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AN EXPLORATION OF RELATIONSHIPS BETWEEN PARENTING STRESS AND  
PRIMARY HEADACHE DISORDERS

A Dissertation presented for the  
Doctorate of Philosophy  
Degree in Clinical Psychology  
The University of Mississippi

YELENA LOUISE JOHNSON, M.A.

May 2019

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## ABSTRACT

Stress is the most commonly reported trigger for headache, and stress affects headache both directly through resulting physiological changes and indirectly through its effects on lifestyle and coping behaviors. However, one form of stress that has been neglected within headache is parenting. Parenting stress is defined as the imbalance between parents' perceptions of available resources and their perceived demands of parenthood. Many variables have been established as predictors of parenting stress (e.g., demographic variables, child behavior, maternal depression), though fewer studies have examined its effects on health. Given that headache and parenthood share similar demographic distributions and risk factors, exploration of the relationship between parenting stress and headache is warranted. The present study aimed to explore associations between mothers' levels of parenting stress and primary headache disorders (i.e., migraine, TTH), as well as the role of sleep quality as a potential moderator of this relationship. The sample consisted of 435 female adults with a mean age of 35.83 years ( $SD = 8.75$ ). Between-group comparisons in parenting stress scores among all three groups (Migraine, TTH, Non-Headache) yielded non-significant results. However, parenting stress was statistically associated with more headache days per month, though not with headache-related disability. Greater parenting stress was associated with poorer sleep quality, but sleep quality was not a significant moderator between parenting stress and headache frequency or headache disability. Although only a small effect was observed, the present findings suggest that parenting stress may be a worthwhile factor for further exploration of its role in headache research. Limitations and clinical implications are discussed.

## DEDICATION

This work is dedicated to my father, Noel Thomas Johnson, for his incredible love and support through thick and thin. That's Dr. Loop to you.

## ACKNOWLEDGMENTS

I would like to thank Dr. Todd A. Smitherman for believing in my potential and pushing me to be a better student, writer, clinician, and scientist. I would also like to thank the Smithereen lab, for your invaluable friendship and collegiality at every step.

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## INTRODUCTION

### **Primary headache disorders**

Primary headache disorders are among the most common medical conditions in the world (WHO, 2012) and cause substantial disability and negative impact on quality of life (Smitherman, Burch, Sheikh, & Loder, 2013; Stovner, 2007). Headache disorders contribute to a considerable amount of missed work hours and reduced productivity, which result in significant financial costs to society (Stovner et al., 2007; WHO, 2012). In addition to tangible costs, headache disorders cause psychosocial and emotional consequences, and negatively impact quality of life, even between headache attacks (Mannix & Solomon, 1998).

The *International Classification of Headache Disorders, Third Edition* (ICHD-3; International Headache Society, 2013) designates 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 (International Headache Society, 2013). The most common primary headache disorders are migraine and TTH. A migraine diagnosis requires at least five attacks fulfilling four conditions: 1) Duration of 4 to 72 hours; 2) The pain is characterized by two or more of the following: unilateral location, pulsating quality, moderate or severe 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). Some individuals with migraine also experience aura symptoms, which are transient sensory sensations, usually visually in nature (e.g., seeing spots or lines, blurry vision, scotoma), that precede the onset of pain and resolve within an hour.

In comparison, TTH differs from migraine with regard to 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: 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—though not both (International Headache Society, 2013). As such, the symptoms of TTH are typically opposite (and less severe compared to) those of migraine.

Migraine affects 18% of women and 6% of men in the United States annually (Lipton, Bigal, Diamond, Freitag, Reed, & Stewart, 2007; Smitherman et al., 2013). For both sexes, prevalence is highest between ages 25 to 55. Migraine prevalence is also greatest in individuals of lower income and higher among Whites than Blacks (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%; prevalence is highest among adults ages 30 to 39 (Lipton et al., 2007).

Headache disorders not only affect many people worldwide but often serve as a risk factor for other illnesses and negative consequences on an individual's well-being, which further compound the negative impact of headache on daily functioning (Saunders, Merikangas, Low,

Von Korff, & Kessler, 2008). Specifically, approximately 4 out of 5 of migraineurs have a comorbid physical or mental health condition (Saunders et al., 2008). Regarding physical conditions, migraine is associated with disproportionate rates of other chronic pain conditions (i.e., arthritis, back pain, neck pain), irritable bowel syndrome, and ulcers (Saunders et al., 2008), among others. Migraineurs also experience significantly higher rates of high blood pressure than individuals without headache (Odds ratio [OR] = 2.1) and individuals with non-migraine headache (OR = 1.8; Saunders et al., 2008). In terms of psychiatric conditions, migraineurs experience higher rates of mood disorders (i.e., major depressive disorder, dysthymia, bipolar), anxiety disorders (e.g., generalized anxiety disorder, panic disorder, PTSD), and prescription drug dependence as compared to those without migraine (Radat & Swendsen, 2005; Saunders et al., 2008).

When comparing migraineurs to those with non-migraine headache, migraineurs exhibit much higher rates of mental health disorders (OR = 1.6; Saunders et al., 2008). More specifically, prevalence of a psychiatric comorbidity is lowest among individuals with episodic tension-type headache, and highest among those with chronic migraine (Mongini et al., 2006). Among psychiatric comorbidities, migraineurs experience especially high rates of anxiety disorders compared to those with non-migraine headache (OR = 1.5; Saunders et al., 2008). Specifically, anxiety disorders are more prevalent among those with chronic migraine than those with chronic TTH (OR = 2.2; Juang, Wang, Fuh, Lu, & Su, 2000). This relationship is further moderated by gender. Among patients with chronic migraine, women are more likely than men to have comorbid anxiety disorders, but not depressive disorders. The opposite is true among patients with chronic TTH, in which women are more likely to have comorbid depressive

disorders, but not anxiety disorders (Juang et al., 2000).

Individuals with headache also experience disproportionate rates of sleep disorders compared to the general population (Rains & Poceta, 2006; Rasmussen, 1993). Insomnia is the most common sleep disorder among headache sufferers (Rasmussen, 1993) and is characterized by difficulty falling asleep or staying asleep, or non-restorative sleep, accompanied by daytime impairment. Daytime impairment may include fatigue, difficulties with memory or concentration, and mood disturbance (American Academy of Sleep Medicine, 2005).

Prior studies indicate that headache sufferers are more likely to sleep shorter durations, experience more difficulty falling asleep, and take longer to fall asleep after nighttime awakenings than non-headache controls (Spierings & van Hoof, 1997). In a study of 1,283 treatment-seeking migraineurs, over half reported difficulty falling asleep, and 61% reported trouble staying asleep at least occasionally (Kelman & Rains, 2005). For 24% of migraineurs and 12% of TTH sufferers, headache onset typically occurs during sleep or upon awakening. Morning headache is more common among migraineurs than individuals with TTH (Rasmussen, 1993), with approximately 71% of migraineurs reportedly experiencing morning headache (Kelman & Rains, 2005). Non-refreshing sleep is associated with migraine for both sexes, and with TTH for females (Rasmussen, 1993). Additionally, women with headache experience greater fatigue than non-headache controls (Spierings & van Hoof, 1997). Not only are problems with sleep associated with the presence of headache disorders, but they are also a known precipitating factor, or “trigger”, for the onset of individual headache attacks (Kelman & Rains, 2005), particularly when occurring in conjunction with high stress (Houle et al., 2012). Overall, comorbid physical and psychiatric conditions play an important role in headache-related

disability and as headache triggers.

Headache 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). Presumably triggers operate to induce headache by prompting changes in headache-related physiology (e.g., via activation of the sympathetic and endocrine systems, nociceptive sensitization). 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). Commonly cited triggers for migraine include stress, menses, skipping meals, and sleep disturbance; as well as odors, neck pain, light, and alcohol use, which are reported less frequently (Kelman, 2007; Walters Pellegrino, et al., 2018). The most frequently reported trigger of migraine is stress, which a majority of migraineurs report triggers their headaches at least occasionally (Walters Pellegrino, 2018).

### **Stress**

Stress is broadly 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. The perceived discrepancy between demands and resources can cause a strain on the body's equilibrium, prompting compensatory physiological responses to restore that equilibrium (Selye, 1955; de Kloet, Joëls, & Holsboer, 2005). The adaptive stress response induces activation of the endocrine system and sympathetic nervous system arousal.

More specifically, the body responds to stress by activation of the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system (SNS). The HPA axis involves an interaction among the hypothalamus, pituitary gland, and adrenal gland—three key structures

responsible for hormone secretion in the body. In response to a stressor, the hypothalamus releases corticotropin-releasing hormone (CRH), which binds to receptors on the pituitary gland. Adrenocorticotrophic hormone (ACTH) is then released from the pituitary gland and binds to receptors on the adrenal cortex, which releases cortisol. Cortisol, the body's primary stress hormone, is a steroid hormone that regulates and influences several major physiological processes (Charmandari, Tsigos, & Chrousos, 2005; Sapolsky, Romero, & Munck, 2000). For instance, cortisol increases blood pressure and cardiac output (Sambhi, Weil, Udhoji, 1965), regulates the immune system, and acts as an anti-inflammatory agent (Nash & Thebarger, 2006). In addition to cortisol release, the hypothalamus also signals the SNS to secrete catecholamines, namely epinephrine and norepinephrine. Sympathetic arousal allows for rapid physiological response to a stressor and includes stimulation of cardiovascular, respiratory, gastrointestinal, and endocrine functions (Tsigos & Chrousos, 2002). Feedback loops act to maintain cortisol and catecholamine secretion within optimal levels for an appropriate, but not excessive, stress response to given demands.

Despite these feedback loops, excessive or chronic exposure to stress can result in a multitude of physical health problems stemming from excessive activation of catecholamines and cortisol (Sapolsky, 1996; Selye, 1955; Tsigos & Chrousos, 2002). The diathesis-stress model suggests that individual gene expression can change in response to stress and may heighten sensitivity to stressful situations (de Kloet et al., 2005). Greater vulnerability to stress can result in many negative health outcomes, such as reduced neurogenesis, impaired learning ability, reduced expression and function of neurotransmitter receptors, and overall cognitive impairment (de Kloet et al., 2005). Chronic dysregulation of the adrenal cortex can lead to the development

of Cushing's syndrome (hypercortisolism) or Addison's disease (hypocortisolism). More broadly, chronic stress is strongly associated with the six leading causes of death in the United States (heart disease, cancer, lung ailments, accidents, cirrhosis of the liver, and suicide; Grohol, 2013), as well as obesity (Sominisky & Spencer, 2014) and impairments in immune functioning (Cohen et al., 2012).

In addition to physiological changes, stress also impacts psychological coping mechanisms in a reciprocal nature. For example, stress may negatively impact one's ability to predict events or one's perceived control over them (de Kloet et al., 2005), which in turn further compounds stress. Richard Lazarus proposed a well-established theory of stress that emphasized individual differences in the stress response from a primarily cognitive perspective. Lazarus' model consists of four essential components: 1) an external, causal event or agent (i.e., stressor); 2) cognitive appraisal or evaluation of the stressful event; 3) coping mechanisms used in response to the stressful demands; and 4) the stress reaction (i.e., behavioral and physiological responses; Lazarus, 1993; Lazarus & Folkman, 1984). In essence, an individual's subjective appraisals and perceptions determine the magnitude of one's stress response (Lazarus, 1993; Lazarus & Folkman, 1984).

Some studies have examined cognitive components of Lazarus' theory in the context of headache. Existing research on cognitive appraisals of pain and stressful situations in headache samples indicates that individuals with headache exhibit higher levels of maladaptive appraisals than headache-free individuals (Chiros & O'Brien, 2011). In a study by Hassinger, Semenchuk, and O'Brien (1999), compared to headache-free controls, migraineurs reported more catastrophizing in response to painful events, more wishful thinking and self-criticism in

response to a mental arithmetic task, and greater use of maladaptive coping strategies (e.g., social withdrawal) in response to stress and pain outside of the laboratory setting. Not only does stress have a unique effect on coping among individuals with headache, it also impacts other aspects of headache.

### **Stress and headache.**

An abundance of research has confirmed a close relationship between stress and headache. Compared to individuals without headache, migraineurs' central nervous systems are hypersensitive between attacks, prompting elevated physiological responses to routine internal and environmental stimuli (Bussone, 2004). Chronic stress itself is a source of nociceptive sensitization (Houle & Nash, 2008). 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 disruptions in homeostasis, migraineurs experience "maladaptive allostatic load," an eventual physiological consequence of those repeated disruptions. According to this model, the effect of stressors is additive and cumulative in migraineurs, which impairs their ability to habituate to stressful stimuli over time. Maleki and colleagues (2012) suggest that repeated migraine attacks themselves thus function as stressors, over time further compromising one's adaptive response to stress and subsequently compounding disease burden.

The mechanisms by which stress affects headache are abundant. 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 & Theborge, 2006). In a study by Houle and colleagues (2012), headache attacks were more likely when preceded by two consecutive days of self-reported high stress. 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, a study by Lipton and colleagues (2014) suggested that a decline in perceived stress from one day to the next is associated with the onset of a migraine attack (i.e., “let-down headache”; Lipton et al., 2014). Perceived stress also affects headache indirectly by fostering maladaptive coping behaviors, such that headache sufferers may develop patterns of disrupted sleep (Rains, 2008), unhealthy eating habits (Nicholson & Bigal, 2008), or excessive use of acute headache medications that further compound headache (Houle & Nash, 2008; Nash & Theborge, 2006; Lake, 2008). Stress thus not only has direct physiological consequences on an already sensitized nervous system, but it contributes to lifestyle behaviors that negatively impact both physical and mental health.

Though much of the literature on stress as a headache trigger has regarded stress as a unidimensional construct, there is growing recognition that different types of stress (e.g., cognitive, physical, social) do not function identically in relation to headache. Cognitive stressors are those which challenge cognitive faculties, such as problem-solving skills, processing speed, and working memory. In experimental tasks, cognitive stress is typically elicited using insoluble anagrams, reaction time tasks, and mental arithmetic. Cognitive stressors have been shown to elicit headache attacks in migraineurs (Martin, Todd, & Reece, 2005; Martin & Teoh, 1999). Studies also indicate that migraineurs experience more pain and take longer to

recover from pain in response to cognitive stressors than do non-headache controls (Leistad, Sand, Westgaard, Nilsen, & Stovner, 2006). Like migraineurs, chronic TTH sufferers also demonstrate lower pain tolerance thresholds and higher pain intensity ratings following a cognitive stressor compared to individuals without headache (Cathcart, Winefield, Lushington, & Rolan, 2009). The effects of physical stress have also been examined in headache samples. A physical stressor involves placing demands on the body, rather than on cognitive functions. In headache studies, physical stress manipulations typically take the form of a cold pressor task, in which participants submerge their hand or arm into extremely cold water for as long as possible and report pain intensity ratings. Overall, the literature suggests that cognitive stress is more likely than physical stress to elicit cardiovascular reactivity associated with headache (Hassinger, Semenchuk, & O'Brien 1999; Domingues, Fonseca, Ziviane, Domingues, & Vassalo, 2009). A third type of stress that has only recently received attention in headache research (Johnson, 2017) is social stress. Social stress involves a “social-evaluative threat”, in which an individual can be negatively judged by others (Dickerson & Kemeny, 2004). In the stress literature more broadly, social stress reliably elicits greater changes in cortisol and ACTH levels than physical or cognitive stressors (Dickerson & Kemeny, 2004). Preliminary data suggest that social stress may be more effective than cognitive stress in eliciting headache-related cardiovascular reactivity for individuals with migraine and TTH (Johnson, 2017), though more research is needed. In sum, the existing literature on stress as a headache trigger has rarely explored different forms of stress beyond those elicited by cognitive and physical stressors, though stress stemming from interpersonal situations represents a potentially fruitful research area for furthering our understanding of the stress-headache relationship.

## **Parenting**

One form of interpersonal stress that has been neglected within headache is parenting. Parenthood places a host of new demands on individuals that were not present before the arrival of the child, which persist and evolve over time (Deater-Deckard, 1998). In the early postpartum stage, parents undergo significant changes to daily routines, engage in unfamiliar tasks, and experience increased fatigue (Alexander & Higgins, 1993). Many mothers experience postpartum depression, especially those of lower income and lower education (Ko, Rockhill, Tong, Morrow, & Farr, 2017). In a study of heterosexual marriages, mothers experienced a steady linear increase in positive mood with regard to their baby between pregnancy and 16 months after birth, but a U-shaped pattern was evident with regard to mood toward their husband (Fleming, Ruble, Flett, & Van Wagner, 1990). Having a child does not always increase emotional distress, though when it does, mothers experience significantly greater difficulty adjusting to first-time childrearing than fathers (Hobbs, 1965; Hobbs & Cole, 1976).

In addition to adjusting to these changes in routine, mood, and the relationship with their partner, parents experience a variety of other concerns. These include parenting as a component of one's identity, their partner's perceptions of their parenting ability, concern about the child's health and safety, and their level of involvement in the child's life. According to a recent survey conducted by the Pew Research Center (2015), a majority of mothers and fathers alike reported that being a parent is "extremely important" or "very important" to their identity, and among parents who are married, most reported that it matters "a lot" to them that their spouse perceives them as a good parent. Many parents also worry about being sufficiently involved in their child's education, especially mothers who work full-time. Not surprisingly, time constraints are a

significant challenge in parenting. Approximately one-third of parents reported “always” feeling rushed, and therefore were more likely to perceive parenting as tiring and stressful than enjoyable. In fact, one in four parents reported that they find parenting stressful all (10%) or most (15%) of the time. Parenthood thus introduces a variety of new demands, as well as emotional and interpersonal challenges, that often exacerbate one’s baseline level of stress.

### **Parenting stress.**

Given the many challenges related to parenthood, “parenting stress” has been studied as its own unique construct. Parenting stress specifically relates to an imbalance between parents’ perceptions of available resources and their perceived demands of parenthood (Deater-Deckard & Scarr, 1996). Such resources may include the knowledge and/or competence necessary for daily and long-term tasks of parenting, as well as financial resources and emotional support. Parenting stress thus is likely greatest for individuals with poor knowledge, low perceived competence, and limited support (i.e., “help” from partners, family members, friends (Deater-Deckard & Scarr, 1996). Of note, parenting stress is considered to be a unique, primary phenomenon rather than a byproduct of other forms of stress (Deater-Deckard, 1998). That is, parenting stress specifically refers to effects of parenting-related stressors, which are distinct from work-related stressors, general life stressors, or co-occurring psychopathology. Parenting stress is considered a multifaceted construct comprised of: 1) the task demands of parenting; 2) the parent’s psychological well-being and behavior; 3) the qualities of the parent-child relationship; and 4) the child’s psychosocial adjustment (Deater-Deckard, 1998). The resulting parenting stress is experienced as negative feelings toward the self and/or the child/children, which are directly related to the demands of parenting (Deater-Deckard, 1998).

Deater-Deckard's (1998) proposed model of parenting stress maps well onto Lazarus' four-component process of stress more broadly. First, in the context of parenting, the stressor is parenthood and/or the child. This includes the imbalance of necessary resources for the demands of parenthood and pressure of societal norms (Deater-Deckard, 1998). Second, cognitive appraisals of these parent-specific stressors vary across, and even within, families. For example, interpreting greater child responsibility and deliberate intention for misbehavior leads to an elevated parenting stress reaction (Dix, Ruble, & Zambarano, 1989). Some appraisals are common across families, however, such as certain infant cries that are universally considered to be aversive (Frodi & Lamb, 1980; Zeslund, Sale, Maio, Huntington, & Weiseman, 1985). Third, coping mechanisms play an integral role in an individual's level of parenting stress. Prior studies indicate that adaptive coping responses (e.g., preparation for pregnancy, knowledge of child/infant development, knowledge of effective parenting strategies) are associated with reductions in parenting stress (Sommer et al., 1993), while maladaptive coping strategies (e.g., passive inactivity in response to problems in the family, denial, rumination) are associated with greater parenting stress (Barnett, Hall, & Bramlett, 1990; Miller et al., 1992). In a study of Polish families of preschool children with autism or Down syndrome, the use of emotion-oriented coping was associated with greater parenting stress than was use of task-oriented coping. In turn, task-oriented coping predicted lower levels of parenting stress among parents with typically developing children (Dabrowska & Pisula, 2010). In addition to reducing both perceived stress and stress-related physiological arousal, adaptive coping responses also quell the negative effects of the stress reaction on the quality of the parent-child relationship (Jarvis & Creasey, 1991). Finally, stress reactions unique to parenting can be measured by the parents' behavior (e.g.,

discipline, coping responses, parenting strategies implemented) and affect (e.g., feelings toward the child, desire for intimacy with the partner) toward the child and partner, and often extend to the parents' overall psychological well-being (Deater-Deckard, 1998).

### **Parenting stress correlates and risk factors.**

A number of child and parent variables have been linked to elevated parenting stress. Demographically, greater parenting stress is associated with lower parent education and lower family income (Deater-Deckard & Scarr, 1996). When comparing adolescent mothers to adult mothers, IQ, socioeconomic status, race, and education mediate the relationship between maternal age and parenting stress. (Sommer et al., 1993). Marital discord has been reliably linked with elevated parenting stress as well (Kersh, Hedvat, Hauser-Cram, Warfield, 2006). Additionally, some data suggest that parenting stress is greatest in single-parent households (Anastopoulos, Guevremont, Shelton, & DuPaul, 1992), which currently represents the second most common family living arrangement for children under 18 in the United States (Census Bureau, 2016).

The majority of data on parenting stress has been gathered primarily among mothers in heterosexual relationships, and the existing findings on differences between mothers and fathers are mixed. Generally, those data suggest few significant differences between mothers and fathers. In a sample of parents with children between ages 1 and 5, mothers and fathers reported nearly identical levels of parenting stress (Deater-Deckard & Scarr, 1996). In a study of parents of children with autism, mothers and fathers also had similar levels of parenting stress overall (Davis & Carter, 2008). Thus, most findings in the current literature suggest that mothers and fathers exhibit equal levels of parenting stress.

Various parent psychological and emotional factors are associated with parenting stress as well. For instance, self-reported maternal psychopathology is a significant predictor of elevated parenting stress (Anastopoulos et al., 1992). For mothers, higher depression scores predict greater parenting stress, which is exacerbated by difficult infant temperament (Gelfand, Teti, & Fox, 1992). For both mothers and fathers, parenting stress has also been linked to anxiety (Delvecchio, Sciandra, Finos, Mazzeschi, & Di Riso, 2015) and decreased self-efficacy (Jones & Prinz, 2005). In abusive families, parenting stress is particularly elevated for individuals who perceive their child's behavior as difficult or problematic, even when those perceptions could not be corroborated observationally (Mash & Johnston, 1990). Fortunately, prior studies have identified social/emotional support as a protective factor, even in groups at risk for experiencing high parenting stress (i.e., adolescent mothers, low-income parents; Passino et al., 1993; Richardson, Barbour, & Bubenzer, 1995). Greater parenting stress has not only been linked to various characteristics of the parents, but to factors related to the child as well.

Child behavior problems and psychological disorders also appear to influence levels of parenting stress. For example, parents of children with externalizing behaviors exhibit greater parenting stress than those of children without (Donenberg & Baker, 1993). For fathers, parenting stress appears to vary by the child's gender and "emotional intensity," such that fathers of girls who infrequently express negative emotions experience lower levels of parenting stress than fathers of more emotionally expressive girls and fathers of boys (McBride, Schoppe, & Rane, 2002). In particular, the presence of frequent aggressive behavior, severe attention-deficit/hyperactivity disorder (ADHD), or developmental disabilities in the child is associated with high parenting stress (Anastopoulos et al., 1992; Gupta, 2007). Additionally, parenting

stress is significantly higher among parents of children with autism spectrum disorder than those of children with Down syndrome or typically developing children (Dabrowska & Pisula, 2010; Hayes & Watson, 2013; Keenan, Newman, Gray, & Rinehart, 2016). Compared to only 8% of parents of typically developing children, 79% of parents of children with autism reported parenting stress scores in the clinical range (i.e.,  $\geq 90^{\text{th}}$  percentile; Keenan et al., 2016). In sum, demographic, parent, and child variables have been established as predictors of parenting stress. However, parenting stress itself also acts to influence other outcomes.

### **Effects of parenting stress.**

The large majority of research on parenting stress has been unidirectional, examining parenting stress as a byproduct of childhood disorders. Very few studies have examined its consequences, and even fewer have focused on the effects of parenting stress on the parents themselves, rather than on the child or the family system. Of note, the relationship between parenting stress and child behavior problems may be bidirectional (Neece, Green, & Baker, 2012), though directionality is difficult to confirm given the mostly correlational studies in this area.

In terms of consequences on the child, parents with higher levels of stress exhibit more irritability and more argumentative responses to their children's behavior, which in turn can exacerbate problematic behavior in the children (Kazdin, 2005). Furthermore, parenting stress can actually interfere with children's response to interventions designed to improve problematic behavior. In a study that examined the role of parenting stress in child behavior interventions, greater parenting stress predicted poorer child outcomes at posttest (Kazdin, 1995). Thus, parenting stress may both contribute to worsening behavior in children and render them less



responsive to treatments for those same behavior problems.

A limited number of studies have attempted to capture the effects of parenting stress on the parent specifically. High parenting stress has been associated with depression (Gelfand, Teti, & Fox, 1992; Kazdin, 2005) and separation anxiety (Deater-Deckard, Scarr, McCartney, & Eisenberg, 1994) in mothers. Findings of a recent longitudinal study of mothers throughout prenatal and postpartum stages suggested that parenting stress is predictive of future depression (Thomason et al., 2014). Parenting stress also contributes to poorer self-reported perceptions of physical health in mothers of children with developmental delays or who exhibit child behavior problems (Eisenhower, Baker, & Blacher, 2009). A self-report study assessing a broad variety of symptoms among parents found that parenting stress was significantly associated with increased psychological (i.e., depression, anxiety, interpersonal problems) and somatic (i.e., headache, lower back pain) complaints (Koeske & Koeske, 1990). However, these findings were not specified beyond the subscale level, thus it is unclear which specific conditions within each domain were most frequently endorsed. To date, no literature exists on the potential role of parenting stress in headache disorders specifically.

### **Parenting and Headache**

The existing literature on parents and headache is limited and narrow in focus, the major limitation being that prior studies have focused almost exclusively on headache in the children (i.e., Esposito et al., 2013; Feldman, Ortega, Joinis-Mitchell, Kuo, & Canino, 2010) rather than in the parents. For instance, Feldman and colleagues studied the effects of acculturative stress among parents of Puerto Rican background and found that greater parental stress was associated with headache in their children. Esposito and colleagues (2013) surveyed mothers of 218 Italian

children diagnosed with migraine without aura and of children without headache (matched for age, gender, and body mass index). Mothers of children with migraine had significantly higher parenting stress than mothers of non-headache controls. These studies suggest that headache disorders in children are associated with increased parenting stress, but studies rarely have explored headache among parent samples.

One study of headache in parents examined outcomes of maternal migraine on child functioning and found that migraine was associated with dysfunctional parenting behaviors (Fagan, 2003). Specifically, greater migraine-related disability was associated with having more inappropriate expectations of children (e.g., expectations that exceed the child's developmental capabilities) and favoring parent-child role reversal (e.g., tendency to use children to meet one's own needs; Fagan, 2003). Another study found that maternal migraine was associated with decreased intra-family communication as compared to communication among families with pain-free mothers and mothers with chronic back pain, such that families with migraine exhibited less openness in expressing their feelings and were less spontaneous (Kopp et al., 1995). Additionally, families of maternal migraineurs were less active in their leisure time. To date, these studies represent the extent of existing research on the relationship between parenting and headache.

This state-of-affairs is striking given that parenthood shares numerous demographic (and other) variables associated with the development and/or progression of a primary headache disorder. Data from the Center for Disease Control (2014) indicates that the mean age of first-time mothers in the United States is 26.3 years old. Thus, migraine prevalence is not only highest during peak years of adult productivity (Stovner et al., 2007; Smitherman et al., 2013) but also

coincides with the same age range as first-time parenting for women. The sex discrepancy of migraine also mirrors the proportion of women who are single-parents (77%; Census Bureau, 2016). Additionally, both headache and parenting stress are most common among individuals of lower education and lower income.

In addition to demographic variables, parenting is associated with several behavioral/lifestyle factors also characteristic of headache sufferers, particularly disturbed sleep. Data from the National Sleep Foundation (NSF; 2002; 2004) indicate that fatigue and changes in sleep patterns are pervasive challenges for parents and caregivers, as evidenced by a myriad of reported sleep disturbances. Almost half of parents (48%) report experiencing symptoms of insomnia more frequently than before they became a parent/caregiver. Parents report frequent nighttime awakenings due to their children, being awoken earlier (by their children), and getting less sleep than adults who do not have children. Specifically, fifty-three percent of parents are awakened by their child at night at least once a week, which is moderated by the age of the child (46% of parents of school-age children vs. 80% of parents with an infant). Nearly half (48%) of parents/caregivers with infants are awakened six or seven nights per week and lose an average of 55 minutes of sleep per night. The percentage of adults reporting insomnia (i.e., trouble falling asleep, trouble staying asleep, waking up too early, being unable to get back to sleep) at least a few nights a week is higher among those who have children in the household (66%) compared to those without (54%). Given the role of sleep disturbance in both parenting stress and headache, exploration of its potential role as a moderator is warranted.

### **Goals of the present study**

Recognizing that parenting and the experience of headache both function as stressors,

share similar demographic and behavioral risk factors and consequences, and may exert reciprocal influence on each other, the paucity of research on relations between parenting stress and headache is notable. Thus, the present study aimed to explore associations between parenting stress and primary headache disorders in parents. Given the strong role of sleep disturbance in both conditions, this study also aimed to explore sleep as a potential moderator of any observed relationship between parenting stress and headache.

## **Hypotheses**

*Study goal 1: To compare parenting stress between parents with and without headache.*

Hypothesis 1a: Parents who meet criteria for a primary headache disorder (migraine or TTH) will report greater levels of parenting stress than non-headache controls.

Hypothesis 1b: Parents with migraine will report greater levels of parenting stress than parents with TTH.

*Study goal 2: To examine associations between parenting stress and frequency of headache attacks and headache-related disability.*

Hypothesis 2: Among parents who endorse a primary headache disorder (migraine or TTH), greater parenting stress will be associated with higher frequency of headache attacks and greater headache-related disability.

*Study goal 3: To explore the potential role of sleep as a moderator variable in the proposed relationship between parenting stress and headache frequency/disability.*

Hypothesis 3: Poorer sleep quality will moderate relations between parenting stress and headache, such that poor sleep quality will strengthen the relationship between parenting stress and both headache frequency and disability.

## METHOD

### **Participants**

Participants were recruited via TurkPrime, a web-based platform for survey administration adopted from Mechanical Turk (MTurk) for use in research (Litman, Robinson, & Abberbock, 2016). MTurk has good-to-excellent psychometric properties and allows for a more demographically diverse and representative sample than studies conducted exclusively with college students (Buhrmester, Kwang, & Gosling, 2011). The platform also allows for recruitment of participants meeting certain demographic inclusion criteria. For the present study, only adults at least 18 years of age who were specified by MTurk as being parents were invited to participate. Status as a parent/caregiver was confirmed at the beginning of the survey, and participants who did not meet full inclusion criteria were excluded from participation. Additional eligibility criteria included female gender (to control for differential rates of migraine among women versus men) and having at least one child age 18 years or under living in the household at least part or most of the time. Parents who were not biologically related to the child (i.e., step-parents, adoptive parents) were included, as long as all other inclusion criteria were met. Each participant was compensated for completion of the questionnaire at levels consistent with prior validation studies on MTurk (Buhrmester et al., 2012). Those who initiated the survey but did

not meet inclusion criteria received prorated compensation, and those who met inclusion criteria but did not complete the full battery received compensation proportionate to their participation.

Results of previous studies examining correlates of parenting stress have generally yielded small-to-medium effect sizes (Deater-Deckard & Scarr, 1996; Jones & Prinz, 2005; Delvecchio et al., 2015). The study by Fagan and colleagues (2003) examining migraine-related disability and parenting attitudes yielded a medium-to-large effect. Thus, the present study conservatively predicted a small-to-medium effect size. Power analyses conducted based on Hypothesis 1 (i.e., 3-group one-way ANOVA) indicated that 303 participants were required for the present study, assuming a small-to-medium effect size ( $f = 0.18$ ), power of .80, and statistical significance of  $p < .05$ . Demographics of the final sample can be found under Results.

## **Materials**

**Eligibility Screener.** A brief screening survey was utilized in order to identify participants who met the aforementioned inclusion criteria. This screener can be found in Appendix A.

**Demographic Questionnaire.** The Demographic Questionnaire solicited basic demographic information such as biological sex, age, race, education, marital status, and family composition (e.g., number of children, average number of days child(ren) live in the participant's household, known medical/psychiatric diagnoses). This measure can be found in Appendix B.

**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) that was adapted for web administration. 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 C for the SDIH-3.

**Headache Impact Test-6.** The Headache Impact Test-6 (HIT-6; Kosinski et al., 2003) is a self-report measure of the impact of headache on an individual's social, cognitive, and psychological functioning. The instrument consists of six items querying the respondent on the frequency with which headache has impaired one's functioning over the last four weeks. Responses to each question are indicated using a 5-point Likert-type scale ranging from "never" to "always". A total score is calculated (range 36 to 78) to classify the respondent's level of headache-related disability into four categories of severity: little impact (scores  $\leq 49$ ), some impact (50-55), substantial impact (56-59), and very severe impact (scores  $\geq 60$ ). The HIT-6 has shown good internal consistency ( $\alpha = 0.90$ ) and test-retest reliability ( $r = 0.78$ ), as well as discriminant validity across headache diagnostic groups (Kosinski et al., 2003). See Appendix D for the HIT-6.

**Pittsburgh Sleep Quality Index.** The Pittsburgh Sleep Quality Index (PSQI) is an 18-item self-report measure of sleep quality (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Respondents provide information regarding their usual sleep habits over the past month, such as those pertaining to sleep efficiency, sleep latency, sleep disturbance, and daytime dysfunction. Examples of open-response items include "When have you usually gone to bed?" and "How long (in minutes) has it taken you to fall asleep each night?" Most items require responses on a Likert-

type scale ranging from 0 (“Not during the past month”) to 3 (“Three or more times a week”) about the frequency of various causes of difficulty sleeping. Scores are totaled to classify overall sleep quality and as a proxy for insomnia. Total possible scores range from 0 to 21, with higher scores being indicative of poorer sleep quality. The PSQI exhibits sufficient sensitivity and specificity in classifying clinical levels of sleep disturbance (Backhaus, Junghanns, Broocks, Riemann, & Hohagen, 2002; Grandner, Kripke, Yoon, & Youngstedt, 2006), and total scores are highly sensitive for identifying sleep disturbance among individuals with insomnia (Buysse et al., 1989). See Appendix E for the PSQI.

**Parental Stress Scale.** The Parental Stress Scale (PSS) is an 18-item measure of parenting stress developed by Berry and Jones (1995). This measure is used to quantify current levels of parenting stress in both mothers and fathers, and is considered valid for use with clinical and non-clinical populations. The PSS has been implemented in samples of parents with children up to 18 years of age (Fernandes, Muller, & Rodin, 2012). Responses to each item are indicated on a 5-point Likert-type scale (1 = Strongly disagree, 5 = Strongly agree) for a possible total score ranging from 18 to 90. Eight of the items are reverse-scored, so that higher total scores indicate greater levels of parenting stress. Examples of items include “I am happy in my role as a parent”, “The major source of stress in my life is my child(ren)”, and “It is difficult to balance different responsibilities because of my child(ren)”. The PSS has demonstrated adequate internal consistency (Cronbach’s  $\alpha = 0.83$ ) and test-retest reliability (0.81; Berry & Jones, 1995). Validation studies indicate that the PSS is significantly correlated with other measures of parenting stress (Berry & Jones, 1995), such as the Parenting Stress Index (PSI; Abidin, 1995). Like the PSI, the PSS specifically distinguishes parenting-related stress from general life stress,



marital stress, and financial stress (Lessenberry & Rehfeldt, 2004). However, standardization samples for the PSS were limited with regard to ethnic diversity (91-95% White; Berry & Jones, 1995), but the PSS has since been adapted for Spanish-speaking (Oronoz, Alonso-Arbiol, & Balluerka, 2007) and Chinese (Leung & Tsang, 2010) populations. See Appendix F for the PSS.

### **Procedure**

The aforementioned battery of surveys was posted to TurkPrime for internet administration between May 2018 and November 2018. Upon initiating the battery, participants were provided with information about study procedures and associated risks and benefits, and informed consent was obtained electronically. Participants were first presented with a brief screener (“Part 1”) to identify individuals who meet the aforementioned inclusion criteria. Individuals who completed Part 1 were compensated \$0.05 and were immediately invited to participate in the full survey. As stated in the consent form, individuals who indicated being male were excluded from the survey without pay. Individuals who indicated being female but having no children under the age of 19 received compensation but were not invited to participate in the full survey. If participants were identified as eligible, they were subsequently invited to participate in the full survey (“Part 2”). Part 2 included two separate attention check items. As stated in the consent form, failing the attention check items resulted in termination of the survey without pay. Participants who completed Part 2 received an additional \$0.25 in compensation.

## RESULTS

### **Statistical Analyses**

Following data collection and screening, preliminary analyses included descriptive statistics of the sample's demographics and scores on self-report measures. For Hypothesis 1, an analysis of variance (ANOVA) was conducted to determine differences in PSS scores among individuals with migraine, TTH, and non-headache controls. For Hypothesis 2, in order to identify associations between parenting stress and headache variables, two separate linear regressions were conducted on the headache sub-samples only. For Hypothesis 3, moderation analyses were conducted to explore the role of sleep quality as a moderator in the relationship between headache variables and parenting stress. Additionally, in order to identify possible covariates, additional one-way ANOVAs and chi-square analyses were conducted to examine between-group differences in the demographic variables. Variables that statistically differed among groups or were significantly associated with PSS scores were then included in analyses of covariation for Hypotheses 1 and 2. SPSS software was used for all analyses, and the criterion for statistical significance was  $p < .05$ .

### **Participant Demographics**

One thousand twenty-six participants completed the initial screener survey. Thirty-three participants were excluded for having unfinished surveys. Of the remaining 993 participants, 159

declined to participate in the full survey study (“Part 2”). Fifteen participants were excluded for failing the first attention check (i.e., “What color is the sky? *Please select Orange for this question*”), and two participants were excluded for failing the second attention check (i.e., “*Please select Reading Instructions from the list below, and not any other hobbies that you may have*”). As such, a remaining 817 participants were considered eligible for data analyses. The scoring algorithm for the SDIH indicated that 203 individuals met criteria for “probable” migraine (i.e., lacking one diagnostic criterion), 91 individuals met criteria for probable ETTH, 62 reported symptoms consistent with another primary headache disorder (i.e., cluster, posttraumatic, medication overuse) and 22 participants had insufficient data for proper placement into a diagnostic category. These 378 participants were thus excluded from the final analyses. Additionally, four participants were excluded for indicating that their child lives in their household fewer than 4 days per week.

The remaining analyzed sample consisted of 435 adult females with a mean age of 35.83 years ( $SD = 8.75$ ). The majority (76%) of the sample was Caucasian, 10% were African American, 5% were multiracial, nearly 4% were Asian, 3% were Hispanic/Latino, and less than 2% of the sample identified as either Native Hawaiian/Pacific Islander, Middle Eastern, American Indian, or Other. Two-thirds (66.1%) of the sample was married, 13% were in a committed relationship, 10% were single, 7% were divorced, 1.8% were separated, and less than 1% were widowed. Regarding education, one-third (34%) of the sample had a bachelor’s degree from a 4-year college, 28% had some college, 13% had an associate’s degree, 12% had a master’s degree, and 7.5% had a high school diploma or GED.

Regarding parenting status, nearly half (47%) of the sample reported having one child, 35% reported having two children, 11% reported having three children, 5% reported having four children, and 2% reported having five children or more. The majority of the sample (93%) reported that their children live in their household seven days of the week. Approximately one-quarter (25.7%) of the sample indicated that their child had been diagnosed with at least one medical or psychological condition. A chi-square test indicated no significant association between headache group and self-reported child medical/psychological condition. One-way ANOVAs indicated no significant between-group differences in number of children or number of days in household. Of note, statistically significant between-group differences were observed for participants' age, reflecting a greater mean age in the TTH group than migraine and non-headache groups by approximately 2 years. Age was thus identified as a covariate for subsequent analyses. The demographic characteristics of the final sample are presented in Table 1, displayed by headache diagnostic status.

### **Headache-Related Variables**

Primary headache diagnoses were made based on ICDH-3 (IHS, 2013) criteria and obtained from responses to the SDIH-3. Responses were scored using an SPSS syntax, which classifies each respondent into one of eight primary headache diagnostic categories or identifies them as non-headache controls. Results of the syntax scoring indicated that 171 participants (39.3%) met ICHD-3 diagnostic criteria for migraine, 120 participants (27.6%) met criteria for TTH, and 144 participants (33.1%) were classified as non-headache.

Means for headache-related disability, headache frequency, and headache severity are summarized in Table 2. Based on SDIH responses, average headache frequency for all

participants with headache was 6.51 days/month ( $SD = 4.89$ ), average headache severity was 5.05 ( $SD = 1.78$ ) out of 10, and average HIT-6 score was 57.94 ( $SD = 8.27$ ). As expected, migraineurs reported significantly more headache days per month, greater pain severity, and greater HIT-6 scores than individuals with TTH.

### **Parental Stress**

Means for PSS scores across diagnostic groups are reported in Table 2. Mean PSS scores for the overall sample was 41.31 ( $SD = 9.77$ ), with scores ranging from 18 to 72. Levene's test indicated no significant differences in homogeneity of variances between groups for PSS scores ( $p = .50$ ). Ancillary analyses indicated that participant age, education, marital status, and number of children were not associated with PSS scores ( $p > .05$ ). However, the reported presence of at least one medical or psychological diagnosis in the participant's child was significantly associated with parental stress  $F(1, 424) = 25.68, p < .001, \eta^2 = .057$ . Participants who endorsed the presence of one or more diagnoses had significantly greater PSS scores ( $M = 45.29$ ) than those who denied the presence of any diagnoses ( $M = 39.97$ ), and thus this variable was also identified as a covariate for subsequent analyses.

### **Sleep Quality**

Mean PSQI scores across diagnostic groups are presented in Table 2. Levene's test indicated no significant differences in homogeneity of variances between groups for PSQI scores ( $p = .071$ ). Significant between-group differences in PSQI total scores were observed,  $F(2, 431) = 9.79, p < .001, \eta^2 = .043$ . Migraineurs reported greater PSQI scores (indicative of poorer sleep quality) than individuals with TTH and non-headache controls. Linear regressions indicated that

PSQI scores were positively associated with headache days per month ( $B = 0.68, p < .01, R^2 = .15$ ) and headache severity ( $B = .09, p = .012, R^2 = .022$ ).

### **Parental Stress among Headache Groups (Study Goal 1)**

The predictions made in Hypotheses 1 and 1b were not supported. A one-way ANOVA examining between-group differences in PSS total scores among all three headache groups (migraine, TTH, non-headache) yielded non-significant results,  $F(2, 432) = 0.556, p = .574, \eta^2 = .003$  (see Table 2 for group means). Additionally, a one-way ANOVA comparing PSS scores between individuals with headache (i.e., collapsing migraine and TTH) and non-headache controls also yielded non-significant results  $F(1, 433) = 0.975, p = .324, \eta^2 = .002$ . Exploratory analyses endeavored to assess whether group differences manifested when accounting for the aforementioned covariates (i.e., participant age and presence of child diagnoses). Between-group differences in PSS scores remained non-significant when controlling for parental age and presence of child diagnoses,  $F(2, 421) = 0.366, p = .694$ .

### **Parental Stress and Headache Variables (Study Goal 2)**

The predictions made in Hypothesis 2 were partially supported. Simple linear regressions on the headache subsample indicated that greater PSS scores were significantly associated with more headache days per month ( $B = .06, p = .049, R^2 = .013$ ). PSS scores were not significantly associated with headache disability as measured by scores on the HIT-6 ( $B = .09, p = .077$ ), though this relationship approached statistical significance. As such, although between-group differences in PSS scores were not significant, results did indicate statistically significant associations between parental stress and headache frequency within the subsample of individuals with headache. An additional hierarchical regression was conducted to determine whether these

findings were maintained after controlling for the presence of reported child diagnoses, given their aforementioned association with PSS scores. As shown in Table 3, the addition of PSS scores did not improve the predictive utility of the model beyond that afforded by the presence of child diagnoses alone (i.e., increasing the variance accounted for by only 0.6%).

### **Parental Stress and Sleep Quality (Study Goal 3)**

Finally, results of a linear regression analysis indicated a significant relationship between PSS scores and PSQI scores ( $B = .09, p < .001, R^2 = .10$ ), such that greater parenting stress was associated with poorer sleep quality. However, the predictions made for Hypothesis 3 were not supported. To test Hypothesis 3, two moderation analyses were conducted using the PROCESS models developed for SPSS by Andrew Hayes (Model 1; Hayes, 2012). In the first analysis, PSQI scores were entered as the moderator variable in the regression model examining the relationship between PSS scores and headache frequency. Results revealed a non-significant interaction term ( $B = .0063, p = .48$ ), suggesting that PSQI scores do not have a statistically significant effect on the strength of the relationship between parental stress and headache frequency. Similar results were obtained when PSQI scores were entered as the moderator variable in the regression model examining the relationship between PSS scores and HIT-6 scores ( $B = -.0069, p = .65$ ).

## DISCUSSION

The present study utilized a nationwide online survey to explore the relationship between parenting stress and primary headache disorders, yielding data from several hundred women with at least one child under the age of 19 in the home on a regular basis. Only a small portion of the results of the present study were consistent with hypotheses; however, the results nevertheless provide useful information for future studies that wish to examine the relationship between parenting stress and headache.

### **Parental Stress among Headache Groups (Study Goal 1)**

Contrary to the primary hypothesis, the present findings suggest that mothers with migraine or TTH do not experience higher levels of parenting stress compared to non-headache controls. The study's adherence to an a priori power analysis defends against inadequate sample size as a contributing factor to these null findings. These data are thus inconsistent with the existing literature that illustrates a close relationship between stress and headache (Houle et al., 2012; Leistad et al., 2006; Martin, Todd, & Reece, 2005; Martin & Teoh, 1999) and the notion that migraineurs experience reduced habituation (heightened sensitivity) to stressful stimuli compared to individuals without headache (Maleki et al., 2012). Of note, the overall mean of PSS scores obtained in the present sample ( $M = 41.31$ ) reflects a rather low level of parenting stress. A review of 25 studies that have utilized the PSS indicated that average pre-intervention



PSS scores ranged from 36.89 to 63.79 (Louie, Cromer, & Berry, 2017). The low PSS scores observed in the present sample may explain the lack of significant differences among diagnostic groups.

Ancillary analyses were consistent with prior research regarding the association between the presence of their child's medical/psychological condition and greater parenting stress (Dabrowska & Pisula, 2010; Hayes & Watson, 2013; Keenan et al., 2016). These results suggest that the relationship between child disorders and parenting stress previously observed in the general population is also relevant among mothers with headache. Further research is needed in order to gain a better understanding of whether parents are more susceptible to the onset or exacerbation of primary headache disorders as a function of their child's psychiatric problems.

### **Parental Stress and Headache Variables (Study Goal 2)**

Consistent with the secondary hypothesis, among participants who endorsed a primary headache disorder, greater parenting stress was associated with a higher frequency of headache (days per month). In combination with the null results of the primary hypothesis, these data suggest that although parenting stress may not be statistically associated with the presence of a primary headache disorder, it is associated with increased frequency of attacks in an existing headache disorder. Of note, however, the observed effect was very small, as parenting stress accounted for only 1% of the variance in headache frequency, and thus should be interpreted accordingly. Given the low PSS scores of the present sample, further research is required in order to understand whether the small effect is a function of the sample or reflects a genuine absence of a meaningful relationship.

Given the large national sample, parenting stress may simply not be as relevant to mothers with primary headache disorders as we hypothesized. The inclusion of a more general measure of stress, such as the Perceived Stress Scale (Cohen & Williamson, 1988), would have allowed for more a specific examination of the effect of parenting stress compared to that of stress more broadly. Further, though many prior studies have established a strong relationship between stress and headache, fewer have compared more specific forms of stress to one another. As discussed previously, an increasing number of studies differentiate cognitive, physical, and social stress as separate constructs rather than treating stress as one conglomerate concept (Hassinger et al., 1999; Domingues et al., 2009; Johnson, 2017). Albeit limited, the existing literature suggests that different types of stress have varying effects on individuals with headache, perhaps even hierarchically. That is, prior studies suggest that cognitive stress is more potent than physical stress for eliciting cardiovascular reactivity among those with headache (Hassinger et al., 1999; Domingues et al., 2009), but that social stress may be even more potent than both (Dickerson & Kemeny, 2004; Johnson, 2017). The present findings indicate that parenting stress may have a place in this hierarchy but not be as potent a contributor to headache variables, but further exploration is necessary to determine how parenting stress compares to other, more well-established forms of stress pertaining to headache. Currently, the majority of the parenting stress literature focuses on comparisons among parents of children with various medical (Pinquart, 2018), developmental (Gupta, 2007), and psychological conditions (Anastopoulos et al., 1992; Gupta, 2007; Stone, Mares, Otten, Engels, & Janssens, 2016). Other studies have examined parenting stress as an outcome variable for interventions (Johnson et al., 2015; Misri et al., 2006) and explored its association with parenting behaviors (Vanschoonlandt,

Vanderfaeillie, Van Holen, De Maeyer, & Robberechts, 2013; Rodgers, 1998). Few studies, if any, have compared parenting stress to other forms of stress beyond the inclusion of a general stress measure. Elucidating the unique contributions of this construct to health outcomes thus remains a much-needed area of research.

The predicted, albeit small, relationship between parenting stress and headache frequency is consistent with the abundance of literature examining stress as a trigger for headache (Cathcart et al., 2009; Domingues et al., 2009; Hassinger et al., 1999; Lipton et al., 2014; Nash & Theborge, 2006). Specifically, results of the present study provide indirect evidence to support multiple studies showing that stress can worsen existing headache disorders by triggering individual headache attacks (Houle et al., 2012; Lipton et al., 2014; Martin et al., 2005) and exacerbating symptoms (Nash & Theborge, 2006). Pending empirical verification that parenting stress may actually trigger headache attacks, these findings could potentially provide valuable contributions to clinical settings. Given that parenting stress is considered a unique type of stress separate from other forms (Deater-Deckard, 1998), increased awareness of the impact of parenting stress on headache frequency might allow clinicians to provide more specialized treatment recommendations and preventive care. For instance, teaching behavioral stress management skills that target parenting stress might have value in affecting headache, though whether this approach would offer incremental utility beyond existing behavioral methods (e.g., relaxation training, biofeedback, general stress management) is unknown.

However, parenting stress was not significantly associated with greater headache-related disability as measured by the HIT-6, although this relationship approached statistical significance. Findings of previous studies suggest that obtaining disability ratings from

participants' family members is a valuable method for capturing headache-related impact and burden more comprehensively. A recent study by Buse and colleagues examined adolescents' perceived burden due to their parents' migraine and found that perceived burden was greater for adolescents of parents with chronic migraine (CM) than episodic migraine (EM; Buse et al., 2018). This perception was true across all five domains measured by the Family Burden Module – Adolescent (FBM-A; Adams et al., 2015) module: Loss of Parental Support and Reverse Caregiving, Emotional Experience, Interference with School, Missed Activities and Events, and Negative Affect. For example, adolescents of parents with CM reported twice as often as those with EM that they needed help from their parent and could not get it and missed a family outing (e.g., movie, game, dance, picnic) at least once in the past 30 days. These data suggest that parental migraine places a significant burden on the family (Buse et al., 2018), and more nuanced data may have been obtained had adolescents been surveyed about parental disability instead of parents.

The discrepancy between parent and child perceptions of headache-related impact may be a relevant consideration. Seng and colleagues (2019) obtained ratings of parental illness impact and migraine disability from parents with migraine as well as their children. Results indicated that correlations between parent and child ratings of parental migraine impact (as measured by the Parental Illness Impact Scale – Revised; PIIS-R; Morley, Selai, Schrag, Thompson, & Jahanshahi, 2010) were at most moderate but otherwise low or nonsignificant (Seng et al., 2019). More specifically, the paired-sample correlation on the Parent/Child Relationship subscale of the PIIS-R was not significant, suggesting a discrepancy between parent and child with regard to perceived impact of migraine. Future studies wishing to explore the relationship between

parenting stress and headache should consider soliciting data from family members in order to capture a broader perspective of headache-related disability as it may relate to parenting stress.

### **Parental Stress and Sleep Quality (Study Goal 3)**

Consistent with existing research, the findings of the present study indicate that greater parenting stress is associated with poorer sleep quality among mothers with and without a primary headache disorder. These data provide further support for the known inverse relationship between stress and sleep quality (Rains & Poceta, 2006) and extend it to parenting stress specifically. Contrary to hypotheses, however, the analyses conducted for the present study suggest that sleep quality is not a significant moderator in the relationship between parenting stress and headache-related variables (i.e., headache frequency and disability). That is, parenting stress was significantly associated with headache frequency regardless of reported sleep quality. However, the coefficient of determination was very small ( $R^2 = .013$ ) for the relationship between PSS scores and headache days per month and non-significant for PSS scores and headache-related disability. These factors, as well as the overall low levels of parenting stress, may explain the absence of an observed effect for sleep quality as a moderator variable.

Few existing studies on parenting stress have examined parental sleep quality as a third variable. In one study of Canadian parents of children with autism, daily diary data revealed that sleep quality moderated the relationship between parenting stressors and negative mood (da Estrela, Barker, Lantagne, & Gouin, 2018). The results of the present study suggest that this pattern may be different for mothers with headache, although those with migraine reported significantly poorer sleep than those with TTH and controls. In the existing literature, the relationship between sleep and headache is well established (Rains, 2008; Rains & Poceta, 2006;

Rasmussen, 1993). A time-series study by Houle and colleagues (2012) found that headache attacks were most likely following two days of either high stress or poor sleep. An additive effect was also observed, in that headache risk was significantly higher following days of concurrent high stress and poor sleep (Houle et al., 2012). As such, we predicted a similar interaction effect within the present sample, in that the combined effect of high parenting stress and poor sleep quality would be associated with increased impact on headache. Additional research is needed in order to determine whether the contradictory findings of the present study are a function of the variables of interest themselves (e.g., parenting stress vs. daily stress more broadly, relatively low PSS scores) or the lack of a truly meaningful interaction effect.

### **Limitations and Future Directions**

While this study is strengthened by its large sample size and adherence to ICHD-3 diagnostic criteria, limitations nevertheless exist. One limitation of the study design is the use of self-report data via an internet-based platform for data collection. Although various studies indicate that TurkPrime provides a valid and reliable method for recruiting participants (Buhrmester et al., 2012; Litman et al., 2016), self-report data using online platforms inherently involve the risk of dishonest responding by participants. Future studies may wish to employ an in-person method of data collection for enhanced verification of participants' responses. However, in terms of strengths, TurkPrime allowed for increased geographic diversity, a broader age range than that afforded by college populations, clinically significant levels of headache frequency, and more efficient data collection.

Another limitation of the present study is the use of the Parental Stress Scale (PSS) as an alternative measure to the Parental Stress Inventory (PSI; Abidin, 1995), which is considered the

gold standard instrument for measuring parenting stress. The prohibitive cost for use of the PSI, combined with limited funding for the present study, presented significant obstacles for its implementation. If feasible, future studies exploring relationships between parenting stress and headache should consider using the PSI in order to more closely match the methods of prior studies examining parenting stress as a main variable of interest. However, the PSS is considered a valid measure of parenting stress and has strong convergent validity with the PSI (Berry & Jones, 1995). Therefore, despite the popularity of the PSI in the parenting stress literature, the results obtained using the PSS in the present study are considered clinically relevant.

The present study restricted participation to women, specifically mothers, in order to control for the significantly higher incidence rate of migraine and TTH among women compared to men. Future studies might include male participants in order to examine potential effects of the parents' gender in the relationship between parenting stress and headache. Additionally, the present study collected data regarding participants' marital status but did not solicit specifics about family structure (e.g., how many caregivers live in the home) or employment (e.g., full-time, part-time, work-from-home). The findings of prior studies suggest that parenting stress does vary as a function of these variables (Deater-Deckard & Scarr, 1996; Anastopoulos et al., 1992). Therefore, the inclusion of family structure, income, and support in future studies might help clarify the null findings of the present study.

One factor that may warrant inclusion in future studies is maternal mental health as a possible covariate, given its established relationships with both parenting stress (Misri, Reebye, Milis, & Shah, 2006; Sheinkopf et al., 2006) and headache (Radat & Swendsen, 2005; Saunders et al., 2008). Parental psychopathology is associated with higher levels of parenting stress (Misri

et al., 2006; Sheinkopf et al., 2006), ineffective parenting practices, and more child behavior problems (Barry, Dunlap, Cotton, Lochman, & Wells, 2005; Cummings, Keller, & Davies, 2005; Qi & Kaiser, 2003). Greater psychological distress has been associated with greater parenting stress specifically among mothers with chronic pain (Evans, Shipton, & Keenan, 2005). Compromised mental health may also impact a parent's ability to utilize adaptive cognitive and social coping skills required to successfully manage parenting stress (Webster-Stratton, 1999). These risk factors for increased parenting stress are analogous to the various identified risk factors of primary headache disorders. As migraineurs experience higher rates of psychiatric disorders than individuals without migraine (Radat & Swendsen, 2005; Saunders et al., 2008), maternal mental health would likely be a relevant third-variable to consider in further exploration of parenting stress in the headache population.

Furthermore, the present study sought to examine the association between parenting stress and headache in the mothers, rather than headache in the child as examined in some prior studies (i.e., Esposito et al., 2013; Feldman, Ortega, Joinis-Mitchell, Kuo, & Canino, 2010). In a study by Esposito and colleagues (2013), greater parenting stress was observed among mothers of children with migraine than mothers of non-headache controls. These data, in combination with the weak findings of the present study, beg the question as to whether parenting stress is actually more relevant for *children* with primary headache disorders than for the parents themselves.

The present study was also limited by the collection of data at a single time-point. A study design that measures parenting stress across multiple time-points would be valuable for several reasons. First, based on the "let-down headache" phenomenon observed by Lipton and



colleagues (2014), as well as the time-series study by Houle and colleagues (2012), it may be necessary to measure parenting stress levels at multiple points across a more prolonged period (e.g., 1 week, 1 month) in order to capture any possible fluctuations. A daily diary study would allow for more comprehensive analyses of the relationship between parenting stress, sleep, and headache, particularly insofar as precipitation of individual attacks on a day-to-day basis is of interest.

Second, existing literature suggests that parenting stress is consistently high during infancy (Crnic & Low, 2002). Longitudinal designs comparing time-points before and after becoming first-time parents might also be a fruitful avenue for exploring the relationship between headache and the novel onset of parenting stress. The aims of the present study stemmed from the abundance of similarities between aspects of parenting and the known triggers and risk factors for headache. These include female sex, early-to-middle adulthood, increased stress, hormonal fluctuations, changes in sleep habits, and changes in eating patterns. Given that parenting suddenly imposes a variety of new stressors and changes in routines (Alexander & Higgins, 1993), future studies could collect data at multiple intervals in order to examine whether the stress of first-time parenting can potentially lead to the onset of a primary headache disorder or exacerbation of an existing headache condition.

Third, prior studies suggest that parenting stress can fluctuate across the child's developmental stages. Kanter and Proulx (2017) interviewed a sample of married mothers and obtained parenting stress data at four time-points across ages 1 and 9 of the child's life and found that parenting stress reportedly increased between ages 1 and 3, but then declined over time (Kanter & Proulx, 2017). Williford, Calkins, and Keane (2007) also observed an overall decline

in parenting stress across the early childhood years (i.e., between ages 2 and 5). Given the amount of variability in the demands placed on parents of children between the ages of 0 to 19, one could expect that parenting stress might vary as a function of children's age within the headache population as well. In addition to examining the relationship between parenting stress and headache before and after the birth of a first child, future studies could also explore the same variables across the child's developmental stages.

Despite the strong presence of existing literature examining the relationship between stress and headache, the role of parenting stress in headache is currently not well understood. Ultimately, parenting stress may not be as relevant to primary headache disorders as other forms of stress (i.e., cognitive, physical, social), but the lack of another stress measure for comparison precludes making that conclusion within the present study. Nevertheless, the present study provides a foundation for further exploration of the relationship between parenting stress and headache, in line with the broader objective of ultimately providing improved clinical care for individuals impacted by primary headache disorders.

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## APPENDICES

## APPENDIX A: Eligibility Screener

Biological Sex:

▼ Male (1) ... Female (2)

Age:

▼ 18 (1) ... 100 or over (83)

Do you have at least one child under 19?

▼ Yes (1) ... No (2)

On average, how many days per week does your child under 19 **live in your household**?

▼ 0 days (1) ... 7 days a week (4)

On average, how many days per month do you experience headaches?

▼ 1 day a month or less (1) ... 2 or more days per month (2)

## APPENDIX B: Demographic Survey

Q1 Biological sex:

▼ Male ... Female

Q2 Age:

▼ 18 ... 100 or over

Q3 Choose one or more races that you consider yourself to be:

- White
- Black or African American
- Latino/a or Hispanic
- Asian
- Native Hawaiian or Pacific Islander
- Middle Eastern
- American Indian or Alaska Native
- Other \_\_\_\_\_

Q4 Marital status:

▼ Married ... Other

Q5 What is the highest level of school you have completed or the highest degree you have received?

▼ Less than high school degree ... Other



Q6 Number of children **under 19** for whom you are a parent/caregiver:

▼ 1 ... More than 10

Q7 On average, how many days per week do your children under 19 **live in your household**?

▼ 1 ... 7

Q8 Have any of your children ever been diagnosed with a psychological disorder and/or a chronic medical condition?

▼ Yes ... Don't know

Q9 Please enter the names of the psychological disorder(s) and/or chronic medical conditions with which your child(ren) have been diagnosed:

- 1. \_\_\_\_\_
- 2. \_\_\_\_\_
- 3. \_\_\_\_\_
- 4. \_\_\_\_\_
- 5. \_\_\_\_\_
- 6. \_\_\_\_\_
- 7. \_\_\_\_\_
- 8. \_\_\_\_\_

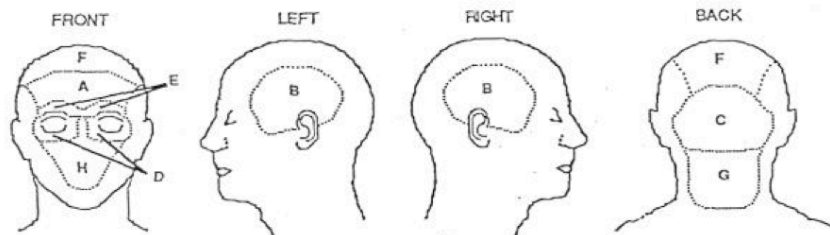
## APPENDIX C: SDIH-3

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

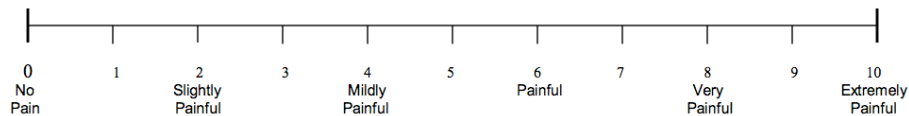
<b>Patient Name:</b>	<b>Age:</b>	<b>Sex:</b> M F
<b>Patient ID:</b>	<b>Interviewer:</b>	<b>Date:</b> / /

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)



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)*
7. How long have these headaches been occurring at this rate? \_\_\_\_ Months or \_\_\_\_ Years
8. What is the total number of this type of headache ever experienced:  1  2-4  5-9  ≥10 \_\_\_\_ *(Indicate total number experienced)*

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 *unremitting* 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  Unremitting

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 pain 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, and/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  $\geq 2$  days/week during this time, complete **Section 4c**  
If **Not**, 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

17a. If aura symptoms are present, has transient ischemic attack been excluded?  Yes  No

## APPENDIX D: HIT-6



### HIT-6™ Headache Impact Test

HIT is a tool used to measure the impact headaches have on your ability to function on the job, at school, at home and in social situations. Your score shows you the effect that headaches have on normal daily life and your ability to function. HIT was developed by an international team of headache experts from neurology and primary care medicine in collaboration with the psychometricians who developed the SF-36® health assessment tool. This questionnaire was designed to help you describe and communicate the way you feel and what you cannot do because of headaches.

To complete, please circle one answer for each question.

When you have headaches, how often is the pain severe?

never rarely sometimes very often always

How often do headaches limit your ability to do usual daily activities including household work, work, school, or social activities?

never rarely sometimes very often always

When you have a headache, how often do you wish you could lie down?

never rarely sometimes very often always

In the past 4 weeks, how often have you felt too tired to do work or daily activities because of your headaches?

never rarely sometimes very often always

In the past 4 weeks, how often have you felt fed up or irritated because of your headaches?

never rarely sometimes very often always

In the past 4 weeks, how often did headaches limit your ability to concentrate on work or daily activities?

never rarely sometimes very often always

+  +  +  +   
 COLUMN 1      COLUMN 2      COLUMN 3      COLUMN 4      COLUMN 5  
 6 points each      8 points each      10 points each      11 points each      13 points each

To score, add points for answers in each column.

**If your HIT-6 is 50 or higher:**

You should share your results with your doctor. Headaches that stop you from enjoying the important things in life, like family, work, school or social activities could be migraine.

TOTAL SCORE

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## APPENDIX E: PSQI

### The Pittsburgh Sleep Quality Index (PSQI)

Name: \_\_\_\_\_ Date: \_\_\_\_\_

**Instructions:** The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions. During the past month,

1. When have you usually gone to bed? \_\_\_\_\_
2. How long (in minutes) has it taken you to fall asleep each night? \_\_\_\_\_
3. When have you usually gotten up in the morning? \_\_\_\_\_
4. How many hours of actual sleep do you get at night? (This may be different than the number of hours you spend in bed) \_\_\_\_\_

Please check the appropriate blank below.

	Not during the past month (0)	Less than once a week (1)	Once or twice a week (2)	Three or More times a week (3)
5. During the past month, how often have you had trouble sleeping because you...				
a. Cannot get to sleep within 30 minutes	a. _____	_____	_____	_____
b. Wake up in the middle of the night or early morning	b. _____	_____	_____	_____
c. Have to get up to use the bathroom	c. _____	_____	_____	_____
d. Cannot breathe comfortable	d. _____	_____	_____	_____
e. Cough or snore loudly	e. _____	_____	_____	_____
f. Feel too cold	f. _____	_____	_____	_____
g. Feel too hot	g. _____	_____	_____	_____
h. Have bad dreams	h. _____	_____	_____	_____
i. Have pain	i. _____	_____	_____	_____
j. Other reason(s), please describe, including how often you have had trouble sleeping because of this reason(s):	j. _____	_____	_____	_____
6. During the past month, how often have you taken medicine (prescribed or "over the counter") to help you sleep?	6. _____	_____	_____	_____
7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?	7. _____	_____	_____	_____
8. During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done?	8. _____	_____	_____	_____
	<b>Very good (0)</b>	<b>Fairly good (1)</b>	<b>Fairly bad (2)</b>	<b>Very bad (3)</b>
9. During the past month, how would you rate your sleep quality overall?	9. _____	_____	_____	_____

Physician Determined Global PSQI Score: \_\_\_\_\_

## APPENDIX F: PSS

### Parental Stress Scale

The following statements describe feelings and perceptions about the experience of being a parent. Think of each of the items in terms of how your relationship with your child or children typically is. Please indicate the degree to which you agree or disagree with the following items by placing the appropriate number in the space provided.

1 = Strongly disagree 2 = Disagree 3 = Undecided 4 = Agree 5 = Strongly agree

1. I am happy in my role as a parent
2. There is little or nothing I wouldn't do for my child(ren) if it was necessary.
3. Caring for my child(ren) sometimes takes more time and energy than I have to give.
4. I sometimes worry whether I am doing enough for my child(ren).
5. I feel close to my child(ren).
6. I enjoy spending time with my child(ren).
7. My child(ren) is an important source of affection for me.
8. Having child(ren) gives me a more certain and optimistic view for the future.
9. The major source of stress in my life is my child(ren).
10. Having child(ren) leaves little time and flexibility in my life.
11. Having child(ren) has been a financial burden.
12. It is difficult to balance different responsibilities because of my child(ren).
13. The behaviour of my child(ren) is often embarrassing or stressful to me.
14. If I had it to do over again, I might decide not to have child(ren).
15. I feel overwhelmed by the responsibility of being a parent.
16. Having child(ren) has meant having too few choices and too little control over my life.
17. I am satisfied as a parent.
18. I find my child(ren) enjoyable.

APPENDIX G: Tables

Table 1

*Demographic Characteristics of the Migraine vs. TTH vs. Non-Headache Groups*

	Migraine ( <i>n</i> =171)	TTH ( <i>n</i> =120)	Non-Headache ( <i>n</i> =144)
Mean age ( <i>SD</i> )	35.13 (7.73)	37.57 (8.36)*	35.22 (9.99)
Mean number of children ( <i>SD</i> )	1.87 (1.07)	1.81 (0.89)	1.72 (0.95)
% Caucasian	80.7	75.8	70.8
% Married	61.9	75.0	63.2
% Bachelor's degree	30.9	35.8	38.2
% One child	44.4	42.5	54.9
% Child in household 7 days/week	94.2	95.0	89.6
% Child has a diagnosed condition	30.9*	27.1	20.3

\**p* < .05 versus comparison groups (migraine and non-headache for age, TTH and non-headache for child diagnoses).

Table 2

*Mean PSS, PSQI, and HIT-6 Scores for Migraine vs. TTH vs. Non-Headache Groups*

	Migraine ( <i>n</i> =171)	TTH ( <i>n</i> =120)	Non-Headache ( <i>n</i> =144)
Mean PSS score ( <i>SD</i> )	41.46 (10.25)	41.89 (8.98)	40.65 (9.85)
Mean PSQI score ( <i>SD</i> )	6.08 (2.93)*	4.81 (2.51)	4.94 (2.75)
Mean HIT-6 score ( <i>SD</i> )	62.29 (5.64)*	51.74 (7.44)	-
Mean headache days/month ( <i>SD</i> )	7.54 (5.01)*	5.04 (4.39)	-
Mean headache pain severity ( <i>SD</i> )	5.85 (1.57)*	3.93 (1.42)	-

\**p* < .05 versus comparison groups (TTH and non-headache for PSQI, TTH for HIT-6 and headache frequency and severity).



Table 3

*Covariate Analyses for PSS Scores and Headache Frequency*

	Beta	R <sup>2</sup>	Δ R <sup>2</sup>	P-value of Δ R <sup>2</sup>	P-value of Model
Block 1		.029			.004
Child diagnoses	1.854				
Block 2		.035	0.6%	.205	.007
PSS scores	.039				

VITA

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**EDUCATION**

- Ph.D.**     **University of Mississippi** (Oxford, MS)     Anticipated May  
Clinical Psychology     2019  
Dissertation: “An Exploration of Relationships between Parenting  
Stress and Primary Headache Disorders”  
Advisor: Todd A. Smitherman, Ph.D.
- M.A.**     **University of Mississippi** (Oxford, MS)     2017  
Clinical Psychology  
Master’s Thesis: “Comparing the Effects of Cognitive and Social  
Stress Among Individuals with Headache”  
Advisor: Todd A. Smitherman, Ph.D.
- B.S.**     **Tulane University** (New Orleans, LA), *Magna Cum Laude*     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.  
**Universidad de Granada** (Granada, Spain)     Fall 2011

**LICENSES AND CERTIFICATIONS**

- Examination for the Professional Practice in Psychology (EPPP)**     2017  
Passed at the Doctoral Level

**FUNDED GRANTS**

- Mississippi Council on Developmental Disabilities (MSCDD)     2015-  
Health Mini-Grant, \$10,000     2016  
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

### CLINICAL EXPERIENCE

**Mt Washington Pediatric Hospital (Baltimore, MD)** August 2018-Present

Predoctoral Intern, Pediatric Psychology Track

Supervisors: Kenneth Gelfand, Ph.D., Bradley Schwimmer, Psy.D.

Providing consultation, cognitive-behavioral intervention, and comprehensive assessment for a diverse outpatient and inpatient pediatric population in an urban hospital setting. Year-long rotations include outpatient psychological evaluation and outpatient psychotherapy. Six-month rotations include pediatric weight management clinic, autism spectrum disorder assessment, and integrated primary care for complex kids. Duties include assisting with program development of the new integrated primary care clinic; providing brief behavioral interventions in collaboration with primary care providers, gastroenterologists, nurse practitioners, and nutritionists; and co-facilitating a behavioral weight management group for overweight and obese children. Upcoming rotations include outpatient diabetes clinic, feeding day treatment program, and inpatient consultation/liaison.

**Delta Autumn Consulting, LLC (Oxford, MS)** 2017- 2018

Therapist, Psychometrist

Supervisor: John Young, Ph.D.

Conducted comprehensive evaluations of cognitive ability, academic achievement, and psychological functioning of public school children. Duties included test administration, scoring, interpretation, and integrated report-writing. Also provided evidence-based interventions in a private practice setting for children and adolescents referred by the local public school district.

**Baptist Children's Village (Water Valley, MS; Independence, MS)** 2017-2018

Psychological and Behavioral Consultant

Supervisor: C. Randy Cotton, Ph.D.

Provided evidence-based interventions and behavioral consultation for children and adolescents residing in temporary and long-term foster care. Additionally, this position required collaboration with the house-parents, campus reverend, and case manager.

- University of Mississippi Psychological Services Center (Oxford, MS)** 2014-2018  
 Graduate Therapist  
 Supervisors: Todd A. Smitherman, Ph.D., Kelly G. Wilson, Ph.D., Laura R. Johnson, Ph.D., John Young, Ph.D., Scott A. Gustafson, Ph.D.  
 Provided individual cognitive-behavioral therapy (CBT) and acceptance and commitment therapy (ACT) intervention for university and community outpatient adults, adolescents, and children with DSM-V disorders.
- **Desoto County School District (Desoto County, MS)** 2016  
 Psychological Examiner  
 Supervisor: Shannon Sharp, Ph.D.  
 Conducted comprehensive evaluations of cognitive ability, academic achievement, and psychological functioning of public school children. Duties included test administration, scoring, interpretation, integrated report-writing, and consultation in Individualized Education Program (IEP) meetings.
  - **Autism Center of North Mississippi (Tupelo, MS)** 2015-2016  
 Diagnostic Services Intern  
 Supervisor: J. Scott Bethay, Ph.D., BCBA  
 Independently conducted comprehensive full-battery assessments for children, adolescents, and adults suspected of having autism or other developmental, behavioral, and emotional disorders.
- North Mississippi Regional Center (Oxford, MS)** 2014-2015  
 Psychological and Behavioral Services Intern  
 Supervisor: J. Scott Bethay, Ph.D., BCBA  
 Provided individual and group interventions for residential patients with severe medical conditions, intellectual impairment, and developmental disabilities (e.g., sickle cell anemia, Prader-Willi Syndrome, Lesch-Nyhan Syndrome).

## **PUBLICATIONS AND PRESENTATIONS**

### Book Chapters

Peck, K. R., **Johnson, Y. L.**, & Smitherman, T. A. (2016). Migraine. In Rosano, Ikram, Ganguli (Eds.), *Neuroepidemiology (Handbook of clinical neurology)*; Vol 138, pp. 283-294). New York: Elsevier.

### Journal Publications

Kurz, A.S., **Johnson, Y.L.**, Kellum, K.K., & Wilson, K.G. (under review). Mobile technology, intensive longitudinal datasets, and inductive behavioral science: Using good ideas from statistics to merge all three. *Journal of Contextual Behavioral Science*.

### Oral Presentations

Polk, A., & **Johnson, Y. L.** (2017, September). *Comparing the effects of cognitive & social stress in individuals with headache*. Paper presented at the Mississippi Psychological Association's 68th Annual Convention; Biloxi, MS.

**Johnson, Y. L.,** Seifen, T. (2016, April). *Evaluation of the MATCH-ADTC protocol in a brief, group-based parent training: A pilot study.* Talk presented at the annual University of Mississippi Conference on Psychological Science, Oxford, MS.

**Johnson, Y. L.,** Seifen, T. (2017, April). *Improvement of parent stress and child behavior through a brief, group-based parent training using the MATCH-ADTC.* Data blitz presented at the annual University of Mississippi Conference on Psychological Science, Oxford, MS.

**Johnson, Y. L.** (2017, April). *Comparing cardiovascular responses to cognitive and social stress among individuals with headache.* Data blitz presented at the annual University of Mississippi Conference on Psychological Science, Oxford, MS.

Simon, S., Hoyt, C., & **Johnson, Y.** (2011, June). *The Effect of Media Images on Women's Leadership Perceptions and Aspirations.* Talk presented at the Society for the Psychological Study of Social Issues Biennial Conference (SPSSI), Charlotte, NC.

#### Poster Presentations

Seifen-Adkins, T., **Johnson, Y. L.** (2017, March). *Improving parental distress and child behavior: A brief, group-based training.* Poster presented at the annual meeting of the Southeastern Psychological Association (SEPA), Atlanta, GA.

**Johnson, Y. L.,** Black, A. K., Kurz A., Kuka, A. J., Smitherman, T. A. (2016, June). *Comparing the effects of cognitive and social stress among individuals with headache: An experimental manipulation.* Poster presented at the annual scientific meeting of the American Headache Society (AHS), San Diego, CA.

**Johnson, Y. L.,** Black, A. K., Kurz, A., Rogers, D. G., Smitherman, T. A. (2015, June). *Stress as a migraine trigger: A review of the experimental literature.* Poster presented at the annual scientific meeting of the American Headache Society (AHS), Washington, DC.

Black, A.K., **Johnson, Y. L.,** Houle, T. T., & Smitherman, T. A. (2014, June). *Perceived headache triggers as a function of headache diagnosis versus headache frequency.* Poster presented at the annual scientific meeting of the American Headache Society (AHS), Los Angeles, CA.

Person, J. M., Wencel, L. W., Crudup, B., **Johnson, Y.,** & Smitherman, T. A. (2014, April). *Establishing a relationship between obesity, stress, and headache disability.* Poster presented at the 2014 UM Conference on Psychological Science, Oxford, MS.

**Johnson, Y.,** Simon, S., O'Brien, L. T., & Major, B. (2013, January). *Exploring the relationship between Blacks' perceptions of Whites' motivations to be nonprejudiced and intergroup experiences.* Poster presented at the annual meeting of the Society for Personality and Social Psychology (SPSP), New Orleans, LA.

#### **EDITING AND REVIEWING EXPERIENCE**

##### Ad-Hoc Reviewing

- *Behaviour Research and Therapy*

- *International Journal of Behavioral Medicine*

**RESEARCH EXPERIENCE**

- **Migraine and Behavioral Health Laboratory** 2013-Present  
 Graduate Research Assistant, University of Mississippi  
 Advisor: Todd A. Smitherman, Ph.D.  
 Research topics covered include psychological and physiological factors of primary headache disorders, insomnia, obesity, and chronic pain.
- **Scientific Infusion That Helps (SITH) Laboratory** 2017-2018  
 Graduate Research Assistant, University of Mississippi  
 Advisor: John Young, Ph.D.  
 Study title: “*Validation of the SAMS and Clinical Assessment of Students with ADHD*”  
 Collaborative study with the UM Department of Pharmacy Administration designed to test the validity of the Subtle ADHD Malingering Screener (SAMS) and to obtain a thorough clinical assessment of students with ADHD.
- **Social Perception Laboratory** 2010-2013  
 Undergraduate Research Assistant, Tulane University  
 Advisor: Laurie O’Brien, Ph.D.  
 Research topics covered include prejudice, stereotyping, stigma, and stereotype threat related to race and gender.

**TEACHING EXPERIENCE**

- Course Instructor of Record, University of Mississippi 2016-2017  
 PSY201 General Psychology
- Course Tutor, University of Mississippi 2017  
 PSY202 Elementary Statistics
- Teaching Assistant, University of Mississippi 2017  
 PSY301 Developmental Psychology

**WORKSHOPS LED**

- **Co-Founder, Empowering You Parenting Workshop** 2015-2016  
 Supervisors: Sharon D. Boudreaux, M.A.T., BCBA, LBA, J. Scott Bethay, Ph.D., BCBA  
 Created and delivered a grant-funded, four-week parent education program derived from the Modular Approach to Therapy for Children with Anxiety, Depression, Trauma, or Conduct Problems (MATCH-ADTC) for parents of the north Mississippi community. Obtained IRB approval to collect baseline and outcome data for parental stress, parental sense of competence, child behavior, and family nutrition and physical activity.

**MEDIA APPEARANCES**

- Interview on *WTVA 9 News* (Tupelo, MS): Empowering You Workshop March 7, 2016

**PROFESSIONAL AND LEADERSHIP EXPERIENCE**

- Peer-Elected Student Representative to the Faculty, Psychology Department 2015-2016  
 University of Mississippi (Oxford, MS)

Vice President of Finance, Autism Speaks University Chapter University of Mississippi (Oxford, MS)	2016-2018
College Prep Mentor, PUENTES Latin Youth Leadership Council Tulane University (New Orleans, LA)	2012
Chapter Secretary, Alpha Lambda Delta Honor Society Tulane University (New Orleans, LA)	2011
Member, Newcomb Leadership Conference: Social Justice and Social Equality Tulane University Newcomb College Institute (New Orleans, LA)	2010
Intern, Residential Treatment Center Reinvestment Program U.S. Department of Behavioral Health (Washington, DC)	2010

**PROFESSIONAL MEMBERSHIPS**

Society of Pediatric Psychology (SPP), Division 54  
American Headache Society (AHS)  
Southeastern Psychological Association (SEPA)