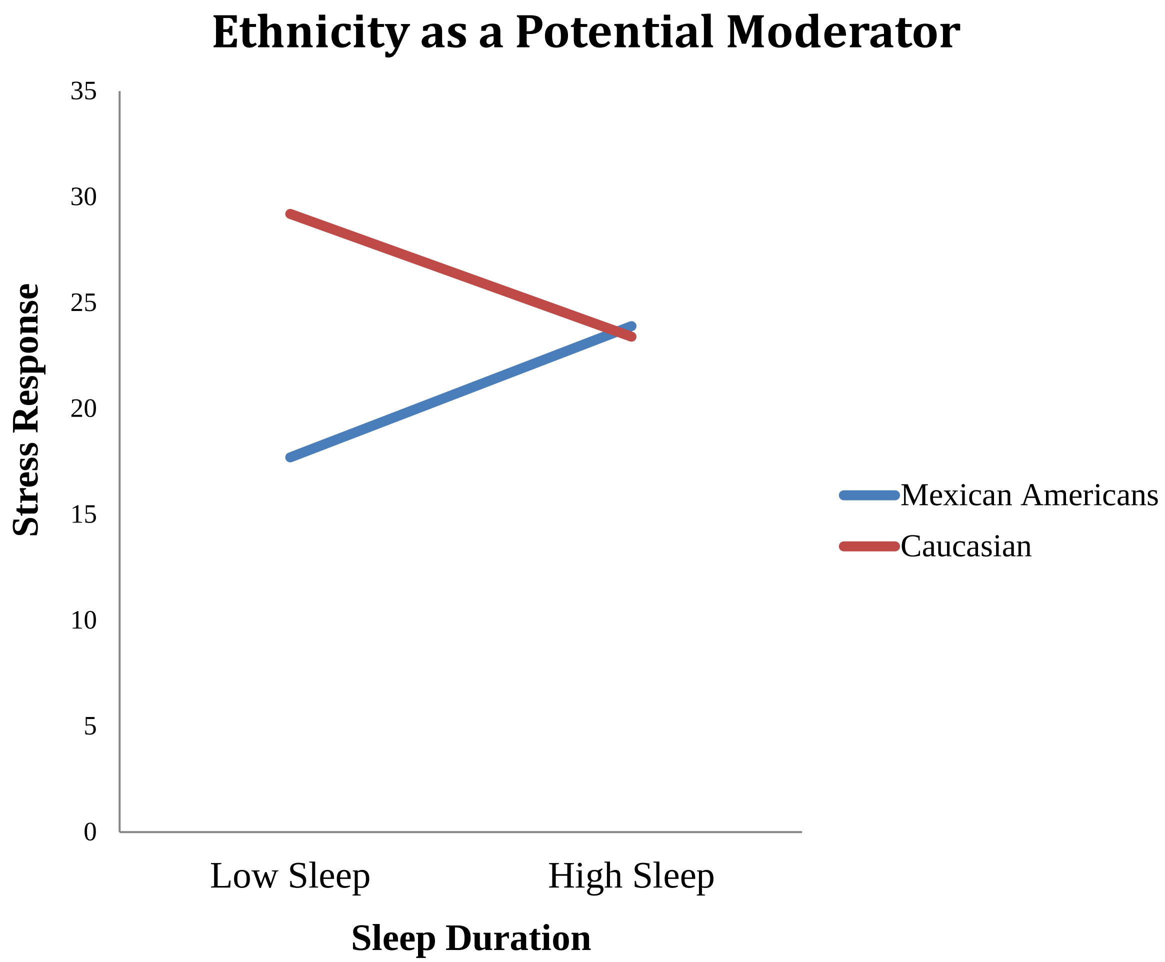


Figure 9. Ethnicity as a moderator for the relationship between sleep duration and stress response, as measured by heart rate.

Appendix A

Daily Diaries

1) Participant ID

The following questions refer to LAST NIGHT:

2) What time did you turn off the lights to go to sleep last night? _________AM PM

3) How long did it take you to fall asleep after turning the lights off last night? ____________

(In minutes)

4) Did you wake up during the night?

1- Yes
2- No

5) How many times did you wake up last night?

6) When you woke up last night, how long were you awake in total? _____________Minutes

7) What time did you wake up and no longer sleep this morning? ___________ AM PM

8) What time did you get out of bed this morning? _____________________ AM PM

9) How difficult was it for you to get out of bed this morning?

1- Not Difficult
2- Slightly Difficult
3- Somewhat Difficult
4- Moderately Difficult
5- Difficult

10) How would you rate the quality of your sleep?

1- Very poor
2- Poor
3- Fair
4- Good
5- Very good

11) How rested or refreshed did you feel when you woke-up this morning?

1- Not at all rested
2- Slightly rested
3- Somewhat rested
4- Well-rested
5- Very well rested

The following questions refer to TODAY:

12) Did you take a nap today?

1- Yes
2- No

13) How long was the nap today (in minutes)?

14) Did you take a prescription medication prescribed to you today?

1- Yes
2- No

15) What kind of prescription medicine did you take?

16) Did you have a caffeinated drink(s)? (e.g., coffee, tea, soda, energy drinks)

17) How many servings of caffeinated drink did you consume today? (Counting a serving as a cup, a bottle, or a can of caffeinated drink).

How many servings did you have before 6pm? _____________servings

How many servings did you have after 6pm? _____________servings

18) Did you drink any alcohol today?

1- Yes
2- No

19) How many drinks containing alcohol did you have yesterday? Consider a “drink” to be a can or bottle of beer, a glass of wine, a wine cooler, or one cocktail or shot of hard liquor (like Scotch, Gin, or Vodka)

20) Did you use/consume a cannabis/marijuana product (e.g., raw plant material, edible cannabis, and concentrate) today?

1- Yes
2- No

21) How many times did you use the cannabis/marijuana product today?

22) Did you use a prescription medication that was NOT prescribed to you today? (e.g., Adderall, Vicodin)

1- Yes
2- No

23) What kind of prescription medication NOT prescribed to you was taken yesterday?

24) Did you take over-the-counter or non-prescription medication for sleep today?

1- Yes
2- No

25) Which over-the-counter or non-prescription medication for sleep did you take today?

Note: Only questions 1-11 were used for the purpose of this study.

Appendix B

Screening for Health Behaviors and Performance Study

Instructions. Please read each question aloud to participants and mark the appropriate answer.

1. Do you classify your ethnic background as Latino? _____Yes _____ No
2. Do you classify your ethnic background as Caucasian? ______ Yes _____ No
3. What generational status are you?

_____1st generation (you immigrated to the US)

_____2nd generation (your parents immigrated to the US)

_____post 2nd generation (your parents were born in the US)

4. Do you use tobacco on a daily basis? _____Yes _____No
5. Do any of the following characterize you? Please wait until I finish naming off options to respond.

_____Yes _____No

   a. I am currently pregnant.
   b. I am currently breast-feeding
   c. I have breast-fed in the past 6 months.

6. Do you have a severe fear of public speaking?

_____Yes _____No

6a. Do you go out of your way to avoid public speaking?

_____Yes _____No

7. Do you have a medical history of any of the following? Please wait until I finish naming off options to respond.

_____Yes _____No

a. Endocrine disorder
b. History of stroke or other neurological disorder likely to affect cognition
c. Diagnosed anxiety, depression, insomnia or post-traumatic stress disorder
d. Hospitalization for a psychiatric condition in the past year
e. Autoimmune, blood, or metabolic disease
f. Any form of cancer
g. Coronary artery disease, significant arrhythmia, uncontrolled hypertension, or any other cardiovascular condition
h. Insulin-dependent diabetes
i. Serious allergies or asthma
j. Use of beta-blockers, inhaled B-agonists, oral or parenteral corticosteroids within the last 3 months
k. Use of psychotropic medication within the past 8 weeks
l. Or any other major health or mental health conditions
m. Previous severe reaction to gel or creams applied to the skin