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Psychological Factors In HIV-Related Headaches

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PSYCHOLOGICAL FACTORS IN HIV-RELATED HEADACHES

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ABSTRACT

Headache is one of the most common medical complaints reported by individuals suffering from HIV/AIDS, but conflicting data exist regarding their prevalence, prototypical characteristics, and relationship to HIV severity. A well-established field of research indicates a strong association between psychiatric comorbidities/psychological factors and headache disorders, but this association has not been explored frequently among HIV patients with headaches. Data on headache symptoms and psychological factors were collected on 200 HIV/AIDS patients from two outpatient clinics using structured interviews and self-report measures. Of these, 107 patients (53.5%) endorsed problematic headaches, most of which (n = 103; 51.5%) were consistent with characteristics of primary headache disorders. Among those who met criteria for primary headaches, 88 (85.44%) met criteria for migraine, while 15 (14.56%) met criteria for tension-type headache. Severity of HIV (as indicated by CD4 cell counts), but not duration of HIV, was strongly predictive of headache severity, frequency, and disability. Those with headache endorsed higher levels of comorbid depression, anxiety, and stress, as well as higher levels of pain catastrophizing, anxiety sensitivity, and fear of pain than did those without headache. These group differences were not attributable to differences in HIV duration, number of prescribed antiretroviral medications, or demographic differences such as age, gender, or race. The results indicate the presence of two distinct groups of individuals: one that is relatively healthy, both physically and emotionally without the presence of headaches or psychological dysfunction, and

one that is relatively unhealthy with frequent disabling headaches and comorbid psychological dysfunction. Implications for treatment and future research are discussed.

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I. INTRODUCTION

Headaches are the number one cause of medical complaints in the United States, accounting for approximately 10 million physician visits each year (The US Headache Consortium, 2000). Human Immunodeficiency Virus (HIV), the precursor to AIDS, has been consistently linked to frequent but varying headache patterns (Evers et al., 2000; Mirsattari, Power, & Nath, 1998). High rates of psychiatric comorbidity exist among both headache and HIV sufferers. As a consequence, the stress and emotional sequelae of headaches is highly problematic among the HIV population. Further research into psychiatric comorbidities and headache-related psychological factors could impact treatment planning and recommendations, in turn improving prognosis for HIV patients with headaches. The following text provides a detailed overview of the classification of headache disorders, and reviews the psychological factors associated with headaches (e.g., psychiatric comorbidities, cognitive factors such as anxiety sensitivity, fear of pain, and self-efficacy). The literature on HIV and HIV-related headaches, as well as that on psychological factors associated with HIV, is then reviewed. The goal of the current study was to determine the nature of headache patterns among HIV patients and the relationship of various psychological factors in their presentation.

Headache Types and Psychiatric Comorbidities

Classification of Headaches

The Headache Classification Subcommittee of the International Headache Society (IHS, 2004) classifies headaches into those that are "primary" and those that are secondary to another

disorder. Primary headache disorders include the most common headache disorders such as migraines, tension-type headaches, cluster headaches, and other headaches not attributable to a secondary cause. Secondary headaches include those that result from other disorders or conditions known to cause headaches, such as those attributable to trauma, to vascular disorder, to substance use or withdrawal, to infection, or to a psychiatric disorder (IHS, 2004). Primary headaches are by far (90%) the most common category of headaches experienced by individuals who present to physicians for treatment (Saper, 1999). Although these categories of headaches are widely accepted across multiple disciplines, much controversy has surrounded their nature and classification (Holroyd, 2002).

Migraine headaches affect nearly 12% of the United States population (Lipton, Hamelsky, & Stewart, 2001; Stewart, Lipton, Celentano, & Reed, 1992). Migraines typically last from 4 to 72 hours and are characterized as unilateral, pulsating headaches that are of moderate to severe intensity (IHS, 2004). They typically are aggravated by or cause avoidance of routine physical activities and are usually associated with nausea and/or vomiting, photophobia (sensitivity to light), and phonophobia (sensitivity to sound; IHS, 2004). Within the migraine category are two main types: migraine with aura and migraine without aura (IHS, 2004). "Aura" refers to the premonitory symptoms that may precede a migraine (i.e. typically visual or sensory disturbances such as flickering lights, spots, and tingling sensations), serving as warning signs that a migraine is soon to develop (IHS, 2004).

In the United States, migraines are three times more common in females than in males (18% vs. 6%; Lawrence, 2004; Silberstein, Lipton, & Dodick, 2008). Unfortunately, although most migraineurs suffer from episodic attacks, some experience a more chronic form, characterized by 15 or more migraines per month, for at least 3 months (IHS, 2004). Despite its

episodic manifestations, recent research findings have contributed to a conceptualization of migraine as a chronic disorder associated with particular predisposing factors, abnormal cortical processing and trigeminovascular functioning, and an overall diminished quality of life (Ambrosini, Rossi, De Pasqua, Pierelli, & Schoenen, 2003; Gantenbein & Sandor, 2006; Granziera, DaSilva, Snyder, Tuch, & Hadjikhani, 2006).

Overall quality of life and daily functioning are negatively impacted by migraines (Lipton & Bigal, 2005). Migraines are the direct cause of billions of dollars lost per year due to decreased work productivity, reduced work attendance, and related medical treatments (Lawrence, 2004). Frequency and intensity of attacks, combined with frequent psychiatric comorbidities such as depressive and anxiety disorders (Radat & Swendson, 2005), are also responsible for reduced quality of life seen in migraineurs.

Tension-type headaches (TTH), the most common primary headaches (Rasmussen, Jensen, Schroll, & Oleson, 1991b; Schwartz, Stewart, Simon, & Lipton, 1998), cause mild to moderate pain as compared to migraines (IHS, 2004). TTHs are frequently described as the feeling of a tight band or belt around the head (Friedman, 1979). These headaches generally present bilaterally with a pressing, nonpulsating quality. Contrary to migraines, aura symptoms are absent and pain is not aggravated by routine physical activity (Rasmussen, Jensen, Schroll, & Oleson, 1991a). There are three sub-types of TTH: infrequent episodic, occurring less than 1 time per month; frequent episodic, occurring from 1 to 14 times per month; and chronic, occurring 15 times or more per month (IHS, 2004). For chronic sufferers in particular, repeated TTHs sometimes intensify pain patterns, in which attacks may progress from moderate to more severe (Rasmussen et al., 1991a; Ulrich, Russell, Jensen, & Oleson, 1996; Scher, Lipton, & Stewart, 2003).

As is the case with migraines, TTHs are also the cause of reduced quality of life and disability. Particularly in chronic TTH sufferers, prevalence is highest during an individual's most active years. Such high frequency can be the cause of much burden, impacting life personally, professionally, and socially (Lenaerts, 2006). To put the disabling effect into perspective, TTH is the cause of 820 lost workdays yearly per 1000 employees (Rasmussen, Jensen, Schroll, & Oleson, 1992).

Cluster headaches (CH) are rare in comparison to migraine and TTH. CHs are characterized by severe unilateral pain, typically centered around and involving edema of the orbital socket, which usually peaks in the first 5 minutes and lasts, on average, for one hour (IHS, 2004). This type of headache causes so much distress that those suffering end up substantially exhausted after it has subsided (Dodick, Rozen, Goadsby, & Silberstein, 2000; Silberstein, Lipton, & Dodick, 2008). Due to the rarity of cluster headaches, the focus of the present study is primarily on headache patterns resembling migraine and TTH.

Headaches and Psychiatric Comorbidity

Not only are depression and anxiety potential causes of headaches (IHS, 1988, 2004; Breslau et al., 2000; Merikangas, Angst, & Isler, 1990), but long-term suffering with headache may place individuals at risk for subsequent psychological problems. Indeed, several studies confirm that individuals experiencing headaches are at a much higher likelihood of suffering from psychological problems than those without headaches. Saunders, Merikangas, Low, Von Korff, and Kessler (2008), in a study conducted using structured interviews for mood, anxiety, and substance use disorders on 5,692 members of the United States population, found that 83% of migraineurs and 79% of other headache sufferers reported some type of psychiatric comorbidity within the past 12 months. Jette, Patten, Williams, Werner, and Wiebe (2008) used

diagnostic interviews on 36,984 individuals to determine the prevalence of various psychiatric conditions in the presence of migraines. They found that major depressive disorder, bipolar disorder, panic disorder, and social phobia were two times more likely in individuals with migraines than those without. Given the high frequency of migraine and other headaches in the general population, the prevalence of psychiatric comorbidities among headache sufferers is quite alarming and worthy of continued study.

Headaches and Depression

The relationship between depression and headaches has been examined most frequently. Hamelsky and Lipton (2006) found that migraine sufferers are 2.2 to 4.0 times more likely to experience depression than the general population. Other studies have found that migraineurs are 4 to 5 times more likely to experience depression, dysthymia, and bipolar disorder than those without headaches (Breslau, 1998; Lake, Rains, Penzien, & Lipchik, 2005). Saunders et al. (2008) found that 18% of migraineurs and 13% of other headache sufferers reported symptoms of major depression, compared to only 5.5% of non-headache sufferers. Most studies have found that the relationship between migraines and depression is bidirectional, with each disorder serving as a risk factor for the other (Breslau, Davis, Schultz, & Peterson, 1994; Breslau et al., 2000; Breslau, Lipton, Stewart, Schultz, & Welch, 2003).

Headaches and Anxiety

Although the majority of research on psychiatric comorbidity and headache focused initially on depression, emerging research has confirmed that anxiety disorders are also uniquely prevalent among headache sufferers. Migraineurs are approximately five times more likely to suffer from an anxiety disorder than is the general population (Hamelsky & Lipton, 2006). Specifically, migraineurs are 3 to 10 times more likely to experience panic attacks, 4 to 5 times

more likely to receive a diagnosis of generalized anxiety disorder (GAD), and 5 times more likely to be diagnosed with obsessive-compulsive disorder (Breslau, 1998; Lake et al., 2005; Breslau & Davis, 1993; Breslau, Schultz, Stewart, Lipton, & Welch, 2001; Maizels, Smitherman, & Penzien, 2006). Saunders et al. (2008) found at least one type of anxiety disorder in 44.5% of migraineurs and 30.9% of other headache sufferers, compared to only 15.5% of non-headache sufferers. Just as a bidirectional relationship has been observed between depression and migraine, a similar relationship is posited for headache sufferers with anxiety (Breslau et al., 2001; Wang, Juang, Fuh, & Lu, 2007; Smitherman, Maizels, & Penzien, 2008; Baskin & Smitherman, 2009).

Headaches and Substance Use Disorders

In addition to elevated rates of depression and anxiety disorders, migraineurs have also been found to be two times more likely to suffer from drug, alcohol, and nicotine use disorders (Breslau, 1998; Lake et al., 2005). However, conflicting results have been obtained across studies. Jette et al. (2008) found that the incidence of drug and alcohol dependence in the migraine population (3.1% and 2.3%, respectively) was statistically similar to that of the general population (3.0% and 2.6%, respectively). Saunders et al. (2008) reported similar rates, with 4.2% of migraineurs experiencing alcohol abuse/dependence and 2.3% experiencing drug abuse/dependence compared to 3.0% and 1.2% (respectively) of the non-headache population. Future research should help clarify which headache patients are at highest risk for substance use disorders. Table 1 summarizes the prevalence rates of various Axis I disorders and migraine, as reported in several large epidemiological studies.

Table 1

Associations Between Migraine Headache and Axis I Psychiatric Disorders (in Odds Ratios [OR] adjusted for sex), as Reported in Five Major Epidemiological Studies

Psychiatric Diagnosis	Breslau (1998), without aura	Breslau (1998), with aura	Breslau & Davis (1992)	Saunders et al. (2008)^	Swartz et al. (2000) *	Zwart et al. (2003)†	Zwart et al. (2003)†, chronic migraine
Major Depressive Disorder (MDD)	2.2	4.0	4.2	2.8	2.25	2.7	6.4
Bipolar I	2.4	7.3	--	3.9[a]	--	--	--
Bipolar II	2.5	5.2	--	3.9[a]	--	--	--
Panic Disorder	3.0	10.4	6.0	3.6	3.40	--	--
Generalized Anxiety Disorder	5.5	4.1	5.1	2.5	--	--	--
Obsessive-Compulsive Disorder (OCD)	4.8	5.0	4.8	--	1.32	--	--
Phobia	1.8	2.9	2.2	2.6	1.43	--	--
Any Anxiety Disorder	2.3	3.1	2.8	3.1	--	3.2	6.9
Alcohol Abuse/Depend.	1.6	2.1	--	1.4	1.05	--	--
Illicit Drug Abuse/Depend.	1.6	3.9	--	2.1	1.05	--	--

^Odds ratios adjusted also for age, ethnicity, employment, and education; *Odds ratios adjusted also for age; †Odds ratios adjusted also for age and education
[a]Odds ratios for Bipolar I or II was 3.9 in this study (not assessed separately)

Negative Impact of Comorbid Psychiatric Disorders

Though many comorbidity studies have focused upon one specific comorbid disorder, depression and anxiety disorders often occur simultaneously in headache sufferers. Lanteri-Minet, Radat, Chautart, and Lucas (2005) found that 19% of migraineurs experienced both anxiety and depression. Other studies have confirmed this shared comorbidity, finding that those with headaches are 2 to 5 times more likely to experience depression and anxiety than are those without headaches (Breslau, 1998; Lake et al., 2005; Hamelsky & Lipton, 2006; Guidetti et al., 1998; Radat & Swendsen, 2005; Smitherman, Maizels, & Penzien, 2008).

Although a majority of the psychiatric comorbidity research has focused upon migraineurs, those with other forms of headache, such as chronic daily headache or medication overuse headache, are at heightened risk as well (Baskin, Lipchik, & Smitherman, 2006). In fact, some have suggested that the risk for psychological problems is higher for those suffering from chronic daily headache or medication overuse headache than for migraineurs (Juang, Wang, Fuh, Lu, & Su, 2000; Verri et al., 1998; Lipchik, Smitherman, Penzien, & Holroyd, 2006), raising the possibility that comorbid psychiatric disorders may serve as one mechanism through which headache becomes chronic. Notably, elevated rates of psychiatric comorbidities occur also among TTH sufferers (Holroyd et al., 2000), who are more vulnerable to attacks when psychological factors such as depression and anxiety are present (Haythornthwaite, 1993).

In addition to high prevalence rates of psychiatric disorders, the presence of psychiatric comorbidity is associated with multiple negative outcomes, foremost among them a poorer treatment prognosis. Psychological problems negatively impact headache treatment adherence and effectiveness, worsening overall prognosis for headache reduction and alleviation (Lake et al., 2005; Siniatchkin, Riabus, & Hasenbring, 1999). Guidetti et al. (1998), in a longitudinal study of 100 participants, found that of headache patients with two or more comorbid psychiatric

disorders in childhood or adolescence, 86% had either no improvement or worsening of headache symptoms at 8-year follow-up. By comparison, patients with no diagnosed psychiatric disorders experienced a remission of headaches after 8 years. The negative prognosis associated with comorbid psychiatric symptoms may in part be a function of patient adherence to their treatment regimens (Rains, Lipchik, & Penzien, 2006), such that patients with comorbid disorders are less likely to take their headache medication as prescribed. Thus, although the study sample sizes have not been very large, existing research evidence suggests that overall prognosis worsens in the combination of headaches and psychiatric disorders.

In addition to poorer prognosis of headache, psychiatric comorbidities are associated also with lower satisfaction with headache treatment, increased headache-related disability, and overall reduced quality of life (Guidetti et al., 1998; Lanteri-Minet et al., 2005; Saunders et al., 2008). Saunders et al. (2008) found that, during the last 30 days, migraineurs experienced role disability 25.2% of the time, compared with other headache sufferers and non-headache participants (17.6% and 9.7% of the time, respectively). Importantly, both migraine and non-migraine headache sufferers with a comorbid psychiatric disorder experienced significantly more disability than their headache counterparts without a comorbid disorder. To put the added impact into a monetary perspective, migraineurs with both anxiety and depressive disorders spend $4,000 to $5,500 more on medical treatment each year than do migraineurs without a psychiatric comorbidity (Pesa & Lage, 2004).

Headaches and Other Psychological Factors

Headaches and Stress

In addition to psychiatric comorbidities, other psychological factors have also been linked to the experience and exacerbation of headache. Among these are stress and negative cognitions

such as catastrophizing, anxiety sensitivity, and fear of pain (Holroyd et al., 1984; Holroyd et al., 2000; Hudzynski & Levenson, 1985; Nicholson, Houle, Rhudy, & Norton, 2007; Nicholson, Nash, & Andrasik, 2005; Scharff, Turk, & Marcus, 1995; Maizels, Smitherman, & Penzien, 2006; Houle & Nash, 2008). Assessment of these variables can be important in evaluating overall headache prognosis.

Stress is an important determinant of an array of psychological and medical problems. Previous research has indicated that the frequency of stress in one's life is directly related to both onset and exacerbation of headache (Holm, Holroyd, Hursey, & Penzien, 1986; Levor, Cohen, Naliboff, McArthur, & Heuser, 1986; Houle & Nash, 2008). Stress is the most commonly reported trigger of headaches (Rasmussen, 1993; Penzien, Rains, & Holroyd, 1993), and both migraineurs (Levor et al., 1986) and TTH sufferers (Holm et al., 1986) report a greater than usual frequency of stressful events prior to headache attacks. Stress has also been linked to headache chronification, or the progression of headache from an episodic to chronic form (Penzien, Rains, & Lipton, 2008; Bigal & Lipton, 2008; Scher, Midgette, & Lipton, 2008).

Presently, the biological mechanisms behind the relationship between headache and stress remain largely unclear. Researchers speculate that stress impacts headache patterns by creating a higher sensitivity to environmental stimuli and a more arousable central nervous system (Drummond & Passchier, 2006). Waldie and Poulton (2002) speculate similar mechanisms linking headache and stress, describing an inheritance of heightened stress sensitivity in migraineurs to higher cortical arousal between headache attacks. Stress and emotional factors are speculated to impact headaches on both peripheral and central levels (Drummond & Passchier, 2006; Oleson & Goadsby, 2006). Peripherally, stress may be the cause of perivascular inflammation and pericranial muscle tenderness (Drummond & Passchier, 2006).

Centrally, stress may impact supraspinal control of neurons, increasing the excitability of trigeminal/spinal levels and decreasing effectiveness of the antinociceptive system (Oleson & Goadsby, 2006). Despite uncertainty surrounding specific biological mechanisms, it is clear that exposure to stress is associated with headache attacks, with increased stress leading to increased headache frequency (Houle & Nash, 2008).

Cognitive Factors

Catastrophizing

As stress and pain interact, increased maladaptive reactions are expected. In chronic pain disorders, maladaptive cognitive responses often involve catastrophizing (Burns, Kubilus, Bruehl, Harden, & Lofland, 2003; Holroyd, Drew, Cottrell, Romanek, & Heh, 2007; Severeijns, Vlaeyen, van den Hout, & Weber, 2001; Sullivan et al., 2001). Catastrophizing involves distortion and overestimation of pain responses as well as underestimation of one's own ability to cope (Severeijns et al., 2001; Smitherman, Nicholson, Schafer, & Houle, in press; Sullivan et al., 2001). Excessive rumination, pessimistic brooding, magnification, and spiraling feelings of helplessness characterize this negative cognitive style. Tsui, Thorn, Rubin, and Alexander (2007), in a study on 15 chronic pain patients, found that catastrophizing was positively associated with both self-reported pain (r=.91) and observed pain (r=.79). Behaviors related to catastrophizing impact accurate understanding of pain expression, which may affect both health care providers' assessment and interpersonal social interactions regarding one's pain (Tsui et al., 2007). Although the topography and parameters of catastrophizing have yet to be studied much in the headache population, relationships between catastrophizing and pain in other chronic pain conditions (Burns et al., 2003; McCracken & Dhingra, 2002) underscore this topic as one worthy of exploration among headache patients.

Anxiety Sensitivity and Fear of Pain

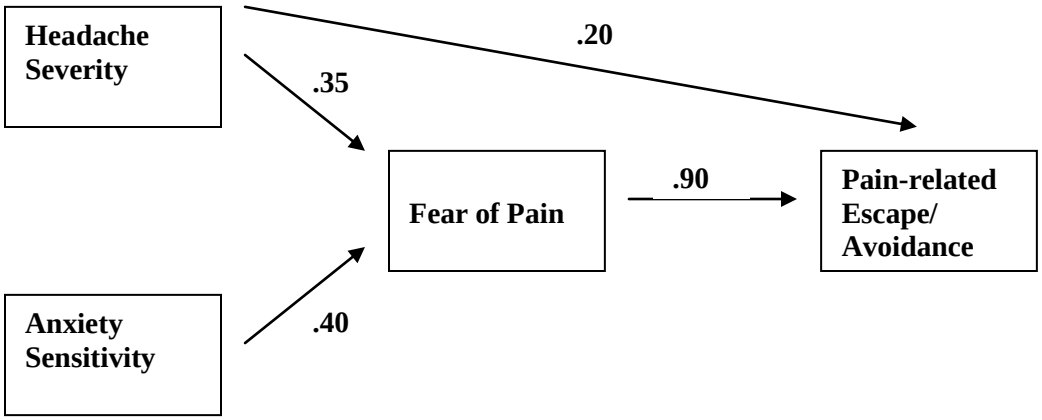
Other maladaptive cognitive responses to pain and pain-related stress have been studied more extensively in headache sufferers, among them the related concepts of anxiety sensitivity and fear of pain. Considered together, these two constructs underscore the notion that individuals suffering from chronic pain conditions are often hypervigilant toward and misinterpret particular bodily sensations. Anxiety sensitivity is a dispositional variable describing the fear of anxiety-related symptoms due to beliefs about their presumed harmful consequences (Asmundson, Norton, & Norton, 1999; Reiss, 1991; Reiss & McNally, 1985). A person high on anxiety sensitivity, for instance, is overly attentive to somatic bodily sensations and often misinterprets such sensations as indicative of a more serious medical problem. Anxiety sensitivity engenders fear associated with anxiety, chronic pain, and other medical problems (Asmundson, Norton, & Norton, 1999).

Anxiety sensitivity is thought to influence pain in two ways: 1) directly by strengthening fear-of-pain beliefs and 2) indirectly by contributing to pain-related escape and avoidance behaviors. Empirical studies have provided support for this model as it applies to migraineurs. Not only are high levels of anxiety sensitivity associated with increased depression and anxiety (Asmundson, Norton, & Norton, 1999), but anxiety sensitivity has been found to be the strongest psychological predictor of fear of pain, alone accounting for 39.8% of the variance in scores on measures assessing fear of pain among a sample of 72 adults with recurrent headaches (Asmundson, Norton, & Veloso, 1999). Anxiety sensitivity was a stronger predictor of pain-related fear than was depression, state and trait anxiety, and headache severity scores.

Fearing (McCracken, Zayfert, & Gross, 1992) and avoiding (Fordyce, 1976) pain have been associated with maintaining pain over time. Empirical studies have confirmed that fear of

pain is a stronger predictor of functional disability than is pain severity itself (McCracken et al.), suggesting that manifestations of fear of pain may be conceptualized as a phobic response that maintains pain and contributing behaviors. Fear of pain is closely linked to anxiety sensitivity. Norton and Asmundson (2004), in a structural-equation modeling study of 156 patients presenting to a neurology clinic (95% migraineurs), confirmed that anxiety sensitivity and severity of pain influenced fear of headache pain, which in turn contributed to resulting escape and avoidance behaviors. Consistent with the aforementioned model, headache severity (z = 0.35) and anxiety sensitivity (z = 0.40) loaded significantly on fear of pain, which loaded directly on headache-related escape and avoidance behaviors (z = 0.90). In turn, headache severity and anxiety sensitivity both contributed indirectly to escape/avoidance behaviors via their influence on fear of pain. This relationship is depicted in Figure 1.

Figure 1. Relationship between psychological variables associated with headache pain (as described by Norton & Asmundson, 2004)



The combination of avoiding and fearing pain creates a vicious cycle by which individuals' inaccurate negative cognitions promote avoidance behaviors, leading to physical deconditioning and further increased pain, and, in turn, perpetuating avoidance (Asmundson,

Norton, & Veloso, 1999; McCracken et al., 1992; Rachman & Arntz, 1991). This cycle contributes to maintenance of disability. Thus, anxiety sensitivity and fear of pain have important prognostic impact on coping with headache pain.

Headache Self-Efficacy

Effective self-management of headaches is a critical health adjustment variable and the primary goal of cognitive-behavioral approaches to treating headache disorders. Coping with headache-related pain involves interpreting and responding to a range of pain-related stimuli along multiple dimensions (Nash & Thebarge, 2006). In addition to the aforementioned cognitive variables associated with headache, self-efficacy has been identified as a crucial factor in headache control (French et al., 2000; Martin, Holroyd, & Penzien, 1990; McGrath, Penzien, & Rains, 2006). Bandura (1977) defined self-efficacy as "belief in one's capabilities to organize and execute the course of action required to produce given attainments" (p.3). In essence, self-efficacy refers to one's confidence or belief in his/her own ability to follow through with a particular course of action. Research indicates that high levels of self-efficacy are important factors in pain management, disability, and overall psychological functioning (Keefe et al., 1997; Lorig, Chastain, Ung, Shoor, & Holman, 1989; Spinhoven, Ter Kuile, Linssen, & Gazendam, 1989).

Changes in levels of self-efficacy moderate (Marlowe, 1998) and are correlated (Bond, Dirge, Rubingh, Durrant, & Baggaley, 2004) with the influence of stress on headache frequency. Self-efficacy also influences management of triggers, predicts response to treatment, and influences coping with pain (Anderson, Dowds, Pelletz, Edwards, & Peeters-Asdourian, 1995; Smith & Nicholson, 2006), such that higher levels of self-efficacy are associated with more favorable outcomes. In pain sufferers, self-efficacy can become enhanced through exposure to

feared activities without the pairing of negative consequences (i.e., experience of pain; Turk & Okifuji, 2002).

Human Immunodeficiency Virus (HIV)

Description of HIV/AIDS

The human immunodeficiency virus (HIV) was first discovered in the United States as the primary cause of acquired immunodeficiency syndrome (AIDS; Clavel et al., 1986; Clavel et al., 1987; Kanki et al., 1987; Eickhoff, 1988). HIV attacks the body's immune system by reducing CD4 white blood cells, disabling the body from fighting off illnesses (Center for Disease Control & Prevention (CDC), 2009). Once the body's CD4 cell count falls below 200 (per cubic millimeter of blood), a diagnosis of AIDS is warranted. During the early stages of HIV, most individuals remain asymptomatic. Those who exhibit signs typically display flu-like symptoms, such as fever, aching muscles, rash, and swollen glands (CDC, 2009). Without treatment, HIV usually progresses into AIDS within the first 8 to 10 years. With treatment, AIDS often can be held off for approximately fifteen years or more. In the later stages of HIV, before AIDS is diagnosed, more severe symptoms can include exhaustion, fever/night sweats, easy bruising, chronic yeast infections, thrush, significant weight loss, and chronic diarrhea. AIDS is commonly developed from opportunistic infections (i.e., tuberculosis, severe bacterial infections, lymphoma, recurrent pneumonia) that occur as HIV progresses (CDC, 2009).

HIV/AIDS Statistics

AIDS, first identified in the United States in 1981, had spread to 46,000 people by 1987 (CDC, 1987) and was estimated to have infected one million people by 2007 (CDC, 2009; Martin, Fain, & Klotz, 2008). Worldwide, it was estimated that 38.6 million people were living with HIV in 2005 (Joint United Nations Programme on HIV/AIDS, 2008). The incidence of HIV

increased in the mid-1990s, declined slightly after 1999, and has been stable since then (Hall et al., 2008). Since its identification almost 30 years ago, HIV has persistently remained one of the greatest global public health challenges (CDC, 2009).

HIV disproportionately affects males and those of minority status. In 2006, males made up 73% of the newly HIV-infected population, while females comprised only 27% (CDC, 2009). Of the newly infected, race splits were as follows: African Americans (45%), Caucasians (35%), Hispanics/Latinos (17%), Asians/Pacific Islanders (2%), and American Indians/Alaskan natives (1%; CDC, 2009; Hall et al., 2008).

HIV-infected individuals were first identified within the homosexual male population (Eickhoff, 1988) and HIV and AIDS were first believed to be exclusively homosexual illnesses. As prevalence spread, though, the routes of contraction and primary populations at risk were accurately identified. Eickhoff (1988) reported that 65% of HIV-infected individuals were homosexual or bisexual males, 8% were homosexual or bisexual intravenous drug users, and 16% were intravenous drug users only. From 2001 to 2006, 46% of new HIV-infections were among males who have sex with males (MSM; CDC, 2008). By 2006, 53% of new HIV-infected individuals were MSM (CDC, 2009). HIV can be contracted through sexual contact with an HIV-infected individual, needle sharing with intravenous drug users, birth to HIV mothers, and receiving multiple blood transfusions (Eickhoff). Fortunately, screening processes were quickly developed and implemented to prevent further HIV contraction from blood transfusions. Approximately 3 to 5% of HIV-infected individuals do not meet any of the aforementioned criteria, leaving a small window for unknown risk factors (Eickhoff).

HIV was originally seen as a problem primarily for younger adults. However, estimates now indicate that 25% of the HIV population in the United States is 50 years and older

(Operskalski, Mosley, Busch, & Stram, 1997). From 2001 to 2005, the number of HIV cases in United States for people 50 years or older increased by 77% (Valenti, 2008). Given the lifelong struggle that most HIV-infected individuals endure, the cost of related medical care is expectedly high. Estimates have been made that lifetime health care costs for HIV-infected individuals range somewhere between $40,000 to $60,000 (Scitovsky & Rice, 1988).

The death rates for older adults are higher than those for younger adults with HIV, and HIV progresses to AIDS more quickly in older adults than in younger adults. More than half of older adults develop AIDS within the first year of HIV infection (Martin et al., 2008). Research has indicated that only 59% of adults over 65 years of age survive past 36 months after developing AIDS, compared to 87-90% of adults aged 20-39 (Operskalski et al., 1997). Fortunately, through the development and widespread use of Antiretroviral (ARV) medications and the progressive Highly Active Antiretroviral Therapy (HAART), associated morbidity and mortality rates began to decrease substantially in the mid-1990s (Palella et al., 1998; Palella et al., 2006). Prognosis for HIV is currently considered as good or better than medical conditions such as hypertension and diabetes (Many, 2009), as it is now labeled more of a chronic than terminal illness (Gifford & Groessl, 2002). As stated by Vervoort et al. (2009), "Starting HAART is seen as a way to get control over HIV instead of being at the mercy of HIV" (p. 435).

HIV and Psychiatric Comorbidity

Along with the dramatic increase in medical problems and stigma related to HIV infection, the likelihood of experiencing mental illness is also much higher than in the general population (Acuff et al., 1999; Bing et al., 2001; Ickovics et al., 2001; Judd et al., 2005; Kemppainen, 2001; Sevard, Laberge, Gauthier, Ivers, & Bergeron, 1998). Living with HIV/AIDS is not easy. Those infected will encounter a variety of medical, social, and

interpersonal difficulties, typically resulting in considerable stress (Larya & Gien, 1993) and in turn occasioning psychological difficulties. According to the HIV Cost and Services Utilization Study (HCSUS), the first major research effort on people being treated for HIV infection, the prevalence of mental illness is significantly higher in the HIV population than the general population (Bing et al., 2001). In this study of 2,864 HIV-infected individuals, results indicated high prevalence rates of substance abuse disorders (50.1%), major depressive disorder (36.0%), dysthymia (26.5%), generalized anxiety disorder (15.8%), and panic disorder (10.5%; Bing et al., 2001).

HIV and Major Depressive Disorder. Depression is a particular problem among individuals living with HIV/AIDS. Acuff et al. (1999) conducted a large, multisite prevalence study and found that over 75% of HIV-infected individuals had major depressive disorder, dysthymia, or both. Using structured diagnostic interviews and self-report measures on 129 HIV/AIDS patients, Judd et al. (2005) found that 27% met criteria for a mood disorder. Using self-report data, Sevard et al. (1998) found that one-fifth of 149 HIV participants described symptoms of depression. Thus, while exact prevalence rates of depression have varied considerably depending upon the study design and method of assessment, these disparate studies all confirm very high rates of depression among the HIV/AIDS population.

Treisman, Fishman, Schwartz, Hutton, & Lyketsos (1998) described two different types of depression in the HIV population: primary and secondary. Primary depressed individuals are those with causes similar to the general population (i.e. family history of mood disorders, personality disorders, and experiences of adverse life events). Secondary depression occurs in those who are suffering from depression as a result of living with HIV/AIDS (Treisman et al., 1998). By contrast, Judd et al. (2005) found no distinct subtype of depression in HIV patients.

Their findings indicated that depressed HIV individuals were within the range of well-recognized risk factors for depression and that social support was among the most important protective factors. However, research has yet to find definitive evidence to describe the typical expression of depression in HIV/AIDS.

The implications of high psychiatric comorbidity in the HIV population are alarming, as depression has harmful negative effects on overall HIV prognosis. The presence of major depressive disorder in an HIV-infected individual not only reduces quality of life, but also reduces adherence to ARVs (Kemppainen, 2001) and results in increased mortality (Ickovics et al., 2001). With the more severely depressed HIV patient, an associated increase in functional impairment likely exacerbates disability, highlighting a need for early diagnosis and intervention (Judd et al., 2005).

Due to the negative implications of psychiatric comorbidity among HIV patients, accurate screening for depression is crucial. Somatic complaints are included on many depression measures, but their presence can often obscure the diagnostic picture when assessing individuals with chronic medical conditions such as HIV or AIDS. Therefore, the most useful tools for measuring depression in this population are those in which somatic questions have been excluded (Cavanaugh, Clark, & Gibbons, 1983; Clark, Cavanaugh, & Gibbons, 1983), such as the Depression, Anxiety, and Stress Scale (DASS; Lovibond & Lovibond, 1995), the BDI-Fast Screen (Beck, Steer, & Brown, 2000), or the POMS Depressive-Dejection subscale (Patterson et al., 2006).

HIV and Other Psychiatric Comorbidities. In addition to depression, anxiety has also been a focus in HIV research. Although virtually all studies have indicated a high prevalence of anxiety disorders in HIV-infected individuals, reported prevalence rates have varied from nearly

half (Sevard et al., 1998) to 70% (Cohen et al., 2002). Hintze, Templer, and Cappelletty (1993) found that family or partner knowledge of an individual's HIV diagnosis positively correlated with anxiety about death. Thus, HIV appears strongly associated with persistent worrying, including fears of mortality.

Substance abuse is also highly prevalent among individuals with HIV/AIDS. Kemppainen et al. (2006), in a study of 502 HIV-infected individuals, found high prevalence of current or past IV drug use (40%) and use of alcohol (45%), marijuana (25%), and cocaine (20%). Pence, Miller, Whetten, Eron, and Gaines (2006) found that alcohol- and drug-related disorders were significantly more likely among HIV clinic patients than among the general population (2.5 times and 7.5 times more common, respectively). One can assume that significant substance abuse and other comorbid disorders in the context of HIV will only exacerbate symptoms and worsen prognosis.

Researching HIV psychiatric comorbidity provides important information to health care providers about providing optimal continuity of care. Not only does it highlight the need for use of psychometrically validated tools by medical health providers, but assessing psychiatric comorbidities also underscores the need for referrals to mental health providers. Neglecting to screen for mental illness or to recommend a psychological consult could make a significant difference in overall prognosis.

HIV and Headaches

Headache in the HIV Population

Headaches are one of the most common medical complaints in the HIV population (Graham & Wippold, 2001; Holloway & Kieburtz, 1995; Newton, 1995). The potential for headache susceptibility is increased in this population due to a higher likelihood of head-pain-

related complications such as meningitis, sinusitis, or neurosyphilis (Wilson & Sande, 2001). Studies have found the prevalence of headaches in the HIV population to range from 40% (Mathews et al., 2000) to 61% (Justice, Rabeneck, Hays, Wu, & Bozzette, 1999). However, previous research has been unable to pinpoint a "typical" HIV headache, with considerable variability of headache patterns reported by HIV-infected individuals (Marchioni et al., 2006). One study found that TTH was considerably more prevalent than migraine (46% vs. 16%, respectively) and other headache types (6%; Evers et al., 2000), while another reported that migraines were more than five times more common than TTH and cluster headaches (76% vs. 14% vs. 10%, respectively; Mirsattari, Power, & Nath, 1998). No specific marker has been found for headache onset in HIV progression either, with headaches occurring both at various points throughout the course of HIV and after AIDS has developed (Holloway & Kieburtz, 1995; Singer, Kim, Fahy-Chandon, Datt, & Tourtellotte, 1996). However, some evidence suggests that as HIV progresses, migraine frequency decreases, while TTH frequency significantly increases (Evers et al., 2000).

The varied headache manifestations and course of headaches in HIV patients underscore the notion that accurate diagnosis of the HIV headache is challenging. In addition to primary headaches that occur in conjunction with (but are not caused by) HIV, it is now well recognized that some headaches in HIV patients are directly attributed to the HIV/AIDS infection itself (IHS code 9.3, "Headache Attributed to HIV/AIDS"). Generally, three patterns of headache attributable to HIV have been described. The first, consisting of a dull, continuous headache without consistent descriptions of duration, site, and response to medication, is a direct symptom of HIV infection (IHS, 2004; Holloway & Kieburtz, 1995; Marchioni et al., 2006). The second pattern is related to the contraction of aseptic meningitis, a rare development that can appear

during different stages of HIV (IHS; Holloway & Kieburtz; Marchioni et al.). The third is a pattern of headache related to the variety of opportunistic encephalic infections (i.e. tuberculosis, severe bacterial infections, lymphoma, recurrent pneumonia; CDC, 2009) that can occur throughout the course of HIV (IHS; Marchioni et al.). Diagnosis of the latter two patterns can be impaired by high costs of medical procedures needed to confirm the diagnosis. When meningitis or other encephalic infections are suspected, defensive medical assessment practices, such as lumbar punctures, MRIs, and CT scans, are implemented (Many, 2009). Further research on the HIV headache could potentially reduce the need for using these expensive and sometimes unnecessary procedures, cutting overall medical costs drastically.

Beyond the aforementioned headache types in the HIV patient, headaches due to medication side effects have been discussed on a limited basis. Headaches sometimes occur as a side effect to HIV medication (Hervey & Perry, 2000; Cvetkovic & Goa, 2003), but findings are not consistent and are thus not included in IHS classification criteria (IHS, 2004; Holloway & Kieburtz, 1995). More severe HIV headaches often appear related to causes such as mass lesions and medication side effects, among other causes (Newton, 1995).

Current Study

As has been illustrated, confusion surrounds the epidemiology and nature of headache patterns in the HIV population, warranting further research in this area. Marchioni et al. (2006) called for further reassessment of the HIV headache, concluding that a definite diagnosis of exclusion would be helpful for treatment considerations. At present, the literature does not provide enough evidence to distinguish HIV-related headaches from headaches not directly related to the infection (Marchioni et al., 2006). The results of the present study will provide

much-needed information about headache symptoms and patterns among HIV patients at varying stages of the HIV or AIDS disease process.

Extant literature has consistently confirmed both high prevalence of headaches in the HIV/AIDS population and elevated rates of psychiatric comorbidities among headache sufferers and individuals with HIV/AIDS. This literature has confirmed also that such comorbidities and other associated psychological factors are significant predictors of headache-related variables and prognosis. However, there is a strong need for extending these findings on the importance of psychological variables to individuals with headache in the HIV/AIDS population.

Therein lie the goals of the present study, which were designed to inform existing gaps in the literature on headaches among individuals with HIV/AIDS. The present study was designed to 1) determine headache patterns characteristic of those with HIV/AIDS; 2) explore levels of comorbid psychiatric symptoms between HIV patients with and without headache; and 3) determine the relationship between psychological variables (e.g., stress, self-efficacy, cognitive factors such as catastrophizing, anxiety sensitivity, and fear of pain) and headache characteristics among those with HIV/AIDS. The study was designed with the applied goals of facilitating recognition of headache characteristics among HIV patients and identifying relevant psychological factors that merit clinical attention in order to improve treatment outcomes.

The following goals and hypotheses were proposed:

Study Goal 1: Description of headache symptoms in HIV patients

Hypothesis 1a: Headache frequency, severity, and disability will increase as CD4 counts decrease and years living with HIV increases.

Hypothesis 1b: Increased relative prevalence of TTH symptoms (vs. migraine symptoms) will be associated with decreased CD4 counts and longer duration of HIV.

Hypothesis 1c: Individuals with preexisting headaches (prior to HIV contraction) will report increased frequency and intensity of headaches since acquiring HIV.

Hypothesis 1d: Headaches characterized by a dull, bilateral ache (IHS code 9.3) will be more prevalent among HIV/AIDS patients than migraine and TTH.

Study Goal 2: Group differences on psychiatric symptoms and psychological variables between headache and non-headache HIV patients

Hypothesis 2a: Psychiatric Symptoms: HIV patients with headache will report higher levels of depression, anxiety, and stress compared to HIV patients without headache.

Hypothesis 2b: Psychological Variables: HIV headache patients will report higher levels of catastrophizing, anxiety sensitivity, and fear of pain than will their non-headache HIV counterparts.

Hypothesis 2c: HIV headache patients will report higher levels of anxiety sensitivity pertaining to physical concerns than will HIV patients without headaches.

Study Goal 3: Determine prediction of headache-related disability and severity afforded by psychological (cognitive) variables. Psychological Variables will predict levels of headache-related disability and severity, even after controlling for levels of depression and anxiety.

Hypothesis 3a: A regression model comprised of 1) demographic factors and 2) depression and anxiety scores will be significant predictors of headache-related disability and severity.

Hypothesis 3b: Psychological variables including levels of headache self-efficacy, catastrophizing, fear of pain, and anxiety sensitivity will offer incremental prediction of headache-related disability and severity (i.e., above and beyond that afforded by demographic variables, CD4 counts, and levels of comorbid depression and anxiety.).

II. METHOD

Study Sites and Participants

Participants were patients diagnosed with HIV or AIDS presenting to two clinics in Montgomery, AL: the UAB Health Center Montgomery Internal Medicine Residency Program and the Montgomery AIDS Outreach (MAO) Program. Recent reports indicate that Montgomery County has the highest per capita rate of HIV infection in the state of Alabama (Alabama Department of Public Health, 2009). The UAB Internal Medicine Residency Program is a 3-year graduate medical training program accredited by the Accreditation Council for Graduate Medical Education and utilizes Baptist Medical Center–South in Montgomery as its primary teaching hospital. MAO is private, non-profit, community-based service organization that offers a variety of services, including medical treatment, mental health counseling, patient education, prevention education, and testing for individuals with or suspected of having HIV. There were no significant differences found between data collection sites on the main dependent variables.

A priori power analyses indicated that 150 participants were sufficient to support the statistical analyses for the present study, assuming a large effect size, power of .80, and alpha level of .05. Data were collected on 200 patients with HIV/AIDS who presented during their routine medical examinations to monitor their HIV/AIDS status. Of these, 101 were male, 98 were female, and 1 identified as transgendered. Their mean age was 43.22 (SD = 12.30), with ages ranging from 18 to 85. One hundred forty-eight (74.0%) were African-American, 42 (21.0%) were Caucasian, 7 (3.5%) were Hispanic/Latino, and the remaining three were of other

ethnicities. Regarding sexual orientation, 148 (74.0%) identified as heterosexual, 43 (21.5%) identified as homosexual, and 9 (4.5%) identified as bisexual. The majority of participants were unmarried (62.0% single, 13.0% divorced, 5.5% widowed). Level of education varied considerably among the sample, with 32.5% having not completed high school, 39.5% having a high school diploma or GED, 18.0% completing some college, and 10.0% receiving a college degree and/or attending graduate school. Notably, 88 (44.0%) participants reported smoking cigarettes, the mean frequency of which was nearly a pack per day (19.86; *SD* = 6.24) and the mean duration of which was 22.65 years (*SD* = 11.08).

Regarding HIV/AIDS characteristics of this sample, participants had been living with HIV for an average of 99.70 (*SD* = 69.29) months, with ranges from 2 to 282 months. Characterization of HIV/AIDS severity was made according to the World Health Organization's (2007) classification for immunological staging based on most recent CD4 counts (number of CD4 cells per mm^3 of blood). Seventy-four participants (37.0%) were classified as Not Significant (CD4 counts of 500 or greater), 40 (20.0%) were classified as Mild (CD4 of 350-499), 58 (29.0%) were classified as Advanced (CD4 of 200-349), and 28 (14.0%) were classified as Severe (CD4 below 200; meeting criteria for AIDS). The mean CD4 count was 443.48 (*SD* = 261.58). While CD4 counts are typically measured every 3 months in clinical settings, participants in this study had a mean of 43.52 (*SD* = 35.06) days since their last measurement. The overwhelming majority of the sample (93.5%) was prescribed ARVs at the time of data collection, 68% of whom were prescribed two or more ARVs. Participants were prescribed an average of 3.91 (*SD* = 1.92) total medications (ARV + non-ARV), and carried an average of 2.19 (*SD* = 1.48) non-HIV comorbid diagnoses. Besides headache, other prominent medical diagnoses were Hypertension (38%), Major Depressive Disorder (20%), Hepatitis (14%),

Hypercholesterolemia (9%), Diabetes (8.5%), Anxiety Disorders (6%), Asthma (6.5%), and Bipolar Disorder (4%).

Measures

Demographic Questionnaire and Medical Records

A demographic questionnaire obtained information pertaining to age, gender, marital status, race, sexual orientation, and level of education. Information obtained from each participant's medical record was also documented on this form. This information included most recent CD-4 counts and viral loads, date of HIV diagnosis, and a list of all prescribed medications and non-HIV diagnoses. This form can be found in Appendix A.

Structure Diagnostic Interview for Headache-Brief Version (SDIH)

The SDIH (Penzien, Rains, & Holroyd, 1993) is a structured diagnostic interview designed to establish headache diagnoses according to the operational criteria of the International Headache Society (1988, 2004). In addition to facilitating the diagnosis of migraine and tension-type headache, the SDIH also inquires about symptoms of cluster headache, posttraumatic headache, and medication overuse headache. We used a revised version of the original SDIH that accords with the most recent diagnostic criteria of the IHS (2004) and included questions about headache symptoms in relation to HIV infection. The formal diagnosis of a primary headache disorder was based on the semiology of the reported headache symptoms. The HIV infection itself and concurrent antiretroviral treatment were not considered to be a symptomatic cause of the headache, unless review of the patient's medical records indicated the presence of opportunistic CNS infections (e.g., toxoplasmosis, meningitis, encephalitis) at the onset of or prior to the headaches, in which case headache secondary to infection was diagnosed. The script for this interview can be found in Appendix B.

Brief Headache Screen

The Brief Headache Screen is a 6-item questionnaire designed to quickly screen for migraine, drug rebound headache, and other disabling headaches (Maizels & Burchette, 2003). The Brief Headache screen assesses frequency of disabling headaches, frequency of mild headaches, and frequency of medication use. Headache diagnoses based on the Brief Headache Screen have been well validated among medical patients presenting within multiple settings (Maizels & Burchette; Maizels & Houle, 2008). This form can be found in Appendix C.

Headache Management Self-Efficacy Scale (HMSE)

The HMSE was developed by French et al. (2000) and is used to measure self-efficacy of one's ability to manage and prevent headaches. The HMSE is a 25-item self-report measure that uses a 7-point Likert type response format (1 = Strongly Disagree to 7 = Strongly Agree). The scale is broken down into 4 subscales: Positive Prevention, Negative Prevention, Positive Pain Management/Disability, and Negative Pain Management/Disability. Regarding the psychometric properties of the measure, French et al. (2000) reported that the scale showed strong internal consistency reliability (α = .90). The HMSE was also found to have high construct validity, correlating positively with the Headache Specific Locus of Control-Internal (HSLC-I; .40; Martin, Holroyd, & Penzien, 1990), the Interview of Coping Efforts-Headache Version-Prevention (ICE-H:P; .54; Hill et al., 1999), and the Interview of Coping Efforts-Headache Version-Tolerance (ICE-H:T; .55; Hill et al., 1999). High scores on the HMSE are associated with lower levels of headache disability (r = -.24), and low scores on the HMSE are associated with increased headache severity (r = -.29; French et al., 2000). This measure can be found in Appendix D.

Migraine Disability Assessment (MIDAS)

The MIDAS is a 5-item questionnaire that quantifies headache-related disability in terms of missed days of work or school, housework, and nonwork (family/leisure) activities over the past 3 months. Questions assess the number of missed days due to headache and the number of days in which headache reduced usual productivity by more than half. The total score reflects the summed total of days that were missed and in which productivity was limited due to headache. The MIDAS has consistently demonstrated satisfactory internal consistency, high test-retest reliability, and good concurrent validity (Lipton, Stewart, Sawyer, & Edmeads, 2001; Stewart, Lipton, Dowson, & Sawyer, 2001; Stewart et al., 1999). This form can be found in appendix E.

Headache Impact Test (HIT-6)

The HIT-6 was developed by Kosinski and colleagues (2003) as a brief questionnaire to measure the impact of headaches on one's functioning and was used in conjunction with the MIDAS as criterion variables of headache-related disability. This 6-item measure asks the respondent to report the frequency with which headaches have limited one's ability to function. Forced-choice responses are assigned a numerical value and summed to calculate the total score of headache impact (Range = 36-78). The HIT-6 has been found to be a reliable, valid, and internally consistent measure of headache impact in research on headache populations (Kawata et al., 2005; Kosinski et al., 2003). The HIT-6 is reprinted in appendix F.

Anxiety Sensitivity Index – 3rd Edition (ASI-3)

The ASI-3 was developed by Taylor et al. (2007) as an updated version of the original ASI (Peterson & Reiss, 1992) and the ASI-R (Taylor & Cox, 1998a, 1998b). The ASI scales were designed to quantify fear of anxiety sensitivity, or the belief that anxiety symptoms should be feared because they portend dangerous consequences. The ASI-3 is an eighteen-item self-report and is rated on a 5-point Likert type scale (0 = Very little to 4 = Very much). The ASI-3

contains three subscales pertaining to anxiety sensitivity toward particular clusters of anxiety symptoms: Physical Concerns, Cognitive Concerns, and Social Concerns. Taylor and colleagues (2007) demonstrated that this most recent ASI has high internal consistency (Physical Concerns: $\alpha = 0.86$, Cognitive Concerns: $\alpha = 0.91$, and Social Concerns: $\alpha = 0.86$) and high construct validity. This form can be found in Appendix G.

Pain Anxiety Symptoms Scale (PASS)

The PASS-20 was developed by McCracken and Dhingra (2002) as a shortened form of the original PASS (McCracken, Zayfert, & Gross, 1992). This 20-item Likert-scale instrument was designed to assess pain-related anxiety/fear of pain. The PASS is designed to assess four components of pain-related anxiety, including fear appraisal, cognitive anxiety, physiological anxiety, and escape/avoidance. Reliability and validity research has confirmed that the PASS-20 is a strong substitute for the PASS when time and effort of participants is a concern, as all four subscales of the PASS-20 correlate with the original instrument above r = .90 (McCracken & Dhingra; Roelofs et al., 2004). Internal consistency of the PASS-20 is high ($\alpha = .91$; McCracken & Dhingra), and the measure has demonstrated convergent validity with other measures of pain-related constructs (Roelofs et al., 2004). The PASS-20 is reprinted in appendix H.

Pain Catastrophizing Scale (PCS)

The PCS was designed to measure exaggerated negative orientations towards pain (Sullivan, Bishop, & Pivik, 1995). This measure is a 13-item self-report measure that is rated on a 5-point Likert type scale (0 = Not at all to 4 = All). The PCS measures catastrophizing along three subscales: rumination, magnification, and helplessness. Internal consistency of the PCS is high ($\alpha = .87$), and research has indicated that it has convergent validity (Sullivan et al., 1995). This form can be found in appendix I.

Depression Anxiety Stress Scale (DASS)

The DASS was designed to assess symptoms of depression, anxiety, and stress within a single self-report measure (Lovibond & Lovibond, 1993). The DASS is a 42-item measure on which responses are rated on a 4-point Likert type scale (0 = Did not apply to me at all to 3 = Applied to me very much, or most of the time). For the Depression scale, scores from 0-9 are considered Normal, from 10-13 are considered Mild, from 14-20 are considered Moderate, from 21-27 are considered severe, and scores of 28 and higher are considered extremely severe (Lovibond & Lovibond, 1993). For the Anxiety scale, scores from 0-7 are considered Normal, from 8-9 are considered Mild, from 10-14 are considered Moderate, from 15-19 are considered Severe, and scores of 20 and higher are considered Extremely Severe (Lovibond & Lovibond, 1993). For the Stress scale, scores from 0-14 are considered Normal, from 15-18 are considered Mild, from 19-25 are considered Moderate, from 26-33 are considered severe, and scores of 34 and higher are considered extremely severe (Lovibond & Lovibond, 1993). The various scales have demonstrated strong internal consistency: DASS total scale (α = .97), Depression subscale (α = .96), Anxiety subscale (α = .92), and Stress subscale (α = .95; Page, Hooke, & Morrison, 2007). Lovibond and Lovibond's component analysis of the DASS found the measure to have high construct validity and demonstrated that the scale items show adequate factor loadings. Further, strong correlations were found between the DASS Depression scale and the Beck Depression Inventory (r = 0.74, Beck & Steer, 1987) and between the DASS Anxiety scale and the Beck Anxiety Inventory (r = 0.81, Beck & Steer, 1990; Lovibond & Lovibond, 1995). The DASS is reprinted in appendix J.

Procedure

All participants in this study were treated in accordance with the ethical guidelines and principles set forth by the American Psychological Association. Participants were recruited during their routine medical examinations at both sites over continuous weeks until complete data were obtained on 200 individuals. Thirty-six participants declined to participate. Participants provided written informed consent with the understanding that their participation was entirely optional and that they could withdraw at any time. After informed consent was obtained, one of the two primary researchers (a trained staff psychologist from the UAB Internal Medicine Program and a psychology graduate student from the University of Mississippi) administered the SDIH in a private setting in order to clarify headache-related symptoms, patterns, and intensity. Prior to administration, participants were screened for reading level using the Reading Scale of the WRAT-4 (Wilkinson & Robertson, 2006) due to documented low reading proficiencies in Central Alabama (Jackson et al., 1991; Bogie, 1995). Participants with a reading level below 7th grade were administered the informed consent documents and questionnaire measures orally (approximately 120 participants). All participants providing consent then completed a packet of the aforementioned questionnaires under the premise that the study's results would help improve prognosis and treatment planning for individuals suffering from HIV. At the conclusion of their participation, all participants were offered a packet of self-help information on dealing with HIV, depression, anxiety, and tips to help improve overall headache prognosis; those reporting significant psychopathology were provided referral information for local outpatient providers.

Data Analysis

Statistical analyses were conducted using PASW by SPSS version 17.0 for participants who had complete data for all variables of interest. Descriptive statistics and frequencies were

used to report demographic statistics and headache symptoms and typologies among HIV patients. Correlational analyses were used to determine the relationship between CD4 counts, HIV duration, and headache severity/disability. An independent samples T-test was used to compare patients on the categorical variables of interest pertaining to headache characteristics (prevalence of migraine vs. TTH). Controlling for multiple comparisons, a MANOVA was conducted to analyze group differences between HIV patients with and without headache on levels of psychiatric comorbidity and the psychological variables of interest (pain catastrophizing, anxiety sensitivity, fear of pain). Significant findings on the MANOVA provided support to parse out results into separate ANOVAs. Determination of those variables predictive of headache-related disability and severity were made using a series of hierarchical linear regression analyses. In such analyses, demographic variables (age, gender) were entered simultaneously as the first block, CD4 counts were entered as the second block, depression and anxiety scores from the DASS were entered simultaneously as the third block, and the psychological variables of interest were entered iteratively and independently (as separate fourth blocks) to determine the proportion of headache-disability (MIDAS, HIT-6 scores) variance accounted for by each psychological factor.

III. RESULTS

Headache symptoms and diagnoses in HIV patients. (Study Goal 1)

Headache prevalence. Of the 200 HIV/AIDS participants, 107 (53.5%) reported having headaches. Four participants' headaches were attributable to secondary causes (2 with toxoplasmosis, 1 with meningitis, and 1 with posttraumatic headache) based on review of medical records and interview data, and these individuals with secondary headache typologies were thus not included in subsequent group comparisons. According to medical records, 39% of all participants carried a pre-existing headache diagnosis. Regarding demographic differences, the headache and non-headache patients did not differ on most demographic variables: mean age, years of education, or on distribution of gender or race. Importantly, the two groups did not differ either on duration of HIV or number of prescribed ARVs. Table 2 presents the data for demographic differences between the headache and non-headache participants. Most notably, the headache participants had significantly lower CD4 counts than did those without headache, but this difference was not attributable to differences in duration of HIV or prescribed ARVs.

Headache diagnoses. Primary headache diagnoses were made based on ICDH-II (IHS, 2004) criteria and obtained from responses to the SDIH. Two researchers reviewed each headache participant's interview data and compared them to ICHD-II criteria to establish diagnoses independently: inter-rater agreement was 99.03% (102/103 primary diagnoses). The one discrepant diagnosis was resolved through discussion. Of the 103 with primary headache disorders, 88 (85.44%) met diagnostic criteria for migraine: 14 (13.59%) with episodic migraine

without aura, 8 (7.77%) with episodic migraine with aura, 11 (10.68%) with probable migraine, and 55 (53.40%) with chronic migraine. Fifteen patients (14.56% of those with headache) met criteria for TTH: 12 (11.65%) with episodic TTH, 1 (0.97%) with probable episodic TTH, 1 (0.97%) with chronic TTH, and 1 (0.97%) with probable chronic TTH. Among participants with headache, 19 (18.45%) identified their headache as fitting the description of a dull, bilateral ache (IHS code 9.3, "Headache Attributed to HIV/AIDS"). Characteristics of reported headache symptoms of all participants, including those with headache attributed to HIV infection, are found in Table 3. The percentage of patients within each headache diagnosis (excluding the 1 participant with chronic TTH and 2 participants with "probable" TTH diagnoses) reporting various headache symptomatology can be found in Table 4.

Table 2

Demographic Characteristics of the Headache vs. Non-Headache Samples

Variable	Headache (*n* =103)	Non-Headache (*n*=93)
Mean CD4 count (*SD*)	326.70** (222.85)	585.51 (228.48)
Mean age (*SD*)	43.28 (11.65)	43.16 (13.26)
Gender (% female)	47.60	50.50
Race (% African American)	74.80	73.10
Marital Status (% single)	66.00	59.10
Sexual orientation (% heterosexual)	66.00*	82.80
Mean education (*SD*)	11.51(1.67)	11.71 (2.01)
% smoker	77.70**	5.40
Mean # ARVs (*SD*)	1.93 (0.95)	1.80 (0.96)
Duration of HIV in months (*SD*)	103.07 (72.15)	95.84 (65.14)

** $p < .0001$; *$p < .05$

Table 3

Headache Characteristics of all Patients with Headache (n=103)

Characteristic	N (%)
Pain Location	
Frontal	65 (63.11)
Temporal	55 (53.40)
Supraorbital	22 (21.36)
Orbital	6 (5.83)
Occipital	5 (4.85)
Pain Distribution	
Unilateral	36 (34.95)
Bilateral	67 (65.05)
Pain Features	
Pulsating	53 (51.46)
Pressing/Tightening (Non-Pulsating)	50 (48.54)
Described as a Dull Bilateral Ache	19 (18.45)
Pain Severity (0-10)	
0-3 (mild)	1 (0.97)
4-5 (moderate)	10 (9.71)
6-10 (severe)	92 (89.32)
Monthly Headache Frequency	
<1 per month	1 (0.97)
1-14 per month	41 (39.81)
15-29 per month	39 (37.86)
30 per month	22 (21.36)
Headaches Began	
Before HIV Diagnosis	18 (17.48)
After HIV Diagnosis	85 (82.52)
Aggravated by Exercise	81 (78.64)
Phonophobia	85 (82.52)
Photophobia	81 (78.64)
Nausea	32 (31.07)
Vomiting	14 (13.59)

Table 4

Headache Characteristics by Diagnosis, Excluding Probable TTH (n=101)

Symptom	EM without Aura n (%)	EM with Aura n (%)	Probable Migraine n (%)	Chronic Migraine N (%)	Episodic TTH n (%)
Number diagnosed*	14 (13.59)	8 (7.77)	11 (10.68)	55 (53.40)	12 (11.65)
Pain Location					
Frontal	10 (71.43)	3 (37.50)	7 (63.64)	39 (70.91)	4 (33.33)
Temporal	6 (42.86)	6 (75.00)	3 (27.27)	32 (58.18)	7 (58.33)
Supraorbital	1 (7.14)	1 (12.50)	3 (27.27)	15 (27.27)	1 (8.33)
Orbital	1 (7.14)	1 (12.50)	0 (0.00)	1 (1.82)	3 (25.00)
Occipital	0 (0.00)	1 (12.50)	0 (0.00)	2 (3.64)	2 (16.67)
Pain Distribution					
Unilateral	6 (42.86)	3 (37.50)	8 (72.73)	14 (25.45)	3 (25.00)
Bilateral	8 (57.14)	5 (62.50)	3 (27.27)	41 (74.55)	9 (75.00)
Pain Features					
Pulsating	6 (42.86)	7 (87.50)	3 (27.27)	33 (60.00)	2 (16.67)
Pressing/ Tightening	8 (57.14)	1 (12.50)	8 (72.73)	22 (40.00)	10 (83.33)
Described as a Dull Bilateral Ache	4 (28.57)	1 (12.50)	3 (27.27)	6 (10.91)	5 (41.67)
Mild Pain	0 (0.00)	0 (0.00)	0 (0.00)	0 (0.00)	1 (8.33)
Moderate Pain	1 (7.14)	1 (12.50)	0 (0.00)	0 (0.00)	6 (50.00)
Severe Pain	13 (92.86)	7 (87.50)	11 (100.00)	55 (100.00)	5 (41.67)
<1 per month	0 (0.00)	0 (0.00)	0 (0.00)	0 (0.00)	1 (8.33)
1-14 per month	13 (92.86)	7 (87.50)	9 (81.82)	0 (0.00)	11 (91.67)
15-29 per month	0 (0.00)	0 (0.00)	2 (18.18)	36 (65.45)	0 (0.00)
30 per month	1 (7.14)^	1 (12.50)^	0 (0.00)	19 (34.55)	0 (0.00)
Before HIV Diagnosis	2 (14.29)	1 (12.50)	3 (27.27)	3 (5.45)	9 (75.00)
After HIV Diagnosis	12 (85.71)	7 (87.50)	8 (72.73)	52 (94.55)	3 (25.00)
Aggravated by Exercise	13 (92.86)	8 (100.00)	6 (54.55)	52 (94.55)	2 (16.67)
Phonophobia	14 (100.00)	8 (100.00)	6 (54.55)	54 (98.18)	2 (16.67)
Photophobia	12 (85.71)	8 (100.00)	5 (45.45)	54 (98.18)	1 (8.33)
Nausea	6 (42.86)	5 (62.50)	4 (36.36)	17 (30.91)	0 (0.00)
Vomiting	2 (14.29)	4 (50.00)	2 (18.18)	6 (10.91)	0 (0.00)

*Percentages within this row refer to percentages of the entire headache sample. All other percentages are in reference to that particular diagnosis.

^ These two participants were diagnosed with episodic migraine despite having daily headaches because they did not meet the 3-month duration criterion for chronic migraine specified in the ICHD-II.

The headache group had a mean pain severity of 7.83 (out of 10; *SD* = 1.60) and a mean of 16.84 (*SD* = 10.57) headaches per month, indicating severe pain and chronic frequency. Confirming the high severity and frequency of headache, scores on the MIDAS and HIT-6 were indicative of severe headache-related disability. The mean MIDAS score of those with headache was 132.13 (*SD* = 91.52) out of 270 maximum. The mean HIT-6 score was 68.58 (*SD* = 10.63) out of 78 maximum.

Relationships between headache and HIV variables. Significant Pearson correlations were also found between CD4 counts and pain severity and headache frequency, indicating that progression of HIV is associated with increased head pain and frequency. Similarly, significant correlations were obtained between both CD4 counts and HIT-6 scores and CD4 counts and MIDAS scores (see Table 4), indicating that the progression of HIV is associated also with greater headache-related disability. Conversely, duration of HIV was not significantly related to headache disability, pain severity, and headache frequency. These correlations can be found in Table 5.

Regarding the prevalence of migraine versus TTH diagnoses as a function of CD4 counts, an independent-samples t-test yielded significant differences ($p < .0001$) between headache diagnoses. Participants with a primary TTH diagnosis had a mean CD4 of 505.23 (*SD* = 370.29) compared to a mean of 293.35 (*SD* = 175.22) for those with a primary migraine diagnosis, indicating an association between the severity of HIV and the type of headache experienced (with migraine being associated with greater severity of HIV/lower CD4 counts). This difference was not a function of HIV duration, as the migraine and TTH groups did not differ on duration of HIV (109.87 months [*SD* = 71.90 vs. 94.54 months [*SD* = 82.49], respectively).

Table 5

Correlations for relationships between headache and HIV variables

	CD4	HIV Duration	MIDAS	HIT-6	Severity	Frequency
CD4	__	NS	-.41**	-.41**	-.33*	-.32*
HIV Duration	NS	__	NS	NS	NS	NS
MIDAS	-.41**	NS	__	.80**	.58**	.79**
HIT-6	-.41**	NS	.80**	__	.69**	.75**
Severity	-.33*	NS	.58**	.69**	__	.53**
Frequency	-.32*	NS	.79**	.75**	.53**	__

** $p < .0001$; *$p < .002$; NS = Not Significant

Only 18 of 103 (17.5%) headache participants reported having headaches prior to being diagnosed with HIV. Of those participants, 11 (61.1%) reported no change in headache frequency, while three (16.7%) reported having headaches at least 10 more days per month after being diagnosed with HIV. Four (22.2%) participants' ratings of headache pain intensity changed from mild to moderate in the months and years following their HIV diagnosis, while 14 (77.8%) reported no change. The majority of headache patients thus reported developing their headache symptoms subsequent to HIV infection, and though limited by a small sample size, the majority of those with preexisting headaches denied an increase in frequency after being diagnosed with HIV.

Psychiatric symptoms and psychological variables between headache and non-headache HIV patients. (Study Goal 2)

Data for group (headache vs. non-headache) comparisons of psychological variables were first analyzed for multivariate assumptions and were found to be acceptably normal and linear. Prior to the multivariate analyses, the 103 primary headache and 93 non-headache HIV/AIDS participants were checked for multivariate outliers by group using Mahalanobis distance. Twelve outliers were found using a conservative $p < .001$ chi-squared cutoff (Mahalanobis distance < 24.322) as suggested by Tabachnick and Fidell (2007). All multivariate analyses were run with and without outliers, resulting in minimal statistical discrepancy. Consequently, the outliers were retained in the results reported below.

The Wilks' lambda multivariate criterion for overall group differences was significant, $F(1, 195) = 62.68$, $p < .0001$ (partial $\eta^2 = .67$), indicating that the headache and non-headache participants differed on the combination of variables pertaining to psychiatric comorbidity and psychological functioning. Subsequent univariate ANOVAs confirmed that the headache group reported significantly higher scores on measures of depression ($p < .0001$; $\eta^2 = .56$), anxiety ($p < .0001$; $\eta^2 = .56$), stress ($p < .0001$; $\eta^2 = .54$), pain catastrophizing ($p < .0001$; $\eta^2 = .66$), anxiety sensitivity ($p < .0001$; $\eta^2 = .53$), and fear of pain ($p < .0001$; $\eta^2 = .63$) than did their non-headache counterparts. Group means are presented in Table 6.

The headache and non-headache groups' scores on the DASS-42 were compared to assess differences in comorbid psychiatric symptoms of depression, anxiety, and stress. On the Depression scale, the headache group's mean score of 27.17 was indicative of severe depressive symptomatology, while the mean score of the non-headache group was in the non-clinical range. On the Anxiety scale, the headache group's mean score of 24.78 was indicative of extremely severe symptomatology, while the mean score of the non-headache group was in the non-clinical

range. On the Stress scale, the headache group's mean score of 26.98 was indicative of severe symptomatology, while the mean score of the non-headache group was in the non-clinical range.

Table 6

Mean Group Scores for Psychiatric Comorbidity and Other Psychological Variables

Variable	Headache (N = 103) *M (SD)*	Non-Headache (N = 93) *M (SD)*	F-value	*p*-value
DASS Depression	27.17 (12.89)	4.03 (6.12)	248.95	< .0001
DASS Anxiety	24.78 (11.98)	3.47 (5.75)	243.29	< .0001
DASS Stress	26.98 (11.68)	5.70 (7.29)	228.47	< .0001
Pain Catastrophizing	34.86 (14.59)	3.09 (6.32)	377.00	< .0001
Anxiety Sensitivity	43.05 (19.55)	9.15 (10.90)	218.32	< .0001
Fear of Pain	66.51 (28.62)	7.78 (13.08)	329.42	< .0001

Note: Higher scores reflect more severe psychological impairment.

As with scores on the DASS, headache patients reported significantly higher levels of pain catastrophizing, fear of pain, and anxiety sensitivity than did HIV patients without headache. On the PCS, participants scoring above 24 are considered catastrophizers, while those scoring below 15 are non-catastrophizers (Sullivan et al., 1995). The headache group scored a mean of 34.86 (*SD* = 14.59), while the non-headache group had a mean of 3.09 (*SD* = 6.32). The PASS-20, a questionnaire used to measure fear of pain and pain anxiety, was originally normed on chronic pain patients with a mean total score of 38.62 (McCracken & Dhingra, 2002). The headache group's mean score on the PASS was 66.51 (*SD* = 28.62), while the non-headache group's mean was 7.78 (*SD* = 13.08). The ASI-3, a questionnaire used to measure anxiety

sensitivity, has mean score of 12.8 for a non-clinical population (Taylor et al., 2007). The headache group's mean score on the ASI-3 was 43.05 (*SD* = 19.55), while the non-headache group scored a mean of 9.15 (*SD* = 10.90). As hypothesized, headache patients reported higher levels of physical concerns related to anxiety sensitivity than did their non-headache counterparts (15.14 [*SD* = 6.63] vs. 3.75 [*SD* = 4.50]; $p < .0001$; $\eta^2 = .50$).

Prediction of headache-related disability by psychological variables. (Study Goal 3) Determination of those variables that predict headache-related disability and severity were made using a series of hierarchical linear regression analyses. In these analyses, demographic variables (i.e., age, gender) were entered simultaneously as the first block, CD4 counts were entered as the second block, depression and anxiety scores from the DASS were entered simultaneously as the third block, and the psychological variables of interest were entered iteratively and independently (as separate fourth blocks). This strategy was chosen to determine the proportion of headache-related disability variance (MIDAS, HIT-6 scores) accounted for by each psychological factor (i.e., headache self-efficacy, pain catastrophizing, fear of pain, anxiety sensitivity) above these other factors that have been shown to be associated with headache disability. The data presented below refer to incremental R^2 changes and significant F-values and *p*-values of these incremental changes.

After the entry of age and gender, CD4 counts accounted for a significant amount of variance ($R^2 = 16.43\%$, $F(1, 99) = 19.83$, $p < .0001$) on HIT-6 scores. Adding in scores from the DASS Depression and Anxiety subscales accounted for the largest amount of incremental variance ($R^2 = 47.54\%$, $F(2, 97) = 66.95$, $p < .0001$) on HIT-6 scores. When each was entered separately as the final block, the psychological factors of pain catastrophizing, fear of pain, and anxiety sensitivity remained significant: the PCS to yield $R^2 = 10.74\%$, $F(1, 96) = 43.52$, $p <$

.0001; the PASS to yield R^2 = 15.00%, F(1, 96) = 74.08, p < .0001; and the ASI to yield R^2 = 9.72%, F(1, 96) = 37.77, p < .0001.

Controlling for age and gender, CD4 counts accounted for a significant amount of variance (R^2 = 16.32%, F(1, 99) = 20.42, p < .0001) on MIDAS scores. Adding in scores from the DASS Depression and Anxiety subscales again accounted for the largest amount of variance (R^2 = 43.78%, F(2, 97) = 60.08, p < .0001) on MIDAS scores. The last blocks added in scores from the varying psychological factors to yield significant incremental variances: the PCS to yield R^2 = 4.42%, F(1, 96) = 13.72, p < .0001; the PASS to yield R^2 = 3.91%, F(1, 96) = 11.95, p < .002; and the ASI to yield R^2 = 4.02%, F(1, 96) = 12.32, p < .002. Age and gender were weak predictors of headache-related disability, accounting for less than 5% of the variance in both the HIT-6 and the MIDAS.

IV. DISCUSSION

The size and diversity of the HIV/AIDS population in this study allowed for considerable confidence in the striking results of the data analysis. With the help of staff from two HIV/AIDS clinics in Montgomery, AL, this study obtained detailed headache data on 200 HIV-infected individuals at varying stages of HIV/AIDS severity. Compared to the few existing studies on HIV-related headache, this study is unique for a number of reasons. Most notably, most of the few existing studies on headache in HIV/AIDS patients were conducted prior to the peak of HAART success, and thus the present study affords more accurate characterization of the current HIV-infected population. Second, the sample's high percentage of ARV prescription and low percentage of diagnosed opportunistic infection provides a unique yet consistent sample in which secondary causes of HIV-related headache appear uncommon. Third, all participants were recruited during routine HIV medical appointments and were therefore presumed not to be under substantially differentiating levels of duress. Lastly, the current study was approached as a headache study on the HIV-infected population from a psychological perspective. With respect to previously published research available, to our knowledge no study has ever been conducted from such a comprehensive multidisciplinary approach.

Although the few previous studies on HIV-related headache have focused primarily on secondary causes of HIV-related headache using neuroimaging technologies (Graham & Wippold, 2001; Goldstein, 1990; Singer et al., 1996), this study was aimed at identifying patterns of headache symptoms, primary headache diagnoses, and related psychological variables. The

results of this study are practical and could enhance the medical and mental health treatment of individuals with HIV/AIDS and comorbid headache disorders.

Description of headache symptoms in HIV patients

Headache prevalence. Previous headache studies on HIV populations have attempted to assess the prevalence of various headache diagnoses. Some studies have found stark contrasts not only in prevalence of headache, but in types of primary headache diagnoses among HIV-infected individuals. Mirsattari et al. (1998) and Singer et al. (1996) obtained headache prevalence rates of 38% and 43%, respectively, among HIV patients, while Evers et al. (2000) found that 60% suffered from headache. As previously noted, other studies have also indicated a range of 40% (Matthews et al., 2000) to 61% (Justice et al., 1999). In the present study, headache prevalence was 53.5%, confirming a broad generalization that approximately half of HIV-infected individuals suffer from headache.

Of the 107 individuals with headache, three had previous diagnoses of opportunistic encephalic infections and one was classified as experiencing posttraumatic headaches (subsequent to a head injury). This evidence suggests that far fewer HIV-infected individuals experience the encephalic infections that have in the past been considered the cause of head pain. Though neuroimaging procedures were not employed in this study to rule out such infections, the overwhelmingly large absence of documented encephalic infections is likely attributable to the current era of HAART therapy, in that fewer HIV patients display neurological symptoms of opportunistic infections that would warrant neuroimaging. It is likely future studies in this area will yield similar results, indicating that while the presence of opportunistic infection in HIV-infected individuals has decreased, headache prevalence has remained consistent.

Headache diagnoses. When considering specific headache diagnoses, results of previous studies are inconsistent. For instance, both Lipton et al. (1991) and Goldstein (1990) reported that headaches secondary to opportunistic infections comprised the majority of headache diagnoses among patients with HIV. Lipton et al. (1991) found only six patients with identifiable migraines and two with TTH out of 49 headache patients. Evers et al. (2000) found that, out of a sample size of 131 HIV-infected individuals, 21 (16%) suffered from migraine while 60 (45.8%) suffered from TTH, indicating a preponderance of TTH among primary headache diagnoses. Within these headaches, preexisting migraines improved while preexisting TTHs worsened over the course of HIV (Evers et al., 2000). Of the previous studies, our findings that migraine (and particularly chronic migraine) is the most common form of primary headache among individuals with HIV are consistent with the results obtained by Mirsattari et al. (1998), who likewise confirmed that the majority of HIV headache sufferers show a migrainous typology (75% migraine vs. 14% TTH).

Notably, both Lipton et al. (1991) and Goldstein (1990) were published before the era of successful HAART, further suggesting that previously documented infectious causes of headache in this population have likely declined substantially. Further, methodological differences were noted between Evers et al. (2000) and the current study. Most notably, 93.5% of participants in the current study were prescribed ARVs, compared to only 65% in Evers et al. Additionally, the participants in the current study were all recruited during their routine examinations, while the participant pool in Evers et al. consisted of asymptomatic individuals presenting for routine examinations, individuals presenting with nonspecific complaints (e.g. dizziness), and individuals with AIDS-defining non-CNS opportunistic infections. The aforementioned methodological differences are of vast importance in terms of generalization to

the modern HIV population. Although during the HAART era, both Evers et al. and Mirsattari et al. (1998) were conducted too early to obtain samples in which almost all participants were prescribed ARVs. However, both their studies and the present study suggest that headache diagnoses in current HIV/AIDS patients are most commonly consistent with primary headache typologies (migrainous and TTH subforms), rather than attributable to the more sinister opportunistic infections that characterized the earlier studies.

Regarding our attempt to characterize the "typical" primary headache pattern in HIV-infected patients, our findings indicate an overwhelming majority of headaches classified as migraine (85.44%) over TTH (14.56%). Strikingly, 53.4% of those with headache were classified as having chronic migraine. The symptom presentation within these migraine diagnoses is somewhat atypical, however. For example, many chronic migraineurs reported pain characterized by a bilateral location (74.55%) and pressing/tightening quality (40%), features typically associated with TTH. Although headache patterns were predicted to be more consistent with the "Headache Attributed to HIV/AIDS" pattern of a dull, bilateral ache variable in onset, site, and intensity (IHS code 9.3), an alternate pattern emerged. Only 19 (18.45%) individuals diagnosed with a primary headache characterized their pain as a dull, bilateral ache. A primarily bilateral pain distribution was indeed evident, but the other most prevalent symptoms were severe pain that was aggravated by activity and co-occurring phonophobia and photophobia, all of which are prototypical features of migraine. Pain site and the presence of nausea and vomiting were variable throughout the sample, perhaps as a function of the wide distribution of HIV severity.

CD4 and headaches. The results of the current study indicate striking differences between the headache and non-headache groups in terms of HIV/AIDS severity. From this

perspective, CD4 counts are clearly of central importance in predicting headache symptoms. As was evidenced anecdotally in this study, many individuals with HIV/AIDS know their most recent CD4 count reading and understand that their overall health is greatly affected by the rise and fall of that number. Consequently, Hypothesis 1a was, as expected, confirmed. The severity of HIV among the non-headache group was on average far less than that of the headache group (mean CD4 in non-significant vs. advanced range, respectively). This discrepancy indicates that the progression of HIV is strongly associated with the presence of headaches. Other headache-related variables were also strongly associated with CD4 counts: headache frequency, headache severity, and headache-related disability were inversely related to CD4 count. That is, existing headaches become more frequent, more intense, and are more disabling as HIV progresses.

HIV duration. In this sample, participants had been living with HIV for an average of 99.70 months (8.31 years). Although originally hypothesized as an important factor in headache presentation, duration of HIV was not significantly associated with any headache-related variable in the present study. In conjunction with our finding that HIV duration was unrelated to CD4 counts, these data suggest that the severity of HIV is a far more potent predictor of headache than is duration of HIV. The lack of a relationship with HIV duration is likely also a function of advances made in identification and distribution of ARVs – most notably the introduction of HAART. Given that this sample was involved in regular medical follow-ups specifically for HIV/AIDS, and that 93.5% of participants were prescribed ARVs, the present sample likely represents a profile of aggressive treatment that may not be reflective of individuals in underserved communities. Nevertheless, these findings speak indirectly to the efficacy of ARVs in transforming HIV/AIDS outcomes from a deadly to a more chronic illness.

The implications of this particular set of results highlight the importance of focusing on medical complaints (e.g. HIV, headaches) from a biopsychosocial perspective (Suls & Rothman, 2004). Approaching medical illnesses from a multidisciplinary perspective can allow for the identification of important psychological factors such as stress, social support, and treatment compliance as a focus of treatment (Suls & Rothman, 2004). Within the context of HIV, emphasizing chronicity and adjustment over terminality may allow for improved treatment success in terms of comorbid physical and mental health outcomes.

Effect of HIV on preexisting headaches. A large majority (82.5%) of the sample indicated that their headaches began after being diagnosed with HIV. Of those with preexisting headaches (n = 18), only three reported an increase in frequency and four reported an increase in pain intensity, indicating little effect of HIV on preexisting headaches. While Evers et al. (2000) found significant increases in frequency and pain intensity on preexisting TTHs following HIV infection, improvement was noted in preexisting migraines. On the other hand, Singer et al. (1996) excluded individuals with preexisting headaches from data analysis, while the majority of other previous studies focused upon HIV headaches either did not report the data or failed to address the issue. Due to such a small number of patients with preexisting headaches in the current study, our results in this particular domain should be considered cautiously.

Smoking and headache. A clear discrepancy between the headache and non-headache group was cigarette use, although this was an ancillary focus of the present study. In previous studies focused on headache prevalence and tobacco use, smokers were more likely to complain of headache than non-smokers (Aamodt, Stovner, Hagen, Brathen, & Zwart, 2006; Waldie, McGee, Reeder, & Poulton, 2008). In the present study, while only 5.4% of the non-headache group identified as smokers, 77.7% of the headache group endorsed cigarette use with an average

of almost one pack per day. While a clear cause-effect relationship cannot be obtained in this cross-sectional design, this finding adds to the practical treatment implications of this study for medical providers when discussing headaches with HIV-infected individuals. This finding also provides indirect evidence linking smoking to headache, but among a much more specific population than has been examined in previous studies on smoking and headache.

Psychological variables between groups. The MANOVA and individual ANOVA results from this study all highlighted striking differences between the headache and non-headache groups on each measure of psychiatric symptoms and cognitive variables. As hypothesized, the headache group scored significantly higher on measures of depression, anxiety, stress, pain catastrophizing, anxiety sensitivity, and fear of pain than the non-headache group. As predicted, anxiety sensitivity pertaining to physical concerns was much greater in the headache group than non-headache group.

These results may be interpreted to indicate that the individuals comprising the non-headache group experienced psychological health comparable to that of the general population. Not only were they without headaches, but they were functioning at an optimal level of psychological well-being and coping well with the stress of HIV, likely because their HIV severity was typically within a non-significant range. Conversely, the headache group endorsed significant psychological distress. They were generally unhappy, anxious, and reacted poorly to their emotional dysfunction and HIV-related stressors, likely related to the fact that their HIV was at a much more advanced stage (i.e., they had higher CD4 counts). These findings can be used to help medical providers inform their patients that HIV is a manageable disease in which infected individuals can expect positive mental health outcomes if they are compliant and proactive with treatment options.

Headaches and depression, anxiety, and stress. Consistent with previous studies indicating high comorbidity between headaches and depression (Breslau, 1998; Hamelsky & Lipton, 2006; Lake et al., 2005), the results of this study found drastic differences between the headache and non-headache groups on endorsement of symptoms of depression. While the participants in the headache group averaged severe depressive symptomatology (83.5% reported at least mild symptoms of depression), the non-headache group indicated non-clinical levels of depression similar to those reported by non-depressed individuals in the general population.

The headache versus non-headache group comparisons also replicated findings from previous studies showing high comorbidity between headaches and anxiety (Hamelsky & Lipton, 2006; Saunders et al., 2008). The headache group in this study endorsed levels of anxiety that are considered in the Extremely Severe range (82.5% considered at least mildly anxious), compared to generally non-clinical levels of anxiety among the non-headache group. Given the polarizing results on symptoms of depression, the similar results for symptoms of anxiety are not surprising. The consistent combined presence of depression and anxiety in a headache population (Breslau, 1998; Lake et al., 2005; Lanteri-Minet et al., 2005), particularly in those infected with HIV, suggests that these common psychiatric comorbidities should be given clinical attention in individuals with HIV, particularly those with advanced disease.

As previously discussed, stress is an important factor in the onset and exacerbation of headaches (Holm et al., 1986; Levor et al., 1986; Houle & Nash, 2008). Consistent with findings on depression and anxiety, the headache group in this study reported significantly higher levels of stress than the non-headache group. Individuals with high comorbid levels of depression, anxiety, and stress, even in the general population, are in need of psychological treatment. When adding in problems related to headaches and HIV, the necessity of treatment increases

considerably. The presence of psychiatric illness, particularly depression (Gordillo, del Amo, Soriano, & Gonzalez-Lahoz, 1999; Kemppainen, 2001; Sledjeski, Delahanty, & Bogart, 2005) has been closely linked to poor HAART compliance (Department of Health and Human Services, 2009) and HIV disease progression (Evans et al., 1997; Judd et al., 2000; Ghebremichael et al., 2009), highlighting the importance of recognizing these issues among HIV-infected individuals. Such results are vastly important for the practical implications of the current study, and should be considered in the context of recommendations made by medical treatment providers.

Headaches and catastrophizing. HIV/AIDS patients with headache reported significant difficulty in responding to and accurately estimating their ability to cope with that pain. This group displayed a catastrophizing profile consistent with that of other types of pain groups in previous studies (Burns et al., 2003; Tsui et al., 2007), adding to a limited body of research between headaches and catastrophizing. The high level of catastrophizing reported by this group is one likely explanation for the overall extreme scores indicated on the entire battery of self-report measures. A resulting response style is characterized by exaggeration of pain, excessive helplessness, and pessimism about coping ability (Severeijns et al., 2001; Smitherman, Nicholson, Schafer, & Houle, in press; Sullivan et al., 2001).

Headaches, anxiety sensitivity, and fear of pain. Regarding symptoms of anxiety sensitivity, the headache group endorsed considerably higher symptoms compared to the non-headache group. Because anxiety sensitivity is associated with fearing ambiguous physical sensations (Asmundson, Norton, & Norton, 1999), it was predicted that such a pattern would be apparent. As previously discussed, anxiety sensitivity is important temporally in the fear of pain (Norton & Asmundson, 2004). Patients with headache in this study also reported higher fear of

pain than did those without headache, and at levels that exceeded those of a treatment-seeking group of chronic pain patients (McCracken & Dhingra, 2002). Patients from McCracken and Dhingra were primarily Caucasian women with back and limb pain of less than 3 years duration, and presumably without a diagnosis of HIV/AIDS. Although subjective pain severity cannot be compared directly between the two studies, duration of pain and reported severity of other medical/mental health problems likely influence fear of pain considerably.

Results of the current study thus provide strong support for previous findings on the role of anxiety sensitivity, fear of pain, and psychological distress among individuals with chronic pain (Asmundson & Norton, 1995; Asmundson & Taylor, 1996; Asmundson, Norton, & Veloso, 1999; Hursey & Jacks, 1992). Previous studies have established and confirmed the role of these factors primarily in samples of headache sufferers from the general population (Asmundson, Norton, & Veloso, 1999; Hursey & Jacks, 1992) and individuals with other chronic pain conditions (Asmundson & Norton, 1995; Asmundson & Taylor, 1996; Vlaeyen & Linton, 2000). The current study, while not focused primarily on the relationships among these variables, provides similar data confirming the role of these psychological factors in HIV patients who also have chronic headaches, a population that remains understudied from a psychological perspective.

Patient Groupings. Clearly, two large but different groups of HIV patients participated in this study: one group that was relatively healthy, optimistic, accepting, and very informed about their illness and their own health status, and another group that was relatively unhealthy, pessimistic, less careful and knowledgeable about their health, and with a poorer treatment prognosis. The latter group tended to consistently report that they experienced both frequent and severe headaches as well as high levels of emotional distress, and catastrophized about their pain

and illness. The former group not only had better HIV lab data results, but they also denied occurrence of problematic headaches, reported much lower levels of emotional distress, and exhibited a greater knowledge of the HIV disease process and the importance of compliance with HAART, the latter of which was evident through the responses of patients during data collection and the clinical observations of the researchers. According to Gifford and Groessl (2002), the most successful self-managing HIV patients are knowledgeable about their medical profile, the HIV illness, and advances in treatment; motivated to change and sustain the necessary behaviors for treatment compliance; and engaged in behaviors to learn, practice, and adapt to new behavioral skills in order to set and achieve goals, rely on social supports, and communicate effectively with treatment providers.

The stark division between the two HIV groups is consistent with findings from HIV treatment studies. Vervoort et al. (2009) studied predictors of HAART adherence based on the importance of accepting or not accepting the HIV diagnosis and necessity of treatment compliance. Patients who successfully adhered to HAART were those who engaged in a decision-making process to adhere to medication, who had thorough knowledge of medication and of the importance of adherence, and who exhibited positive behaviors and thoughts surrounding HAART, the HIV infection, and social support. Judd et al. (2000) confirmed the importance of adherence to HAART in a 2-year study that tracked changes in depression during treatment. In that study, mean CD4 counts rose while self-reported symptoms of depression decreased, as did the number of patients who endorsed concerns about their health. These data indicate that adherence to HAART not only improves HIV parameters, but is effective also at improving comorbid mood symptoms and health perceptions. Data from these studies confirm that adherence to HAART is multiply determined by a variety of patient factors and is associated

with better overall health and less psychological distress. Although the current study did not investigate HAART compliance, the patient subgroupings and response styles are consistent with the notion that those HIV patients whose illness is well-controlled with HAART have much better specific and global outcomes than do their sicker counterparts.

Psychological variables as predictors of headache. The hierarchical regressions performed on this data set yielded important results. Age and gender were found to be largely unrelated to headache-related disability among this sample of individuals with HIV/AIDS. Instead, CD4 count and the psychological variables were important predictors of disability. Importantly, comorbid symptoms of depression and anxiety afforded significant prediction of disability beyond that accounted for by CD4 counts and the demographic variables. After accounting for depression and anxiety, each psychological variable (e.g., catastrophizing, anxiety sensitivity, fear of pain) independently resulted in significant prediction of headache-related disability, though the variances accounted for were far smaller than those explained by comorbid depression and anxiety.

Given that cognitive psychological factors have never been addressed in research on HIV-infected individuals with headache, this set of results is particularly important. The findings of this study indicate that although the overriding influence of depression and anxiety are of primary importance, the influence of maladaptive cognitive processing remains an important predictor of headache-related disability. Although maladaptive cognitive processing is common in both depression and anxiety, as evidenced in Leung and Poon (2001), such symptoms in this study independently predicted headache-related disability specifically. These findings are consistent with those from previous studies, which have noted a strong relationship between catastrophizing and pain-related disability among patients with arthritis (Keefe et al., 2004) and

migraine patients without HIV (Holroyd et al., 2007). Practically, these results suggest that specific thought patterns and cognitive distortions should be addressed in treatment, perhaps in turn improving the psychological functioning and the impact of headache among individuals with HIV.

Practical Implications

One of the primary motives for this research was to provide meaningful, practical data to primary care physicians, infectious disease specialists, neurologists, HIV health care teams, and individuals diagnosed with HIV about headache patterns and related psychological functioning. The ultimate goal was to provide more detailed information about headache symptomatology in an attempt to answer questions about "typical" headaches in HIV/AIDS patients and the relative importance of various psychological factors that are often overlooked (Marchioni et al., 2006). The approach taken in this study was geared at displaying a transdisciplinary biopsychosocial model of collaboration between health professions (Suls & Rothman, 2004) in an attempt to discover more about HIV and its complications pertaining to headache.

Among others, one practical benefit from the results of this study is the ability to present the data in the form of feedback to HIV-infected individuals. Accurate epidemiological data leads to improved illness management. When first diagnosed, an individual usually receives information about the importance of medication compliance. The results of this study highlight the importance of medication compliance due to strong correlations between CD4 counts, headache frequency, and psychological dysfunction, and in consideration of extant literature on HAART compliance and positive physical and mental health outcomes. By looking at these implications in such a broad manner, it seems practical for medical providers to attempt to

convince HIV-infected individuals that the most important factor is not the presence of HIV, but how the patient responds to the illness, its complications, and its management.

The purpose of this applied research was to enhance understanding of headaches in the HIV/AIDS population in order to improve patient education and treatment strategies. Based on the results, the headache suffering HIV population would benefit from an increase in referrals for mental health treatment. In order to optimize such transdisciplinary treatment opportunities, mental health professionals would benefit from learning about the modern nature of HIV, its relationship with headaches, and the vicious cycle the two factors participate in with psychological problems. With a combination of rigid adherence to HAART through improved self-management and consistent psychotherapy (when indicated), the negative impact of HIV could potentially be greatly reduced through increases in CD4 counts, improved overall mental health, and education in ways to prevent and cope with headaches.

Another important benefit of clarifying the prevalence of headache and its "typical" presentation in HIV patients is a potential reduction in medical costs associated with sometimes unnecessary invasive medical procedures (e.g., lumbar punctures, MRIs, CT scans) for which patients and the health care system incur high costs. These defensive practice techniques have traditionally been employed subsequent to complaints of head pain to rule out opportunistic infections such as meningitis, sinusitis, or neurosyphilis among individuals with HIV/AIDS (Many, 2009). Our data suggest that symptom patterns and causes of headache in HIV patients likely have changed over the years, coinciding with the development and proliferation of HAART that has reduced disease burden and improved the prognosis of living with HIV. With the added knowledge about typical patterns of headache in HIV-infected individuals, suggesting

that the majority of these headaches are consistent with migrainous subforms and not opportunistic infections, reductions in these costly medical procedures may result.

Limitations and Directions for Future Research

The most prominent limitation in this study was the length of the questionnaire packet administered in the context of high-traffic medical clinics, which in conjunction with the interview sometimes took more than an hour to complete. Screening with the WRAT-4 was initiated to increase validity among those of low reading levels, but added to the burden involved in data collection. Both in oral and written administration, some participants seemed to develop a bifurcated response style characterized by either a "defensive pride" in which they reported a healthy medical profile and denied comorbid problems, or in which they endorsed virtually all problems associated with HIV, headache, and emotional distress. These extremes in response styles embody the distinct HIV patient groups articulated by Vervoort et al. (2009) and are reflected in the highly significant group differences. Nonetheless, continued replication with similar samples is warranted given the extreme group differences observed.

Another possible confounding variable was that the comparisons of data gathered from oral versus paper administration could not be analyzed. A significant portion of the sample in this study was not able to read above a 6th grade level and were thus administered the informed consent documents and lengthy questionnaire measures orally, which further increased administration time. Participant reading level and readability forms and measures is an often-ignored factor in psychological and health care research, yet is vital to address in the context of medical settings, particularly those in underserved, rural, and low income areas. Although group differences could not be analyzed on the basis of oral versus self-administered versions of data collection, previous studies have found no difference in mode of self-report administration

(Edwards, Holmes, & Carvajal, 1998; Hahn, Rao, Cella, & Choi, 2008; Kendrick & Hatzenbuehler, 2006; Weinberger, Oddone, Samsa, & Landsman, 1996). It appeared to the researchers that the length of the questionnaire was more explanatory in accounting for more the extreme response styles than was the method of administration. Regardless, when evaluating the results of this study, readers should consider these factors.

Another limitation was the absence of neuroimaging procedures typically used to rule out secondary headache causes. As previously discussed, earlier studies in this area employed the use of neuroimaging in conjunction with self-reported headache symptoms and medical records to exclude secondary causes of headache. However, due to the scope and goals of the current study, such procedures were not employed. Our conclusions about secondary causes were informed by review of patient medical records, patient self-report of past or current encephalitic infections, and the characteristics of reported headache symptoms. Although our results suggest that most HIV patients with headache problems do not show evidence of secondary causes (or merit neuroimaging), future studies confirming these findings with neuroimaging techniques are clearly needed for more definitive confirmation. Future research could model the methodology of the current study while adding neuroimaging procedures to definitively rule out opportunistic encephalic infections in the modern HIV-infected individual. Although such a study would come at high cost, convincing results could lead to an overall reduction in future costs both to the HIV patient and society as a whole.

Another minor limitation was that the population of this study consisted of a relatively small number of individuals with preexisting headaches. Given the nature of this study and its primary goals, this was not of great concern, but nonetheless limited conclusions pertaining to changes in headache patterns subsequent to HIV diagnosis. Because little research to date has

focused on this issue, future studies should look at HIV-infected individuals with headaches prior to diagnosis, preferably using longitudinal designs.

In addition to the directions mentioned previously, future research in this area should include treatment studies that focus on aggressively treating HIV among patients with comorbid headache. Such studies would include both HAART and mental health treatment, with close monitoring of changes not only in HIV-related lab data but in headache characteristics and psychological functioning. In addition to extensive psycho-education regarding HIV, mental health treatment should focus on addressing comorbid psychopathology, improving coping skills, and teaching skills in behavioral migraine management (e.g., stress management, trigger identification, relaxation training). Other treatment studies could focus on the role of triptans and other migraine-specific medications in improving headache and quality of life among these individuals. The results of such studies could be used to develop protocols by which practitioners from multiple disciplines could efficiently teach newly identified HIV-infected individuals about medication compliance, managing comorbid headaches, and maintaining a healthy emotional state.

In conclusion, research on headache among individuals with HIV has been sporadic and limited, which is striking given both its well-documented prevalence among this population and significant treatment advancements coinciding with the success of HAART. Our data suggest that slightly more than half of all patients with HIV have problematic headaches; that these headaches are highly frequent and typically of a migrainous presentation (with some atypical features); that their presence, frequency, severity, and resulting disability is strongly related to the severity (but not duration) of HIV/AIDS; and that these headaches are strongly associated with numerous psychological variables.

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APPENDICES

APPENDIX A: DEMOGRAPHIC QUESTIONNAIRE AND MEDICAL RECORDS

Demographic Questionnaire and Medical Records

Participant ID: _____

Date: ____/____/_____ Study/Clinic Site (circle one): 1. UAB 2. MAO

Age: _____

Most recent CD4 cell count: _____ **Date Taken**: ____/____/_____

Most recent viral load: _____ **Date Taken**: ____/____/_____

Date of HIV diagnosis: ____/____/_____

Gender (Circle One): 1. Male 2. Female 3. Transgender

Relationship Status (Circle One): 1. Single 2. Married 3. Divorced

4. Separated 5. Widowed 6. Unknown

Race (Circle One): 1. African American 2. Asian 3. Hispanic/Latino

4. Native American/Pacific Islander 5. Caucasian

6. Other:_____

Sexual Orientation (circle one)**:** 1. Heterosexual 2. Homosexual 3. Bisexual

Highest Level of Education (Circle One):

1^{st} 2^{nd} 3^{rd} 4^{th} 5^{th} 6^{th} 7^{th} 8^{th} 9^{th} 10^{th} 11^{th}

12) H.S. Diploma/GED 13) Some College

14) College Diploma 15) Some Graduate School

16) Graduate Degree

Medications Taken by Patient:

Medications	Dosage	Frequency

Medical Diagnoses:

_____

_____

_____

_____

_____

_____

_____

APPENDIX B: STRUCTURE DIAGNOSTIC INTERVIEW FOR HEADACHE (SDIH)

Structured Diagnostic Interview for Headache – Revised (Brief Version)

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.

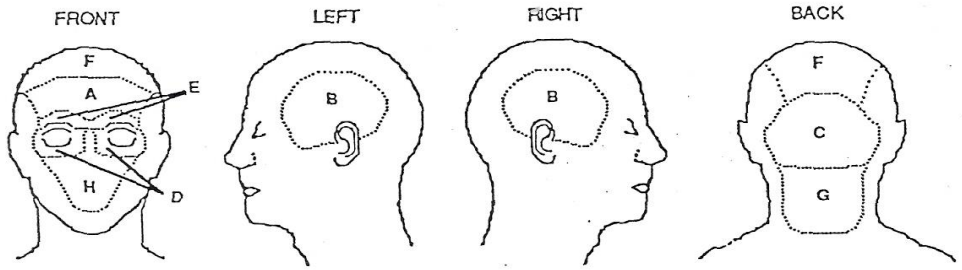
1. Do you ever get headaches? ☐ Yes ☐ No

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

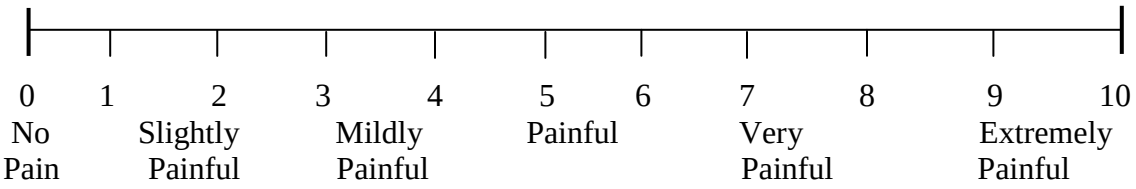
2. Select all pain locations that apply to this type of headache: ***(You must check at least one)***

☐ 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)



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



On average, how many **headache-free days** do you have each <u>week</u>? _____ days

5. Which of the following symptoms are a "predominant feature" of this headache type (presume that the headache is untreated)?

<u>Pain Location</u> ***(Select <u>only</u> one)***: ☐ Unilateral ☐ Not Unilateral
<u>Pain Features</u> ***(Select <u>only</u> one)***: ☐ Pulsating ☐ Pressing/Tightening (non-pulsating)
☐ Other:____

*If pressing/tightening (non-pulsating) in quality:
Could this headache be best described as a dull ache that occurs on both sides of the head?
☐ Yes ☐ No

6. How often does the patient experience this type of headache pain? ____ days per month

7. How long have these headaches been occurring at this rate? ____ Months Years

Did these headaches begin before or after you were diagnosed with HIV?
_____ Before HIV diagnosis _____After HIV diagnosis

*If these headaches began before you were diagnosed…
How often did they occur? _____ days with headache per month
How intense was the pain, on average? _____ (1-10 scale used earlier)

Since you were diagnosed with HIV, have you ever also been diagnosed with:

_____ Meningitis (Month/year of diagnosis: _____)
_____ Encephalitis (Month/year of diagnosis: _____)
_____ Other severe infection (Month/year of diagnosis: _____)

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 ONLY if patient reports headaches always last at least 7 days in duration). ***(Indicate duration in minutes)***

☐ 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)? ☐ 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 begin? ☐ 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

Complete the respective Appendices below if the patient "YES" to #13 (Aura Symptoms—Appendix 1), #14 (Cluster headache Symptoms—Appendix 2), #15-C (Medication Overuse Symptoms—Appendix 3), or #16 (Posttraumatic Headache Symptoms—Appendix 4)

APPENDIX 1	Migraine Aura Symptoms

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

2. What best describes the aura symptoms? ***(Select all that apply)***

☐ At least one aura symptom develops gradually over more than 4 minutes, **AND/OR** 2 or more symptoms occur in succession over 4 minutes

☐ Each aura symptom lasts longer than 4 minutes but less than 60 minutes

☐ Headache begins during aura **OR** follows aura with a headache-free interval of less than 60 minutes

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

X	SYMPTOM	X	SYMPTOM
☐	Loss of sight (scotoma)	☐	Uncoordinated movements (ataxia)
☐	Scintillation (rapidly oscillating pattern)	☐	Dizziness (vertigo)
☐	Blurred vision	☐	Ringing in ears (tinnitus)
☐	Fortification spectra (zig-zag	☐	Decreased hearing acuity
☐	Double vision	☐	Decreased level of consciousness
☐	Tingling or numbness (paresthesias)	☐	Aphasia or unclassifiable speech
☐	Weakness (paresis)	☐	Poorly articulated speech (dysarthria)

APPENDIX 2	Cluster Headache Symptoms

1. Have the headaches occurred in cluster periods? ☐ Yes ☐ No
   If **YES**, complete #1a
   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
☐	Red eyes (conjunctival injection)	R L	☐	Forehead and facial sweating	R L
☐	Tearing of the eyes (lacrimation)	R L	☐	Pupillary constriction (miosis)	R L
☐	Nasal congestion	R L	☐	Drooping eyelids (ptosis)	R L
☐	Runny nose (rhinorrhoea)	R L	☐	Eyelid swelling (oedema)	R L
☐	Restlessness or agitation		☐		

APPENDIX 3	Medication-Overuse Headache Symptoms

1. Has the patient withdrawn from the overused medication? ☐ Yes ☐ No
   If **YES**, complete #1a and #1b
   If **NO**, skip to #2

1a. Did headache resolve or revert to its previous pattern within 2 months after discontinuation of overused medication? ☐ Yes ☐ No

1b. Has medication overuse ceased within the last 2 months, but headache has not resolved or reverted back to its previous pattern? ☐ Yes ☐ No

2. Has intake of ergotamine, triptan, opioid **OR** combination of ergotamine, triptan, opioid, or analgesic occurred on 2 or more days per week, for 10 or more days per month, for greater than 3 months ***(Must not have combination overuse of any single class alone)***? ☐ Yes ☐ No
If **YES**, indicate drug(s): ☐ ergotamine ☐ triptan ☐ opioid ☐ analgesic ___

3. Has the patient's intake of analgesic occurred on 2 or more days per week, for 15 or more days per month, for greater than 3 months? ☐ Yes ☐ No
   If **YES**, indicate drug: _____

4. Has the patient's intake of combination analgesics occurred on 2 or more days per week, for 10 or more days per month, for greater than 3 months? ☐ Yes ☐ No
If **YES**, indicate drugs: _____

5. Has the patient's intake of medication other than ergotamine, triptan, analgesic, or opioid occurred on a regular basis for greater than 3 months? ☐ Yes ☐ No
If **YES**, indicate drug: _____

APPENDIX 4	Post-Traumatic Headache Symptoms

1. Was there a loss of consciousness associated with head trauma? ☐ Yes ☐ No
If **YES**, complete #1a
If **NO**, skip to #2

1a. What was the duration of unconsciousness?____ m h d ***(Indicate duration in minutes, hours, or days)***

2. Is head injury attributed to whiplash? ☐ Yes ☐ No
If **YES**, skip #5 through #8
If **NO**, complete #3 through #8

3.Did headache develop within 7 days after head trauma (or after regaining consciousness)? ☐ Yes ☐ No

4. 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

5. Did coma develop? ☐ Yes ☐ No
If **YES**, indicate severity on Glasgow Coma Scale:
☐ GCS <13 *[moderate/severe]* ☐ GCS ≥13 *[mild]*

6. Did post-traumatic amnesia develop and continue for longer than 48 hours? ☐ Yes ☐ No

7. Did symptoms/signs develop diagnostic of a concussion? ☐ Yes ☐ No

8. Were abnormal neuroimaging results attained suggestive of a traumatic brain lesion? ☐ Yes ☐ No

Appendix C: BRIEF HEADACHE SCREEN

Brief Headache Screen

Check the best answer for each question below.

1. How often do you get *severe* headaches (difficult or unable to continue normal function)?
   _____ Daily or near daily
   _____ 3-4 days per week
   _____ Between 2 days per week and 2 days per month
   _____ Once a month or less
   _____ Almost never

2. How often do you get mild or less severe headaches?
   _____ Daily or near daily
   _____ 3-4 days per week
   _____ Between 2 days per week and 2 days per month
   _____ Once a month or less
   _____ Almost never

3. How often do you take pain relievers, or any medication to relieve headache symptoms?
   _____ Daily or near daily
   _____ 3-4 days per week
   _____ Between 2 days per week and 2 days per month
   _____ Once a month or less
   _____ Almost never

4. How often do you miss some work or leisure time because of headache?
   _____ Daily or near daily
   _____ 3-4 days per week
   _____ Between 2 days per week and 2 days per month
   _____ Once a month or less
   _____ Almost never

5. Are you satisfied with the current medication you use to relieve your headaches?
   _____ Yes
   _____ No

6. Are you taking daily prescription medication to prevent headaches?
   _____ Yes
   _____ No

7. If no, do your headaches trouble you enough to take daily preventive medication?
   _____ Yes
   _____ No

APPENDIX D: HEADACHE MANAGEMENT SELF-EFFICACY SCALE

HMSE

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

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 *mild* 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 E: MIGRAINE DISABILITY ASSESSMENT (MIDAS)

MIDAS

Write in your answer for each question below.

1. On how many days in the last 3 months did you miss work or school because of headaches?

_____ days

2. How many days in the last 3 months was your productivity at work or school reduced by half or more because of your headaches?
(Do not include days you counted in question 1 where you missed work or school.)

_____ days

3. On how many days in the last 3 months did you not do household work because of your headaches?

_____ days

4. How many days in the last 3 months was your productivity in household work reduced by half or more because of your headaches?
(do not include days you counted in question 3 where you did not do household work.)

_____ days

5. On how many days in the last 3 months did you miss family, social or leisure activities because of your headaches?

_____ days

6. On how many days in the last 3 months did you have any headache?
(If a headache lasted more than 1 day, count each day.)

_____ days

7. On a scale of 0 - 10, on average how painful were these headaches?
(where 0 = no pain at all and 10 = pain as bad as it can be.)

_____

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

HIT-6

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

APPENDIX G: ANXIETY SENSITIVITY INDEX – THIRD EDITION (ASI-3)

ASI-3

Please circle the number that best corresponds to how much you agree with each item. If any items concern something that you have never experienced (e.g., fainting in public), then answer on the basis of how you think you might feel *if you had* such an experience. Otherwise, answer all items on the basis of your own experience. Be careful to circle only one number for each item and please answer all items.

	Very little	**A little**	**Some**	**Much**	**Very much**
1. It is important for me not to appear nervous.	0	1	2	3	4
2. When I cannot keep my mind on a task, I worry that I might be going crazy.	0	1	2	3	4
3. It scares me when my heart beats rapidly.	0	1	2	3	4
4. When my stomach is upset, I worry that I might be seriously ill.	0	1	2	3	4
5. It scares me when I am unable to keep my mind on a task.	0	1	2	3	4
6. When I tremble in the presence of others, I fear what people might think of me.	0	1	2	3	4
7. When my chest feels tight, I get scared that I won't be able to breathe properly.	0	1	2	3	4
8. When I feel pain in my chest, I worry that I'm going to have a heart attack.	0	1	2	3	4
9. I worry that other people will notice my anxiety.	0	1	2	3	4
10. When I feel "spacey" or spaced out I worry that I may be mentally ill.	0	1	2	3	4
11. It scares me when I blush in front of people.	0	1	2	3	4
12. When I notice my heart skipping a beat, I worry that there is something seriously wrong with me.	0	1	2	3	4
13. When I begin to sweat in a social situation, I fear people will think negatively of me.	0	1	2	3	4
14. When my thoughts seem to speed up, I worry that I might be going crazy.	0	1	2	3	4
15. When my throat feels tight, I worry that I could choke to death.	0	1	2	3	4
16. When I have trouble thinking clearly, I worry that there is something wrong with me.	0	1	2	3	4
17. I think it would be horrible for me to faint in public.	0	1	2	3	4
18. When my mind goes blank, I worry there is something terribly wrong with me.	0	1	2	3	4

APPENDIX H: PAIN ANXIETY SYMPTOMS SCALE (PASS-20)

PASS-20

Individuals who experience pain develop different ways to respond to that pain. We would like to know what you do and what you think about when in pain. Please use the rating scale below to indicate how often you engage in each of the following thoughts or activities. Circle any number from 0 (NEVER) to 5 (ALWAYS) for each item.

NEVER ALWAYS

1. I think that if my pain gets too severe, it will never decrease……. . .0 1 2 3 4 5
2. When I feel pain I am afraid that something terrible will happen……0 1 2 3 4 5
3. I go immediately to bed when I feel severe pain …..…..…….0 1 2 3 4 5
4. I begin trembling when engaged in activity that increases pain……0 1 2 3 4 5
5. I can't think straight when I am in pain ……. …… 0 1 2 3 4 5
6. I will stop any activity as soon as I sense pain coming on ……..……0 1 2 3 4 5
7. Pain seems to cause my heart to pound or race ……..…….. 0 1 2 3 4 5
8. As soon as pain comes on I take medication to reduce it ……. …….0 1 2 3 4 5
9. When I feel pain I think that I may be seriously ill ……. …… 0 1 2 3 4 5
10. During painful episodes it is difficult for me to think of anything else besides the pain…….0 1 2 3 4 5
11. I avoid important activities when I hurt ……... 0 1 2 3 4 5
12. When I sense pain I feel dizzy or faint ……..0 1 2 3 4 5
13. Pain sensations are terrifying ……0 1 2 3 4 5
14. When I hurt I think about the pain constantly ……. 0 1 2 3 4 5
15. Pain makes me nauseous (feel sick)……. 0 1 2 3 4 5
16. When pain comes on strong I think I might become paralyzed or more disabled ……. 0 1 2 3 4 5
17. I find it hard to concentrate when I hurt …….. 0 1 2 3 4 5
18. I find it difficult to calm my body down after periods of pain …….0 1 2 3 4 5

19. I worry when I am in pain ……0 1 2 3 4 5

20. I try to avoid activities that cause pain ……0 1 2 3 4 5

Appendix I: PAIN CATASTROPHIZING SCALE

Pain Catastrophizing Scale

Instructions:
We are interested in the types of thoughts and feelings that you have when you are in pain. Listed below are thirteen statements describing different thoughts and feelings that may be associated with pain. Using the following scale, please indicate the degree to which you have these thoughts and feelings when you are experiencing pain.

RATING	**0**	**1**	**2**	**3**	**4**
MEANING	Not at all	To a slight degree	To a moderate degree	To a great degree	All the time

	When I'm in pain...	RATING
1.	I worry all the time about whether the pain will end.	_____
2.	I feel I can't go on.	_____
3.	It's terrible and I think it's never going to get any better.	_____
4.	It's awful and I feel that it overwhelms me.	_____
5.	I feel I can't stand it anymore.	_____
6.	I become afraid that the pain will get worse.	_____
7.	I keep thinking of other painful events.	_____
8.	I anxiously want the pain to go away.	_____
9.	I can't seem to keep it out of my mind.	_____
10.	I keep thinking about how much it hurts.	_____
11.	I keep thinking about how badly I want the pain to stop.	_____
12.	There's nothing I can do to reduce the intensity of the pain.	_____
13.	I wonder whether something serious may happen.	_____

APPENDIX J: DEPRESSION ANXIETY STRESS SCALE (DASS)

DASS 42

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.**

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 a considerable degree, or a good part of the time.

3 Applied to me very much, or most of the time.

1. I found myself getting upset by quite trivial things	**0**	**1**	**2**	**3**
2. I was aware of dryness of my mouth	**0**	**1**	**2**	**3**
3. I couldn't seem to experience any positive feeling at all	**0**	**1**	**2**	**3**
4. I experienced breathing difficulty (e.g., excessively rapid breathing, breathlessness in the absence of physical exertion)	**0**	**1**	**2**	**3**
5. I just couldn't seem to get going	**0**	**1**	**2**	**3**
6. I tended to over-react to situations	**0**	**1**	**2**	**3**
7. I had a feeling of shakiness (e.g., legs going to give way)	**0**	**1**	**2**	**3**
8. I found it difficult to relax	**0**	**1**	**2**	**3**
9. I found myself in situations that made me so anxious I was most relieved when they ended	**0**	**1**	**2**	**3**
10. I felt that I had nothing to look forward to	**0**	**1**	**2**	**3**
11. I found myself getting upset rather easily	**0**	**1**	**2**	**3**
12. I felt that I was using a lot of nervous energy	**0**	**1**	**2**	**3**
13. I felt sad and depressed	**0**	**1**	**2**	**3**
14. I found myself getting impatient when I was delayed in any way (e.g., lifts, traffic lights, being kept waiting)	**0**	**1**	**2**	**3**
15. I had a feeling of faintness	**0**	**1**	**2**	**3**
16. I felt that I had lost interest in just about everything	**0**	**1**	**2**	**3**
17. I felt I wasn't worth much as a person	**0**	**1**	**2**	**3**
18. I felt that I was rather touchy	**0**	**1**	**2**	**3**
19. I perspired noticeably (e.g., hands sweaty) in the absence of high temperatures or physical exertion	**0**	**1**	**2**	**3**
20. I felt scared without any good reason	**0**	**1**	**2**	**3**

21. I felt that life wasn't worthwhile	**0**	**1**	**2**	**3**
22. I found it hard to wind down	**0**	**1**	**2**	**3**
23. I had difficulty in swallowing	**0**	**1**	**2**	**3**
24. I couldn't seem to get any enjoyment out of the things I did	**0**	**1**	**2**	**3**
25. 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)	**0**	**1**	**2**	**3**
26. I felt down-hearted and blue	**0**	**1**	**2**	**3**
27. I found that I was very irritable	**0**	**1**	**2**	**3**
28. I felt I was close to panic	**0**	**1**	**2**	**3**
29. I found it hard to calm down after something upset me	**0**	**1**	**2**	**3**
30. I feared that I would be "thrown" by some trivial but unfamiliar task	**0**	**1**	**2**	**3**
31. I was unable to become enthusiastic about anything	**0**	**1**	**2**	**3**
32. I found it difficult to tolerate interruptions to what I was doing	**0**	**1**	**2**	**3**
33. I was in a state of nervous tension	**0**	**1**	**2**	**3**
34. I felt I was pretty worthless	**0**	**1**	**2**	**3**
34. I was intolerant of anything that kept me from getting on with what I was doing	**0**	**1**	**2**	**3**
36. I felt terrified	**0**	**1**	**2**	**3**
37. I could see nothing in the future to be hopeful about	**0**	**1**	**2**	**3**
38. I felt that life was meaningless	**0**	**1**	**2**	**3**
39. I found myself getting agitated	**0**	**1**	**2**	**3**
40. I was worried about situations in which I might panic and make a fool of myself	**0**	**1**	**2**	**3**
41. I experienced trembling (e.g., in the hands)	**0**	**1**	**2**	**3**
42. I found it difficult to work up the initiative to do things	**0**	**1**	**2**	**3**

APPENDIX K: CONSENT TO PARTICIPATE IN EXPERIMENTAL STUDY

Consent to Participate in an Experimental Study
Title: Psychological Factors in HIV-Related Headaches

Investigator
Kale E. Kirkland, M.A.
Department of Psychology
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Faculty Advisor
Todd A. Smitherman, Ph.D.
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Peabody Hall
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Description
Many individuals with HIV experience headaches. We are asking you to participate in a research study that will evaluate the relationship between your headaches and psychological factors. This study requires you to undergo a brief interview about your headaches and to complete a packet of questionnaires related to depression, anxiety, stress, and pain. The administration of the instruments will take approximately 30-45 minutes to be completed. We will also be gathering some data from your medical records, including the date of your HIV diagnosis and your most recent CD4 and viral load counts.

Risks and Benefits
Your participation in this study is entirely voluntary. Interviews will be conducted in a private setting to assure your privacy. You may feel uncomfortable answering questions about symptoms of depression or anxiety, but your data will remain entirely confidential. You will be assigned a code number so that your name is not associated with any of your data. If your responses suggest serious thoughts of suicide, we will arrange for you to speak with an available clinician or escort you to a nearby emergency room, if appropriate. You may benefit from taking part in this study, as we will provide you with some educational materials for coping with headaches, depression/anxiety, and HIV. Participants reporting significant symptoms of depression and anxiety will also be provided contact information for local mental health providers. There are no other benefits to participation. However, this study may help us better understand headache patterns and associated factors in individuals with HIV.

Cost and Payments
This study will take approximately 30-45 minutes of your time (15 minutes for the interview, 15-30 minutes for the questionnaires), most of which will occur while you are waiting to see your doctor. There are no other costs for helping us with this study.

Confidentiality
We will not put your name on any of your tests. The only information that will be on your test materials will be your assigned code number. This code number will be linked to your name, your gender (whether you are male or female), your ethnicity, and your age. The list of code numbers will be kept at all times within a locked room and will be provided to no one other than the study personnel. The code list will be destroyed at the conclusion of the study. Any publications or presentations resulting from this study will not include any names or information that could lead to your identification. Therefore, we do not believe that you can be identified from any of your tests.

Right to Withdraw
You do not have to take part in this study. If you start the study and decide that you do not want to finish, all you have to do is to tell your physician or the researchers. We want to assure you that whether or not you choose to participate or to withdraw will not affect your care at the clinic. No medical treatments will be added or withheld based on participation in this study. The researchers may terminate your participation in the study without regard to your consent and for any reason, such as protecting your safety and protecting the integrity of the research data.

Protected Health Information
Protected health information is any personal health information which identifies you in some way. The data collected in this study includes your age, date of HIV diagnosis, most recent CD4 cell count and date taken, most recent viral load and date taken, sexual orientation, level of education, **relationship status, a list of the medications you take, and other diagnoses**. A decision to participate in this research means that you agree to the use of your health information for the study described in this form. This information will not be released beyond the purposes of conducting this study. The information collected for this study will be kept until the study is complete. While this study is ongoing you may not have access to the research information, but you may request it after the research is completed.

IRB Approval
This study has been reviewed by The University of Mississippi's Institutional Review Board (IRB). The IRB has determined that this study fulfills the human research subject protections obligations required by state and federal law and University policies. If you have any questions, concerns, or reports regarding your rights as a participant of research, please contact the IRB at (662) 915-7482.

Statement of Consent
I have read the above information. I have been given a copy of this form. I have had an opportunity to ask questions, and I have received answers. I consent to participate in the study.

_____
Signature of Participant

_____
Signature of Investigator

NOTE TO PARTICIPANTS: DO <u>NOT</u> SIGN THIS FORM
IF THE IRB APPROVAL STAMP ON THE FIRST PAGE HAS EXPIRED.

APPENDIX L: CONSENT TO RELEASE INFORMATION FROM MEDICAL RECORDS

Consent to Release Information from Medical Records

Investigator
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Faculty Advisor
Todd A. Smitherman, Ph.D.
Department of Psychology
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University of Mississippi
(662) 915-1825

Description
Signing this form will authorize researchers to obtain specific information from your medical records: Age, date of HIV diagnosis, most recent CD4 cell count and date taken, most recent viral load and date taken, sexual orientation, level of education, **relationship status, a list of the medications you take, and other diagnoses.**

Confidentiality
Although the medical records used will be linked to your name, we will be recording medical data of interest on a demographic sheet that will not have identifiers or your name; you will instead be assigned a code number to ensure confidentiality. This code number will be linked to your name, your gender (whether you are male or female), your ethnicity, and your age. The list of code numbers will be kept at all times within a locked room and will be provided to no one other than the study personnel. The code list will be destroyed at the conclusion of the study. Any publications or presentations resulting from this study will not include any names or information that could lead to your identification. Therefore, we do not believe that you can be identified from any of your information.

Protected Health Information
Protected health information is any personal health information which identifies you in some way. The data collected in this study includes your age, date of HIV diagnosis, most recent CD4 cell count and date taken, most recent viral load and date taken, sexual orientation, level of education, **relationship status, a list of the medications you take, and other diagnoses**. A decision to participate in this research means that you agree to the use of your health information for the study described in this form. This information will not be released beyond the purposes of conducting this study. The information collected for this study will be kept until the study is complete. While this study is ongoing you may not have access to the research information, but you may request it after the research is completed.

IRB Approval
This study has been reviewed by The University of Mississippi's Institutional Review Board (IRB). The IRB has determined that this study fulfills the human research subject protections obligations required by state and federal law and University policies. If you have any questions, concerns, or reports regarding your rights as a participant of research, please contact the IRB at (662) 915-7482.

Statement of Consent
I have read the above information. I have been given a copy of this form. I have had an

opportunity to ask questions, and I have received answers. I consent to release information from my medical records as part of my participation in this study.

Signature of Participant

Signature of Investigator

NOTE TO PARTICIPANTS: DO NOT SIGN THIS FORM
IF THE IRB APPROVAL STAMP ON THE FIRST PAGE HAS EXPIRED.

VITA

Kale Edney Kirkland was born April 17, 1984, in Montgomery, Alabama. He graduated in 2002 with honors from Saint James High School in Montgomery, Alabama. From 2002 to 2005, Kale majored in Psychology at Auburn University, where he graduated Summa Cum Laude with a Bachelor of Arts degree (3.905 Cumulative GPA).

Upon graduation from Auburn University, Kale was accepted and enrolled in the clinical psychology doctoral program at the University of Mississippi, where he completed his Master of Arts work with his major professor, David S. Hargrove, Ph.D. Upon completion of his thesis, Kale began working under a new major professor, Todd A. Smitherman, Ph.D., at which point the ideas for this project was formulated. Kale is currently completing his pre-doctoral internship with the Department of Justice Bureau of Prisons at the Federal Correctional Institution Fort Worth, which he will complete in August 2011.