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EXAMINING SLEEP AS A MODERATOR OF PHYSIOLOGICAL RESPONSE TO STRESS  
AMONG MIGRAINEURS

A Dissertation Defense  
presented in partial fulfillment of requirements  
for the degree of Doctor of Philosophy  
in the Department of Psychology  
The University of Mississippi

by

VANESSA LEE MOYNAHAN

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## ABSTRACT

Stress and sleep disturbance are among the most frequently reported triggers of migraine. Several studies have found migraineurs have heightened physiological response and decreased rates of habituation to stressors compared to those without headache. A smaller body of literature suggests that poor sleep and high stress can interact to influence migraine, though a larger literature across chronic pain populations and the general population also supports an interactive effect between stress and sleep. No study to date has examined the extent to which sleep disturbance moderates physiological response to stress among migraineurs, the findings of which may help to inform understanding of migraine mechanisms and treatment development strategies. The present study sought to experimentally examine the effect of sleep quantity and quality on cardiovascular reactivity to a repeated stressor (i.e., speech task) and to determine if an observed stress-sleep relationship varies as a function of headache diagnosis (i.e., migraine vs. non-headache). Results indicated that participants with migraine and those without headache had different systolic blood pressure in response to stress between timepoints depending on their quantity of sleep ( $F(2, 130) = 4.742, p = .010, R\text{-squared change} = .068$ ), indicating a significant interaction effect. However, the nature of the interaction was different from expected, as there was an inverse relationship between sleep quantity and SBP during the initial stressor task among participants without headache, while no such relationship existed for migraineurs. Rather, migraineurs' SBP during the initial stressor task was similar regardless of their sleep quantity. Interactive effects were not found for other physiological measures or for sleep quality.

Results suggest that sleep may not be a protective factor against heightened stress response among migraineurs. Future studies are needed to further examine relationships between these factors and possible mechanisms behind any interaction effects.

## LIST OF ABBREVIATIONS

CM	Chronic Migraine
EM	Episodic Migraine
TTH	Tension-type Headache
ETTH	Episodic Tension-type Headache
CTTH	Chronic Tension-type Headache
ICHD	International Classification of Headache Disorders
TSST	Trier Social Stress Test
SBP	Systolic Blood Pressure
DBP	Diastolic Blood Pressure
HR	Heart Rate
PHQ-9	Patient Health Questionnaire-9
GAD-7	General Anxiety Disorder-7
PSQI	Pittsburgh Sleep Quality Index
STAI	State Trait Anxiety Inventory
PSS-10	Perceived Stress Scale-10
SDIH-3	Structured Diagnostic Interview for Headache-3

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## I. INTRODUCTION

### **Migraine: Definition and Impact**

The International Classification of Headache Disorders (ICHD-3; International Headache Society, 2013) differentiates the diagnostic criteria for all headache disorders, including the most common primary headache disorders of migraine and tension-type headache (TTH). Migraine pain is characterized by a unilateral location, pulsating quality, moderate or severe intensity, and/or causing interference with routine physical activity. Headache attacks last 4-72 hours and may be accompanied by nausea or vomiting or both photophobia and phonophobia. While a diagnosis of episodic migraine (EM) requires at least five lifetime attacks, a diagnosis of chronic migraine (CM) requires at least 15 days with headache per month for a period of several months.

Migraine is a common neurological disorder that can be very disabling, with a 12% lifetime prevalence in the U.S (Lipton et al., 2007). Migraine is three times more common among women than men (18% vs. 6%, respectively; Lipton et al., 2007; Lipton, Bigal, Hamelsky, & Scher, 2008). Although migraine can affect people throughout their lives, peak prevalence is between the ages of 30-39 among both men and women, with lowest occurrence in adults aged 60 or over. Research has consistently confirmed that individuals with migraine experience many negative consequences as a result of the disorder, including reduced quality of life (Lipton, Hamelsky, Kolodner, Steiner, & Stewart, 2000; Terwindt et al., 2000), reduced work performance (Von Korff, Stewart, Simon, & Lipton, 1998), reduced school functioning (Smitherman, McDermott, & Buchanan, 2011), frequent medical visits (Edmeads et al., 1993), and negative impact on family relationships (Smith, 1996).

## **Migraine Comorbidities and Triggers**

Extensive literature supports a greater-than-chance association between migraine and psychiatric disorders, including major depressive disorder, panic disorder, phobias, and bipolar disorder (Baskin, Lipchik, & Smitherman, 2006; Baskin & Smitherman, 2009; Jette, Patten, Williams, Becker, & Wiebe, 2008; Lipton, Hamelsky, Kolodner, Steiner, & Stewart, 2000; Ratcliffe, Enns, Jacobi, Belik, & Sareen, 2009). Other common comorbid disorders include generalized anxiety disorder and obsessive-compulsive disorder, although prevalence of these disorders among migraineurs varies between studies (Breslau, Davis, & Andreski, 1991; Swartz, Pratt, Armenian, Lee, Eaton, 2000; Jette et al., 2008; Ratcliffe et al., 2009). Studies comparing EM and CM have found that rates of psychiatric disorders, including depression, anxiety, and bipolar disorder, are higher among individuals with CM (Antonaci et al., 2011; Buse, Manack, Serrano, Turkel, & Lipton, 2010). Migraineurs are also at increased risk for sleep disorders, especially insomnia (Rains & Poceta, 2006).

Triggers, or precipitating factors of migraine, are reported by the majority of migraineurs. Kelman (2007) found that 76% of migraineurs claimed that they could identify specific triggers for their migraine attacks; this proportion rose to 95% when participants were asked to select from a list of triggers. A meta-analysis of headache triggers examined results from 85 articles and over 27 thousand participants and found the two most commonly endorsed headache triggers to be stress (58%) and sleep (41%; Pellegrino, Davis-Martin, Houle, Turner, & Smitherman, 2018). Another literature review by Peroutka (2014) identified the most commonly reported migraine triggers, among them stress, auditory stimuli, sleep disturbance (lack of sleep, excess sleep), missing meals/fasting, and menstruation. Across studies, stress is the most common

perceived migraine trigger, with 58-84% of migraineurs endorsing it as such (Pellegrino et al., 2018; Martin, 2010; Peroutka).

## **Stress**

The National Institute of Mental Health (NIMH) defines stress as the brain's response to any demand (NIMH, 2016). Similarly, McEwen and Gianaros (2011) cite the brain as the central organ of stress processes; specifically, the brain determines which experiences are stressful, dictates how individuals will cope, and changes in both function and structure due to stressful experiences. Stimuli perceived as stressors activate an innate physiological stress response termed allostasis (de Kloet, Joels, & Holsboer, 2005; McEwan & Stellar, 1993). The body's first response to a perceived stressor is sympathetic nervous system arousal, leading to increased arousal, alertness, attention, and cognitive processing (de Kloet et al.). If the perceived threat continues, the hypothalamus activates the adrenal glands to release epinephrine which results in increased heart rate, increased blood pressure, rapid breathing, and the release of glucose into the bloodstream. A short time later, the hypothalamus releases two neuropeptides that coordinate a second stress response: corticotropin-releasing hormone (CRH) and vasopressin (AVP). The pituitary gland then releases adrenocorticotrophic hormone (ACTH), the net effect of which is the release of cortisol from the adrenal gland (de Kloet et al.).

Once the perceived stressor has subsided, feedback loops are triggered to shut down the hypothalamus-pituitary-adrenal HPA axis, and the parasympathetic nervous system aids in returning the organism to homeostasis or pre-stressor state (Lupien, McEwen, Gunnar, & Heim, 2009). However, the return to homeostasis depends on a variety of factors, namely stressor type, magnitude, and duration. Exposure to chronic stress can cause either persistent activation of the HPA axis and excess cortisol production or the opposite effect: a blunted HPA response and

hypocortisolism (Miller, Chen, & Zhou, 2007). An important finding in Miller et al.'s meta-analysis of stress's effect on cortisol output was that the more time that had passed since the stressor ended, a person's cortisol decreased eventually to below baseline level. Similarly, when chronic stressors remained present in a person's life or environment, daily cortisol output was higher compared to baseline. Thus, when chronic stress begins and persists, the HPA axis is overactive and produces excess cortisol, but if the stressor stops, cortisol secretion rebounds to below normal as time passes. The constant elevated cortisol concentration and other negative physiological sequelae of chronic stress are termed "allostatic load." An "allostatic state" occurs when the systems involved in allostasis are elevated in a sustained manner, and allostatic load – the cumulative effects of an allostatic state – refers to the physiological consequences of chronic exposure to stressors or the associated heightened neuroendocrine response (de Kloet et al., 2005; McEwen, 2004; McEwen & Stellar, 1993).

Allostatic load resulting from prolonged stress is associated with many negative physiological and psychological consequences. For example, stress hormones such as cortisol contribute to the suppression of Th1 cytokines which activate cellular immunity to protect against infection and disease. Suppression of Th1 cytokines in turn allow increased production of Th2 cytokines which can exacerbate autoimmune diseases and allergies (Chiappelli, Manfrini, Franceschi, Cossarizza, & Black, 1994; Segerstrom and Miller, 2004). Thus, chronic stress can compromise the immune system and contribute to illness or disease onset. Chronic stress also has negative effects on the cardiovascular system. Although a rise in blood pressure is a normal and adaptive response to stress, prolonged increases in blood pressure due to chronic stress promote generation of atherosclerotic plaques (McEwen, 2004). Hypertension is associated with many health problems, including myocardial infarction, stroke, heart failure, and renal failure

(Ong, Cheung, Man, Lau, & Lam, 2007). Another psychological consequence of chronic stress is memory change. Adrenaline and cortisol promote memory for the stressful situation, although as stress continues, neurons atrophy, which can lead to memory impairment. Other brain structures that undergo change include the hippocampus, prefrontal cortex, and amygdala, which can lead to increased anxiety, impaired memory, and impaired decision-making (McEwen). Overall, stress has many effects on the body and when prolonged can contribute to a variety of health problems.

### **Stress Responses in Migraine**

Many studies have supported associations between stress and migraine. Specifically, stress can contribute to migraine onset, exacerbate the progression of migraine frequency, precipitate and intensify individual attacks, and worsen headache-related disability. Frequent migraine itself also serves as a chronic stressor that impacts quality of life, functioning at home and work, and affective state (Nash and Thebarg, 2006).

The most commonly endorsed trigger of migraine is stress (Martin, 2010). Specifically, the existing literature indicates that daily hassles and daily stress, rather than major life stressors, are most likely to contribute to headache attacks or symptoms (Kohler, 1990). Several studies have experimentally manipulated stress to examine its effects on individual headache attacks, including their onset and severity. For example, after completing a lab stressor task that consisted of difficult-to-solve anagrams accompanied by failure feedback, 85% of participants with headache reported the task triggered a headache attack (Martin et al., 2007). Further, participants reported greater headache intensity in response to cognitive and noise stressors than to non-stress control conditions (Martin et al., 2005). Most recently, Lipton et al. (2014) assessed whether a day of low stress following a day of high stress was associated with migraine (i.e., “let-down headache”). Among their sample of 22 migraineurs completing daily diaries, though general

stress level was not associated with migraine onset, a decline in perceived stress was associated with migraine onset the next day. Thus, sufficient evidence exists to suggest that fluctuations in stress can trigger migraine attacks.

Although the mechanisms by which stress may trigger migraine attacks are not fully understood, Borsook and colleagues (2012) propose migraine to be a disease of allostatic load affecting the brain, such that migraine alters brain processing and structure. This model suggests that migraineurs face repeated stressors (e.g., migraine attacks themselves, exposure to triggers) but fail to habituate to them and thus experience a dysregulation of normal adaptive stress responses in which allostasis is not achieved (Borsook et al., 2012). As a result of this perpetual stress response, the nervous system of migraineurs becomes hypersensitive to routine stressors and other stimuli (Maleki, Becerra, & Borsook, 2012). For example, compared to non-headache controls, migraineurs are more sensitive to thermal (Schwedt et al., 2011), light (Purdy, 2011), sound (Sjostrand et al., 2010), and olfactory stimuli (Demarquay et al., 2008). Further evidence of altered allostatic regulation comes from numerous studies showing that migraineurs commonly experience pain upon response to non-painful stimuli (i.e., allodynia; such as while brushing one's hair or touching one's scalp). For example, 43-100% of migraineurs in experimental studies and 63% in a population-based study experienced allodynia (Ashkenazi, Sholtzow, Shaw, Burstein, & Young, 2007; Burstein, Yarnitsky, Goor-Aryeh, Ransil, & Bajwa, 2000; Lipton et al., 2008; Lovati et al., 2008; Mathew, Cutrer, & Garza, 2016). Maleki et al. (2012) suggest that stress leads to this heightened pain sensitivity via changes in regions that influence pain modulation, such as the periaqueductal gray (PAG), basal ganglia, and medial prefrontal cortex. According to the allostatic load model, migraine attacks function as stressors, the cumulative effects of which are perpetual neuronal and nociceptive hypersensitivity.

In addition to a heightened physiological response to stress-related stimuli, studies also suggest that migraineurs experience difficulty with habituation, or diminishing of a physiological response to a frequently repeated stimulus. This phenomenon has been demonstrated with different stimuli and physiological processes. The first study demonstrating decreased habituation among migraineurs examined contingent negative variation (CNV), a slow cortical electroencephalography (EEG) potential generated in a reaction-time paradigm (Coppola, Pierelli, & Schoenen, 2009; Maertens de Noordhout, Timsit-Berthier, Timsit, & Schoenen, 1986; Schoenen, Maertens, Timsit-Berthier, & Timsit, 1985). Researchers found CNV habituation to be markedly decreased among people with migraine. Other neurophysiological tests, such as those that measure other event-related potentials, visual-evoked potentials, auditory-evoked potentials, and somatosensory evoked potentials have also demonstrated reduced habituation among migraineurs compared to healthy controls (Coppola, Pierelli, & Schoenen, 2009).

In the context of stress or stressful stimuli, a relatively smaller body of literature has explored habituation or adaptation among migraineurs. Huber, Henrich, and Gundel (2005) compared 30 migraineurs to 30 non-headache controls when examining habituation to auditory stimuli and a mental arithmetic performance task. They found that habituation was impaired among migraineurs in the electrodermal (i.e., skin conductance), vasomotor (i.e., pulse volume amplitude), and cardiovascular systems during a mental arithmetic task; habituation of the vasomotor system only was impaired during presentation of auditory stimuli. Huss et al. (2008) compared physiological differences before/during/after a stressor task between 21 children with migraine and 32 non-headache controls. Participants completed an emotional arousal task that consisted of describing a negative stressful event from their past. Compared to controls, children with migraine exhibited slower physiological recovery after the emotional stressor, as evidenced



by higher diastolic blood pressure and higher LF/HF (low frequency/high frequency) ratio scores after a 5-minute recovery period. Similarly, in a study comparing migraine, TTH, and non-headache control groups in their response during and after a social stressor (i.e., delivering a news story in front of a video camera while receiving performance feedback), heart rate among migraineurs took longer than that of controls to return to baseline during the recovery period (Holm, Lamberty, McSherry, & Davis, 1997). Results suggest that migraineurs take longer than nonclinical populations to adapt and recover from stressful tasks or situations.

Although many studies provide support for increased physiological response to stress and decreased adaptation or habituation among migraineurs, other studies have failed to find such differences or yielded inconclusive results. Leistad and colleagues subjected participants with migraine, TTH, or non-headache to a 60-minute “low-grade cognitive stressor” intended to simulate real-life ongoing stress and then a 30-minute recovery period. The stressor task involved matching object positions to verbal instructions (e.g., two up, four right); items were administered via a computer program that also provided feedback on performance. Although participants with TTH took longer to recover from pain as quantified by EMG response and had higher cortisol levels during the recovery period, migraineurs showed similar EMG response and cortisol levels as non-headache controls (Leistad et al., 2007a; Leistad et al., 2007b; Leistad et al., 2008). Martin and Teoh (1999) compared mean temporal pulse amplitude, mean interbeat interval, and mean R wave to pulse interval scores across visual stimuli, stress, and control conditions among three groups: migraine, TTH, and non-headache control. The stress condition consisted of difficult-to-solve anagrams accompanied by failure feedback. Although negative affect was associated with changes in all vascular measures from baseline to the stressor phase, no significant cardiovascular differences were found between diagnostic groups. In a similar

study, women with migraine, TTH, or non-headache controls participated in a five-phase experiment including baseline, three stress periods (i.e., mental arithmetic tasks with negative feedback), and recovery (Stronks et al., 1998). Although mean norepinephrine levels, systolic blood pressure, diastolic blood pressure, and frontalis EMG were higher among migraineurs compared to non-headache controls, no significant group by period (stressors and recovery) interactions were found, indicating that migraineurs do not respond differently to stress compared to people without migraine.

Inconsistencies in the literature regarding whether migraineurs indeed exhibit a unique response to stress may be attributable to study design. For example, although Leistad et al. (2007a, 2007b, 2008) did not find differences in response between migraineurs and controls, they used a comparatively mild stressor. Studies finding migraineurs respond differently to stress compared to non-headache controls employed more severe stressor tasks. Additionally, the experiments discussed in this section used relatively small sample sizes, and larger sample sizes may have yielded more consistent differences between groups. Despite inconsistent findings regarding migraineurs' unique response to stress, differences in sleep have been more consistently observed.

### **Sleep in Migraine**

Sleep disturbances or disorders, including insomnia, restless leg syndrome, and breathing, movement, and circadian rhythm disorders, are common among migraineurs (Cevoli et al., 2012; Rains and Poceta, 2006), and up to half of headache patients have chronic sleep difficulties (Kelman & Rains, 2005). Sleep deprivation, lack of sleep, excessive sleep, or sleep disturbance is also frequently cited by migraineurs (36-74%) as a trigger (Martin, 2010; Peroutka, 2014). Among migraineurs presenting for treatment at headache clinics, one-half to two-thirds report

insomnia, making it the most common sleep disorder among migraineurs (Rains and Poceta, 2006). Kelman and Rains (2005) found insomnia symptoms were three times more common among migraineurs compared to the general population; specifically, 38% of migraineurs reported chronically shortened sleep periods compared to the 10.8% incidence in the general population. Non-refreshing sleep is more common among migraineurs than the general population [Odds ratio (OR) = 2.98; Rasmussen, 1993], and excessive daytime sleepiness is more prevalent among migraineurs than non-headache controls (OR = 3.1; Barbanti, 2007) and associated with increased migraine disability. Findings from the National Comorbidity Survey demonstrated that compared to healthy participants, migraineurs had greater difficulty with daytime fatigue (OR = 2.6), falling asleep (OR = 2.2), remaining asleep (OR = 2.8), and awakening early (OR = 2.0; Lateef et al., 2011). Although Seidel et al. (2009), for example, did not find a positive association between migraine diagnosis and fatigue, the majority of studies have consistently found that migraine is associated with significantly increased risk for sleep problems.

The relationship between migraine and sleep disturbance is such that either can exacerbate the other. For example, Kelman and Rains (2005) found that 71% of 1283 treatment-seeking adult migraineurs endorsed headaches awakening them from sleep. Additionally, much literature supports the notion that sleep disturbance or lack of sleep precipitates headache or worsens headache symptoms. In a review of studies examining headache triggers, Peroutka (2014) determined that 43% of 5347 migraineurs across 25 studies reported sleep loss or sleep problems as a migraine trigger. In a prospective daily diary study of 33 chronic migraineurs and 22 CTTH sufferers, two days of decreased sleep was associated with increased risk for headache, and headache severity increased as sleep decreased (Houle et al., 2012). While findings suggest

that poor sleep quality and migraine symptoms worsen each other, other studies have examined whether the relationship between disordered sleep and migraine can be explained by psychiatric comorbidity.

Although sleep disorders and migraine commonly co-occur with depression and anxiety, Walters, Hamer, and Smitherman (2014) found that, among 78 migraineurs, sleep quality was independently associated with migraine frequency after controlling for symptoms of depression and anxiety. Further, sleep quality had the greatest association with headache frequency and disability, while sleep hygiene and daytime sleepiness did not account for any further variance in headache frequency, severity, or disability. In a similar study by Vgontzas, Cui, and Merikangas (2008), migraineurs completed self-report measures of sleep problems and psychiatric symptoms. Compared to those without headache, migraineurs reported more complaints of inadequate sleep (OR = 2.5) and difficulty falling asleep (OR = 3.0), and these associations remained when controlling for lifetime depression and anxiety symptoms. Thus, despite the common co-occurrence of psychiatric symptoms and sleep problems, the association between migraine and sleep problems does not appear to be merely a function of comorbid psychiatric symptoms. Though research suggests an interaction between sleep and migraine, an additional factor that may interact with sleep to exacerbate migraine symptoms is stress.

### **Stress and Sleep Interaction in Migraine**

Despite the disproportionate prevalence of both stress and sleep disturbance among migraineurs, there is surprisingly limited literature examining interactions between stress and sleep among migraineurs. Houle et al. (2012) examined relationships between self-reported stress, sleep duration, and headache intensity among patients with chronic migraine or chronic tension-type headache. Not only were low sleep and high stress each independently associated

with increased risk for next-day headache, but they had an interactive effect such that low sleep and high stress most strongly predicted next-day headache when they co-occurred. In another prospective diary study, Spierings et al. (1996) also found an interactive effect among 20 female migraine patients who kept a daily diary of sleep, mood, and stress. Specifically, daily hassles and mood changes, including feeling tired, were significantly increased in the two days before a migraine headache compared to other non-headache days. Interactions between stress and sleep have also been demonstrated in laboratory studies. In one, pressure, heat, and cold pain thresholds and sleep quality [i.e., questionnaires, sleep diaries, and polysomnography (PSG)] were examined among participants with migraine, TTH, and non-headache controls (Engstrom et al., 2014). Migraineurs had lower pain thresholds compared to controls, and increased pain sensitivity was associated with increased sleep problems (e.g., insomnia, subjective sleep quality, pain-related sleep trouble) among migraineurs. Thus, results suggest an interaction between sleep and response to physical stress among migraineurs, although authors considered findings to be preliminary due to their sample size of 53 patients. Overall, these studies among migraineurs indicate a stress-sleep interaction.

According to the allostatic load model of migraine, sleep deprivation and circadian disruption are themselves sources of stress (Spiegel, Leproult, & Van Cauter, 1999), and migraine is associated with alterations in sleep, particularly increased prevalence of sleep disorders (Rains, Poceta, & Penzien, 2008). Thus, inadequate sleep among migraineurs likely exacerbates stress, making migraineurs a susceptible population to stress-sleep interactions. Though studies of this type are few within migraine, those among chronic pain populations and the general population indicate a need to further examine sleep-stress interactions among clinical populations.

## **Stress and Sleep Interaction in Chronic Pain**

Several studies have addressed stress and sleep interactions among chronic pain populations, many of which have used a pain task as the stressor. As many as one in three people suffer from chronic pain (Rosenzweig et al., 2010), and 50-88% of these patients report significant sleep disturbance (Smith & Haythornthwaite, 2004). Several self-report diary studies have indicated associations between stress and sleep. For example, among 50 women with fibromyalgia, previous night's sleep quality accounted for 5% of the variance in the present day's attention to pain when controlling for pain intensity (Affleck et al., 1996). Thus, independent of the intensity of pain, poor sleep quality influenced patients' awareness and ability to focus attention elsewhere. Pain intensity may also be affected by sleep, as demonstrated in a study of 28 adult burn victims (Raymond et al., 2001). A night of poor sleep quality was significantly associated with higher pain intensity the next day (partial  $r = -.25$ ), and the partial correlations between pain intensity and symptoms of sleep disorders were even higher (partial  $r = .37-.56$ ). These results suggest that while inadequate sleep one night partly predicts pain intensity the following day, the correlation between existing sleep conditions and pain intensity may reflect a chronic effect of sleep disturbance on physiological sensitivity.

In addition to impacting pain intensity and attention to pain, sleep may also impact affective response to stress. Hamilton, Catley, and Karlson (2007) used twice-daily diaries to assess stress, sleep, and pain variables among 49 women with fibromyalgia or rheumatoid arthritis, and negative affect in response to stress varied as a function of sleep quality. Specifically, participants who had good sleep quality showed little affective response to stressful events, while those with poor sleep quality experienced more negative affect, which increased as level of stress increased. Similar results were found among 287 chronic pain patients, 88.9% of

whom reported at least one sleep disturbance and 62.1% who endorsed inadequate or reduced sleep (McCracken and Iverson, 2002). Sleep disturbance was associated with pain intensity ( $r = .26$ ), depression or emotional distress ( $r = .41$ ), and physical symptoms of distress ( $r = .34$ ). Haythornthwaite, Hegel, and Kerns (1991) also found that among their sample of 46 chronic pain patients who completed a daily sleep diary for five days, hours of sleep were negatively correlated with depression ( $r = -.40$ ), anxiety ( $r = -.48$ ), and pain severity ( $r = -.33$ ). The broader implication of these self-report diary and assessment studies is that poor sleep quality promotes heightened stress reactivity, and high-quality sleep may facilitate adjustment to stress. Consistent with the allostatic load model, inadequate sleep may limit available resources to adapt to and manage stress and pain.

Experimental studies on chronic pain populations have yielded mixed findings. Sixteen adults with fibromyalgia participated in physical stressor task in which a manual algometer applied pressure to the finger (Agargun et al., 1999). Pain thresholds were negatively correlated with sleep quality ( $r = -.58$ ), habitual sleep efficiency ( $r = -.56$ ), sleep disturbance ( $r = -.57$ ), and Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) global scores ( $r = -.56$ ), although negative associations between pain threshold and sleep latency/duration were not statistically significant. Similarly, studies employing total sleep deprivation, NREM sleep deprivation, or slow-wave sleep disruption among chronic pain patients have also demonstrated decreased pain thresholds during physical stressor tasks (Moldofsky, Scarisbrick, England, & Smythe, 1975; Lentz, Landis, Rothermel, & Shaver, 1999). In an experimental study examining psychological stress (Shaver et al., 1997), 11 women with fibromyalgia self-reported poorer sleep quality and higher psychological distress than 11 women without symptoms (controls). However, there were no differences between groups in

somnographic sleep variables of sleep quality, depth, or continuity for the total night.

Additionally, catecholamines and cortisol urine concentrations were similar for both groups after a Stroop stress challenge, thus not supporting group differences in physiological response to psychological stress. It should be noted, however, that physiological stress response was not statistically analyzed as a function of sleep quality or quantity, so a stress-sleep interaction is uncertain. Overall, studies provide evidence that poor sleep is associated with increased pain sensitivity and intensity among chronic pain patients, although the associations between sleep quality and psychological stress are less clear.

### **Sleep and Stress Interaction in the General Population**

Although limited literature exists on sleep and stress interactions among migraineurs and stress-sleep studies among chronic pain populations have focused largely on pain and physical stress, stress and sleep interactions have been studied more extensively among the general population. Psychological stress and worry negatively impact sleep quality (Borkovec, Lane, & VanOot, 1981; Hall et al., 1997). According to Kim and Dimsdale's (2007) review of the effects of stress on sleep, studies have yielded inconsistent findings although several have identified decreases in REML, increases in REM, and decreases in slow-wave sleep among people confronted with stressful life events. Conversely, sleep deprivation elicits physiological stress responses and increases energy expenditure, blood pressure, cortisol, and both insulin and blood (Gangwisch et al., 2006; McEwen, 2006). Several studies have examined relationships between physiological responses to acute experimental stressors and sleep.

Studies using cardiovascular measures as outcome variables have yielded mixed findings. Hall and colleagues (2004) compared heart rate variability among 59 undergraduates who were assigned to a stress condition involving a speech task or a non-stress control group. EKG was



collected the following night and, compared to controls, participants who underwent the stress task exhibited greater mean arterial pressure, decreases in parasympathetic modulation during NREM and REM sleep, and increases in sympathovagal balance during NREM sleep.

Conversely, parasympathetic modulation increased throughout REM cycles among the non-stress control group. A different study examined sleep deprivation's effect on cardiovascular reactivity to acute psychological stressor tasks administered the next day: a Stroop color-word naming task and a speech task (Franzen et al., 2011). During the speech task, both groups (one night of total sleep deprivation vs. one night of normal sleep) exhibited increased systolic blood pressure reactivity, and sleep deprived individuals also exhibited greater systolic blood pressure during the task than participants who had slept. Subjective stress ratings were similar between groups, suggesting sleep deprivation has a unique influence on physiological response that cannot be accounted for by psychological stress. Kato et al. (2000) conducted a similar study using four stressor tasks: sustained handgrip, maximal forearm ischemia, mental stress, and a cold pressor test. Participants completed the stressor tasks twice – once after a normal night of sleep and once after a night of sleep deprivation. Contrary to Franzen et al.'s findings, although sleep deprivation resulted in higher resting blood pressure the following morning, sleep deprivation did not potentiate cardiovascular responses to any stressful stimuli. It should be noted, however, that Kato et al.'s sample size was 8, and a larger sample size (or one of a clinical sample) could have yielded different results.

In addition to cardiovascular measures, psychological stress and cortisol have also been used as outcome variables and have supported an interaction between stress and sleep. For example, in one study 53 adults were assigned to either a night of sleep-deprivation or a nine-hour window for sleep and then completed both high and low stressor conditions (Minkel et al.,

2012). The former consisted of a difficult serial subtraction task, a difficult version of the Stroop task, and a divided attention task with negative performance feedback. The low stress condition included an easy subtraction task and an easy Stroop color-naming task. Sleep-deprived participants scored significantly higher on subjective measures of stress, anger, and anxiety in response to the low-stressor condition, but differences between groups in the high-stressor condition were not significant. This suggests sleep loss lowers the threshold at which a person experiences an event as stressful although findings could be attributable to order effects, as all participants completed the low stress condition first. In addition to impacting psychological stress, sleep also affects cortisol levels. Minkel et al. (2014) subjected a group allowed normal sleep and one under total sleep deprivation to the Trier Social Stress Test (TSST), which has been demonstrated to evoke autonomic and HPA axis stress responses (Granger, Kivlighan, Eibenstein, Sheikh, Gordis, & Stroud, 2007; Nater et al., 2005). Cortisol measurements were taken at baseline, throughout the TSST, and during recovery. Cortisol was significantly higher in the sleep-deprived group than the normal sleep group, and sleep deprivation amplified response to the stressor after controlling for baseline cortisol levels. Considered collectively, results from these experimental studies provide further support that reduced sleep affects physiological response to stress.

### **Goals of the Present Study**

While recent studies of non-migraine samples demonstrate that sleep deprivation affects physiological response to stressors, further research on sleep-stress interactions is needed among migraineurs given the significant roles of stress and sleep disturbance among this common and disabling primary headache disorder. The real-world applicability of the prior studies on sleep and stress among migraineurs is questionable given that existing studies have employed total

sleep deprivation, while sleep loss or inadequate sleep is much more likely to occur in real-world settings. Existing studies have confirmed an interaction effect between stress and sleep among migraineurs, but an experimental study could help identify potential mechanisms behind these effects, and use of a repeated stressor would afford assessment not only of sleep's effect on initial stress response but also on habituation or adaptation to stressful stimuli. The present study thus sought to compare physiological response to a stressor between migraineurs and non-headache controls and to determine if physiological stress response is moderated by sleep duration the prior night. Results from this study may be used to gain further understanding of how stress and sleep affect headache-related physiology and has implications for increasing knowledge of headache triggers and treatment development strategies.

## **Hypotheses**

*Study Goal 1: To determine if sleep duration moderates physiological response and adaptation to stress among migraineurs and non-headache controls.*

Hypothesis 1: Migraineurs would exhibit greater physiological response to the second administration of a stressor compared to non-headache controls.

Hypothesis 2a: An inverse relationship between sleep quantity and physiological response to stress (i.e., heart rate and both systolic and diastolic blood pressure) would be observed among migraineurs and non-headache controls.

Hypotheses 2b: Sleep quantity would have a greater impact on heart rate and blood pressure reactivity to a stress task among migraineurs than non-headache controls.

*Study Goal 2: To determine associations between physiological adaptation to a stressor and self-reported sleep quality.*

Hypothesis 3: An inverse relationship would exist between self-reported sleep quality and physiological response to the second administration of a stressor (i.e., heart rate and both systolic and diastolic blood pressure) among migraineurs and non-headache controls.

## II. METHODS

### **Participants**

The initial sample consisted of 133 undergraduate students age 18 and older enrolled in psychology courses at the University of Mississippi who received modest course credit for participation. Students meeting ICHD-3 criteria for migraine (with a frequency of at least 2 headache days/month) and those not meeting criteria for any headache disorder were identified as candidates for the study following their completion of a series of online questionnaires via Qualtrics, and they were invited by email to participate. Assuming a moderate effect size ( $f=.25$ ), a power level of 0.80, and an alpha level of 0.05, a total sample size of 34 participants was required in a repeated-measures, within-between interaction design with 2 groups and 3 measurements. Because G Power cannot account for moderating variables, 25% additional participants were required (43 participants total) in order to determine if sleep was a moderating variable in physiological response to an experimental stressor.

### **Measures**

*Structured Diagnostic Interview for Headache – 3 (Brief Version)*. The Structured Diagnostic Interview for Headache (SDIH-3; Smitherman, Penzien, Rains, Nicholson, & Houle, 2015) is a modified version of the original computer-administered and well-validated SDIH (Andrew, Penzien, Rains, Knowlton, & McAnulty, 1992), revised to comport with ICHD-3 diagnostic criteria. The SDIH-3 is a 17-item instrument that assesses for primary headache disorders by querying headache symptoms, frequency, severity, and other diagnostic

characteristics. Additionally, the SDIH-3 includes appendix questions for assessing aura symptoms, cluster headache, medication overuse, and post-traumatic headache.

***Blood Pressure and Heart Rate.*** Diastolic and systolic blood pressure and heart rate were measured with a digital sphygmomanometer and heart rate monitor (Omron HEM-907XL) at 3-minute intervals throughout baseline, stressor, and recovery periods, and averaged within each phase.

***Sleep.*** Sleep quantity was measured via an online self-report sleep diary for the two days preceding the stressor task.

***Pittsburgh Sleep Quality Index.*** The Pittsburgh Sleep Quality Index (PSQI) is an 18-item self-report measure of sleep quality developed by Buysse, Reynolds, Monk, Berman, and Kupfer (1989). The measure has 89.6% sensitivity and 86.5% specificity in distinguishing “good” from “poor” sleepers and has been validated in both clinical and non-clinical samples (Grandner, Kripke, Yoon, & Youngstedt, 2006; Backhaus, Junghanns, Brooks, Riemann, Hohagen, 2002).

***Stressor Task.*** The Trier Social Stress Task (TSST; Kirschbaum, Pirke, & Hellhammer, 1993) is a standardized and well-validated paradigm for inducing social stress in laboratory settings. The task begins by informing the participant she has 10 minutes to prepare an impromptu speech explaining why she is a good candidate for her ideal job (Birkett, 2011). The participant is also informed at this time that the subsequent speech will be videorecorded and presented in front of a panel of judges trained in public speaking. After the 10-minute preparation/anticipatory period, the participant is given 5 minutes to deliver the speech, and immediately thereafter is asked to subtract 13 from 1,022 for 5 minutes. If a mistake is made, the participant is asked to start over, and during both tasks the judges provide no verbal or visual feedback or encouragement to the participant.

Experiments requiring a repeated-measures design have modified the TSST to allow for repeated administrations (Schommer, Hellhammer, & Kirshbaum, 2003; Kudielka et. al, 2006). All procedures are the same for each administration, with the only variations being the job that participants are asked to apply for and the initial number used for the serial subtraction task. In order to preserve the social evaluative nature of the TSST and allow for repeated measures within a relatively short time frame, the present study omitted the subtraction task and included two administrations of the speech task. Participants were given 5 minutes to prepare each speech to allow this experimental session to be completed within one hour.

***State Trait Anxiety Inventory.*** The State Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) is a self-report measure that consists of two subscales assessing state and trait anxiety. The state anxiety subscale is frequently used to measure psychological distress in studies employing the TSST, and it has been shown to have high reliability, concurrent validity, and construct validity (Spielberger, 1989). This study used only the state anxiety subscale.

***Headache Intensity.*** Headache intensity during the 2 days following the experimental stressor was queried via an online brief self-report survey.

## **Procedures**

Young adults who were invited and who agreed to participate met with an experimenter in order to complete the SDIH-3 and confirm a diagnosis of migraine (with at least 2 headache days/month) or absence of headache. Participants also received instructions for the online self-report sleep diary and were instructed to complete the diary for the 2 nights prior to the experimental session.

At the experimental session 2 days later, participants completed the PSQI and the first administration of the STAI. After 10-minute baseline, participants completed the 5-minute preparation period and the 5-minute speech task. Upon completion, participants had a recovery period for 10 minutes, and then were told to prepare a speech for a different job (i.e., second most desired job). After the second 5-minute preparation period they delivered a second 5-minute speech, and the STAI was administered the second time following the completion of the second stressor task. A second 10-minute recovery period followed. HR and BP were measured 4 times (every 3 minutes) during each phase: baseline, stressor 1, recovery 1, stressor 2, and recovery 2. After the experimental session was complete, participants were debriefed and those with migraine were asked to record ratings of headache intensity for two subsequent days. The follow-up headache intensity data were collected for additional analyses that are not a part of the current dissertation.

### **Statistical Analyses**

In order to assess sleep as a moderator of physiological response to a stressor, a series of repeated-measures multiple regressions with Bonferroni correction were performed. Specifically, within a general linear model repeated measures design, migraine and non-headache groups were compared after entering three timepoints (baseline, stressor task 1, stressor task 2) for each physiological variable as within-subject factors, with a mean-centered sleep variable as a covariate. Separate analyses were run for total sleep quantity over the two nights immediately preceding the experiment and sleep quality as reported on item 9 of the PSQI. An interaction term was added to each analysis to address the main hypothesis, and each sleep variable was broken into three levels (mean, one standard deviation below the mean, one standard deviation above the mean) to conduct follow-up analyses of any potential interaction effects. Each analysis



of significance was then run a second time, after controlling for depression (PHQ-9) and anxiety (GAD-7) scores.

### III. RESULTS

#### **Participant Demographics and Primary Headache Diagnosis Prevalence**

One hundred thirty-three students ages 18 - 41 participated in the study. Of these, 31 were dismissed after part 1 of the study after they provided responses to the SDIH-3 indicative of a headache diagnosis other than migraine or no headache ( $n = 30$ ), or that precluded establishing a clear diagnosis ( $n=1$ ). Specifically, 28 participants met diagnostic criteria for tension-type headache, 1 reported a prior diagnosis of cluster headache, and 1 reported symptoms of post-traumatic headache. Additionally, 1 participant was dismissed after part 1 due to a language barrier preventing adequate communication of informed consent and instructions. Of the 101 participants invited to complete the experimental session, 19 (15 migraineurs, 4 non-headache) missed their scheduled appointments and thus did not complete the study. An additional 8 participants (6 migraineurs, 2 non-headache controls) withdrew during the experimental session due to reported anxiety over giving the first or second speech. Of the 74 participants who completed the study, 2 participants (both non-headache controls) were excluded from analysis due to missing data. Additionally, 3 participants with migraine reported migraine frequencies of one day or less per month; these participants were excluded to ensure distinct subgroups of migraine and non-headache. The final sample thus included 69 participants: 37 (53.6% of retained sample) without headache and 32 (46.4%) with migraine, which exceeded that specified in the a priori power analysis.

Demographics of the retained sample are presented in Table 1. Most participants were female (81.2%), and the mean age was 19.1 years ( $SD = 3.0$ ). The majority of the sample was Caucasian (78.3%), followed by 10.1% African American, 7.2% Asian, 2.9% multiracial or other, and 1.4% Hispanic/Latino. Participants meeting diagnostic criteria for migraine reported, on average, experiencing 6.1 headache days per month ( $SD = 3.6$ ), a pain severity of 6.4 out of 10 ( $SD = 1.3$ ), and moderate levels of both anxiety and depression that were significantly higher than the control group.

### **Sleep as a Moderator of Physiological Response to Stress**

#### *Sleep quantity*

For total sleep quantity on both nights preceding the experiment, repeated-measures multiple regression revealed significant differences in systolic blood pressure (SBP) between baseline, stressor task 1, and stressor task 2 ( $F(2, 130) = 91.62, p < .001$ , R-squared change = .585). As expected, SBP was higher during both stressor tasks compared to baseline, and SBP decreased during the second stressor task compared to the first. Thus, the speech task was effective in inducing stress and increasing SBP, and participants appeared to habituate to stress given the decrease in SBP during the second stressor task. Systolic blood pressure did not change between timepoints as a function of headache diagnosis ( $F(2, 130) = .626, p = .536$ , R squared change = .01) or as a function of sleep quantity ( $F(2, 130) = .038, p = .963$ , R-squared change = .001).

However, consistent with study hypotheses, participants with migraine and those without headache had different SBP in response to stress between timepoints depending on their quantity of sleep ( $F(2, 130) = 4.742, p = .010$ , R squared change = .068), indicating a significant interaction effect. To determine the specific nature of this interaction, analyses were repeated

using on z-scores of sleep quantity, with 11.28 hours indicating low sleep (z-score = -1), 13.80 hours indicating average sleep (z-score = 0), and 16.32 hours indicating high sleep (z-score = 1). As Figure 1 displays, participants without headache had highest SBP at baseline and the first stressor task when they had the least amount of sleep (11.28 hours) and lowest SBP when they had the highest amount of sleep (16.32 hours). However, such a pattern was not seen in migraineurs. Rather, migraineurs appeared to respond similarly to stress regardless of their sleep quantity, as little variance in SBP was seen between low, average, and high sleep quantity. When each timepoint in this interaction was analyzed, this relationship was significant during stressor task 1 ( $p = .008$ ) but not significant at baseline or the second stressor task. Furthermore, when examining sleep quantity based on z-scores, significance was only found at a z-score of 1, such that this interaction was significant with participants whose sleep was 1 standard deviation above the mean ( $SD = 2.52$ ) or who slept 16.32 hours or more over two nights ( $F(1, 65) = 4.845, p = .031, R \text{ squared change} = .069$ ). There was not a significant interaction effect during baseline or the second stressor task. When PHQ-9 and GAD-7 scores were included in the interaction term, none of the aforementioned findings for differences in systolic blood pressure between groups were significant.

Differences in diastolic blood pressure (DBP) between timepoints were significant ( $F(2, 130) = 132.38, p < .001, R \text{ square change} = .671$ ), indicating that the speech task was also effective in increasing DBP among participants. Similar to SBP, DBP increased during both stressor tasks compared to baseline, and DBP during the second stressor task decreased compared to the first stressor task. DBP did not change as a function of sleep quantity ( $F(2, 130) = 1.202, p = .304, R \text{ square change} = .018$ ) nor as a function of headache diagnosis ( $F(2, 130) = .418, p = .659, R\text{-squared change} = .006$ ). Contrary to hypotheses, participants with migraine and

those without headache did not have different DBP in response to stress between timepoints depending on their quantity of sleep, as there was not a significant interaction effect ( $F(2,130) = .994, p = .373, R \text{ squared change} = .015$ ). However, despite the interaction being statistically insignificant, a similar pattern to the SBP results is visually evident (i.e., where only participants without headache showed the predicted inverse relationship between sleep quantity and DBP at all time points).

Heart rate (HR) was also significantly different between timepoints ( $F(2, 130) = 88.749, p < .001, R\text{-square change} = .577$ ), indicating participants' HR increased in response to the speech task. In the main model, HR did not vary as a function of headache diagnosis ( $F(2, 130) = 1.792, p = .171, R\text{-squared change} = .027$ ) or sleep quantity ( $F(2, 130) = .416, p = .660, R \text{ squared change} = .006$ ), and the diagnosis-sleep interaction term was not significant ( $F(2, 130) = 1.905, p = .153, R \text{ squared change} = .028$ ). Thus, results for HR were inconsistent with the study's hypothesis.

#### *Sleep quality*

Additional repeated-measures multiple regressions were run to test for an interaction effect involving sleep quality, as measured by participants' self-report of sleep quality on the PSQI. However, none of these analyses revealed significant results. Specifically, the diagnosis-sleep interaction term was not significant for systolic blood pressure ( $F(2, 130) = .376, p = .688, R\text{-squared change} = .006$ ), diastolic blood pressure ( $F(2, 130) = .629, p = .535, R\text{-squared change} = .010$ ), or heart rate ( $F(2, 130) = .754, p = .472, R\text{-squared change} = .011$ ).

#### *Sleep quantity and quality for one night preceding the experiment*

Although goals for this study included examining two nights of sleep when assessing for interaction effects, exploratory analyses were run to assess for different outcomes involving only

one night of sleep. Overall, results were similar to those from two nights of sleep, with a significant diagnosis-sleep interaction term for the main models only found for systolic blood pressure and not for diastolic blood pressure or heart rate.

## IV. DISCUSSION

Existing literature has presented much evidence that reduced sleep affects physiological response to stress, particularly among the general population and chronic pain populations. An interaction of sleep and stress response among migraineurs has been less researched, although studies conducted indicate that low sleep and high stress may trigger headache attacks (Houle et al., 2012; Spierings et al., 1996) and that migraineurs' increased pain sensitivity in physical stressor tasks is associated with sleep problems (Engstrom et al., 2014). Given the sparsity of literature on stress-sleep interactions among migraineurs and the employment of sleep deprivation in prior studies among the general or chronic pain population, this study sought to examine real-world sleep conditions in an experimental study, specifically analyzing any differences in a stress-sleep interaction between migraine and non-headache groups as a function of exposure to a laboratory stressor. Additionally, a repeated stressor was used to examine sleep's effect on adaptation to stressful stimuli. Based on findings of previous studies and the prevalence of sleep disturbance and high stress among migraineurs, we hypothesized that sleep quantity and quality would have a greater impact on blood pressure and heart rate reactivity to a stress task among migraineurs than non-headache controls.

### **Sleep as a Moderator of Physiological Response to Stress**

Although systolic blood pressure did not change as a function of headache diagnosis alone, there was a significant interaction effect of headache diagnosis and sleep quantity on SBP, a result consistent with hypotheses. However, the nature of that interaction and the specific results obtained were unexpected. It is unclear why there was an inverse relationship between

sleep quantity and SBP among non-migraineurs, while no such relationship was seen for migraineurs. Based on previous literature examining migraineurs' stress response, we expected low sleep among migraineurs to be associated with heightened stress response compared to both migraineurs with greater sleep quantity and non-headache controls. There are possibly confounding variables unaccounted for by this study, such as weight or physical activity (e.g., perhaps higher sleep quantity among migraineurs is associated with less physically active lifestyle and therefore different cardiovascular response). Alternatively, while insufficient sleep has been demonstrated among the general population as a risk factor for increased stress response (cardiovascular, cortisol, etc.; Gangwisch et al., 2006; McEwen, 2006) and those results were replicated in this study among participants without headache, results of this study also seem to suggest that sleep quantity may not have the same impact on SBP among migraineurs. If these results are replicated in future studies, the clinical implication may be that while treating insomnia has a host of benefits for migraineurs (Smitherman et al, 2018; Rains, 2018), decreased SBP in response to stress may not be among them. However, when PHQ-9 and GAD-7 scores were added as controls to the interaction analyses, results were no longer significant, suggesting that increased anxiety and depression associated with migraine may account for differences in physiological reactivity between groups.

Contrary to hypotheses, there was no significant interaction effect of sleep quantity and headache diagnosis on diastolic blood pressure. Thus, DBP in response to stress could not be predicted by sleep quantity and headache diagnosis. Similar to results for DBP, there was also no interaction effect for heart rate in response to stress. While there is little research on the interaction of sleep and physiological stress response between headache diagnoses, existing literature indicates that in a physical stressor task, migraineurs exhibit decreased pain thresholds



compared to participants without headache and that this increased pain sensitivity is associated with more sleep problems (Engstrom et al., 2014). Considering the multitude of studies that have identified increased stress response and sleep problems among migraineurs, it therefore seemed plausible we would observe a similar interaction for DBP or HR during our stressor tasks, with low sleep associated with increased DBP or HR among migraineurs especially. However, this study's results indicated no such interaction effect involving sleep and headache diagnosis for these specific cardiovascular measures, and there was no main effect of headache status on reactivity more generally (i.e., both groups evidenced similar increases from baseline). Further studies are needed among these populations examining interactive effects involving sleep and other physiological measures of stress.

The unexpected pattern of SBP results and non-significant interaction effects for DBP converge around the notion that sleep may not be a protective factor against heightened blood pressure reactivity for migraineurs, further contributing to literature on relations between migraine and cardiovascular disease. Existing studies have yielded contradicting findings regarding an association between migraine and hypertension, with some suggesting no relationship and others indicating a positive or negative relationship between the conditions (Agostoni & Aliprandi, 2008; Mancia et. al., 2011). A review and meta-analysis of migraine and cardiovascular disease determined that migraine with aura is associated with an increased risk of ischemic stroke, while no such association was found between any migraine and myocardial infarction or death due to cardiovascular disease (Schurks et. al., 2009). However, the authors also concluded that too few studies were available to reliably evaluate potentially modifying factors, such as sleep. A hospital-based registry study including a sample of nearly 125,000 surgical patients found that migraineurs were at increased risk of perioperative ischemic stroke

compared to patients without migraine and that this risk was highest among migraineurs with aura (Timm et al., 2017). Given that studies examining hypertension and sleep duration among healthy adults have provided evidence that sleep deprivation acutely increases blood pressure and that prolonged short-sleep durations are associated with increased risk of hypertension (Gangwisch et al., 2006), it is surprising that the current study's results suggest sleep was a protective factor against heightened blood pressure among non-headache controls but *not* among migraineurs during the stress task.

Regarding habituation to stress, results of this study did not provide evidence for any differences between migraineurs and participants without headache. This was inconsistent with this study's hypothesis that migraineurs would consistently exhibit reduced habituation to stress compared to non-headache. Such a finding was unexpected, given existing research demonstrating that migraineurs have reduced habituation to stressful stimuli compared to non-headache samples (Huber, Henrich, & Gundel, 2005; Huss et al., 2008; Holm, Lamberty, McSherry, & Davis, 1997). Specifically, heart rate among migraineurs took longer than that of controls to return to baseline during the recovery period of a social stressor task (Holm et al., 1997), and cardiovascular habituation to stress was impaired among migraineurs (Huber, Henrich, & Gundel, 2005; Huss et al., 2008). Differences in results between past studies and the current study could be attributable to stressor task or participant sample. For example, previous studies used auditory stimuli and a mental arithmetic task (Huber, Henrich, & Gundel, 2005) or included a study sample of children (Huss et al., 2008). The present study instead utilized a modified version of the well-validated Trier Social Stress tasks and young adults as participants. Additionally, while the current study included participants with at least two headache days per month, migraineurs in Huber et al.'s (2005) sample had at least one migraine attack per week per

and the duration of illness was a minimum of two years. It is possible that the difference in severity of migraine between studies could explain the opposing findings, but this seems unlikely given the high mean frequency of attacks in the present sample.

Finally, contrary to hypotheses, no interactions involving sleep quality were found, despite sleep quality (on the PSQI) being lower among migraineurs compared to participants without headache. The difference between groups is consistent with existing literature on sleep quality among migraineurs (Rasmussen, 1993), although results suggest that sleep quality does not play a role in cardiovascular reactivity to stress among this sample.

Overall, despite the original hypothesis that an inverse relationship would exist between sleep duration and physiological reactivity, results indicate that this pattern was seen among non-headache controls but that longer sleep duration was not beneficial for migraineurs. One possible explanation is that the depressive symptoms often comorbid with migraine may be a contributing factor to the association between longer sleep duration and increased physiological reactivity in this study. Depressive symptoms have an association with sleep disorders including hypersomnia (Dauvilliers, Lopez, Ohayon, & Bayard, 2013). Additionally, depressed mood is associated with higher SBP and DBP, and depressed patients have been found to have more cardiovascular diseases and hypertension than other psychiatric patients (Scalco, Scalco, Azul, & Lotufo Neto, 2005). Given that the interaction effect of sleep and headache diagnosis found for SBP was no longer significant when controlling for depression and anxiety, psychiatric symptoms among migraineurs may account for the differences between groups and thus explain why migraineurs demonstrated a positive association between sleep duration and physiological reactivity to stress. If this is the case, treatment for affective symptoms rather than for sleep could be more likely to

protect against heightened physiological reactivity among migraineurs, though this notion awaits empirical verification.

Alternatively, perhaps migraineurs with longer sleep duration and higher blood pressure and heart rate lead more sedentary lifestyles. Low physical activity is associated with increased risk for and greater frequency of migraines (Ahn, 2013) and also increases risk for cardiovascular diseases (Thompson et al, 2003). Although this study did not assess activity level, a more sedentary lifestyle among migraineurs could have partly explained the positive association between sleep duration and physiological reactivity to stress. In this case as well, a program of aerobic exercise or increased physical activity could be an effective area of intervention.

### **Limitations and Future Directions**

Strengths of this study include strict adherence to ICHD diagnostic criteria via in-person structured interviews (SDIH-3), use of a medical-grade monitor for measuring heart rate and blood pressure, and the use of a validated social stressor task to induce stress in participants. However, this study includes several limitations, and caution should be used when generalizing these findings to the broader population. First, the final sample consisted of 69 participants, which although sufficient assuming a moderate effect size, would be insufficient to detect smaller effects. Thus, it is possible that a larger sample would have yielded different results and perhaps significant sleep-diagnosis interaction effects for more variables than solely systolic blood pressure. Second, all participants were non-treatment-seeking undergraduate students with an average age of 19 years, and thus results may not generalize to older or clinical populations, or those who have suffered from migraine for decades. However, young adults are a desirable population in headache research, given their high prevalence of primary headache disorders and

low frequency of variables that complicate conclusions from headache studies, such as long histories of medication overuse and chronification (Smitherman et al., 2011). Additionally, participants with low headache frequencies (1 day or less per month) were omitted from analyses, and retained migraine participants had an average of approximately 6 headache days per month. In actuality then, our sample was quite severe regarding headache frequency, as only 14% of adults with migraine experience headache on 5 or more days per month (Lipton et al., 2007). Third, although this study included multiple measures of sleep including quantity for one and two nights and sleep quality, it relied on self-report measures rather than lab manipulation, actigraphy, or overnight polysomnography for quantifying sleep. Thus, it is possible that different effects would have been found between diagnosis, sleep, and physiological measures had more objective measures of sleep been employed. However, the utilization of self-reported sleep in the natural environment most closely mirrors real-world sleep behavior.

Another possible explanation of results involves self-selection bias, which likely occurred at three separate points in the study. Potential participants were emailed invitations and the study relied on volunteers for its sample. It is possible that participants with the most sensitivity or aversion to stress declined to respond to the initial study invitation. Once volunteers were given more instructions and details, participants with heightened aversion to stress may have self-selected out of the study. For example, as detailed in the results section, 19 people missed their scheduled experimental session after being screened for headache and receiving detailed study instructions, and 15 of those people were migraineurs. Especially given migraineurs' increased response to stress as detailed in existing literature, it seems likely that an increased sensitivity to stress or fear of triggering a migraine may have contributed to migraineurs declining to participate in the experimental session involving the stressor tasks. An increased sensitivity or

aversion to stress or fear of triggering migraine may also explain why 6 out of the 8 participants who withdrew from the study during the experimental session were migraineurs. All 8 of these participants cited anxiety over the speech tasks as their reason for withdrawing. If all 27 of these participants (21 of which were migraineurs) who missed their appointments or withdrew their participation had completed the study, results may have yielded different findings. Perhaps the recruitment method led to an unrepresentative sample of migraineurs and that a study deliberately sampling those with strong aversion to stress or public speaking may have produced results more consistent with this study's hypotheses. As such, this may explain why migraineurs in the current sample did not demonstrate the expected inverse relationship between sleep quantity and blood pressure, while non-headache controls did.

Given the established prevalence of sleep disturbance and chronic stress among migraineurs in the existing literature, future studies are needed to further examine relationships between these factors and possible mechanisms behind any interaction effects. Studies including clinical populations or older adults would yield results more relevant to a wider range of migraineurs, and those with larger samples and objective measurement of sleep would also be able to detect smaller effects of sleep and headache condition on physiological response. A treatment study examining CBTi's impact on physiological response to stress among migraineurs is another possible area of research, as prior studies of this type have focused exclusively on headache and sleep outcome variables. Such findings would determine if treatment addressing sleep concerns could also promote healthy physiological adaptation to stress. Given the current study's unexpected findings that among migraineurs, longer duration of sleep was associated with increased SBP in response to stress, further studies examining mechanisms behind this interaction would yield information for effective treatment (i.e., increasing physical activity or

addressing depressive symptoms). Last, given existing literature and findings from the current study, migraineurs' habituation to stress is an area worthy of further exploration and clarification, possibly with different measures than strictly cardiovascular ones (e.g., cortisol). Further studies will help clarify the roles headache diagnosis and sleep play in stress response and may help identify targets for both behavioral and pharmacological treatments.

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## APPENDIX

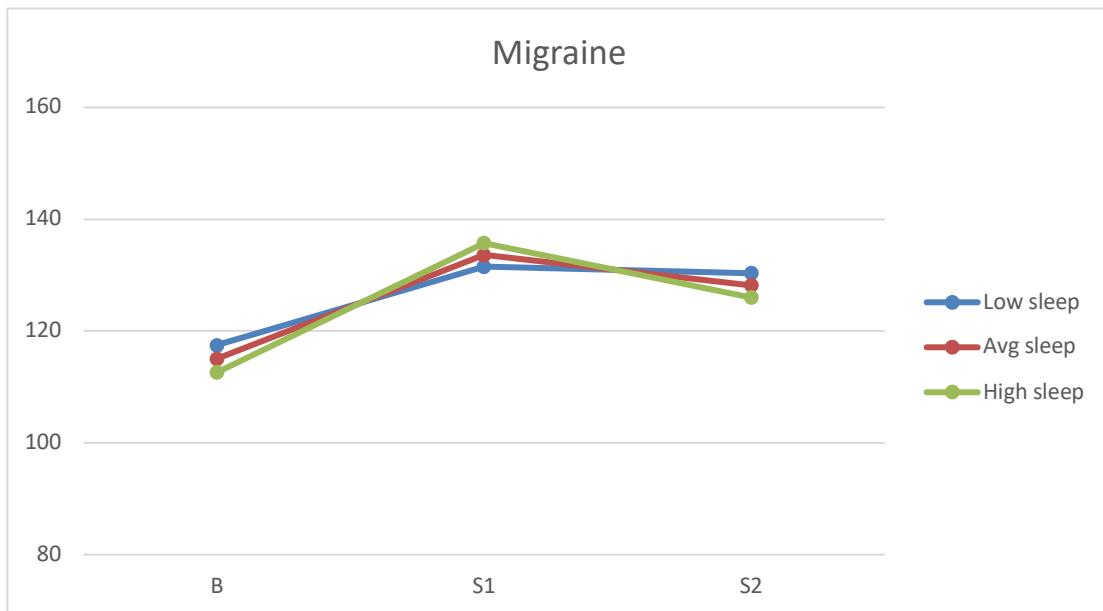
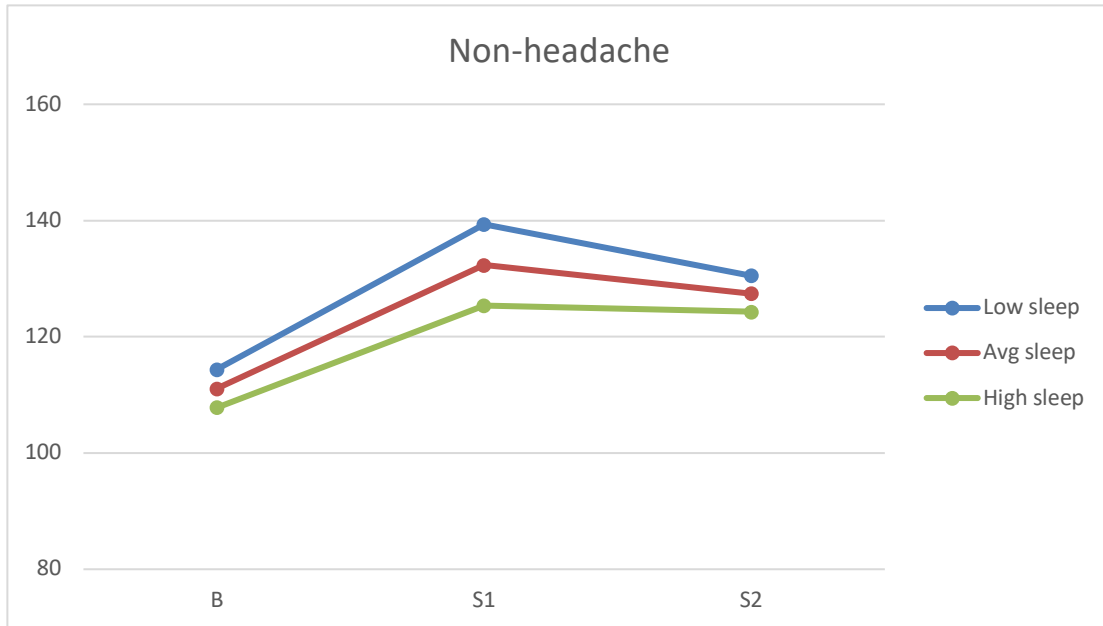


Table 1. *Demographic and Baseline Variables Across Groups*

		No Headache (n = 37)	Migraine (n=32)
Demographics			
Age	M	18.9	19.4
	(SD)	(1.6)	(4.0)
Female	N	26	30
	%	(70.3%)	(93.8%)
Male	N	11	2
	%	(29.7%)	(6.3%)
Caucasian	N	26	28
	%	(70.3%)	(87.5%)
Headache Days/Month	M	N/A	6.1
	(SD)		(3.6)
STAI Score (First admin)	M	45.4	44.2
	(SD)	(5.2)	(5.2)
STAI Score (Second admin)	M	42.2	41.0
	(SD)	(5.7)	(5.9)
PHQ-9 Score	M	4.8	7.2*
	(SD)	(4.2)	(3.4)
GAD-7 Score	M	4.3	7.7*
	(SD)	(4.2)	(4.4)
PSS-10 Score	M	20.4	21.7
	(SD)	(3.3)	(3.0)
Physiological/Sleep Variables at Baseline			
Baseline SBP (mm Hg)	M	111.3	114.9
	(SD)	(12.1)	(10.0)
Baseline DBP (mm Hg)	M	69.8	74.9*
	(SD)	(11.3)	(8.6)
Baseline HR (bpm)	M	77.4	83.8*
	(SD)	(12.6)	(11.5)
Sleep Duration Night 1 (hours)	M	7.0	7.1
	(SD)	(1.6)	(1.7)
Sleep Duration Night 2 (hours)	M	6.6	6.9
	(SD)	(1.4)	(1.7)
Sleep Quality (PSQI)	M	1.05	1.47*
	(SD)	(.58)	(.62)
Sleep Quality Night 1	M	6.6	5.8
	(SD)	(1.8)	(1.7)
Sleep Quality Night 2	M	6.2	5.5
	(SD)	(1.9)	(2.0)

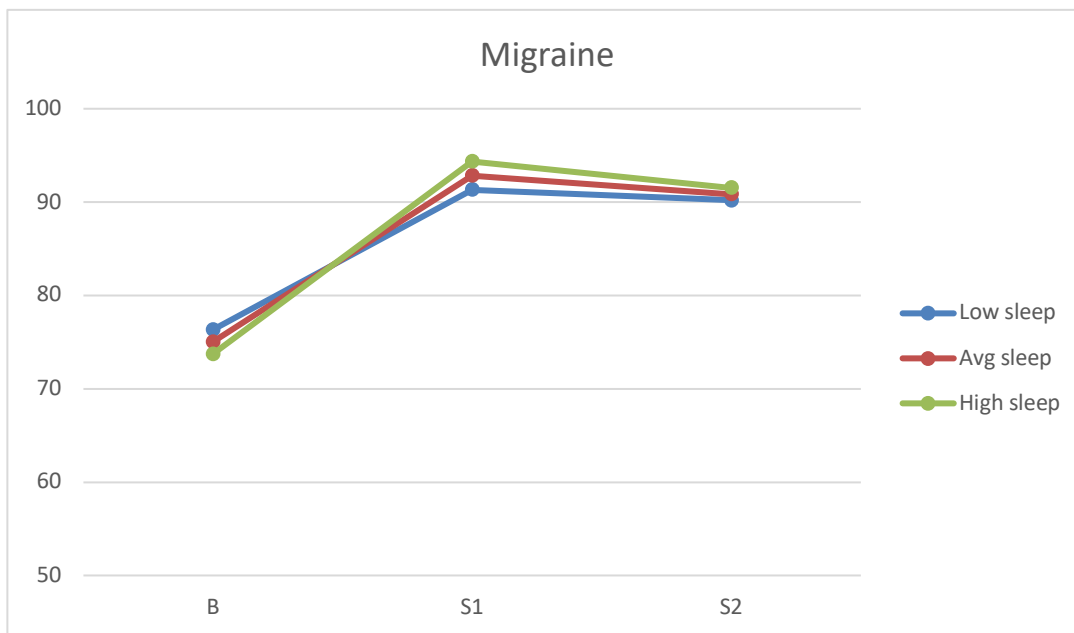
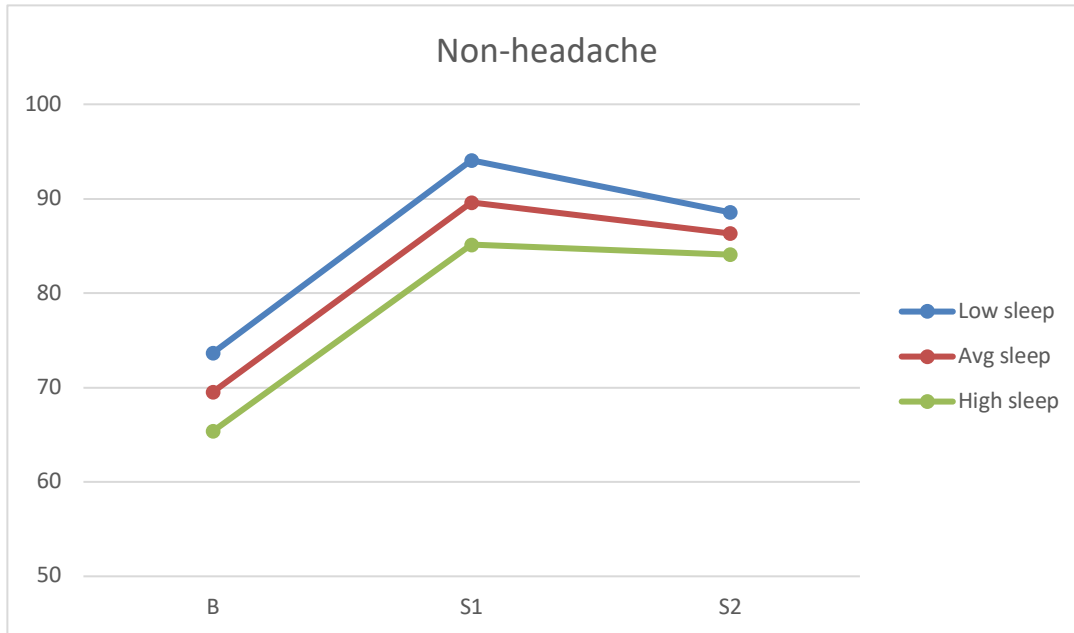
*Note.* M = mean, SD = standard deviation, N = number of participants, % = percentage of participants, SBP = systolic blood pressure, DBP = diastolic blood pressure, HR = heart rate.  
 \* = significant difference between groups ( $p < .05$ ).

Figure 1. Systolic blood pressure  
(Total sleep quantity on both nights preceding experiment)



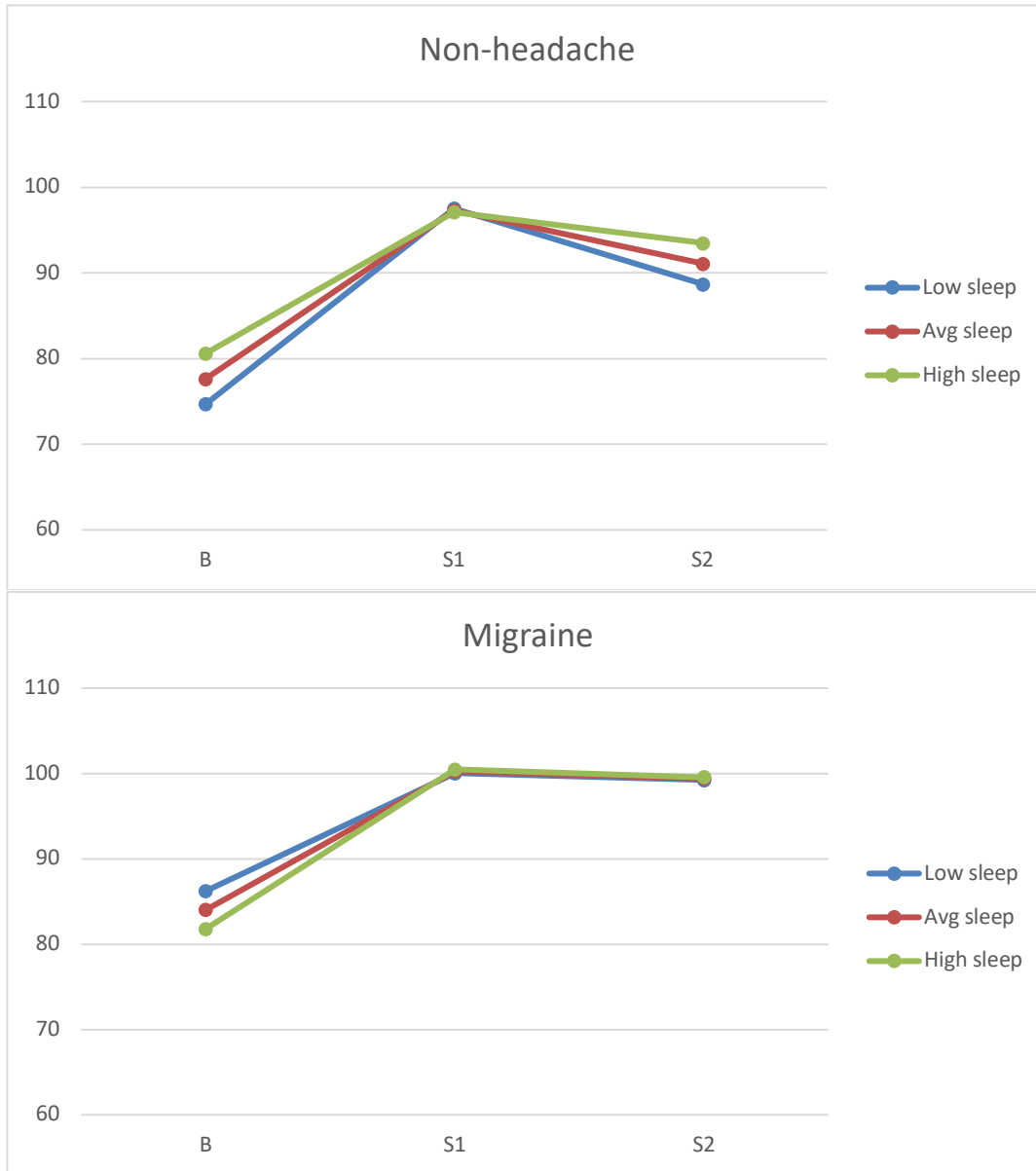
\*low sleep = 11.28 hours, average sleep = 13.80 hours, and high sleep = 16.32 total hours on both nights preceding experiment

Figure 2. Diastolic blood pressure  
(Total sleep quantity on both nights preceding experiment)



\*low sleep = 11.28 hours, average sleep = 13.80 hours, and high sleep = 16.32 total hours on both nights preceding experiment

Figure 3. Heart rate  
(Total sleep quantity on both nights preceding experiment)



\*low sleep = 11.28 hours, average sleep = 13.80 hours, and high sleep = 16.32 total hours on both nights preceding experiment

VITA

**Vanessa Lee Moynahan**

University of Mississippi  
Department of Psychology  
University, MS 38677

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**EDUCATION**

**Doctor of Philosophy, Clinical Psychology** (May 2019)  
University of Mississippi, Oxford, MS (APA-accredited)  
*Dissertation: Examining sleep as a moderator of physiological response to stress among migraineurs*  
Dissertation Chair: Todd A. Smitherman, Ph.D.  
GPA 3.84

**Master of Arts, Clinical Psychology** (July 2015)  
University of Mississippi, Oxford, MS (APA-accredited)  
*Thesis: Comparing anthropometric methods to quantify relations between adiposity and headache*  
Thesis Chair: Todd A. Smitherman, Ph.D.  
GPA 3.81

**Bachelor of Science, Psychology** (June 2008)  
University of Washington, Seattle, WA  
Honors Program  
GPA 3.67

**LICENSES/CERTIFICATIONS**

**Examination for Professional Practice in Psychology (EPPP)**  
Passed at the Doctoral Level for all states: July 2016  
**American Red Cross Disaster Training in Psychological First Aid – Certification**  
Northwest Mississippi Chapter (certified September 2011)

## CLINICAL EXPERIENCE

**United States Navy, Naval Medical Center San Diego** (November 2018-Present)  
San Diego, CA

*Lieutenant, Clinical Psychology Postdoc*

- Provide treatment for active duty Sailors and Marines
- Complete psychological assessments and fitness for duty evaluations
- Manage patient cases, including returning service members to duty or submitting PEB or ADSEP documentation
- Co-manage Central Referral Office to schedule patients with treating providers and appropriate level of care

**United States Navy, Naval Medical Center San Diego** (September 2017-September 2018)  
San Diego, CA

*Lieutenant, Pre-doctoral Psychology Intern, APA Accredited Internship*

- Provided individual and group therapy for active duty Sailors and Marines in inpatient and outpatient settings
- Completed psychological assessments, fitness for duty evaluations, and disability rating assessments (TDRL evaluations)

**University of Mississippi Counseling Center** (August 2015-May 2016)  
Oxford, MS

*Therapist*

- Provided individual therapy for adults with a range of presenting problems
- Co-led two therapy groups for eating disorders and substance abuse
- Conducted intake assessments and developed treatment plans
- Supervisor: Quinton Edwards, Ph.D.

**Autism Center of North Mississippi** (July 2014-July 2015)  
Tupelo, MS

*Applied Behavioral Analysis (ABA) Therapist and Diagnostician*

- Administered ABA therapy to children with autism spectrum disorder, developmental delay, or behavioral problems
- Conducted comprehensive psychological evaluations for children and adolescents with autism spectrum disorder, developmental delay, intellectual disability, learning disability, and anxiety and mood disorders
- Worked with SPED directors, teachers, and parents to assist with Individualized Education Program (IEP) development
- Supervisors: Scott Bethay, Ph.D. and Matthew Davison, M.S., BCBA

**University of Mississippi Psychological Assessment Clinic** (August 2014-August 2017)  
Oxford, MS

*Psychological Examiner*

- Conduct comprehensive psychological evaluations to assess for learning disabilities, Attention-Deficit/Hyperactivity Disorder, mood/anxiety disorders, personality disorders, and psychotic disorders

- Conduct University Police Department fitness for duty evaluations and bariatric evaluations
- Supervisors: Scott Gustafson, Ph.D. and Shannon Sharp, Ph.D.

**The Baddour Center**, Department of Education and Research (July 2012-July 2013)

Senatobia, MS

*Psychology Intern*

- Provided individual and group therapy for adults with intellectual disabilities in a residential facility
- Conducted assessments for cognitive ability, adaptive behavior, medication side-effects, and dementia
- Consulted with interdisciplinary professionals and developed/implemented behavior plans
- Provided staff trainings on resident empowerment
- Supervisor: Shannon Hill, Ph.D.

**University of Mississippi Psychological Services Center** (July 2012-August 2017)

Oxford, MS

*Graduate Therapist*

- Provide individual, group, and family therapy for adults and children in the greater community
- Manage a caseload with a wide range of presenting problems, including mood and personality disorders, severe psychopathology, and concerns related to health psychology such as insomnia and chronic pain
- Supervisors: Todd A. Smitherman, Ph.D., Scott Gustafson, Ph.D., Stefan Schulenberg, Ph.D., Alan Gross, Ph.D., and Tom Lombardo, Ph.D.

**Safe Place Domestic Violence Shelter** (January 2010-July 2011)

Olympia, WA

*Support Group Facilitator*

- Facilitated weekly in-house support group for domestic violence survivors

**Crisis Clinic of Thurston and Mason Counties** (January 2010-July 2011)

Olympia, WA

*Crisis Intervention Specialist*

- Provided crisis intervention and community resource referrals
- Trained new volunteers in crisis intervention skills and clinic procedures
- Supervisor: Jill Joanis, LMHC

### **TEACHING AND ADMINISTRATIVE EXPERIENCE**

**University of Mississippi** (August 2016-May 2017)

Oxford, MS

*Instructor of Record*

- Teach PSY 201 Introduction to Psychology to a class of 102 students

- Prepare and give lectures and tests, meet and provide individual feedback to students
- Supervisor: Todd Smitherman, Ph.D.

**University of Mississippi Office of Student Disability Services** (August 2014-May 2015)  
Oxford, MS

*Disability Specialist*

- Reviewed and verified applications for student accommodations and disability services
- Supervisor: Stacey Reycraft, M.Ed.

**University of Mississippi Psychological Services Center** (July 2013-July 2014)  
Oxford, MS

*Assistant to Clinic Director*

- Provided administrative oversight of graduate student therapists and trained graduate students in clinic procedures
- Managed record keeping, Titanium software, and client notes throughout department
- Advertised clinic services to the community, designed clinic publication and outreach materials
- Supervisor: Scott Gustafson, Ph.D., ABPP

### **RESEARCH EXPERIENCE**

**Behavioral Health Lab, University of Mississippi** (2012-2017)  
Oxford, MS

- Collaborate on a variety of research projects in the field of behavioral health, health psychology, and headache disorders and co-author posters presented at conferences
- Supervisor: Todd Smitherman, Ph.D.

**Trauma Lab, University of Mississippi** (2011-2012)  
Oxford, MS

*Graduate Research Assistant*

- Administered CAPS structured interviews for a study of the Pennebaker writing paradigm to reduce PTSD symptomology
- Supervisor: Tom Lombardo, Ph.D.

**St. Martin's University** (2010-2011)  
Lacey, WA

*Research Assistant*

- Assisted with studies on childhood sexual abuse
- Collected, coded, entered, cleaned, and analyzed data
- Supervisor: Katia Shkurkin, Ph.D.

**Reconnecting Youth Program, University of Washington School of Nursing** (2007-2008)  
Seattle, WA

*Research Assistant*



- Assisted with studies on school-based models designed to prevent maladaptive behaviors and depression among high-risk youth
- Recruited and interviewed parent and child participants, collected and entered data, designed visual aids, and completed office administration tasks
- Supervisor: Deborah Thomas-Jones, Ph.D.

**Project MARS (Motivating Adolescents to Reduce Sexual Risk) (2006-2008)**

University of Washington, Seattle, WA

*Research Assistant*

- Entered data, conducted literature searches, wrote article summaries, and helped maintain and update EndNote database
- Supervisor: Joshua Ginzler, Ph.D.

**University of Washington (2005-2006)**

Seattle, WA

*Research Assistant*

- Assisted with study on eating disorders among college undergraduates
- Administered surveys to participants, entered data, moved questionnaires to online format using DatStat
- Supervisor: Ursula Whiteside, doctoral candidate

**PRESENTATIONS**

**Oral Presentations**

**Moynahan, V. L.** & Smitherman, T. A. (April 2016). *Differences in perceived stress between headache disorders and headache-related variables*. Oral presentation at the 3<sup>rd</sup> annual Conference on Psychological Science, University of Mississippi, Oxford, MS.

Black, A. K., **Moynahan, V. L.**, Fulwiler, J., Smitherman, T. A. (June 2015). *The role of fear of pain in headache*. Paper presented in the New Investigator/Trainee Research Tournament at the annual scientific meeting of the American Headache Society, Washington, DC.

Hamer, J., **Moynahan, V. L.**, & Smitherman, T. A. (April 2015). *Traumatic event exposure and headache-related variables*. Oral presentation at the 2nd annual Conference on Psychological Science, University of Mississippi, Oxford, MS.

**Poster Presentations**

Hamer, J. D., Hooks, C., **Moynahan, V. L.**, & Smitherman, T. A. (June 2016). *The role of psychological acceptance in headache*. Poster presented at the 58<sup>th</sup> annual scientific meeting of the American Headache Society, San Diego, CA.

**Moynahan, V. L.**, Rogers, D. G., & Smitherman, T. A. (November 2015). *Comparing anthropometric methods to quantify relations between adiposity and headache*. Poster presented at the 49th annual meeting of the Association for Behavioral and Cognitive Therapies, Chicago, IL.

Foote, H. L., Crudup, B. M., **Moynahan, V. L.**, Landy, S. H., Roland, M., & Smitherman, T. A. (June 2014). *Psychological flexibility in migraine: acceptance and values-based action*. Poster presented at the 56<sup>th</sup> annual scientific meeting of the American Headache Society, Los Angeles, CA.

Peck, K. R., **Moynahan, V. L.**, & Smitherman, T. A. (November 2013). *Relationships among headache, self-efficacy, and impairment*. Poster presented at the 47th annual meeting of the Association for Behavioral and Cognitive Therapies, Nashville, TN.

**Moynahan, V. L.**, McDermott, M., Tull, M. T., Gratz, K. L., Houle, T. T., & Smitherman, T. A. (June 2013). *Comorbidity of migraine and affective disorders among substance dependent inpatients*. Poster presented at the 2013 International Headache Congress, Boston, MA.

### **EDITING AND REVIEWING**

#### **Ad-Hoc Reviewing**

*Behaviour Research and Therapy*

*International Journal of Behavioral Medicine*