EXPLORING PERPETUATING FACTORS BY WHICH PERCEIVED STRESS CONTRIBUTES TO POOR SLEEP QUALITY: THE ROLE OF PRE-SLEEP AROUSAL, BEHAVIORS, AND COGNITIVE PROCESSES

by

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ABSTRACT

CHRISTINE E. SMITH MASON. Exploring perpetuating factors by which perceived stress contributes to poor sleep quality: The role of pre-sleep arousal, behaviors, and cognitive processes. (Under the direction of DR. VIRGINIA GIL-RIVAS)

Young adults are the fasting growing but least understood at-risk group reporting poor sleep. Stress and developmental processes related to the transition to college may negatively alter sleep quality and sleep patterns that continue into adulthood and contribute to poor health outcomes. The current project tested a conceptual model of associations among perpetuating factors hypothesized to influence the relationship between stress and poor sleep. Self-reported measures of perceived stress, sleep quality, cognitive-affective states, and pre-sleep cognitive, somatic and behavioral processes were administered at two time points, baseline (P1) and 30 days later (P2). A total of 142 non-clinical, college-aged young adults completed the measures at both time points. Results demonstrated that a majority of participants reported poor-quality sleep at baseline and thirty days later. Age, female gender, and employment were significantly associated with poor sleep quality at P2. Perceived stress had a significant direct and indirect effect on sleep quality at P2 through pre-sleep arousal. Emotion dysregulation, ruminative thinking, sleep-related worry, and poor sleep hygiene were associated with higher levels of pre-sleep arousal. Beliefs and attitudes about sleep did not contribute to self-reported sleep arousal. Contrary to expectations, the relationship between perceived stress and pre-sleep arousal was not moderated by maladaptive beliefs about sleep. Furthermore, sleep-related worry and emotion dysregulation did not moderate the mediational effects of pre-sleep arousal on poor sleep quality. These results parallel findings from studies exploring associations among individuals with clinical insomnia diagnoses, as well as those

including objective measures of sleep. These findings may be used to inform the development of brief interventions to prevent and treat poor sleep in college-aged young adults.

DEDICATION

This dissertation is dedicated to my husband, *Alexander John Mason, III,* and to my parents, *Stephen Harold Smith* and *Martha Ann Hackl Smith.*

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LIST OF ABBREVIATIONS

APA	American Psychological Association
BAS	Beliefs and Attitudes about Sleep Scale
BSM	Behavioral Sleep Medicine
CBT-I	Cognitive Behavioral Treatment for Insomnia
CDC	Centers for Disease Control and Prevention
CI	Confidence Interval
CNS	Central Nervous System
CPAP	Continuous Positive Airway Pressure
DERS	Difficulties with Emotion Regulation Scale
DV	Dependent Variable
ECG	Electrocardiogram
ED	Emotion Dysregulation
EEG	Electroencephalogram
EMG	Electromyography
HPA	Hypothalamic-Pituitary-Adrenal
IV	Independent Variable
LLCI	Lower Limit Confidence Interval
MBSR	Mindfulness-Based Stress Reduction
NSF	National Sleep Foundation
OSA	Obstructive Sleep Apnea
P1	Part 1
P2	Part 2

PC-PTSD	Primary Care Post-Traumatic Stress Disorder Screen
PNS	Parasympathetic Nervous System
PSA	Pre-Sleep Arousal
PSAS	Pre-Sleep Arousal Scale
PSG	Polysomnography
PSQI	Pittsburg Sleep Quality Index
PSR	Perceived Stress Reactivity
PSRS	Perceived Stress Reactivity Scale
PSS	Perceived Stress Scale
PTSD	Post-Traumatic Stress Disorder
REM	Rapid Eye Movement Sleep
RTS	Ruminative Thinking Style
SHI	Sleep Hygiene Index
SNS	Sympathetic Nervous System
SOL	Sleep Onset Latency
SRW	Sleep-Related Worry
ULCI	Upper Limit Confidence Interval
UNCC	University of North Carolina at Charlotte

CHAPTER ONE: INTRODUCTION

1.1 The problem of poor sleep

The Centers for Disease Control and Prevention (CDC, 2012) have identified poor sleep as a public health epidemic. Poor sleep is characterized by symptoms such as difficulty falling asleep, frequently waking after sleep onset, waking too early without being able to fall back to sleep, and/or having feelings of lethargy or distress following a night of insufficient sleep duration (CDC, 2012). The National Sleep Foundation (NSF, 2014) reports that each month 58% of adults complain of at least one of these symptoms and that 28% report getting fewer than six hours of sleep on at least 15 out of every 30 days. In total, an estimated 40 million adults in the United States experience chronic poor sleep; an additional 20 million report intermittent sleep difficulties (NSF, 2014). It is estimated that approximately one in 10 adults in the United States suffers from chronic sleep difficulties in which these symptoms persist for three or more months, and higher rates occur among women, older adults, and medical and psychiatric populations (NSF, 2014).

The prevalence of sleep difficulties increases with age, though young adults are the fastest growing group of individuals reporting poor sleep quality (American Psychological Association [APA], 2015), placing them at risk for adverse health outcomes. Ironically, this age group is the least studied and understood in sleep intervention and health outcome research (Lund, Reider, Whiting, & Prichard, 2010), perhaps due to lack of clinical presentation in primary care facilities and sleep disorders clinics where treatment for poor sleep is most likely to occur. Instead, a majority of research on college-aged young adults has been observational, with the goal of exploring

sleep patterns in relation to their contribution to fatigue, daytime sleepiness, and educational performance (Oginska & Pokorski, 2006; Tsai & Li, 2004), which are common complaints among college-aged young adults.

Lund and colleagues (2010) sought to move beyond studies examining the prevalence of sleeping difficulties among college students and aimed to identify predictors and exacerbating factors of poor sleep in this population. They found that 60% of college students were classified as poor-quality sleepers and that they were significantly more likely to report a greater number of physical and mental health complaints than their good-quality sleeper counterparts (Lund et al., 2010). Recent studies have also revealed that mental health complaints including depressive symptoms and anxiety episodes are more severe and frequent for students with sleep difficulties than compared to those without sleep difficulties (Gress-Smith et al., 2015; Nyer et al., 2013). College students with poorer sleep also report increased use of alcohol and drug use, often as means to self-medicate for poor sleep, putting these young adults at increased risk for substance abuse and dependence (Brown, Buboltz, & Soper, 2010; Taylor & Bramoweth, 2010).

Researchers have identified a pattern that sleep difficulties in college students appear to start before (Cheng et al., 2012) and worsen soon after the transition to college. What is more, these difficulties have also been found to worsen over time during their college years, highlighting the vulnerability of this group to the progression of sleep difficulties from acute to chronic (Milojevich & Lukowski, 2016). Additionally, in a large sample size of 1,039 college students, only 57.1% were considered “normal sleepers”, in which individuals had no complaints of poor sleep, and objective criteria for

chronic insomnia were not met (Taylor, Bramoweth, Grieser, Tatum, & Roane, 2013). The remaining 42.9% of students either met the Diagnostic and Statistical Manual of Psychiatric Disorders, Fifth Edition criteria (APA, 2013) for chronic insomnia (9.5%), complained of difficulty sleeping but did not meet full criteria for chronic insomnia (6.5%), or met the diagnostic criteria for chronic insomnia for frequency, severity, and duration without complaint of poor sleep (26.9%). Almost half of those assessed students were identified as at risk for poor sleep, with nearly 27% unaware of their poor sleep patterns (Taylor et al., 2013). These statistics are alarming, as they are significantly higher than those reported for the general population of adults at risk for poor sleep. Furthermore, Taylor and colleagues (2013) put forth the concerning assertion that college students may not even recognize that they have insomnia or insomnia symptoms; rather, they may assume that their poor sleep is a normal experience. Hence, early identification, prevention, and intervention efforts are likely to be critical for young adults as a means to delay or negate the detrimental effects of poor sleep over time.

1.2 Health implications of poor sleep

Lack of sufficient sleep has been associated with deficits in normal cognitive, emotional, behavioral, and physical functioning. Indeed, poor sleep in young adults is related to decreased attention, poorer concentration, excessive emotional arousal, impaired interpersonal functioning, and increased negative emotional reactivity (Buboltz, Brown, & Soper, 2001; Sawyer & Weaver, 2010; Vandekerckhove & Cluydts, 2010). An estimated 67% of young adults have expressed subjective concerns over lack of sleep as they relate to successful daytime cognitive and physical functioning (Gradisar et al., 2013). These alterations contribute to increased daytime sleepiness (Alapin et al., 2000),

increased motor vehicle accidents (Taylor & Bramoweth, 2010), and decreased school performance (Thacher, 2008).

Regarding decreased school performance, the American College Health Association (2007) has ranked sleep as a leading significant health issue related to impaired academic performance in a large population sample of 80,000 college students. In fact, Trockel, Barnes, and Egget (2000) found that sleep patterns predicted academic performance above and beyond all other health-related behaviors. What is more, findings from another large-scale sample of nearly 2,000 participants found that nearly 27% of undergraduate students were at risk for sleep disorders (Gaultney, 2010). This risk significantly predicted an objective grade point average less than 2.0 out of a 4.0 scale. These findings suggested that students at greater risk of sleep disorders are also at greater risk of academic failure (Gaultney, 2010) and consequent retention, dropout, or transfer to another institution (O'Connell, 2014). This relationship was particularly significant among females, African American and White students compared to males and participants of other racial backgrounds (Gaultney, 2010). Thus, lack of sleep should be interpreted as a primary concern for universities.

The trend of sleep-related impairment continues beyond college into the adult workforce. Daytime sleepiness, sleep-related errors resulting in accident or injury, and decreased overall job performance make lack of sleep also costly for employers (Kessler et al., 2011). Work productivity losses amount to 11.3 missed days of work and $2,280 each year for each employed person reporting sleeping difficulties (Institute of Medicine, 2006; Kessler et al., 2011; Rosekind et al., 2010). Nationwide, sleep-related impairment results in $63.2 billion in economic losses annually (Kessler et al., 2011).

Regarding behavioral and physical functioning, lack of sufficient sleep has been associated with maladaptive health practices including smoking, alcohol use, and physical inactivity, among all ages (CDC, 2012; Schoenborn & Adams, 2008). These associations are amplified for young adults in college, given lifestyle changes allowing for minimal adult supervision, increase in risk-taking activities, and easy access to tobacco products, alcohol, and recreational, prescription, and over-the-counter drugs (Lund et al., 2010). Furthermore, trends observed in college students have associated poor sleep with the onset and maintenance of obesity (Melton, Langdon, & McDaniel, 2013). One explanation for this may be that shorter sleep duration over time leads to impairment of the body's ability to use insulin, which suggests that poor sleep also contributes to changes in metabolic processes that result in excess body weight, increased body mass index, and subsequent obesity (Vargas, Flores, & Robles, 2014; Vgontzas et al., 2008).

Poor sleep has also been associated with the onset, management, and treatment outcomes of chronic physical and mental health conditions including severe symptoms associated with pain, rheumatoid and osteoarthritis, cardiovascular and pulmonary disease, diabetes mellitus, anxiety, and depression (Institute of Medicine, 2006; Kasebah, Chi, & Krishnaswamy, 2006; Zimmerman, McGlinchey, Young, & Chelminski, 2006). At present, chronic illness, obesity, and tobacco use rank as three of the more financially and physically costly public health challenges facing American citizens (CDC, 2012). Health Psychology as a discipline emphasizes that incremental risk factors over time contribute to the onset and development of later illness. Thus, attention to these problem areas in early adulthood will be crucial to enact change for these larger health

consequences more often associated with later life. Consequently, as a result of its high comorbidity with each of these problem areas, poor sleep in young adults is rapidly advancing as a significant public health issue in need of further exploration.

1.3 Overview of sleep

To understand how poor sleep influences health outcomes, it is first necessary to recognize the role of sleep as a physiological process. Described by Maslow (1943) as a fundamental human need, sleep is defined as "a reversible behavioral state of perceptual disengagement from and unresponsiveness to the environment" (Carskadon & Dement, 2000, p.15). Humans follow an intrinsic biological rhythm that lasts approximately 24 to 25 hours, naturally progressing from wakefulness to sleep – a process known as circadian rhythm (Markov & Goldman, 2006). This wake-sleep cycle typically synchronizes with environmental cues, or "zeitgebers," such as light exposure and/or temperature, to follow Earth's 24-hour light-dark cycle (Aschoff, Hoffman, Pohl, & Wever, 1974).

Normal sleep architecture is characterized by a repeating four-stage cycle (Iber et al., 2007). Stages 1-3 are known as non-rapid eye movement sleep. Sleep progresses from shallow sleep in stages 1 and 2 to deep, slow-wave sleep in stage 3 when the restorative effects of sleep have been observed to take place (Harris, 2005). The final stage of sleep is referred to as rapid eye movement (REM) sleep. REM sleep is associated with dreaming and has been hypothesized to be responsible for learning and memory consolidation (Siegel, 2001; Stickgold, 2005). Upon completion of REM sleep, the cycle progresses back into shallow sleep before returning to slow-wave sleep. Each complete sleep cycle lasts from one to two hours. Sleep disruption can occur at varying intervals throughout the four stages, though it is most likely to take place during superficial sleep

associated with stages 1 and 2 (Tassi & Muzet, 2000). Through the observation of how bodily systems function during sleep, researchers have been able to theorize as to the purpose and function of sleep as a physiological process.

Unlike other physiological processes in the body, sleep appears to be a unique process in that its origins remain disputed. Experts in the field of sleep research have yet to agree on the evolutionary purpose and function of sleep, though the history of the field indicates multiple theories have gained support over time. Krueger, Frank, Wisor, and Roy (2016) were recently funded to investigate various multidisciplinary perspectives regarding the purpose of sleep. Their results, summarized below, posit that six theories most accurately explain the function of sleep due to evolutionary value, substantial auxiliary evidence, and supporting co-occurring biochemical mechanisms.

Firstly, the authors suggest that sleep serves an immune function, with the purpose of enhancing the body's defenses against pathogens. Secondly, it is suggested that sleep functions to reduce caloric use, for those times historically and at present when sustenance was and is not readily available. Thirdly, sleep is theorized to restore brain energy stores, for brain-specific cellular metabolism occurring as neurons fire repeatedly during waking. Fourthly, sleep is unique to vertebrates; non-vertebrates and simple organisms have been observed to rest as opposed to sleep. Thus, sleep has been theorized to serve a glymphatic function, allowing for opportunity to clean waste created by the central nervous system, unique to vertebrates, during waking. Fifthly, sleep functions to restore performance degradations induced during wakefulness, allowing for post-work recovery of processes and organ systems. Lastly, it is suggested that sleep functions to support brain connectivity (Krueger et al., 2016).

Each of these independent theories highlights that sleep is an important mechanism in human health; however, of these six leading theories, Krueger and colleagues (2016) suggest that the brain connectivity theory is the leading theory for the elemental function of sleep. Further, considering the brain and the growth, expansion, and maintenance of its neuronal connections are critical for every aspect of functioning, processing, and growth within the body, it is then even more imperative that researchers pay attention to the epidemic of insufficient sleep threatening optimal connectivity during sleep and daytime functioning.

1.4 Sleep and health

Evidence for the theories, function, and health implications of poor sleep comes primarily from studies of sleep deprivation and restriction. Sleep first became a focused area of medical research interest through systematic laboratory demonstrations of the lethal effects of complete sleep deprivation in rats (Rechtschaffen & Bergmann, 1989). These studies demonstrated that sleep deprivation had significant deleterious mental and physical health consequences and that total sleep restriction could ultimately result in death. As sleep is believed to serve as a mechanism for homeostasis, it may be viewed as an active contributor to allostatic processes that restore the body to balance after natural exposure to stressful stimuli during waking (Sapolsky, 2004).

In order to investigate potential consequences associated with intermittent and chronic lack of sleep in human subjects, Pilcher and Huffcut (1996) conducted a meta-analysis that compared sleep-deprived subjects with non-sleep-deprived controls. Nineteen studies were included in the meta-analysis, and sleep deprivation categories were divided into three groups: partial deprivation (examining studies in which

participants received less than 5 hours of sleep per night over the course of several days), short-term deprivation (examining studies in which participants were assigned to less than 45 consecutive hours of deprivation), and long-term deprivation (examining studies in which participants were assigned to greater than 45 consecutive hours of sleep deprivation). All three deprivation conditions resulted in mood, motor ability, and cognitive functioning deficits when compared to control conditions not exposed to systematic sleep deprivation. Interestingly, partial deprivation conditions revealed the greatest negative effects (Pilcher & Huffcut, 1996), suggesting that intermittent periods of less sleep may have greater consequences for individuals than periods of longer, consecutive hours without sleep.

Similar outcomes have been noted when comparing groups of individuals with modest reductions in sleep over time (i.e., 6 hours compared with 8 hours per night; Van Dongen, Maislin, Mullington, & Dinges, 2003), and with incremental increases in deprivation in the consecutive number of hours restricted (i.e., 3 hours, 4 hours, 5 hours, etc.; Banks & Dinges, 2007). These results offer evidence that even minor reductions in daily total sleep time have implications for poorer sleep outcomes, and that even one day of minimal increase to the number of hours of restricted sleep may be more significantly related to cognitive, affective, and physical deficits in functioning.

As these studies on sleep deprivation and restriction have demonstrated, sleep has been traditionally measured and reported by examining hours of sleep duration during the course of a night's sleep. For adults, 6 to 8 hours of sleep has been the recommended sleep duration per night (NSF, 2014). However, younger college-aged adults have a slightly higher self-reported ideal of 8.5 hours per night to accommodate an exit away

from a circadian rhythm shift that occurs earlier in adolescence during puberty (Taylor & Bramoweth, 2010).

Recent attention has shifted away from this emphasis on sleep duration to a more global focus on sleep efficiency, patterns of falling asleep and waking, daytime functioning, and individual appraisal of sleep patterns (Carney et al., 2012). These aspects of sleep may be more strongly related to health outcomes; they comprise facets of a complex construct referred to as sleep quality (Krystal & Edinger, 2008). Multiple lifestyle and environmental factors are likely to influence sleep quality, with stress being a reliable anecdotal and evidence-based contributor.

1.5 Stress and poor sleep

Stress (Selye, 1956) is a term used to characterize the complex emotional, cognitive, and biological effects of an event or process that threatens homeostasis. It is known that sleep is a homeostatic process that occurs during times of non-wakefulness; therefore, it is important to deduce that stress affects the psychological and physiological mechanisms that contribute to and alter the process of sleep (McEwen, 1998). Despite repeated associations between high stress and poor sleep quality, the relationship between poor sleep quality and stress has yet to be fully explained (Orzel-Gryglewska, 2010).

What is known suggests that when an event is psychologically perceived by an individual as acutely stressful or threatening, the body responds with the immediate introduction of physiological changes through activation of the hypothalamic-pituitary-adrenal (HPA) axis (Fries, Dettenborn, & Kirschbaum, 2009; Sapolsky, 2004). This action activates nervous system, endocrine, respiratory, metabolic, and other organ systems within the body to convert all resources to energy to function, respond to, and

relieve the perceived experience of stress. This biological response temporarily disrupts regulatory processes of normal functioning, including sleep, through physiological activation of the stress response (Sapolsky, 2004).

Under conditions of chronic stress, the return to pre-stress homeostatic balance does not occur. Instead, the parasympathetic nervous system (PNS) response is slow to initiate and insufficient to regain balance. The sympathetic nervous system (SNS) remains activated, and energy stores in the brain in the form of glucose are actively converted for use during sleep to sustain the ongoing SNS response. This consequently interferes with cognitive processing, memory consolidation, and normal physiological functioning (Akerstedt & Nilsson, 2003; Sapolsky, 2004), which is likely to decrease the recuperative nature of sleep in both the short- and the long-term. Over time, this leads to dysregulation of the endocrine, metabolic, and immune systems that are evidenced by physiological indicators of chemical and cellular functioning (Sapolsky, 2004), thus allowing for increased risk for disease susceptibility and mortality (Danese & McEwen, 2012; Orzel-Gryglewska, 2010). Therefore, the detrimental physiological responses brought about by perceived stress over time make it crucial to further examine the mechanisms by which perceived stress influences poor sleep quality. With greater theoretical knowledge, individuals can protect from these risks posed by acute and chronic perceived stress as they influence poor sleep quality, and consequent health. Such mechanisms may be influenced by learned coping, or protective, cognitive, emotional, and behavioral processes that help individuals manage stress (Schneiderman, Ironson, & Siegel, 2005).

1.6 Spielman's 3-P model of insomnia

A diathesis-stress model for the development of poor sleep offers a framework that captures the critical role of stress and coping processes in the onset and maintenance of sleep difficulties. One such model, Spielman's 3P model of insomnia, proposes that underlying predispositional vulnerabilities may allow for precipitating stressors that naturally arise in life experiences to produce disease, in this case poor sleep (Spielman, Caruso, & Glovinsky, 1987). Such vulnerabilities, or predisposing factors, can be genetic, biological, psychological, or situational (Ingram & Luxton, 2005). The model suggests that predisposing factors may make an individual more likely to experience acute sleep difficulties under conditions of a precipitating event (e.g., stress); sleep difficulties are then exacerbated and maintained over time by perpetuating maladaptive behavioral and cognitive patterns, which are implemented to alleviate stress but adversely contribute to the perpetuation of chronic sleep difficulties.

Spielman's 3P model is used primarily to guide clinical treatment for insomnia, which is a sleep disorder defined as "the symptom[s] of difficulty falling asleep, repeated awakenings with difficulty returning to sleep, [waking too early without being able to fall back to sleep], or sleep that is nonrestorative or poor in quality" (Buysse, Germain, Hall, Monk, & Nofzinger, 2011, p. 129). Known predisposing factors for chronic insomnia include female gender, anxiety, neuroticism, and psychobiological hyperarousal (e.g., extreme physiological cortical arousal in response to psychological trauma) (LeBlanc et al., 2009). These predisposing factors have been mostly identified in observational, population research exploring relationships in clinical insomnia treatment in adults, without due attention to how these factors influence poor sleep specifically under

conditions of stress and to how these factors relate to young adults with sleep difficulties who do not meet the criteria for insomnia disorders.

Spielman's 3P model also suggests that perpetuating factors, or maladaptive thought and behavior patterns that individuals engage in to alleviate stress and improve sleep during times of stress, contribute to the maintenance of sleep difficulties even after the stressor has been resolved. Thus, increased attention to risk and perpetuating factors of poor sleep, such as pre-sleep behaviors, and cognitive processes, may help us understand the modifiable mechanisms in the relationship between perceived stress and sleep quality specifically among young adults (Espie, 2002; Morin, 1993; Spielman, Caruso, & Glovinsky, 1987).

Cognitive Behavioral Therapy for Insomnia (CBT-I; Bootzin & Nicassio, 1978; Harvey, 2001; Morin, 1993; Spielman, Saskin, & Thorpy, 1987), based on the processes put forth by Spielman's 3P model, is the leading treatment for complaints of poor sleep in sleep clinics where Behavioral Sleep Medicine (BSM) is practiced. CBT-I targets predisposing, precipitating, and perpetuating processes involved in the relationship between stress and poor sleep quality. College-age individuals are largely remiss from the literature on sleep disorders, as they do not regularly present for insomnia treatment where BSM is offered; thus, this model may benefit as a guide for the exploration into of the nature of and mechanisms of associations influencing poor sleep quality in this typically non-clinical group of young adults.

1.7 Perceived stress reactivity

Following Spielman's 3P model, it is important to explore additional predisposing and perpetuating factors that may specifically contribute to the development and

continuation of poor sleep quality in this young adult group. One potential predisposing factor that has to date remained separate from the literature on poor sleep quality is perceived stress reactivity (PSR; Schulz, Jansen, & Schlotz, 2005). A more recently identified and related construct to perceived stress, PSR suggests individuals may have a tendency towards increased psychological and physiological responses across specific stressful situations; different individuals are likely to perceive different scenarios as uniquely stressful. PSR is also an underexplored area of research on stress, for researchers have only recently developed a measure to capture the nature of this construct and differentiate it from overall stress perceptions (Schultz et al., 2011). Specifically, different from the state-like nature of perceived stress, PSR is a dispositional, trait-like characteristic in which individuals inherently differ on how they response to individual stressors in both immediate and long-term reactions (Limm et al., 2010). Studies exploring measurement invariance have confirmed independence of constructs between PSR and perceived stress (Schlotz, Yim, Zoccola, Jansen, & Schulz, 2011).

Six specific domains of PSR have been identified – work overload, social conflicts, social stress, failure, anticipation, and prolonged reactivity – suggesting that individuals may react more intensely and differently to each of these domains of stressors as opposed to perceiving and reacting similarly to different stressors (Schlotz et al., 2011). PSR has been found to be significantly associated with sleep difficulties (Mezick et al., 2009); however, PSR has yet to be used within a theoretical framework that explores the relationship between stress and poor sleep. Furthermore, exploration into which specific domains of PSR that may have greater influence on poor sleep quality has yet to occur.

Evidence for age-related differences in domains of psychobiological stress

reactivity suggests that younger adults may react more strongly than middle and older adults to stressful events in domains related to interpersonal and work-related performance (Neupert, Almeida, & Charles, 2007; Schlotz, Hammerfald, Ehlert, & Gaab, 2011). Due to this biological response to psychological stressors, college students may also perceive greater stress and experience greater psychological stress reactivity in life domains that carry greater meaning or in which they have little prior experience in their age group (Rith-Najarian, McLaughlin, Sheridan, & Nock, 2014). In sum, PSR as a construct may help researchers better understand the unique role of perceived stress as it contributes to poor sleep quality specifically among college students (Schlotz, Hammerfald, Ehlert, & Gaab, 2011).

1.8 Pre-sleep arousal

Though not frequently targeted in cognitive behavioral interventions, pre-sleep arousal (PSA; Nicassio, Medlowitz, Fussell, & Petras, 1985) is an important factor in the relationship between perceived stress and sleep quality. PSA encompasses the combination of somatic and cognitive arousal an individual generally experiences during attempts to fall asleep in the evening (i.e., being mentally alert, racing thoughts, muscle tenseness, rapid heart rate; Nicassio et al., 1995). High PSA is commonly observed in individuals with complaints of poor sleep, which suggests that individuals who report difficulty sleeping may be more likely to experience higher general levels of somatic and cognitive arousal when attempting to fall asleep (Ong, Shapiro, & Manber, 2008).

Furthermore, observational studies on stress and sleep have demonstrated that increased stress in the time prior to sleep contributes to difficulties falling asleep, an increased number of awakenings after sleep initiation, and premature awakenings from

sleep (Fries, Dettenborn, & Kirschbaum, 2009). Fries and colleagues (2009) have hypothesized this relationship may occur as a result of elevations in hormones, such as cortisol, released during events perceived as stressful during waking that remain in the body and are activated as the body calms in preparation for sleep, and thus interferes with sleep. This is thought to occur because perceived stress triggers the activation of the SNS resulting in physiological and cognitive arousal, which in turn contribute to poor sleep (Charles et al., 2011). PSA captures the evening somatic and cognitive elevations of arousal that occur after perceiving stress during the day, which, in turn, contributes to difficulties with sleep onset latency (SOL) and influences sleep quality. Thus, we view PSA as a mediator of the relationship between perceived stress and sleep quality.

1.9 Perpetuating cognitive and behavioral processes

Sleep theorists have long considered cognitive and behavioral processes as important contributors to poor sleep (Morin, Stone, Trinkle, Mercer, & Remsberg, 1993; Spielman, Saskin, & Thorpy, 1987). When these patterns are maladaptive, they are considered to be perpetuating factors of poor sleep, as they contribute to further difficulties with falling and/or staying asleep. Poor sleep hygiene behaviors (Spielman, Saskin, & Thorpy, 1987) and maladaptive cognitions such as dysfunctional beliefs and attitudes about sleep (Morin et al., 1993) contribute to the maintenance of poor sleep quality, as individuals alter thoughts, behavior, and hygiene in attempts to cope or "make up" for lost or broken sleep after the precipitation of stress. Cognitive processes regarding beliefs and attitudes related to sleep may impact individuals' sleep-related behaviors responsible for poor sleep. For example, Morin and colleagues (1993) found that in a

sample of older adults, dysfunctional beliefs and attitudes toward sleep were associated with feelings of helplessness related to making successful improvements in sleep quality.

Morin and colleagues (1997) also identified sleep-related behaviors that perpetuate poor sleep by contributing to PSA and increased arousal during awakenings from sleep, which interfere with an individual's ability to fall back asleep. Such sleep hygiene behaviors have since been found to be strongly associated with poor sleep quality (Mastin, Bryson, & Corwyn, 2006; Morin et al., 1993; Spielman, Saskin, & Thorpy, 1987). Specifically, Spielman's 3P model of insomnia suggests, that among other behaviors, behaviors such as staying in bed longer than planned, using the bed for activities other than sleep, and varying bedtimes and rise times contribute to poor sleep quality through the perpetuation of poor sleep habits that interfere with healthy sleep behavioral practices. Furthermore, lifestyle changes common for college-aged young adults such as increasing caffeine, alcohol, and substance use (Gellis, Stotsky, & Taylor, 2014), are likely to contribute to shifts in a regular sleep schedule; thus, these maladaptive behaviors may be more pronounced in this age group.

Maladaptive sleep-related beliefs and behaviors are prevalent and modifiable. What is less explored in sleep literature and not included in traditional models of poor sleep which focus primarily on sleep-related constructs is attention to general psychological cognitive processes such as emotion dysregulation (ED; Gross, 1998) and ruminative thinking style (RTS; Brinker & Dozois, 2009). Though not specific to sleep difficulties, these processes are likely to be important, as they more generally capture the psychological processes of how individuals respond to and cope with perceived stress during the day.

RTS captures an individual's tendency to over-think or ruminate on general events (Brinker & Dozois, 2009). This tendency for enhanced focus on certain experienced or imagined events has been found to be related to higher levels of perceived stress, as well as activation of the SNS. For example, prior to sleep, rumination over school and work-related and personal plans and situations has been associated with sleep-interference in young adults (Querstret & Cropley, 2012), perhaps as a result of increasing pre-sleep arousal.

Additionally, ED strategies employed by individuals may also influence the degree to which perceived stress interferes with poor sleep quality. Specifically, ED refers to an individual's inability to regulate his or her emotional responses when exposed to provocative stimuli in the environment (Thompson, 1994) and describes the regulatory, cognitive processes individuals employ to voluntarily modify emotional experience and behavior (Gross, 2013). Indeed, a strong link between perceived stress and the inability to manage negative emotions has been established. Specifically, under conditions of increased perceived stress, individuals are less likely to manage successfully negative emotions than under conditions of less perceived stress (Prakash, Hussain, & Schirda, 2015). Moreover, individuals under chronic stress are likely to have lower ED reserves than when experiencing acute stress (Sliwinski & Scott, 2016).

Difficulties with ED in response to stress are likely to contribute to greater cognitive and physiological activation as a result of poor control over the experience and expression of one's emotions (Gross, Richards, & John, 2006), which may contribute to poorer sleep quality through increasing PSA (Babson & Feldner, 2015). This may be especially true for young adults, as the plastic, neurological connections of the brain

associated with the ability to regulate and interpret emotions continue to develop well into adulthood (Johnson, Blum, & Giedd, 2009).

Poor ED is directly linked to poor sleep quality (Markarian, Pickett, Deveson, & Kanona, 2010); as one prepares for sleep, emotion centers of the brain are heightened, suggesting that under conditions of stress, increased activation of these somatic and cognitive PSA areas contributes to difficulty initiating and maintaining sleep. Thus, under conditions of greater perceived stress, ED is likely to moderate the relationship between perceived stress and PSA. We additionally believe that difficulties with ED are likely to later interact with PSA to influence poorer sleep quality through the inability to regulate emotions and ruminations over poor sleep.

One specific ED process that has not been adequately explored, though commonly reported in individuals with sleep difficulties (specifically insomnia), is sleep-related worry (SRW; Sunnhed & Jansson-Fröjmark, 2014). SRW, defined as repetitive, negative concerns about not being able to fall or stay asleep, has been shown to contribute to poor sleep quality (Sunnhed & Jansson-Fröjmark, 2014). Recently, researchers have proposed SRW as an important factor influencing the relationship between perceived stress and poor sleep quality, for it captures the nature of pre-sleep repetitive thoughts about the causes, consequences, and symptoms of not being able to sleep (Smith & Alloy, 2009).

Further, Carney, Harris, Moss, and Edinger (2010) identified SRW as a separate construct from RTS as it relates to insomnia, and their recommendations suggest that interventions to treat poor sleep quality would benefit from targeting these two constructs independently. It is likely that tendencies to engage in SRW play a role after PSA has already been elevated. As individuals experience difficulties with falling asleep and

staying asleep, more generally, pre-sleep RTS tendencies shift away from rumination over general events and situations and instead focus on the daytime and health consequences of poor sleep. Worry over difficulty sleeping contributes to increased cognitive arousal, which interacts with physiological activation occurring as a result of stress; it has been hypothesized to be "key factor in the relationship between repetitive thought and [poor] sleep quality" (Babson & Feldner, 2015, p. 279). In the contribution of SRW to poor sleep in young adults, however, it may be likely that SRW will yield similar positive relationships with poor sleep quality as have been found in clinical samples of middle-aged adults, with a sample of participants with a mean age of 50 years (Sunnhed & Jansson-Fröjmark, 2014).

Thus, it is important to include ED, RTS, and SRW in models of the relationship between stress and poor sleep quality, as ED, generalized ruminative thoughts and worries regarding poor sleep have all been found to respond well to psychological interventions that aim to combat maladaptive cognitions (Kloss, Nash, Horsey, & Taylor, 2010). For example, studies on interventions for anxiety have demonstrated that cognitive behavior therapy for anxiety focused on decreasing worry and improving emotion regulation strategies is associated with reductions in both ED and rumination (Mennin, Heimberg, Turk, & Fresco, 2002). Generalizing these findings to the sleep literature, one can imagine that addressing RTS, SRW, and ED is likely to contribute to improvements in sleep quality via their interactive relationship with perceived stress. In sum, exploring the roles of these three cognitive processes on the relationships between perceived stress and poor sleep quality has the potential for informing cognitive-behavioral sleep

interventions such that perpetuating factors can be targeted in interventions to decrease stress and improve sleep quality.

1.10 The current study

Researchers recognize the contribution of sleep quality to the development and management of health conditions. Current treatments to improve sleep quality involve a combination of pharmacological and nonpharmacological interventions. Unfortunately, sleep medications affect areas of the brain involved in central nervous system (CNS) activation (e.g., GABA receptors) and circadian rhythm (e.g., melatonin). The available evidence suggests that these medications have short-term effects, are not the treatment of choice for chronic sleep problems, and can result in medication dependency (NSF, 2014). Nonpharmacological interventions frequently target behavioral (e.g., sleep hygiene, sleep consolidation) and cognitive processes that contribute to sleep problems (e.g., dysfunctional beliefs about sleep). In young adults, educational programs to increase sleep hygiene have been significantly helpful when compared to groups with no education; however, outcomes do not target the cognitive components that also contribute to increased pre-sleep arousal (Kloss et al., 2016).

Additionally, effectiveness studies on CBT-I and Mindfulness-Based Stress Reduction (MBSR) have demonstrated considerable success in affordably improving sleep problems (Andersen et al., 2013; Sharma & Andrade, 2012). Importantly, these interventions include stress reduction components, supporting the hypothesized relationship between perceived stress and sleep. However, targeting stress and modifications to behavioral and cognitive processes to improve sleep quality yields mixed findings, with CBT-I success rates of only up to 60-70% (Edinger, 2016).

Despite high rates of poor sleep due to environmental, lifestyles changes, and developmental tasks, college-aged young adults have been largely ignored in CBT-I and mindfulness-based advances in sleep research due to their absence in clinical sleep disorders centers (Taylor et al., 2014). It is important to note, however, that brief interventions to improve sleep quality in this young group have more recently been conducted to explore modifiable pre-sleep behaviors and cognitive processes. These outcomes have yielded mixed findings, perhaps because they are more focused on outcomes and not fully based on relationships among variables based in theory (Kloss et al., 2010; Lillehei, Hacon, Savik, & Reis, 2015).

In light of these recent advances in brief interventions, we still know relatively little about the mechanisms related to the onset and development of sleep difficulties in this age group. Mullan (2014) suggests that what is missing in research on stress and sleep is "the conduct of more theoretically driven research to determine the predictors of poor sleep (Mullan, 2014, p. 434; Mullan, Todd, Chatzisarantis, & Hagger, 2014). Consistent with this idea, Nuendorf and colleagues (2015) suggest the relationship between stress and sleep quality is more complex and in need of further exploration to optimize and generalize treatment to all individuals and groups. Commonly used models that guide clinical treatment for poor sleep, such as Spielman's 3-P model of insomnia, may also guide research examining factors that contribute to poor sleep quality among young adults.

Specifically, the present study aimed to explore the additional contribution of PSR to pre-sleep arousal and poor sleep quality. Though research suggests that PSR is a predisposing trait, similar to personality factors, there is some evidence suggesting that

individuals can be taught to change their responses to various perceived stressors through training (Morgan, Umberson, & Hertzog, 2014). Further, as Morgan and colleagues (2014) suggest, understanding stress perceptions in various life domains (e.g., failure, social conflict, social evaluation, prolonged stress, and work overload) can inform treatment efforts. Lastly, perpetuating behavioral (i.e., poor sleep hygiene behaviors) and cognitive processes (i.e., dysfunction beliefs about sleep, emotion dysregulation, ruminative thinking-style, and sleep related worry) may determine how individuals respond to, cope with, and manage stress.

Furthermore, Espie (2002) hypothesizes there are likely to be interactive effects of stress with perpetuating factors of poor sleep to influence acute and chronic sleep disturbances. To our knowledge, researchers have not yet examined the interactive role of perceived stress and perceived stress reactivity with perpetuating sleep-related behaviors (e.g., sleep hygiene behaviors) and cognitions (e.g., dysfunctional beliefs about sleep and SRW) and more general psychological cognitive processes (e.g., ED and RTS). More modifiable than personality, beliefs, attitudes, and behaviors related to sleep and sleep hygiene and state-like cognitive processes such as dysfunctional beliefs about sleep, ED strategies, RTS, and SRW may influence the degree to which perceived stress and perceived stress reactivity influence pre-sleep arousal and poor sleep quality (Babson & Feldner; 2015; Markarian et al., 2013).

Thus, the purpose of this study was to test an integrative, conceptual model to understand the relationships among perpetuating factors of poor sleep as they interact with self-reported stress (See Figure 1). Results from the study will contribute to the development of theory and the implementation of interventions to help young adults

better manage stress in various domains to improve sleep quality. The practice implications of this research are vast, from the screening of patients with sleep problems, to the selection of cognitive and emotion regulation processes targeted in current interventions. Answering the question of "for whom and under what conditions" interventions may be more or less effective is of relevance for efforts to improve sleep quality among young adults.

1.11 Research aim and hypotheses

Aim. To test a conceptual model (See Figure 1) of the mediating role of pre-sleep arousal (PSA) on the relationship between perceived stress, perceived stress reactivity (PSR), and poor sleep quality in a non-clinical sample of college adults. Further, several cognitive and behavioral factors were examined as potential moderators of these relationships. Specifically, we expected that:

Hypothesis 1. PSA would fully mediate the relationship between perceived stress, PSR, and poor sleep quality.

Hypothesis 2. Cognitive and behavioral factors related to sleep would moderate the relationship between perceived stress, PSR, and PSA, such that:

H2a) Individuals who reported more dysfunctional beliefs about sleep under conditions of greater perceived stress would be more likely to report higher levels of PSA;

H2b) Individuals who reported more dysfunctional beliefs about sleep under conditions of greater PSR would be more likely to report higher levels of PSA;

H2c) Individuals who reported poorer sleep hygiene behaviors under conditions of greater perceived stress would be more likely to report higher levels of PSA;

H2d) Individuals who reported poorer sleep hygiene behaviors under conditions of greater PSR would be more likely to report higher levels of PSA;

H2e) Individuals who reported higher levels of ruminative thinking style under conditions of greater perceived stress would be more likely to report increased PSA; and

H2f) Individuals who reported higher levels of ruminative thinking style under conditions of greater PSR would be more likely to report increased PSA;

H2g) Individuals with greater emotion dysregulation under conditions of greater perceived stress would be more likely to report increased PSA; and

H2h) Individuals with greater emotion dysregulation under conditions of greater perceived stress would be more likely to report increased PSA.

Hypothesis 3. Emotion dysregulation (ED) and sleep-related worry (SRW) would moderate the relationship between PSA and poor sleep quality.

H3a) ED would moderate the relationship between PSA and poor sleep quality, such that the mediation of PSA on poor sleep quality would be conditional on levels of ED; and

H3b) SRW would moderate the relationship between PSA and poor sleep quality, such that the mediation of PSA on poor sleep quality would be conditional on levels of SRW.

CHAPTER TWO: METHODOLOGY

2.1 Participants

A power analysis using G*Power 3 software (Faul, Erdfelder, Buchner, & Lang, 2007) for multiple regression with multiple predictors, an alpha of 0.05, and a power of 0.80 was conducted to calculate the minimum recommended sample size of 89 for the study. A total of one hundred forty-two undergraduate full- or part-time students at the University of North Carolina at Charlotte (UNC Charlotte) between the ages of 18 and 24 years completed baseline (P1) and follow-up questionnaires 30 days later (P2).

Exclusion criteria included a) pregnancy, b) high risk for having a diagnosis of obstructive sleep apnea (OSA), c) treatment for a sleep disorder within the past six months (e.g., Cognitive Behavior Therapy for Insomnia [CBT-I]), Continuous Positive Airway Pressure [CPAP]), d) regular reported use of sedative-hypnotic medications or mood-altering medications for depression or anxiety, or e) diagnosis of a significant psychiatric condition that may interfere with normal sleep (e.g., bipolar disorder, schizophrenia, acute stress disorder, post-traumatic stress disorder [PTSD]).

2.2 Procedure

2.2.1 Recruitment

Participants were recruited through the use of an online system (SONA) used by the Department of Psychological Science to offer course credit for participation in research studies to students enrolled in undergraduate courses at UNC Charlotte. Paper fliers advertising the study were posted throughout the campus (e.g., student health center, library, cafeteria, gymnasium, etc.), and electronic fliers were distributed through online recruitment listservs for undergraduate research and the Department of Psychological

Science. The measurement schedule and measures utilized in the study are described below.

2.2.2 Original measurement schedule

At the commencement of data collection, interested students were originally required to complete a brief consent and 7-minute online eligibility screen; eligible students were invited to participate in the study. Instructions informed participants that they consented for researchers to contact them via email regarding eligibility and subsequent assessments. Eligible participants were then invited to complete the two-part study as described in the revised measurement schedule below. Those who were not eligible were informed that with completion of screening questions they had consented for responses to be kept confidential for use in secondary analyses.

Initially, a high response rate between screening, study participation, and completion was expected due to feasibility of completion and the provision of course credit with an additional opportunity for monetary reward. We anticipated that to reach the target sample size of 200 as is recommended for structural equation modeling analyses (Boomsma, 1982,1985; Wolf et al., 2013), fewer than 270 students would need to be screened for eligibility with full participation of Part 1 (P1) and Part 2 (P2) of the study. However, this was an underestimation.

From January 2017 to May 2017, only 96 students participated in the eligibility screen. Of these 96 students, 71 (74.0%) were eligible to participate in P1. Of the 71 eligible, only 40 (56.3%) participated in P1 of the study. Of the 40 who consented to participate at P1, only 25 (62.5%) completed P2 for full study participation. At this rate,

data collection would have required approximately two full years to reach the targeted sample size of 89.

2.2.3 Revised measurement schedule

Permissions to revise the study measurement and incentive schedule were unanimously granted through the Doctoral Dissertation Committee, the UNC Charlotte Department of Psychological Science, and the UNC Charlotte IRB. Following the revised measurement schedule, the eligibility screen was removed and instead embedded into P1 of the study.

Following these changes, all students interested in participating in the study were asked to complete an informed consent prior to beginning the survey and were informed that P1 included an embedded eligibility screen. Participants were informed that full participation would include the completion of two brief electronic assessments, scheduled 30 days apart. P1 measures took approximately one hour to complete. After the completion of P1, eligible participants were informed that they met eligibility criteria for the study and would be emailed 30 days later with an access code to complete P2 of the study. Those who were not eligible were informed that with completion of P1 questions, they consented that responses would be kept confidential for use in secondary analyses.

P2 measures were completed electronically 30 days after the completion of P1 and took approximately 15 minutes to complete. An email message was sent seven days prior to and again on the anticipated P2 date (P1 + 30 days) to remind and invite participants to complete the follow-up questionnaires within 7 days. After this time, all data were securely downloaded from the SONA system then cleared from the website.

Participant incentives. Participants enrolled in undergraduate Psychological Science courses receiving research credit through the SONA system were compensated for their time and involvement in the study. In the original measurement schedule, participants received no credits for the screening assessment, and they earned one credit for the assessment at P1 and one-half additional credit for the follow-up assessment at P2. Credit amounts were later increased with approval to encourage full participation in the study such that students were awarded 1.5 credits at the completion of P2 (2.5 credits in total). For both measurement schedules, students who completed assessments at both P1 and P2 were eligible for a drawing with an award of a $200 Amazon gift card.

With approved changes to the measurement and incentive schedule, participation increased significantly. From August 2017 to December 2017, 316 students consented to participation in P1. Of the 316 who consented to participate, 224 (70.9%) met a priori eligibility criteria and were invited to complete P2. Of the 224 eligible participants, 123 (54.9%) completed P2. The total number of study completers was 148 (25 from original measurement schedule; 123 from revised measurement schedule). Total study attrition among eligible participants from P1 to P2 was approximately 43.9%. See Figure 2 for flow diagram.

2.3 Materials

All measures were self-reported, completed electronically, and remained unchanged throughout the process of data collection. Eligible, consented participants completed all measures electronically using the SONA system. P2 measures were completed 30 days after the completion of P1 measures.

2.3.1 Screen variables

Demographic information. Information related to sex, age, race, ethnicity, marital status, employment status, housing, enrollment status (full- or part-time), education, and mother's education was collected.

History of sleep difficulties and treatment. Participants responded to questions related to past and present history of sleep difficulties and whether or not they had received clinical treatment for a sleep disorder within the past six months. Additionally, participants completed the 8-item, self-reported STOP-Bang Questionnaire (Chung et al., 2008) to determine obstructive sleep apnea (OSA) risk. Participants who scored 5 or greater were considered at high risk for OSA and were excluded from the study.

Current medications. Participants were asked to complete a checklist assessing whether or not they used specific sedative-hypnotic, antidepressant, or anxiolytic medications known to interfere with normal sleep patterns. Those who had reported taking these medications within the last six months were excluded.

Mental health history. Participants completed a checklist assessing whether or not they had ever been or were currently diagnosed by a medical professional with a list of mental health conditions (e.g., depression, anxiety, etc.). Participants who endorsed having an unlisted condition were asked to specify their diagnosis. Participants were excluded if they endorsed they were diagnosed at present or in the past by a medical professional with schizophrenia or bipolar disorder. All interested participants additionally completed the brief, 4-item Primary Care PTSD Screen (PC-PTSD; Prins et al., 2004) assessing PTSD symptoms in response to a particular stressor during the past month. Individuals who scored higher than 3 were at risk for a diagnosis of current PTSD and were excluded from the study.

2.3.2 Dependent variable

There are multiple ways to measure sleep quality. First, polysomnography (PSG) objectively measures the quality of an individual's physiological pattern of sleeping over the course of a night with use of multiple measurement devices, including electroencephalogram (EEG; measurement of brain activity), electrocardiogram (ECG; measurement of cardiovascular activity), electromyography (EMG; measurement of eye movement), respiratory and carbon dioxide monitoring equipment, and pulse oximetry (Krystal & Edinger, 2008). Resulting objective indices include information regarding total sleep time, number of times awoken, and time to fall asleep, in addition to numerical results demonstrating the percentages or amounts of sleep occurring during each of the four stages of sleep. PSG is particularly useful for identifying specific areas of sleep in which individuals experience difficulty and for diagnosing differential clinical disorders of sleep, making it the gold standard of sleep quality measurement (Marino et al., 2013). Objective measures of sleep such as polysomnography (PSG) were not collected for this study as they represent a burden to participants (i.e., we did not expect to include participants with sleep disorders that would be best diagnosed through PSG), and as they have been shown to poorly measure patterns of poor sleep related to insomnia-like sleep difficulties. Thus, the self-report approach in the current study most accurately mirrored feasible implementation of identifying poor sleep among non-clinical, young college students presenting with complaints of stress or poor sleep in a university primary care clinic or counseling center.

Actigraphy, or accelerometry-based measurement of sleep, is a second approach to measuring sleep quality. Similar to PSG, actigraphy offers researchers an objective

index of how well an individual sleeps during the course of a night. These indices include output indicating individuals' amount of time to fall asleep, number of times awoken, sleep fragmentation, and overall sleep efficiency – the overall sleep quality during the course of the night (Lockley, Skene, & Arendt, 1999; Mezick et al., 2009). Compared to PSG, actigraphy is the preferred method to measure poor sleep and insomnia-related difficulties, as individuals can wear the actigraphic devices on their wrists and monitor sleep onset and wakefulness difficulties in their natural sleeping environment at home for days or weeks, as opposed to a single night of measurement in a laboratory environment. Advantages to actigraphy include portability, low cost, and the ability to collect sleep data with multiple time points.

Though PSG and actigraphy are preferred methods of collecting objective data related to sleep quality in research, using them in this study would not realistically identify non-clinical, healthy young adults with poor sleep quality since they typically do not present to academic medical centers or sleep disorders clinics where these measurement options would be available. Additionally, wearable devices and portable fitness trackers (e.g., Fitbit®, Jawbone®, etc.), though popular and affordable, have been criticized for poor validity in sleep research due to low specificity in overestimating total sleep time and underreporting number of awakenings during sleep (Kolla, Mansukhani, & Mansukhani, 2016). Thus, our decision to use the validated and widely-used Pittsburgh Quality Sleep Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) as the sole outcome measure of poor sleep quality reflects a cost-effective, naturalistic method of capturing self-reported sleep quality. This option offers a reliable and valid measure of poor sleep when objective measures are not available or realistic, as is likely the case in

primary care, counseling, and urgent care facilities where young adults would be likely to present with complaints of poor sleep.

The PSQI consists of 19 items with 7 composite scores that assess subjective sleep quality and sleep disturbances over the course of the past month. Items were scored on a 0 to 3 scale. Seven resulting component scores targeting different sleep domains were added to create a global sleep quality score. Higher scores indicate poorer sleep quality; global scores above 5 indicate significant impairment. The PSQI is a widely utilized, valid, and reliable measure of sleep quality for clinical and non-clinical sleep disturbances (Carpenter & Andrykowski, 1998). Criterion validity for the PSQI has been established for use with young adults as a measure of sleep difficulties (Grandner, Kripke, Yoon, & Youngstedt, 2006). The PSQI was administered at P1 and P2 in the current study, with adequate test-retest reliability across measurements ($r = 0.7$, $p < 0.01$). The outcome variable in the current study was PSQI at P2, measured approximately 30 days after the PSQI at P1.

2.3.3 Part 1 measures

Baseline sleep quality. Participants completed the PSQI described above to assess subjective sleep quality in the 30 days prior to P1 measurement.

Perceived stress. The 10-item Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983) was used to assess perceptions of stress through statements about feelings and thoughts related to stressful events during the last month (0 = never; 4 = very often). Examples of questions include “In the past month, how often have you been upset because of something that happened unexpectedly” and “In the past month, how often have you felt nervous and stressed.” The PSS has demonstrated high internal reliability,

test-retest reliability, concurrent validity, predictive validity among samples in the general population (Roberti, Harrington, & Storch, 2006), with adequate validity, reliability and stability among independent samples of college students across cultures (Dias, Silva, Maroco, & Campos, 2015). Reliability in the current sample was $\alpha = 0.81$.

Perceived stress reactivity. Perceived stress reactivity was measured as an exploratory variable using the Perceived Stress Reactivity Scale (PSRS; Schulz et al., 2005). The PSRS is a 23-item scale that differentiates stress originating from six specific domains: reactivity to failure, social evaluation, social conflict, anticipatory stress, work overload, and prolonged reactivity. The domains of the PSRS have demonstrated reliability, stability across cultures, and construct validity when compared to measures of perceived stress (Scholtz et al., 2011; Scholtz et al., 2011). Furthermore, each of the six domains of PSR have been found to be uniquely independent from each other in their relationship with perceived stress (Morgan et al., 2014). In the current sample, reliability of the PSRS was $\alpha = 0.86$.

Pre-sleep arousal. The 16-item Pre-Sleep Arousal Scale (PSAS; Nicassio et al., 1985) is a brief, self-report measure in which participants rated the intensity of arousal experienced prior to sleep. The PSAS is rated on a 5-point Likert-type scale (1 = not at all; 5 = extremely), with a total score range from 8 to 40. The scale is divided into somatic (8 items) and cognitive (8 items) subscales. Sample items include "shortness of breath or labored breathing," "review or ponder events of the day," and "being distracted by noises or sounds in the environment (e.g., ticking of the clock, house noises, traffic)." The PSAS has demonstrated satisfactory internal consistency for subscales and adequate construct validity (Kohn & Espie, 2005). Young adults have been found to have less

intra-individual variability in daily PSAS scores over a two-week period than older adults, suggesting that scores in this age group are generally consistent from day-to-day (Shoji, Tighe, Dautovich, & McCrae, 2015). In the current sample, reliability of the PSAS was $\alpha = 0.86$.

Beliefs and attitudes about sleep. The 30-item Beliefs and Attitudes about Sleep Scale (BAS; Morin, 1994) was used to measure participants' general dysfunctional beliefs and attitudes towards sleep. The scale was developed to identify maladaptive cognitions associated with poor sleep quality and insomnia. Item responses were scored on a scale of 0 (strongly disagree) to 10 (strongly agree). Sample items include "when I sleep poorly on one night, I know it will disturb my sleep schedule for the whole week" and "when I have trouble sleeping, I should stay in bed and try harder." Scores were calculated averaging the 30 items. Higher scores are associated with greater dysfunctional beliefs and attitudes towards sleep. The BAS is used frequently in research on clinical and non-clinical sleep populations and has demonstrated high reliability and adequate validity across studies (Morin, Vallieres, & Ivers, 2007). Reliability in the current sample was $\alpha = 0.86$.

Sleep hygiene. The self-report Sleep Hygiene Index (SHI; Mastin, Bryson, & Corwyn, 2006) was used to assess participants' behavioral practices of sleep hygiene. Participants were asked to specify the frequency in which they engage in 13 behaviors. Sample items include "I take daytime naps lasting two or more hours" and "I use alcohol, tobacco, or caffeine within 4 hours of going to bed or after going to bed." Items were rated on a 5-point Likert-type scale (0 = never; 4 = always), with total scores ranging from 0 to 52. Higher scores indicate a global assessment of poorer sleep hygiene. The

SHI has demonstrated high reliability and construct validity and correlates highly with poor sleep quality and diagnoses of insomnia related to poor sleep hygiene (Cho, Kim, & Lee, 2013). Reliability of the SHI in the current sample was $\alpha = 0.72$.

Ruminative thinking style. The Ruminative Thought Style Questionnaire (RTS; Brinker & Dozois, 2009) is a 20-item measure of thinking and ruminative style that captures positive, negative, and neutral facets of ruminative thinking. The RTS has been considered to capture the nature of global ruminative thinking, as opposed to depressive or anxious rumination styles. Sample items include "I can't stop thinking about some things" and "if I have an important event coming up, I can't stop thinking about it." Responses were scored on a 1 (not at all descriptive of me) to 7 (describes me very well) scale. Higher scores indicated greater ruminative thinking styles. The RTS has demonstrated very high internal consistency and strong convergent validity with other measures of specific ruminative thinking styles (Tanner, Voon, Hasking, & Martin, 2013), which is consistent with the current sample ($\alpha = 0.95$).

Emotion dysregulation. The Difficulties with Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) was used to assess participants' typical levels of emotion dysregulation. The DERS is a 36-item self-report measure that results in a total score representing overall difficulties with emotion regulation. It includes the following six subscales: a) awareness and understanding of emotions, b) acceptance of emotions, c) emotional clarity, d) positive beliefs about one's ability to handle negative emotions, e) the ability to engage in goal-directed behavior when experiencing negative emotions, and f) impulse control. Participants responded to items on a 5-point Likert-type scale from 1 (almost never) to 5 (almost always). A total score was calculated by reverse scoring

appropriate items, then summing responses from all items. Higher values indicated greater emotional dysregulation. The DERS has been found to have high internal consistency, adequate construct and predictive validity, and consistency across race and gender groups (Ritschel, Tone, Schoemann, & Lim, 2015). In the current sample, the reliability of the DERS was $\alpha = 0.93$.

Sleep-related worry. Sunnhed and Jansson-Fröjmark's (2014) 6-item measure of sleep-related worry was administered. Factor analysis results have indicated that this scale is comprised of two factors, worry for sleeplessness (e.g., "I worry about my sleep when I cannot fall asleep") and worry for health as a result of not sleeping ("I worry that my body will be harmed if I sleep poorly). Participants responded using a 5-point Likert-type scale (1 = totally disagree; 5 = totally agree). Higher summed scores indicated greater SRW. Reliability in the current sample was $\alpha = 0.90$.

2.4 Statistical analyses

The SPSS Version 24.0 (IBM, 2017) statistical software package was used for data management and statistical analyses. Using SPSS, descriptive statistics were conducted to identify missing data, out-of-bound values, and outliers. Invalid responses were identified and removed from analyses. Skew and kurtosis were assessed to determine normality. Reliability coefficients were calculated across all measurement scales. Pearson's bivariate correlations were conducted to explore the basic linear relationship among continuous variables. Independent samples' t-tests were used to evaluate group differences among categorical demographic variables with the dependent variable (DV), poor sleep quality at P2. Significant demographic variables were included as covariates in secondary analyses.

To test Hypothesis 1, path analyses were conducted to examine the mediating role of pre-sleep arousal on the relationship between perceived stress and perceived stress reactivity and poor sleep quality. Bootstrapping analyses following Hayes' (2012) SPSS PROCESS macro for multiple mediation with 5,000 bootstrap iterations with 0.95 confidence intervals were used to determine confidence intervals and the significance of indirect effects.

For testing the moderation presented in Hypotheses 2 and 3, product terms were created using centered variables to explore the interaction of each moderator with the predictor and mediator on the outcome variable. For H2, a product variable was created for the each of the interactions of perceived stress with beliefs and attitudes about sleep (H2a; perceived stress x BAS), poor sleep hygiene behaviors (H2c; perceived stress x SHI), ruminative thinking style (H2e; perceived stress x RTS), and emotion dysregulation (H2g; perceived stress x DERS) on PSA. For H3, product variables using centered variables were created for the interactions of emotion dysregulation (H3a; PSA X DERS) and PSA with SRW (H3b; PSA X SRW) on poor sleep quality and were included in moderated mediation analyses following the conditional PROCESS analysis macro for SPSS (Hayes, 2017). Any significant interactions were planned to be plotted for significant path coefficients between product variables and the dependent variable using the equations $M = a_1 + b_1X_1 + b_2X_2 + b_3X_1X_2$ and $Y = a_1 + b_1X_1 + b_2M_1 + b_3X_1X_2 + b_4M_1X_2 + b_5X_2$.

CHAPTER THREE: RESULTS

3.1 Descriptive findings and preliminary analyses

Of the 148 participants who completed the study, one did not meet the age restriction criteria and five were identified as having limited response variability or missing data greater than 50% for scaled measures. Thus, six cases were removed from analyses, and the final sample size consisted of 142 participants.

Demographic characteristics are provided in Table 1. A majority of the sample identified as female, white, and non-Hispanic (91.6%). Participants ranged in age from 18 to 24 years (M = 18.85, SD = 1.13). The sample consisted primarily of unmarried, employed, full-time enrolled, first-year undergraduate students living in an on-campus apartment or dormitory.

Means and standard deviations for all variables included in planned analyses are reported in Table 2. Histograms and frequency analyses indicated that all variables demonstrated a normal distribution and adequate variability. Participants reported a mean of 6.82 hours (SD=1.18) of sleep per night at P1 and 6.80 hours (SD=1.26) at P2. While 52.1% and 56.4% reported receiving 6-8 hours of sleep per night respectively at P1 and P2. Surprisingly, only 8.5% of participants at P1 and 7.7% at P2 endorsed receiving the self-reported ideal of 8.5 hours per night for college-aged adults. On average, participants reported poor-quality sleep (PSQI score > 5) at assessments for both Part 1 and Part 2, with 91.5% of the sample endorsing significant sleep disturbance at P2 (see Table 2). Mean PSQI scores increased from P1 to P2, indicating slightly poorer average sleep quality 30 days later. However, 97.9% of the current sample reported PSQI change scores within 3-point values from P1 to P2, which is not clinically significant and is consistent

with the literature suggesting that self-reported sleep quality remains unchanged longitudinally in the absence of interventions to improve sleep (Backhaus et al., 2002; Buysse et al., 1989).

Zero-order correlations were conducted among demographic and study variables (see Table 3). Increased age was associated with poorer sleep quality P1 and P2. Female sex was positively associated with higher levels of perceived stress and perceived stress reactivity, as well as poorer sleep quality at P2. White race was associated with healthier beliefs and attitudes about sleep; Black race was significantly associated with increased sleep-related worries, greater emotional dysregulation, and less accurate beliefs and attitudes about sleep. Employment was associated with poorer sleep quality at P2, while unemployment was associated with better reported sleep quality. Living off campus was associated with higher pre-sleep arousal. T-tests indicated that females reported poorer sleep quality at P2 than did males ($t(140) = -1.97$, $p = .05$) and that employed participants reported significantly poorer sleep quality at P2 than did unemployed participants ($t(140) = -2.27$, $p < .05$). Living location and racial/ethnicity identification were not significantly associated with poor sleep quality and were thus not included as covariates in secondary analyses. Gender and employment status were not predictive of variance in sleep quality in regression analyses and were removed as covariates in analyses.

Table 4 shows correlations among study variables. The pattern of associations was consistent with expectations, indicating moderate-to-strong associations among all variables. As expected, sleep quality at P1 was highly correlated with sleep quality at P2. Given the magnitude of the association between P1 and P2 sleep quality, sleep quality at P1 was removed as a covariate in subsequent analyses. Of note, the two predictor

variables, perceived stress and perceived stress reactivity, were also highly and positively correlated with each other, as well as with ruminative thinking style and emotion dysregulation.

3.2 Regression analyses

Hypothesis 1. The first hypothesis postulated that pre-sleep arousal (PSA) would mediate the relationships between both perceived stress and perceived stress reactivity (PSR) and poor sleep quality at P2. A path analysis was originally conducted including both predictors with PSA as the mediator (see Figure 3). Results indicated that both perceived stress reactivity and perceived stress had significant direct effects on pre-sleep arousal, though only perceived stress had a significant direct effect on poor sleep quality (see Table 5). PSR did not have a significant direct effect on poor-sleep quality. Pre-sleep arousal, as expected, demonstrated a significant direct effect on poor sleep quality.

Due to the high correlation between perceived stress and perceived stress reactivity, independent path analyses were run to explore both the direct and indirect associations of perceived stress and perceived stress reactivity with sleep quality at P2 (see Figure 4). These analyses uncovered that the path coefficients for the direct effects between both perceived stress (PS) and PSR with PSA increased significantly and with similar magnitude. Second, the direct effect of PSR on poor sleep quality increased slightly and was borderline significant. Furthermore, the magnitude of the path coefficient from PSA to poor sleep quality, significance of the indirect effects, and the variance as measured by R^2 remained consistent in both independent models (R^2 PS = 0.37; R^2 PSR = 0.33). Because similar patterns and directions among relationships emerged, it was determined that including both perceived stress and PSR as independent

predictors of PSA and poor sleep quality was redundant, without adding variance to the model. Thus, for parsimony and based on available empirical support for the construct of perceived stress, it was decided to move forward with a mediational model including only perceived stress for subsequent moderation and conditional process analyses.

Table 6 lists the direct, indirect, and total effects of the updated model (as seen in Figure 4a) exploring the mediation of pre-sleep arousal on the relationship between perceived stress and poor sleep quality. Bootstrapping analyses indicated that the indirect effect of perceived stress on poor sleep quality was significant ($b = 0.06$, $p < 01$, CI = 0.03 - 0.09). The magnitude of the path weight of perceived stress on pre-sleep arousal was strong as hypothesized. However, contrary to expectations, pre-sleep arousal only partially accounted for the relationship between perceived stress and poor sleep quality. Thus, Hypothesis 1 was partially supported.

Hypothesis 2. The second hypothesis postulated that cognitive (i.e., dysfunctional beliefs and attitudes about sleep, ruminative thinking style, and emotion dysregulation) and behavioral factors (poor sleep hygiene behaviors) associated with sleep quality would moderate the relationship between perceived stress and PSA. Four models were tested. Perceived stress and each of the four centered moderators were entered at Step 1. Step 2 included the addition of two-way interaction terms using centered variables for each of the four moderators with perceived stress (see Table 7).

As expected, sleep hygiene, emotion dysregulation, and ruminative thinking style were significant predictors of pre-sleep arousal at both Step 1 and Step 2 (see Table 7). However, dysfunctional beliefs and attitudes about sleep (BAS) did not significantly predict pre-sleep arousal. Furthermore, contrary to hypotheses, beliefs and attitudes about

sleep (H2a), poor sleep hygiene behaviors (H2c), ruminative thinking style (H2e), or emotion dysregulation (H2g) did not moderate the relationship between perceived stress and pre-sleep arousal. Hypothesis 2 was not supported.

Hypothesis 3. The third hypothesis postulated that emotion dysregulation and sleep-related worry (SRW) would independently moderate the mediating effect of pre-sleep arousal on the relationship between perceived stress and poor sleep quality. Two moderated mediation analyses were conducted using the PROCESS macro for SPSS (Hayes, 2017), with poor sleep quality as the dependent variable, perceived stress as the independent variable, pre-sleep arousal as the mediator, and age as covariate.

In the first model, emotion dysregulation (ED) was included as a moderator of the mediating effect of pre-sleep arousal on poor sleep quality (H3a). Findings indicated direct effects of perceived stress ($b = 0.10$, $p < .01$) and pre-sleep arousal ($b = 0.13$, $p < .10$) on sleep quality at P2. Further, a partial direct effect of age ($b = 0.50$, $p < .01$) on sleep quality at P2 emerged. Contrary to expectations (H3a) the indirect effect of perceived stress on poor sleep quality through pre-sleep arousal was not conditional on emotion dysregulation, and the mediation effect was significant across all levels of the moderator (Index = -.0003, Boot SE = .0005, Boot LLCI = -.0014, Boot ULCI = .0006). This model accounted for 37.5% of the variance in poor sleep quality at P2.

In the second model, sleep-related worry (SRW) was included as a moderator of the hypothesized mediational effect of pre-sleep arousal (H3b). Significant direct effects of perceived stress ($b = 0.10$, $p < .01$) and pre-sleep arousal ($b = 0.09$, $p < .10$) on poor sleep quality at P2 emerged. In addition, a partial effect of age ($b = 0.49$, $p < .01$) on poor sleep quality at P2 was identified. Contrary to expectations, the results indicated that the

effect of perceived stress on poor sleep quality at P2 through pre-sleep arousal was not conditional on levels of sleep-related worry, and the indirect effect was significant across all levels of SRW (Index = -.0007, Boot SE = .0072, Boot LLCI = -.0049, Boot ULCI = 0.002). This model accounted for 39.9% of the variance in poor sleep quality. However, SRW did significantly predict pre-sleep arousal ($b = 0.42$, $p < .01$).

Overall, contrary to expectations, neither emotion dysregulation nor SRW moderated the mediation effect of pre-sleep arousal on poor sleep quality. Thus, Hypothesis 3 was not supported.

CHAPTER FOUR: DISCUSSION

4.1 Discussion of findings

The purpose of this research was to examine the processes by which stress contributes to poor sleep among a non-clinical sample of young adults. Stress is well-documented in its dysregulatory effects on homeostasis (Chrousos & Gold, 1992), and the sleep-wake cycle is one recognized homeostatic process disrupted by the physiological and psychological mechanisms of stress (McEwen, 1998). Young adults are the fastest growing cohort reporting poor sleep, yet theoretical pathways by which stress contributes to poor sleep are less documented in the literature. Therefore, we aimed to test a hypothesized conceptual model that may help guide our understanding of the relationship between both perceived stress and PSR with poor sleep in this age group.

Surprisingly, the majority (91.5%) of study participants met the criteria for classification as poor-quality sleepers (PSQI > 5). In past studies of young adults with larger sample sizes, closer to 40 – 60% were classified as poor-quality sleepers (Lund, 2010; Taylor, Bramoweth, Grieser, Tatum, & Roane, 2013). Though the overall range of variablility across the scale for the current study was minimal for good-quality sleepers (range score 3 to 5), there was a high degree of variability across scores at higher levels of poor sleep quality (range score 6 to 17) which suggests that among poor-quality sleepers, the degree to which young adults endorse sleep disturbances ranges from borderline-poor to extremely poor. Overall, it appears that clinical symptom elevations remain untreated among healthy young adults, supporting the need for intervention with this group. Further, this study assessed one month of self-reported sleep quality, which is considered an acute time period. Change in PSQI scores was minimal (< 3 points) and

not clinically significant, suggesting that those who reported poor sleep at baseline are also reporting poor sleep 30 days later. Importantly, acute sleep difficulties progress to what is considered chronic sleep difficulties after 3 months.

One explanation for the high proportion of students reporting poor sleep quality could be the rapid changes in amount and frequency of technology use since the rise of smartphone and social media usage in the past decade (Twenge, Martin, & Campbell, 2018), and more screen time has been associated less sleep in adolescents (Barlett, Gentile, Barlett, Eisenmann, & Walsh, 2012). In one example, Exelmans and Van den Bulck (2017) found that binge viewing of television shows and movies via Internet streaming services by smartphone and laptop was significantly associated with sleep disturbance in college students. What is more, they found that binge viewing also contributed to higher levels of pre-sleep arousal (PSA). Additionally, in a recent study of 249 college students (a sample which includes 25 participants also enrolled in the current study), 83% of participants endorsed using three or more electronic devices in the hour prior to sleep (Smith-Mason & Gil-Rivas, 2018). Furthermore, the number of devices used and pre-sleep laptop use significantly predicted pre-sleep arousal, and pre-sleep laptop use was associated with insomnia symptoms.

A second explanation for the proportion of students reporting poor sleep quality may be explained by demographic characteristics. The sample majority identified as female and employed. Female gender has been a known predisposing characteristic for the development of insomnia (LaBlanc et al., 2009), and in our sample women reported poorer sleep quality than did men. In addition, the finding that those who were employed had poorer sleep quality at P2 compared to those who were not employed was

unexpected. Specifically, unemployment at P1 was associated with better sleep quality at P2, and employment at P1 was associated with poorer sleep quality at P2. Given the academic demands expected of full-time university students (97.9% of the sample), it appears that the added stress or burden of employment (52.1% of the sample) may contribute to poorer sleep quality 30 days later significantly more than those who are unemployed. This suggests there are multiple, conflicting demands required of young adults. These individuals may not yet have the skills to navigate the demands and stressors of work in addition to the demands and stressors of school, thus contributing to noticeable differences in sleep quality among the two employment status groups.

This study tested a hypothesized conceptual model examining two indicators of stress, a situational, state-like form of stress (perceived stress), and a predisposing, trait-like form of stress (PSR) and their influence on poor sleep quality. First, we proposed that pre-sleep arousal (PSA) would fully mediate the relationship between perceived stress, perceived stress reactivity, and poor sleep quality, since high cognitive and somatic arousal is observed frequently among individuals reporting clinical sleep difficulties in the hour before sleep. Beyond this mediation, we also hypothesized that dysfunctional beliefs and attitudes about sleep (BAS), sleep hygiene behaviors, emotion dysregulation, and ruminative thinking style (RTS) would moderate the relationship between perceived stress and PSA. Each of these processes has been hypothesized to influence how individuals cope with stress or manage pre-sleep thoughts and behaviors, which may contribute to differences in levels of PSA prior to falling asleep. Finally, we hypothesized that the mediating effect of PSA on poor sleep quality would be conditional

on levels of emotion dysregulation and sleep-related worry (SRW), for their likely roles in maintaining arousal that may disrupt sleep quality.

Preliminary analyses revealed that contrary to expectations PSR had considerable overlap with the more commonly used construct of perceived stress and demonstrated a similar pattern of associations with pre-sleep arousal and poor sleep quality. Jackowska, Fuchs, and Klaperski (2017) reported similar conflicting patterns when including both PSR and a measure of perceived chronic stress as predictors of sleep disturbance. Parallel to our findings, they found that while PSR predicted poor sleep, the addition of chronic stress attenuated that relationship such that only chronic stress accounted for the variance in sleep. Thus, as a singular variable, PSR appears to capture a similar relationship with PSA and poor sleep quality as does perceived stress; however, exploration of the six specific domains of PSR that may greater influence poor sleep quality could help better understand the relationship between stress and poor sleep among young adults. This approach was first suggested by Morgan, Umberson, and Hertzog (2014), and Herr and colleagues (2018) indeed found that an intervention to reduce stress reactivity among adult males yielded outcomes which identified three domains of stress specifically related to work overload, social conflicts, and prolonged reactivity – or the inability to recover to baseline after a period of prolonged stress – as significant contributors to sleep difficulties.

For our primary hypothesis, we had expected that PSA would fully mediate the association between perceived stress and poor sleep quality at P2 for its ability to capture evening cognitive and somatic elevations stemming from stress during daytime wakefulness. As expected, high associations among perceived stress, PSA, and poor sleep

quality were observed. However, we uncovered a partial – rather than full – mediation of PSA. This finding is theoretically important, for it suggests that a while a portion of perceived stress directly influences poor sleep quality, a significant indirect effect of that relationship is also accounted for by one's level of pre-sleep arousal. What is more, this means that perceived stress continues to predict poor sleep quality independently of potential influencing factors, including pre-sleep arousal.

Similar associations have been observed among clinical sleep disorder samples (e.g., Chen, 2017). However, to our knowledge our study was the first to examine this relationship among a non-clinical sample with both female and male participant young adults. A study by Winzeler and colleagues (2014) also explored PSA as a mediator of stress and sleep quality among non-clinical young adults (*M* age = 21.74); however, their sample included only females, and data collection occurred over a two week period using a ecological momentary assessment approach. Their findings indicated pre-sleep arousal mediated the relationship between daily stress and subjective sleep quality, both at the within- and between-participant levels; they also found that on days where participants reported higher stress, they reported increased levels of pre-sleep arousal. This finding suggests that treatments for poor sleep in young adults should incorporate psycho-education about the role of stress on pre-sleep arousal and sleep difficulties, as well as intervention components for effective stress management (Morin, Rodrigue, & Ivers, 2003).

Taken together, these findings support the use of a stress-diathesis model, such as Speilman's 3-P model of insomnia (Spielman, Caruso, & Glovinsky, 1987) to understand factors that contribute to poor sleep. Specifically, the model suggests that individual

differences in biological and psychological characteristics may increase susceptibility or vulnerability of developing later sleep difficulties under conditions of stress.

Guided by this model, we expected that pre-sleep cognitive, affective, and behavioral processes would perpetuate poor sleep quality by moderating the relationship between perceived stress and PSA. Specifically, we hypothesized that under conditions of perceived stress, individuals reporting more dysfunctional BAS, poorer sleep hygiene behaviors, higher levels of RTS, and greater emotion dysregulation would be more likely to report increased PSA; however, only sleep hygiene behaviors, emotion dysregulation, and RTS had significant direct effects on pre-sleep arousal.

We were initially surprised to see no moderating effects, particularly among sleep hygiene behaviors, since under conditions of stress, individuals have been observed to engage in behaviors such as shifting sleep schedules (Mastin, Bryson, & Corwyn, 2006), using alcohol or other substances to induce sleep (Stepanski & Wyatt, 2003), and spending time in bed prior to sleep actively planning for future events (Querstret & Cropley, 2012), which likely contribute to pre-sleep arousal. However, the lack of interactive effects in the sample is not as surprising. Instead, our findings indicated that in addition to perceived stress, sleep hygiene behaviors, RTS, and emotion dysregulation directly contribute to PSA.

Upon closer examination of these effects, RTS and emotion dysregulation are state-like cognitive-affective processes that are not typically included in research on poor sleep quality. We found that young adults who report higher levels of ruminative thinking and higher emotion dysregulation are also reporting increased PSA. In the past, sleep research has centered on rumination over poor sleep (Carney et al., 2010), not generalized

rumination. Our findings indicate that ruminating on general situations or events – positive or negative – similarly interferes with sleep quality by directly increasing arousal in the time prior to sleep. Furthermore, our outcomes demonstrating difficulties with emotion regulation contribute to pre-sleep arousal is consistent with Babson and Feldner (2015)'s assertion that poor management of affective responses contributes to greater arousal prior to sleep, which contributes to poorer sleep quality.

One explanation put forth from research by Cox, Ebesutani, and Olatunji (2016) posits the relationship between sleep and rumination may be due to deficits in executive functioning. Executive functioning is typically attributed to the frontal lobe, and frontal-cortical functioning is also critical in the modulation of emotional regulation (Banks et al., 2007). Frontal lobe development continues to form throughout late adolescence and young adulthood until the approximate age of 25; thus, targeting college-aged young adults for intervention highlights the plasticity of this age group to potential lifetime improvements in ruminative thinking and emotion regulation as a means to improve sleep and future health.

In contrast, dysfunctional beliefs and attitudes about sleep did not significantly predict PSA. This is not surprising, as to our knowledge this relationship had not previously been explored or noted in the sleep literature. BAS represent a measure individual's core beliefs about sleep. Thus, they may represent a stable cognitive set that may not influence perceived stress and does not contribute to fluctuating pre-sleep arousal in the time prior to sleep. Instead, BAS may have an alternative role as an independent cognitive predictor of poor sleep quality. This would be consistent with other research that has found, when maladaptive, these core BAS contribute directly to

insomnia symptoms (Morin, 1997; Hertenstein et al., 2015; Vand, Gharraee, Farid, & Bandi, 2014). Furthermore, BAS have been shown to be improved with psychoeducation and cognitive interventions that do not target arousal (Morin, 1997; Ong, Shapiro, & Manber, 2008).

In sum, our findings are important, as they add to the literature on pre-sleep arousal, suggesting that ruminative thinking, emotion dysregulation, and sleep hygiene are processes that contribute directly to pre-sleep cognitive and somatic arousal in the hour before sleep, regardless of one's perception of stress. In turn, these findings suggest that interventions to improve PSA and subsequent sleep quality may need separate components to teach stress management and affective regulation in addition to the core cognitive and behavioral components that Cognitive Behavioral Treatment for Insomnia (CBT-I) currently targets as perpetuating factors of poor sleep.

Furthermore, the study also explored if the mediational effects of PSA on poor sleep quality at P2 were conditional on levels of emotion dysregulation or SRW; these hypotheses were not supported. However, the inability of the study to detect these effects was not surprising (Whisman & McClelland, 2005) given that individuals reporting higher levels of PSA were also reporting high levels of emotion dysregulation and sleep-related worry. The magnitude of these associations likely explained the difficulties detecting the moderating effects and the conditional effects of PSA on poor sleep quality. One way to improve the power to detect these effects in future studies would be to increase sample size and examine these associations using structural equation modeling (SEM). Using SEM, highly associated measures such as PSA, SRW, and emotion dysregulation could be created into a single latent construct using confirmatory factor

analysis. These latent constructs would then allow for simultaneous measurement of mediating and moderating effects between constructs and underlying factors.

We did not uncover direct effects of emotion dysregulation or SRW on poor sleep quality, nor did we detect moderating effects of emotion dysregulation or SRW on the mediation of PSA on poor sleep quality at P2. However, the finding that SRW significantly predicted PSA was an unanticipated outcome. This finding is new to the literature on sleep, as SRW has been primarily hypothesized to directly affect poor sleep quality. However, SRW is still a relatively new construct (Sunnhed & Jansson-Fröjmark, 2014); thus, its inclusion in models of poor sleep quality has been missing. Our findings add to what is known about the relationship between worry and sleep, confirming that SRW is indeed a separate cognitive construct from rumination (RTS), as was suggested by Carney and colleagues (2010). Furthermore, our findings suggest that SRW, in part, may also contribute to PSA.

Age was significant as a covariate across all analyses with poor sleep quality as the outcome. Participants in this study were primarily in their first two years of college, a period of time when young adults report sleep difficulties shortly after their transition to college (Cheng et al., 2012). Further, sleep quality was worse among older students which suggests that poor sleep continues beyond the immediate transition into college. These same difficulties have been found to continue after college in the workforce, where daily work demands – or stressors – have been found to increase pre-sleep arousal and contribute to greater disruption of sleep patterns (Loft & Cameron, 2014). This finding adds support to our suggestion that early identification, prevention, and intervention

efforts are likely to be critical for young adults as a means to delay or negate the detrimental effects of poor sleep over time.

4.2 Limitations

Though the present study offers benefits for advancing the body of knowledge related to the pathways by which perceived stress contributes to poor sleep quality in college-aged young adults, limitations exist. Firstly, data were collected at two time points over a thirty-day period, which may not adequately represent the temporal relationship among variables as is necessary to determine true mediation. Importantly, the Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983) and the Pittsburgh Quality Sleep Index (PSQI; Buysse et al., 1989) are intended to capture perceived stress and subjective sleep quality over the past thirty days, so our use of these valid measures at appropriate intervals does in fact capture a temporal relationship from Part 1 (P1) to Part 2 (P2).

Secondly, perceived stress was not assessed at P2 which may be perceived as a limitation, for one might argue that stress levels may have changed between the two measurement time points due to potential confounds of participants' experience of heightened stress due to traumatic or other stressful events such as midterm or final exams. Participants who met criteria for risk of post-traumatic stress on the PC-PTSD (Prins et al., 2004) at either time of measurement were excluded from data analyses (no participant met these criteria at P2). Also, participant enrollment was on a rolling admission, with thirty days (+/- 7 days) from P1 to P2, so it is unlikely that all students were experiencing heightened stress at similar times of P1 and P2 completion. Summed perceived stress scores across participants indicated that students experienced on average

mild to moderate stress at P1 (M = 15.83, SD = 6.61, range 0 – 40); further, perceived stress showed a similar association with poor sleep quality scores at both P1 (r = 0.44, p < .01) and P2 (r = 0.47, p < .01), suggesting that perceived stress at P1 had similar associations with PSQI scores at P1 and P2.

Thirdly, the study is limited by the lack of objective measurement of sleep. The standard approaches to objective sleep measurement include polysomnography (PSG) and actigraphy, or accelerometry-based measurement of sleep. These options are expensive and are not frequently used outside of sleep disorders research or in clinics as assessment methods. Using these objective measures would not have realistically yielded identification of healthy individuals with poor sleep quality who do not present to academic medical centers or research clinics. Therefore, the decision to use the validated and widely-used PSQI as self-report measures of sleep quality reflects a cost-effective, naturalistic method of capturing stress levels and sleep quality. This self-report approach most accurately mirrors feasible implementation of identifying poor sleep among non-clinical, young college students presenting with complaints of stress or poor sleep in a university student health center, primary care clinic, or counseling center where PSG testing would not likely be available. Despite this limitation, our findings using self-reported measures in the exploration of a conceptual model including pre-sleep arousal as a partial mediator of the relationship between stress and poor sleep among healthy young adults parallel the pattern of findings from a recently published dissertation (Chen, 2017) that used objective measures of sleep quality (PSG + accelerometry) and of the stress response (cortisol + heart rate variability) in a sample of young adults meeting the diagnostic criteria for insomnia. This demonstrates how our results that pre-sleep arousal

partially mediates the relationship between stress and poor sleep from a non-clinical sample with self-reported data mirrors outcomes from a clinical sample with objective data.

Furthermore, the present study has two noticeable strengths. First, to our knowledge, no other study has examined the relationship between stress and sleep quality at multiple time points among healthy young adult men and women. Second, this is a theory-based project, with results that have the ability to guide the development of brief interventions for college-aged young adults – the majority of whom appear to be experiencing difficulties with sleep. Indeed, this study respond to Mullan's (2014) suggestion that what is missing in our understanding of stress and sleep *a priori* is theoretical-guided research to identify predictors of poor sleep.

4.3 Future directions

Future research will benefit from the inclusion of objective, physiological measures of sleep quality, as well as daily diaries exploring the timing and frequency of daytime stressors along with patterns of sleep latency, awakenings, and duration, and details regarding pre-sleep technology use. Objective measurement using PSG and actigraphy approaches, though expensive, may provide details into the physiological patterns and effects of poor sleep on the body over time in an age group that is not often observed in sleep laboratories. These measures can be use in combination with self-reported sleep diaries to capture information related to both objective and subjective sleep quality. An example of one such measure is the Consensus Sleep Diary (Carney et al., 2012), which has become the top choice of daily diaries to monitor sleep patterns for its inclusion of detailed information regarding time in bed, out of bed, sleep latency, number of

wakenings, and time spent awake in the middle of the night. This measure also has the option of monitoring pre-sleep eating, exercise, caffeine intake, and substance use (e.g., nicotine, alcohol) behaviors, which are known facilitators of poor sleep hygiene (Stepanski & Wyatt, 2003). Currently, validated sleep diaries do not measure daytime stressors, which would provide additional information that could guide combined interventions to improve stress management and sleep quality.

Lastly, the current study offers a clearer understanding of the role of pre-sleep arousal as a mediator of stress and poor sleep, of which pre-sleep technology and social media use may contribute a key role as perpetuators of pre-sleep cognitive, somatic, and affective arousal (Exelmans & Van den Bulck, 2017). With high percentages of college-aged young adults reporting pre-sleep technology use, exploration into detailed patterns of screen time and social media use will be crucial for understanding sleep difficulties in this age group moving forward. Future studies can use these findings in three ways: to guide the development of validated measurement devices which will permit researchers to investigate mechanisms by which pre-sleep screen time contributes to patterns of stress and arousal prior to sleep; to aid in the development of interventions that educate and manage daytime or pre-sleep responses to technology; or even use technology as means by which we can improve poor sleep through web- or application-based interventions, among which the United-Kingdom based program Sleepio® has been gaining evidence-based support as being an effective digital tool in the dissemination of CBT-I (Luik & Kyle, 2017).

4.4 Conclusions

Taken in aggregate, the current findings indicate that healthy, young adults are reporting high levels of poor sleep quality. Further, even in the context of mild-to-moderate, self-reported stress we are seeing that perceived stress is influencing PSA which in turn contributes to poor sleep quality 30 days later. Moreover, our findings that emotion dysregulation, RTS, sleep hygiene behaviors, and SRW significantly contribute to PSA add to what is known about the pathways by which cognitive, affective, and behavioral processes perpetuate poor sleep quality. College-age individuals have been largely remiss from the literature on sleep disorders, as they do not regularly present for insomnia treatment at locations where offered; thus, these findings offer insight into the nature and mechanisms of the influence of stress on poor sleep quality in this typically non-clinical group of young adults. However, what is concerning is that these findings parallel relationships observed among samples of patients meeting clinical diagnostic criteria and receiving treatment for insomnia disorders. Consequently, these results may benefit future research in that they can serve as the baseline for the development of brief interventions for stress management, affective regulation, and improvement of poor sleep in this age group.

Cognitive Behavioral Treatment for Insomnia is the intervention of choice for complaints of poor sleep (Spielman, Caruso, & Glovinsky, 1987). The primary factors of treatment using a CBT-I approach include sleep compression/restriction, stimulus control, sleep hygiene, and cognitive therapy, which can target sleep-related worries and ruminative thinking style. Not only has PSA been found to influence sleep quality; it has also been identified as a mediator to the effects of CBT-I (Sunnhed & Jansson-Fröjmark, 2015) on sleep quality. Thus, pre-sleep arousal can be viewed as a mechanism to improve

sleep quality as well as a barrier to the effects of CBT-I when not adequately addressed in clinical treatment.

CBT-I includes pre-sleep relaxation components (e.g., progressive muscle relaxation) aimed at decreasing unwanted somatic tension and intrusive thoughts at night (Morin, 1994) that are the core of pre-sleep arousal. However, evidence increasingly indicates that daytime mindfulness practices (Kabat-Zinn, 1990), rather than night-time pre-sleep relaxation, are more effective in decreasing PSA and daytime hyperarousal (Ong, Xia, Smith-Mason, & Manber, 2018), with stable reductions in Pre-Sleep Arousal Scale (PSAS; Nicassio et al., 1985) scores lasting up to six-months post-intervention (Ong et al., 2014). Nested within the scope of mindfulness-based treatments, Dialectical Behavior Therapy (DBT; Linehan, 1993) may offer an additional component that teaches young adults, whose amygdala and frontal lobe growth are still in development, skills to manage affective ruminative and emotion regulation responses that have been found in the present study to predict pre-sleep arousal. Furthermore, a review (Ong & Smith, 2017) of the seven, completed randomized controlled trials of mindfulness-based treatments for insomnia found support for their efficacy for decreasing pre-sleep arousal and hyperarousal and for improving sleep quality and reducing sleep disturbance, supporting that these interventions could be implemented as feasible complements or alternatives to CBT-I or pharmacological treatment for clinical insomnia. Thus, mindfulness- based and DBT skills training interventions may be what are missing in the prevention and treatment of poor sleep.

In conclusion, the findings from the current study add to our theoretical understanding of what is known about the predisposing and perpetuating factors that

influence sleep quality among an understudied, healthy, non-clinical, young adult, student population. These findings can be used to guide the implementation of interventions to help this group better manage stress to improve sleep quality, which has implications for consequent health outcomes. Specifically, a proposed future project will be to develop and test a brief intervention that includes psychoeducation of the stress response as part of the protocol for CBT-I, mindfulness meditation for the management of stress and ruminative thinking, and DBT strategies for the development of emotion regulation strategies critical to this developmental stage – given the vulnerable window of young adults for increased stress – to improve present and future health and to protect from the development and progression of disease over time.

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APPENDIX A: TABLES

Table 1. Demographic Characteristics of the Sample

		%
Gender		
	Male	33.6
	Female	66.4
Race		
	White/non-Hispanic	68.1
	Black/African American	14.9
	Asian	7.1
	Native American/Alaska Native	1.4
	More than one race	4.3
	Other/Not reported	4.2
Mother's education		
	Some high school/No GED	6.3
	High school diploma/GED	16.1
	Some college (less than 4 years)	29.4
	Bachelor's degree	36.4
	Graduate/Professional degree	11.8
Undergraduate level		
	Freshman	55.2
	Sophomore	30.8
	Junior	10.5
	Senior	3.5
Enrollment status		
	Full-time	97.9
	Part-time	2.1
Employment status		
	Unemployed/Not working	47.9
	Employed	52.1
Marital status		
	Single	97.2
	Married/Civil union	0.7
	Live-in-partner	1.4
	Did not report	0.7
Housing		
	Apartment/Dorm on campus	49.3
	Apartment/House off campus	23.9
	Lives with parents	26.8

Note. N=142.

Table 2. Descriptive Statistics of Study Variables

	M	*SD*	Range
P2 Poor sleep quality	8.44	2.58	3.00 - 17.00
P1 Poor sleep quality	8.00	2.12	4.00 - 12.00
Pre-sleep arousal	27.99	9.51	15.00 - 56.00
Perceived stress	15.83	6.61	2.00 - 34.00
Perceived stress reactivity	21.12	7.68	1.00 - 40.00
Beliefs and attitudes about sleep	3.67	1.42	0.80 - 6.97
Sleep hygiene behaviors	21.32	6.71	6.00 - 38.00
Ruminative thinking style	78.42	26.32	20.00 - 140.00
Emotion dysregulation	81.00	21.17	43.00 - 130.00
Sleep-related worry	14.40	6.38	6.00 - 30.00

Note. N=142. P2 = Part 2; P1 = Part 1. All scales are measured such that higher scores indicate greater dysfunction.

Table 3. Correlations of Demographic Variables with Stress and Sleep Variables

	P2 Sleep Quality	P1 Sleep Quality	PSA	SRW	BAS	SHI	RTS	DERS	PSS	PSR
Age	**0.28****	**0.28****	0.08	0.06	0.11	-0.09	0.11	0.07	0.12	0.06
Female	**0.16***	0.12	0.08	0.01	0.04	0.08	0.16	0.04	**0.20***	**0.30****
White	-0.14	-0.04	0.04	-0.08	**-0.18***	-0.01	-0.15	-0.07	-0.16	-0.10
Black	0.01	-0.02	-0.02	**0.19***	**0.24****	0.12	0.00	**0.18***	0.03	0.03
Other race	0.14	0.10	-0.04	-0.06	0.02	-0.12	0.13	-0.06	0.10	0.05
Ethnicity	0.01	-0.05	0.02	0.15	0.05	0.10	0.14	-0.05	0.08	0.01
Mother's education	0.04	0.07	-0.08	-0.07	-0.04	0.00	0.02	-0.03	-0.05	-0.10
Class Standing	**0.18***	0.13	0.08	0.08	0.03	-0.08	0.04	0.08	0.11	0.13
Employed	**0.19***	0.13	0.06	0.07	0.11	-0.06	-0.03	-0.11	0.11	-0.01
Lives on campus	-0.06	-0.08	-0.15	-0.13	0.01	0.13	-0.02	0.02	-0.02	0.01
Lives off campus	0.07	0.14	**0.24****	0.15	0.08	-0.12	0.10	0.04	0.15	0.15
Lives with parents	-0.01	-0.05	-0.06	0.00	-0.08	-0.03	-0.08	-0.07	-0.13	-0.16

Note. * $p < .05$. ** $p < .01$. All primary study variables are scored such that positive correlation coefficients signify greater dysfunction. P2 = Part 2; P1 = Part 1; PSA = Pre-Sleep Arousal; SRW = Sleep-Related Worry; BAS = Beliefs and Attitudes about Sleep; SHI = Sleep Hygiene Index; RTS = Ruminative Thinking Style; DERS = Difficulties with Emotion Regulation Scale; PSS = Perceived Stress Scale; PSRS = Perceived Stress Reactivity.

Table 4. Correlations between Stress and Sleep Variables

	P2 Sleep Quality	P1 Sleep Quality	PSA	SRW	BAS	SHI	RTS	DERS	PSS	PSR
P2 Sleep Quality	-	-	-	-	-	-	-	-	-	-
P1 Sleep Quality	0.66**	-	-	-	-	-	-	-	-	-
PSA	0.49**	0.53**	-	-	-	-	-	-	-	-
SRW	0.42**	0.50**	0.48**	-	-	-	-	-	-	-
BAS	0.42**	0.29**	0.35**	0.46**	-	-	-	-	-	-
SHI	0.32**	0.31**	0.48**	0.27**	0.29**	-	-	-	-	-
RTS	0.44**	0.40**	0.53**	0.36**	0.32**	0.45**	-	-	-	-
DERS	0.35**	0.38**	0.40**	0.25**	0.36**	0.26**	0.46**	-	-	-
PSS	0.47**	0.44**	0.42**	0.32**	0.36**	0.21**	0.40**	0.45**	-	-
PSR	0.37**	0.41**	0.43**	0.26**	0.27**	0.24**	0.53**	0.57**	0.61**	-

Note. * $p < .05$. ** $p < .01$. P2 = Part 2; P1 = Part 1; PSA = Pre-Sleep Arousal; SRW = Sleep-Related Worry; BAS = Beliefs and Attitudes about Sleep; SHI = Sleep Hygiene Index; RTS = Ruminative Thinking Style; DERS = Difficulties with Emotion Regulation Scale; PSS = Perceived Stress Scale; PSR = Perceived Stress Reactivity.

Table 5. Mediational Analysis of Perceived Stress, Perceived Stress Reactivity, Pre-Sleep Arousal, and Poor Sleep Quality at P2

Predictors	Effects	Outcomes	
		PSA	P2 Sleep Quality
	Total R^2	0.22**	0.33**
Perceived Stress			
	Direct	0.36**	0.11**
	Indirect via PSA		0.04
	Total		0.15
Perceived Stress Reactivity			
	Direct	0.34**	0.01
	Indirect via PSA		0.03**
	Total		0.04
Pre-Sleep Arousal			
	Direct/Total		0.10**
	Spurious		0.03

Note. * $p < .05$. ** $p < .01$. P2 = Part 2.

Table 6. Path Analysis of Perceived Stress, Pre-Sleep Arousal, and Poor Sleep Quality

Predictor	Effects	Outcomes	
		PSA	Sleep Quality
	Total R^2		0.37**
Covariates			
Age	Partial Effect		0.50**
Perceived Stress			
	Direct Effect	0.59**	0.12**
	Indirect Effect via PSA (CI = 0.03 – 0.09)		0.06**
	Total Effect		0.17**
Pre-Sleep Arousal (PSA)			
	Direct/Total Effect		0.09**

Note. * $p < .05$. ** $p < .01$. PSA = Pre-Sleep Arousal. Age was included as a covariate.

Table 7. Multiple Regression Analysis Examining the Moderating Effects of Perceived Stress with Beliefs and Attitudes about Sleep, Sleep Hygiene Behaviors, Ruminative Thinking Style, and Emotion Dysregulation on Pre-Sleep Arousal

	Pre-Sleep Arousal (PSA)			
	B	*SE*	*B*	ΔR^2
Beliefs & attitudes about sleep (BAS)			Model 1	
Step 1: Predictor variables				0.18**
Perceived stress	0.57	0.12	0.40**	
BAS	0.33	0.55	0.05	
Step 2: Two-way interaction				0.03
Perceived stress	0.57	0.12	0.40**	
BAS	0.43	0.55	0.07	
Perceived stress x BAS	-0.15	0.07	-0.17	
Sleep hygiene behaviors (SHI)			Model 2	
Step 1: Predictor variables				0.33**
Perceived stress	0.30	0.13	0.21*	
SHI	0.54	0.10	0.38**	
Step 2: Two-way interaction				0.00
Perceived stress	0.31	0.13	0.21*	
SHI	0.54	0.10	0.38**	
Perceived stress x SHI	0.00	0.01	0.02	
Ruminative thinking style (RTS)			Model 3	
Step 1: Predictor variables				0.33**
Perceived stress	0.35	0.11	0.24**	
RTS	0.16	α0.03	0.44**	
Step 2: Two-way interaction				0.00
Perceived stress	0.35	0.11	0.24**	
RTS	0.16	0.03	0.44**	

	Perceived stress x RTS	0.00	0.00	-0.05	
Emotion dysregulation (ED)				Model 4	
Step 1: Predictor variables					0.23**
	Perceived stress	0.43	0.12	0.30**	
	Emotion dysregulation	0.12	0.04	0.27**	
Step 2: Two-way interaction					0.00
	Perceived stress	0.43	0.12	0.30**	
	Emotion dysregulation	0.12	0.04	0.27**	
	Perceived stress x ED	0.00	0.01	0.00	

Note. * $p < .05$. ** $p < .01$. BAS = Beliefs and Attitudes about Sleep; ED = Emotion Dysregulation; PSA = Pre-Sleep Arousal; PSR = Perceived Stress Reactivity; RTS = Ruminative Thinking Style; SHI = Sleep Hygiene Index. Age was not included as a covariate due to lack of association with PSA.

Table 8. Multiple Regression Analysis Examining the Moderated Mediation Effects of Emotion Dysregulation

(a) Consequent					
Antecedent	Poor sleep quality (outcome)				
	B	*SE B*		*p*	
Perceived stress (predictor)	0.11***	0.03		0.00	
Age (covariate)	0.50***	0.15		0.00	
Emotion Dysregulation (moderator)	0.02	0.03		0.41	
Pre-sleep arousal (mediator)	0.13*	0.08		0.09	
Interaction (pre-sleep arousal x emotion dysregulation)	0.00	0.00		0.58	
b) Direct effect of perceived stress on poor sleep quality					
B	*SE B*	*t*	*p*	LLCI	ULCI
0.11***	0.03	3.37	0.00	0.043	0.166
(c) Conditional indirect effects of perceived stress on poor sleep quality at values of emotion dysregulation					
Emotion dysregulation (moderator)	Effect	Bootstrap SE		BootLLCI	BootULCI
56.00	0.06	0.02		0.022	0.114
78.00	0.06	0.02		0.027	0.092
104.12	0.05	0.02		0.015	0.082

Note. * $p < .10$. ** $p < .05$. *** $p < .01$. LLCI = Lower limit confidence interval. ULCI = Upper limit confidence interval.

Table 9. Multiple Regression Analysis Examining the Moderated Mediation Effects of Sleep-Related Worry

(a) Consequent					
Antecedent	Poor sleep quality (outcome)				
	B	*SE B*		*p*	
Perceived stress (predictor)	0.10***	0.03		0.00	
Age (covariate)	0.49	0.15		0.00	
Sleep-related worry (moderator)	0.11	0.08		0.16	
Pre-sleep arousal (mediator)	0.09*	0.05		0.07	
Interaction (pre-sleep arousal x sleep-related worry)	0.00	0.00		0.65	
b) Direct effect of perceived stress on poor sleep quality					
B	*SE B*	*t*	*p*	LLCI	ULCI
0.10***	0.03	3.52	0.00	0.045	0.161
(c) Conditional indirect effects of perceived stress on poor sleep quality at values of pre-sleep arousal					
Sleep-related worry (moderator)	Effect	Bootstrap SE		BootLLCI	BootULCI
7.00	0.05	0.02		0.013	0.104
14.00	0.05	0.02		0.019	0.079
22.00	0.04	0.02		0.008	0.069

Note. * $p < .10$. ** $p < .05$. *** $p < .01$. LLCI = Lower limit confidence interval. ULCI = Upper limit confidence interval.

APPENDIX B: FIGURES

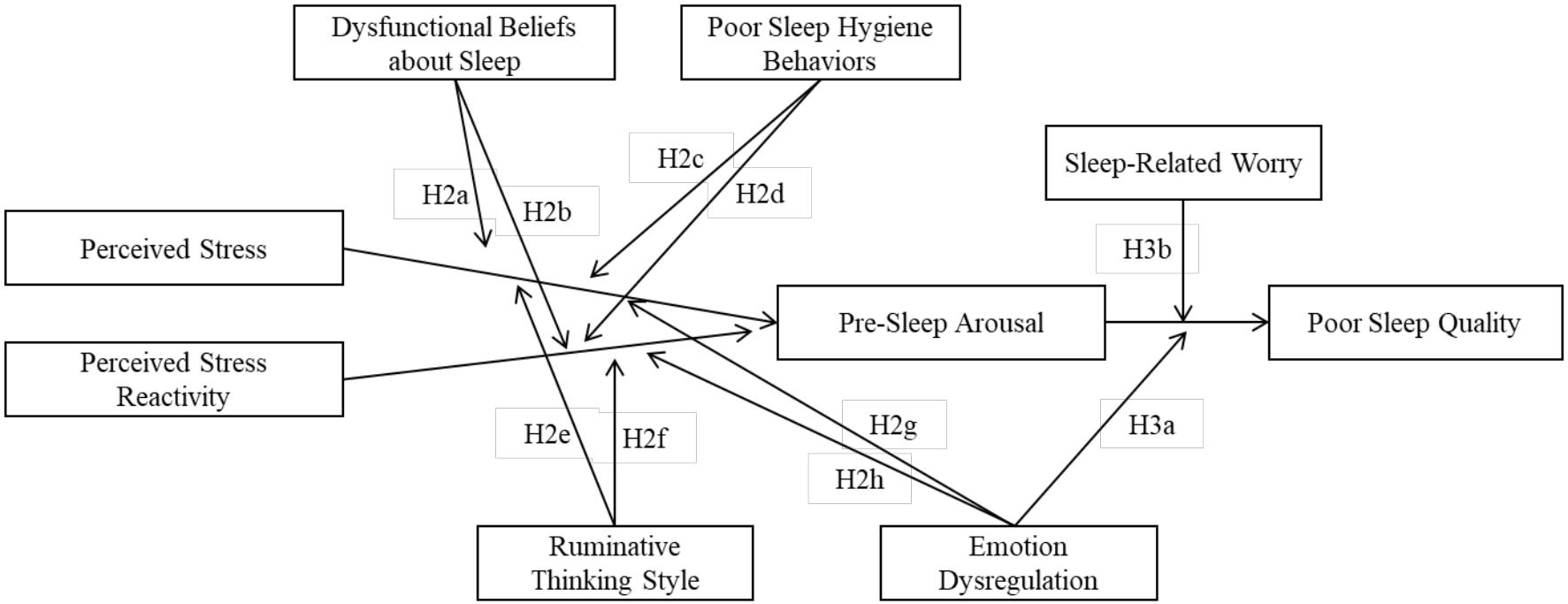

Figure 1. Conceptual model for the contribution of pre-sleep arousal and perpetuating cognitive-emotional-behavioral factors to the relationship between perceived stress, perceived stress reactivity, and poor sleep quality.

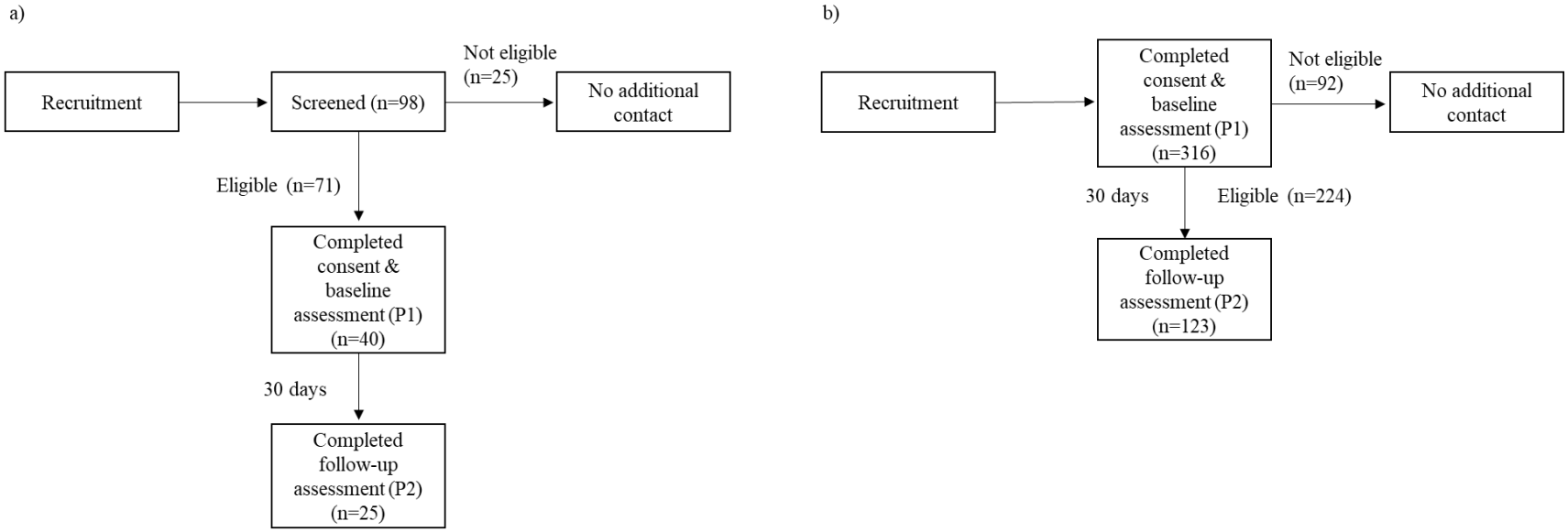

Figure 2. Flow diagram of study protocol for a) original and b) revised measurement schedules. Note. P1 = Part 1; P2 = Part 2.

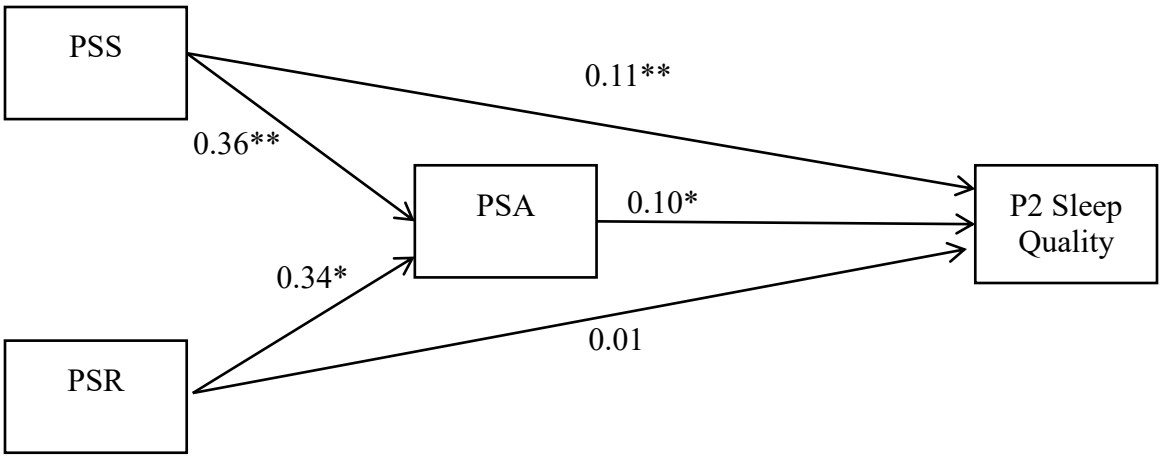

Figure 3. Original path analysis model for Hypothesis 1, in which pre-sleep arousal mediated the relationship between two predictors, perceived stress and perceived stress reactivity, and poor sleep quality 30 days later. Note. P2 = Part 2; PSA = Pre-Sleep Arousal; PSR = Perceived Stress Reactivity; PSS = Perceived Stress Scale.

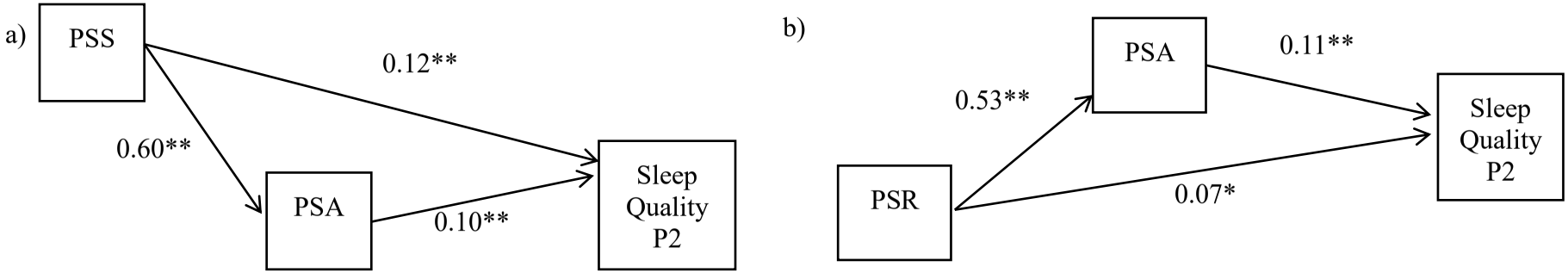

Figure 4. Alternative path analysis models for Hypothesis 1, in which a) pre-sleep arousal mediated the relationship perceived stress and poor sleep quality 30 days later and b) pre-sleep arousal mediated the relationship between perceived stress reactivity and poor sleep quality 30 days later. Note. P2 = Part 2; PSA = Pre-Sleep Arousal; PSR = Perceived Stress Reactivity; PSS = Perceived Stress Scale.

APPENDIX C: MEASURES

I. ELIGIBILITY SCREEN

DEMOGRAPHIC INFORMATION

What is your gender?	
Male	0
Female	1
Trans-	2

If female, are you currently pregnant?	
No	0
Yes	1

What is your race?	
American Indian or Alaska Native	0
Asian	1
Black/African American	2
Native Hawaiian or Other Pacific Islander	3
White	4
More than one race (write in all that apply)	5
Other	[String Variable]

How do you identify yourself?	
Hispanic/Latino	0
Not Hispanic/Latino	1

What is your current academic class standing (based on number of credit hours)?	
Freshman	0
Sophomore	1
Junior	2
Senior	3
Post-Baccalaureate	4
Graduate	5

Are you enrolled as a full-time or part-time student?	
Part-time (less than 12 credit hours/semester)	0

Full-time (12 or more credit hours/semester)	1

What is your current employment status?	
Unemployed/Not working	0
Full-time employed	1
Part-time employed	2
Other (please describe; i.e., 2 jobs, during the summer, etc.)	3
If employed, what is your position?	[String Variable]

What is your marital status?	
Married / Civil Union	0
Single	1
Divorced	2
Live-in-partner	3
Widowed	4

Are you currently living	
In an apartment on campus?	0
In an apartment or house off campus?	1
With your parents?	2

What is your MOTHER's highest level of education?	
Some high school / No GED	0
High school diploma / GED	1
Some college (less than 4 years)	2
Bachelor's degree	3
Graduate/Professional degree (Masters, Doctorate, etc.)	4

MEDICAL HEALTH HISTORY

Has a medical doctor or health professional (i.e., nurse practitioner, physician assistant) ever diagnosed you as having any of the following conditions? Choose all that apply.		
(Diagnosis)	(No)	(Yes)
Asthma	0	1
Diabetes (type 2)	0	1
Diabetes (type 1)	0	1
High blood pressure	0	1
Heart problems	0	1
Chronic pain	0	1
GERD	0	1
Thyroid disease	0	1
Epilepsy	0	1
Any medical condition not listed (i.e., autoimmune disorder, cancer, etc.)?	0	1
Please describe:	[String Variable]	

HISTORY OF SLEEP DIFFICULTIES AND TREATMENT

Has a medical doctor or health professional (i.e., nurse practitioner, physician assistant) ever diagnosed you as having any of the following conditions? Choose all that apply.		
(Diagnosis)	(No)	(Yes)
Insomnia	0	1
Sleep apnea	0	1
Restless leg syndrome	0	1
Periodic limb movement disorder	0	1
Another sleep disorder not listed?	0	1
Please describe:	[String Variable]	
What treatment were you given? Please describe:	[String Variable]	

STOP – Bang

	Yes (1)	*No (0)*
1. Do you snore loudly (loud enough to be heard through closed doors)?	1	0
2. Do you often feel tired, fatigued, or sleepy during the daytime?	1	0
3. Has anyone observed you stop breathing during your sleep?	1	0
4. Do you have or are you being treated for high blood pressure?	1	0
5. Is your BMI more than 35?	1	0
a) What is your height?	[String variable]	
b) What is your weight?	[String variable]	
6. Is your age over 50 years old?	1	0
7. Is your neck circumference greater than 40 cm?	1	0
8. Is your gender male?	1	0

CURRENT MEDICATIONS

Are you CURRENTLY using any of the following treatment/medications to help you sleep or to help with anxiety or depression?			
(Medication/ Treatment)	*(No - Never)*	*(Yes - CURRENT)*	*(Yes – In the past / In the past six months)*
Cognitive Behavioral Therapy for Insomnia	0	1	2
Continuous Positive Airway Pressure (CPAP)	0	1	2
Ambien / Zolpidem	0	1	2
Lunesta / Eszopliclone	0	1	2
Melatonin	0	1	2
Rozerem / Remalteon	0	1	2
Desryl / Trazadone	0	1	2
Elavil / Amitriptyline	0	1	2
Dalmane / Flurazepam	0	1	2
Klonopin / Clonazepam	0	1	2
Doral / Quazepam	0	1	2
Ativan / Lorazepam	0	1	2
Xanax / Alprazolam	0	1	2
Restoril / Temazepam	0	1	2
Halcion / Triazolam	0	1	2
Benadryl / Diphenhydramine	0	1	2
Any treatment or medication not listed?	0	1	2
Please describe:	[String Variable]		

MENTAL HEALTH HISTORY

Has a psychologist or a mental health professional (i.e., counselor or psychiatrist) ever diagnosed you as having any of the following conditions? Choose all that apply.			
(Diagnosis)	(No)	(Yes - CURRENT)	(Yes – In the past)
Anxiety Disorder (panic disorder, obsessive compulsive disorder, generalized anxiety)	0	1	2
Schizophrenia or another psychotic disorder	0	1	2
Depression	0	1	2
Bipolar disorder	0	1	2
Posttraumatic stress disorder (PTSD)	0	1	2
Alcohol abuse/dependence	0	1	2
Substance abuse/dependence (i.e., illicit drugs, prescription medication, etc.)	0	1	2
Any other mental health condition not listed?	0	1	2
Please describe:	[String Variable]		

Primary Care – Post-Traumatic Stress Disorder Checklist (PC-PTSD)

In your life, have you ever had any experience that was so frightening, horrible or upsetting that, IN THE PAST MONTH, you:		
	(No)	(Yes)
1. Have had nightmares about it or thought about it when you did not want to?	0	1
2. Tried hard not to think about it or went out of your way to avoid situations that reminded you of it?	0	1
3. Were constantly on guard, watchful, or easily startled?	0	1
4. Felt numb or detached from others, activities, or your surroundings?	0	1

II. P1 Measures

Pittsburgh Sleep Quality Index (PSQI)

The following instructions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.				
1. During the past month, what time have you usually gone to bed at night?				[String Variable]
2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night?				[String Variable]
3. During the past month, what time have you usually gotten up in the morning?				[String Variable]
4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spend in bed.)				[String Variable]
5. During the past month, how often have you had trouble sleeping because you…	*Not during the past month (0)*	*Less than once a week (1)*	*Once or twice a week (2)*	*Three or more times a week (3)*
a. Cannot get to sleep within 30 minutes	0	1	2	3
b. Wake up in the middle of the night or early morning	0	1	2	3
c. Have to get up to use the bathroom	0	1	2	3
d. Cannot breathe comfortably	0	1	2	3
e. Cough or snore loudly	0	1	2	3
f. Feel too cold	0	1	2	3
g. Feel too hot	0	1	2	3
h. Had bad dreams	0	1	2	3
i. Have pain	0	1	2	3
	Not during the past month (0)	*Less than once a week (1)*	*Once or twice a week (2)*	*Three or more times a week (3)*
j. Other reason(s), please describe and check how often you have had trouble sleeping because of each reason: Reason 1: [String Variable]	0	1	2	3
Reason 2: [String Variable]	0	1	2	3
6. During the past month, how often have you taken medicine to help you	0	1	2	3

sleep (prescribed or "over the counter")?				
7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?	0	1	2	3
8. During the past month, how would you rate your sleep quality overall?	0	1	2	3
9. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?	*No problem at all (0)*	*Only a very slight problem (1)*	*Somewhat of a problem (2)*	*A very big problem (3)*
Do you have a partner or roommate?	*No bed partner or room-mate (0)*	*Partner or Room-mate in other room (1)*	*Partner or roommate in same room, but not same bed (2)*	*Partner in same bed (3)*

Perceived Stress Scale (PSS)

The questions in this scale as you about your feelings and thoughts during the last month. In each case, you will be asked to indicate by circling how often you felt or thought a certain way					
	Never (0)	*Almost never (1)*	*Some-times (2)*	*Fairly often (3)*	*Very often (4)*
1. In the last month, how often have you been upset because of something that happened unexpectedly?	0	1	2	3	4
2. In the last month, how often have you felt that you were unable to control the important things in your life?	0	1	2	3	4
3. In the last month, how often have you felt nervous and "stressed"?	0	1	2	3	4
4. In the last month, how often have you felt confident about your ability to handle your personal problems?	0	1	2	3	4
5. In the last month, how often have you felt that things were going your way?	0	1	2	3	4
6. In the last month, how often have you found that you could not cope with all the things that you had to do?	0	1	2	3	4
7. In the last month, how often have you been able to control irritations in your life?	0	1	2	3	4
8. In the last month, how often have you felt that you were on top of things?	0	1	2	3	4
9. In the last month, how often have you been angered because of things that were outside your control?	0	1	2	3	4
10. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?	0	1	2	3	4

Perceived Stress Reactivity Scale (PSRS)

	(0)	*(1)*	*(2)*
1. When tasks and duties build up to the extent that they are hard to manage…	I am generally untroubled (0)	I usually feel a little uneasy (1)	I normally get quite nervous (2)
2. When I want to relax after a hard day at work…	This is usually going to be quite difficult for me (0)	I usually succeed (1)	I generally have no problem at all (2)
3. When I have conflicts with others that may not be immediately resolved…	I generally shrug it off (0)	It usually affects me a little (1)	It usually affects me a lot (2)
4. When I make a mistake…	In general, I feel confident (0)	I sometimes feel unsure about my abilities (1)	I often have doubts about my abilities (2)
5. When I'm wrongly criticized by others…	I am normally annoyed for a long time (0)	I am annoyed for just a short time (1)	In general, I am hardly annoyed at all (2)
6. When I argue with other people…	I usually calm down quickly (0)	I usually stay upset for some time (1)	It usually takes me a long time until I calm down (2)
7. When I have little time for a job to be done…	I usually stay calm (0)	I usually feel uneasy (1)	I usually get quite agitated (2)
8. When I make a mistake…	I am normally annoyed for quite a long time (0)	I am normally annoyed for quite a while (1)	I generally get over it easily (2)
9. When I am unsure what to do or say in a social situation…	I generally stay cool (0)	I often feel warm (1)	I often begin to sweat (2)
10. When I have spare time after working hard…	It is often difficult for me to unwind and relax (0)	I usually need some time to unwind properly (1)	I am usually able to unwind effectively and forget about the problems of the day (2)

11. When I am criticized by others…	Important arguments come to my mind when it is too late to still make my point (0)	I often have difficulty finding a good reply (1)	I usually think of a reply to defend myself (2)
12. When something does not go the way I expected…	I usually stay calm (0)	I often get uneasy (1)	I usually get very agitated (2)
13. When I do not attain a goal…	I usually remain annoyed for a long time (0)	I am usually disappointed but recover soon (1)	In general, I am hardly concerned at all (2)
14. When others criticize me…	I generally don't lose confidence at all (0)	I generally lose a little confidence (1)	I generally feel very unconfident (2)
15. When I fail at something…	I usually find it hard to accept (0)	I usually accept it to some degree (1)	In general, I hardly think about it (2)
16. When there are too many demands on me at the same time…	I generally stay calm and do one thing after the other (0)	I usually get uneasy (1)	Usually, even minor interruptions irritate me (2)
17. When others say something incorrect about me…	I usually get quite upset (0)	I normally get a little bit upset (1)	In general, I shrug it off (2)
18. When I fail at a task…	I usually feel very uncomfortable (0)	I usually feel somewhat uncomfortable (1)	In general, I don't mind (2)
19. When I argue with others…	I usually get very upset (0)	I usually get a little bit upset (1)	I usually don't get upset (2)
20. When I am under stress…	I usually can't enjoy my leisure time at all (0)	I usually have difficulty enjoying my leisure time (1)	I usually enjoy my leisure time (2)
21. When tasks and duties accumulate to the extent that they are hard to cope with…	My sleep is unaffected (0)	My sleep is slightly disturbed (1)	My sleep is very disturbed (2)
22. When I have to speak in front of other people…	I often get very nervous (0)	I often get somewhat nervous (1)	In general, I stay calm (2)

23. When I have many task and duties to fulfill…	In general, I stay calm (0)	I usually get impatient (1)	I often get irritable (2)

Pre-Sleep Arousal Scale (PSAS)

Please describe how intensely you generally experience each of these symptoms as you attempt to fall asleep in your own bedroom.					
	Not at all (1)	*Slightly (2)*	*Moderately (3)*	*A lot (4)*	*Extremely (5)*
1. Heart racing, pounding, or beating irregularly.	1	2	3	4	5
2. A jittery, nervous feeling in your body.	1	2	3	4	5
3. Shortness of breath or labored breathing.	1	2	3	4	5
4. A tight, tense feeling in your muscles.	1	2	3	4	5
5. Cold feeling in your hands, feet or your body	1	2	3	4	5
6. Have stomach upset (knot or nervous feeling, heartburn, nausea, etc.)	1	2	3	4	5
7. Perspiration in the palms of your hands or other parts of your body.	1	2	3	4	5
8. Dry feeling in your mouth or throat.	1	2	3	4	5
9. Worry about falling asleep.	1	2	3	4	5
10. Review or ponder events of the day.	1	2	3	4	5
11. Depressing or anxious thoughts.	1	2	3	4	5
12. Worry about problems other than sleep.	1	2	3	4	5
13. Being mentally alert, active.	1	2	3	4	5
14. Can’t shut off your thoughts.	1	2	3	4	5
15. Thoughts keep racing through your head.	1	2	3	4	5
16. Being distracted by sounds, noise in the environment (e.g., ticking of the clock, house noises, traffic).	1	2	3	4	5

Dysfunctional Beliefs and Attitudes about Sleep Scale (BAS)

Several statements reflecting people's beliefs and attitudes about sleep are listed below. Please indicate to what extent to you personally agree or disagree with each statement. There is no right or wrong answer. For each statement, circle the number that corresponds to your own personal belief. Please respond to all items even though some may not apply directly to your own situation.											
	Strongly Disagree	*0*	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10* *Strongly Agree*
1. I need 8 hours of sleep to feel refreshed and function well during the day.	0	1	2	3	4	5	6	7	8	9	10	
2. When I do not get the proper amount of sleep on a given night, I need to catch up on the next day by napping or on the next night by sleeping longer.	0	1	2	3	4	5	6	7	8	9	10	
3. Because I am getting older, I need less sleep.	0	1	2	3	4	5	6	7	8	9	10	
4. I am worried that if I go one or two nights without sleep, I may have a nervous breakdown.	0	1	2	3	4	5	6	7	8	9	10	
5. I am concerned that chronic insomnia may have serious consequences for my physical health.	0	1	2	3	4	5	6	7	8	9	10	
6. By spending more time in bed, I usually get more sleep and feel better the next day.	0	1	2	3	4	5	6	7	8	9	10	
7. When I have trouble getting to sleep, I should stay in bed and try harder.	0	1	2	3	4	5	6	7	8	9	10	
8. I am worried that I may lose control over my abilities to sleep.	0	1	2	3	4	5	6	7	8	9	10	
9. Because I am getting older, I should go to bed earlier in the evening.	0	1	2	3	4	5	6	7	8	9	10	
10. After a poor night's sleep, I know that it will interfere with my daily activities on the next day.	0	1	2	3	4	5	6	7	8	9	10	

11. In order to be alert and function well during the day, I am better off taking a sleeping pill rather than having a poor night's sleep.	0	1	2	3	4	5	6	7	8	9	10
12. When I feel irritable, depressed, or anxious during the day, it is mostly because I did not sleep well the night before.	0	1	2	3	4	5	6	7	8	9	10
13. Because my bed partner falls asleep as soon as his or her head hits the pillow and stays asleep through the night, I should be able to do so too.	0	1	2	3	4	5	6	7	8	9	10
14. I feel that insomnia is basically the result of aging, and there isn't much that can be done about the problem.	0	1	2	3	4	5	6	7	8	9	10
15. I am sometimes afraid of dying in my sleep.	0	1	2	3	4	5	6	7	8	9	10
16. When I have a good night's sleep, I know that I will have to pay for it the following night.	0	1	2	3	4	5	6	7	8	9	10
17. When I sleep poorly on one night, I know that it will disturb my sleep schedule for the whole week.	0	1	2	3	4	5	6	7	8	9	10
18. Without an adequate night's sleep, I can hardly function the next day.	0	1	2	3	4	5	6	7	8	9	10
19. I can't ever predict whether I will have a good or poor night's sleep.	0	1	2	3	4	5	6	7	8	9	10
20. I have little ability to manage the consequences of disturbed sleep.	0	1	2	3	4	5	6	7	8	9	10
21. When I feel tired, have no energy, or just seem not to function well during the day, it is generally because	0	1	2	3	4	5	6	7	8	9	10

I did not sleep well the night before.											
22. I get overwhelmed by my thoughts at night and often feel I have no control over my racing mind.	0	1	2	3	4	5	6	7	8	9	10
23. I feel I can still lead a satisfactory life despite sleep difficulties.	0	1	2	3	4	5	6	7	8	9	10
24. I believe that insomnia is essentially a result of a chemical imbalance.	0	1	2	3	4	5	6	7	8	9	10
25. I feel that insomnia is ruining my ability to enjoy life and prevents me from doing what I want.	0	1	2	3	4	5	6	7	8	9	10
26. I avoid or cancel obligations (social, family, occupational) after a poor night's sleep.	0	1	2	3	4	5	6	7	8	9	10
27. A "nightcap" before bedtime is a good solution for sleep problems.	0	1	2	3	4	5	6	7	8	9	10
28. Medication is probably the only solution to sleeplessness.	0	1	2	3	4	5	6	7	8	9	10
29. My sleep is getting worse all the time and I don't believe anyone can help.	0	1	2	3	4	5	6	7	8	9	10
30. It usually shows in my general appearance if I haven't slept well.	0	1	2	3	4	5	6	7	8	9	10

Sleep Hygiene Index (SHI)

The following questions list a series of 13 behaviors. For each one, choose how often it applies to you.					
	Never (0)	*Rarely (1)*	*Some-times (2)*	*Frequent -ly (3)*	*Always (4)*
1. I take daytime naps lasting two or more hours.	0	1	2	3	4
2. I go to bed at different times from day to day.	0	1	2	3	4
3. I get out of bed at different times from day to day.	0	1	2	3	4
4. I exercise to the point of sweating within 1 hour of going to bed.	0	1	2	3	4
5. I stay in bed longer than I should two or three times a week.	0	1	2	3	4
6. I use alcohol, tobacco, or caffeine within 4 hours of going to bed or after going to bed.	0	1	2	3	4
7. I do something that may wake me up before bedtimes (for example: play video games, use the internet, or clean).	0	1	2	3	4
8. I go to bed feeling stressed, angry, upset, or nervous.	0	1	2	3	4
9. I use my bed for things other than sleeping or sex (for example: watch television, read, eat, or study).	0	1	2	3	4
10. I sleep on an uncomfortable bed (for example: poor mattress or pillow, too much or not enough blankets).	0	1	2	3	4
11. I sleep in an uncomfortable bedroom (for example: too bright, too	0	1	2	3	4

stuffy, too hot, too cold, or too noisy).					
12. I do important work before bedtime (for example: pay bills, schedule, or study).	0	1	2	3	4
13. I think, plan, or worry when I am in bed.	0	1	2	3	4

Ruminative Thinking Style Scale (RTS)

For each of the items below, please rate how well the item describes you.							
	Not at all (1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*	*Very well (7)*
1. I find that my mind often goes over things again and again	1	2	3	4	5	6	7
2. When I have a problem, it will gnaw on my mind for a long time	1	2	3	4	5	6	7
3. I find that some thoughts come to mind over and over throughout the day	1	2	3	4	5	6	7
4. I can't stop thinking about some things	1	2	3	4	5	6	7
5. When I am anticipating an interaction, I will imagine every possible scenario and conversation	1	2	3	4	5	6	7
6. I tend to replay past events as I would have like them to happen	1	2	3	4	5	6	7
7. I find myself daydreaming about things I wish I had done	1	2	3	4	5	6	7
8. When I feel I have had a bad interaction with someone, I tend to imagine various scenarios where I would have acted differently	1	2	3	4	5	6	7
9. When trying to solve a complicated problem, I find that I just keep coming back to the beginning without ever finding a solution	1	2	3	4	5	6	7
10. If there is an important event coming up, I think about it so much that I work myself up	1	2	3	4	5	6	7
11. I have never been able to distract myself from unwanted thoughts	1	2	3	4	5	6	7

12. Even if I think about a problem for hours, I still have a hard time coming to a clear understanding	1	2	3	4	5	6	7
13. It is very difficult for me to come to a clear conclusion about some problems, no matter how much I think about it	1	2	3	4	5	6	7
14. Sometimes I realize I have been sitting and thinking about something for hours	1	2	3	4	5	6	7
15. When I am trying to work out a problem, it is like I have a long debate in my mind where I keep going over different points	1	2	3	4	5	6	7
16. I like to sit and reminisce about pleasant events from the past	1	2	3	4	5	6	7
17. When I am looking forward to an exciting event, thoughts of it interfere with what I am working on	1	2	3	4	5	6	7
18. Sometimes even during a conversation, I find unrelated thoughts popping into my head	1	2	3	4	5	6	7
19. When I have an important conversation coming up, I tend to go over it in my mind again and again	1	2	3	4	5	6	7
20. If I have an important event coming up, I can't stop thinking about it	1	2	3	4	5	6	7

Difficulties in Emotion Regulation Scale (DERS)

Please indicate how often the following 36 statements apply to you by writing the appropriate number from the scale of 1 to 5 alongside each item.					
	Almost Never (0 -10%)	*Sometimes (11-35%)*	*About half the time (36-65%)*	*Most of the time (66-90%)*	*Almost always (91-100%)*
1. I am clear about my feelings	0	1	2	3	4
2. I pay attention to how I feel	0	1	2	3	4
3. I experience my emotions as overwhelming and out of control	0	1	2	3	4
4. I have no idea how I am feelings	0	1	2	3	4
5. I have difficulty making sense out of my feelings	0	1	2	3	4
6. I am attentive to my feelings	0	1	2	3	4
7. I know exactly how I am feeling	0	1	2	3	4
8. I care about what I am feeling	0	1	2	3	4
9. I am confused about how I feel	0	1	2	3	4
10. When I'm upset, I acknowledge my emotions	0	1	2	3	4
11. When I'm upset, I become angry with myself for feeling that way	0	1	2	3	4
12. When I'm upset, I become embarrassed for feeling that way	0	1	2	3	4
13. When I'm upset, I have difficulty getting work done	0	1	2	3	4
14. When I'm upset, I become out of control	0	1	2	3	4
15. When I'm upset, I believe that I will remain that way for a long time	0	1	2	3	4

16. When I'm upset, I believe that I will remain that way for a long time	0	1	2	3	4
17. When I'm upset, I believe that my feelings are valid and important	0	1	2	3	4
18. When I'm upset, I have difficulty focusing on other things	0	1	2	3	4
19. When I'm upset, I feel out of control	0	1	2	3	4
20. When I'm upset, I can still get things done	0	1	2	3	4
21. When I'm upset, I feel ashamed with myself for feeling that way	0	1	2	3	4
22. When I'm upset, I know that I can find a way to eventually feel better	0	1	2	3	4
23. When I'm upset, I feel like I am weak	0	1	2	3	4
24. When I'm upset, I feel like I can remain in control of my behaviors	0	1	2	3	4
25. When I'm upset, I feel guilty for feeling that way	0	1	2	3	4
26. When I'm upset, I have difficulty concentrating	0	1	2	3	4
27. When I'm upset, I have difficulty controlling my behaviors	0	1	2	3	4
28. When I'm upset, I believe that there is nothing I can do to make myself feel better	0	1	2	3	4
29. When I'm upset, I become irritated with myself for feeling that way	0	1	2	3	4
30. When I'm upset, I start to feel very bad about myself	0	1	2	3	4
31. When I'm upset, I believe that wallowing in it is all I can do	0	1	2	3	4

32. When I'm upset, I lose control over my behaviors	0	1	2	3	4
33. When I'm upset, I have difficulties thinking about anything else	0	1	2	3	4
34. When I'm upset, I take time to figure out what I'm really feeling	0	1	2	3	4
35. When I'm upset, it takes me a long time to feel better	0	1	2	3	4
36. When I'm upset, my emotions feel overwhelming	0	1	2	3	4

Sleep-Related Worry Scale (SRW)

Please rate the degree to which you agree or disagree with each of these statements.					
	Totally disagree (1)	*(2)*	*(3)*	*(4)*	*Totally agree (5)*
1. I worry about my sleep when I go to bed.	1	2	3	4	5
2. I worry about my sleep when I cannot fall asleep.	1	2	3	4	5
3. I worry about my sleep when I am awake at night.	1	2	3	4	5
4. I worry that my body will be harmed if I sleep poorly.	1	2	3	4	5
5. Because of my poor sleep, I worry that I will get sick more easily.	1	2	3	4	5
6. Because of my poor sleep, I worry that there is something wrong in my body.	1	2	3	4	5

III. P2 Measures

Pittsburgh Sleep Quality Index (PSQI)

The following instructions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.				
1. During the past month, what time have you usually gone to bed at night?				[String Variable]
2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night?				[String Variable]
3. During the past month, what time have you usually gotten up in the morning?				[String Variable]
4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spend in bed.)				[String Variable]
5. During the past month, how often have you had trouble sleeping because you…	*Not during the past month (0)*	*Less than once a week (1)*	*Once or twice a week (2)*	*Three or more times a week (3)*
a. Cannot get to sleep within 30 minutes	0	1	2	3
b. Wake up in the middle of the night or early morning	0	1	2	3
c. Have to get up to use the bathroom	0	1	2	3
d. Cannot breathe comfortably	0	1	2	3
e. Cough or snore loudly	0	1	2	3
f. Feel too cold	0	1	2	3
g. Feel too hot	0	1	2	3
h. Had bad dreams	0	1	2	3
i. Have pain	0	1	2	3
	Not during the past month (0)	*Less than once a week (1)*	*Once or twice a week (2)*	*Three or more times a week (3)*
j. Other reason(s), please describe and check how often you have had trouble sleeping because of each reason: Reason 1: [String Variable]	0	1	2	3
Reason 2: [String Variable]	0	1	2	3
6. During the past month, how often have you taken medicine to help you	0	1	2	3

sleep (prescribed or "over the counter")?				
7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?	0	1	2	3
8. During the past month, how would you rate your sleep quality overall?	0	1	2	3
9. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?	*No problem at all (0)*	*Only a very slight problem (1)*	*Somewhat of a problem (2)*	*A very big problem (3)*
Do you have a partner or roommate?	*No bed partner or room-mate (0)*	*Partner or Room-mate in other room (1)*	*Partner or roommate in same room, but not same bed (2)*	*Partner in same bed (3)*

Primary Care – Post-Traumatic Stress Disorder Checklist (PC-PTSD)

In your life, have you ever had any experience that was so frightening, horrible or upsetting that, IN THE PAST MONTH, you:		
	(No)	(Yes)
1. Have had nightmares about it or thought about it when you did not want to?	0	1
2. Tried hard not to think about it or went out of your way to avoid situations that reminded you of it?	0	1
3. Were constantly on guard, watchful, or easily startled?	0	1
4. Felt numb or detached from others, activities, or your surroundings?	0	1