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Factors Associated with a Longer Length of Stay and Minimal Improvement in an Inpatient Headache Unit

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FACTORS ASSOCIATED WITH A LONGER LENGTH OF STAY AND MINIMAL IMPROVEMENT IN AN INPATIENT HEADACHE UNIT

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Dissertation Approval

This is to certify that the thesis presented to us by Kristen Wenrich on the 29th day of November, 2011, in partial fulfillment of the requirements for the degree of Doctor of Psychology, has been examined and is acceptable in both scholarship and literary quality.

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Abstract

The purpose of the present study is to identify psychological factors that predict a longer length of stay (LOS) and minimal improvement in a headache inpatient unit. Research shows that some psychological factors associated with headache disorders, such as anxiety, depression, and maladaptive coping skills, can complicate the disorder. The present study theorized that psychological factors that complicate the headache disorder would predict a longer LOS and minimal improvement. The present study used a quasi-experimental, prospective, cross-sectional survey research design, with multiple regression analyses. There was a total of 51 completed protocols. Of those completed protocols, 78% of participants were age 35 years and older. Of consenting participants, 82% were Caucasian women. Results indicated that the hypothesized model to predict LOS and minimal improvement was not significant. Paired-samples *t*-test analyses indicated that there were significant reductions of BPI-SF interference and severity scores after inpatient treatment, presenting a basis for further research involving comparison groups.

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Chapter One: Introduction

Statement of the Problem

Headache for most people, although a nuisance, is usually infrequent and resolvable. However, for a proportion of the population, headache is a chronic disorder that is disabling, significantly impeding quality of life (Lipton & Newman, 2003). According to Jensen and Stovner (2008), "the scope and scale of the burden of headache is underestimated, and headache disorders are universally under-recognized and undertreated" (p. 354). Globally, of the adult population, 47% have a general headache condition, 10% have a migraine disorder, 38% have Tension-Type Headache, and 3% have a chronic headache disorder (Jensen & Stovner, 2008). Disability from Tension-Type Headache is greater than that from migraine because of its higher prevalence, despite migraine being considered the ninth most costly neurological condition for both genders, and the third most costly for women (Jensen & Stovner, 2008). Headache is one of the 10 most disabling disorders for both genders, and the fifth most disabling for women (Jensen & Stovner, 2008).

Prevalence rates for debilitating headache disorders vary depending on demographic characteristics, such as age, region of the world, and gender (Diamond et al., 2007). A study conducted from 1992 to 2001 found that visits for headaches in emergency departments occurred with average rates of 10.9 for women, versus 4.6 for men, per 1,000 persons in the United States population (Goldstein, Camargo, Pelletier, & Edlow, 2006). In terms of race, Goldstein et al. (2006) found that the rate was 7.5 for Caucasian patients, 12.0 for African American patients, and 2.2 for other races. The rate

for Hispanic patients was 4.6, while the rate for nonHispanic patients was 6.5. They also found that emergency department visits for headaches were most common for patients in the age range of 18 - 49 years. According to Freitag, Lake, Lipton, and Cady (2004), African American men and women have prevalence rates for Chronic Tension-Type Headache, Chronic Migraine, and Frequent Headache of Other Types that are lower than those for Caucasian men and women. In regards to region, Migraine is more prevalent in Europe (14.8%) and North America (11.1%) than it is in Africa (4%) (Jensen & Stovner, 2008). Tension-Type Headache is more prevalent in Europe (80%) than it is in Asia and the Americas (20 - 30%). Chronic headache is most common in Central and South America (5%) and least common in Africa (1.7%). According to Jensen and Stovner (2008), the male to female ratio for Migraine varies from 1:2 to 1:3, with women having more Migraine without Aura than Migraine with Aura. For Tension-Type Headache, women are only slightly more affected, with a ratio of 4:5. Interestingly, there is no gender difference in incidence for prepubertal children. In terms of age, Tension-Type Headache peaks between the ages of 30 and 39 years, for both genders. Onset of migraine occurs in the 20's and 30's; it peaks in the 40's and declines thereafter. Chronic headache disorders span a lifetime, increasing until the 50's, with only a minor decline with age.

In addition to physical pain and suffering, patients suffer economic, occupational, and social losses (Krymchantowski, Adriano, De Góes, Moreira, & Da Cunha Jevoux, 2007). The second American Migraine Study conducted in 1999 found that only 9% of participants were able to work or function normally with their headache condition (Lipton & Newman, 2003). Likewise, 59% of participants reported that they missed family

functions or social leisure activities. Stovner and Hagen (2006) found that headache presents more of a burden for patients than do disorders such as Epilepsy, Multiple Sclerosis, and Parkinson's disease, as measured by the Disability-Adjusted Life-Years. Yet, despite the burden of migraine, there are still many underdiagnosed cases that do not receive appropriate treatment (Diamond et al., 2007).

Aside from the impact headaches have on patients' lives, they also create significant direct and indirect costs for society, as reviewed in the study by Munakata et al., (2009). The most recent American Migraine Prevalence and Prevention Study, a five-year longitudinal study, evaluated the impact of migraine on utilization of health-care resources and on productivity loss (Munakata et al., 2009). Data from the first 2 years of the study showed that participants with transformed migraine reported more primary-care visits, neurology and headache-specialist visits, emergency-room visits, and pain clinic visits as compared to participants with episodic migraine. In terms of productivity, participants with transformed migraine reported significantly more time missed from work or school because of headaches. Participants with transformed migraine also reported more time at work or school when productivity was reduced by half because of headaches. Participants with transformed migraine incurred significantly more direct and indirect costs ($7,750 per patient, per year) as compared to participants with episodic migraine ($1,757 per patient, per year). Lost productivity accounted for 55.7% of the costs for participants with migraine and 69.6% of the costs for participants with transformed migraine. Specifically, with indirect costs resulting from absenteeism, participants with migraine lost 13.7 hours per person, per year, while participants with

transformed migraine lost 85.7 hours per person, per year. With indirect costs caused by reduced productivity, participants with migraine lost 48.3 hours per person, per year, while participants with transformed migraine lost 256 hours per person, per year.

To reduce costs from headache, patients must be treated by appropriate professionals in appropriate settings (Saper, Silberstein, Gordon, Hamel, & Swidan, 1999). Inpatient treatment for patients with headache is sometimes a warranted and necessary evil, that is, "the severity of the illness must match the intensity of the service" (Saper et al., 1999, p.13). According to Saper et al. (1999), when hospitalization is appropriate to patients' conditions, it can reduce both direct and indirect costs. For example, successful hospitalizations can lead to longer periods of headache-free time and consequently less frequent medical care, thus reducing associated direct costs. Likewise, more headache free time results in less frequent work absences, thus reducing indirect costs. On the other hand, if hospitalization is not appropriate, or if there is an unsuccessful outcome, both direct and indirect costs increase. Examining factors associated with a longer length of stay (LOS) and minimal improvement potentially can provide an efficient way to identify patients who need more support before or during hospitalization in order to prevent and/or remediate factors that impede success of inpatient treatment.

The relationship between LOS and treatment outcome for inpatients with headache is an important area of study, not only because of the limited research on the topic but also because of the potential to reduce costs associated with headaches. Likewise, few studies have found treatment parameters that predict success for inpatient

treatment (Grazzi et al., 2002). Identifying factors associated with a longer LOS and minimal improvement can lead to the development of a rubric for teams who treat inpatients with headache that can help them to better decide specialized care for refractory cases in order to promote a timely, successful hospitalization.

Purpose of the Study

Research focused on factors associated with minimal improvement has not been clear about the way factors interact to predict a poorer outcome or about the stipulations of individual differences that may preclude predictive value for certain individuals. According to Grazzi et al. (2002), "multiple factors interact in varied ways to determine a patient's response to treatment" (p. 7). The purpose of the present study is to determine whether a combination of psychological variables predicts a longer LOS and minimal improvement for inpatients with headache, in order to create a rubric that directs specialized care for refractory cases. The study will analyze the significance of potential predictors in determining a longer LOS and minimal improvement. This author proposed that psychological factors that complicate the headache disorder will be associated with a longer LOS and minimal improvement.

Overview of Literature Review

Owing to the dearth of research on psychological factors that predict inpatient treatment outcome and LOS, the literature review focuses primarily on psychological and demographic factors that complicate headache disorders, and may lead to a poorer treatment outcome. The literature review describes headache classifications and the medical and psychological pathologies of and treatments for headaches. Research on the

relationships among pain and psychological and demographic factors will be presented. Likewise, the biopsychosocial model and stress diathesis model will be presented to review the connection between biology and psychology in the pathology of medical conditions.

Relevance to the Broad and General Knowledge Base of Psychology

The present study emphasizes the goal to expand the value of psychology to other disciplines, specifically the medical field. This study aims to incorporate the use of psychological measures in hospital settings in order for the field of psychology to define and expand further its role in the medical field. This study emphasizes the need for medical and mental-health professionals to collaborate to discuss the ways medical and psychological factors interact to result in minimal treatment outcome.

Chapter Two: Literature Review

Headache Disorders

Headache is a complex disorder with various etiological contributors, both genetic and environmental (International Headache Society, IHS; 2004). The lack of a single clear-cut headache etiology caused frustration for patients. There are many variations of headache disorders, likely caused by the numerous potential combinations of etiological contributors. As is applicable to this study, headache diagnoses will be reviewed to reveal their impact on quality of life.

According to the *International Classification of Headache Disorders, 2nd edition,* the first step in diagnosing headache is to determine whether the headache is a primary or secondary disorder (IHS, 2004). Primary refers to headaches that are not caused by another disorder known to produce headaches. Secondary refers to headaches that are caused by another disorder. Both primary and secondary diagnoses can be given simultaneously (IHS, 2004). Basic primary headache categories, such as migraine and tension-type, will be reviewed. Secondary headaches, such as Medication Overuse Headache, will be discussed also, as they frequently appear in inpatient settings (Saper, 2008).

Migraine is divided into two main categories, Migraine with Aura and Migraine without Aura (IHS, 2004). To meet diagnostic criteria for Migraine without Aura, an individual must have at least five attacks lasting between 4-72 hours per month (untreated or unsuccessfully treated). Headache pain also must meet two of the following characteristics: unilateral, pulsating, moderate to severe intensity, and/or aggravated by

physical activity. Additionally, during a headache, an individual must have at least one of the following symptoms: nausea and/or vomiting or photophobia and/or phonophobia (sensitivity to light and sound, respectively).

Migraine with Aura is characterized by focal neurological symptoms that precede and/or accompany the headache (IHS, 2004). To meet diagnostic criteria, an individual must have at least two attacks per month with no motor weakness and at least one of the following symptoms: fully reversible visual homonymous symptoms, including flickering lights, spots, lines, loss of vision; fully reversible unilateral sensory symptoms, including pins and needle, numbness; and/or a fully reversible dysphasic speech disturbance. At least one aura symptom must develop in 5 minutes or less, and/or aura symptoms must occur in succession for at least 5 minutes. Each aura symptom must last for at least 5 minutes.

There are diagnoses to account for disorders that are similar to Migraine with Aura and Migraine without Aura (IHS, 2004). Typical Migraine With Aura is diagnosed when there is aura with a headache meeting the criteria for Migraine without Aura. Typical Nonmigraine Headache with Aura is diagnosed when typical visual, sensory, and/or auditory aura symptoms occur without a headache characterized by the criteria of Migraine without Aura.

Differentiations of migraine include diagnoses such as Familial Hemiplegic Migraine, Sporadic Hemiplegic Migraine, Basilar-Type Migraine, and Retinal Migraine (IHS, 2004). Familial Hemiplegic Migraine is diagnosed if the aura associated with Typical Migraine with Aura includes motor weakness, and at least one first- or second-

degree relative has migraine aura with motor weakness. Sporadic Hemiplegic Migraine is diagnosed when there is motor weakness without a familial link. Basilar-Type Migraine is distinguished from other migraine disorders by its simultaneous effect on both hemispheres. Basilar-Type Migraine is diagnosed when at least two attacks per month occur with aura, without motor weakness, and with two of the following symptoms: dysarthria (difficulty articulating words), vertigo, tinnitus, hypacusia (hearing impairment), diplopia (double vision), visual symptoms in both temporal and nasal fields of both eyes, ataxia, decreased level of consciousness, and/or simultaneous bilateral paraesthesias. Retinal Migraine is characterized by reversible monocular visual disturbance, such as scintillations (flashing or sparkling), scotomata, and/or blindness.

Complications also can occur with migraine disorders that warrant additional diagnoses, such as Chronic Migraine, Persistent Aura without Infarction, Migrainous Infarction, Status Migrainosus, and Migraine-Triggered Seizure (IHS, 2004). Chronic Migraine is diagnosed when a migraine headache occurs on 15 or more days per month for more than 3 months in the absence of medication overuse. Persistent Aura without Infarction is diagnosed when one or more of the aura symptoms typical of a previous Migraine with Aura attack lasts for more than a week without evidence of an infarction. Migrainous Infarction is diagnosed when one or more migrainous aura symptoms are associated with an ischaemic brain lesion. Status Migrainosus is diagnosed when a severe migraine typical of Migraine without Aura lasts more than 72 hours. Migraine-Triggered Seizure is diagnosed when a migraine aura typical of Migraine with Aura triggers a seizure.

Tension-Type Headache is divided into the following categories: Infrequent Episodic Tension-Type Headache, Frequent Episodic Tension-Type Headache, Chronic Tension-Type Headache, and Probable Tension-Type Headache (IHS, 2004). Infrequent Episodic Tension-Type Headache is diagnosed when two of the following characteristics are met: bilateral location, pressing or tightening quality that is nonpulsating, mild or moderate intensity, and/or not aggravated by routine physical activity. While there may be anorexia, there is no nausea or vomiting, and there is not more than one symptom of photophobia or phonophobia. Headaches must last at least 30 minutes and can last up to 7 days. At least 10 episodes must occur, but no more than once a month and 12 days per year. Frequent Episodic Tension-Type Headache has the same criteria as Infrequent Episodic Tension-Type Headache except that headaches occur more than once a month but fewer than 15 days per month, for at least 3 months.

Chronic Tension-Type Headache, also labeled as New Daily-Persistent Headache, is diagnosed after 3 days of unambiguous symptoms similar to Frequent Episodic Tension-Type Headache symptoms that last for hours or are continuous (IHS, 2004). Probable diagnoses for infrequent and frequent episodic headaches (Probable Infrequent Episodic Tension-Type Headache and Probable Frequent Episodic Tension-Type Headache) are given when patients meet all but one criterion. Probable Chronic Tension-Type Headache is diagnosed when all criteria are met for Chronic Tension-Type Headache, and within the past 2 months some form of Medication-Overuse Headache is present.

Medication-Overuse Headache is divided into several categories: Ergotamine-Overuse Headache, Analgesic-Overuse Headache, Triptan-Overuse Headache, Opioid-Overuse Headache, and Combination Medication-Overuse Headache (IHS, 2004). Ergotamine-Overuse Headache is diagnosed when a headache is present for more than 15 days per month with at least one of the following characteristics: bilateral, pressing/tightening (nonpulsating) quality and/or mild or moderate in intensity (IHS, 2004). Likewise, the headache must have developed or worsened as a result of ergotamine overuse. Headache also must resolve or revert to its usual pattern within 2 months after stopping the ergotamine. Analgesic-Overuse Headache follows the same diagnostic criteria as Ergotamine-Overuse Headache. Triptan-Overuse Headache is diagnosed when a headache is present on more than 15 days per month and meets at least one of the following characteristics: predominantly unilateral, pulsating quality; moderate or severe intensity; and/or aggravated by or causing avoidance of routine physical activity (IHS, 2004). Headache also must be associated with at least one of the following symptoms: nausea, vomiting, photophobia, or phonophobia. Additionally, headache frequency must have increased during triptan overuse. Headache also must revert to its usual pattern within 2 months after stopping the triptan.

Opioid-Overuse Headache is diagnosed when headache is present for more than 15 days per month and is associated with development of headache or worsening of headache during opioid overuse (IHS, 2004). Likewise, headache must resolve or revert to its usual pattern after 2 months of stopping opioids. Combination Medication-Overuse Headache is diagnosed when headache is present for fewer than 15 days per month and

meets at least one of the following characteristics: bilateral, pressing/tightening (nonpulsating) quality, or mild or moderate intensity (IHS, 2004). Headache also must develop or worsen during combination-medication overuse and resolve or revert to its usual pattern within 2 months after stopping combination medications.

There are several reasons for the development of Medication-Overuse Headache. Patients may overuse medication for pain relief and to function with daily responsibilities because of inadequate control from medical care (Saper, 2008). Patients with headache commonly overuse medication to function, rather than to escape from problems or to get a high (Primavera & Kaiser, 1993; Saper, 2008). Patients with headache may also overuse because of a low pain tolerance, a fear of pain, and/or a belief of entitlement to be pain free (Saper, 2008). Medication-Overuse Headache is less likely in patients with Chronic Tension-Type Headache. However, Episodic Tension-Type Headache in patients who overuse medications can transform easily to a chronic headache disorder (IHS, 2004). Patients who overuse acute medications rarely respond to preventative medications.

Headache Pathology

Medical

The pathology of pain in general involves tissue damage, injury, or inappropriate activation of the pain-producing pathways of the central nervous system (CNS) or the peripheral nervous system (PNS; Goadsby & Raskin, 2008). Tissue damage stimulates peripheral nociceptors (pain reception), which is a normal response of a healthy nervous system (Goadsby & Raskin, 2008).

While the key structures involved in headache are known, the pathology is more obscure (Goadsby & Raskin, 2008). The trigeminovascular system, the large intracranial vessels, dura mater, and the trigeminal nerve that innervates them, is involved in headache. The caudal portion of the trigeminal nucleus and the pain modulatory systems in the brain that receive input from the trigeminal nociceptors also are involved in headache (Goadsby & Raskin, 2008). Several accepted etiological models for migraine include explanations of multimechanisms, genetic mutations, cortical spreading depression (CSD), and neurotransmitters (Barbas & Schuyler, 2006). The pathology of Tension-Type Headache is more obscure (Edmeads, 1998). There are few known contributing factors to Tension-Type Headache (Barbas & Schuyler, 2006).

Migraine is no longer conceptualized as simply a vascular condition resulting from blood vessel dilation (Thomas Jefferson University, n.d.). Multiple mechanisms contribute to the onset of migraine. Specifically, a chain of events, beginning with inflammation, leads to norciception, vasodilation, and central and peripheral sensitization. Inflammation involving hormone-like prostaglandins and neuropeptides results in vasodilation—the widening of the blood vessels. Vasodilation activates norciception, which transmits pain signals to the thalamus and cerebral cortex, the location of the first sensation of pain. Activation of the nociceptors leads to increased stimulation of the nerve cells in the trigeminal nerve (the main sensory nerve). Prolonged stimulation of the trigeminal nerve results in central sensitization. The multimechanism explanation accounts for migraine symptoms, such as sensitivity to light and noise and/or

nausea and vomiting. For example, stimulation of the trigeminal nerve may affect any of the nerve's three branches, resulting in sinus, facial, and/or neck pain.

Even when genetics do not appear to play a role in migraine, they are implicated (Barbas & Schuyler, 2006). In addition to the increased prevalence of migraine among family members, five susceptible gene locations on four chromosomes have been found to be linked significantly to Migraine with Aura and Migraine without Aura. Likewise, Familial Hemiplegic Migraine may provide insight on whether there is a shared cellular pathogenesis between the Familial Hemiplegic Migraine, more common migraine subtypes, and other headache conditions (Barbas & Schuyler, 2006; Sanchez-del-Rio, Reuter, & Moskowitz, 2006). However, there is no single gene responsible for Migraine with Aura and Migraine without Aura (Barbas & Schuyler, 2006).

According to Sanchez-del-Rio et al., (2006), genetic mutations lead to migraine aura through CSD. CSD starts with cortical stimulation, followed by a slow wave of neuronal depolarization that travels across the cortex, resulting in a long-lasting suppression of neuronal activity. The Familial Hemiplegic Migraine mutations make the brain more susceptible to CSD through excessive synaptic glutamate release, decreased removal of glutamate and potassium from the synaptic cleft, or persistent sodium influx. CSD can be induced experimentally by trauma to the cortex, high extracellular concentrations of glutamate or potassium, inhibition of NA+/ K+ -ATPase, and other stimuli. Overall, migraine develops from environmental and genetic causes that enhance susceptibility to hyperactivity in the cortex, resulting in CSD. However, the complete picture of the initiation of migraine is still a mystery.

Neurotransmitters, such as serotonin, are implicated in migraines (Hamel, 2007). While most studies have not supported a genetic role in serotonin synthesis, there is strong support for a link between low brain serotonin neurotransmission and migraine headache (Hamel, 2007). A disposition for low serotonin levels facilitates CSD-induced trigeminal nociception (Hamel, 2007). Similar to migraine, depression is also a disorder of low serotonin. Therefore, the comorbidity between depression and migraine is not surprising (Cahill & Murphy, 2004). However, one family study did not find a genetic basis for the association between migraine and depression (Cahill & Murphy, 2004). Twin studies, when conducted, may prove otherwise. As of now, there is no conclusive evidence for a common genetic etiology for both disorders (Cahill & Murphy, 2004). The neurobiological relationship between headaches and psychological factors will be discussed in more detail in the cognitive/affective and biological components section.

While the etiology of Tension-Type Headache is more obscure, several known psychological, environmental, and neurological factors are linked to Tension-Type Headache (Bendtsen & Jensen, 2006; Edmeads, 1998). Neurologically, pericranial myofascial pain sensitivity, which is common in patients with Tension-Type Headache, could be a result of peripheral sensitization of myofascial nociceptors (Bendtsen & Jensen, 2006). Studies also suggest that with Chronic Tension-Type Headache, the central nervous system is sensitized at both the spinal dorsal horn/trigeminal nucleus and supraspinally (Bendtsen & Jensen, 2006). However, central pain processing seems to be normal in patients with Episodic Tension-Type Headache (Bendtsen & Jensen, 2006). Stress and fatigue are known to aggravate Tension-Type Headache, and depression is

also a common factor (Edmeads, 1998). Epidemiological and twin studies found no evidence of a strong genetic susceptibility for Tension-Type Headache (Barbas & Schuyler, 2006).

Psychological/environmental mechanisms

Biopsychosocial model. The model for understanding headaches has progressed past the biomedical model to the biopsychosocial model (Nicholson, Houle, Rhudy, & Norton, 2007). The main theme of the biopsychosocial model is the interplay among biological, psychological, and social processes. Each factor has a unique influence on headaches; not one is more or less important than the others (Nicholson et al., 2007). Psychological factors, such as depression, anxiety, coping skills, stress, neuroticism, catastrophizing, locus of control, self-efficacy, emotional inhibition, and negative emotions, can play a role in the development, course, and outcome of headaches (Nicholson et al., 2007). Overall, research shows that psychological problems have an important influence on the suffering patients with pain (Dersh, Polatin, & Gatchel, 2002; Korff & Simon, 1996). For example, studies show that psychological factors can trigger headache potentiation, pain perception, disability, and treatment outcome (Korff & Simon, 1996; Nicholson et al., 2007).

Biopsychosocial theories categorize the concept of pain into four dimensions: nociception, pain, suffering, and pain behavior (Gatchel, 2004). Nociception occurs when nerves send messages conveying tissue damage to the brain. Pain is a subjective experience that involves the transduction, transmission, and modulation of sensory input. Sensory input may be filtered through genetic composition, learning history,

psychological status, and sociocultural influences. Suffering is defined as an emotional reaction to nociception or an aversive event associated with nociception. Pain behavior is defined as the reactions people have when they are suffering or in pain, such as avoiding activities.

Gatchel proposed a biopsychosocial interactive theory of health and illness (Gatchel, 2004). It proposes that affective, cognitive, biological, and somatic processes influence the autonomic, endocrine, and immune systems through afferent and efferent feedback. Underlying all these processes are genetic predispositions. The interactive processes can influence activities of daily living, environmental stressors, interpersonal relationships, family environment, social support/isolation, social expectations, cultural factors, insurance issues, previous treatment experiences, and work history, all of which also can influence psychological and biological factors.

Gatchel developed a biopsychosocial theory to explain the progression of acute pain to chronic pain (Dersh et al., 2002). He proposed three stages: normal emotional reactions, a wider range of psychological problems, and habituation to or acceptance of some aspects of the sick role. The first stage is characterized by normal emotional reactions to pain, such as anxiety, fear, and worry. During the second stage, after 2 to 4 months of persistent pain, emotional reactions can persist into psychological problems, such as learned helplessness, anger, distress, and somatization. According to the stress diathesis model, psychological problems that develop in the context of pain depend on an individual's preexisting psychological and personality characteristics, socioeconomic factors, and other environmental conditions. The stress of coping with chronic pain

exacerbates the individual's preexisting characteristics. During the third stage, psychological problems persist to the point where the individual's life revolves around pain as a result of chronicity. During this stage, patients can habituate to some aspects of the sick role. They may use the chronic condition as an excuse to avoid responsibility and social obligations, which could become a reinforcer for maintaining the chronic condition.

Stress diathesis model. The stress diathesis model is a component of the broader biopsychosocial model (Korszun, 2002). It is a widely accepted model for stress-related disorders (Korszun, 2002). The model proposes that stress interacts with underlying predispositions, thereby causing disorders. Stress can include physical stress, such as a virus or injury, or psychological stress, such as an event that threatens an individual's homoeostasis (Korszun, 2002). For example, an individual who develops a chronic pain condition may have predispositions, such as negative schemas or deficits in instrumental skills (Dersh et al., 2002). The stress of chronic pain then may activate the predispositions, resulting in depression (Dersh et al., 2002).

The stress diathesis model can specify how stress aggravates and/or contributes to the development of a headache disorder (Nash & Thebarge, 2006). For example, a preexisting acquired or inherited vulnerability to developing a headache disorder, when aggravated by stress, can develop into a headache disorder. Electrophysiological studies suggest stress sensitivity may be a contributor to migraine disorders. Individuals with migraine have higher levels of cortical arousal between migraine episodes with a lack of

habituation. Stress sensitivity may develop from the headache disorder, or it may be a correlate of shared pathophysiology (i.e., serotonin dysfunction).

Link between cognitive/affective and biological components. Studies show that neural circuits of both headache and cognitive-affective activity are highly intertwined (Nicholson et al., 2007). The pain circuit comprises the periaqueductal gray, serotonin (5-HT) neurons of the rostral ventromedial medulla, and norepinephrine of the dorsolateral pontomesenchephalic tegmentum (Nicholson et al., 2007). Serotonin is involved in pain and cognitive-affective activity in a few important ways: production of emotional pain reactions; obsessive rumination; sleep/awake cycles; long-term, low-level pain transmission; and regulation of neurotransmitters responsible for alertness (Buelow, Herbert, & Buelow, 2000). Norepinephrine is involved in maintaining wakefulness and alertness and the fight-or-flight response (Buelow et al., 2000). Norepinephrine also has an analgesic effect on pain by slowing the release of substance P (Buelow et al., 2000). Likewise, some of the pain-relieving effects of opioid analgesics, serotonin agonists, and norepinephrine agonists occur through the pain circuit (Buelow et al., 2000).

According to Nicholson et al. (2007), pain involves multiple brain regions, which are also the same regions involved in emotion, attention, and stress, among other psychological phenomena. Specifically, the amygdala area of the limbic system can activate the pain circuit, and it is also an important area for emotion. The anterior cingulated cortex, orbitofrontal cortex, insula, and hippocampus are implicated in pain modulation through attention, placebo, expectation, perceptions of control, and anxiety. The interaction between psychological and biological systems may explain the influence

of long-term changes through neuroplasticity and sensitization on the development of headache chronification and comorbid mood disorders.

Delgado (2006) reported that dysfunction in ascending and descending pathways of the serotonergic and noradrenergic systems can result in depression and chronic pain. When the serotonergic and noradrenergic pathways are not working properly, heightened sensitivity to pain and pain from normally nonpainful stimuli can occur. Antidepressant medications that act on the serotonergic and/or noradrenergic systems not only improve mood but also treat symptoms of chronic pain.

According to Cahill and Murphy (2004), the serotonin dysfunction involved in migraine and depression represents an underactive serotonergic system. Serotonin is proposed to be involved in migraine as evidenced by changes in serotonin and its metabolites during migraine attacks, the ability of serotonin to trigger migraine attacks, and the implication of serotonin in treatment (Cahill & Murphy, 2004). To date, seven serotonin receptors have been identified: 5-HT1 through 5-HT7 (Sheftell & Atlas, 2002). Evidence suggests that 5-HT1 is involved in migraines, since effective triptan medications are 5-HT1 agonists (Sheftell & Atlas, 2002). Serotonin also has been implicated in anxiety disorders, eating disorders, obsessive-compulsive disorder, (OCD), and Tension-Type Headache (Sheftell & Atlas, 2002).

Theories propose that psychological factors modulate pain through shared circuits (Nicholson et al., 2007). Negative affect may influence headaches by activating a defensive system that creates neural and physiological changes triggering or exacerbating a headache. Nicholson et al. (2007) defined negative affect as including emotions, such as

anxiety, dysphoria (transient feelings of depression), and anger. Serotonin and gamma-aminobutyric acid (GABA) have been implicated as a link in the neurochemistry between negative affect and headaches. Anxiety is linked to GABA dysregulation. Correspondingly, some preventative migraine medications reduce cortical excitability by enhancing GABA-ergic function. At a peripheral level, negative affect can lead to adrenaline release, sugar infused in the bloodstream, change in blood lipid levels, and increases in heart rate, respiration, and muscle tension, any of which alone or in combination can instigate a headache.

The neurotransmitter dopamine also has been implicated in migraine (Sheftell & Atlas, 2002). Dopamine plays a role in mood, cognition, aggression, pleasure seeking, motivation, impulse control, substance abuse, and aggression. Likewise, migraine prodrome symptoms, such as yawning, mood changes, nausea, and vomiting, are treated with antidopaminergic medications. Patients with migraine also have an increased density of dopamine receptors on peripheral lymphocytes when compared to the density in controls, which may reflect an underactive dopaminergic system. In terms of genetics, there is evidence that migraine is associated with the dopamine allele ß-hydroxylase.

Psychological Factors Associated with Headaches

Depression

Depression often is associated with the presence of headaches (Nicholson et al., 2007). As described by Nicholson et al., (2007), depression is characterized by feelings of sadness, despair, emptiness, and/or lack of pleasure in activities that occur almost every day for more than 2 weeks. A prospective designed study, measuring the incidence

of the first onset of major depression and headaches during a 2-year follow-up study found a comorbidity between headache and depression (Breslau, Lipton, Stewart, Schultz, & Welch, 2003). There are several hypotheses regarding the relationship between the two disorders: (a) there is a shared etiology (whether inherited or acquired), thereby creating vulnerability for the development of both disorders; (b) migraine leads to worry and dysphoria, thereby increasing the risk for depression; (c) depression leads to migraines and/or impairs the ability to cope with pain; and (d) there is a bidirectional relationship between depression and migraine, with one disorder increasing the risk for the other (Breslau et al., 2003). Much of the following research is aimed at shedding light on the relationship between pain and depression.

Research has not led to a clear understanding about whether there is a shared etiology or a bidirectional relationship between depression and headaches (Breslau et al., 2003). When patients with migraine with and without major depression were compared, patients with major depression had headaches at a greater severity but were not at risk for headache persistence (Breslau et al., 2003). Likewise, the risk for a first onset of major depression increased six-fold with the presence of migraine and a history of one or more depressive symptoms (Breslau et al., 2003). However, patients with and without major depression did not differ on the frequency of migraine attacks or on the persistence of migraine after a 2-year follow-up study (Breslau et al., 2003). Likewise, Breslau et al. (2003) found that there was no increase in headache-related disability over time for people with persistent migraine and a history of comorbid major depression. Interestingly, a correlational study involving inpatient treatment of chronic headache

disorders found that reductions in depression scores as measured by the Beck Depression Inventory (BDI) were not correlated with a reduction in the frequency of severe headache (Hoodin, Brines, Lake, Wilson, & Saper, 2000). Research supporting a bidirectional relationship between depression and headaches is conflicting, perhaps because of the length of the follow-up period in the studies.

More definite evidence for the shared etiology hypothesis is found when researchers compared patients with migraine with patients with other severe types of headache (Breslau et al., 2003). Unlike with migraine, the presence of other severe headache disorders did not predict the transition of one or more depressive symptoms to the full criteria of major depression (Breslau et al., 2003). Therefore, one can assume that the major depression associated with migraine is not simply a reaction of distress from pain, because people with severe headaches then also would have depressive symptoms transformed into major depression (Breslau et al., 2003). According to Breslau et al. (2003), there is a shared etiology between migraine and depression, which is probably not a result of a predisposition to experience or report both physical and psychological symptoms of distress. Instead, they propose that the shared etiology is an imbalance of hormones or neurotransmitters. Other studies suggest that the shared etiology results from a common pathophysiology between both disorders (Cahill & Murphy, 2004). For example, both migraine attacks and depressive episodes have a sudden onset, a similar course over time, and a partially overlapping treatment responsiveness (Cahill & Murphy, 2004).

Even in the absence of a major depressive disorder, there is a relationship between negative affect and headaches (Nicholson et al., 2007). Negative affect can increase the likelihood of having a headache, the intensity of pain, and the amount of headache disability. Research suggests that sufferers from headache experience more dysphoria as compared to people who do not suffer from headache. Likewise, negative affect creates more opportunities for stress to trigger a headache. Some studies suggest that negative affect increases severity of head pain (Nicholson et al., 2007). The presence of depression and dysphoria is a negative treatment indicator. Not only do they negatively influence patients' satisfaction with health care, but in terms of treatment response with general medical conditions, depression and dysphoria can interfere with patients' ability to adapt to lifestyle changes, recover from procedures, and adhere to medication regimens (Cruess, Minor, Antoni & Millon, 2007; Nicholson et al., 2007). It appears that perhaps with some headache types, dysphoria negatively influences the disorder (Nicholson et al., 2007).

Researchers analyzed the relationship between mood and pain in experimental studies (Keefe, Lumley, Anderson, Lynch, & Carson, 2001). Pain-free participants acted roles of positive, negative, or neutral mood states and then engaged in a cold-pressure pain tolerance task. Results showed that participants who acted the negative mood state showed a significant decrease in cold-pressure pain tolerance. Conversely, participants acting the positive mood condition showed a significant increase in cold-pressure pain tolerance. Results suggest that negative mood leads to increased reported experimental pain and potentially lower pain tolerance.

To sort out the relationship between pain and depression, some studies examined the effects of a depressive history on pain disorders (Tennen, Affleck, & Zautra, 2006). Even years after having a major depressive episode, patients with rheumatoid arthritis reported higher levels of pain, as compared to patients without a history of depression. However, higher levels of pain were reported only during high levels of distress. Likewise, patients with primary fibromyalgia syndrome (PFS) and a history of depression vented emotions more and perceived a decline in coping ability with rises in pain, as compared to patients with PFS and no history of depression. Interestingly, depressive symptoms in patients with PFS in the absence of a history of depression did not impact pain significantly unless the patients had a history of depression. However, higher levels of depressive symptoms were associated with lower moods on painful days. Patients with PFS who were formerly depressed used ineffective coping strategies when pain increased. Perhaps previously depressed patients had an erosion of resources and/or a vulnerability for using ineffective coping strategies, especially when in pain.

Another study investigated the relationship between depression and chronic pain by looking at the similar and different characteristics of depression and pain and at the impact of depression on pain outcomes (Korff & Simon, 1996). Korff and Simon (1996) found that not all dimensions of chronic pain are associated with depression. Contrary to previously mentioned studies, pain intensity was not significantly associated with increased depression. However, Korff and Simon (1996) found that interference with activities was a strong predictor of higher levels of depression. Additionally, the number of pain days in the previous 6 months combined with the number of pain sites was

significantly associated with depressive symptoms. Diffuse pain also was associated with increased depressive symptoms. In fact, the combination of diffuse somatic symptoms (other than pain) and interference with activities (disability) was associated with psychological distress. Korff and Simon (1996) suggested that people reporting diffuse somatic symptoms may have a heightened responsiveness to physical and psychological stressors. They proposed that the responsivity is related to the same process that creates comorbidity between physical and psychological problems.

Another study found connections among somatic symptoms, depression, and headache (Tietjen et. al., 2007). Chronic headache was associated with increased severity of somatic symptoms and increased frequency of major depressive disorder. Common somatic symptoms other than head pain included extremity/joint pain, back pain, and stomach pain. Somatic symptoms that overlapped with depressive symptoms were almost twice as likely as pain symptoms for sufferers of chronic headache in the study. Tietjen et al. (2007) also found that increases in somatic symptoms were associated with increases in depression.

The interrelationship among somatic symptoms, depression, and headache should be interpreted with caution (Tietjen et al., 2007). For example, the increase in depression could result from an overlap of similarity between depressive and somatic symptoms (Tietjen et al., 2007). Along the same lines, another study found a strong association between headaches and somatoform and psychological disorders (Bensenor, Tofoli, & Andrade, 2003). Bensenor et al. (2003) attributed the strong association between headaches and somatoform disorders to the overlap of similar symptomatology.

However, one study suggested that somatic and depressive symptoms are distinct owing to the finding that somatic symptoms predicted major depression in the following year (Tietjen et al., 2007).

According to Tietjen et al. (2007), there is probably a synergistic relationship between the related variables of depression and headache. For example, while one or two pain-related variables may not impact mood, many variables combined may result in depression. Tietjen et al. (2007) found that people with frequent headache, severe headache-related disability, and multiple somatic symptoms are likely to have active major depressive disorder. They also found that as education and income decreased, severity of headache frequency, headache impact, somatic symptoms, and depression increased. Apparently, rather than a simple, bidirectional relationship between headache and depression, there is probably a synergistic relationship between the two variables, which may be linked to a dysfunction of the serotonergic system.

According to Korff and Simon (1996), the associations among diffuse physical symptoms, migraines, and depression are the result of a common predisposition between migraine and psychological disorders rather than of a causal relationship between the disorders. In support against a causal relationship, Korff and Simon (1996) reported that people with chronic depression did not have an increased risk for the first onset of back pain, stomach pain, or temporomandibular pain, as compared to a control group. However, Korff and Simon (1996) did not present research specific to migraine and chronic depression.

While some research suggests that depression does not increase the risk for developing pain disorders, other research suggests that pain may increase the risk for developing depression under certain circumstances (Korff & Simon, 1996). Research suggests that pain influences depression over time through loss of social reinforcement and learned helplessness. However, the length of chronic pain did not predict the onset of depression. Rather, unimproved pain predicted the onset of depression. For example, after 7 weeks, patients with unimproved back pain were significantly more depressed than patients whose pain had improved. According to Koroff and Simon (1996), depression seems to manifest as a result of an incomplete recovery, rather than from chronicity. Again, Koroff and Simon (1996) did not present research specific to migraine and depression.

Anxiety

Anxiety and migraine are frequently comorbid disorders, begging the same question asked about depression and headache: are they part of the same spectrum or separate disorders? (Sheftell & Atlas, 2002). Both disorders sometimes can be treated with the same medications, suggesting a related etiology. Likewise, anxiety disorders are more prevalent among migraine patients than among the general population. According to Sheftell and Atlas (2002), while there is no definite answer, research suggests that there are shared mechanisms of action that account for the etiology of anxiety, depression, and headaches.

While research has focused more on the comorbidity between depression and headache, anxiety may be even more prominent in patients with headache (Nicholson et

al., 2007). Anxiety is defined as worry, fear, uneasiness, and apprehensiveness (Nicholson et al., 2007). It can result from real or perceived situations for which outcomes are unpredictable, uncontrollable, or unobtainable (Nicholson et al., 2007). Anxiety is more chronic during unpredictable events, such as headaches (Sheftell & Atlas, 2002). Situations provoking anxiety can be either specific (i.e., a work evaluation) or more nebulous (i.e., future career) (Nicholson et al., 2007). Nicholson et al. (2007) reported that individuals with headaches have more anxiety than individuals without them. Correspondingly, feelings of anxiety and stress are the most common headache triggers (Nicholson et al., 2007). Nicholson et al. (2007) reported that anxiety also can exacerbate intensity and frequency of head pain.

Anxiety significantly contributes to headache-related disability (Nicholson et al., 2007). According to Nash, Williams, Nicholson, and Trask (2006), patients with headache and increased anxiety had more disability, a poorer quality of life, and higher health care costs than did patients without anxiety. Specifically, one study found that pain-related anxiety accounted for about 14% of the variance of headache-related disability (Nash et al., 2006). Likewise, even when controlling for pain, headache-control beliefs, and emotional distress, physiological anxiety uniquely and significantly contributed to disability (Nash et al., 2006). Additionally, less anxiety after a period of 6 months predicted a lesser impact of headache interference as compared to a reduction of headache frequency and medication change (Nicholson et al., 2007).

Nash et al. (2006) proposed that pain-related anxiety contributes to disability. According to the fear avoidance model, pain can trigger cognitive, emotional, and

physiological anxiety responses. Pain may trigger cognitions regarding inability to cope, activity exacerbating pain, or worry about the inability to complete scheduled activities. Pain also may create the physiological responses of anxiety through sympathetic arousal. In turn, the psychological and physiological responses of anxiety may create a fear of pain, avoidance of activities, and hypervigilance for the preliminary signs of pain.

Hypervigilance for pain can result from fear of pain, severity of pain, and avoidance of pain (Keefe et al., 2001). Fear of pain can lead to avoidance of stimuli that may trigger pain, such as movement, social activities, employment, and daily routine activities (Keefe et al., 2001). A fear of pain contributed to greater disability and a worsening of pain in patients with chronic pain (Keefe et al., 2001; Siedliecki & Good, 2006). Likewise, one study found that a high fear of pain and severity of pain interfered with attention, potentially as a result of hypervigilance for pain (Keefe et al., 2001).

While research on the relationship between headaches and anxiety sensitivity is in its infancy, anxiety sensitivity may increase headache-related disability (Nicholson et al., 2007). Anxiety sensitivity is a cognitive process defined as the tendency to react fearfully to unusual bodily sensations, (i.e., a headache is a brain tumor). The main component of AS is the catastrophic interpretation, (thinking the worst about bodily sensations), which creates fear. Disability can occur when innocuous sensations misinterpreted as headache triggers lead to an avoidance of the specific activities occurring around the sensations and/or of activities in general.

According to Nicholson et al. (2007), anxiety sensitivity also can increase the likelihood of precipitating a headache, making headache pain worse. Nicholson et al.

(2007) proposed that the negative effects of anxiety sensitivity occur when the catastrophic interpretation leads to sympathetic activation, which may trigger head pain or precipitate a headache. The increased pain reinforces the catastrophic interpretation, thereby increasing sympathetic arousal and consequently perpetuating a vicious cycle that may lead to panic attacks (Nicholson et al., 2007). One laboratory study found that anxiety sensitivity accounted for higher pain ratings among patients with panic disorder as compared to controls (Keefe et al., 2001).

Some research suggests that fear and anxiety have different relationships with pain (Keefe et al., 2001). One study examined the differences. Participants were assigned to one of three emotion conditions: fear by a brief shock, anxiety by a threat of shock, and a neutral condition. Before and after emotional inductions, participants were tested on their pain threshold to radiant heat. Anxiety and fear produced different effects on pain threshold as measured by finger withdrawal. Anxiety resulted in increased pain reactivity, while fear resulted in decreased pain reactivity. While the results coincide with animal-study findings, a previously mentioned study found that fear worsens pain in patients with pain. Therefore, more research is needed to determine the role of fear and anxiety during the pain process in experimental and naturalistic settings with the general population and patients with pain.

Pain Sensitization

Negative emotions, such as depression and anxiety, are not only consequences of pain but also part of the pain process, particularly the development of pain sensitization (Janssen, 2002). Negative emotions serve several functions during the pain process.

Avoidance caused by depression or anxiety promotes recovery. Anxiety causes vigilance that prevents further harm. However, once pain becomes chronic, negative emotions are no longer adaptive. Negative emotions caused by chronic pain may cause ongoing physiological reactivity (i.e., enhanced sympathetic activation and muscle tension), along with low vagal tone and hypervigilance for pain, contributing to a pain sensitization. In turn, pain sensitization causes misdirected attempts to escape or avoid pain, which reinforces negative emotions, resulting in functional disability.

Psychiatric Comorbidity

With the presence of comorbid psychiatric and medical conditions, health-care utilization increases and health perception declines (Kalaydjian & Merikangas, 2008). However, psychiatric comorbidity has more of a detrimental effect on the headache disorder (Kalaydjian & Merikangas, 2008). Untreated depression may account for some pain-treatment failures (Kalaydjian & Merikangas, 2008). Of one study sample, 19 % had at least one comorbid psychiatric disorder (Kalaydjian & Merikangas, 2008). Dysthymia was the most common comorbid disorder of the depressive disorders, while Generalized Anxiety Disorder and Panic Disorder were the most common comorbidities of the anxiety disorders (Bensenor et al., 2003; Kalaydjian & Merikangas, 2008). Interestingly, both Generalized Anxiety Disorder and Dysthymia are the most chronic disorders in their respective categories.

Suicide is a significant concern among patients with migraine. Cahill and Murphy (2004) reported an elevated risk of suicide among patients with Migraine with Aura and Migraine without Aura. Of particular risk are patients with Migraine with Aura, with

comorbid depressive disorders. For example, with depression alone, the odds of a suicide attempt are 7.8 % as compared to 23.2 % for depressive disorders comorbid with Migraine with Aura. Even after controlling for psychiatric disorders, both Migraine with Aura and Migraine without Aura were significantly associated with suicidal ideation and attempts.

Learned Helplessness

According to Sheftell and Atlas (2002), helplessness is a psychological state that can occur from an uncontrollable situation or a situation perceived as uncontrollable, such as headaches. A response pattern of helplessness can lead to a general response of learned helplessness, which can contribute to depression and anxiety (Sheftell & Atlas, 2002). Overmier (2002) reported that learned helplessness is a state resulting from a loss of control over one's environment or over events that are especially noxious or painful. Learned helplessness is a state characterized by impaired motivation to initiate coping behaviors or learn new ways to cope (Overmier, 2002). A state of learned helplessness can last hours, days, or weeks (Overmier, 2002). It is likely to develop when an individual is exposed to unpredictable, aversive events outside of one's control (Overmier, 2002). One theory proposed that helplessness is responsible for the comorbidity between migraine and psychiatric disorders (Sheftell & Atlas, 2002).

Stages of Change

Some studies found that readiness to manage pain, as measured by the Pain Stages of Change Questionnaire (PSOCQ), affected treatment outcomes (Hadjistavropoulos & Shymkiw, 2007). The precontemplation subscale measures a lack of intention to use self-

management techniques for pain, while the action subscale measures an active engagement in managing pain. Unexpectedly, a high intention to manage pain predicted dropout of a 10-session cognitive-behavioral treatment program. However, Hadjistavropoulos and Shymkiw (2007) reported that high intention to manage pain and high active engagement were associated with less depression and more use of coping strategies. Patients who entered treatment with high active engagement and continued to increase their engagement early in treatment benefited the most from multidisciplinary treatment. Likewise, individuals with high active engagement for managing pain reported higher self-efficacy, internal control over pain, and more satisfaction with information given by their primary-care provider.

On the other hand, a low intention to self-manage pain correlated significantly with high levels of pain severity, pain-related interference, depression, and pain-related anxiety (Hadjistavropoulos & Shymkiw, 2007). Likewise, individuals with low intention to self-manage pain reported less control and self-efficacy over pain. Interestingly, Hadjistavropoulos and Shymkiw (2007) found that a powerful-others or chance locus of control predicted low intention to self-manage pain.

Headache-treatment outcome studies found psychological indicators for medication response and nonresponse (Lucas et al., 2007). Specifically, anxiety and high emotional distress, as measured by the Hospital Anxiety and Depression Scale (HAD), were associated with a greater likelihood of not responding to medication treatment. Lucas et al. (2007) proposed that anxiety and depression play a role in poor medication compliance, which contributes to nonresponse to medication treatment. More

specifically, they reported that catastrophizing, less use of positive reinterpretation to cope, and avoidance were characteristics associated with nonresponders. Interestingly, acceptance as a coping strategy also was associated with nonresponders. According to Lucas et al. (2007), acceptance may reflect helplessness and unwillingness to take control of headaches. On the other hand, it may reflect a transition from seeking treatment to adaptation to the disorder. Lucas et al. (2007) also reported that the use of positive reinterpretation as a coping strategy was associated with treatment response.

Neuroticism

Neuroticism is a personality characteristic associated with maladaptive coping skills, the onset of new migraine headache disorder, depression, and other pain-related variables (Keefe et al., 2001; Tennen et al., 2006). Neuroticism is defined as the disposition to experience and report aversive emotions (Keefe et al., 2001). People with high neuroticism may respond to pain in maladaptive ways, particularly with catastrophizing (Keefe et al., 2001). One prospective study assessed neuroticism at baseline along with the new onset of migraine headaches over a 5-year period. The study found that the risk of developing migraine increased directly with neuroticism scores among women (Keefe et al., 2001). Therefore, according to Keefe et al. (2001), negative emotional states not only are correlated with pain problems, but also are risk factors for pain onset and/or exacerbation. Neuroticism also is linked to pain intensity, pain-related appraisals, low self-efficacy beliefs, and low pain control appraisals (Hadjistavropoulos & Shymkiw, 2007; Tennen et al., 2006).

In addition to neuroticism, Huber and Henrich (2003) researched the association between personality characteristics and headaches. They found that neuroticism, as measured by the neurotic triad on the Minnesota Multiphasic Personality Inventory (MMPI-2), and introversion are elevated in patients with migraine. However, headache and bodily symptoms are shared items on the neuroticism score, which contributed somewhat to elevations. Huber and Henrich (2003) also reported a significant correlation between the neuroticism score and headache duration (total time of headaches per week). Social conformity also is a personality characteristic associated with patients with migraine. Additionally, Huber and Henrich (2003) reported that longitudinal studies showed a strong and consistent association between migraines and a stress-reactive personality, characterized by nervousness, sensitivity, and tendency to worry.

Huber and Henrich (2003) reported evidence against trait stability indefinitely influencing headaches. According to Huber and Henrich (2003), the elevated introversion and neuroticism scores reversed with successful headache treatment. Likewise, the fact that stress can trigger the onset of migraines and increase migraine attacks is evidence against a “migraine personality.” Instead, Huber and Henrich (2003) proposed an interaction between personality and environment. Specifically, the personality traits associated with migraine are proposed to result from a lower tolerance to stress, ineffective coping strategies, a limited ability to relax, and an increased focus on achievement.

Stress and Coping

Appraisals of environmental events can create negative emotional states, which can lead to stress (Keefe et al., 2001). Stress is defined as a process of appraisal of events and the resources to cope with the events (Keefe et al., 2001). There are two types of appraisal: primary appraisal and secondary appraisal (Hassinger, Semenchuk, & O'Brien, 1999). In primary appraisal, the person determines the significance of the event. In secondary appraisal, the person determines available resources (Hassinger et al., 1999). The combined primary and secondary appraisals lead to an evaluation of the stress level (Hassinger et al., 1999). The appraisal of events can affect the manifestation of the stress and the coping efforts used to reduce stress (Hassinger et al., 1999). Additionally, coping mechanisms can influence stress-related health symptoms, such as pain (Hassinger et al., 1999).

Hassinger et al. (1999) reported that headache sufferers' appraisal of stress is different from the appraisal by headache-free controls (Hassinger et al., 1999). For example, those who suffered with headache reported more daily hassles and rated the hassles as more distressing and disturbing when compared to the reports and ratings by headache-free individuals (Hassinger et al., 1999). Hassinger et al. (1999) proposed that when the perception of stress by those who suffer with headache is more negative than the typical perception, they may exacerbate their head pain.

According to Hassinger et al. (1999), headache-free individuals and those with tension headache cope with pain differently. For example, Hassinger et al. (1999) reported that those with tension headache responded to acute pain with more

catastrophizing than that of the control group. Hassinger et al. (1999) also reported that those with headache cope with stress less effectively than do controls. According to Huber and Henrich (2003), those with headache cope with stress by using coping skills, such as wishful thinking, problem avoidance, and self-criticism, which are thought to maintain stress rather than to reduce it. Likewise, those with headache use the stress-reducing coping strategy of social support less frequently than do headache-free controls (Hassinger et al., 1999). While there were differences in appraisal of and coping with pain among the headache-free, headache, and migraine groups, there were no differences among the groups in coping with cognitive stressors (Hassinger et al., 1999).

Differences in appraisal of and coping with stress were found also between patients with migraine and controls, with patients with migraine using maladaptive coping skills (Hassinger et al., 1999; Huber & Henrich, 2003). In a laboratory study, while patients with migraine reported a cold pressor task as more painful than did controls, they did not indicate the experience of pain any sooner than did controls (Hassinger et al., 1999). Results suggest that patients with migraine do not have a lower pain tolerance to the acute pain task (Hassinger et al., 1999). Hassinger et al. (1999) proposed that patients with migraine may have learned to appraise pain as more troublesome to lessen responsibility. In clinical studies, patients with migraine used more maladaptive coping strategies when dealing with pain and stress, such as catastrophizing and social withdrawal (Hassinger et al., 1999). However, in the laboratory study, catastrophizing was not found to be a significant reliant coping strategy for patients with

migraine. In terms of coping with cognitive stressors, the laboratory study found that patients with migraine used wishful thinking and self-criticism (Hassinger et al., 1999).

There are several possibilities for the relationship between headaches and maladaptive coping strategies (Hassinger et al., 1999). One research-based theory proposed that using less effective coping strategies to deal with stress and pain can exacerbate stress and pain (Hassinger et al., 1999). However, because most studies are quasiexperimental, other possibilities have been considered. For example, pain may create a mobilization effect to use any coping strategy available, whether adaptive or not (Hassinger et al., 1999). Additionally, maladaptive strategies may work for dealing with headaches, but could be maladaptive when used in other situations (Hassinger et al., 1999). Another theory proposed that there is a reciprocal relationship between maladaptive coping strategies and headaches, specifically that they both may increase the risk for the other to occur (Hassinger et al., 1999).

Studies show that maladaptive coping strategies and stress are associated with poorer adjustment and outcomes for patients with pain (Hassinger et al., 1999). With patients suffering from chronic low-back pain and arthritis, catastrophizing predicted increased pain levels, psychological distress, and decreased health status (Hassinger et al., 1999). Longitudinal studies with patients with rheumatoid arthritis and fibromyalgia found that passive coping was associated with depression, psychosocial impairment, and increased pain behaviors (Hassinger et al., 1999). Huber and Henrich (2003) reported that with patients with migraine, fatigue-induced stress led to longer recovery time from tension and depression. According to Hassinger et al. (1999), prolonged stress may lead

to an increased comorbidity of depressive and anxiety disorders for patients with headache.

Rothermund and Brandtstädter (2003) conducted research about accommodative and assimilative ways of coping with loss of functional deficits. They found that when functional impairments presented later in life, compensatory strategies that are assimilative in nature, (i.e., obtaining additional resources to avoid losses in order to maintain personal standards) increased for individuals up until the age of 70 years, and declined thereafter. They proposed that functional impairments initially prompt individuals to seek out compensatory strategies. When those strategies are no longer efficient in maintaining previous levels of functioning, there is a reduction in their use. When compensatory strategies are no longer efficient, self-evaluations become negative. However, Rothermund and Brandtstädter (2003) proposed that with accommodative coping, the negative effect can be buffered by adjusting personal standards.

Catastrophizing. Catastrophizing is a maladaptive coping mechanism associated with negative pain-related outcomes (Keefe et al., 2001). Despite the negative outcomes, this mechanism seems to serve a purpose for patients with pain. Catastrophizing is defined as the tendency to focus on and exaggerate pain as a threat and negatively evaluate the ability to deal with pain. The coping perspective proposes that people who catastrophize may use it to communicate their need for emotional support. Specifically, people may catastrophize to elicit the help of others and to reduce future expectations of behavioral activity. Catastrophizing is linked to higher self-reported pain, higher levels of

overt pain behavior, more pain-related disability, and increased use of pain medication and health-care services.

Kemp, Ersek, and Turner (2005) found associations between demographic characteristics and coping strategies for patients with pain. Both female and male older adult patients reported that spiritual practice and physical exercise were the most helpful coping strategies. Spiritual practice was reported as the third most common pain-coping strategy, while regular exercise was reported as the second most common. Religious coping strategies for pain management were most commonly reported by women and racial minorities.

Spirituality. In a literature review, Rippentrop (2005) found that patients with chronic pain use prayer as one way to cope with pain. In one study of individuals with musculoskeletal pain, prayer was the most used nonconventional pain management remedy. Another study found that minorities with arthritis used prayer to cope with pain. For example, 92% of African Americans and 50% of Hispanics with arthritis used prayer to cope with pain. Likewise, a qualitative study of Latina women with arthritis found that prayer and religious beliefs or activities were the second most used coping strategies for pain.

While prayer and spirituality are used frequently as coping mechanisms, spiritual coping strategies may not be effective ways to cope with pain (Rippentrop, 2005). Some research suggests that prayer characterized by hope is associated with increased pain, whereas other research suggests that prayer is associated with reduced pain. Spiritual coping is complex, and many coping questionnaires have a religious coping subscale with

a few items that capture negative and/or positive effects of religious coping. Some studies found no relationship between spirituality and pain. One study found that while spirituality religious coping was not associated with quality of life, it was associated with positive affect and psychological well-being. For example, spirituality as measured by the Spiritual Transcendence Scale, was an independent predictor of positive affect and self-ratings of health on the Short Form Health Survey (SF-36) in a sample of patients with rheumatoid arthritis.

Social support. For some patients with pain, social support leads to positive outcomes, while social isolation leads to negative outcomes (Zautra, Hamilton, & Burke, 1999). Social isolation predicted greater pain and psychological dysfunction in patients with fibromyalgia. For some individuals, social support plays a critical role in the choice of coping strategies they use. For example, women with rheumatoid arthritis chose coping strategies based on the responses of their social network and personal disposition. Women with critical spouses used more maladaptive coping strategies. Older adults with negative social relations reported having more difficulty coping with chronic health problems. Zautra, Hamilton, and Burke, (1999) reported that positive interpersonal interactions may preserve quality of life for individuals with fibromyalgia, while social withdrawal during pain episodes may make the individual more vulnerable to stress over time. Continual withdrawal from social interaction may weaken the ability to cope effectively, thus leading to maladaptive ways of coping. Helping patients with fibromyalgia find ways to interact positively with others even during pain attacks can improve well-being.

Hurwitz, Goldstein, Morgenstern, and Chiang (2006) also reported positive effects of social support. In a study of patients with neck pain, those who reported using social support and positive self-assurance had less pain and disability 6 months after treatment. Patients who had a lack of support from co-workers, family, and friends had more symptoms. Likewise, active coping, such as seeking out social support, is associated with reduced levels of pain, or no change in pain. On the other hand, passive coping, such as social isolation, is associated with high levels of ongoing pain.

Optimism. Ferreira and Sherman (2007) defined optimism as having a positive outlook on life, which is part of one's temperament and related to adaptation to chronic illness. According to Ferreira and Sherman, optimism is related to a better reported quality of life and promotes healthier habits and coping with chronic illness. Depending on the circumstances of the chronic illness, the role of optimism may vary. For example, if the chronic illness can be controlled at least in part by the patient's behaviors, optimism is associated with better functioning. Having positive outcome expectancies predicted positive outcomes, such as positive affect, psychological well-being, and the use of problem-focused coping strategies.

Studies show relationships among optimism, pain conditions, and chronic illness (Affleck, Tennen, & Apter, 2001; Treharne, Kitas, Lyons, & Booth, 2005). For patients in the early stages of rheumatoid arthritis, a chronic, painful arthritic condition, optimism was related to less depression, more life satisfaction, and lower levels of pain (Treharne et al., 2005). However, when the disease was established, optimism was associated with higher pain levels (Treharne et al., 2005). Affleck et al. (2001) reported that patients with

rheumatoid arthritis reported more pessimism than optimism being associated with negative daily mood, more functional disability, negative daily events, and poorer sleep. Individuals who were more optimistic reported having more cognitive coping strategies, social support, and control over pain, despite still having pessimistic moments (Affleck et al., 2001). Affleck et al. (2001) reported that negative daily experiences, coping strategies, and neuroticism in combination with pessimism or optimism affected pain-related outcomes. Optimists may have better health outcomes because they cope better with stress and difficult situations, resulting in less distress and impact on the physical condition (Affleck et al., 2001).

Demographic Factors Associated with Headaches

Bensenor et al. (2003) reported demographic and psychological factors associated with headache. Specifically, they found that headache attributed to psychological factors (nervousness or mental illness) peaked for both genders in the 25-34 year age range. However, after age 54 year, the frequency of a psychological contribution to headaches decreased. Headache attributed to lifestyle and/or physical conditions (i.e., fever, clinical, or neurological disorders) or to psychological conditions were 1.5 to 2.9 times more common for women than for men (Bensenor et al., 2003). Additionally, the frequency of headache problems attributed to lifestyle and/or physical conditions increased with age, up until the 55-64 year age range (Bensenor et al., 2003). In 1998, chronic headache disorders, such as Chronic Tension Type Headache, Chronic Migraine, and Frequent Headache of Other Types, occurred at a median age of 39 years in both men and women (Bensenor et al., 2003). Prevalence for Chronic Tension Type Headache, Chronic

Migraine, and Frequent Headache of Other Types appeared to be higher for people in their 40's and 50's and dropped to the lowest levels after age 55 years (Freitag et al., 2004). Demographic risk factors associated with Chronic Daily Headache include female gender; divorced, separated, or widowed marital status; and an education level of less than high school graduate (Marmura, Rosen, Abbas & Silberstein, 2009).

According to Bigal, Liberman, and Lipton (2006), headache disorders change with age. Patients in younger age ranges have a higher proportion of unilateral pain, pain aggravated by exercise, photophobia, and phonophobia (Bigal et al., 2006). Moreover, with increasing age, migraine attacks are typically less frequent and milder (Bigal et al., 2006; Kunkel, 2006;). However, while nausea and migraine disability lessen with age, total global amnesia and transient migrainous accompaniments are more frequent (Kunkel, 2006). Bigal et al. (2006) also found that aura was more common with age. With older patients, new-onset migraine is more likely to have secondary causes (Kunkel, 2006).

Bigal et al. (2006) reported that the prevalence of headache disorders varies among age groups. The peak prevalence of migraine occurred between the ages of 30 to 39 years (Bigal et al., 2006). Probable Migraine was more common than migraine in the younger and older ages (18-29 years; 70+ years) (Bigal et al., 2006). Transformed migraine to Chronic Daily Headache was more prevalent in middle- and older-age subjects, suggesting that over time some patients transform to Chronic Daily Headache (Bigal et al., 2006). Tension-Type Headache is more common before the age of 45 years, but it also can occur after the age of 45 years (Bigal et al., 2006). Late-onset Tension-

Type Headache is likely caused by excessive muscle tension aggravated by arthritis, poor posture, visual abnormalities, and temporomandibular joint disorders (Kunkel, 2006). Kunkel (2006) reported that Tension-Type Headache before age 45 years is usually associated with depression and stress.

According to Thorn et al. (2004), there are pain differences between men and women. Particularly, women report more intense pain and have a lower threshold for pain than men. Thorn et al. reported that there are both biological and psychosocial explanations for the gender differences. Catastrophizing and gender roles are two psychosocial variables proposed to influence the relationship between gender and pain. According to Thorn et al. (2004), gender differences may account for the higher prevalence of headache disorders among women.

According to Thorn et al. (2004), the impact of societal gender roles on personality contributes to the pain responsivity differences between genders. They defined gender roles as characteristics of gender differences proscribed by society. For example, in the United States, stoicism is attributed to men and sensitivity is attributed to women. Individuals classified as masculine had higher pain thresholds and significantly lower pain-intensity ratings as compared to individuals classified as feminine (Thorn et al., 2004).

Some studies suggest that catastrophizing is a mediator between gender and pain-related outcomes (Keefe et al., 2001; Thorn et al., 2004). According to Keefe et al. (2001), catastrophizing may explain some gender differences of pain reporting. Compared to men, female patients with osteoarthritis who catastrophized had higher

reported levels of pain, physical disability, and pain behavior (Keefe et al., 2001). However, when catastrophizing was controlled for, the gender effects were eliminated (Keefe et al., 2001). Contrarily, Thorn et al. (2004) reported that in a cold pressor task, catastrophizing measured before the task showed no significant effect on subjective pain reports. Likewise, there was only a slight effect from catastrophizing on pain tolerance (Thorn et al., 2004).

The emotional vulnerability trait, which is more prevalent in women, may partially explain the gender pain differences (Thorn et al., 2004). Thorn et al. (2004) reported that when the emotional vulnerability trait was controlled for statistically, the gender differences in pain responses were reduced. Thorn et al. (2004) proposed that the emotional vulnerability trait is a mediating link between gender differences, pain tolerance, and subjective pain ratings. Interestingly, individuals classified as androgynous showed no significant differences in pain ratings as compared to masculine and feminine groups.

Thorn et al. (2004) reported that the emotional vulnerability trait is responsible for catastrophizing and the gender differences of pain. When the emotional vulnerability trait and the factor of a whiny, complaining attitude were statistically controlled for, men and women did not differ in catastrophizing. According to Thorn et al. (2004) catastrophizing is likely a characteristic of the emotional vulnerability trait. Therefore, someone with a stable pattern of emotional vulnerability may have the tendency to catastrophize in response to pain, which is consistent with the stress diathesis model of chronic pain.

According to Thorn et al. (2004), women are more likely than men to develop the emotional vulnerability trait as a result of a history of pain and/or negative life events. Women are more likely than men to have persistent recurrent pain from chronic nonlife-threatening conditions. Additionally, the multiple role responsibilities women have may lead to greater perceived stress, a higher prevalence of depression, and more negative life events in general. However, Thorn et al. (2004) acknowledged that women may become more emotionally vulnerable at least partly because of biological predispositions for pain sensitivity.

Minority patients have unique chronic pain experiences (Baker, Buchanan, & Corson, 2008). African American people reported more pain and less control over pain than reported by Caucasian people. Additionally, African American women are disproportionately affected by more chronic medical diseases, rate their health as poor more often, and report less functional capacities when compared to Caucasian women. The differences between Caucasian and African American women exist even after controlling for income and education.

Baker et al. (2008) examined pain characteristics of a sample of African American women aged 61 - 80 years to determine unique pain characteristics. They found that women who were younger reported greater pain intensity. Baker et al. proposed that the age difference may be related to experience in developing coping skills. Older women may have learned better ways to cope with physical or psychological health issues. Baker et al. also proposed that older women may have acclimated to higher pain thresholds over the years, thereby reducing the perception of pain intensity. The age

effect also could have resulted from a cohort effect. For example, the group of older women may have had biological vitality or they may have developed psychological strength or coping abilities that allowed them to mitigate pain intensity. Baker et al. did not report the age range they considered as younger or older.

Baker et al. (2008) found differences with locus of control (LOC), depression, and pain between African American women reported in prior research and other demographic populations. In the sample of African American women aged 61 - 80 years, those who experienced greater pain intensity reported more depressive symptoms and an internal LOC. In other studies, an internal LOC was related to better pain outcomes, perhaps suggesting that LOC functions differently for this sample. Baker et al. (2008) proposed that for older African American women, perceived responsibility for pain may create self-blame instead of motivation to reduce pain severity. They reported that depressive symptoms in this population may be misrepresented. Chronic medical symptoms common in the older African American population of women can resemble depressive symptoms, making depression difficult to be distinguished from a medical condition.

Psychological and Medical Factors Associated with Treatment Outcome

Research has found relationships between treatment outcome and health LOC, Medication Overuse Headache, and personality disorders (Lake, Saper, & Hamel, 2009; Primavera & Kaiser, 1993). Primavera and Kaiser (1993) found that inpatients with a balanced health LOC were discharged an average of 1 day earlier than patients with a primary LOC of chance, trust in self, or trust in others. Individuals with a balanced health LOC had no extreme differences among health beliefs of chance, trust in self, and trust in

others (Primavera & Kaiser, 1993). Lake et al. (2009) found that Medication Overuse Headache, particularly from simple analgesics or triptans, was associated with significant improvement at discharge from inpatient treatment. Patients least likely to benefit from inpatient treatment were those with personality disorders, without Medication Overuse Headache (Lake et al., 2009). Inpatients with opioid dependency, which was more common among patients with Borderline Personality Disorder (BPD), had a longer LOS than the average stay of 13 days. Lake et al.'s (2009) inpatient treatment included medical intervention, cognitive-behavioral therapy (CBT), family therapy, relaxation, and psychoeducational groups. The majority of the inpatient study population was diagnosed with Chronic Daily Headache. Of the study population, 60% were diagnosed with Cluster B personality disorders. While Lake et al. (2009) acknowledged that their study was neither controlled nor randomized, they retorted that their inpatient population consisted of treatment failures, some from respected experts in headache management.

Lake, Saper and Hamel (2009) described several theories for the relationships among negative treatment outcome, BPD, and opioid dependence. One theory is that patients with pain and BPD may be more attentive to internally induced pain, rather than external pain. Another theory is that patients with pain have deficits in their ability to regulate and tolerate distress in order to prevent escalation. Additionally, patients with BPD have doctor-patient relationships that are difficult to manage, which may negatively affect treatment outcome. Several hypothesized factors may explain the increased rates of opioid dependence in inpatients with BPD. Physicians may be more likely to administer opioids to patients with BPD because of the patient's demanding, insistent interactions

with physicians. Likewise, patients with BPD may be more likely to request opioids as a result of a dysregulation in their pain and affective systems, general emotional distress, a need to control relationships, and beliefs of entitlement. Lake et al. (2009) proposed that the relationship among the presence of BPD, opioid Medication Overuse Headache, and negative outcome in their study may be related to the frequency of substance-abuse disorders in individuals with BPD. Specifically, opioids not only may trigger Medication Overuse Headache but also may exacerbate psychological symptoms, such as flashbacks, dissociation, a feeling of emptiness, and self-injurious behaviors.

Few studies have been conducted regarding the relationships between inpatient treatment outcome and scales as measured on the MMPI-2 and the Millon Behavioral Medicine Diagnostic (MBMD) (Diaz, 2004; Grazzi et al., 2002). In a study conducted in an inpatient setting with patients with chronic headache, MMPI-2 scales 2, 7, and 8 predicted LOS (Diaz, 2004). Particularly, scale 8, which measures confusion, disorganization, poor judgment, anxiety, and pessimism, predicted a longer LOS. Individuals scoring high on scale 8 may lack basic information to problem solve, and they may feel socially and emotionally alienated (Butcher et al., 2001; Diaz, 2004). Interestingly, individuals with elevations on scale 7 tended to have a shorter LOS (Diaz, 2004). Scale 7 measures anxiety, tension, insecurity, sadness, pessimism, and fatigue. Individuals with elevations on scale 7 also may be meticulous and organized (Butcher et al., 2001; Diaz, 2004). They also may worry about social acceptance or have difficulty coping with stress (Diaz, 2004). Scale 2, measuring depression, hopelessness, and insecurity, also predicted a longer LOS (Diaz, 2004). Individuals scoring high on scale 2

may be indecisive, lack self-confidence, or give up easily. Grazzi et al. (2002) found that scale 1, which measures concern with physical and bodily complaints, cynicism, and pessimism, predicted a negative outcome. Grazzi et al. (2002) reported that findings for scales F, 6, and 8 have been difficult to interpret. Diaz (2004) found that the forceful scale on the MBMD predicted a longer LOS (Diaz, 2004). Individuals scoring high on the forceful scale tend to be domineering, stubborn, and suspicious when interacting with others (Diaz, 2004). They also seek out challenges and are often risk-takers.

Headache Treatments

Medical

Headache disorders are treated in a variety of settings: outpatient headache centers, inpatient headache units, and emergency departments (Kwiatkowski & Alagappan, 2006). According to Kwiatkowski and Alagappan (2006), headache treatment in an emergency department may be successful but is often costly and frustrating for patients. Outpatient treatment can include medication therapy, nerve blocks, botulinum toxins, and nerve blocks (Kwiatkowski & Alagappan, 2006). Inpatient treatment can involve any of the previously listed outpatient treatments, along with intravenous therapy to break the pain cycle (Kwiatkowski & Alagappan, 2006; Saper, 2008). Inpatient therapy is reserved for intractable, severe, or complicated headache disorders (Kwiatkowski & Alagappan, 2006; Saper, 2008).

Preliminary treatment of headache disorders usually involves abortive and preventative medication therapy in an outpatient setting (Kwiatkowski & Alagappan, 2006). Abortive medications are used to limit the intensity and duration of an attack. For

mild to moderate attacks, abortives, such as acetaminophen, aspirin, ibuprofen, naproxen sodium, and tolfenamic acid, are suggested. For moderate to severe attacks, abortives, including dihydroergotamine, triptans, prochlorperazine, metoclopramide, ketorolac, and meperidine are suggested, some of which are administered intravenously or intramuscularly.

Preventative medications are used to decrease the intensity and frequency of attacks (Kwiatkowski & Alagappan, 2006). Preventative medications are used when there are more than two to three attacks a month, attacks last longer than 48 hours, or attacks are severe or debilitating. Preventative medications are usually 55 - 65% effective but have significant side effects. Therefore, when headaches decrease, tapering and discontinuing the preventative medication is recommended. Preventative medications include β-adrenergic blocking agents; calcium channel blockers; tricyclic antidepressants; anticonvulsants, such as divalproex sodium and sodium valproate; monoamine oxidase inhibitors; and methysergide.

Intractable, severe, or complicated headache disorders are best treated in an inpatient setting (Kwiatkowski & Alagappan, 2006; Saper, 2008). If aggressive outpatient or emergency department treatment is not effective, inpatient treatment is recommended (Saper, 2008). Specifically, inpatient treatment is necessary when detoxification is needed, when the presence of comorbid medical or psychological disorders interferes with treatment efficacy, or when unstable vital signs or dehydration is present (Saper, 2008). If not already completed, the preliminary steps of inpatient treatment involve laboratory and imaging tests to rule out headaches caused by secondary conditions

(Saper, 2008). Next, patients are given intravenous fluids and medications to break the pain cycle (Saper, 2008). If present, Medication Overuse Headache is treated by reduction of the overused drug while controlling withdrawal symptoms (Saper, 2008). Psychological assessment is offered early during inpatient treatment, followed by corresponding psychological treatment (Saper, 2008). Preventative and abortive medications may be tried experimentally with in order to establish an outpatient regimen offering more headache control (Saper, 2008).

Psychological

Psychological treatments for headache can influence the course and outcome of headache disorders (Sheftell & Atlas, 2002). Psychological treatments complement the efficacy of pharmacological treatments (Sheftell & Atlas, 2002). Treatments include emotional disclosure, CBT, for stress management, relaxation, biofeedback, and patient education.

Emotional disclosure. The negative effects of emotional inhibition on physical symptoms have led to the development of therapeutic interventions to increase emotional awareness (Keefe et al., 2001). For example, Keefe et al. (2001) described a treatment used to increase awareness of emotions and the relationship between physical symptoms and emotions. While the treatment has not been tested in controlled treatment outcome studies, according to Keefe et al. (2001), one study found that participants showed a reduction in symptoms (less temporomandibular joint pain or low back pain) and a greater awareness of emotions.

Emotional disclosure has varying outcomes, both positive and negative (Keefe et al., 2001). Treatment during which intense emotion was elicited with gestalt techniques reduced symptoms of depression but did not reduce long-term pain (Keefe et al., 2001). While pain levels were lower after sessions, the average pain level of patients increased between sessions (Keefe et al., 2001). Another approach that may be better suited for patients with pain is disclosure of troubling emotions at the patient's own pace (Keefe et al., 2001). Patients can be encouraged to write about stressful experiences in a self-directed and self-paced manner in order to avoid symptom exacerbation (Keefe et al., 2001). Overall, according to Keefe et al. (2001), emotional disclosure seems to result in short-term distress but long-term improvement taking several months.

Emotional disclosure has shown mostly positive effects for patients with rheumatoid arthritis (Keefe et al., 2001). Keefe et al. (2001) described one study that found that patients with rheumatoid arthritis reported significantly less affective disturbance and improved ability to conduct daily activities after 3 months of disclosure about stressful experiences. Although patients reported increased negative mood after verbal disclosure, they had the most improvement in their joint condition after 3 months as determined by a physician examination and physical tests. Patients with rheumatoid arthritis participating in emotional disclosure also had objective health improvements. Patients who wrote for 3 days about stressful experiences had significantly lower global impairment scores after 4 months as compared to those of controls, as assessed by physicians who were blind to groups.

According to Keefe et al. (2001), patients with rheumatoid arthritis are not the only individuals who can benefit from emotional disclosure. College students and healthy people in the community experienced health benefits by expressing emotions through private writing or verbal disclosure. Decreased health-care visits, enhanced immunological response, improved grade-point average, and faster reemployment after being laid off were some positive benefits. Emotional disclosure also may help individuals who catastrophize about dental procedures. Patients who participated in emotional disclosure and scored high on catastrophizing reported less pain and better mood than participants who scored high on catastrophizing and were in the control group.

Along with the emotional component of emotional disclosure, physiological and cognitive mechanisms are involved (Keefe et al., 2001). Venting emotions may decrease pain by changing the physiology that contributes to pain. Keefe et al. (2001) proposed that emotional processing activates the emotional schema, leading to cognitive changes. Emotional disclosure creates disconfirming evidence when negative affective states do not lead to harm. Additionally, repeated disclosure creates habituation to negative affective states. However, emotional catharsis in itself, without cognitive change, seems to be counterproductive.

CBT. CBT can help patients with headache cope better with everyday stresses that may trigger, exacerbate, or maintain headaches (Penzien et al., 2005). CBT helps patients identify cognitive and affective components of the stress response and understand the relationship among headaches, stress, and coping ability. Patients are guided to find the cognitive, emotive, and behavioral triggers of headaches and use more effective strategies

to deal with headache-related stress. For example, patients often respond to a debilitating headache condition with depression or anxiety, which CBT can reduce. CBT is used often in conjunction with relaxation techniques and biofeedback.

More specifically, CBT can be helpful for psychological problems, such as low self-efficacy, low internal LOC, or anxiety that can make managing headaches difficult (Nicholson et al., 2007). Patients may think they have no influence on headache attacks (low internal LOC) or that they are not able to manage headache triggers (low self-efficacy). Educating patients about ways to manage headache triggers to reduce the number of headache attacks and about their role in managing triggers can increase internal LOC. In addition, socratic questioning can be helpful for patients with anxiety-related disability who have catastrophic fears. For example, therapists can guide patients who limit their activity because of fear of provoking a headache to find instances when activity did not result in debilitating headaches or when medications aborted the headache.

Relaxation and biofeedback. Relaxation techniques and biofeedback involve the practice of controlling physiological responses that contribute to headaches (Penzien et al., 2005). Relaxation training and biofeedback can help patients gain better control over headache-related physiology in general by lowering sympathetic arousal. Relaxation is taught as a headache preventative, not an abortive. Practice is emphasized before seeing results, as training may take as many as 10 sessions. Three types of relaxation training are widely used—progressive muscle relaxation, autogenic training, and mindfulness. Progressive muscle relaxation involves alternating between tensing and relaxing muscles;

autogenic training involves using heaviness and warmth; and mindfulness involves calming the mind by maintaining focus on repeating words.

Biofeedback is the practice of controlling physiological responses, such as skin temperature and muscle tension (Penzien et al., 2005). Patients are taught to warm finger temperature and/or reduce arousal and muscle tension (Penzien et al., 2005). They are provided feedback on their performance through sensors that detect their physiological responses (Holroyd et al., 1984). Relaxation is usually a main component of biofeedback (Penzien et al., 2005). Like relaxation training, biofeedback is a preventative treatment that must be practiced at home regularly to be effective (Penzien et al., 2005).

Outcomes of relaxation and biofeedback practice vary, depending on the research (Freitag et al., 2004; Grazzi et al., 2002; Holroyd et al., 1984; Hoodin et al., 2000). According to Holroyd et al. (1984), successful performance feedback was associated with headache reduction. Holroyd et al. (1984) proposed that successful performance feedback leads to cognitive changes in self-efficacy and LOC, which are both associated with headache reductions. Freitag et al. (2004) reported that patients who practiced relaxation more frequently during severe headaches and to prevent headaches showed the greatest decrease on the Beck Depression Inventory (BDI) at discharge during inpatient treatment. The change in BDI scores was correlated with the frequency of relaxation practice (Freitag et al., 2004). However, there was no significant correlation between BDI changes and reduction of severe headache. Grazzi et al. (2002) reported that during inpatient treatment, patients who received biofeedback and relaxation training in conjunction with pharmacologic intervention experienced a clear advantage from treatment over a 3 year

period, as compared to patients receiving only pharmacologic intervention. Levels of improvement for the combined treatment group remained the same at posttreatment (Grazzi et al., 2002). Likewise, the relapse rate was significantly lower in the combined group at 3 years (Grazzi et al., 2002). Patients receiving the combined group treatment were also more careful in their use of analgesic medications (Grazzi et al., 2002). According to Hoodin et al. (2000), the practice of relaxation during inpatient medical intervention was associated with decreased depression as measured by BDI scores. Hoodin et al. (2000) proposed that an internal health LOC may have contributed to the increased adherence to relaxation practice and that treatment gains may have contributed to the decrease in depression.

Patient education. Patient education can help patients to manage headache episodes, become independent with self-care, initiate activities to manage headaches, manage medical therapies better, and alter daily routines to manage headaches (Sheftell & Atlas, 2002). Education should help patients understand how the underlying biology of the headache disorder is influenced by triggers, such as diet, hormones, environmental changes, sensory stimuli, and stress (Sheftell & Atlas, 2002). Likewise, teaching patients about the functions of abortive and preventative medications can help patients become more involved in their treatment plan (Sheftell & Atlas, 2002). Patient education can be used to improve medication compliance, especially for patients who have low self-efficacy for taking medication (Nicholson et al., 2007). Providers should discuss barriers to adherence and give reinforcement for adherence in order to increase self-efficacy and medication compliance (Nicholson et al., 2007).

Chapter Three: Hypotheses

The purpose of this study is to predict a longer LOS and minimal improvement by regressing LOS and minimal improvement on a number of psychological variables as measured on the MBMD (Millon, Antoni, Millon, Minor, & Grossman, 2006). LOS is measured by the difference between the admission and discharge dates. Both the admission and discharge dates are included in the calculation of the LOS. Minimal improvement is measured by the difference between admission and discharge scores on the Brief Pain Inventory-Short Form (BPI-SF; Cleeland, 2009) interference and severity scales and the difference between preadmission headache index scores and discharge headache index scores. The headache index score assesses frequency, duration, and severity of headaches.

Hypothesis 1

Ho: A combination of variables on the MBMD (i.e., anxiety-tension, depression, a forceful coping style, pain sensitization, social isolation, future pessimism, and spiritual absence) does not significantly and independently predict a longer LOS.

H1: A combination of variables on the MBMD (i.e., anxiety-tension, depression, a forceful coping style, pain sensitization, social isolation, future pessimism, and spiritual absence) significantly and independently predicts a longer LOS.

Hypothesis 2

Ho: A combination of variables on the MBMD (i.e., anxiety-tension, depression, a forceful coping style, pain sensitization, social isolation, future pessimism, and spiritual absence) does not significantly and independently predict minimal improvement.

H2: A combination of variables on the MBMD (i.e., anxiety-tension, depression, a forceful coping style, pain sensitization, social isolation, future pessimism, and spiritual absence) significantly and independently predicts minimal improvement.

Hypothesis 3

Ho: After inpatient treatment, there was not a significant difference between admission and discharge BPI-SF interference scores.

H3: After inpatient treatment, there was a significant difference between admission and discharge BPI-SF interference scores.

Hypothesis 4

Ho: After inpatient treatment, there was not a significant difference between admission and discharge BPI-SF severity scores.

H4: After inpatient treatment, there was a significant difference between admission and discharge BPI-SF severity scores.

Rationale

Psychological factors, such as depression, anxiety, personality traits, and maladaptive coping skills, can complicate headache disorders. Individuals with comorbid depression experienced more disability from migraine headaches than individuals without comorbid depression (Breslau et al., 2003). Likewise, depression is a risk factor for increasing the likelihood of the onset of pain and/or the exacerbation of pain (Keefe et al., 2001). With medical conditions in general, depression and dysphoria are obstacles for some patients attempting to adapt to lifestyle changes, recover from procedures, and adhere to medication regimens (Cruess et al., 2007). According to Nash et al. (2006),

patients with headache and increased anxiety have more disability, a poorer quality of life, and higher health-care costs than those without increased anxiety. Pain sensitization causes misdirected attempts to escape or avoid pain, which reinforces negative emotions, resulting in functional disability (Janssen, 2002). Social isolation predicted greater pain and psychological dysfunction in patients with fibromyalgia (Zautra et al., 1999). According to Ferreira and Sherman (2007), there are relationships between improved psychosocial and physical-health outcomes and optimism. Rippentrop (2005) found that spirituality was an independent predictor of positive affect in a sample of patients with rheumatoid arthritis. This author is assuming that the psychological factors discussed in the literature review that complicate the headache disorder will lead to a longer LOS and/or minimal improvement. Likewise, discovering whether this study replicates the results found by Diaz (2004), that the forceful scale on the MBMD predicted a longer LOS, would be beneficial.

Hypotheses 3 and 4 regarding the efficacy of inpatient treatment as measured by the BPI-SF were proposed in order to confirm minimal improvement as a valid, accurate construct in reference to the inpatient treatment provided. For example, if inpatient treatment was not effective for the majority of inpatients, the ability of the proposed psychological variables to predict minimal improvement would be skewed. However, the efficacy of inpatient treatment was not the focus of this study. Therefore, experimental controls were not implemented for treatment-efficacy hypotheses.

Chapter Four: Methods

This quasi-experimental, prospective, cross-sectional, survey research study used multiple regression to predict psychological factors associated with a longer LOS and minimal improvement in a headache inpatient unit. Survey research is the most efficient way to collect data for this study. It is the least obstructive and time-consuming method for patients. Collecting data prospectively avoided recall errors that patients may have had if they had been required to rely on their memory.

Study Overview

This study was conducted at the Jefferson Headache Center at Methodist Hospital between the months of August 2010 and May 2011. The headache inpatient unit at Methodist Hospital practices an interdisciplinary-team approach composed of attending physicians, the nurse coordinator, a headache fellow, a neurology resident, a psychologist, a psychiatrist, unit nurses, and a physician assistant, all of whom coordinate care. As a standard of care, patients must have a neurological evaluation before admission to rule out causes of pain other than headache. All patients are diagnosed with a headache condition within the standards of the International Headache Society before admission. As a part of the inpatient admission process, patients are required to complete a calendar to record their headache frequency, severity, and duration for at least 1 week before planned admission.

Assuming two-tailed $\alpha = 0.05$ and power = 0.95, a sample of 153 patients would allow a determination of whether the seven hypothesized psychological factors predict minimal treatment outcome and a longer LOS (Faul, Erdfelder, Buchner, & Lang, 2009).

The headache center at Methodist averages 10-13 inpatients a week. Recruitment was estimated to take approximately 4 to 8 months. However, glitches with recruitment precluded following the planned schedule. From August 2010 through October 2010, key personnel tried to increase the response rate of the preadmission calendars used to obtain baseline headache index scores. At the start of collecting data in August, key personnel realized that calendars were not being included in the inpatient packets mailed to patients prior to planned admissions. Likewise, about half of inpatients were admitted on an emergency basis, precluding the possibility of completing the preadmission calendar during the week before admission. As a result, staff circulated calendars, encouraging all patients to complete a calendar on a daily basis and to bring the calendar when admitted for inpatient treatment. Data collection resumed in November despite the continued poor response rate of preadmission calendars. Study procedures were not changed as a result of the poor response rate because reliable outcome measures, such as LOS and BPI-SF, were being collected as well. Preadmission calendars continued to be collected from participants who had them. Consenting inpatients were included regardless of whether they had the preadmission calendar.

Data collection outcome

By the end of May 2011, there was a total of 87 consenting participants. Approximately two to four inpatients consented to the study each week. Several factors could have attributed to the difficulty of amassing consenting participants in the numbers as estimated. Many patients were sleeping or in pain during the consenting opportunities. Additionally, some patients declined because they had visitors at the time. Some patients

declined because they reported that they did not have the cognitive ability to complete the surveys. There were 36 participants who did not complete the discharge BPI-SF for several reasons. Some patients did not complete the discharge BPI-SF because they refused or did not stay for the entirety of treatment as recommended. However, a majority of the discharge BPI-SF forms were not completed because of communication issues between key personnel and headache center inpatient staff.

Inclusion criteria

Patients admitted into the headache inpatient unit between November 2010 and May 2011 were approached with opportunities to consent to being a participant in the study. Participants were 18 years or older. Individuals younger than the age of 18 years are not admitted into this inpatient unit. In order to be included in the study, patients were required to have had the headache disorder for 3 or more months, demonstrate the ability to consent, complete measures at admission and discharge, and complete the entire stay as recommended.

Exclusion criteria

Patients with the following characteristics were excluded from the study: patients who had a cluster headache disorder, did not speak and read English, were not able to consent, failed to complete measures at both admission and discharge, left early against recommendations because of insurance or personal reasons, and did not have the headache disorder for 3 or more months.

Consent process

Key personnel visited new patients no longer than 1 day after admission and asked several questions in order to assess the patients' ability to consent. Patients were asked questions to determine whether they spoke and read English and were oriented to date, time, place, and persons. Patients were visited again at a later date if they were unable to consent because of sleep, fatigue, and/or disorientation during the initial attempt.

Upon meeting initial inclusion criteria of speaking English and having the ability to consent, key personnel briefly explained the purpose of the study and the consent process to patients. Key personnel reviewed the risks and benefits and emphasized that participation was voluntary, did not affect care, and could be withdrawn at any time without any consequences. Patients were given as much time as needed to read the informed consent, ask questions about the study, and consent or decline. After providing written informed consent, key personnel reviewed patients' records to ensure that they met the additional inclusion criteria (having the headache disorder for 3 or more months, no cluster headache diagnosis) and ensured that the patient planned to stay for the entirety of treatment. Patients who did not meet inclusion criteria were notified of the reason they could not participate. A copy of the informed consent was kept in a separate file for patients who qualified. The consent forms for those who did not qualify were shredded.

Procedures

Key personnel gave patients who consented and met inclusion criteria a copy of their signed informed consent, the MBMD, the BPI-SF, the calendar to complete during

the stay, and a demographic profile created for this study. The demographic questionnaire included questions about age, gender, race, education level, and work status. The admission measures typically took 60 minutes to complete. While patients had as long as 90 minutes to complete the measures, arrangements were made to accommodate patients who needed more time. Key personnel picked up the measures after 90 minutes or at a later date if more time was needed. No sooner than 1 day before discharge, patients were given the BPI-SF again, which typically took 5 minutes to complete.

After completion of admission and discharge measures, the patient's name, dates of admission and discharge, LOS, and medical and psychiatric diagnoses were recorded on a form labeled, "Patient Information." Medical and psychiatric diagnoses were obtained from the current inpatient hospital records and recorded on the Patient Information form. Additional survey data (MBMD, admission and discharge BPI-SF, preadmission and discharge headache index score) were matched with the patient information data and entered on a spreadsheet. The spreadsheet file was password protected. Upon completion of the study, the file was deleted and survey data were shredded. Until completion of the study, survey data were secured in a locked filing cabinet kept on site. While names were collected, they were neither reported nor published.

Measures

MBMD. The MBMD (Millon et al., 2006) is a self-report measure developed to reflect the attitudes, behaviors, and concerns of medical patients. It measures factors such as psychiatric indicators, coping styles, stress moderators, treatment prognostics, and

management guides (Cruess et al., 2007). Three scales, Debasement, Desirability, and Disclosure, are designed to correct for response patterns that may distort scores (Cruess et al., 2007). The measure includes 165 true-false items, estimated to take approximately 20-25 minutes to complete (Cruess et al., 2007). The MBMD was normed on more than 700 patients, from the age range of 18 to 85 years, with medical conditions, such as heart disease, cancer, diabetes, chronic pain, and HIV/AIDS (Cruess et al., 2007). The MBMD demonstrates both internal reliability and consistency, with an internal consistency coefficient mean of $\alpha = .79$ for all scales and a test-retest reliability mean of .83 for all scales (Cruess et al., 2007). With convergent validity, the MBMD depression scale correlated at .87 with the BDI. Prevalence scores reflect a comparison to the normed population (Cruess et al., 2007). Of the coping styles, a prevalence score of 60 or higher should be considered in analyses (Millon et al., 2006). Of the psychiatric indicators, a prevalence score of 75 - 84 suggests the presence of the scale's disorder, while a prevalence score of 85 and higher suggests a prominence of the scale's disorder (Millon et al., 2006). Prevalence scores between 60 and 74 are suggestive of the presence of symptom pathology but not sufficiently indicative unless the score is the highest score of the psychiatric indicators (Millon et al., 2006).

For this study, the scales analyzed will be a) anxiety-tension and depression from the psychiatric indicators, b) forceful coping style, and c) pain sensitization, social isolation, future pessimism, and spiritual absence from the stress moderator scales. For all categories of scales, this study included scores of 60 or higher in analyses. Items on the anxiety-tension scale include "I'm on edge a lot lately" and "I feel jumpy and under

strain, but I don't know why" (Millon et al., 2006). The depression scale measures poor appetite, social withdrawal, discouragement, guilt, behavioral apathy, self-depreciation, and anhedonia. The forceful scale measures domineering, tough-minded, and distrustful characteristics. The pain sensitization scale measures the tendency to be overly sensitive and reactive to pain. The social isolation scale measures perception of social support. The future pessimism scale measures outlook towards health status. The spiritual absence scale measures spiritual or religious resources to cope with stress, fear, or uncertainties associated with medical conditions.

Headache index score. The headache index score measures headache frequency, duration, and severity. A calendar with an intensity scale will obtain frequency, duration, and severity of headaches. The headache index score was calculated by multiplying each day's average intensity, by duration of headache in hours for that day, adding the week's total multiplications of hours and intensity, and dividing by the total number of days. Intensity is measured on a scale from 0 (no pain) to 10 (the worst pain you can imagine). Outcome was determined by the difference between preadmission and discharge headache index scores. The higher the difference, the better the outcome. Zero indicated no change from treatment. A negative difference indicated a regression from the admission condition.

BPI-SF. The BPI-SF is a widely used self-report measure to assess clinical pain (Cleeland, 2009). The BPI-SF is composed of two factors: severity and interference. The interference factor has two subdimensions, an affective subdimension and an activity subdimension. The affective subdimension measures pain interference in reference to

relationships, enjoyment of life, and mood. The activity subdimension measures pain interference in reference to walking, general activity, and work. A five-option verbal descriptor scale, with ratings of 0 = not at all, 1 = a little bit, 2 = moderately, 3 = quite a bit, and 4 = extremely, is used to measure pain interference. Pain severity is assessed by asking questions about pain over time: at its "worst," "least," "average," and "now." The recall period of the severity and interference scale is a week. A numerical rating scale is used to measure pain severity, where 0 = no pain and 10 = worst pain you can imagine. For this study, outcome was determined by the differences between admission and discharge interference and severity scores. The higher the difference, the better the outcome. Zero indicates no change from treatment. A negative difference indicates a regression from the admission condition.

Factor analysis in a large outpatient metastatic cancer study (N = 1,261) verified the two separate factors of pain severity and interference. In the same study, internal stability was good, ranging from .80 to .87 for the four pain severity items and .89 to .92 for the seven interference items. Multidimensional scaling provided strong psychometric support for the independent measurement of pain interference and severity. Initial short-term (1 day to 1 week) test-retest reliability for ratings of "worst," and "average," pain severity was acceptable (.78). Test-retest reliability for pain "now" is less (.59). Several more recent studies found similar test-retest coefficients. An outpatient German pain clinic study with retest occurring 30-60 minutes after the first administration found coefficients of .98 for pain severity and .97 for pain interference. Reliability coefficients for daily administration of pain severity "worst" "average" and "current" ranged from .83

to .88. Test-retest reliabilities for pain interference in the same study ranged from .83 to .93.

Analysis of Risk and Benefits

While patients in the study will not directly benefit from participation, the study may help future patients by providing data potentially to implement programs and/or procedures aimed to ameliorate factors associated with a longer LOS and minimal improvement. New programs and/or procedures aimed at specialized care could lower costs incurred by headache both to patients and society. There is a rare risk that patients may experience psychological distress from completing the measures. In the case that patients experience distress, psychological support is available on the unit to remediate the situation.

Chapter Five: Results

Data Collection Outcome

The difference between preadmission and discharge headache index scores was not used as a criterion variable because of the low response rate of the preadmission calendar. The response rate for the preadmission calendar used to obtain baseline headache index scores was only 6.9% among consenting participants. Of the 87 consenting participants, 36 were lost owing to failure to obtain discharge data, leaving a total of 51 completed protocols (see Table 1 for specific demographic information of consenting participants). Of the sample population, 78% was age 35 years and older. For analyses purposes, adults aged 18 to 34 years and adults aged 35 years and older were coded into two separate categories.

Table 1

Demographics of Consenting Participants

Gender	Hispanic	African American	Caucasian
Female	3	4	71
Male	0	1	8

Analyses and Results for Hypotheses 1 and 2

A multiple linear regression analysis was performed to determine the proportion of variance accounted for by the model, the significance of the model, and the significance of the predictor variables to predict the criterion variables (LOS and minimal improvement as measured by BPI-SF interference and severity differences scores). The model included the following scales: anxiety-tension, depression, forceful coping style, pain sensitization, social isolation, future pessimism, and spiritual absence. Using the simultaneous "enter" method on SPSS, version 19.0, all seven predictor variables in the model were entered in three separate analyses for each criterion variable. The proportion of the variance accounted for by the hypothesized model to predict each criterion variable (LOS and minimal improvement as measured by BPI-SF interference and severity difference scores) was not significant (see Table 2). Specifically, hypothesis 1, stating

Table 2

Significance of Hypothesized Model to Predict LOS and Minimal Improvement

Dependent Variables	*R* Square	*F*	Adjusted *R*
LOS	.034	.219	-.123
Severity	.089	.601	-.059
Interference	.170	1.256	.035

Note. Model 1 predictors: Pain Sensitization, Depression, Anxiety, Social Isolation, Spiritual Absence, Pessimism, Forceful Coping Style. Severity and Interference scores are measured by the Brief Pain Inventory (Short Form). LOS= length of stay.
$p < .05$.

that the model predicted a longer LOS, was not supported. Hypothesis 2, stating that the model predicted minimal improvement also was not supported. Additionally, there were no significant correlations between individual predictors of the model and each criterion variable.

Exploratory Analyses and Results

As observed during data review, a majority of the sample had elevations on the Pessimism and Pain Sensitization scales (see Table 3). Likewise, a pattern emerged among the refractory cases. A case was determined refractory when there was a LOS of 8 days or longer and BPI-SF interference or severity scores were less than or equal to 1.5. The refractory criteria were determined based on review of the outcome means (see Table 4 for means and standard deviations of outcome variables). Nonmarried participants with low Confident and Sociable scores and a high Denigrated score on the MBMD coping skills scales were observed to have minimal improvement after inpatient treatment in this sample population (see Table 5 for sample percentages of elevations and absences of elevations of exploratory variables). Marital status was coded as either married or not married. Participants labeled single, divorced, or separated were coded as not married for analyses purposes. Supplemental analyses were conducted using two exploratory models based on the aforementioned findings. Predictors of the first exploratory model included marital status and denigrated coping style. Predictors of the second exploratory model included sociable and confident coping styles. Only two predictors were included in each exploratory model because of the small sample size in order to increase the chances of detecting significance. Using the simultaneous “enter” method on SPSS, version 19.0,

predictor variables in the models were entered in three separate analyses for each criterion variable. The proportions of variance accounted for by the exploratory models

Table 3

Percentages of Inpatients with Elevations on Predictor Variables

Forceful coping style	Depression	Pain sensitization	Anxiety	Spiritual absence	Social isolation	Pessimism
8	69	94	59	31	37	84

Note. $n = 51$. Decimals rounded to whole numbers. Prevalence score ≥ 60.

Table 4

Mean and Standard Deviation of LOS, BPI-SF Severity, and Interference Difference Scores

Dependent Variables	Mean	Standard deviation
LOS	7.29	2.11
BPI-SF interference score	3.29	2.75
BPI-SF severity score	3.39	2.26

Note. $n = 51$. Decimals rounded to tens place. LOS = length of stay; BPI-SF = Brief Pain Inventory (Short Form).

Table 5

Percentages of Inpatients with Elevations and Absences of Elevations on Exploratory Predictors in Supplemental Analyses

Denigrated	Sociable	Confident	Not married
63	63	69	39

Note. $n = 51$. Denigrated, Sociable, and Confident are coping skills scales on the Millon Behavioral Medicine Diagnostic. Decimals rounded to whole numbers. Sociable and Confident scales percentages represent an absence of elevation.

to predict each criterion variable (LOS and minimal improvement as measured by BPI-SF interference and severity difference scores) were not significant (see Tables 6 and 7).

Owing to the high percentage of older adults in the population sample, exploratory analyses were conducted on the disabled status collected on the demographic questionnaire and the outcome variables. Participants had the following options on the demographic questionnaire regarding work status: employed part-time or full-time, student, unemployed, or disabled. For analyses purposes, disabled status was coded into two categories, disabled and not disabled. Any category other than disabled was considered not disabled. On the demographic questionnaire, the disabled category did not specify whether someone was receiving public assistance. A Pearsons *r* bivariate correlation was conducted using SPSS, version 19.0, to determine correlation between the disabled category and each outcome variable (LOS and minimal improvement as measured by BPI-SF interference and severity difference scores). There were no correlations between the disabled category and the outcome variables (see Table 8).

Table 6

Significance of Exploratory Model of Marital Status and Denigrated Coping Style to Predict LOS and Minimal improvement

Dependent Variables	*R* Square	*F*	Adjusted *R*
LOS	.231	1.348	.014
Severity	.093	.208	-.033
Interference	.077	.143	-.036

Note. Exploratory model predictors: Married Marital Status and Denigrated Coping Style. Severity and Interference scores are measured by the Brief Pain Inventory Short - Form. LOS = length of stay.
$p < .05$.

Table 7

Significance of Exploratory Model of Sociable and Confident Coping Styles to Predict LOS and Minimal Improvement

Dependent Variables	*R* Square	*F*	Adjusted *R*
LOS	.024	.578	-.017
Severity	.097	.230	-.032
Interference	.015	.376	-.026

Note. Exploratory model predictors: Sociable and Confident Coping Styles. Severity and Interference scores are measured by the Brief Pain Inventory Short - Form. LOS = length of stay.
$p < .05$.

Table 8

Correlations Between Disabled Category and Outcome Variables

Dependent Variables	Pearson correlation	Significance
LOS	-.026	.428
Severity	-.153	.141
Interference	-.071	.311

Note. Severity and Interference scores are measured by the Brief Pain Inventory Short - Form. LOS = length of stay. $n = 51$. Significance = 1-tailed.

Analyses and Results for Hypotheses 3 and 4

For analysis of the third and fourth hypotheses, a dependent, paired-samples *t*-test using SPSS, version 19.0, was performed to determine the efficacy of inpatient treatment by comparing the difference between admission and discharge BPI-SF interference and severity scores. The mean admission BPI-SF interference score ($M = 6.0188$), significantly exceeded the discharge BPI-SF interference score ($M = 2.7316$), $t(50) = 8.545$, $p = .000$. After inpatient treatment, the difference between BPI-SF interference admission and discharge scores was a little larger than one standard deviation ($d = 1.1963$). The third hypothesis, stating that after inpatient treatment there was a significant difference between admission and discharge BPI-SF interference scores, was supported. Therefore, after inpatient treatment, interference of headaches during daily activities was significantly reduced. The mean admission BPI-SF severity score ($M = 6.00$), significantly exceeded the discharge BPI-SF severity score ($M = 2.6373$), $t(50) = 10.765$,

p = .000. After inpatient treatment, the difference between BPI-SF severity admission and discharge scores was a little larger than one standard deviation (d = 1.5112). The fourth hypothesis, stating after inpatient treatment there was a significant difference between admission and discharge BPI-SF severity scores, was supported. Therefore, after inpatient treatment there was a significant reduction in severity of headaches. Additionally, 59% of the sample obtained a score of 0 or 1 at discharge on the BPI-SF severity item asking on a scale of 1-10 for a rating of current pain.

Chapter Six: Discussion

Summary of Findings

In review, the first and second alternate hypotheses were not supported. Specifically, the combination of psychological factors hypothesized (anxiety-tension, depression, a forceful coping style, pain sensitization, social isolation, future pessimism, and spiritual absence) did not predict a longer LOS or minimal improvement for this sample of inpatients with headache. The third and fourth alternate hypotheses were supported. Overall, inpatients in this sample experienced improvements in headache severity and interference of functioning from headaches after inpatient treatment. The confirmation of the third and fourth alternate hypotheses supports the contention that minimal improvement was a valid, accurate construct used in this study. Inpatient treatment efficacy was likely not a confounding variable conflicting with the ability of hypothesized psychological variables to predict minimal improvement. Inpatient treatment for this headache center may be an effective treatment option for patients with difficult-to-treat headache disorders.

While reviewing data, a pattern was observed among refractory cases. Nonmarried participants with low Confident and Sociable scores and a high Denigrated score on the coping skills scales had minimal improvement after inpatient treatment in this sample. Therefore, exploratory analyses were conducted, which were not supported. Specifically, a nonmarried marital status in combination with a high Denigrated coping skills score was not significant in predicting minimal improvement or a longer LOS.

Likewise, low Confident and Sociable coping skills scores were not significant in predicting minimal improvement or a longer LOS.

Significance of Findings

Support for the efficacy of inpatient treatment for headache at this headache center was confirmed. While confirmatory research is needed, patients with difficult-to-treat headache conditions should be considered for inpatient treatment. Depending on future research on long-term outcomes of inpatient treatment, not only may inpatient treatment be effective for difficult-to-treat headache disorders, but it also may be a more cost-efficient option.

As observed during the data review, a majority of the sample had elevations on the Pessimism and Pain Sensitization scales. Of the sample population, 94% had elevated Pain Sensitization scores and 84% had elevated Pessimism scores. For this sample, Pain Sensitization and Pessimism were likely not predictors of LOS and minimal improvement. The prevalence of the elevations likely precluded the predictive ability of those factors. Additionally, there were no significant correlations between Pessimism or Pain Sensitization scores and the outcome variables. Pessimism and Pain Sensitization may simply be factors characteristic of a difficult-to-treat headache disorder, rather than predictors of treatment outcome.

The MBMD measure may be helpful in identifying patients who have or will have a difficult-to-treat headache disorder that requires inpatient treatment. As mentioned previously, Pain Sensitization and Pessimism scales may be indicators of a difficult-to-treat headache disorder. A neurologist considering inpatient treatment for a patient could

refer during the decision-making process to the MBMD to check the Pain Sensitization and Pessimism scales. If the patient has elevations on these scales, he or she may have a difficult-to-treat headache disorder requiring more intensive inpatient treatment.

There are several explanations for the unsupported first, second, and exploratory hypotheses. There may be factors that do not complicate the headache disorder that predict a longer LOS and minimal improvement. The logical conclusion that factors that complicate headache disorders predict minimal improvement or a longer LOS may not stand true. Commonplace factors, such as the schedule of the patient, the patient-doctor relationship, and the schedule of the doctor, should be explored in future studies for their ability to predict LOS and minimal improvement. Additionally, study limitations and the potential for medical factors to hold more weight in predicting LOS and minimal improvement are considerations for the unsupported hypotheses.

Findings Related to Literature Review

The sample demographics are important to consider in reference to the findings. As discussed in the literature review, headache disorders influenced by psychological comorbidity were more common for individuals in the 25 to 34 year age range (Bensenor et al., 2003). This sample, composed of 78% of adults ages 35 years and older, may have not had psychological comorbidity as a relevant factor in complicating the headache disorder. With adults from ages 55 to 64 years, functional deficits, such as physical limitations and lifestyle changes, influenced headache disorders (Bensenor et al., 2003). If the logic is true behind the theory driving the first two alternative hypotheses, the demographic characteristics of this sample may have precluded findings that could

support hypotheses composed of psychological constructs. Future research with older inpatient populations should explore functional deficits as predictors of treatment outcome. Likewise, considering that the prevalence of comorbid medical disorders is higher in an older adult population, future research should examine the types of medical disorders, the severity of medical disorders, and the number of medical disorders as predictors of minimal improvement and/or LOS.

Relevance of Findings to the Theory and Practice of CBT

When considering CBT's value to patients who suffer from headache, the function of psychological symptoms for patients with a headache disorder should be considered. Anxiety and depression are considered maladaptive coping strategies. However, anxiety and depression about the future is reasonable for someone with a debilitating headache disorder, who has experienced many failed treatment attempts. Perhaps anxiety and depression did not predict a negative outcome because, despite complicating the headache disorder, they serve the function for the patient of staying persistent with treatment efforts. For example, while thinking of better times can make someone depressed, it also may reflect hope that improvement is possible. While anticipating pain can make someone anxious, it also may reflect a desire to try to plan for a future without pain. Hassinger et al. (1999) proposed similar theories. They stated that pain may create a mobilization effect to use any coping strategy available, whether adaptive or not (Hassinger et al., 1999). Additionally, they proposed that maladaptive strategies may work for dealing with headaches but may not be effective when used in other situations (Hassinger et al., 1999). Certainly, there are adaptive ways to cope with

pain that exclude depression and anxiety. Encouraging optimistic thinking that reflects small, achievable, incremental steps of improvement is best for patients with headache.

Findings Applicable to Future Research

As opposed to depression and anxiety, which, while maladaptive, could serve a function of persistence with treatment, coping skills that function to maintain the headache condition rather than to ameliorate it may be critical in predicting a negative treatment outcome. A construct discussed in the literature review that may represent a coping skill that maintains the headache condition is what Gatchel described as the "sick role" (Dersh et al., 2002). Gatchel proposed that acute pain transforms into chronic pain when the patient begins to habituate to aspects of the sick role. Patients habituate to the sick role when they use the chronic condition as an excuse to avoid responsibility and social obligations. Interestingly, Lucas et al. (2007) reported that a lack of response to headache treatment was correlated with using acceptance as a coping strategy. In response to the finding, Lucas et al. (2007) proposed that acceptance may reflect helplessness, an unwillingness to take control of headaches, or a transition from seeking treatment to adaptation to the disorder.

Suggestions for Future Research

Researching the coping skills that patients with headache use throughout the progression of their headache disorder could be influential in determining the interaction among coping skills, medical characteristics of the headache disorder, and treatment outcome. Psychologists should collaborate with neurologists in order to identify stages in the progression of the headache disorder as determined by medical characteristics that

define the severity of the disorder. Neurologists also could define stages as they see fit that appropriately define the progression of the headache condition.

Research also is needed to determine the coping skills that are specific to use by patients with headache. While many coping skills are identified as used by patients with pain and/or patients with functional impairments, these coping skills may or may not generalize to patients with headache. One area of interest is the assimilative and accommodative coping process as described by Rothermund and Brandtstädter (2003). They reported the process of coping with functional impairments in aging adults as a combination of assimilative and accommodative coping skills, used in conjunction with available resources. They proposed that when assimilative coping skills were no longer efficient to avoid losses of functional impairments, aging adults used accommodative coping skills to change their personal standards. Patients with headache may parallel this coping process. In the beginning of the headache disorder, patients may use assimilative coping skills, gathering all their resources and putting all their effort into avoiding losses associated with the headache condition. However, after many failed treatment efforts, patients with headache may change their personal standards to reflect acceptance of the headache disorder. While Rothermund and Brandtstädter (2003) proposed that accommodative coping strategies serve as a buffer, Lucas et al. (2007) proposed that acceptance may reflect a negative process involving helplessness.

Tracking coping skills used by patients with during the progression of their headache disorder is preliminary in nature. Therefore, a case study design with only a few participants would be appropriate. The focus would be on determining the interaction

between the stages of the headache disorder and the function of the coping skills used. Finding valid, reliable measures of coping skills can help to determine predictors of negative treatment outcome. As mentioned previously, commonplace factors, such as the schedule of the patient, the patient-doctor relationship, and the schedule of the doctor, should be explored in future research.

Research should be conducted to provide further support for the third and fourth hypotheses. To verify the efficacy of this headache center's inpatient treatment program, a control and comparison group should be used. A wait-list control group, as well as an outpatient infusion treatment group, could be used in further research, comparing short-term and long-term outcomes. Samples should be followed up every several months after discharge to determine the length of treatment outcome maintenance. Research comparing short-term and long-term outcomes of headache inpatient treatment with alternative treatment options is essential in determining whether inpatient treatment is a cost-efficient treatment option.

Limitations

While the alternative and exploratory hypotheses were not supported, null hypotheses were not confirmed. Despite the plausible explanations discussed to account for the absence of significant findings, the following limitations may have interfered with the model's ability to predict minimal improvement and/or a longer LOS. Of most importance, the sample size was not large enough to detect significance. Another potential confounding variable is that the sample population consisted of individuals willing to participate. The characteristic of willingness to participate could be a factor

that precluded the sample from being representative of the inpatient population with headache, in addition to precluding experimental control of the study. Contrarily, Huber and Henrich (2003) reported an association between patients with migraine and social conformity, in which case a willingness to participate may be a characteristic that reflects a representative sample of the inpatient population. Additionally, the sample was homogenous, consisting primarily of Caucasian women. The homogenous sample, despite being similar to the general headache population, may be a confounding variable precluding experimental control of the study. The demographics of being a Caucasian woman may have been a characteristic interfering with the models' ability to predict LOS and/or minimal improvement. In specific reference to the exploratory hypotheses, the cases observed to be refractory during data review may not have occurred frequently enough to warrant significance, or the sample size was not large enough to detect significance.

Considering the limitations, another study with a larger, heterogeneous sample would be required to confirm or disconfirm the models' ability to predict LOS and minimal improvement. Likewise, psychological factors may be lesser predictors, secondary to medical variables (i.e., response to medication) or environmental variables. Therefore, a larger than recommended sample size may be required to detect a psychological factor's significance amongst potentially stronger variables.

In reference to limitations related to the supported third and fourth alternative hypotheses, there was no control group used for comparison against the inpatient treatment. Therefore, the inpatient treatment may not have caused the significant

improvement in headache severity and interference of daily functioning from headaches. One cannot rule out that a sample under control-group conditions without treatment (in which inpatients are excused from responsibilities and under the care of nursing staff) would not demonstrate the same level of improvement as that of the treatment group. Likewise, considering that this headache center's inpatient treatment program is adjusted to meet patients' needs, there is variability in the treatment that each patient receives. The variability of each inpatient's treatment protocol could represent confounding variables. This study obtained short-term outcomes; unfortunately, long-term outcomes of inpatient treatment efficacy were not a focus of this study.

When replicating this study with an ample sample size, several considerations should be made. Additional key personnel should be trained; data on inpatient demographic characteristics should be collected prior to the start of data collection; measures used should be brief, simple, and completed on-site; and participants should be given time accommodations to complete measures. In order to accommodate for the conditions of inpatient treatment, an ample key personnel staff is required. For example, inpatients are often sleeping or unable to consent because of sedative effects of medication. Key personnel should be available to check-in throughout the day in order to obtain consenting participants. Likewise, key personnel are needed to collect outcome data in order to prevent loss of participants as a result of uncompleted discharge measures. Headache center staff have many responsibilities and should not be relied upon for study protocol.

Data regarding the inpatient sample demographics, particularly age, should be collected prior to the beginning of data collection in order to determine whether the hypotheses proposed are applicable to the characteristics of the sample population. However, despite collecting demographic data on inpatients with headache prior to data collection, determining the characteristics of inpatients willing to participate could still be difficult.

The best practice is to use outcome measures that can be completed briefly in one time period, rather than measures that have to be completed on a daily basis, over the duration of the stay. Inpatients often did not complete the calendar in its entirety during the duration of the stay. Participants required time accommodations for completion of the MBMD. Inpatients were more willing to participate in the study if they had the option of completing the MBMD on a day when their condition permitted more functionality. Despite efforts to circulate the headache calendars to obtain the headache index score, at the conclusion of data collection, the obtaining of calendars did not seem to improve enough to permit their use as a reliable measure. If inpatients are required to bring from home a calendar or any other kind of measure to be used as a baseline measure, they will likely not bring the measure with them to the hospital. Baseline data are best collected at admission.

Summary and Conclusions

The stages of creating a model that includes medical, environmental, and psychological factors to predict negative treatment outcome for inpatients with headache are still preliminary. The complex nature of the interaction of factors to predict treatment

outcome is still largely unknown. Theories proposed regarding functional deficits and coping skills should be explored in an effort to discover a psychological factor amenable to treatment efforts that can, at least partly, predict a negative treatment outcome. Neurologists and psychologists should collaborate their research efforts in order to propose a model that includes medical, environmental, and psychological factors. Particularly, neurologists and psychologists should examine the interactions among medical, environmental, and psychological factors that may influence treatment outcome.

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