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The Role Of Fear Of Pain In Headache

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THE ROLE OF FEAR OF PAIN IN HEADACHE

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

by
ANNA KATHERINE BLACK

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ABSTRACT

Recurrent headache sufferers are often fearful of pain, which disrupts cognitive thought processes, interferes with daily activities, and may maintain headache-related disability through avoidance and associated negative reinforcement. The aim of this cross-sectional study was to 1) examine differences in fear of pain between headache sufferers and non-headache controls; 2) examine differences in fear of pain across primary headache diagnostic groups; 3) assess the extent to which fear of pain predicts headache variables (e.g., severity, frequency, disability); and 4) determine whether fear of pain mediates the relationship between pain severity and headache-related disability. The sample consisted of 908 young adults ages 18-49 (M = 19.52 years; 64.9% female). Of those, 237 (26.1%) met the diagnostic criteria for episodic tension-type headache, 232 (25.6%) for episodic migraine (167 [18.4%] without aura and 65 [7.2%] with aura), 38 (4.2%) for chronic migraine, and 19 (2.1%) for chronic tension-type headache; 382 (42.1%) served as non-headache controls. Fear of pain differed among groups, with headache sufferers reporting greater fear of pain than those without headache; migraineurs typically endorsed greater fear of pain than those with tension-type headache. Among those with headache, fear of pain significantly predicted headache severity ($R^2 = 6.1\%$) and frequency ($R^2 = 4.5\%$), and accounted for more variance in disability ($R^2 = 17.5\%$) than gender, anxiety, and depression combined (13.8%). Pain severity and disability were strongly associated ($r = .61, p < .001$), and fear of pain partially mediated this association (indirect effect point estimate = 0.38; 95% confidence interval [CI]: 0.23 to 0.57). Fear of pain differentiates migraineurs from those without headache and plays a significant role in primary headache, particularly in headache-related
disability. Findings build upon and extend those from previous chronic pain studies and highlight the need for longitudinal and experimental studies to further explore this construct in headache.

*Key words:* Headache, fear of pain, mediator, disability, migraine, tension-type headache
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<tr>
<td>TTH</td>
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<td>ETTH</td>
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I. INTRODUCTION

Primary Headache Diagnoses

Headache is one of the primary reasons for medical consultation (Linet, Celentano, & Stewart, 1991) and results in lost wages, decreased productivity, and high health care costs at both an individual and societal level (Edmeads & Mackell, 2002; Stewart, Lipton, Celentano, & Reed, 1992). Stewart and colleagues (2003) estimated that headache disorders account for approximately 9% of the annual lost labor productivity in the United States. According to a review of population-based studies, headache is among the 10 most disabling medical conditions worldwide and among the fifth most disabling conditions among women specifically (Stovner et al., 2007). In the United States, 46% of the population receives a diagnosis of a “primary” headache disorder each year (Edmeads et al., 1993; Stovner et al., 2007), which denotes those headache disorders that are not attributable to other diseases and lack an identifiable cause (International Headache Society, 2004; Lipton, Silberstein, & Dodick, 2008). The ICHD-II divides primary headaches into four categories based on attack duration and symptoms: migraine, tension-type headache (TTH), cluster headache, and other primary headaches.

Migraine and tension-type headache (TTH) are the two most common primary headache disorders, with lifetime prevalence rates of 14% and 42%, respectively (Stovner et al., 2007). According to the 2010 Global Burden of Disease study, TTH is the second most common disorder in the world, followed immediately by migraine (Vos et al., 2013). Migraine is characterized by recurrent, moderate to severe head pain, often unilateral and pulsating in nature,
that typically lasts 4-72 hours if untreated or unsuccessfully treated. In addition, head pain is often aggravated by routine physical activity (e.g., walking) and accompanied by nausea and/or vomiting, and sensitivity to both light (photophobia) and sound (phonophobia). Migraine may occur with or without aura symptoms, which are temporary neurological features that occur prior to onset of pain and resolve thereafter. Typical aura symptoms include visual (e.g., seeing flickering lights, spots, or zigzag lines), sensory (e.g., tingling or numbing sensations), or speech disturbances (Lipton, Scher, Silberstein, & Bigal, 2008). Two percent of migraineurs experience frequent migraine attacks on 15 or more days per month (“chronic migraine”; Lipton & Bigal, 2005; Lipton, Silberstein, & Dodick, 2008). Epidemiological data indicate a threefold female to male preponderance of migraine (18% vs. 6%; Lipton, Stewart, Diamond, Diamond, & Reed, 2001; Lipton, Stewart, & Simon, 1998; Stewart et al., 1992; Schwartz, Stewart, & Simon, & Lipton 1998), with peak prevalence between 25 and 55 years of age (Lipton et al., 2001; Lipton et al., 2002). Migraine is most common among Caucasians, followed by African Americans and Asian Americans (Stewart, Lipton, & Liberman, 1996).

Tension-type headache, though less disabling than migraine, is the single most prevalent primary headache disorder, with one-year prevalence ranging from 31% to 74% (Schwartz et al., 1998). In comparison to migraine, TTH symptoms are mild to moderate in severity, and thus many individuals with episodic TTH (ETTH, <15 days/month) rarely seek treatment (Lenaerts & Newman, 2008). Unlike the severe, throbbing, one-sided pain of migraine, TTH is characterized predominantly by pressing or tightening head pain (i.e., non-pulsatile), is typically bilateral in nature, and is not aggravated by routine physical activity. Another requirement for a diagnosis of TTH is an absence of nausea or vomiting, and only one of either photophobia or phonophobia may be present (Lipton, Silberstein, & Dodick, 2008). Frequency and duration of TTH vary
extensively; these headaches may last anywhere from 30 minutes to 7 days and can be 1) infrequent, occurring 1 day or less per month (infrequent episodic TTH, or ETTH), 2) frequent, occurring 1 to 14 days per month (frequent ETTH), or 3) chronic, occurring 15 or more days per month (CTTH). Like migraine, TTH is more prevalent among women (42%) than men (36%), with the highest prevalence among Caucasian individuals (Schwartz et al., 1998). Typical onset of TTH is between 20 and 30 years of age and peaks between 30 and 39 years of age (Schwartz et al., 1998).

Impact of Headache

The negative impact associated with the symptoms, frequency, and severity of headache attacks can be quite substantial (Lipton, Bigal, Hamelsky, & Scher, 2008). Solomon and colleagues (1993) found that chronic headache sufferers reported more functional impairment than did diabetes, arthritis, and chronic back pain patients. Data from the 2010 Global Burden of Disease survey indicated that migraine is the eighth leading cause of disability in the world (Vos et al., 2013). In a Canadian study of migraineurs, 90% of participants reported postponing household work because of their migraines (Edmeads et al., 1993). In addition, more than 50% of migraineurs in the US reported severe impairment or bed rest during attacks, and 35% indicated at least one day of activity restriction due to migraine over a 3-month period (Lipton et al., 2007). As such, the economic impact of headache is substantial and includes both direct costs (e.g., utilization of health care services) and indirect costs (e.g., absenteeism, reduced work productivity; Lipton, Bigal, Hamelsky, & Scher, 2008). Migraineurs have higher direct medical costs than the general public due to higher rates of emergency room visits and physician appointments, as well as costs associated with prescription migraine medications (Edmeads & Mackell, 2002). Although TTH does not often cause pronounced functional impairment
(McGeeney, 2009), Schwartz, Stewart, and Lipton (1997) reported that 19% of individuals with ETTH missed workdays and 22% reported reduced effectiveness at work during their headache episodes. Thus, although migraine is more burdensome to the individual sufferer, the societal burden of TTH is actually greater because of its much higher prevalence (Stovner et al., 2007). Regardless of differences in prevalence, clearly both migraine and TTH are substantial causes of disability.

Psychological Variables

Although migraine and TTH are conceptualized as neurological disorders, they often co-occur with psychiatric disorders such as anxiety and depression (Breslau, 1998; Radat & Swendsen, 2005). Compared to those without migraine, episodic and chronic migraineurs are 2-5 times more likely to have a comorbid mood or anxiety disorder (Breslau, 1998; Jette, Patten, Willam, Becker, & Wiebe, 2008, Radat & Swendsen, 2005), with prevalence of these disorders highest among those with more frequent headache (i.e., CM; Karakurum, Soylu, & Karatas, et al., 2004). These comorbid mental disorders are associated with increased headache-related disability (Lanteri-Minet, Radat, Chautard, & Lucas, 2005; Saunders, Merikangas, Low, Von Korff, & Kessler, 2008), and anxiety appears to be more related to disability than depression (Hamelsky & Lipton, 2006; Lanteri-Minet et al., 2005; Smitherman, Penzien, & Maizels, 2008; Zwart et al., 2003). In addition, many headache sufferers often report anxiety and stress as triggers of individual headache attacks (Kelman, 2007; Martin, Milech, & Nathan, 1993) or worrying about the causes and consequences of headache (Penzien, Holroyd, Holm, & Hursey, 1985). Specifically, Smith and Nicholson (2006) found that reductions in anxiety after a 6-month period was a better predictor of reductions in headache-related disability than changes in headache frequency or medication usage. Psychiatric comorbidity is an important factor in
understanding headache-related disability, as are other psychological constructs.

For years, the medical model of disease conceptualized pain-related disability as a function primarily of pain severity (Asmundson et al., 1999). For example, in a sample of treatment-seeking migraineurs, Magnusson and Becker (2003) observed that headache intensity was more predictive of headache-related disability than attack frequency. Despite these findings, other evidence suggests that headache-related disability cannot be fully explained by intensity or frequency (Stewart et al., 2003). Indeed, the last 30 years of pain research have been marked by an expansion of interest into the psychological (e.g., cognitions, affective states, avoidance behaviors) factors that influence pain (Asmundson et al., 1999). Weisenburg (1998) highlighted the importance of cognitive appraisals (i.e., one’s interpretation of painful stimuli), patient beliefs (e.g., one’s thoughts about the meaning of symptoms, ability to control pain, or the impact of pain on one’s life), and coping styles as significant factors in the individual experience of pain. In a headache sample, French and colleagues (2000) found that increased levels of self-efficacy, or one’s confidence in her abilities to successfully prevent and manage headache, were associated with utilization of more effective coping strategies. The authors also found that affective factors, such as increased levels of emotional distress, were associated with increased disability. One multidimensional construct that incorporates all four aspects of pain experience (physiological, cognitive, behavioral, and affective) is fear of pain (McCracken, Zayfert, & Gross, 1992). In the broader chronic pain literature, fear of pain appears to be as, if not more, disabling than pain itself (Crombez, Vlaeyen, Heuts, & Lysens, 1999; McCracken, Gross, Sorg, & Edmunds, 1993; Vlaeyen & Linton, 2000; Waddell, 1996).

Fear of Pain
Fear of pain refers to a fear of physical movement or activity due to a presumed susceptibility to pain (Swinkels-Meewisse, Roelofsm, Verbeek, Oostendorp, & Vlaeyen, 2003). If pain is perceived as being threatening or harmful, pain-related fear evolves. The cognitive, affective, and physiological components of pain-related fear play an integral role in pain-related escape and avoidance (Leeuw, Goossens, Linton, Crombez, Boersma, & Vlaeyen, 2007). Specifically, individuals with high fear of pain manifest hypervigilance to the physiological symptoms of pain (e.g., sympathetic arousal) and to environmental threat stimuli (Crombez, Eccleston, Van Damme, Vlaeyen, & Karoly, 2012; Nash, Williams, Nicholson, & Trask, 2006). Hypervigilance prompts avoidance of activities and contexts often unrelated to pain and leads to physical deconditioning, ultimately increasing functional impairment. This fear-avoidance (FA) model of pain, first proposed by Lethem and colleagues (1983), has been used to explain the transition from acute to chronic pain syndromes. Pain-related avoidance behaviors and attentional allocation initially play an adaptive role in protecting the body from re-injury but become maladaptive when they persist beyond the point at which tissue damage has healed, ultimately fostering a greater sensitivity to pain (Lethem, Slade, Troup, & Bentley, 1983; Vlaeyen & Linton, 2000; Norton & Asmundson, 2003). Avoidance minimizes the likelihood of pain onset, which negatively reinforces the avoidance behavior and maintains or strengthens fear of pain over time (Vlaeyen & Linton, 2000; 2012). Pain-related disability increases as unwarranted avoidance prevents fear extinction, interferes with physical performance, disrupts attentional processing, and contributes to social withdrawal (Asmundson, Kuperos, & Norton, 1997; Norton & Asmundson, 2003; Vlaeyen & Linton, 2000).

Several studies have since validated the cyclical pattern of the FA model and support the notion that pain-related fear restricts physical performance (Crombez, Vervaet, Lysens, Baeyens,
& Eelen, 1998; Crombez et al., 1999; McCracken, Zayfert, & Gross 1992; McCracken et al.,
1993; Vlaeyen, Kole-Snijders, Boeren, & van Eek, 1995; Vlaeyen & Linton, 2000), leads to
over-predictions of pain (Crombez, Vervaet, Lysens, Eelen, & Baeyens, 1996; McCracken et al.,
1993), and correlates strongly with self-reported disability (Crombez et al., 2012; Linton and
Buer, 1995; Waddell et al., 1993; Vlaeyen & Linton, 2000; 2012). Fear-avoidance constructs
also predict disability in chronic low back pain patients, even after controlling for initial pain
ratings (Fritz, George, & Delitto, 2001).

Further support for the FA model was evidenced in a chronic musculoskeletal pain
sample, wherein Asmundson and Taylor (1996) used structural equation modeling to examine
the role of anxiety sensitivity (AS), a fear of benign anxiety-related bodily sensations, on fear of
pain. The authors found that although pain severity accounted for 13% of the variance in fear of
pain, fear of pain directly influenced escape/avoidance behavior. In addition, Vlaeyen and
colleagues (2001, 2002) found that in vivo graded exposure to potentially threatening situations
effectively reduced pain-related fear and disability in individuals with chronic low back pain
(Vlaeyen, de Jong, Geilen, Heuts, & van Breukelen, 2001; Vlaeyen, de Jong, Geilen, Heuts, &
van Breukelen, 2002), further underscoring the detrimental effects of avoidance in establishing
and maintaining pain-related fear and disability. Theoretically, early detection of fear of pain in
chronic pain patients may help spawn more effective treatment programs that could prevent or
reduce subsequent disability.

Though the FA model was originally developed to describe the pain process among
individuals with chronic musculoskeletal pain, some researchers have attempted to apply this
model to persistent headache disorders (Hursey & Jacks, 1992). Headache sufferers often use
avoidance as a coping strategy for head pain and, consistent with the FA model of chronic pain,
this avoidance is related more strongly to chronicity than to pain intensity (Phillips & Jahanshahi, 1985; 1986). Unlike musculoskeletal pain, however, migraine and TTH are not caused by injury, and thus the clinical recommendations for rest upon injury and then for activity (upon healing) are perhaps not as applicable for headache patients. However, the FA model may be of particular relevance insofar as fear of pain may foster pain-related attentional biases (Lethem et al, 1983; Vlaeyen & Linton, 2000; Norton & Asmundson, 2003), and restriction of activities unrelated to pain may increase disability, reduce social support, and contribute to depression (Crombez et al., 2012; Vlaeyen & Linton, 2000; 2012).

Most recently, Martin and MacLeod (2009) reviewed evidence suggesting that avoidance of headache triggers may actually increase pain sensitivity. Despite common clinical advice to avoid headache triggers, the mechanisms by which triggers precipitate headache have not been established empirically, and for some triggers continued avoidance may actually perpetuate headache (Martin & MacLeod). As Martin and MacLeod (2009) argue, the common advice to avoid triggers is in fact contrary to the principles of the FA model, as avoiding a trigger could decrease tolerance for the trigger and thus strengthen its potential to induce a headache in the future (i.e., avoiding triggers may prevent habituation). Thus, future research is warranted to determine the role that fear and avoidance play in the maintenance of head pain specifically.

Although fear of pain has been studied infrequently among headache patients, existing evidence suggests that recurrent headache sufferers often report high fear of pain and that this fear disrupts cognitive thought processes, interferes with daily activities (Hursey & Jacks, 1992), and maintains pain (Philips & Jahanshahi, 1986). Fear of pain may be influenced by headache diagnosis, as Hursey and Jacks found that ETTH sufferers reported less fear of minor pain but greater fear of severe or medical pain than non-headache sufferers. The authors hypothesized that
the relatively lower fear ratings of minor pain experienced by ETTH sufferers resulted from more frequent exposure to minor pain in the form of their ETTH attacks. In another study among persistent headache sufferers, Asmundson, Norton, and Veloso (1999) examined the role of AS on cognitive, behavioral, and affective components of fear of pain in a sample of 72 treatment-seeking headache patients recruited from an outpatient neurology clinic. The majority of participants were middle-aged females with a long history of migraine headache. Anxiety sensitivity was a significant predictor of fear of pain and alone accounted for 39.8% of the variance in fear of pain. Notably, the authors found that fear of pain uniquely accounted for significant variance in “lifestyle change” (i.e., disability) even after controlling for pain severity. Though these results support the idea that pain-related fear plays a salient role in functioning among headache sufferers, a major limitation is that a single questionnaire item was used to assess disability.

In a subsequent study, Norton and Asmundson (2004) used structural equation modeling to examine relations between fear of pain, AS, and avoidance behavior in treatment-seeking headache patients. Participants consisted of 171 headache patients who were primarily middle-aged women with migraine and had headaches for an average of over 17 years. The authors found that fear of pain loaded directly on headache-related escape and avoidance ($z = 0.90$) and that headache pain severity indirectly influenced escape/avoidance behavior via a direct loading on fear of pain ($z = 0.35$). However, as in their previous study (Asmundson et al., 1999), escape/avoidance was only assessed with one unvalidated questionnaire item.

Until recently, the relations between fear of pain and headache-related disability had not been assessed using a validated measure of disability. In 2006, Nash, Williams, Nicholson, and Trask used the Migraine Disability Assessment Scale (MIDAS; Stewart et al., 2001) to examine
the influence of fear of pain on disability after controlling for pain, emotional distress, self-efficacy, and locus of control among 96 middle-aged primary headache sufferers, 90% of whom had migraine with or without TTH. Fear of pain (specifically physiological anxiety) had a strong and unique association with headache-related disability, accounting for 14% of unique variance after controlling for the aforementioned covariates. In addition to its role in headache-related disability, fear of pain also corresponds with medication use. Fear of pain “predicts” prescription and over-the-counter analgesic use beyond pain severity (Asmundson, Wright, Norton, & Veloso, 2001), such that high fear of pain may serve as a risk factor for overuse of acute headache medications. Thus, understanding of and attention to the role of fear of pain in headache-related variables are essential and may have significant therapeutic implications.

Goals of the Present Study

As described earlier, studies have indicated that fear of pain is a stronger predictor of disability among clinical pain samples than is pain intensity or frequency (McCracken et al., 1993) and plays a significant role in the maintenance of pain (McCracken et al., 1992; Vlaeyen et al., 1995). Consistent with FA models of chronic pain disorders, Hursey and Jacks (1992) found that headache sufferers with greater fear of pain reported more disruptions in activities than individuals with lower fear of pain. The studies conducted by Nash et al. (2006) and Asmundson and colleagues (1999; 2004) highlighted the importance of fear of pain in headache-related disability, but most studies to date have not utilized a validated measure of disability (Asmundson et al., 1999; 2004). Further, no study has compared fear of pain across primary headache diagnostic groups or assessed fear of pain among younger adults without long headache histories potentially confounded by medication overuse. The aims of the present study thus were to assess fear of pain across headache diagnostic groups and quantify relations with
headache variables (e.g., severity, frequency, disability) among a sample of young adult headache sufferers.

Hypotheses

The following goals and hypotheses were proposed:

*Study Goal 1: To examine potential differences in fear of pain between headache sufferers and non-headache controls.*

Hypothesis 1: Headache sufferers would report greater fear of pain than non-headache controls.

*Study Goal 2: To examine differences in fear of pain across five primary headache groups (EM with and without aura, CM, ETTH, CTTH).*

Hypothesis 2a: Migraineurs would report more pain-related fear than individuals with ETTH and CTTH, with individuals with CM endorsing higher fear of pain than those with EM.

Hypothesis 2b: Observed differences in fear of pain would remain even after controlling for relevant covariates (i.e., anxiety, depression, and gender).

*Study Goal 3: To assess the extent to which fear of pain “predicts” headache variables (e.g., severity, frequency, disability).*

Hypothesis 3a: Fear of pain would be positively associated with headache frequency, severity, and disability.
Hypothesis 3b: Fear of pain would be positively associated with headache frequency, severity, and disability after controlling for relevant covariates.

Study Goal 4: To examine whether fear of pain acts as a mediator between pain severity and headache-related disability.

Hypothesis 4: Fear of pain would partially mediate relations between pain severity and headache-related disability.

Figure 1: Proposed mediation model with fear of pain as the mediator.
II. METHODS

Participants

The initial sample consisted of undergraduate students 18 years of age and older who completed a variety of computer-administered measures assessing headache symptoms, headache-related disability, pain severity, and fear of pain as part of a larger battery of measures. Those with an identifiable ICHD-II primary headache disorder (ETTH, CTTH, EM with or without aura, CM) or those who did not suffer from headache were retained for the present study. Young adult headache sufferers were of particular interest due to their high prevalence of headache and resulting functional impairment (Smitherman, McDermott, & Buchanan, 2011), as well as their comparatively lower probability of confounding variables such as medication overuse and headache chronification. Individuals with suspected cluster headache, medication overuse headache (MOH), or posttraumatic headache resulting from head injury were excluded. Other exclusion criteria include suspect effort (i.e., highest 10% of completion time; see Smitherman & Kolivas, 2013) and incomplete batteries (i.e., missing data). Assuming a small effect size ($f^2 = .02$), a power level of 0.80, and an alpha level of 0.05, a total sample size of 245 participants was required.

Materials

*Depression, Anxiety, and Stress Scale (DASS-21).* The DASS-21 is a shortened 21-item version of the original 42-item DASS (Lovibond & Lovibond, 1995), which uses Likert-type scales to assess depression, anxiety, and stress over the past week. Each of the DASS-21
subscales has strong reliability (Cronbach alphas .87 to .94) and convergent validity with other measures of depression and anxiety. In addition, the original DASS was co-normed with non-clinical samples and is thus appropriate for use with our sample (Antony, Bieling, Cox, Enns, & Swinson, 1998). This measure can be found in Appendix B.

*Headache Impact Test-6 (HIT-6).* The HIT-6 (Kosinski et al., 2003) is a 6-item self-report measure on which respondents indicate the impact of headache on emotional and cognitive functioning, as well as daily activities, over the past 4 weeks. Respondents estimate frequency of impairment using a 5-point Likert-type scale ranging from “never” to “always”. The overall impact of headache is indicated by the total scores, which range from 36 to 78 (Mild impairment 50-55; Moderate impairment 56-59; Severe impairment 60+). This instrument provides reliable and valid assessment of headache-related disability among episodic and chronic headache sufferers (Yang, Rendas-Baus, Varon, & Kosinski, 2011). This measure can be found in Appendix C.

*Pain Anxiety Symptoms Scale-20 (PASS-20).* The PASS-20 is a condensed 20-item version of the original 40-item PASS (McCracken, Zayfert, & Gross, 1992), which was developed to assess fear of pain. This 5-point Likert-type scale quantifies fear of pain across four areas: cognitive anxiety, escape and avoidance responses, fearful appraisals, and physiological anxiety responses. Total scores range from 0-100. Item reduction from the original measure yielded a minimal reduction of validity correlations with other measures of pain, depression, and disability, indicating that the PASS-20 has good convergent and construct validity (McCracken & Dhingra, 2002). In addition, the PASS-20 has good internal consistency ($\alpha = 0.81$) and strong convergent ($r = 0.95$) and divergent ($r = 0.57$) validity with the original PASS. This measure can be found in Appendix D.
Structured Diagnostic Interview for Headache-Revised (SDIH-R). The SDIH-R is a revision of the original computer-administered Structured Diagnostic Interview for Headache (Andrew, Penzien, Rains, Knowlton, & McAnulty, 1992) that was adapted to adhere to the diagnostic criteria of the ICHD-II. The 17-item SDIH-R assesses headache diagnostic features and includes additional questions to assess the presence of aura, cluster headache, MOH, and posttraumatic headache. The SDIH has shown strong validity for identifying primary headache disorders (Andrew, Penzien, Rains, Knowlton, & McAnulty, 1992). The SDIH-R was used for the purposes of quantifying headache frequency and severity and for establishing ICHD-II headache diagnoses for participants, with the exception that the minimum required duration for migraine was decreased from 4 to 2 hours given that younger migraineurs often have otherwise prototypical attacks lasting less than 4 hours (Rains, Penzien, Lipchik, & Ramadan, 2001). This measure can be found in Appendix E.

Procedure

Undergraduate students in psychology courses were recruited from an online research announcement program at the University of Mississippi. Participants who chose to participate in the study provided informed consent and completed the aforementioned measures online in exchange for modest course credit.

Statistical Analyses

Descriptive statistics were summarized and distributions examined. Mahalanobis distance was used to determine and exclude multivariate outliers. To examine how the different aspects of fear of pain (cognitive anxiety, escape and avoidance, fearful appraisals, and physiological anxiety) varied between specific headache diagnostic groups and non-headache controls, a
multivariate analysis of variance (MANOVA) was conducted using the four PASS-20 subscale scores as dependent variables and diagnostic status as the independent variable. Post-hoc tests were run as appropriate to identify specific differences between groups. A subsequent MANCOVA assessed whether any observed differences remained after controlling for relevant covariates (which were identified using t-tests [for gender] and Pearson correlations [for depression and anxiety]). Next, among the headache sufferers only, three separate linear regressions were conducted to determine the predictive utility of the PASS-20 total score on headache severity, frequency (day/month), and disability. These analyses were repeated after first entering relevant covariates in an initial block to identify variance uniquely attributable to fear of pain.

Finally, the effects of headache severity on headache disability through fear of pain were assessed, both directly and indirectly, using standard path-analytic approaches described by Preacher and Hayes (2004, 2008), which has a lower Type II error rate and greater statistical power than the Baron and Kenny (1986) method (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002; Preacher & Hayes, 2004). Unlike the former approach (Baron & Kenny, 1986), Preacher and Hayes (2004) utilize a bootstrapping method, which can be applied to small sample sizes and makes no assumptions about the shape of the sampling distribution. Bootstrapping involves randomly sampling observations with replacement from the original data set and computing the statistic of interest in each resample. In mediation, bootstrapping is used to generate the sampling distribution of the indirect effect and this empirical representation is then used to construct a percentile-based confidence interval (95% CI). Consistent with Preacher and Hayes’ (2008) recommendation, 5,000 bootstrapped samples were employed.
III. RESULTS

Participant Demographics and Primary Headache Diagnosis Prevalence

Twenty-two hundred and sixty students ages 18-49 participated in the study. Of these, 288 did not provide sufficient information to determine a headache diagnosis, 109 failed to complete the entire battery, and 307 evidenced suspect effort by taking less than 30 minutes to complete the items in the battery (i.e., ≥ 90th percentile of completion time speed; see Smitherman & Kolivas, 2013). These participants were excluded from analyses, as were those who reported symptoms indicative of probable migraine (n = 294), probable TTH (n = 202), cluster headache (n = 29), MOH (n = 4), and posttraumatic headache resulting from head injury (n=62). Of the 965 remaining participants, 38 were missing subscale scores on at least one of the measures of interest and thus were excluded from the analyses, as is standard in the estimation of path analytic models (Preacher & Hayes, 2004). The remaining 927 participants were screened by group for multivariate outliers using Mahalanobis distance, and 19 outliers (2.04%) were identified using a conservative $p < .001$ chi-square cut-off (Tabachnick & Fidell, 2007). Fifty-three percent of these outliers were non-headache controls (10 participants), and the remaining 47% met criteria for the primary headache disorders of ETTH (6 participants), EM without aura (2 participants), and EM with aura (1 participant). These outliers were deleted prior to analysis, leaving a final sample size of 908.

The demographic characteristics of the retained sample are presented in Table 1. The sample was predominantly female (64.9%) with a mean age of 19.52 years ($SD = 2.79$). The majority (75.7%) were Caucasian, followed by 15.6% African American, 2.4% Hispanic/Latino,
3.7% Asian, 2.2% multiracial, and 0.3% Native American/Pacific Islander. Regarding headache, 382 participants (42.1%) denied headache (i.e., non-headache controls), 237 participants (26.1%) met the diagnostic criteria for ETTH, 232 (25.6%) for episodic migraine (167 [18.4%] without aura and 65 [7.2%] with aura), 38 (4.2%) for chronic migraine, and 19 (2.1%) for CTTH. On average, participants with a primary headache disorder reported experiencing headache on 6.66 days per month ($SD = 5.28$), and mean severity of these headaches was 4.72 out of 10 ($SD = 1.68$). In addition, headache participants reported a mean fear of pain score of 23.33 ($SD = 15.69$) on the PASS-20 and a mean score of 52.51 ($SD = 9.09$) on the HIT-6, with nearly 36% of those with headache endorsing substantial to very severe headache-related disability.

Covariates

As expected, fear of pain had significant moderate associations with both anxiety, $r(899) = .40, p < .001$, and depression, $r(889) = .31, p < .001$. In addition, females reported significantly higher fear of pain on the PASS-20 than males ($M = 21.82 [15.20] \text{ vs } 16.28 [11.85], t(906) = -5.66, p < .001$). As such, anxiety, depression, and gender were used as covariates in the subsequent regression analyses.

Differences in Fear of Pain

Due to unequal group sample sizes and the resulting homogeneity assumption violation of the MANOVA (Box’s $M = 212.46, p < .001$), Pillai’s trace was used to assess the significance of overall group differences, as recommended by Tabachnick and Fidell (2001). The one-way MANOVA across diagnostic groups yielded a significant omnibus effect, Pillai’s $V = .16, F(20, 3608) = 7.53, p < .001$. Given the significance of the overall test, the univariate main effects for headache diagnosis were assessed and are presented in Table 2. Scores on all four PASS-20
subscales differed significantly between groups: Cognitive Anxiety, \( F(5, 902) = 20.33, p < .001 \); Escape and Avoidance, \( F(5, 902) = 16.17, p < .001 \); Fearful Appraisals, \( F(5, 902) = 12.94, p < .001 \); and Physiological Anxiety, \( F(5, 902) = 24.12, p < .001 \). Pertinent to Hypothesis 1, migraineurs reported greater fear of pain than non-headache controls. Those with CM and EM with aura evidenced higher fear of pain on all PASS-20 subscales than those without headache; episodic migraineurs without aura reported higher anxiety than controls on two subscales (Cognitive Anxiety and Escape and Avoidance). Individuals with TTH, whether episodic or chronic, however, did not report higher fear of pain than those without headache. Pertinent to Hypothesis 2, those with CM reported significantly higher fear of pain than EM participants without aura and those with TTH. Compared to those with CM, episodic migraineurs with aura reported similarly high levels of fear of pain, except on the Physiological Anxiety subscale (\( p < .05 \)). Chronic migraineurs differed significantly from tension-type headache sufferers on all four subscales (\( ps < .05 \)). In addition, chronic migraineurs endorsed higher fear of pain than episodic migraineurs without aura on all subscales (\( ps < .05 \)) except Escape/Avoidance (\( p = .052 \)).

A subsequent MANCOVA assessed whether these differences remained after controlling for gender, depression, and anxiety. The MANCOVA yielded a significant omnibus effect, Pillai’s V = .09, \( F(20, 3488) = 4.06, p < .001 \), indicating that overall group differences remained after incorporating these covariates. Univariate tests confirmed that scores on all four PASS-20 subscales differed significantly between groups: Cognitive Anxiety, \( F(5, 872) = 7.80, p < .001 \); Escape and Avoidance, \( F(5, 872) = 8.40, p < .001 \); Fearful Appraisals, \( F(5, 872) = 5.19, p < .001 \); and Physiological Anxiety, \( F(5, 872) = 12.10, p < .001 \). Specifically, migraineurs reported greater fear of pain than non-headache controls on all four PASS-20 subscales (\( ps < .05 \)), and individuals with ETTH differed from controls only on the Escape/Avoidance subscale (\( p < .05 \)).
In general the major group differences from the MANOVA remained, except those with CM and EM without aura no longer differed on Cognitive Anxiety (p = .066), and EM participants with aura scored significantly higher than those without aura on Physiological Anxiety (p < .05).

Predictive Utility of Fear of Pain

Among headache sufferers, separate linear regression analyses confirmed that fear of pain, as measured by the PASS-20 total score, significantly “predicted” headache severity and frequency (headache days/month) from the SDIH-R, accounting for 6.1% and 4.5% of variance in these variables, respectively (ps < .001). In addition, fear of pain significantly predicted headache-related disability, accounting for 17.5% (p < .001) of variance in HIT-6 scores (see Table 3). After controlling for gender, anxiety, and depression, fear of pain remained a significant but smaller unique predictor of headache severity (ΔR² = 2.7%; p < .001), frequency (ΔR² = 1.0%; p < .001), and disability (ΔR² = 8.7%; p < .001).

Mediation Analysis

The effect of headache severity on disability (HIT-6) was assessed directly and indirectly through fear of pain (PASS-20 total score) using the INDIRECT procedure for SPSS (Preacher and Hayes, 2008). Each path is represented in terms of an unstandardized ordinary least squares (OLS) regression coefficient (see Table 4). The total effect (path c in Figure 2) indicated that for every 1-point increase in headache severity, HIT-6 scores increased on average by more than 3 points (3.29, p < .001), indicating that individuals who experience more severe headache pain also report greater headache disability. The direct effect (path c’) of headache severity on headache disability indicated that when controlling for fear of pain, a 1-point increase in headache severity was associated with a 2.90 unit increase in HIT-6 scores (p < .001).
The estimate of the indirect effect \((ab)\) of headache severity on disability through fear of pain was most relevant to the mediation hypothesis. This effect was quantified as the product of two OLS regression coefficients, one estimating fear of pain from headache severity (path \(a\)) and the other estimating headache disability from fear of pain while controlling for severity (path \(b\)). Path \(a\) indicates that a unit increase in headache severity was associated with a 2.21 unit increase in fear of pain \((a = 2.21)\), and path \(b\) shows that a unit increase in fear of pain was associated with a 0.17 unit increase in disability \((b = 0.17)\). As reported in Table 4, the difference between the total and direct effects \((c - c')\) is the indirect effect \((ab)\) through fear of pain, which yielded a point estimate of 0.38 (95% CI = 0.23 - 0.57). As this bias-corrected and accelerated bootstrap CI for the indirect effect did not include zero, the null hypothesis that the total indirect effect equals zero was rejected, and the mediation effect was significant. Although a small amount of the variance in fear of pain was explained by changes in headache severity \((R^2 = 6.1\%)\), a substantial amount of variance in HIT-6 scores was accounted for by both fear of pain and headache severity \((R^2 = 44.5\%)\). These results provide support for the hypothesis that headache severity is partially associated with headache-related disability through its effect on fear of pain.
Figure 2: Path coefficients for simple mediation analysis on headache-related disability

Note: c denotes the total effect, while c’ denotes the effect of headache severity on disability when fear of pain is not included as a mediator. ** p < .001
IV. DISCUSSION

The fear-avoidance model was originally developed to explain the transition from acute to chronic pain, and most research on this model has been conducted with chronic musculoskeletal pain patients. With a scarcity of research in headache samples, the present study sought to examine the role of pain-related fear in primary headache disorders.

Fear of Pain across Diagnostic Groups

As hypothesized, headache sufferers reported greater fear of pain than non-headache controls, consistent with previous cross-sectional designs, indicating greater fear of severe and medical pain (Hursey & Jacks, 1992) and cognitive anxiety (Bishop, Holm, Borowiak, & Wilson, 2001) among headache sufferers than those without headache. However, results from the present study revealed that non-headache controls differed significantly only from migraineurs, not individuals with TTH, on reported levels of pain-related fear. This finding may be a function of the higher pain severity characteristic of migraine versus TTH, as prior studies from the chronic musculoskeletal pain literature have shown positive associations between pain severity and fear of pain (Leeuw et al., 2007; Vlaeyen & Linton, 2000).

In addition to differences in pain severity, migraine and TTH are associated with differing levels of disability and avoidance behavior (Bigal, Bigal, Betti, Bordini, & Speciali, 2001; Cassidy, Tomkins, Hardiman, & O’Keane, 2003; Lenaerts & Newman, 2008; Waldie & Poulton, 2002), both of which are influenced by fear of pain (Crombez et al., 2012; Linton & Buer, 1995; Perry & Francis, 2013; Waddell et al., 1993; Vlaeyen & Linton, 2000; 2012) and
pain chronicity (Phillips & Jahanshahi, 1985; 1986). We thus endeavored to compare fear of pain also across specific headache diagnoses. As hypothesized, migraineurs reported greater fear of pain than tension-type headache sufferers, with chronic migraineurs reporting higher fear of pain than episodic migraineurs on most PASS-20 subscales. These findings are consistent with the FA model of chronic pain, indicating that avoidance is related more strongly to chronicity than to pain intensity (Leeuw et al., 2007; Lethem et al., 1983; Phillips & Jahanshahi, 1985; 1986).

Fear of Pain as a Predictor

In addition to comparing fear of pain across headache groups, the present study highlighted the utility of fear of pain in predicting headache-related variables. Given that headache is among the top 10 most disabling medical conditions worldwide (Stovner et al., 2007), perhaps the most striking finding was the proportion of variance that fear of pain accounted for in headache-related disability (17.5%). Specifically, fear of pain accounted for more variance in disability than gender, anxiety, and depression combined (13.8%). Even after controlling for these covariates, fear of pain remained a significant unique predictor of headache disability, accounting for 8.7% of unique variance. This approaches a medium effect size (Cohen, 1988) and is consistent with findings of Nash et al. (2006), in which fear of pain (specifically physiological anxiety) accounted for 14% of variance in disability after controlling for pain, emotional distress, self-efficacy, and locus of control. Considered in conjunction, these findings suggest that fear of pain is of relevance not only to musculoskeletal pain, but in headache as well, further confirming its role in fear-avoidance models of pain (Leeuw et al., 2007; Vlaeyen & Linton, 2000; 2012).

Most studies on fear of pain have examined the predictive utility of fear of pain on
disability (Asmundson, Norton, & Veloso, 1999; Crombez et al, 1999; McCracken, Faber, & Janeck, 1998; McCracken, Gross, Aikens, & Carnkike, 1996; Nash et al., 2006; Vlaeyen & Linton, 2000) and neglected other pain variables. In the field of headache, to our knowledge fear of pain has not been previously studied as a predictor of headache severity and frequency, despite their established contributions to avoidance and disability (Asmundson, Norton, & Veloso, 1999; Nash et al., 2006). As hypothesized, fear of pain significantly predicted these variables, accounting for 6.1% and 4.5% of variance in severity and frequency, respectively. These findings indicate that increases in fear of pain are associated with more frequent and more painful headaches, which further explains the aforementioned differences in fear of pain across primary headache diagnostic groups. They also parallel those from several chronic pain studies showing similar positive associations between fear of pain measures and measures of severity or disability (Leeuw et al., 2007; Perry & Francis, 2013; Vlaeyen & Linton, 2000). For instance, Asmundson and Taylor (1996) found that pain severity accounted for 13% of the variance in fear of pain. In the present study fear of pain remained a significant (albeit modest) predictor of headache severity and frequency, even after controlling for the aforementioned covariates, but was most strongly associated with disability. Collectively, these findings provide theoretical support for the FA model as relevant to headache, to the extent that heightened fear of pain promotes escape and avoidance behaviors (e.g., avoiding headache triggers or situations putatively associated with headache), which may compound pain-related disability by preventing fear extinction and fostering sensitivity to headache triggers. Indeed, emerging research using experimental designs suggests that avoidance of headache triggers may actually sensitize headache sufferers to these triggers over time (Martin & MacLeod, 2009).
Understanding associations between fear of pain and headache is essential and may have significant therapeutic implications. Theoretically, early detection of fear of pain may reduce the likelihood of headache chronification and ultimately improve functioning, if fear of pain can be targeted therapeutically before becoming severe. In vivo graded exposure to feared stimuli is effective in reducing fear of pain and disability in chronic lower back pain patients (Vlaeyen et al., 2001; 2002), but studies on headache patients are lacking.

Fear of Pain as a Mediator

As hypothesized, headache severity and headache-related disability were positively associated with each other, indicating the presence of an effect to be mediated. The mediation analysis showed that this association was partially driven by fear of pain, consistent with findings from a cross-sectional study of chronic lower back pain patients with an older mean age (range: 25-80 years; Karoly, Okun, Ruehlman, & Pugliese, 2008). Individuals with higher fear of pain were more disabled by headache than those with lower fear of pain scores, even when pain severity was held constant. These results indicate that pain severity influences fear of pain, which in turn influences disability. According to the FA model of pain, these individuals with high fear of pain engage in more avoidance of activities than those with less fear of pain, activities that may or may not be directly contributing to their pain. Thus, targeting fear of pain and escape/avoidance behaviors may result in better treatment outcomes than targeting pain symptoms alone.

Despite the fact that fear of pain was a statistically significant mediator, the clinical significance of the mediation effect was rather modest. While only 6% of the variance in fear of pain was explained by headache severity, similar to the findings of French and colleagues
(2000), 45% of the variance in headache disability was accounted for by both fear of pain and headache severity. Nonetheless, there was a substantial amount of unexplained variance in disability. These modest findings may in part be due to the fact that our sample consisted of non-treatment seeking headache sufferers with uncomplicated headache histories (i.e., without frequent medication use and headache chronification). Treatment-seeking headache sufferers would likely report greater headache impact and fear of pain, which in turn could account for larger proportions of variance in disability. Additionally, other potential mediators or moderators not included in the model may help account for remaining variance. For example, coping strategies influence how well individuals are able to adjust to pain (Asmundson, Norton, & Norton, 1999) and perceived self-efficacy affects pain-coping strategies (French et al., 2000). Low self-efficacy has also been associated with increased disability in chronic low-back pain patients (Costa, Maher, McAuley, Hancock, & Smeets, 2011) as well as in CTTH sufferers (French et al., 2000). In fact, Costa and colleagues (2011) found that pain self-efficacy partially mediated the relationship between pain severity and disability, but fear of movement beliefs did not mediate this relationship. Given the importance of coping and self-efficacy in pain-related functioning, future studies should endeavor to explore the relationship between fear of pain and self-efficacy to determine how these psychological constructs may influence pain-coping strategies.

Central to the FA model is the notion that reductions in fear of pain foster improvements in pain-related disability, and thus fear of pain is often a target of treatment (Leeuw et al., 2007). In the chronic pain literature, several cognitive behavioral therapies have shown to be effective in reducing pain-related fear, thereby decreasing disability and improving overall functioning (Bailey, Carleton, Vlaeyen, & Asmundson, 2010; McCraken, Gross, & Eccleston, 2002; Woby,
Watson, Roach, & Urmston, 2004; Zale, Lange, Fields, & Ditre, 2013). Given the observed role of fear of pain in headache, behavioral interventions targeting this fear may foster functional improvement in this population. However, most headache-specific behavioral therapies aim primarily to reduce headache frequency by recognizing headache stressors, reducing physiological responses, developing coping skills, fostering self-efficacy, and modifying headache-related cognitions (Rains, Penzien, McCrory, & Gray, 2005). An increased focused on headache-related disability as a primary outcome could foster interest in addressing constructs such as fear of pain in treatment, as well as attempts to integrate exposure-based treatments of fear of pain into well-established behavioral headache therapies.

Limitations and Future Directions

Strengths of the current study include a large sample size composed of young primary headache sufferers without complicated headache histories, a strong statistical methodology, utilization of a validated measure of headache-related disability, and adherence to ICHD diagnostic criteria. However, caution is advised when drawing generalizations from the current study, as some limitations exist. First, the study used retrospective reports of headache histories gathered from self-report questionnaires. Though the computer-administered SDIH-R inquired about ICHD-II diagnostic criteria, daily headache diaries and interview-based diagnoses would have strengthened the findings of the present study. Second, results may not generalize to older adults and clinical settings due to the limited diversity of the sample. However, young adult headache sufferers are a desirable population for investigating relations between headache and potential mediator and moderator variables, given their low frequency of medication overuse and headache chronification that characterizes (and complicates) study of treatment-seeking patients. Although the sample consisted of non treatment-seeking headache sufferers, a substantial
number of participants reported being significantly impaired by their headaches as evidenced by interference in everyday responsibilities at home, work, school, and social activities. Nonetheless, future studies should utilize treatment-seeking headache sufferers to confirm the relevance of fear of pain in these individuals. As behavioral interventions designed to decrease escape and avoidance behaviors are effective in reducing fear of pain among chronic pain patients (Vlaeyen et al., 2001; 2002), treatment studies assessing the viability and utility of these approaches in reducing headache-related disability may be warranted if the present findings are confirmed in clinical samples.

Finally, the present study was cross-sectional in nature and thus the observed associations should be interpreted as correlational rather than causal. Although it was assumed that increases in fear of pain lead to increases in headache-related disability, increases in disability could instead lead to increases in fear, or both may be influenced by a third variable. Mediator analyses by definition involve assumptions about temporal ordering of variables, which in the present study were based on the sequential ordering of these variables in the FA model (Vlaeyen & Linton, 2000) and previous research showing that fear of pain predicts pain-related disability (Asmundson et al., 1999; Crombez et al., 1999; Leeuw et al., 2007; McCracken et al., 1992; Nash et al., 2006). Future studies that experimentally manipulate fear of pain are needed also, in order to assess the role of fear of pain in fostering attentional biases and escape/avoidance behaviors. Further research is needed also to examine other potential psychological mediators of the headache-disability relationship, such as locus of control and self-efficacy. The need for these future studies is highlighted by the results of the present study, suggesting that fear of pain plays a significant role in primary headache, particularly headache-related disability.
LIST OF REFERENCES


LIST OF APPENDICES
APPENDIX A: TABLES
Table 1

*Demographic Characteristics of the Sample (n = 908)*

<table>
<thead>
<tr>
<th>Variable</th>
<th>% or Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (% Female)</td>
<td>64.9</td>
</tr>
<tr>
<td>Mean Age (SD)</td>
<td>19.52 (2.79)</td>
</tr>
<tr>
<td>Race (% Caucasian)</td>
<td>75.7</td>
</tr>
<tr>
<td>Marital Status (% Never Married)</td>
<td>97.8</td>
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<tr>
<td>Education (% Some college)</td>
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</tr>
<tr>
<td>Employment (% Unemployed)</td>
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</tr>
<tr>
<td>Income (% &gt;$50,000)</td>
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</tr>
<tr>
<td>Greek (% Greek)</td>
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</tr>
<tr>
<td>Religion (% Protestant/Evangelical)</td>
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<tr>
<td>Mean Headache Severity (SD)</td>
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</tr>
<tr>
<td>Mean HIT-6 Score (SD)</td>
<td>52.51 (9.09)</td>
</tr>
<tr>
<td>Mean PASS-20 Score (SD)</td>
<td>23.33 (15.69)</td>
</tr>
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</table>
Table 2. MANOVA Results Examining Group Differences in Fear of Pain (PASS-20) Subscales

<table>
<thead>
<tr>
<th></th>
<th>No Headache</th>
<th>CM</th>
<th>EM with aura</th>
<th>EM without aura</th>
<th>CTTH</th>
<th>ETTH</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
</tr>
<tr>
<td><strong>n = 382</strong></td>
<td>n = 38</td>
<td>n = 65</td>
<td>n = 167</td>
<td>n = 19</td>
<td>n = 237</td>
<td></td>
</tr>
<tr>
<td>Cognitive Anxiety</td>
<td>5.94 (4.71)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>12.21 (5.71)&lt;sup&gt;d&lt;/sup&gt;</td>
<td>9.55 (6.30)&lt;sup&gt;cd&lt;/sup&gt;</td>
<td>9.07 (5.40)&lt;sup&gt;bc&lt;/sup&gt;</td>
<td>7.68 (5.23)&lt;sup&gt;abc&lt;/sup&gt;</td>
<td>6.85 (4.57)&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>Escape and Avoidance</td>
<td>5.68 (4.36)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>10.21 (5.05)&lt;sup&gt;c&lt;/sup&gt;</td>
<td>8.91 (5.18)&lt;sup&gt;bc&lt;/sup&gt;</td>
<td>8.01 (4.41)&lt;sup&gt;bc&lt;/sup&gt;</td>
<td>5.32 (3.60)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>6.59 (3.82)&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>Fearful Appraisals</td>
<td>2.42 (3.06)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>6.39 (5.24)&lt;sup&gt;c&lt;/sup&gt;</td>
<td>4.69 (4.46)&lt;sup&gt;bc&lt;/sup&gt;</td>
<td>3.69 (4.01)&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>3.00 (4.43)&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>2.87 (3.33)&lt;sup&gt;ab&lt;/sup&gt;</td>
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<tr>
<td>Physiological Anxiety</td>
<td>1.88 (2.77)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>6.87 (5.34)&lt;sup&gt;c&lt;/sup&gt;</td>
<td>4.71 (4.78)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>3.35 (3.42)&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>2.37 (2.99)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.36 (2.93)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

*Note:* groups with different superscripts differed significantly (p < .05) on the subscale of interest; CM = chronic migraine; EM = episodic migraine; CTTH = chronic tension-type headache; ETTH = episodic tension-type headache.
Table 3. *Fear of Pain as a Predictor of Headache Variables (n = 526)*

<table>
<thead>
<tr>
<th>Predictor</th>
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<th>$SE$</th>
<th>$t$</th>
<th>$P$ value</th>
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<td>Severity</td>
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<td>.028</td>
<td>.005</td>
<td>5.822</td>
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<tr>
<td>Frequency (days/month)</td>
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<td>.075</td>
<td>.015</td>
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<td>Disability (HIT-6)</td>
<td>.175 (.174)</td>
<td>.253</td>
<td>.024</td>
<td>10.561</td>
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Table 4.

*Regression Results for Mediation Effect*

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<th>t</th>
<th>p</th>
<th>95% CI</th>
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<td>8.58–16.05</td>
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<td></td>
<td>$I_Y$</td>
<td>34.78</td>
<td>.92</td>
<td>37.86</td>
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<td>32.98–36.59</td>
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<tr>
<td>Severity</td>
<td>$a$</td>
<td>2.21</td>
<td>.38</td>
<td>5.82</td>
<td>&lt;.001</td>
<td>1.46–2.95</td>
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<tr>
<td></td>
<td>$R^2 = .06$</td>
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<td>-</td>
<td>&lt;.001</td>
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<td>HIT-6 on:</td>
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<tr>
<td>FOP</td>
<td>$b$</td>
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<td>.02</td>
<td>8.53</td>
<td>&lt;.001</td>
<td>.13–.21</td>
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<td>Severity</td>
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<td>.18</td>
<td>15.94</td>
<td>&lt;.001</td>
<td>2.54–3.26</td>
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<td>Indirect</td>
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<td>via:</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FOP</td>
<td>$ab$</td>
<td>.38</td>
<td>.09</td>
<td>-</td>
<td>-</td>
<td>.23–.57</td>
</tr>
<tr>
<td>Total</td>
<td>$c$</td>
<td>3.29</td>
<td>.19</td>
<td>17.46</td>
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<td>-</td>
<td>-</td>
<td>&lt;.001</td>
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</tbody>
</table>

*Note.* CI = confidence interval; LL = lower limit; UL = upper limit; FOP = fear of pain.
### DASS 21

Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you over the past week. There are no right or wrong answers. Do not spend too much time on any statement.

**The rating scale is as follows:**

- 0 Did not apply to me at all
- 1 Applied to me to some degree, or some of the time
- 2 Applied to me to a considerable degree, or a good part of the time
- 3 Applied to me very much, or most of the time

<table>
<thead>
<tr>
<th>Statement</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
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</thead>
<tbody>
<tr>
<td>1 I found it hard to wind down</td>
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<tr>
<td>2 I was aware of dryness of my mouth</td>
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<tr>
<td>3 I couldn't seem to experience any positive feeling at all</td>
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<td>4 I experienced breathing difficulty (e.g., excessively rapid breathing, breathlessness in the absence of physical exertion)</td>
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<td>5 I found it difficult to work up the initiative to do things</td>
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<td>6 I tended to over-react to situations</td>
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<td>7 I experienced trembling (e.g., in the hands)</td>
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<tr>
<td>8 I felt that I was using a lot of nervous energy</td>
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<td>9 I was worried about situations in which I might panic and make a fool of myself</td>
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<td>10 I felt that I had nothing to look forward to</td>
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<tr>
<td>11 I found myself getting agitated</td>
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<tr>
<td>12 I found it difficult to relax</td>
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<td>13 I felt down-hearted and blue</td>
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<tr>
<td>14 I was intolerant of anything that kept me from getting on with what I was doing</td>
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<tr>
<td>15 I felt I was close to panic</td>
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<tr>
<td>16 I was unable to become enthusiastic about anything</td>
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<tr>
<td>17 I felt I wasn't worth much as a person</td>
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<tr>
<td>18 I felt that I was rather touchy</td>
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<tr>
<td>19 I was aware of the action of my heart in the absence of physical exertion (e.g., sense of heart rate increase, heart missing a beat)</td>
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<td>20 I felt scared without any good reason</td>
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<tr>
<td>21 I felt that life was meaningless</td>
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</table>
APPENDIX C
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.

1. When you have headaches, how often is the pain severe?
   - Never
   - Rarely
   - Sometimes
   - Very Often
   - Always

2. 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

3. When you have a headache, how often do you wish you could lie down?
   - Never
   - Rarely
   - Sometimes
   - Very Often
   - Always

4. 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

5. In the past 4 weeks, how often have you felt fed up or irritated because of your headaches?
   - Never
   - Rarely
   - Sometimes
   - Very Often
   - Always

6. 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

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.
PASS-20

Please read each item carefully, and then rate how often each statement applies to your life using the following scale:

<table>
<thead>
<tr>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never</td>
<td>Always</td>
<td></td>
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</tbody>
</table>

1) I can’t think straight when in pain. _____
2) During painful episodes it is difficult for me to think of anything besides the pain _____
3) When I hurt I think about pain constantly _____
4) I find it hard to concentrate when I hurt _____
5) I worry when I am in pain _____
6) I go immediately to bed when I feel severe pain _____
7) I will stop any activity as soon as I sense pain coming on _____
8) As soon as pain comes on I take medication to reduce it _____
9) I avoid important activities when I hurt _____
10) I try to avoid activities that cause pain _____
11) I think that if my pain gets too severe, it will never decrease _____
12) When I feel pain I am afraid that something terrible will happen _____
13) When I feel pain I think that I might be seriously ill _____
14) Pain sensations are terrifying _____
15) When pain comes on strong I think that I might become paralyzed or more disabled _____
16) I begin trembling when engaged in an activity that increases pain _____
17) Pain seems to cause my heart to pound or race _____
18) When I sense pain I feel dizzy or faint _____
19) Pain makes me nauseous _____
20) I find it difficult to calm my body down after periods of pain _____
Structured Diagnostic Interview for Headache – Revised (Brief Version)

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

The following items are selected from the long version of the Structured Diagnostic Interview for Headache (SDIH). The SDIH is 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, benign headaches according to both IHS (2004) and Ad Hoc Committee (1962) diagnostic criteria. Optimal use of this interview requires expertise with the diagnostic classifications and familiarity with the computer software and manual that accompany the interview.

1. Does the patient get more than one type of headache? □ Yes □ No

(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)

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

<table>
<thead>
<tr>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>Slightly</td>
<td>Mildly</td>
<td>Painful</td>
<td>Very</td>
<td>Extremely</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain</td>
<td>Painful</td>
<td>Painful</td>
<td>Painful</td>
<td>Painful</td>
<td>Painful</td>
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</tbody>
</table>

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? ____ d w m y

(Indicate frequency in x per day, week, month, or year)

7. How long have these headaches been occurring at this rate? ____ months 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)

- [ ] Unremitting OR

____ m h d Typical Average  
____ m h d Typical Minimum  
____ m h d Typical Maximum
10. Has anything about this headache (except freq.) changed in the last 6 months? □ Yes □ No

   If YES, explain: ________________________________

11. Is the patient’s typical headache pain aggravated by routine physical activities (i.e., walking, lifting, bending, etc.)?

   □ Yes □ No

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

   □ Loss of appetite/Anorexia
   □ 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 of the reported symptoms provide evidence of focal cerebral cortical, and/or brainstem dysfunction, complete Appendix 1

   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 Appendix 2

   If NO, skip to #15

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

   If YES, complete #15a, #15b, #15c

   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

15c. Did this headache develop or markedly worsen during medication overuse?  □ Yes  □ No

If **YES**, complete **Appendix 3**

If **NO**, skip to #16

16. Is this headache related to any head injury or trauma?  □ Yes  □ No

If **YES**, complete **Appendix 4**

If **NO**, skip to #17

17. Is this headache suspected to be attributed to a physical or other neurological disorder?  □ Yes  □ No
VITA

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University of Alabama: ADHD and Anxiety Program, Tuscaloosa, AL (2011-2012)
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University of Alabama: Teaching Assistant, Freshman Honors Psychology Class (2011)

PUBLICATIONS:


MANUSCRIPTS UNDER REVIEW:


POSTER PRESENTATIONS:


AD HOC REVIEWING:

Behaviour Research and Therapy