Comparing the Effects of Cognitive and Social Stress among Individuals with Headache

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COMPARING THE EFFECTS OF COGNITIVE AND SOCIAL STRESS AMONG INDIVIDUALS WITH HEADACHE

A Thesis
presented in partial fulfillment of requirements
for the degree of Master of Arts
in Clinical Psychology
The University of Mississippi

by
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ABSTRACT

Stress is the most frequently reported trigger of headache. A number of studies have examined responses to cognitive and physical stressors among individuals with headache, primarily using self-report and various physiological measurements as outcome variables. In the stress literature more broadly, the Trier Social Stress Test (TSST) consistently has been shown to be a valid and reliable method of eliciting laboratory stress. However, this popular stress manipulation has not been previously used or promoted within the headache literature. The present study aimed to introduce the TSST to the headache literature and to experimentally compare the TSST to a cognitive stressor in its ability to elicit cardiovascular reactivity, perceived stress, and headache activity. The present sample consisted of 50 young adults (82% female) with a mean age of 18.84 years ($SD = 1.54$). Significant within- and between-group differences were observed for systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR). Mean SBP, DBP, and HR were not statistically different between conditions at Baseline but diverged during the stress manipulation. Individuals in the Social condition experienced significantly higher SBP, DBP, and HR than those in the Cognitive condition. SBP and DBP also remained elevated during the Recovery phase for those in the Social condition only. No significant differences were observed in self-reported state anxiety, perceived stress, acceptance, or headache activity. Results of the present study suggest that the TSST is a superior method of inducing a cardiovascular stress response than cognitive stress tasks among individuals with headache. Limitations and clinical implications are discussed.
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INTRODUCTION

Headache diagnoses and headache-related disability

Headache disorders are some of the most common disorders of the nervous system (WHO, 2012) and present a prevalent health issue around the world (Stovner et al., 2007). Nearly half of the adult population has experienced headache at least once in the past year, and headache disorders cause substantial disability, negatively impact quality of life, and lead to increased healthcare costs (WHO, 2012; Smitherman, Burch, Sheikh, & Loder, 2013). In the United States, headache is the fifth leading cause of emergency department visits, accounting for an estimated four million visits per year (Smitherman et al., 2013). Headache disorders can also contribute to predisposition for other illnesses. For example, depression is three times more common among individuals with migraine or severe headaches compared to headache-free individuals (WHO, 2012).

The *International Classification of Headache Disorders, Third Edition* (ICHD-3; International Headache Society, 2013) delineates four categories of primary headache disorders (i.e., those not attributable to other causes): migraine, tension-type headache (TTH), cluster headache and other trigeminal autonomic cephalalgias, and other primary headaches (Silberstein, Lipton, & Dodick, 2007). Diagnosis of migraine requires that one has experienced at least five
attacks fulfilling four conditions: 1) Migraine attacks last between 4 and 72 hours; 2) The pain is characterized by two or more of the following: unilateral location, pulsating quality, moderate or severe pain intensity, and aggravation by or avoidance of physical activity; 3) During the headache attack, at least one of the following is present: nausea, vomiting, or both photophobia (sensitivity to light) and phonophobia (sensitivity to sound); and 4) The symptoms must not be attributed to another disorder (International Headache Society, 2013).

Comparatively, TTH differs from migraine in duration and distinguishing features of the headache attacks. TTH attacks last between 30 minutes and 7 days, with pain characterized by at least two of the following attributes: bilateral location, pressing or tightening quality, mild or moderate intensity, and not aggravated by routine physical activity. Individuals with TTH do not experience nausea or vomiting but may experience either photophobia or phonophobia (not both; International Headache Society, 2013).

Migraine affects 18% of women and 6% of men in the United States (Lipton, Bigal, Diamond, Freitag, Reed, & Stewart, 2007; Smitherman et al., 2013). For both sexes, prevalence is highest between ages 25 to 55 and higher among Caucasians than African Americans. Migraine prevalence is also highest in individuals of lower income (Lipton et al., 2007; Smitherman et al., 2013). Tension-type headache is the most common of the four primary headache disorders, with a global prevalence of 42% (Stovner et al., 2007; WHO, 2012). Like migraine, TTH is also most likely among adults ages 30 to 39. However, TTH is more prevalent in Europe than in the U.S. (Stovner et al., 2007), and is only slightly more common in women (46.9%) than in men (42.3%; Crystal & Robbins, 2010). Population-based studies reveal that TTH is less burdensome to the individual than migraine but causes as much, if not more, disability at a societal level due to its greater prevalence (Stovner et al., 2007). Migraine and
TTH both impose substantial financial costs to society as a result of missed work hours or reduced productivity (Stovner et al., 2007; WHO, 2012).

**Headache triggers**

A study of 1,207 patients with primary headache disorders revealed that 75.9% reported experiencing one or more “triggers,” or precipitating stimuli, for their headache attacks (Kelman, 2007). Triggers can be categorized as internal physiological changes (e.g., hormones) or changes in the external environment (e.g., climate, stressful events, missing meals; Rothrock, 2008). It is important to note that no single factor acts as a trigger for all migraineurs, and it is rare for an individual’s reported trigger to always provoke headache attacks (Rothrock, 2008). The most commonly cited triggers include stress, menses, skipping meals, and sleep disturbance, though odors, neck pain, light, and alcohol use are reported less frequently (Kelman, 2007). The most commonly reported trigger of migraine is physical or emotional stress, which 80-90% of migraineurs report affects at least some of their headaches (Kelman, 2007; Penzien, Rains, & Holroyd, 1993).

Most patients are advised clinically to avoid their headache triggers, but some evidence suggests that avoidance may actually be maladaptive in the long-term as it can foster sensitivity to the trigger and promote social isolation and withdrawal (Martin, Lae, & Reece, 2007; Martin & MacLeod, 2009). Martin and colleagues (2007) found that among participants exposed to varying durations of a stressful task (difficult anagrams with failure feedback), those who experienced longer exposure to stress reported less negative affect than those who experienced shorter exposure. These findings suggest that participants experienced habituation or learned to cope in response to the extended stressful tasks, perhaps indicating that longer exposure may produce adaptive coping strategies. Subjective ratings of headache intensity followed a different
pattern, reflecting a cubic trend. Headache intensity ratings were highest in the shortest and longest exposure times (5 and 35 minutes, respectively), but lower in no exposure (0 minutes) and intermediate exposure times (15 and 25 minutes). Thus headache intensity varied as a function of duration of stress exposure but did not reflect a linear trend as did negative affect. It remains unclear whether avoidance of triggers or progressive exposure with the intent of desensitization or habituation is most effective in reducing frequency of headache attacks, and likely this varies as a function of trigger type (Martin et. al, 2007).

**Stress**

Stress is typically defined as the effects of an actual or perceived discrepancy between the imposed demands of the environment and the necessary resources for adapting to those demands (Houle & Nash, 2008). This discrepancy may cause a strain on the body’s equilibrium, prompting the body to engage in compensatory physiological responses to restore that equilibrium (Houle & Nash, 2008; de Kloet, Joëls, & Holsboer, 2005). The adaptive stress response induces sympathetic nervous system arousal leading to alertness, vigilance, heightened attention, and enhanced cognitive processing (de Kloet et al., 2005). Physical and psychological stressors, particularly those that are prolonged or frequent, also activate the endocrine system (i.e., hypothalamus-pituitary-adrenal axis), culminating in the release of cortisol and catecholamines via the adrenal glands. In addition to biological responses, stress also affects psychological coping mechanisms for stressful situations. For example, stress may impact one’s ability to predict events or one’s perceived control over situations (de Kloet et al., 2005).

The long-term effects of stress on the body contribute to mental illness and physical health problems (Selye, 1955). As articulated in the diathesis-stress model, individual gene expression is subject to change in response to stress and may increase sensitivity to stressful
situations (de Kloet et al., 2005). Increased vulnerability to stress can lead to a number of health consequences, such as reduced neurogenesis, impaired learning ability, reduced expression and function of neurotransmitter receptors, and overall cognitive impairment (de Kloet et al., 2005). Additionally, ample evidence suggests that chronic stress leads to a host of physical health consequences. Stress is strongly associated with the six leading causes of death (heart disease, cancer, lung ailments, accidents, cirrhosis of the liver, and suicide; Grohol, 2013), as well as obesity (Sominsky & Spencer, 2014) and impairments in immune functioning (Cohen et al., 2012).

**Stress as a headache trigger.** A close relationship exists between stress and headache. Stress can trigger individual headache episodes, act as a predisposing factor for the de novo onset of a headache disorder, exacerbate symptoms of an existing headache disorder, and worsen disability resulting from headache (Nash & Thebarge, 2006). One study found that headache attacks were more likely when preceded by two consecutive days of self-reported high stress (Houle et al., 2012). When two consecutive days were divergent in stress level, headache attacks were more likely when individuals experienced high stress the day before, but not the day of, their headache attack. Similarly, Lipton and colleagues found that a decline in perceived stress from one day to the next is associated with the onset of a migraine attack (Lipton et al., 2014). In addition to real-life stress, individuals experience headache attacks in response to experimental stress tasks from a single laboratory session (Martin, et al., 2007). These data support the clinical lore that stress is a predictor for onset of headache attacks.

Compared to non-headache individuals, migraineurs’ brains are hypersensitive between attacks, prompting intense physiological responses to routine environmental stimuli. As a result, migraineurs may exhibit diminished habituation to stressful stimuli and develop “central
“sensitization,” in which the central nervous system becomes increasingly sensitive to pain and pain-related stimuli (Maleki, Becerra, & Borsook, 2012). Maleki and colleagues (2012) proposed that while a healthy brain state reflects “adaptive allostasis,” or an adaptive response to disturbances in homeostasis, migraineurs experience “maladaptive allostatic load,” an eventual physiological cost resulting from those repeated disturbances. According to this model, the effect of stressors is additive and cumulative in migraineurs, which may impair habituation to stressful stimuli over time. Maleki and colleagues suggest that repeated migraine attacks thus function as stressors, over time further compromising one’s response to stress and compounding disease burden.

Perceived stress also affects headache indirectly by fostering maladaptive coping behaviors. For example, individuals may develop patterns of disrupted sleep (Rains, 2008), unhealthy eating habits (Nicholson & Bigal, 2008), or more frequent and excessive use of acute headache medications (Houle & Nash, 2008; Nash & Thebarge, 2006; Lake, 2008). Fear of pain and anxiety resulting from stressful life events can lead to an excessive use of acute medication, thus avoiding the onset of headache and further reinforcing unnecessary medication use. This reinforcement prevents the individual from learning to prevent or moderate headache triggers such as stress, therefore promoting a cycle of excessive medication use (Houle & Nash, 2008; Nash & Thebarge, 2006; Lake, 2008).

Laboratory stress manipulations

The literature to date consists of a number of different ways to manipulate and measure stress among individuals with headache, most of which represent cognitive or physical stressors.

Cognitive stress tasks. Insoluble anagrams, mental arithmetic, and reaction time tasks are among the most commonly used cognitive stress manipulations in the headache literature to
date. Martin, Todd, and Reece (2005) tested the effects of noise and cognitive stress (i.e., insoluble anagrams) on participants with TTH. Participants in this study provided forehead electromyography (EMG), electrocardiographic (ECG), temporal arterial distention, and systolic blood pressure measurements, as well as self-report ratings throughout the session of negative affect, aversion to noise, and headache intensity. Martin and colleagues (2005) also obtained ratings of the presence and severity of headache after the manipulation. Both noise and stress elicited headache, but neither produced significant physiological changes. A similar study by Martin and Teoh (1999) compared the effects of visual disturbance (i.e., flicker, glare, and eyestrain) and cognitive stress (i.e., insoluble anagrams) in migraineurs, TTH, and controls, using the same dependent variables. Both visual disturbance and stress precipitated headache, though their effect on physiological changes was inconclusive (Martin & Teoh, 1999). Martin, Lae, and Reece (2007) later examined the effect of insoluble anagrams on heart rate, temporal pulse amplitude, and forehead EMG. This study focused primarily on the effect of duration of exposure to the stressor, and revealed that the shortest and longest durations of exposure increased headache intensity ratings compared to intermediate durations (Martin et al., 2007).

Leistad and colleagues examined participants’ physiological responses to a 60-minute two-choice computerized reaction time test intended to elicit cognitive stress. Migraineurs experienced more pain in the frontal and temporal regions of the skull based on electromyography (EMG) responses, took longer to recover from pain during the rest period, and rated higher self-reported neck pain compared to non-headache controls (Leistad, Sand, Westgaard, Nilsen, & Stovner, 2006). Migraineurs also had significantly higher cortisol levels during pre- and post-test phases than controls (Leistad, Stovner, White, Nilsen, Westgaard, &
Sand, 2007) and exhibited less vasoconstriction and higher heart rate during the task compared to fibromyalgia patients (Leistad, Nilsen, Stovner, Westgaard, Rø, & Sand, 2008).

Prowse and Wilson (1992) recorded muscle tension via EMG responses during mental arithmetic and insoluble anagram tasks, finding that migraineurs and TTH participants had greater increases in muscle tension than controls. In contrast, Stronks and colleagues collected heart rate, blood pressure, and pulse amplitude measurements in response to a mental arithmetic stress task among an all-female sample but found no differences in physiological reactivity among migraineurs, TTH sufferers, and non-headache controls (Stronks et al., 1998). These two conflicting findings reflect minor inconsistencies in the literature with regard to physiological responses to cognitive stress. These differences could be attributable to methodological variations, as the Prowse and Wilson study incorporating an anagram task in addition to the mental arithmetic. Additionally, they included males in their sample, whereas Stronks and colleagues studied an all-female sample during the second half of their menstrual cycles. In sum, the effects of cognitive stress in individuals with headache have been quantified using several different paradigms and by examining a myriad of subjective and physiological outcome measurements to quantify stress.

**Comparing cognitive and physical stress.** The most commonly used method for inducing physical stress in individuals with headache is a cold pressor task, in which participants are instructed to submerge their hand or arm into ice water (typically 0-4º C; Hines & Brown, 1936) for as long as possible and report pain intensity ratings. Takeshima and colleagues examined the effects of a cold pressor task on platelet activation, norepinephrine, and plasma free fatty acids in blood among migraineurs, individuals with TTH, and non-headache controls (Takeshima, Takao, Urakami, Nishikawa, & Takahashi, 1989). Migraineurs and TTH sufferers
in this study exhibited increased heightened platelet activation during the stress task compared to controls. The authors posited that platelet overactivation and lower norepinephrine levels in migraine and MCH groups may reflect hypofunction of the sympathetic nervous system (Takeshima et al., 1989).

Cathcart, Winefield, Lushington, and Rolan (2009) compared chronic TTH sufferers to non-headache controls on their pain tolerance and intensity ratings in response to a cold pressor task after exposure to either cognitive stress (anagrams and mental arithmetic) or a waiting room condition. Headache sufferers in the stress condition demonstrated lower pain tolerance thresholds and reported higher pain intensity ratings compared to non-headache controls in the stress condition and to headache sufferers in the waiting room condition (Cathcart et al., 2009). These results suggest that cognitive stress produces heightened sensitivity to pain in TTH sufferers.

Hassinger, Semenchuk, and O’Brien (1999) compared responses of migraineurs and controls to cold pressor and mental arithmetic tasks. For both groups, mental arithmetic elicited a greater increase in systolic blood pressure, heart rate, and cardiac output than the cold pressor (Hassinger et al., 1999). Similarly, Domingues and colleagues compared the effects of a cold pressor task to a cognitive stressor (i.e., Stroop task) between migraineurs and non-headache controls (Domingues, Fonseca, Ziviane, Domingues, & Vassalo, 2009). Migraineurs in this experiment exhibited significant changes in heart rate and systolic blood pressure in response to the cognitive stress task compared to controls, but the groups did not differ in response to the cold pressor (Domingues et al., 2009). Collectively, the findings of these studies suggest that migraineurs experience greater cardiovascular reactivity to cognitive stressors than to physical stressors such as the cold pressor task.
**Social stress.** A third type of stress manipulation largely absent from the headache literature is provocation of social stress. According to Dickerson and Kemeny (2004), “social-evaluative threat” occurs when an individual’s self-identity can be negatively judged by others. In laboratory manipulations, this threat involves performance tasks that require cognitive effort with potential for evaluation, such as mental arithmetic and speech tasks in front of observers (Dickerson & Kemeny, 2004). However, not all mental arithmetic tasks present social-evaluative threat, such as those in which participants engage in mental arithmetic via computer (Stronks et al., 1999). In the stress literature broadly, tasks that contain both uncontrollable circumstances (i.e., outcomes not contingent on the participant’s behavior) and social-evaluative components have been consistently associated with the largest changes in cortisol and adrenocorticotropin hormone levels, as well as the longest time to physiological recovery (Dickerson & Kemeny, 2004) when compared to other stress tasks. Specifically, the combination of public speaking and cognitive stress is associated with an effect size almost twice as large as those of other stressor types (i.e., physical or cognitive; Dickerson & Kemeny, 2004).

Social stress paradigms also offer a number of advantages for experimental use. These methods are more reliable and potent in eliciting physiological reactivity than cognitive stress tasks and allow for numerous modifications in design (Allen et al., 2014). Unlike the cold pressor task, social stress does not also induce pain, which may confound interpretation of outcome variables (Allen et al., 2014). Finally, social stress has been validated across a variety of populations, including children and adolescents (Buske-Kirschbaum et al., 1997) and individuals with psychiatric disorders (Petrowski, Herold, Joraschky, Mück-Weymann, & Siepmann, 2010; Rouach et al., 2007) and substance abuse (Starcke, Holst, Brink, Veltman, & Goudriaan, 2013).
While headache researchers have often compared physical and cognitive stress manipulations, very few studies have explored social stress manipulations among individuals with headache. Some of the aforementioned headache studies have incorporated variations of social stress, such as counting backward aloud (Prowse & Wilson, 1992; Hassinger et al., 1999) or delivering a 2-minute speech in front of a video camera (Holm, Lamberty, McSherry, & Davis, 1996). Holm and colleagues (1996) had participants deliver a news story in front of a camera with continuous feedback in the form of color-coded lights instead of a panel of experimenters. Results suggested that migraineurs exhibited a longer time to recovery following heightened pulse rate during the task compared to TTH and controls, though the authors did not claim a causal relationship for this association. However, while few studies of stress as a headache trigger have incorporated a social-evaluative aspect to the laboratory manipulation, none have formally implemented the most widely used and well-validated acute stress manipulations in the literature: the Trier Social Stress Test.

The Trier Social Stress Test

The Trier Social Stress Test (TSST) was designed by Kirschbaum, Pirke, and Hellhammer at the University of Trier (1993). In response to other stress paradigms identified as producing inconsistent results or being insufficiently effective in eliciting stress-related reactivity, Kirschbaum and colleagues (1993) endeavored to establish a stress manipulation that would reliably induce activation of the hypothalamus-pituitary adrenal (HPA) axis. Formal protocol for the TSST consists of multiple phases: a waiting period (45 minutes), pre-stress physiological measurements, a stress task (20 minutes), post-stress physiological measurements, and a recovery period (20 minutes). The stress task consists of a 10-minute preparation period for an impromptu speech about why they would be a strong candidate for their dream job, a 5-
minute speech on this topic, and then 5 minutes of mental arithmetic performed aloud. Both the speech and mental arithmetic take place in front of a panel of multiple confederate experimenters and a video camera. To facilitate threat of social evaluation, participants are also told that the panel has been trained in public speaking and will be reviewing their performance. The experimenters remain expressionless throughout the speech and provide no positive or negative feedback either verbally or via body language, only instructions to continue speaking or counting for the full time allotted if the participant finishes early.

Kirschbaum and colleagues (1993) examined effects of the TSST on plasma cortisol, salivary cortisol, hormone levels (i.e., adrenocorticotropic, prolactin, and growth hormone), and heart rate in healthy volunteers. They found that the TSST produced significant increases on plasma cortisol, salivary cortisol, and all three hormone levels, and that heart rate significantly increased during the stress task. Mean peak heart rate reached 26.5 beats per minute (bpm) above baseline during the stress task period and then returned to baseline levels shortly after cessation of the task. Since its development, researchers have investigated responses to the TSST in numerous populations, including those with depression, anxiety disorders, eating disorders, and gastrointestinal disorders (Allen, Kennedy, Cryan, Dinan, & Clarke, 2014). The TSST is reliably associated with increased HPA axis activity, cardiovascular arousal, sympathetic-adrenal-medullary system activity, and ratings of subjective stress (Allen et al., 2014).

The TSST has also been compared to alternative methods of inducing stress. The TSST results in greater activation of the HPA axis and increased perceived stress ratings compared to physical stress induced by cold pressor (McRae et al., 2006), and the TSST induces larger cortisol responses than cognitive tasks or public speaking alone (Dickerson & Kemeny, 2004). The TSST also appears to induce stress more reliably than cognitive manipulations, the effects of
which vary considerably depending on the task. For instance, the Stroop test increases perceived stress and heart rate but the effect of noise on perceived stress and physiological reactivity is unclear (Allen et al., 2014). In sum, the TSST is established as a valid and reliable method for inducing stress in a majority of human participants (Allen et al., 2014), yet strikingly this task has not been formally utilized in headache.

**Goals of the present study**

Because stress is the most commonly reported trigger of migraine and TTH attacks, better understanding its relation to headache is important clinically. The literature suggests that cognitive stressors elicit headache-related physiological changes in headache sufferers more so than physical stressors. In the stress literature broadly, social-evaluative stressors have been established as the most potent means for eliciting a stress response in humans. However, this category of stress paradigms is relatively unexplored in headache. The question thus arises as to what effect social stress might have on physical and psychological variables relevant to headache sufferers as compared to cognitive stress. The present study thus sought to compare cognitive and social stress manipulations in their ability to elicit physiological reactivity, perceived stress, and headache in headache sufferers.

**Hypotheses**

*Study Goal 1: To compare the effects of social and cognitive stressors on indices of physiological reactivity.*

Hypothesis 1a: Participants in the social stress condition would exhibit greater increases in heart rate compared to individuals in the cognitive stress condition.

Hypothesis 1b: Participants in the social stress condition would exhibit greater increases in blood pressure compared to individuals in the cognitive stress condition.
Study Goal 2: To compare the effect of social and cognitive stressors on state anxiety.

Hypothesis 2: Participants in the social stress condition would report higher increases in anxiety following the manipulation compared to individuals in the cognitive stress condition.

Study Goal 3: To compare the effect of social and cognitive stressors on headache activity in the following 48 hours.

Hypothesis 3: Participants in the social stress condition would report higher mean increased headache severity during the 2 days following the stress manipulation compared to those in the cognitive stress condition.
METHOD

Participants

Undergraduate students from the University of Mississippi were recruited via Sona Systems, a web-based participant management software. Only participants who met ICHD diagnostic criteria for episodic or chronic migraine (with or without aura) or TTH were retained for participation. Power analyses indicated that 46 participants would be required for the present repeated-measures study, assuming an effect size of $f = .175$ (small to medium), power of .80, and statistical significance of $p < .05$.

Materials

Structured Diagnostic Interview for Headache-3 (Brief Version). The Structured Diagnostic Interview for Headache (SDIH) is a well-established diagnostic interview for identifying primary headache disorders (Andrew, Penzien, Rains, Knowlton, & McAnulty, 1992). The present study used a revised version of the SDIH that comports with ICHD-3 diagnostic criteria (SDIH-3; Smitherman, Penzien, Rains, Nicholson, & Houle, 2015). The measure is comprised of 17 items that assess for key characteristics of headache (e.g., location, pain intensity, frequency), as well as appendices to assess for aura, cluster headache, medication overuse, and post-traumatic headache. See Appendix A for the SDIH-3.

Perceived Stress Scale. The Perceived Stress Scale (PSS-10) is a 10-item self-report questionnaire designed to measure an individual’s perception of stress, adapted from the original 14-item version (Cohen & Williamson, 1988). Items inquire as to what degree respondents feel that situations in their lives are unpredictable, uncontrollable, and make them feel overloaded.
Responses to each question are indicated using a 0 to 4 Likert-type scale. The PSS-10 has demonstrated reliability (internal consistency alpha = .78) and validity for assessing perceived stress among community samples of at least a junior high school education (Cohen & Williamson, 1988). See Appendix B for the PSS.

**Acceptance and Action Questionnaire-II (AAQ-II).** The Acceptance and Action Questionnaire-II (AAQ-II) is a self-report questionnaire designed to measure experiential avoidance and psychological inflexibility (Bond et al., 2011). The instrument consists of 7 statements, each rated on a 1-to-7 Likert-type scale ranging from “Never true” to “Always true”. Examples of items include “I’m afraid of my feelings” and “Worries get in the way of my success”. The AAQ-II has demonstrated reliability (internal consistency alpha ranging from .78 to .88). See Appendix C for the AAQ-II.

**State Trait Anxiety Inventory.** The State Trait Anxiety Inventory (STAI) is a self-report questionnaire designed to measure anxiety and distress (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). Its most commonly used version, Form Y, consists of 20 items assessing trait anxiety (e.g., “I am a steady person”) and 20 items assessing state anxiety (e.g., “I am worried”). Responses to each question are indicated using a 4-point Likert-type scale (e.g., from “Almost Never” to “Almost Always”). The STAI has demonstrated reliability (internal consistency alpha ranging from .86 to .95), as well as construct and concurrent validity (Spielberger, 1989). For the present study, only the State Anxiety subscale (Form Y) was administered. See Appendix D for the STAI.

**Heart rate/Blood pressure.** Cardiovascular measures (heart rate, systolic blood pressure, and diastolic blood pressure) were collected via digital physiological monitoring equipment (Omron HEM-907XL Intellisense blood pressure monitor) in 2-minute intervals during each
phase of the procedure (Baseline, Stress 1, Stress 2, Recovery) and averaged within each phase. This method is consistent with the procedures of prior studies using the TSST (Kirschbaum et al., 1993; Birkett, 2011; Starcke et al., 2013).

**Headache severity measure.** Information about participants’ headache severity was obtained by means two Likert-type items to assess the intensity and disability of headache. The items were administered via Qualtrics, a web-based survey platform. See Appendix E for items of the headache severity measure.

**Trier Social Stress Test.** The social stress task followed standard protocol for the Trier Social Stress Test (TSST; Kirschbaum, Pirke, Hellhammer 1993; Birkett, 2011). The first portion of the stress task was an impromptu five-minute speech in front of a panel of confederate experimenters. Participants were informed that they would have 10 minutes to prepare a 5-minute speech describing why they would make a good candidate for their ideal job, that they would speak for 5 full minutes in front of a panel of judges who are trained in public speaking (confederate experimenters), and that the speech would be videotaped. After 10 minutes, participants were timed for 5 minutes while speaking in front of the panel. If at any point during the 5 minutes the participants were silent for more than 20 seconds, confederates prompted them to continue speaking by saying only, “You still have time remaining”.

At the end of the 5-minute speech period, participants then began the mental arithmetic task. The experimenter informed them that they would be given 5 minutes to sequentially subtract the number 13 from 1,022 and to say their answers aloud in front of the panel of confederates. If a mistake was made or zero was reached, the participants were asked to start over from 1,022. After the mental arithmetic task, participants returned to the waiting room and were asked to wait comfortably for 20 minutes.
**Cognitive stress task.** Participants in the cognitive stress condition completed a computerized task using E-Prime, designed by the first author, of matched duration as the TSST (20 minutes). The task involved 15 minutes of anagrams followed by 5 minutes of mental arithmetic, with false accuracy feedback provided visually and including periodic speed prompts. The 15-minute anagram task consisted of five blocks of 10 anagrams, adapted from the task designed by Martin and Teoh (1999). Each set of 10 anagrams included a combination of “easier”, “hard”, and “insoluble” 8- to 10-letter anagrams. After each block, one of two false feedback screens appeared, stating, “Your performance on the preceding 10 trials of anagrams was Average” or “Your performance on the preceding 10 trials of anagrams was Below Average” (Martin & Teoh, 1999). In accordance with Martin and Teoh’s (1999) protocol, participants were shown the “Average” false feedback screen following the first, second, and fourth blocks of anagrams, and the “Below Average” screen after the third and fifth blocks. The instructions and prompts for the 5-minute mental arithmetic task were analogous to that of the TSST (i.e., subtract the number 13 from 1,022), only the task was conducted entirely via computer. Participants were instructed to speak their responses to the anagrams and mental arithmetic aloud in order to mirror the speaking required in the TSST.

**Design**

The present study implemented a two-group (Social vs. Cognitive stressor) repeated measures design. Immediately following the baseline session, participants were randomly assigned to condition. Randomization was stratified as a function of gender (male vs female) and headache diagnosis (migraine vs TTH) and occurred in permuted blocks of four to one of the two stress conditions.
Procedure

Students enrolled in introductory psychology courses completed a university-wide prescreening battery that included an online version of the SDIH. Participants meeting criteria for episodic or chronic migraine (with or without aura) or TTH (minimum 4 headache days per month) were recruited through the Sona Systems software and invited to schedule and attend two individual laboratory visits (“Part 1” and “Part 2”) in exchange for modest course extra credit. In Part 1, the experimenter obtained informed consent, administered the SDIH-3 orally to confirm headache diagnosis, and provided instructions for how to complete the headache severity measures. Participants were also instructed not to eat, drink, smoke, or exercise 1 hour prior to their scheduled Part 2 session. After Part 1, participants completed daily headache severity ratings (“Self-Monitoring”) for two consecutive days preceding Part 2. The measures were sent via email and completed electronically via Qualtrics. Participants were sent daily prompts to facilitate the self-monitoring.

Figure 1 depicts the timeline of Part 2 for both conditions. During Part 2, participants were reminded of potential risks of participation that were outlined in the consent form; asked whether they had eaten, drank, or exercised in the hour prior to arrival; and asked whether they were currently experiencing a headache attack. Participants responding in the affirmative to one of these questions were asked to reschedule their Part 2 session for a later date. Participants then completed a brief demographics questionnaire and were administered the PSS-10, AAQ-II, and STAI Form Y electronically. They then began the 10-minute rest period. At the end of the 10-minute rest period, baseline blood pressure and heart rate measurements were collected for 10 minutes (“Baseline”). The blood pressure cuff required 30 seconds to reach full inflation and was programmed to inflate two times per 5-minute interval, with a 2-minute rest between each
inflation. As such, the four measurements obtained during the 10-minute Baseline period were taken at minutes 0:30, 3:00, 5:30, and 8:00.

After Baseline, participants began their assigned 20-minute stress task. Both conditions were comprised of a 15-minute activity (“Stress 1”) followed by a 5-minute mental arithmetic portion (“Stress 2”). Participants in both conditions remained seated during the stress task. Blood pressure and heart rate measurements were collected at the aforementioned 2.5-minute intervals during the stress tasks. Immediately following the stress manipulation, the STAI Form Y was re-administered (without the presence of panelists). Finally, a 10-minute rest period was allotted during which blood pressure and heart rate were again measured at four 2.5-minute intervals (“Recovery”). This completed Part 2. Participants were asked to complete another set of daily headache severity ratings on each of the two subsequent days. Upon completion of the final self-monitoring measures, participants received full debriefing information via email and provided post-debriefing re-consent.

*Figure 1:* Part 2 session procedure.
RESULTS

Statistical Analyses

Following data collection and screening, preliminary analyses included descriptive statistics of the sample and assessment of PSS scores in relation to the outcome variables and as a potential covariate. In order to identify associations between the two stress task conditions (Social vs. Cognitive) and the outcome variables of interest (heart rate, systolic blood pressure, diastolic blood pressure, state anxiety, and headache severity), five repeated-measures analysis of variance (ANOVA) were conducted, with a separate ANOVA for each dependent variable. The repeated measures term was Time (Baseline, Stress 1, Stress 2, and Recovery for the physiological variables; Baseline and Recovery for state anxiety; and pretest and posttest for the 2 days of headache severity self-monitoring). Within- and between-subjects effects, as well as interaction effects, were examined. SPSS software was used for all analyses, and the criterion for statistical significance was $p < .05$.

Participant Demographics

Sixty-eight participants completed the study. One participant opted to withdraw during the experiment, and one participant declined re-consent after debriefing. Sixteen other participants did not meet criteria for minimum headache frequency of 4 days per month upon interview and thus were excluded. The remaining analyzed sample consisted of 50 young adults (82% female) with a mean age of 18.84 years ($SD = 1.54$). The majority (76%) of the sample
was Caucasian, 16% were African American, 4% were Hispanic/Latino, 2% were Asian, and 1% identified as “Other.” Regarding headache diagnosis, 29 participants (58%) met ICHD-3 diagnostic criteria for migraine and 21 participants met criteria for TTH. Average headache frequency reported at Part 1 on the SDIH for all participants was 9.96 days/month (SD = 5.71). There were 19 participants in the Cognitive condition (12 migraine, 7 TTH) and 31 participants in the Social condition (17 migraine, 14 TTH). The demographic characteristics of the final sample are presented in Table 1 (See Appendix F). Although group sizes were unequal after excluding those without sufficient headache frequency, Levene’s test indicated no significant differences in homogeneity of variances between groups for any of the dependent variables. Additionally, analysis of the full sample of 66 participants indicated an identical pattern of results as those presented below for the 50 participants meeting inclusion criteria.

**Cardiovascular measurements**

For two participants, one blood pressure and heart rate measurement was missing during Stress 2 due to a technical error with the monitor. In these cases, their Stress 2 average was computed using last observation carried forward for the missing data points. No significant physiological differences were observed as a function of an interaction between headache diagnosis (migraine vs TTH) and time, although the present sample was not powered to detect such interaction effects.

**Systolic blood pressure.** Significant results were obtained for repeated-measures analyses regarding systolic blood pressure (SBP). Within-subject analyses indicated a significant quadratic trend in SBP across the four phases of the manipulation (Baseline, Stress 1, Stress 2, Recovery), $F(1, 48) = 20.76, p < .001, \eta^2 = .295$. These data suggest that participants in both conditions experienced significant changes in SBP over the course of the manipulation, with
bends in the regression line at Stress 1 and Stress 2. The between-subject repeated measures ANOVA was also significant, with individuals in the Social condition experiencing higher SBP than those in the Cognitive condition, $F(1, 48) = 10.124, p = .003$, partial $\eta^2 = .174$. One-way ANOVAs for between-group analyses at each individual phase of the manipulation indicate that mean SBP was not statistically different between conditions at Baseline (M social = 112.08, M cognitive = 111.16, $F(1, 49) = .089, p = .767$ (Time 1 in Figure 2), but diverged during the stress manipulation. In the Stress 1 phase (Time 2 in Figure 2), participants in the Social condition had significantly higher SBP (M = 122.57, SD = 13.77) than those in the Cognitive condition (M = 106.96, SD = 9.82), $F(1, 49) = 18.58, p < .001$. This discrepancy was maintained during the Stress 2 phase (Time 3 in Figure 2), as SBP peaked for individuals in the Social condition (M = 125.81, SD = 14.93), and increased slightly for those in the Cognitive condition (M = 109.05, SD = 14.49), $F(1, 49) = 15.164, p < .001$. In the Recovery phase (Time 4 in Figure 2), SBP decreased both for those in the Social condition (M = 116.72, SD = 12.11), and in the Cognitive condition (M = 107.68, SD = 9.55); however, a significant difference between conditions persisted $F(1, 49) = 7.64, p = .008$. These data suggest that the Social condition not only elicited a significantly higher SBP than the Cognitive condition during the stress task, but also that individuals in the Social condition continued to experience significantly elevated SBP compared to those in the Cognitive task during Recovery.
Figure 2: Repeated measures ANOVA for systolic blood pressure during experimental session.

**Diastolic blood pressure.** Significant results were also obtained for analyses regarding diastolic blood pressure (DBP), and DBP findings followed a pattern analogous to that observed for SBP. Within-subject analyses also indicate a significant quadratic trend in DBP across the four phases of the manipulation $F(1, 48) = 35.038, p < .001$, partial $\eta^2 = .422$. The between-subjects analysis was also significant, with individuals in the Social condition experiencing higher DBP than those in the Cognitive condition, $F(1, 48) = 6.932, p = .011$, partial $\eta^2 = .126$. One-way ANOVAs for between-group analyses at each individual phase of the manipulation indicated that measures of DBP for the two conditions were not statistically different at Baseline (M Cognitive = 70.79, M Social = 71.40), $F(1, 49) = .042, p = .838$, but differed during the stress manipulation. In the Stress 1 phase, participants in the Social condition had significantly higher DBP (M = 82.98, SD = 14.58) than those in the Cognitive condition, for whom DBP remained unchanged (M = 70.67, SD = 9.59), $F(1, 49) = 10.681, p = .002$. This discrepancy was
maintained during the Stress 2 phase, as DBP peaked for individuals in the Social condition (M = 86.13, SD = 13.69) and slightly increased for those in the Cognitive condition (M = 72.24, SD = 11.29), F (1, 49) = 13.776, p = .001. In the Recovery phase, DBP decreased both for those in the Social condition (M = 77.69, SD = 11.47), and in the Cognitive condition (M = 71.18, SD = 7.86); however, a significant difference between conditions remained present during Recovery, F (1, 49) = 4.736, p = .034. Consistent with the trends observed for SBP, individuals in the Social condition also continued to experience significantly elevated DBP during Recovery compared to those in the Cognitive condition.

*Figure 3*: Repeated measures ANOVA for diastolic blood pressure during experimental session.

**Heart rate.** Results concerning within-subject changes in heart rate (HR) across the four phases of manipulation yielded a significant linear trend F (1, 48) = 8.622, p = .005, partial η²=.152, and quadratic trend, F (1, 48) = 55.745, p < .001, partial η²=.537. Unlike SBP and DBP, between-subjects repeated measures analyses for HR were not significant, F (1, 48) =
1.387, \( p = .245 \), partial \( \eta^2 = .028 \). However, one-way ANOVAs for between-group analyses at each individual phase of the manipulation indicated that measures of HR were not different at Baseline (M cognitive = 78.96, M social = 79.32), \( F(1, 49) = .009, p = .923 \), but diverged during the stress manipulation. In the Stress 1 phase, participants in the Social condition had significantly higher HR (M = 89.02, SD = 12.49) than those in the Cognitive condition (M = 81.50, SD = 9.81), \( F(1, 49) = 4.98, p = .03 \). This discrepancy was maintained during the Stress 2 phase, as HR peaked for individuals in the Social condition (M = 91.74, SD = 14.53), and slightly increased for those in the Cognitive condition (M = 83.04, SD = 10.73), \( F(1, 49) = 5.088, p = .029 \). Unlike the results observed for SBP and DBP, measures of HR were not statistically different in the Recovery phase, (M cognitive = 80.97, M social = 80.31, \( F(1, 49) = .038, p = .846 \)).

*Figure 4*: Repeated measures ANOVA for heart rate during experimental session.
Self-Report Measures

**Cohen’s Perceived Stress Scale (PSS-10).** Average total score on the PSS-10 for the overall sample was 18.74 (SD = 5.93). Scores did not vary significantly as a function of sex, $F(1, 48) = 1.351, p = .251$, or headache diagnosis, $F(1, 48) = .014, p = .907$. Additionally, there were no differences in mean scores between the two conditions, $F(1, 48) = .235, p = .630$. These data suggest that participants did not differ in baseline levels of perceived stress at the time of participation.

**Acceptance and Action Questionnaire-II (AAQ-II).** Average total score on the AAQ-II for the overall sample was 22.0 (SD = 9.14). Scores did not vary significantly as a function of sex, $F(1, 48) = 2.03, p = .161$, or headache diagnosis, $F(1, 48) = .035, p = .853$. Additionally, there were no differences in mean scores between the two conditions, $F(1, 48) = .362, p = .550$. These data suggest that participants did not differ in baseline levels of emotional acceptance.

Linear regressions indicated that AAQ scores were not statistically predictive of SBP, DBP, or HR measurements at any of the four time points, although higher AAQ scores were significantly correlated with higher STAI scores at Recovery, $F(1,48) = 11.79, p = .001 (R^2 = .201)$.

**State Trait Anxiety Inventory (STAI).** For the overall sample, mean scores on the STAI were 35.74 (SD = 11.23) at Baseline and 48.31 (SD = 12.81) immediately following the stress task. There were no significant differences between conditions at Baseline, $F(1, 48) = .484, p = .490$, or after the stress task $F(1, 48) = .022, p = .882$. Scores on the STAI also did not differ by headache diagnosis at Baseline, $F(1, 48) = .019, p = .891$, or after the stress task, $F(1, 48) = .517, p = .476$. Additionally, a repeated-measures ANOVA indicated no significant between-group differences for change in STAI scores between time points $F(1, 47) = .037, p = .849$. However, a paired-samples t-test comparing means of pretest and posttest scores indicated
a significant increase in state anxiety following the manipulation for both conditions, (M pretest = 35.53, M posttest = 48.31), $t = 8.00 \ p < .001$. Although these data do not support the hypothesis that individuals in the Social condition would report greater increases in subjective anxiety on the STAI, the significant increase in scores for both conditions suggests that the manipulations were equally effective in increasing self-reported anxiety.

**Headache Self-Monitoring**

**Intensity ratings.** Average ratings of headache intensity for the overall sample were 3.71 ($SD = 1.74$) in the two days prior to the manipulation, and 3.58 ($SD = 2.1$) in the two days following. There were no significant differences between conditions for mean intensity ratings in the days prior, $F (1, 48) = 1.012, p = .320$, nor in the days after, $F (1, 48) = .402, p = .529$. A repeated-measures ANOVA indicated no significant between-group differences for change in mean headache intensity ratings $F (1, 46) = .591, p = .446$.

**Disability ratings.** Average ratings of headache disability for the overall sample were 2.65 ($SD = 1.78$) for the two days prior to the manipulation, and 2.54 ($SD = 1.78$) in the two days following. There were no significant differences between conditions for mean intensity ratings in the days prior, $F (1, 46) = 2.05, p = .159$, nor in the days after, $F (1, 46) = .209, p = .649$. A repeated-measures ANOVA indicated no significant between-group differences for change in mean headache disability ratings $F (1, 46) = .773, p = .384$. 

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DISCUSSION

The present study sought to compare cognitive and social stress manipulations in their ability to elicit cardiovascular reactivity, perceived anxiety, and headache intensity and disability in a sample of headache sufferers. To address the first study goal, we recorded measurements of blood pressure and heart rate throughout each of the stress manipulation procedures. For the second study goal, we obtained ratings of state anxiety immediately preceding and following the stress task. Regarding the third study goal, we collected ratings of headache intensity and disability on the two days preceding and two days following the experimental manipulation. Several results of the present study were consistent with hypotheses and provide relevant information for future studies that wish to examine the relationship between stress and headache.

Physiological Reactivity

Consistent with the primary hypotheses, individuals in the Social condition experienced significantly higher blood pressure and heart rate during the stress manipulation than those in the Cognitive condition. These results corroborate the findings of previous studies that have demonstrated significant increases in heart rate and blood pressure in response to the TSST among non-headache groups (Kirschbaum et al., 1993; Childs, Vicini, & De Wit, 2006; Campisi, Bravo, Cole, & Gobeil, 2012), as well as studies showing nonsignificant cardiovascular changes during a cognitive stressor among individuals with headache (i.e. difficult-to-solve anagrams; Martin et al., 2005; Martin et al., 2007). In conjunction they suggest that a social stressor such as the TSST is more potent in eliciting physiological reactivity than cognitive stressors.
The absence of differences between the Social and Cognitive conditions at Baseline indicates that the observed group differences were not attributable to preexisting discrepancies between the two groups. In the Social condition, SBP, DBP and HR rose sharply in the Stress 1 phase and then continued to rise in the Stress 2 phase. In the Cognitive condition, however, the same three measures actually decreased from baseline levels during Stress 1 and then increased only slightly between Stress 1 and Stress 2. Nearly identical patterns for blood pressure and heart rate were evident during Baseline, Stress 1, and Stress 2 phases, but not during Recovery.

In the Recovery phase, HR returned to baseline levels for both conditions and did not statistically differ from one another. This finding is consistent with results of prior TSST studies of non-headache samples (Buske-Kirschbaum, Geiben, Höllig, Morschhäuser, & Hellhammer, 2002; Childs et al., 2006) and headache studies using a cognitive stressor (Hassinger et al., 1999; Leistad et al., 2007) in which heart rate returned to baseline levels during recovery. In prior studies, blood pressure has also been observed to return to baseline levels (Hassinger et al., 1999; Campisi et al., 2012). However, in the present study, those in the Social condition maintained elevated SBP and DBP levels into the Recovery phase. These discordant patterns of blood pressure and heart rate between Stress 2 and Recovery raise some important considerations. A potential explanation for the observed delays in returning to baseline blood pressure could be a function of headache diagnoses. One study using a stress task that resembles elements of the TSST found that migraineurs took longer than healthy controls to recover following heightened pulse rate, but not blood pressure (Holm et al., 1996). Thus, more research is needed to determine whether prolonged elevation in blood pressure among headache sufferers is unique to the TSST.
While cognitive stressors have previously dominated the literature on experimental stress manipulations among headache sufferers, the present study suggests that the TSST is a superior method of inducing a cardiovascular stress response. Considering the well-established role of stress in headache, in combination with robust evidence for experimental use of the TSST, implementation of this paradigm into studies of stress within headache populations is warranted. The present findings potentially reflect a pivotal change for current approaches to studying stress as a trigger for headache. Future studies examining stress as a headache trigger would benefit from using this more potent manipulation for eliciting physiological responses to better understand the role of stress in headache. Ideally, the use of more effective simulations of stress in experimental settings can eventually translate to valuable applications in clinical settings. For instance, given that in the present study social stress elicited a notably more pronounced physiological response than cognitive stress, perhaps patients would benefit from behavioral treatments that differentiate among different forms of stress and tailor stress management strategies accordingly, rather than treating stress as a unidimensional trigger. Additional clinical contributions could eventuate from investigating whether the different forms of stress precipitate headache differentially, and improving specificity in identifying triggers could lead to more effective management of headache disorders.

**State Anxiety**

Results of the STAI revealed that participants in both conditions rated their level of state anxiety as significantly higher immediately following the manipulation compared to Baseline. These data do not support the initial hypothesis that state anxiety ratings would vary by condition. However, these data do serve as a valuable manipulation check. Given that that the two manipulations were equally successful in eliciting perceived state anxiety, the results
regarding blood pressure and heart rate can be interpreted with more confidence. Given the analogous ratings of state anxiety across both conditions, the heightened cardiovascular reactivity observed in the Social condition was not the result of the other condition being ineffective. Had individuals in the Cognitive condition reported significantly lower or unchanged ratings of state anxiety as predicted, we may have questioned whether the task was an adequate manipulation to use for comparison.

Another important consideration for these results lies in the observed discrepancy between objective and subjective measurements. Despite the significant group differences observed in heart rate and blood pressure, subjective ratings of state anxiety were nearly identical between conditions. These conflicting patterns raise questions as to why subjective ratings would be incongruent with objective measurements, what variables may influence that discrepancy, and which form of measurement is more useful for research and clinical applications. Previous studies comparing somatic measurements to participants’ subjective ratings of anxiety found a weak positive correlation between patient ratings and HR upon exposure to a cognitive stress task (i.e., Stroop task), but no correlation between ratings and SBP (McLeod, Hoehn-Saric, & Stefan, 1986). With the exception of skin conductance and HR, participants’ subjective ratings of somatic symptoms were not reliable (McLeod et al., 1986). Further research is needed to explore this phenomenon among individuals with headache, but the observed discordance between different anxiety measurement methods is a well-established phenomenon (Rachman & Hodgson, 1974) that does not invalidate the present results.

**Headache Intensity and Disability**

Contrary to our predictions, headache-related disability and pain intensity did not differ significantly by condition. The absence of a relationship between condition and subsequent
headache represents a study limitation regarding clinical application, but can perhaps be explained by referencing aspects of the study methodology. Participants were asked to record headache diary data for only two days prior and two days following the experimental manipulation. Considering that inclusion criteria for the present sample required only a minimum of 4 headache days per month, the likelihood of most participants experiencing headache in the two days following the experiment was low, such that our study was not powered to adequately address this question. Martin and Teoh (1999) collected diary data for one week after the experimental stressor, and found that headache activity among their sample was significantly greater 48-72 hours after the stress manipulation than in the initial 48 hours. Similarly, the “let-down headache” hypothesis outlined by Lipton and colleagues (2014) suggests that the decline in PSS scores from one day to the next is predictive of a migraine attack on the third day (Lipton et al., 2014). The 2-day window used in the present study might have been too narrow to capture such delayed effects. Extending the number of days on which participants record headache data would have increased the likelihood of observing subsequent effects of the manipulation, as would requiring a higher baseline headache frequency for inclusion. However, doing so could also pose challenges in inferring causal relationships between the stressor and headache activity observed several days later.

Additionally, in the present study headache ratings were not collected on the actual day of the task. It is possible that some participants experienced subsequent headache later the same day of the experimental session, which the present design would have failed to capture. Interestingly, the same study by Martin, Todd, and Reece (2005) that produced nonsignificant physiological effects observed significant results for subsequent headache activity. Martin and colleagues (2005) had participants rate the presence and intensity of headache during the
manipulation and found that 74% of individuals in their cognitive stress condition developed a headache during the task. Perhaps collecting headache ratings throughout the task, as well as on a wider and more inclusive range of days surrounding the stressor, could provide a more comprehensive examination of the effects of the TSST on headache activity.

**Limitations and Future Directions**

In addition to implementing an experimental design, the present study is strengthened by its adherence to ICHD-3 diagnostic criteria, use of both subjective and objective measures of stress/anxiety, and adoption of previously validated tasks for inducing stress in a comparative fashion. Still, our findings should be considered within the context of the study’s limitations.

First, because this sample was comprised exclusively of younger adults, it is unknown whether results of the present study are generalizable to a population of older adults. Young adults were chosen because of their high prevalence of headache and relative lack of complicating factors such as medication overuse and headache chronification, but migraine and TTH are most prevalent within a slightly older age range than that of the present sample (Lipton et al., 2007). Participants also were not screened for the presence of psychiatric comorbidities that may influence subjective ratings of perceived stress and anxiety. Future studies would benefit from including screening tools for relevant medical and psychological conditions.

Another limitation lies in the minor modifications to the formal TSST procedure that were made in favor of study feasibility. In the present study, Baseline and Recovery periods were shortened to 10 minutes each. In the protocol outlined by Kirschbaum and colleagues (1993), participants are asked to wait for up to 30 minutes during the Baseline phase and are given 30-70 minutes for Recovery. Also in Kirschbaum and colleagues’ original procedure (1993), participants enter a separate room used exclusively for the speech delivery and mental arithmetic portions of the
task. This was not possible in the present study due to space limitations and mobility constraints imposed by the blood pressure cuff. Future studies may benefit from implementing a more exact replication of the original protocol, although even the modified TSST used herein was sufficient to produce substantial physiological changes.

Overall, the present study served to introduce experimental implementation of the TSST to the headache literature. Our findings indicate that the TSST elicited significantly greater blood pressure and heart rate than the cognitive task, suggesting it is more effective for producing a cardiovascular stress response in headache sufferers and that its effects are more durable than those of a comparable cognitive stressor. These results provide considerable evidence in favor of using a social rather than cognitive stress task in future headache-stress studies and substantiate the need for further exploration in this area. Given the high base rate of headache sufferers who cite stress as a trigger, using experimental manipulations that most accurately capture resulting physiological responses could ultimately be quite valuable for clinical purposes.
LIST OF REFERENCES


Headache (Advances in Psychotherapy: Evidence-Based Practice.) Cambridge, MA: Hogrefe.


APPENDICES
Appendix A

### Structured Diagnostic Interview for Headache – 3 (Brief Version)

<table>
<thead>
<tr>
<th>Patient Name:</th>
<th>Age:</th>
<th>Sex: M F</th>
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<tbody>
<tr>
<td>Patient ID:</td>
<td>Interviewer:</td>
<td>Date: / /</td>
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</table>

The following items are adapted from the Structured Diagnostic Interview for Headache (SDIH), part of the Headache Evaluation and Diagnostic System (HEDS), which includes software for data entry and diagnostic decision-making. These materials are intended to facilitate diagnosis of selected recurrent headaches according to ICHD-3 beta (2013) diagnostic criteria. Optimal use of this interview requires expertise with the diagnostic classification.

1. Does the patient get more than one type of headache? [ ] Yes [ ] No  
   (If YES, complete a separate brief interview form for each type of headache)  
   Headache #1 #2 #3

2. Select all pain locations that apply to this type of headache: (You must check at least one)  
   [ ] frontal (A) [ ] temporal (B) [ ] occipital (C) [ ] orbital (D) [ ] supraorbital (E)

3. Select all that apply: [ ] top of head (F) [ ] base of neck (G) [ ] nasal/facial (H)

![Headache Diagram](image-url)

4. What is the intensity of pain that the patient experiences with a typical headache?  
   [ ] (Indicate rating from 0-10)

5. Which of the following symptoms are a “predominant feature” of this headache type (presume that the headache is untreated)?  
   Pain Location (Select only one): [ ] Unilateral [ ] Not Unilateral
   Pain Features (Select only one): [ ] Pulsating [ ] Pressing/Tightening (non-pulsating) [ ] Other:

6. How often does the patient experience this type of headache pain?  
   [ ] w m y (Indicate frequency in DAYS with headache per week, month, or year; query headache-free days if patient has very frequent attacks or difficulty specifying days with headache)

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9. How long does this headache last if untreated or unsuccessfully treated? (If patient falls asleep and wakes up without headache, duration of attack is until waking up. Check unrelenting if patient reports never experiencing headache less than 7 days in duration). (Indicate duration in minutes, hours, or days)

_____ m  h  d  Typical Average  _____ m  h  d  Typical Minimum  _____ m  h  d  Typical Maximum

OR  ☐ Unrelenting

10. Has anything about this headache (except frequency) changed in the last 6 months?  ☐ Yes  ☐ No

   If YES, explain:

11. Is the patient’s typical headache pain aggravated by (or cause avoidance of) routine physical activities (e.g., walking, climbing stairs, lifting, bending)?

   ☐ Yes  ☐ No

12. Do any of the following symptoms occur with this headache?

   ☐ Headache worsened by conversational noise levels (phonophobia)
   ☐ Headache worsened by normal light (photophobia)
   ☐ Nausea (Indicate intensity)  ☐ Mild  ☐ Moderate  ☐ Severe
   ☐ Vomiting (Indicate intensity)  ☐ Mild  ☐ Moderate  ☐ Severe

13. Does the patient ever experience symptoms before this headache begins?  ☐ Yes  ☐ No

   If YES, and if any reported symptoms provide evidence of visual, sensory, or other CNS symptoms, complete Section 4a

   If NO, skip to #14

14. Does this headache have severe unilateral orbital, supraorbital, or temporal pain, and/or does the interviewer suspect a cluster-type headache?  ☐ Yes  ☐ No

   If YES, complete Section 4b

   If NO, skip to #15

15. Does the patient use any medications to relieve headache pain?  ☐ Yes  ☐ No

   If YES, complete #15a, #15b

   If NO, skip to #16

15a. How long has the patient been using the medication(s) to relieve headache pain?  _____ d  w  m  y  (Indicate duration in days, weeks, months, or years)

15b. What is the frequency of medication use?  _____ days per week  _____ days per month  _____ times per day

   If use has been occurring for >3 months and at a frequency of ≥2 days/week during this time, complete Section 4c

   If NO, skip to #16

16. Did this headache develop or worsen significantly (if pre-existing) after any trauma or injury to the head or neck?  ☐ Yes  ☐ No

   If YES, complete Section 4d

   If NO, skip to #17

17. Is this headache suspected to be attributed to another ICHD-3 disorder?  ☐ Yes  ☐ No

   If aura symptoms are present, has transient ischemic attack been excluded?  ☐ Yes  ☐ No

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Section 4a  Migraine Aura Symptoms

1. How many aura attacks has the patient experienced? _____

2. Which of the following apply to the aura symptoms? (Select all that apply)
   - At least one aura symptom spreads gradually over 2-5 minutes, AND/OR 2 or more symptoms occur in succession
   - Each individual aura symptom lasts 5-60 minutes
   - At least one aura symptom is unilateral
   - The aura is accompanied, or followed within 60 minutes, by headache

3. Indicate which of the following aura symptoms are present during this type of headache: (Select all that apply)

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<th>SYMPTOM</th>
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<tbody>
<tr>
<td></td>
<td>Partial loss of sight (scotoma)</td>
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<td>Uncoordinated movements (ataxia)</td>
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<td></td>
<td>Scintillation</td>
<td></td>
<td>Dizziness (vertigo)</td>
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<td></td>
<td>Blurred vision</td>
<td></td>
<td>Ringing in ears (tinnitus)</td>
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<td></td>
<td>Fortification spectra (zig-zag lines)</td>
<td></td>
<td>Decreased hearing acuity</td>
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<td></td>
<td>Double vision</td>
<td></td>
<td>Decreased level of consciousness</td>
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<td></td>
<td>Tingling or numbness (paresthesias)</td>
<td></td>
<td>Aphasia or unclassifiable speech</td>
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<tr>
<td></td>
<td>Motor weakness (paresis)</td>
<td></td>
<td>Poorly articulated speech (dysarthria)</td>
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<td>Other:</td>
<td></td>
<td>Other:</td>
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</table>

Section 4b  Cluster Headache Symptoms

1. Have the headaches occurred in cluster periods?  □ Yes  □ No
   - If YES, complete #1a and #1b
   - If NO, skip to #2

1a. What is the total number of cluster periods experienced? _____

1b. What is the duration of cluster periods? _____ d w m y (indicate duration in days, weeks, months, or years)

2. Are the headaches separated by remission periods?  □ Yes  □ No
   - If YES, complete #2a
   - If NO, skip to #3

2a. What is the duration of remission periods? _____ d w m y (indicate duration in days, weeks, months, or years)

3. Indicate which of the following symptoms are present, as well as side affected, during this type of headache: (Select all that apply)

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<thead>
<tr>
<th>X</th>
<th>SYMPTOM</th>
<th>SIDE</th>
<th>X</th>
<th>SYMPTOM</th>
<th>SIDE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Red eye (conjunctival injection)</td>
<td>R L</td>
<td></td>
<td>Forehead and facial sweating</td>
<td>R L</td>
</tr>
<tr>
<td></td>
<td>Tearing of the eye (lacrimation)</td>
<td>R L</td>
<td></td>
<td>Forehead and facial flushing</td>
<td>R L</td>
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<tr>
<td></td>
<td>Nasal congestion</td>
<td>R L</td>
<td></td>
<td>Eyelid swelling (oedema)</td>
<td>R L</td>
</tr>
</tbody>
</table>

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### Section 4c  Medication-Overuse Headache Symptoms

1. Has intake of ergotamine, triptans, or opioids occurred on 10 or more days per month, for over 3 months? □ Yes □ No  
   If YES, indicate drug(s): □ ergotamine □ triptan □ opioid ________________

2. Has the patient’s intake of simple analgesics (e.g., acetaminophen, acetylsalicylic acid, other NSAID), occurred on 15 or more days per month, for over 3 months? □ Yes □ No  
   If YES, indicate drug: ________________

3. Has the patient’s intake of combination analgesics occurred on 10 or more days per month, for over 3 months? □ Yes □ No  
   If YES, indicate drug(s): ________________

4. Has intake of any combination of ergotamine, triptans, simple analgesics, NSAIDs, and/or opioids occurred on 10 or more days per month, for over 3 months (without overuse of any single class alone)? □ Yes □ No  
   If YES, indicate drug(s): ________________

### Section 4d  Post-Traumatic Headache Symptoms

1. Did headache develop within 7 days after head trauma (or after regaining consciousness, or after regaining the ability to sense and report pain)? □ Yes □ No

2. Was there a loss of consciousness associated with head trauma? □ Yes □ No  
   If NO, complete #2a  
   If YES, skip to #3

2a. What was the duration of unconsciousness? _____ m h d (Indicate duration in minutes, hours, or days)

3. How long has the headache continued? (Select most representative category)  
   □ Resolves within 3 months after head trauma  
   □ Persists for greater than 3 months after head trauma  
   □ Persists but 3 months have not passed since head trauma

4. Is head injury attributed to whiplash? □ Yes □ No  
   If YES, skip #5 through #9  
   If NO, complete #5 through #9

5. Did coma develop? □ Yes □ No  
   If YES, indicate severity on Glasgow Coma Scale (GCS): □ GCS <13 [moderate/severe] □ GCS >13 [mild]

6. Did post-traumatic amnesia develop and continue for longer than 24 hours? □ Yes □ No

7. Was there alteration in level of awareness for longer than 24 hours? □ Yes □ No

8. Were abnormal neuroimaging results attained suggestive of a traumatic head injury? □ Yes □ No

9. Immediately after the head injury, were any of the following present? (Select all that apply)  
   □ Transient confusion, disorientation, or impaired consciousness  
   □ Loss of memory for events immediately before or after the head injury  
   □ At least two symptoms suggestive of mild traumatic brain injury (nausea, vomiting, visual disturbances, dizziness and/or vertigo, impaired memory and/or concentration) (Circle all symptoms that apply)

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INSTRUCTIONS:

The questions in this scale ask you about your feelings and thoughts during THE LAST MONTH. In each case, please indicate your response by placing an “X” over the circle representing HOW OFTEN you felt or thought a certain way.

<table>
<thead>
<tr>
<th></th>
<th>Never</th>
<th>Almost Never</th>
<th>Sometimes</th>
<th>Fairly Often</th>
<th>Very Often</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. In the last month, how often have you been upset because of something that happened unexpectedly?</td>
<td></td>
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<tr>
<td>2. In the last month, how often have you felt that you were unable to control the important things in your life?</td>
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<tr>
<td>3. In the last month, how often have you felt nervous and “stressed”?</td>
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<tr>
<td>4. In the last month, how often have you felt confident about your ability to handle your personal problems?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. In the last month, how often have you felt that things were going your way?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. In the last month, how often have you found that you could not cope with all the things that you had to do?</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>7. In the last month, how often have you been able to control irritations in your life?</td>
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<td></td>
</tr>
<tr>
<td>8. In the last month, how often have you felt that you were on top of things?</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>9. In the last month, how often have you been angered because of things that were outside your control?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?</td>
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<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix C

AAQ-II

Below you will find a list of statements. Please rate how true each statement is for you by circling a number next to it. Use the scale below to make your choice.

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>never true</td>
<td>very seldom true</td>
<td>seldom true</td>
<td>sometimes true</td>
<td>frequently true</td>
<td>almost always true</td>
<td>always true</td>
</tr>
</tbody>
</table>

1. My painful experiences and memories make it difficult for me to live a life that I would value. 1 2 3 4 5 6 7
2. I’m afraid of my feelings. 1 2 3 4 5 6 7
3. I worry about not being able to control my worries and feelings. 1 2 3 4 5 6 7
4. My painful memories prevent me from having a fulfilling life. 1 2 3 4 5 6 7
5. Emotions cause problems in my life. 1 2 3 4 5 6 7
6. It seems like most people are handling their lives better than I am. 1 2 3 4 5 6 7
7. Worries get in the way of my success. 1 2 3 4 5 6 7

This is a one-factor measure of psychological inflexibility, or experiential avoidance. Score the scale by summing the seven items. Higher scores equal greater levels of psychological inflexibility.

Appendix D

SELF-EVALUATION QUESTIONNAIRE

Please provide the following information:

Name________________________ Date________ S____

Age________________________ Gender (Circle) M F T____

DIRECTIONS:
A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you feel right now, that is, at this moment. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best:

1. I feel calm_________________________ 1 2 3 4

2. I feel secure ________________________ 1 2 3 4

3. I am tense __________________________ 1 2 3 4

4. I feel strained ______________________ 1 2 3 4

5. I feel at ease ________________________ 1 2 3 4

6. I feel upset _________________________ 1 2 3 4

7. I am presently worrying over possible misfortunes __________ 1 2 3 4

8. I feel satisfied ______________________ 1 2 3 4

9. I feel frightened _____________________ 1 2 3 4

10. I feel comfortable ___________________ 1 2 3 4

11. I feel self-confident _________________ 1 2 3 4

12. I feel nervous ______________________ 1 2 3 4

13. I am jittery _________________________ 1 2 3 4

14. I feel indecisive ____________________ 1 2 3 4

15. I am relaxed ________________________ 1 2 3 4

16. I feel content ______________________ 1 2 3 4

17. I am worried ________________________ 1 2 3 4

18. I feel confused _____________________ 1 2 3 4

19. I feel steady ______________________ 1 2 3 4

20. I feel pleasant _____________________ 1 2 3 4
Appendix E

Headache severity measure

Please answer the following questions based on TODAY ONLY.

1. **How would you rate the intensity of your headache today?**
   
   (0 = no headache, 10 = excruciating)

<table>
<thead>
<tr>
<th></th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
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<tbody>
<tr>
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<td></td>
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</tr>
</tbody>
</table>

2. **How would you rate the disability caused by your headache today?**
   
   (0 = no disability, 10 = severe impairment/bedrest required)

<table>
<thead>
<tr>
<th></th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
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<td></td>
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<td></td>
</tr>
</tbody>
</table>
Appendix F

Demographic Characteristics of the Sample (n = 50)

<table>
<thead>
<tr>
<th>Variable</th>
<th>% or Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (% Female)</td>
<td>82.0%</td>
</tr>
<tr>
<td>Mean Age (SD)</td>
<td>18.84 (1.54)</td>
</tr>
<tr>
<td>Race (% Caucasian)</td>
<td>76.0%</td>
</tr>
<tr>
<td>Year in College (% 1st year)</td>
<td>80%</td>
</tr>
<tr>
<td>Headache days/month</td>
<td>9.96 (5.71)</td>
</tr>
<tr>
<td>PSS-10 Score</td>
<td>18.74 (5.93)</td>
</tr>
<tr>
<td>AAQ-II Score</td>
<td>22.0 (9.14)</td>
</tr>
<tr>
<td>Baseline STAI Score</td>
<td>35.53 (11.24)</td>
</tr>
<tr>
<td>Recovery STAI Score</td>
<td>48.31 (12.82)</td>
</tr>
<tr>
<td>Pretest Headache Intensity Ratings</td>
<td>3.71 (1.74)</td>
</tr>
<tr>
<td>Posttest Headache Intensity Ratings</td>
<td>3.58 (2.1)</td>
</tr>
<tr>
<td>Pretest Headache Disability Ratings</td>
<td>2.65 (1.77)</td>
</tr>
<tr>
<td>Posttest Headache Disability Ratings</td>
<td>2.54 (1.77)</td>
</tr>
</tbody>
</table>
VITA

Yelena L. Johnson, B.S.

Education

B.S. Tulane University (New Orleans, LA) 2009-2013
Major in Psychology; Minors in Spanish and Business
Honors Thesis: “To Speak or Not to Speak: Examining the effects of sexism on women’s self-esteem and well-being”
Advisor: Laurie O’Brien, Ph.D.
GPA: 3.7, Magna Cum Laude
Study Abroad: Universidad de Granada (Granada, Spain) Fall 2011

Clinical Experience

• University of Mississippi Psychological Services Center 2014-present
  Graduate Therapist
  Supervisors: Todd A. Smitherman, Ph.D., Kelly G. Wilson, Ph.D., Laura R. Johnson, Ph.D.
• Autism Center of North Mississippi (Tupelo, MS) 2015-2016
  Diagnostic Services Intern
  Supervisor: J. Scott Bethay, Ph.D., BCBA
• North Mississippi Regional Center (Oxford, MS) 2014-2015
  Psychological and Behavioral Services Intern
  Supervisor: J. Scott Bethay, Ph.D., BCBA

Workshops

• Co-Founder, Empowering You Parenting Workshop 2015-2016
  Supervisors: Sharon D. Boudreaux, M.A.T., BCBA, LBA, J. Scott Bethay, Ph.D., BCBA

Research Experience

• Graduate Research Assistant, University of Mississippi 2013-present
  Migraine and Behavioral Health Laboratory
Advisor: Todd A. Smitherman, Ph.D.

- Undergraduate Research Assistant, Tulane University 2010-2013
  Social Perception Laboratory
  Advisor: Laurie O’Brien, Ph.D.

Publications and Presentations

Book Chapters


Oral Presentations


Poster Presentations


**Professional Memberships**

- Member, American Headache Society (AHS)
- Member, Southeastern Psychological Association (SEPA)

**Teaching Experience**

Graduate Instructor, University of Mississippi 2016-2017
PSY201 General Psychology
Course Tutor, University of Mississippi 2017
PSY202 Elementary Statistics

**Editing and Reviewing Experience**

Ad-Hoc Reviewing

*Behaviour Research and Therapy*
*International Journal of Behavioral Medicine*

**Funded Grants**

- Mississippi Council on Developmental Disabilities (MSCDD) 2015-2016
  Health Mini-Grant, $10,000
  Project: Empowering You Parenting Workshop
- Newcomb-Tulane Summer Honors Research Grant 2011
  Tulane University, $6,000
  Project: Effects of Stereotype Threat on Women’s Performance in Leadership Tasks

**Awards and Achievements**

- Diversity Fellowship, University of Mississippi 2013-2018
- Semi-finalist, Three-Minute Thesis (3MT) Competition, University of Mississippi 2015
• Anne M. McPherson Memorial Student Award, Tulane University 2012
• Newcomb College Institute Conference Attendance/Presentation Grant, Tulane University 2012
• Agnes Landor Lewis Award, Tulane University 2011
• Dean’s List, Tulane University 2009-2013

Professional Experience

• Student Representative to the Full Faculty 2015-2016
  University of Mississippi Psychology Department
• Intern, Residential Treatment Center Reinvestment Program 2010
  U.S. Department of Behavioral Health (Washington, DC)
  Supervisor: James Ballard, III, Ph.D.

Leadership Experience

• Vice President of Finance, Autism Speaks University Chapter 2016-present
  University of Mississippi
• Community Service Committee Chair, Chi Omega Fraternity 2011
  Tulane University
• Chapter Secretary, Alpha Lambda Delta Honor Society 2011
  Tulane University
• College Prep Mentor, PUENTES Latin Youth Leadership Council 2012
  Tulane University
• Member, Newcomb Leadership Conference: Social Justice and Social Equality 2010
  Tulane University