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ALLODYNIA AND SELF-EFFICACY IN MIGRAINEURS

A Thesis

presented in partial fulfillment of requirements

for the degree of Master of Arts

in Clinical Psychology

The University of Mississippi

Ashley N. Polk

May, 2019

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## ABSTRACT

Headache disorders are among the most common disorders of the nervous system, with migraine alone affecting 14% of women and 6% of men worldwide each year. Headache-related self-efficacy, or one's confidence in preventing and managing headache, is particularly important for prevention and management of headache disorders and predicts response to behavioral and pharmacological treatments. Allodynia, the perception of non-noxious stimuli as painful due to central sensitization, compounds headache-related disability and compromises efficacy of triptans. Therefore, migraineurs with allodynia may perceive headache treatments as less efficacious and thus have reduced perceived headache-related self-efficacy. However, no literature to date has explored the relationship between self-efficacy and allodynia and the possible mediating role of fear of pain. The present study aimed to explore the relationship between allodynia and self-efficacy and whether fear of pain mediated this relationship among a non-treatment seeking sample of young adults with migraine. A significant negative relationship was observed between allodynia and self-efficacy in migraineurs, however the indirect effect of the mediation model was not statistically significant. Results of the present study suggest migraineurs with allodynia experience reduced headache-related self-efficacy, which may be an important target for behavioral intervention.

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## INTRODUCTION

### **Headache.**

Headache disorders are among the most common disorders of the nervous system (WHO, 2016), currently affecting 22.7% of people in the United States (Smitherman, Burch, Sheikh, & Loder, 2013). Global prevalence of current headache disorders is 47%, while lifetime prevalence is 66% (Stovner et al., 2007). However, men and women are affected differentially. Headache disorders affect 27.6% of women and 14.8% of men in the United States, are associated with increased use of healthcare services (Smitherman et al.), and are the second highest cause of years lost due to disability (YLD) worldwide (Global Burden of Disease Study Collaborators, 2016). Additionally, people suffering from headache disorders are 2.8 and 2.3 times more likely than people without headache to have a comorbid physical or psychiatric diagnosis, respectively (Smitherman et al.). Although head pain is the fifth most common reason for emergency room hospital visits (Burch, Rizzoli, & Loder, 2018), headache disorders often go under-diagnosed and untreated (Stovner et al.), despite their high prevalence and global burden.

### **Migraine.**

Headache disorders broadly represent a number of more specific disorders. “Primary” headache disorders, or those not attributable to other causes, include migraine, tension-type headache (TTH), trigeminal autonomic cephalalgias such as cluster headache, and other primary headache disorders (ICHD-3; International Headache Society [IHS], 2018). The focus herein is on migraine, as it is the most common disabling primary headache disorder. Migraine is



characterized by severe, pulsating pain that is typically experienced on one side of the head and may be aggravated by, or cause avoidance of, routine physical activity. Migraine typically lasts between 4 and 72 hours if untreated, with attack frequency (days/month with headache) varying greatly. Further, migraineurs must experience at least one additional symptom of nausea, vomiting, or both photophobia (i.e., sensitivity to normal light levels) and phonophobia (i.e., sensitivity to conversational noise levels). A significant minority of individuals with migraine sometimes experience aura, or temporary neurological symptoms that typically precede pain onset and gradually resolve. Migraine aura symptoms are visual or sensory in nature. Negative visual aura symptoms include vision loss; positive visual symptoms include flashing or shimmering lights perceived in one or both eyes (Rothrock, 2009). Sensory aura includes numbness and tingling across the face and toward the extremities (Rothrock). Migraine is further differentiated by frequency of headache attacks: Episodic migraine is characterized by less than 15 attacks per month, whereas chronic migraine is characterized by 15 or more attacks per month.

**Prevalence and Impact.** Migraine is a common and disabling condition. In the United States, 17.3% of females and 5.3% of males meet criteria for migraine, according to the American Migraine Prevalence and Prevention (AMPP) Study, with rates highest among individuals aged 30 to 39 years old (Buse et al., 2013). A recent review of United States government studies obtained even higher prevalence rates, such that 20.7% of US women and 9.7% of men suffered from migraine in the past three months (Burch et al., 2018). These higher rates are likely attributable to the fact that government surveillance studies rarely employ full diagnostic criteria and are thus more liberal in their estimates. Globally, 10% of the world's

population currently suffers from migraine, with a reported 14% lifetime prevalence (Stovner et al., 2007).

Migraine results in severe impairment during and between attacks, contributing to absenteeism, reduced productivity at work, and increased healthcare costs (Holroyd, 2010). For men and women of all ages worldwide, migraine is the sixth leading cause of years of life lived with disability (YLD), and third leading cause for people aged 15-49 years (Steiner, Stover, & Vos, 2016). In large part, migraine's impact on functioning is attributable to the fact that peak prevalence is highest in individuals aged 18-44 years (Burch et al., 2018), a time typically marked by high productivity, employment, and childrearing. Although many headache disorders are disabling, migraine appears to impact individuals' functioning most severely. Migraineurs experience more lost productive time at work or school than individuals with other headache diagnoses (Stewart, Wood, & Razzaghi, 2008), and those with chronic migraine are most profoundly affected given their high frequency of attacks (Serrano et al., 2013).

Migraine-related disability often deters individuals from participating in routine activities, including household responsibilities, social events, and exercise. Women in particular are more likely to report mild to severe migraine-related disability leading to impairment of routine activities (Buse et al., 2013). Though migraine alone impacts functioning, comorbid psychiatric diagnoses may compound its effects on functioning. Specifically, migraineurs with a comorbid psychiatric diagnosis are more frequently admitted to the hospital, dispensed more medications, and incur greater healthcare costs than migraineurs without psychiatric comorbidity (Lafata et al., 2004). As such, psychiatric conditions serve as moderator variables in the relationship between migraine and disability, prompting clinical consideration when migraineurs seek health services.

**Psychiatric Comorbidity.** Assessment and consideration of psychiatric comorbidities in migraine patients is particularly important for understanding migraine prognosis. Although migraine is classified as a neurological disorder, migraine often occurs with psychiatric disorders including depression (Breslau, Lipton, Stewart, Schultz, & Welch, 2003; Hamelsky & Lipton, 2006; Louter et al., 2015; Nicholson, Houle, Rhudy, & Norton, 2007), generalized anxiety disorder (GAD), panic disorder (Smitherman, Penzien, & Maizels, 2008), and bipolar spectrum disorder (Hamelsky & Lipton).

Depression and anxiety disorders are the most common comorbid presentations among migraineurs (Hamelsky & Lipton, 2006; Smitherman, Penzien, & Rains, 2013), and these relationships appear bidirectional in nature. For instance, individuals with a history of depression have a three-fold higher risk of migraine onset than those without prior depression, and those with migraine demonstrate a five-fold greater risk for onset of major depression than those without migraine histories (Breslau et al., 2003). The presence of comorbid psychopathology compounds functional impact (Breslau et al.; Tietjen, Herial, Hardgrive, Utley, & White 2007), such that depressed migraineurs show poorer response to treatment, decreased satisfaction with medical care, and increased hopelessness and disability (Nicholson et al., 2007). Though depression is common among migraineurs, similar findings exist for anxiety disorders comorbid with migraine.

Anxiety and stress are commonly reported as migraine triggers, and anxiety disorders, such as GAD, may have an even stronger association with migraine than affective disorders (Hamelsky & Lipton, 2006; Smitherman et al., 2008). Migraineurs with a comorbid anxiety disorder report increased pain intensity and migraine-related disability accompanied by decreased quality of life (Nicholson et al., 2007; Seng et al., 2017). Further, they experience

more negative affectivity and symptoms of hyperarousal than migraineurs without anxiety (Louter et al., 2015). Adding to heightened disability, and thus affecting quality of life, is one's locus of control. External locus of control is one's perception that an event is due to chance or the power of others, while internal locus of control is one's perception that an event is due to her own behaviors or characteristics (Rotter, 1966). Anxiety mediates the relationship between internal headache-specific locus of control (HSLC) and migraine-related quality of life (Grinberg & Seng, 2017), such that greater HSLC leads to greater anxiety symptoms, which increases migraine-related quality of life impairments. Although high internal HSLC typically bolsters migraineurs' problem-solving and self-efficacy, the unexpected results of this study suggest that HSLC may prove maladaptive in the absence of an ability to enact behavioral coping strategies (Grinberg & Seng), leading to increased anxiety symptoms. Increased anxiety contributes to avoidance of both activities and headache triggers and increases physiological reactivity (Nicholson et al.). As such, anxiety inhibits migraineurs' behavioral repertoire for coping with migraine, contributing to migraine progression. An important component of behavioral migraine treatment thus includes teaching patients methods of coping with anxiety-related migraine triggers (e.g., stress, poor sleep, missing meals, menstruation).

### **Associated Psychological Variables**

**Self-Efficacy.** In addition to psychiatric comorbidity and HSLC, other psychological variables influence migraine and migraine-related disability. Self-efficacy is one's belief that she can enact specific behaviors in specific contexts (Bandura, 1977). Specifically, self-efficacy includes both efficacy expectations and outcome expectations (Bandura). Efficacy expectations include one's belief that she can appropriately perform a behavior needed to produce an outcome. An outcome expectation, then, is one's assessment of her behavior as capable of

producing the specific outcome. Therefore, one can intelligibly understand the correct behavior path for producing a desired outcome without believing she is capable of executing the behavior. Self-efficacy is paramount to successful behavioral coping strategies (Bandura) and has demonstrated utility in predicting response to headache disorders.

Within headache disorders, self-efficacy includes one's beliefs in her ability to prevent and manage both headache episodes and headache-related disability. Individuals with headache who possess high self-efficacy beliefs take appropriate actions to prevent and manage headache pain, and thus typically possess an internal locus of control pertaining to headache. However, those with an external locus of control often have low self-efficacy pertaining to headache management (French et al., 2000), as they believe there is little they can do self-manage headache. Further, French and colleagues found that individuals who use positive psychological strategies (e.g., reducing muscle tension, stress management) to prevent and manage headache reported higher perceived self-efficacy than those who did not use such strategies. Additionally, self-efficacy favorably impacts headache-related disability, headache frequency, and pain severity. High self-efficacy is associated with reduced headache-related disability (French, et al.; Peck & Smitherman, 2015), headache frequency (Nicholson et al., 2005), and headache severity (Peck & Smitherman). Self-efficacy is a unique and significant predictor of headache disability beyond severity and locus of control (French et al.), underscoring the importance of distinguishing between self-efficacy and locus of control in migraineurs. More specifically, locus of control represents one's beliefs about both internal and external factors that influence migraine, whereas self-efficacy represents one's beliefs in her ability to affect change on internal and external factors (French et al.). Further, the effect of pain severity on disability is mediated

by one's self-efficacy beliefs, such that individuals with high self-efficacy are less disabled than those with low self-efficacy when controlling for pain severity (Peck & Smitherman).

In addition to playing a role in headache prevention and management, self-efficacy is implicated in psychological correlates of headache. Though stressful life events are positively correlated with headache frequency, self-efficacy moderates this relationship (Marlowe, 1998), such that the relationship weakens as self-efficacy increases. This finding suggests that self-efficacy may act as a partial buffer between experience of stressful events and headache frequency. Further, high self-efficacy is inversely associated with depression and anxiety symptomatology, somatic symptoms, and use of maladaptive coping strategies after controlling for headache severity and chronicity (Martin, Holroyd, & Rokicki, 1993). These findings demonstrate the importance of self-efficacy in improving headache-related disability.

As related to response to treatment, headache-specific self-efficacy improves following behavioral interventions for migraine, as would be expected given that efficacious behavioral migraine interventions (progressive muscle relaxation, biofeedback, stress and trigger management training) focus on teaching patients skills to better self-manage and prevent attacks (Rains, Penzien, McCrory, & Gray, 2005). Similarly, migraineurs who receive concurrent behavioral and pharmacological treatment show significantly greater improvement in headache self-efficacy than those who receive pharmacological treatment alone over a 12-month period (Seng & Holroyd, 2010). Importantly, improvement in self-efficacy following treatment is associated with greater reductions in post-treatment migraine frequency (Nicholson et al., 2005). Nicholson et al. (2007) noted that self-efficacy in migraineurs aids in managing hyperarousal, facilitating treatment adherence, and decreasing migraine-related burden on the healthcare system, as enhancing self-efficacy promotes individuals' ability to prevent and cope with

migraine and its associated triggers. Approaching and coping with migraine-related stimuli not only bolsters self-efficacy but reduces avoidance behaviors typically found in pain patients, and growing evidence suggests that progressive exposure to headache triggers may reduce trigger sensitivity over time (Martin & MacLeod, 2009; Martin et al., 2014). One factor that may contribute to migraineurs' avoidance behaviors, and thus negatively affect self-efficacy, is fear of pain.

**Fear of Pain.** The fear avoidance model (FAM) of pain was originally developed to describe the role of beliefs and behavioral responses in progression versus improvement of musculoskeletal pain following acute injury. The FAM posits that patients' pain sensations and subsequent emotional reactions may become dysynchronous, leading to an exaggerated pain response (Lethem, Slade, Troup, & Bentley, 1983). According to the FAM (Lethem et al.), patients either confront or avoid their pain experiences as a function of their beliefs about harmfulness of pain sensations. Abstaining from activities putatively associated with pain is motivated by "fear of pain," a perceived susceptibility to pain or reinjury resulting in avoidance of pain-related stimuli (Lethem et al.). Fear of pain leads to both cognitive and behavioral avoidance of pain experiences and activities that can persist well beyond the time needed for tissue healing. Prolonged avoidance leads to decreased social and physical activity, resulting in decreased exposure to pain, restricted behavioral coping repertoires, and physical decline stemming from deconditioning of the musculoskeletal system. A patient's propensity for avoidance is influenced by a number of psychosocial factors including stressful life events, previous pain history, coping strategies, behavioral patterns, and anxiety sensitivity (Lethem et al.; Vlaeyen & Linton, 2000). These avoidance behaviors are negatively reinforcing (Lethem et al., 1983) but over time function to exacerbate pain (via deconditioning and increased sensitivity

to pain stimuli) and contribute to the development and maintenance of pain-related disability (Vlaeyen & Linton). Attention to patients' fear of pain and resulting avoidance behaviors may inform effective interventions that promote confrontation of pain-related experiences (Bailey, Carleton, Vlaeyen, & Asmundson, 2010) and bolster self-efficacy.

Though the FAM originally described pain-related behaviors resulting from musculoskeletal injury, some research aims to apply the model to headache disorders (Hursey & Jacks, 1992). Fear of pain and avoidance behaviors have long been studied in chronic pain conditions (Philips & Jahanshahi, 1985; Philips, 1987) and are particularly important in headache disorders given that patients often avoid triggers/stimuli associated with head pain. An early factor analytic study (Philips & Jahanshahi, 1986) examining pain behaviors in headache sufferers identified thirteen factors contributing to pain behavior, six of which were manifestations of avoidance behavior (and accounted for 42.6% of the total variance in pain behavior). Of the six factors, social avoidance accounted for the most variance (21.9%), such that headache sufferers avoided social activities more than other daily activities due to head pain (Philips & Jahanshahi). Social avoidance likely diminishes quality of life and contributes to headache-related disability, compromising headache-related self-efficacy and increasing risk for depression.

Fear of pain is strongly associated with headache-related disability, even after controlling for pain severity, locus of control, self-efficacy, and emotional distress (Nash, Williams, Nicholson, & Trask, 2006). Building on this finding, Black, Fulwiler, and Smitherman (2015) explored fear of pain among young adults with headache and found that migraineurs endorsed greater fear of pain than non-headache controls and individuals with tension-type headache. This relationship remained after controlling for gender, depression, and anxiety. The same study also



found that fear of pain accounted for more variance in headache-related disability than the combination of anxiety, depression, and gender. Further, fear of pain mediated the relationship between pain severity and headache-related disability. Increases in fear of pain were also associated with more frequent and painful migraines. Though this study illuminated the role of fear of pain in headache-related disability, unexplained variance in disability remained.

One variable that may account for additional variance in this relationship is self-efficacy, given the strong aforementioned association between disability and self-efficacy. Self-efficacy mediates the relationship between fear of pain and functional disability, but not depressive symptoms, in children with chronic headache disorders (Carpino et al., 2014). Increases in fear of pain predicted decreases in self-efficacy, which in turn predicted increased functional impairment. These findings suggest that self-efficacy may serve as a protective factor between fear of pain and resulting disability and that self-efficacy is more important for behavioral, rather than emotional, outcomes related to headache pain. Though a relationship between fear of pain and self-efficacy has been shown in children with chronic headache, no literature to date has explored this association in adult migraineurs, despite the fact that adults are far more likely to experience migraine than are children (Lipton et al., 2007). Taken together, the literature demonstrates potential importance of both fear of pain and self-efficacy as targets for treatment for reducing headache-related disability.

**Allodynia.** A physiological aspect of migraine likely related to fear of pain is cutaneous allodynia. Cutaneous allodynia, the perception of non-noxious stimuli as painful on the skin (e.g., mild heat/cold, light touch or pressure; Burstein, Yarnitsky, Goor-Aryeh, Ransil, & Bajwa, 2000), further contributes to one's pain experience. Physiologically, allodynia results partially from central sensitization, a phenomenon resulting from lowered pain thresholds of central

trigeminal neurons receiving input from the skin and dura. These lowered thresholds result in hypersensitivity to stimuli that would typically occasion little to no pain response (Burstein et al., 2000). Allodynia in pain patients is differentiated into three types: thermal, dynamic mechanical, and static mechanical. Thermal allodynia occurs as a result of hypersensitivity to hot and cold stimuli. Dynamic mechanical allodynia, commonly termed brush allodynia, is the perception of brushing the skin as painful and is likely mediated by A $\beta$  mechanoreceptive and capsaicin-insensitive A $\beta$  fibers. Finally, static mechanical allodynia is the perception of pressure on the skin as painful, a response mediated by A $\delta$  nociceptive fibers (Lipton et al., 2008). A wide variety of pain patients experience allodynia (Jarrell & Arendt-Nielsen, 2016; Jensen & Finnerup, 2014), including migraineurs. The association of allodynia with migraine is likely due to similar physiological mechanisms stemming from sensitization of nociceptors and activation of meningeal perivascular pain fibers (Mathew, Kailasam, & Seifert, 2004).

Burstein and colleagues (2000) were the first to note the occurrence of cutaneous allodynia in migraineurs via quantitative sensory testing, following the identification of allodynia in animal models of migraine. Up to 79% of migraineurs may experience allodynia during migraine attacks (Burstein et al.). The relationship between migraine and allodynia is both temporal and site-specific, with pain sensitivity typically presenting ipsilateral to head pain (Mathew et al., 2004). Many migraineurs report allodynia symptoms at the peak of head pain, though allodynia may also occur between pain onset and peak intensity (Mathew et al.). Allodynia duration is most often confined to migraine attacks, though 20% of migraineurs experience persistent allodynia for 6 to 48 hours following migraine cessation (Mathew et al.). Further, allodynia is associated with a number of migraine characteristics.

Cutaneous allodynia is associated with increased migraine-related disability and is most prevalent in migraineurs aged 18 to 36 (Baykan et al., 2016; Kalita, Yadav, & Misra, 2009; Lipton et al., 2008). Allodynia appears to be a risk factor for migraine progression (Baykan et al.; Lipton et al.), as both migraine duration and frequency of attacks are positively associated with allodynia (Kalita et al.). Migraine aura is associated with a 3.5 fold increased risk for allodynia (Lipton et al., 2008), such that 70.5% of migraineurs with aura also experience allodynia during migraine attacks (Baykan et al.). Other migraine characteristics associated with allodynia include family history of headache, nausea, vomiting, phono- and photophobia, and prodromal symptoms (Baykan et al.). Allodynia may also be associated with hormonal changes in women, (Baykan et al.), though this association is less surprising given that women are disproportionately affected by migraine. Given the association of disabling migraine symptoms with allodynia, treatment regimens that reduce disability and enhance quality of life are of particular importance.

Despite an array of efficacious pharmacological and behavioral migraine treatments, additional research is needed for understanding the role of allodynia in migraine treatment. Triptan medications, which act as 5-HT<sub>1B/1D</sub> receptor agonists, are common and effective acute treatments for migraine (Thorlund et al., 2014). However, treatment for migraineurs with allodynia is much less successful. A study comparing triptan therapy response in migraineurs with and without allodynia during attacks found that presence of allodynia predicted response to triptans, such that migraineurs who did not experience allodynia were more likely to be pain-free both one and four hours after treatment (Burstein, Collins, & Jakubowski, 2004). Additionally, Burstein and colleagues found that 83% of those without allodynia during migraine were pain-free within 2 hours of sumatriptan treatment. Among those with allodynia symptoms, however, only 15% were pain-free if treatment was initiated after allodynia began, and 80% of these

individuals had a recurrence of pain within 8 to 16 hours of treatment. Conversely, 63% of those with allodynia were pain-free if sumatriptan administration preceded onset of allodynia symptoms. These findings suggest that allodynia moderates triptan response.

Burstein and colleagues (2004) thus concluded that triptan treatment is highly effective for migraineurs in the absence of allodynia and may alleviate some pain for individuals with allodynia if pain is treated prior to allodynia onset (i.e., when head pain is first noticed). These findings suggest that individuals experiencing allodynia present with a narrow window of opportunity for alleviating migraine with triptans. Migraineurs with allodynia thus likely perceive triptan treatment as less efficacious, thereby reducing their ability to effect change and diminishing headache-related self-efficacy. As such, migraineurs who experience allodynia may benefit from behavioral interventions focused on increasing self-efficacy for managing migraine attacks and accompanying allodynia. However, additional research is warranted concerning the role of self-efficacy and other psychological factors in allodynia.

Given the prevalence of psychological symptoms and disorders among migraineurs, those with allodynia often present with psychological comorbidities, though the literature is somewhat mixed. Allodynia uniquely contributes to the likelihood of depression among migraineurs (Louter et al., 2014; Louter et al., 2015). Individuals with allodynia also endorse more symptoms of anxiety and depression than those without allodynia, and symptom severity varies as a function of allodynia severity (d'Agostino, Francia, Licursi, & Cerbo, 2010; Kao et al., 2014). Notably, migraineurs who experience allodynia may experience more psychological difficulties than psychiatric patients. For instance, migraineurs with allodynia obtain significantly higher scores on measures of negative affect, lack of positive affect, and somatic arousal compared to

those without allodynia, non-headache controls, and both individuals with prior and current psychological diagnoses (Louter et al., 2014).

By comparison, other studies have found no association between allodynia and psychological symptoms among migraineurs (Lovati et al., 2009; Lovati, D'Amico, Brambilla, Mariani, & Bussone, 2008). These discrepancies may result from differing methods of assessing allodynia and psychological symptoms. Studies that found an association between allodynia and psychological factors (Louter et al.; d'Agostino et al.; Kao et al., 2014) used a validated measure for assessing presence of allodynia, which provide severity cutoff scores. However, studies that found no association between allodynia and psychological factors (Lovati et al.; Lovati et al.) employed less rigorous methods of assessing allodynia symptoms, requiring experience of only one allodynia symptom for inclusion. Therefore, studies employing more rigorous methods of allodynia symptom assessment likely contained participants who experience allodynia during migraine. Further, studies finding significant relationships used multiple validated measures for assessing specific psychological symptoms, while those with null results used a single cumulative questionnaire for assessing broad psychopathology.

Though self-efficacy is especially compromised in migraineurs with depression and anxiety, less is known about self-efficacy among migraineurs experiencing allodynia. Further, both allodynia and self-efficacy are influenced by migraine frequency and disability, though no literature to date has explored the relationship between self-efficacy and allodynia among migraineurs. Given that both migraine and allodynia are attributable to processes of central sensitization that occasion hypersensitivities to pain and resulting avoidance behaviors, increased fear of pain and decreased headache-related self-efficacy likely characterize allodynia in migraineurs. However, literature on this relationship is lacking.

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

Allodynia, fear of pain, and self-efficacy contribute to migraineurs' pain experience and coping behaviors. Though literature to date has explored the relationship of each variable with a myriad of migraine characteristics and comorbidities, no integrative study exists. Principally, despite the role of allodynia in response to acute pharmacotherapies, the relationship between allodynia and self-efficacy remains unknown and unexplored. Allodynia likely exacerbates fear of pain and contributes to pain-related avoidance behaviors consistent with tenets of the FAM, likely decreasing adaptive coping and compromising self-efficacy. The current study thus aimed to explore the relationship between allodynia and self-efficacy among migraineurs and to assess whether fear of pain mediates this relationship. A potential clinical implication of the present study includes that migraineurs with allodynia may benefit from interventions targeting self-efficacy as well as fear of pain (Bailey, Carleton, Vlaeyen, & Asmundson, 2010; Woods & Asmundson, 2008).

## **Hypotheses**

*Study Goal 1: To investigate whether allodynia is associated with self-efficacy in migraineurs.*

- Hypothesis 1a: Allodynia would be negatively associated with self-efficacy in migraineurs.
- Hypothesis 1b: The association would remain after controlling for migraine frequency and disability.

*Study Goal 2: To investigate whether fear of pain mediates the association between allodynia and self-efficacy in migraineurs.*

- Hypothesis 2a: Fear of pain would mediate the association between allodynia and self-efficacy in migraineurs, such that allodynia would predict increased fear of pain, which in turn would predict reduced self-efficacy.

Hypothesis 2b: The association would remain after controlling for migraine frequency and disability in the mediation model

## METHODS

### Participants

The initial sample consisted of undergraduate students aged 18 years and older who completed computer-administered measures assessing headache symptoms, disability, allodynia, fear of pain, and self-efficacy among a larger battery of measures. Individuals meeting ICHD-3 criteria for migraine with or without aura occurring on at least 2 days per month were retained for the present study. Individuals with or suspected of having another headache disorder (episodic or chronic tension type headache, posttraumatic headache, or cluster headache) were excluded. Other exclusion criteria included suspect effort, defined as participants completing the battery in the fastest 10% of completion time (i.e., <30 minutes). Assuming a medium effect size of  $f^2 = .15$ , power of .80, and statistical significance of  $p < .05$ , a total sample size of 85 participants was required.

### Materials

**Demographics Questionnaire.** Participants completed a questionnaire to provide information about race, ethnicity, gender, and other demographic information. This measure can be found in Appendix A.

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



frequency, severity, and other diagnostic characteristics that aid in differential diagnosis. Additionally, the SDIH-3 includes appendix questions for assessing aura symptoms, cluster headache, medication overuse, and post-traumatic headache. This measure can be found in Appendix B.

**Headache Management Self-Efficacy (HMSE-25).** The HMSE-25 (French et al., 2000) is a 25-item self-report measure that assesses headache-related self-efficacy, including individuals' ability to prevent their headache episodes and manage head pain. Each item is scored using a 7-point Likert scale ranging from 1, "strongly disagree," to 7, "strongly agree" with 9 reverse-scored items. HMSE-25 scores range from 25 to 175, with higher scores indicative of higher self-efficacy. The HMSE-25 exhibits excellent internal consistency ( $\alpha = .90$ ) and a positive correlation with the Headache-Specific Locus of Control Internal Subscale ( $r = .40$ ), another measure of headache self-management (French et al.). This measure can be found in Appendix C.

**Headache Impact Test-6 (HIT-6).** The HIT-6 (Kosinski et al., 2003) is a 6-item self-report measure that assesses disability resulting from headache. Specifically, the HIT-6 assesses the impact of headache on psychological, cognitive, occupational, and social functioning over the past 4 weeks. The 6 items query responses on a 5-point Likert-type scale from "Never" to "Always" to assess frequency and severity of impairment. Scores range from 36 to 78, with scores of 60 and above indicating very severe impact of headache on functioning. Kosinski and colleagues found the HIT-6 to have good internal consistency, alternate forms, and test-retest reliability (0.89, 0.90, and 0.80, respectively), as well as high discriminate validity across headache diagnostic groups. This measure can be found in Appendix D.

**Allodynia Symptom Checklist-12 (ASC-12).** The ASC-12 (Lipton et al., 2008) is a 12-item measure that queries cutaneous allodynia symptom frequency during headache. Each item represents a common context in which allodynia is experienced, rated as “does not apply to me” or occurring “never,” “rarely,” “less than 50% of the time,” or “ $\geq$  50% of the time.” The first three responses receive a score of 0, symptoms that occur less than half the time receive a score of 1 point, and symptoms occurring half the time or more are scored 2 points. ASC-12 scores range from 0-24, with scores of 0-2 considered indicative of an absence of allodynia, scores of 3-5 indicating mild allodynia, scores of 6-8 suggesting moderate allodynia, and scores of 9 or more indicating severe allodynia. The ASC-12 has a three-factor structure, such that it represents thermal, mechanical static, and mechanical dynamic allodynia. Finally, the ASC-12 demonstrates high sensitivity (84.8%) and low to moderate specificity (52.2%) compared to quantitative sensory testing (QST), the gold standard for assessing cutaneous allodynia. This measure can be found in Appendix E.

**Pain Anxiety Symptom Scale (PASS-20).** The PASS-20 (McCracken & Dhingra, 2002) is an abbreviated version of the original 40-item PASS (McCracken, Zayfert, & Gross, 1992), which was developed to evaluate fear of pain across four domains: pain-related anxious physiological reactions, fearful cognitions, cognitive anxiety, and avoidance/escape behaviors. The PASS-20 retains good internal consistency, criterion validity, and construct validity from the original PASS. The subscales mirror the original PASS subscales with high intercorrelations (mean  $r = .95$ ) and more modest correlations between nonmatching subscales (mean  $r = .57$ ), supporting both good convergent and divergent validity. The PASS-20 items are rated on a 6-point Likert scale ranging from 0, “never,” to 5, “always.” Possible scores range from 0 to 100, with higher scores indicative of greater fear of pain. This measure can be found in Appendix F.

## **Procedure**

The aforementioned measures were included as part of a larger online survey battery administered through SONA Systems via Qualtrics, used with undergraduate students enrolled in psychology courses. Students completed the online battery over three consecutive semesters and received modest course credit.

### **III. RESULTS**

#### **Statistical Analyses**

Descriptive statistics were summarized and distributions examined. Mahalanobis distance was used to determine and exclude multivariate outliers. Prior to conducting primary analyses, bivariate correlations between variables of interest were tested using Pearson correlation analyses. The primary analyses were then conducted in three steps. First, a simple linear regression was conducted to assess the relationship between allodynia and self-efficacy among migraineurs. Second, a hierarchical linear regression was conducted to assess the relationship between allodynia and self-efficacy after controlling for migraine frequency and disability. Finally, the effect of allodynia on headache self-efficacy through fear of pain was assessed, both directly and indirectly using methods outlined by Hayes (2018). Bootstrapping procedures were used to estimate the 95% confidence interval of the indirect effect of allodynia on self-efficacy through fear of pain.

#### **Participant Demographics**

A total of 2003 students participated. Three-hundred twenty-four failed to complete the entire battery, 122 evidenced suspect effort by completing the battery in less than 30 minutes, and 3 were under 18 years old. These participants were excluded from analyses. Additional exclusions included participants who did not complete headache-specific items necessary to assign headache diagnosis (n = 87), denied headache (n = 357), endorsed symptoms consistent with probable migraine (n = 350) or probable TTH (n = 272), or reported symptoms consistent with episodic TTH (n = 197), chronic TTH (n = 10), cluster headache (n = 20), less than 2

migraine days per month ( $n = 10$ ), or headache attributable to head injury ( $n = 97$ ). One participant did not complete the Allodynia Symptom Checklist-12 and was excluded from analyses.

Table 1 presents demographic characteristics of the retained sample. The final sample consisted of 147 college students (87.80% female) with a mean age of 18.98 years old ( $SD = 2.39$ ). The majority of the sample was Caucasian (86.4%); 11.6% were African American, 1.4% identified as multiracial, and 0.7% were Hispanic/Latina. Regarding headache diagnosis, of the 147 migraineurs retained, 56 (38.1%) met diagnostic criteria for episodic migraine without aura, 49 (33.3%) met diagnostic criteria for episodic migraine with aura, and 42 (28.6%) met criteria for chronic migraine. On average, participants reported experiencing nearly ten headache days per month, with severe headache-related disability and mild levels of allodynia. Participants' mean headache management self-efficacy scores were moderate, and their mean fear of pain scores were elevated. Regarding allodynia, 44.2% of the sample reported experiencing no allodynia symptoms, while 55.8% reported experiencing some level of allodynia symptoms.

### **Data Analytic Assumptions**

Histograms and descriptive statistics (i.e., kurtosis, skewness) were used to assess analytic assumptions for variables of interest (ASC-12, HMSE-25, PASS-20, and HIT-6) and found to be satisfactory. All participants were assessed for multivariate outliers on total scores of interest by Mahalanobis distance prior to performing statistical analyses; six multivariate outliers were found using a conservative  $p < .001$  cutoff and were excluded from all analyses. Twenty-two percent of the remaining 147 participants had missing total scores due to missing values (total missing data = 0.48%). Missing values were imputed using mean substitution by group (i.e. with and without allodynia).

## Correlations Among Variables of Interest

Significant correlations were found between each of the variables. Table 2 presents correlations among allodynia and associated headache variables. Allodynia showed a small positive association with fear of pain and headache-related disability (both  $r$ s = .21). Headache-related disability showed a small positive relationship with fear of pain and a moderate-to-large positive relationship with headache frequency and severity. Finally, fear of pain showed a small positive relationship with both headache frequency and severity. Differences between migraineurs with and without allodynia are presented in Table 3, revealing those with allodynia reporting significantly greater fear of pain than those without ( $M = 37.00$  vs.  $31.81$ , respectively). Those with and without allodynia did not differ on headache-related disability or self-efficacy.

## Regression Analyses

First, a simple linear regression was performed to “predict” headache-related self-efficacy based on allodynia among migraineurs. A significant negative relationship between allodynia and self-efficacy emerged ( $R^2 = .03, p = .049$ ), such that for each one-point increase on the ASC-12, participants’ HMSE-25 scores decreased .93 points. Next, a hierarchical multiple regression was performed to “predict” headache-related self-efficacy based on allodynia after adding migraine frequency and disability into the model. Complete results of the hierarchical multiple regression are presented in Table 4. Block 1, which included migraine frequency and disability, was significant ( $R^2 = .16, p < .001$ ), with headache variables accounting for 16% of the variance in headache-related self-efficacy. Block 2, which incorporated allodynia, was not significant ( $\Delta R^2 = .01, p = .21$ ), as allodynia accounted for only 1% of the change in variance in headache-related self-efficacy. The overall model remained significant ( $p < .001$ ), although

headache-related disability was the only significant predictor of self-efficacy in the final model ( $\beta = -.393, p < .001$ ), indicating that as disability increases, self-efficacy decreases.

### **Mediation Analysis**

Using the PROCESS macro for SPSS (Hayes, 2018), the effect of allodynia on self-efficacy was assessed directly and indirectly through fear of pain. Bootstrapping procedures were used to estimate the 95% confidence interval of the indirect effect of allodynia on self-efficacy through fear of pain using 5,000 bootstrapped samples. Mediation results are presented in Figure 1. The total effect of allodynia on self-efficacy was  $-.93, p = .049$ , as was expected given it replicates results from the aforementioned regression, indicating that self-efficacy is reduced for migraineurs with allodynia. For every one-point increase on ASC-12 scores, HMSE-25 scores decrease by nearly one point on average. Allodynia was positively associated with fear of pain (path  $a = 0.99, p < .01$ ), but fear of pain was not associated with self-efficacy (path  $b = .01, p = .93$ ). The direct effect of allodynia on self-efficacy was  $-.94, p = .05$ , indicating that holding fear of pain constant does not result in statistically significant changes in self-efficacy as a result of allodynia, although this effect fell just shy of statistical significance. Finally, the indirect effect of allodynia on self-efficacy through fear of pain was assessed in the mediation model. The indirect effect of allodynia on self-efficacy through fear of pain was  $.01, 95\% CI(-.27, .22)$ , which is not statistically significant given that the confidence interval contains zero. Therefore, fear of pain does not appear to mediate the relationship between allodynia and self-efficacy among migraineurs.

### **Supplementary Analyses**

Supplementary analyses were performed to explore whether null results from the mediation model might be attributable to collapsing episodic and migraineurs together.

Differences in allodynia scores were analyzed between migraine diagnostic groups (i.e., migraine, migraine with aura, chronic migraine) by performing a one-way analysis of variance (ANOVA). The ANOVA yielded non-significant results ( $F[2, 144] = .87, p = .420$ ), indicating that allodynia scores were not statistically different between diagnostic groups. Results from this analysis are presented in Table 5.



## IV. DISCUSSION

The impact of allodynia on pain conditions is often considered throughout headache and chronic pain literature, though little research to date has explored psychological correlates of allodynia. The present study sought to examine the relationship between allodynia and self-efficacy among migraineurs and the possible influence of fear of pain on the hypothesized relationship.

### **Relationships Between Allodynia and Self-Efficacy**

Consistent with Hypothesis 1a, allodynia and self-efficacy showed a small but significant negative relationship, such that as allodynia severity increased, migraineurs' self-efficacy decreased. This is the first study to our knowledge to demonstrate a relationship between allodynia and self-efficacy, either within headache literature or pain literature more broadly. Prior research demonstrated that migraineurs with allodynia present with a narrow window for relieving head pain with triptan treatment (Burstein, Collins, & Jakubowski, 2004), suggesting counseling in medication adherence and alternative methods of treatment, such as behavioral self-management, may be promising interventions for such patients. Chronic disease literature suggests that self-management skills are superior to information-only education and improve functional outcomes (Bodenheimer, Lorig, Holman, & Grumbach, 2002). Further, self-efficacy is a critical component of self-management among chronic pain conditions broadly (Bodenheimer et al.; Marks, Allegrante, & Lorig, 2005), indicating that enhancing and promoting self-efficacy results in improvements in disability and disease prognosis more generally.

Efficacy of non-pharmacological migraine management is well established for improving migraine frequency and disability (Rains, Penzien, McCrory, & Gray, 2005; Nicholson, Nash, & Andrasik, 2005). More recent research (Seng & Holroyd, 2010) exploring the impact of treatment expectancies on headache outcomes found that the addition of behavioral migraine management to pharmacotherapy yielded greater increases in headache-related self-efficacy than pharmacotherapy alone. Seng and Holroyd also demonstrated that behavioral management improved internal locus of control and decreased chance locus of control, regardless of pharmacotherapy condition (i.e., beta blocker or placebo). Early work on biofeedback suggested that resulting improvements in TTH derived not from physiological changes, but from cognitive changes stemming from perceptions of success and increases in self-efficacy and internal locus of control (Holroyd et al., 1984). Further evidence suggests that self-efficacy itself is the mechanism of change in cognitive behavioral interventions. Specifically, self-efficacy at two months mediated the relationship between stress management and disability outcomes at eight months among individuals with TTH (Holroyd, Labus, & Carlson, 2009), underscoring the mechanistic role of psychological processes in self-management approaches to treatment. The present negative relationship between allodynia and self-efficacy in migraineurs suggests that self-efficacy's influence on functional outcomes and treatment response is a variable worth investigating among patients with allodynia.

To the extent allodynia is associated with reduced headache self-efficacy, migraineurs with allodynia (especially those responding poorly to pharmacotherapy) may benefit from the addition of behavioral migraine management and corresponding improvements in self-efficacy. Because low self-efficacy is associated with high external locus of control (French et al., 2000), migraineurs with allodynia may in fact view themselves as poor candidates for behavioral

management (Seng & Holroyd, 2010), given that they believe migraine is influenced by factors other than their own behavior. In fact, though, patients with high external locus of control exhibit greater improvements in self-efficacy following behavioral migraine management than those with a high internal locus of control (Seng & Holroyd). Though no studies to our knowledge have evaluated the efficacy of behavioral migraine interventions for individuals with allodynia, work in this area holds promise for increasing available treatment options to this challenging subgroup of migraine patients.

Contrary to Hypothesis 1b, however, the observed relationship between allodynia and self-efficacy proved non-significant after accounting for migraine frequency and disability. Only 17% of total variance in self-efficacy was accounted for in the model—most of which was attributable to disability—leaving a large proportion of variance in self-efficacy unexplained. Perhaps anxiety sensitivity and medication overuse would account for additional variance in self-efficacy among migraineurs with allodynia. Anxiety sensitivity (AS) is characterized by hypersensitivity to physiological sensations and misinterpretation of these sensations as dangerous (Reiss, 1991). Applied to headache, AS may present as misinterpretation of sensations as indicative of headache onset, leading to subsequent avoidance of activities occurring during misinterpretation (Nicholson et al., 2007). Evidence shows that AS influences escape and avoidance behaviors in headache patients (Norton & Asmundson, 2004) and predicts sensitivity to headache triggers (Smitherman, Davis, Walters, Young, & Houle, 2015). Taken together, these findings suggest that migraineurs with high AS avoid pain-related stimuli, which likely increases sensitivity to pain. In an attempt to prevent or attenuate head pain, such patients may unnecessarily utilize acute headache medications, resulting in medication overuse. Among headache patients, medication overuse headache (MOH) not only impacts attack frequency and

disability (Peck, Roland, & Smitherman, 2018), but also likely develops in part from central sensitization (De Felice, Ossipov, & Porreca, 2011), thus increasing risk for allodynia. Though AS and medication overuse were not considered in the present study, they may account for some unexplained variance in self-efficacy among migraineurs with allodynia.

### **Mediation Analysis**

In accordance with hypotheses, allodynia and self-efficacy were negatively associated with one another, presenting an effect to be mediated. Contrary to Hypothesis 2a, however, the mediation analysis showed that fear of pain does not serve a mediating role between allodynia and self-efficacy in migraineurs. However, a positive relationship between allodynia and fear of pain was found, indicating that FOP increases with allodynia symptom severity. Given that allodynia results from central sensitization and is characterized by hypersensitivity to non-painful stimuli, migraineurs experiencing allodynia may engage in more pain-avoidant behaviors, consistent with the FAM, than those without allodynia. As mentioned above, one such maladaptive avoidance behavior includes taking acute medication prematurely, leading to medication misuse and possibly MOH as it confers increased risk for progression of headache frequency. Indeed, patients meeting diagnostic criteria for MOH are increasingly likely to engage in pain avoidant behaviors compared to those without MOH (Peck, Roland, & Smitherman, 2018). Though the exact mechanisms underlying MOH are unknown, central sensitization is likely implicated (De Felice, Ossipov, & Porreca, 2011), suggesting that MOH and allodynia share physiological underpinnings that likely contribute to maladaptive avoidance and escape behaviors characterizing fear of pain. Moreover, repeated administration of pharmacotherapy in the absence of pain relief likely fosters reduced self-efficacy for headache self-management and

increased reliance on medication. Should the proposed cycle exist, decreased self-efficacy may maintain medication overuse over time.

Perhaps allodynia and fear of pain are better considered in the context of other pain-related variables, such as medication overuse, rather than self-efficacy given that no significant bivariate correlation was found between fear of pain and self-efficacy. This particular finding is inconsistent with results found in the broader chronic pain literature, which indicate that increases in self-efficacy are associated with reductions in fear of pain (Perry & Francis, 2013; Woby et al., 2007). Further, findings suggest that self-efficacy mediates the relationship between fear of pain and disability in a sample of adult chronic pain participants (Perry & Francis, 2013), which included a small percentage of chronic migraineurs. However, participants in these studies had complex pain histories and were on average 20 years older than the participants in the present study, making generalizations and comparisons difficult. Further, participants in the aforementioned studies endorsed markedly lower self-efficacy than the present sample, which may account for disparate findings.

Finally, considering the positive association between allodynia and fear of pain found in the present study, the predicted mediation effect may present when considering the role of FOP in the relationship between allodynia and *disability*, rather than self-efficacy. The positive relationship between allodynia and disability is well established (Baykan et al., 2016; Lipton et al., 2008), and recent research exploring psychological mediators of headache variables show that both self-efficacy (Peck & Smitherman, 2015) and fear of pain (Black, Fulwiler, & Smitherman, 2015) mediate the relationship between headache severity and disability. Allodynia is closely related to migraine severity, such that migraineurs with more severe allodynia experience more severe head pain (Jakubowski, Silberstein, Ashkenazi, & Burstein, 2005). The

close relationship between allodynia and pain severity suggests that exploring the mediating role of psychological variables, such as fear of pain, on the relationship between allodynia and disability is worth exploring in future research. The present study explored self-efficacy, rather than disability, as a dependent variable because evidence suggests that self-efficacy is both an important mechanism of change and treatment outcomes in migraine management.

### **Supplementary Analysis**

Prior research demonstrates that patients with aura and chronic migraine experience more severe allodynia than those with episodic migraine and without aura (Lipton et al., 2008). To test whether the predicted mediation effect was attributable to collapsing episodic and chronic migraineurs, supplemental analyses were conducted exploring differences in allodynia as a function of headache diagnosis. Contrary to prior findings, the current study found no significant differences in allodynia between participants with migraine, migraine with aura, and chronic migraine diagnoses. However, allodynia severity in the current sample was, on average, somewhat lower than that from prior studies (Bigal et al., 2008), though many studies simply report the presence versus absence of allodynia rather than severity ratings.

### **Limitations and Future Directions**

The present study is strengthened by adherence to ICHD-3 diagnostic criteria; use of validated measures of headache disability, headache-related self-efficacy, fear of pain, and allodynia; and a sample comprised of young adults with frequent migraine. However, some limitations exist and should be considered when interpreting the study's conclusions. First, the study relied on retrospective report concerning headache symptomatology and associated psychological variables. While the computer-administered SDIH-3 adheres to ICHD-3 diagnostic criteria, interview-based diagnoses would strengthen confidence in validity of the current study.

Similarly, quantitative sensory testing (QST) is the gold standard method for assessing allodynia. While the ASC-12 is a well-validated self-report measure of allodynia symptoms, the use of QST evaluative methods would provide more objective data concerning allodynia symptoms experienced during and between migraine attacks. Second, given that the sample primarily consisted of non-treatment seeking, young undergraduate students, results may not generalize to clinical populations or older adults given that treatment seeking samples present with both more severe pain quality and allodynia symptoms. However, the current sample endorsed nearly ten headache days per month, a strikingly high frequency for an undergraduate, non-treatment seeking sample. Further, given that allodynia is associated with migraine chronicity (Bayken et al., 2016; Lipton et al, 2008), the current sample may not present with highly complex headache histories, though they reported high migraine frequency and marked disability resulting from head pain. Young adults in their mid-twenties to early thirties may present with more extensive headache and allodynia histories, given that allodynia prevalence is highest among adults aged 18-36 (Baykan et al.). However, mean age of the present sample falls within this age range. Though it is possible that the predicted mediation effect may not prove significant if the study is underpowered, the present study utilized a sample nearly twice that required based on an a priori power analysis. Finally, the present study employed cross-sectional methods and as such, associations should be recognized as correlational rather than causal given uncertainty regarding temporal relationships among variables.

Future research should utilize treatment-seeking migraineurs and physiologically based methods for assessing allodynia, such as QST. Additionally, future research should endeavor to understand the relationship between allodynia and self-efficacy through other pain-related variables, such as medication overuse, or attend to outcomes variables more consistently linked

to allodynia such as disability or headache frequency (Baykan et al., 2016; Lipton et al., 2008). The significant negative association between allodynia and self-efficacy suggests that self-efficacy may be an important target for and mechanism of change in behavioral interventions for patients with allodynia, although this awaits empirical verification. Studies examining the utility of behavioral interventions broadly, and on self-efficacy specifically, among migraineurs with allodynia may highlight important clinical considerations in treating these patients.

Allodynia may also show relationships with variables such as fear of pain and medication overuse due to disproportionately high rates of avoidance behaviors, consistent with the FAM. Exploring the topography of avoidance behaviors among migraineurs with and without allodynia could expose meaningful differences in pain-related avoidance behaviors among patients with allodynia. Such differences could inform targeted behavioral interventions for allodynia. Additionally, given the shared physiological underpinnings of migraine, allodynia, and medication overuse, psychological variables leading to central sensitization, such as chronic or prolonged stress, may be relevant clinically. Finally, literature to date is mixed regarding psychological correlates of allodynia, such as depression and anxiety, likely due to varying methodological approaches. More conclusive research in this area is needed to improve understanding of psychological comorbidities among migraineurs with allodynia.



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**Table 1.** Demographic Characteristics of the Sample

<b>Variable</b>	<b>% or Mean (SD)</b>
Gender (% Female)	87.8
Mean Age (SD)	18.98 (2.39)
Race (% Caucasian)	86.4
Episodic Migraine % (w/o aura)	38.1
Episodic Migraine % (w/ aura)	33.3
Chronic Migraine %	28.6
Mean Headache Days/Month (SD)	9.90 (5.85)
Employment (% Unemployed)	70.7
Income (% <\$10,000)	36.1
Religion (% Protestant Christian)	40.1
Relationship (% Not In Relationship)	60.5
Father's Education (% $\geq$ bachelor's degree)	55.1
Mother's Education (% $\geq$ bachelor's degree)	65.9
Mean Headache Severity (SD)	6.02 (1.50)
Mean ASC-12 Score (SD)	3.69 (3.46)
Mean HMSE Score (SD)	101.18 (19.72)
Mean HIT-6 Score (SD)	60.76 (6.68)
Mean PASS-20 Score (SD)	34.70 (15.72)

**Table 2.** Pearson correlations among allodynia and associated headache variables

Measure	1	2	3	4
1. ASC-12				
2. HIT-6	.18*			
3. PASS-20	.22**	.22**		
4. Frequency	.14	.46**	.25**	
5. Severity	.50**	.49**	.17*	.25**

\*  $p < .05$

\*\*  $p < .01$

**Table 3.** Group differences between migraineurs with and without allodynia.

	<b>Allodynia group mean (SD)</b>	<b>No allodynia group mean (SD)</b>	<b><i>t</i>-tests</b>
<b>HIT-6</b>	61.60 (6.29)	59.71 (7.05)	<i>t</i> (145) = -1.71
<b>PASS-20</b>	37.00 (17.07)	31.81 (13.40)	<i>t</i> (145) = -2.01*
<b>HMSE-25</b>	99.79 (17.57)	102.93 (22.15)	<i>t</i> (145) = .96

\* *p* = .04

**Table 4.** Regression Analysis Results

			Adjusted			
	Beta	95% CI for Beta	R <sup>2</sup>	ΔR <sup>2</sup>	P- value of ΔR <sup>2</sup>	P- value of Model
<b>Block 1</b>			15.5%			<.001
Frequency	-.002	-.59 – .58				
HIT-6	-.393	-1.67 – -.65				
<b>Block 2</b>			16.4%	1%	.214	<.001
ASC-12	-.097	-1.43 – .32				

CI = confidence interval

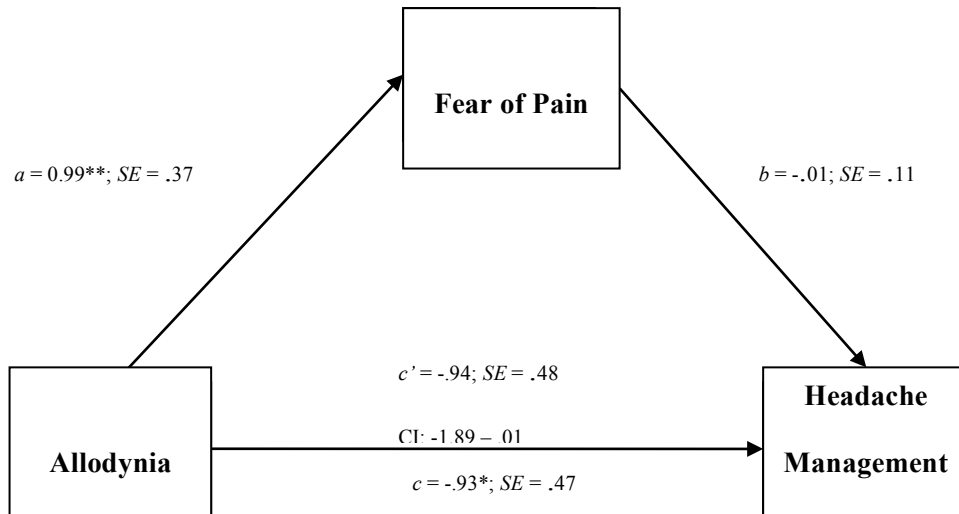


**Table 5.** One-way ANOVA comparing allodynia across migraine diagnoses

	<b>Sum of Squares</b>	<b>df</b>	<b>Mean Square</b>	<b>F</b>	<b>Sig.</b>
<b>Between Groups</b>	20.912	2	10.456	.872	.420
<b>Within Groups</b>	1726.313	144	11.988		
<b>Total</b>	1747.224	146			

## LIST OF FIGURES

**Figure 1.** Path coefficients for simple mediation analysis on self-efficacy



*Note:*  $c$  denotes the total effect while  $c'$  denotes the effect of allodynia on HMSE when fear of pain is not included as a mediator.

\*  $p < .05$

\*\*  $p < .01$

## LIST OF APPENDICES

## APPENDIX A: DEMOGRAPHIC INFORMATION

**Please answer the following questions.**

1. What is your biological sex?  
0 = Male  
1 = Female
2. How old are you? \_\_\_\_ (Ranges from 18 to 64)
3. What is your marital status?  
0 = Never married  
1 = Married  
2 = Divorced/Annulled  
3 = Separated  
4 = Widowed  
5 = Not married, but living with partner
4. Who do you currently live with? Check all that apply.  
0 = Alone  
1 = Spouse or romantic partner  
2 = Children (under age 18)  
3 = Other relative  
4 = Friend or roommate
5. What is your *highest* education level completed?  
0 = Elementary (8<sup>th</sup> grade or less)  
1 = Some High School  
2 = High School Diploma  
3 = Some College  
4 = Bachelor's Degree  
5 = Master's Degree  
6 = Doctoral or professional degree (PhD, MD, etc.)
6. What best describes your current employment status?  
0 = Unemployed  
1 = Home Maker  
2 = Part-Time  
3 = Full-time (40 hours per week or more)
7. What best describes your total household income (before taxes)?  
0 = Less than 10,000  
1 = \$10,000 to \$20,000  
2 = \$21,000 to \$30,000  
3 = \$31,000 to \$50,000  
4 = \$51,000 to \$100,000  
5 = Greater than \$100,000
8. Do you describe yourself as a Hispanic or Latino?  
0 = No  
1 = Yes
9. What is your race?  
0 = White  
1 = Black/African American  
2 = Asian  
3 = Native American, Alaskan Native  
4 = Asian or Pacific Islander  
5 = Native Hawaiian or Other Pacific Islander  
6 = Multiracial (list numbers \_\_\_\_ & \_\_\_\_ & \_\_\_\_)
10. What is your religious affiliation?
  1. Protestant Christian
  2. Roman Catholic
  3. Evangelical Christian
  4. Jewish
  5. Muslim
  6. Hindu
  7. Buddhist
  8. Other: \_\_\_\_\_
  9. I am not religious

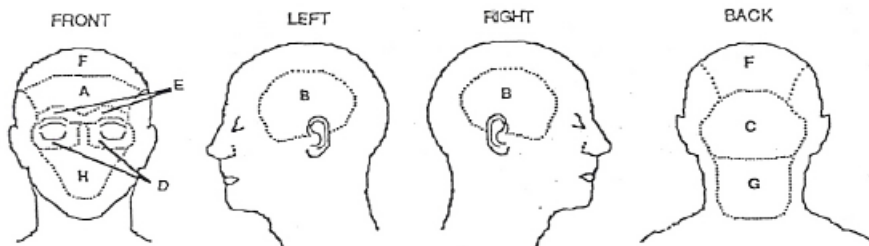
APPENDIX B: STRUCTURED DIAGNOSTIC INTERVIEW FOR HEADACHE – 3 (BRIEF  
VERSION (SDIH-3))

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

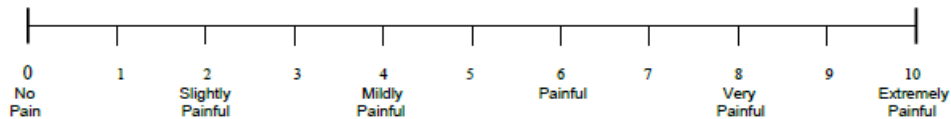
Patient Name:	Age:	Sex: M F
Patient ID:	Interviewer:	Date: / /

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 ≥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

## Section 4a

## Migraine Aura Symptoms

1. How many aura attacks has the patient experienced? \_\_\_\_

2. Which of the following apply to the aura symptoms? (*Select all that apply*)

- At least one aura symptom spreads gradually over  $\geq 5$  minutes, AND/OR 2 or more symptoms occur in succession
- Each individual aura symptom lasts 5-60 minutes
- At least one aura symptom is unilateral
- The aura is accompanied, or followed within 60 minutes, by headache

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

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

## Section 4b

## Cluster Headache Symptoms

1. Have the headaches occurred in cluster periods?  Yes  No

If **YES**, complete #1a and #1b

If **NO**, skip to #2

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

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

2. Are the headaches separated by remission periods?  Yes  No

If **YES**, complete #2a

If **NO**, skip to #3

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

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

X	SYMPTOM	SIDE	X	SYMPTOM	SIDE
<input type="checkbox"/>	Red eye (conjunctival injection)	R L	<input type="checkbox"/>	Forehead and facial sweating	R L
<input type="checkbox"/>	Tearing of the eye (lacrimation)	R L	<input type="checkbox"/>	Forehead and facial flushing	R L
<input type="checkbox"/>	Nasal congestion	R L	<input type="checkbox"/>	Eyelid swelling (oedema)	R L

<input type="checkbox"/>	Runny nose (rhinorrhoea)	R L	<input type="checkbox"/>	Drooping eyelids (ptosis)	R L
<input type="checkbox"/>	Pupillary constriction (miosis)	R L	<input type="checkbox"/>	Sensation of fullness in the ear	R L
<input type="checkbox"/>	Restlessness or agitation		<input type="checkbox"/>	Other:	

<b>Section 4c</b>	<b>Medication-Overuse Headache Symptoms</b>
-------------------	---

1. Has intake of ergotamine, triptans, or opioids occurred on 10 or more days per month, for over 3 months?  Yes  No  
If **YES**, indicate drug(s):  ergotamine  triptan  opioid \_\_\_\_\_
2. Has the patient's intake of simple analgesics (eg, acetaminophen, acetylsalicylic acid, other NSAID), occurred on 15 or more days per month, for over 3 months?  Yes  No  
If **YES**, indicate drug: \_\_\_\_\_
3. Has the patient's intake of combination analgesics occurred on 10 or more days per month, for over 3 months?  
 Yes  No  
If **YES**, indicate drugs: \_\_\_\_\_
4. Has intake of any combination of ergotamine, triptans, simple analgesics, NSAIDs, and/or opioids occurred on 10 or more days per month, for over 3 months (without overuse of any single class alone)?  Yes  No  
If **YES**, indicate drug(s): \_\_\_\_\_

<b>Section 4d</b>	<b>Post-Traumatic Headache Symptoms</b>
-------------------	---

1. Did headache develop within 7 days after head trauma (or after regaining consciousness, or after regaining the ability to sense and report pain)?  Yes  No
2. Was there a loss of consciousness associated with head trauma?  Yes  No  
If **YES**, complete #2a  
If **NO**, skip to #3
- 2a. What was the duration of unconsciousness? \_\_\_\_ m h d (*Indicate duration in minutes, hours, or days*)
3. How long has the headache continued? (*Select most representative category*)  
 Resolves within 3 months after head trauma  
 Persists for greater than 3 months after head trauma  
 Persists but 3 months have not passed since head trauma
4. Is head injury attributed to whiplash?  Yes  No  
If **YES**, skip #5 through #9  
If **NO**, complete #5 through #9
5. Did coma develop?  Yes  No  
If **YES**, indicate severity on Glasgow Coma Scale (GCS):  GCS <13 [*moderate/severe*]  GCS ≥13 [*mild*]
6. Did post-traumatic amnesia develop and continue for longer than 24 hours?  Yes  No
7. Was there alteration in level of awareness for longer than 24 hours?  Yes  No
8. Were abnormal neuroimaging results attained suggestive of a traumatic head injury?  Yes  No
9. Immediately after the head injury, were any of the following present? (*Select all that apply*)  
 Transient confusion, disorientation, or impaired consciousness  
 Loss of memory for events immediately before or after the head injury  
 At least two symptoms suggestive of mild traumatic brain injury (nausea, vomiting, visual disturbances, dizziness and/or vertigo, impaired memory and/or concentration) (*Circle all symptoms that apply*)

APPENDIX C: HEADACHE MANAGEMENT SELF-EFFICACY SCALE – 25 (HMSE-25)

**Instructions:** You will find below a number of statements related to headaches. Please read each statement carefully and indicate how much you agree or disagree with the statement by circling a number next to it. Use the following scale as a guide:

Strongly Disagree	Moderately Disagree	Slightly Disagree	Neither Agree or Disagree	Slightly Agree	Moderately Agree	Strongly Agree
1	2	3	4	5	6	7

- 1) I can keep even a *bad* headache from disrupting my day by changing the way I respond to the pain. 1 2 3 4 5 6 7
- 2) When I'm in some situations, nothing I do will prevent headaches.\* 1 2 3 4 5 6 7
- 3) I can reduce the intensity of a headache by relaxing. 1 2 3 4 5 6 7
- 4) There are things I can do to reduce headache pain. 1 2 3 4 5 6 7
- 5) I can prevent headaches by recognizing headache triggers. 1 2 3 4 5 6 7
- 6) Once I have a headache there is nothing I can do to control it.\* 1 2 3 4 5 6 7
- 7) When I'm tense, I can prevent headaches by controlling the tension. 1 2 3 4 5 6 7
- 8) Nothing I do reduces the pain of a headache.\* 1 2 3 4 5 6 7
- 9) If I do certain things every day, I can reduce the number of headaches I will have. 1 2 3 4 5 6 7
- 10) If I can catch a headache before it begins I often can stop it. 1 2 3 4 5 6 7

*Please complete reverse side*

Strongly Disagree	Moderately Disagree	Slightly Disagree	Neither Agree or Disagree	Slightly Agree	Moderately Agree	Strongly Agree
1	2	3	4	5	6	7

- 11) Nothing I do will keep a mild headache from turning into a bad headache.\* 1 2 3 4 5 6 7
- 12) I can prevent headaches by changing how I respond to stress. 1 2 3 4 5 6 7

13)	I can do things to control how much my headaches interfere with my life.	1	2	3	4	5	6	7
14)	I <u>cannot</u> control the tension that causes my headaches.*	1	2	3	4	5	6	7
15)	I can do things that will control how long a headache lasts.	1	2	3	4	5	6	7
16)	Nothing I do will keep a bad headache from disrupting my day.*	1	2	3	4	5	6	7
17)	When I'm not under a lot of stress I can prevent many headaches.	1	2	3	4	5	6	7
18)	When I sense a headache is coming, there is nothing I can do to stop it.*	1	2	3	4	5	6	7
19)	I can keep a <i>mild</i> headache from disrupting my day by changing the way I respond to the pain.	1	2	3	4	5	6	7
20)	If I am under a lot of stress there is nothing I can do to prevent headaches.*	1	2	3	4	5	6	7
21)	I can do things that make a headache seem not so bad.	1	2	3	4	5	6	7
22)	There are things I can do to prevent headaches.	1	2	3	4	5	6	7
23)	If I am upset there is nothing I can do to control the pain of a headache.*	1	2	3	4	5	6	7
24)	I can control the intensity of headache pain.	1	2	3	4	5	6	7
25)	I can do things to cope with my headaches.	1	2	3	4	5	6	7

APPENDIX D: HEADACHE IMPACT TEST – 6 (HIT-6)

# HIT-6™

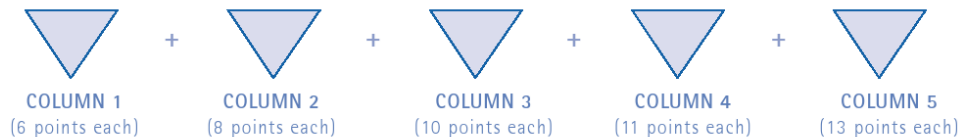
(VERSION 1.1)

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.



<b>1</b>	<b>When you have headaches, how often is the pain severe?</b>				
	Never	Rarely	Sometimes	Very Often	Always
<b>2</b>	<b>How often do headaches limit your ability to do usual daily activities including household work, work, school, or social activities?</b>				
	Never	Rarely	Sometimes	Very Often	Always
<b>3</b>	<b>When you have a headache, how often do you wish you could lie down?</b>				
	Never	Rarely	Sometimes	Very Often	Always
<b>4</b>	<b>In the past 4 weeks, how often have you felt too tired to do work or daily activities because of your headaches?</b>				
	Never	Rarely	Sometimes	Very Often	Always
<b>5</b>	<b>In the past 4 weeks, how often have you felt fed up or irritated because of your headaches?</b>				
	Never	Rarely	Sometimes	Very Often	Always
<b>6</b>	<b>In the past 4 weeks, how often did headaches limit your ability to concentrate on work or daily activities?</b>				
	Never	Rarely	Sometimes	Very Often	Always



To score, add points for answers in each column.  
Please share your HIT-6 results with your doctor.

**Total Score**

Higher scores indicate greater impact on your life.

Score range is 36-78.

HIT-6™ US (English) Version 1.1  
©2000, 2001 QualityMetric, Inc. and GlascoSmithKline Group of Companies



APPENDIX E: ALLODYNIA SYMPTOM CHECKLIST – 12 (ASC-12)

<b>How often do you experience increased pain or an unpleasant sensation on your skin during your most severe type of headache when you engage in each of the following?</b>	<b>Does not apply to me (0)</b>	<b>Never (0)</b>	<b>Rarely (0)</b>	<b>Less than half of the time (1)</b>	<b>Half of the time or more (2)</b>
Combing your hair					
Pulling your hair back (e.g., ponytail)					
Shaving your face					
Wearing eyeglasses					
Wearing contact lenses					
Wearing earrings					
Wearing a necklace					
Wearing tight clothing					
Taking a shower (when shower water hits your face)					
Resting your face or head on a pillow					
Exposure to heat (e.g., cooking, washing your face with hot water)					
Exposure to cold (e.g., using an ice pack, washing your face with cold water)					
<b>Total score</b>					
<b>Sum of total scores</b>					

**Allodynia Symptom Checklist – 12 (ASC-12)**

Scoring:

- None            0-2
- Mild            3-5
- Moderate      6-8
- Severe         9 or more

APPENDIX F: PAIN ANXIETY SYMPTOM SCALE – 20 (PASS-20)



## CURRICULUM VITA

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**Ashley N. Polk, B.A.**

CURRICULUM VITAE

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### EDUCATION

- M.A. University of Mississippi** (Oxford, MS) Anticipated May 2019  
Clinical Psychology  
Thesis: "Allodynia and Self-Efficacy in Migraineurs"  
Advisor: Todd A. Smitherman, Ph.D.  
GPA: 3.93
- B.A. University of Mississippi** (Oxford, MS) 2016  
Major in Psychology; Minor in Neuroscience  
GPA: 3.86, Magna Cum Laude

### CLINICAL EXPERIENCE

- Institute of Community Services Head Start** September 2018-present  
Mental Health Consultant  
Supervisor: Alan Gross, Ph.D.  
Created and implemented behavioral interventions and programs for children aged five and younger enrolled in Head Start programs throughout North Mississippi. Consulted with teachers, administrators, and parents concerning children's behavior at home and in the classroom. Created and led parenting workshops.
- University of Mississippi Psychological Assessment Center** August 2018-present  
Graduate Assessment Team  
Supervisor: Scott Gustafson, Ph.D., ABPP  
Administered full battery cognitive and achievement assessments, ADHD and learning disability assessments, law enforcement fit-for-duty assessments, bariatric assessments, and athletic assessments for university and community outpatient adults and children.
- North Mississippi Regional Center** July 2017-June 2018  
Psychological and Behavioral Services Intern  
Supervisors: Giovanni Biffle, M.A., BCBA & Kim Sallis, Ph.D.

Provided individual interventions for inpatient clients with intellectual and developmental disabilities including Prader-Willi Syndrome and Lesch-Nyhan Syndrome. Planned and implemented individually tailored interventions using applied behavior analysis. Conducted formal full-battery assessments with children and adults suspected of having developmental, intellectual, and behavioral disorders.

**University of Mississippi Psychological Services Center** August 2017-present  
Graduate Therapist

Supervisors: Todd Smitherman, Ph.D., Scott Gustafson, Ph.D., ABPP, John Young, Ph.D.  
Provided individual cognitive-behavioral therapy (CBT) for university and community outpatient adults and children with DSM-5 disorders.

## RESEARCH EXPERIENCE

**Graduate Research Assistant, University of Mississippi** 2016-present  
Migraine & Behavioral Health Laboratory  
Advisor: Todd Smitherman, Ph.D.

**Undergraduate Research Assistant, University of Mississippi** 2015-2016  
Migraine & Behavioral Health Laboratory  
Advisor: Todd Smitherman, Ph.D.

**Undergraduate Research Assistant, University of Mississippi** 2014-2016  
Psychopharmacology Research Laboratory  
Advisor: Kenneth Sufka, Ph.D.

**Summer Research Intern, Center of Biomedical Research Excellence** 2015  
Grant Number: NIGMS P20GM104932  
Supervisor: Michael Repka, D.D.S., Ph.D.

## PUBLICATIONS & PRESENTATIONS

### Journal Articles

Davis-Martin, R. E., **Polk, A. N.**, & Smitherman, T. A. (2017). Alcohol use as a comorbidity and precipitant of primary headache: Review and meta-analysis. *Current Pain and Headache Reports*, 21(42). DOI: <https://doi.org/10.1007/s11916-017-0642-8>

Tiwari, R., **Polk, A.**, Patil, H., Ye, X., Pimparade, M., Repka, M. (2015). Rat palatability

study for taste assessment of caffeine citrate formulation prepared via hot-melt extrusion technology. *AAPS PharmSciTech*. Advanced online publication. DOI: 10.1208/s12249-015-0447-1

### **Oral Presentations**

**Polk, A.** & Smitherman, T. (2019, April). *Allodynia, stress, and disability in migraineurs: A mediation analysis*. Presented at the 2019 UM Conference on Psychological Science, Oxford, MS.

**Polk, A.** (2018, April). *What's your path? M.A., Ph.D., Psy.D.* Presented at the 2018 UM Conference on Psychological Science, Oxford, MS.

**Polk, A.**, Johnson, Y., & Smitherman, T. (2017, September). *Comparing the effects of cognitive & social stress in individuals with headache*. In M. Perry (Chair), *The age of anxiety: Exploring and assessing anxiety and its' problematic health correlates*. Presented at the Mississippi Psychological Association's 68th Annual Convention; Biloxi, MS.

Sharpe, K. M., **Polk, A. N.**, & Smitherman, T. A. (2017, April) *Perceptions of headache trigger potency*. Presented at the 2017 UM Conference on Psychological Science, Oxford, MS.

### **Poster Presentations**

Wittig, C., Cochran, S., Mullen, C., **Polk, A.**, & Smitherman, T. (2019, April). *Psychological correlates of allodynia*. Presented at the 2019 UM Conference on Psychological Science, Oxford, MS.

Sharpe, K. M., Crow, M. K., **Polk, A. N.**, & Smitherman, T. A. (2016, April). *Trigger stimuli in young adults with migraine and tension-type headache*. Presented at the 2016 UM Conference on Psychological Science, Oxford, MS.

### **ATTENDED WORKSHOPS & TRAININGS**

Hayes, A. F. (2018, October). *Introduction to Mediation, Moderation, and Conditional Process Analysis*. Presented at the University of Mississippi.

### **PROFESSIONAL MEMBERSHIPS**

Student Member, Association for Behavioral and Cognitive Therapies (ABCT)

Student Member, Mississippi Psychological Association (MPA)  
Member, Women in Behavior Analysis (WIBA)

### **TEACHING EXPERIENCE**

Instructor of Record PSY 410, Health Psychology	July 2018
Graduate Teaching Assistant PSY 309, Learning PSY 201, General Psychology	2018-present 2016-2018
Undergraduate Teaching Assistant PSY 341, Multicultural Psychology	2015-2016

### **AWARDS & ACHIEVEMENTS**

Graduate Honors Fellowship	2016-present
Chancellor's Honor Roll, University of Mississippi	2012-2016
Phi Kappa Phi Honor Society	
Phi Beta Kappa Honor Society	
Provost Scholar, University of Mississippi	

### **PROFESSIONAL EXPERIENCE**

<b>Administrative Assistant to the Clinical Training Program</b> DCT & Supervisor: Todd Smitherman, Ph.D. Assisted with incoming graduate student orientation; planned, coordinated, and executed clinical and experimental programs' Interview Weekend; assisted with APA self-study; coordinated details of faculty candidate interviews	July 2018-present
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### **LEADERSHIP EXPERIENCE & SERVICE**

<b>Chancellor's Standing Committees</b> University of Mississippi LGBTQ Affairs Artist Series	August 2018-present
<b>Graduate Student Faculty Representative</b>	August 2018-present



University of Mississippi Psychology Department

**Graduate Student Council**

May 2018-present

Director of Social & Philanthropic Affairs  
University of Mississippi

**Chair's Graduate Student Advisory Council**

2016-2017

University of Mississippi Psychology Department

**Judicial Board**

2015-2016

University of Mississippi

**RELEVANT COURSEWORK**

ACT Seminar

Spring 2019

Instructor: Kelly Wilson, Ph.D.

Seminar on College Teaching

Spring 2018

Instructor: Kenneth Sufka, Ph.D.

Evidence Based Treatments Seminar

Fall 2017

Instructor: John Young, Ph.D.

**REFERENCES**

References available upon request.