Published in final edited form as:
Headache. 2007 March ; 47(3): 413–426.

Psychological Risk Factors in Headache

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Abstract

Headache is a chronic disease that occurs with varying frequency and results in varying levels of disability. To date, the majority of research and clinical focus has been on the role of biological factors in headache and headache-related disability. However, reliance on a purely biomedical model of headache does not account for all aspects of headache and associated disability. Using a biopsychosocial framework, the current manuscript expands the view of what factors influence headache by considering the role psychological (i.e., cognitive and affective) factors have in the development, course, and consequences of headache. The manuscript initially reviews evidence showing that neural circuits responsible for cognitive–affective phenomena are highly interconnected with the circuitry responsible for headache pain. The manuscript then reviews the influence cognitions (locus of control and self-efficacy) and negative affect (depression, anxiety, and anger) have on the development of headache attacks, perception of headache pain, adherence to prescribed treatment, headache treatment outcome, and headache-related disability. The manuscript concludes with a discussion of the clinical implications of considering psychological factors when treating headache.

Keywords

headache; self-efficacy; locus of control; biopsychosocial; psychological; negative affect

Headache is currently conceptualized as a chronic disorder with acute episodes of pain occurring intermittently lasting anywhere from minutes to days. For a significant number of patients, these attacks occur once a month or more and result in varied levels of disability.[1–4] Clinicians thus need to consider what factors influence the development, course, and severity of individual headache attacks and subsequent disability in order to minimize the frequency of attacks, reduce their severity, and limit their impact on functioning. To date, the overwhelming majority of research and clinical interest has focused on biological influences. These efforts have resulted in significant steps forward in the treatment and prevention of headache and its related disability; however, this research has also revealed that biological factors alone fail to account for all aspects of headache and disability. Psychological factors such as headache management locus of control and self-efficacy, and negative affect/emotional states can alter the likelihood of a headache attack being triggered, the perceived severity of headache pain, the impact headache has on functioning, and treatment prognosis.[5,6] Unfortunately, psychological factors are typically considered relevant only in cases where the patient presents

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Conflict of Interest: None

with significant psychopathology.[7–9] The purpose of the current manuscript is to describe the rationale for envisioning headache within a biopsychosocial framework, review evidence supporting this view, and consider clinical implications of considering psychological factors when treating headache.

A BIOPSYCHOSOCIAL MODEL OF HEADACHE

The biopsychosocial model can be summarized as considering the multidirectional relationships between biological (physiological), psychological (behavioral), and social (environmental) factors in the explanation of disease.[10,11] Whereas a biomedical model of disease focuses exclusively on biological processes and relinquishes psychological processes to the theatre of the mind, the biopsychosocial model attempts to overcome Cartesian dualism by recognizing that biological, psychological, and social/environmental influences are scientifically inseparable.

To date, the overwhelming majority of empirical and theoretical efforts to elucidate the development, course, and consequences of individual headache attacks have used a biomedical model. However, the clinical scientist and practicing clinician who regularly treat headache recognize that the experiences of many headache patients do not match the expectations emanating from a biomedical model. As a result, there is growing attention to the interplay of biological, psychological (made up primarily of cognitive and affective processes), and social processes in headache.[14–16] Viewing headache as a biopsychosocially influenced disease does not discount the importance of biological factors; instead, it provides a more complete account of what influences the development, course, and consequences of headache in the clinical setting. Given the emphasis on interdependent influences of biological, psychological, and social factors, it may seem surprising that the current manuscript focuses almost exclusively on psychological factors. This is not intended to suggest that psychological factors are more important than biological or social factors (since the biopsychosocial model holds that each factor's influence is fluid), but is rather a function of the paucity of literature to date reviewing the influence of psychological factors on headache.

CENTRAL-LEVEL PSYCHOLOGICAL INFLUENCES ON PAIN

Prior to focusing on specific psychological (cognitive and affective) factors that influence headache, the manuscript briefly reviews evidence showing that neural circuits responsible for cognitive-affective phenomena are highly interconnected with the circuitry responsible for headache pain.

By definition, pain is a psychological construct referring to the perception of unpleasant or aversive sensations.[17,18] Multiple brain regions process different aspects of a pain message, making pain an event stemming from a complex neuromatrix.[19–22] Many of the regions associated with pain processing are also involved with other psychological phenomena (eg, emotions, attention, stress),[23–30] therefore, modulation of pain by psychological factors may occur through these shared circuits, altering the pain signal within brain.

The best known CNS mechanism that modifies pain is a circuit that comprises the periaqueductal gray (PAG), 5-Hydroxytryptamine (5-HT) neurons of the rostral ventromedial medulla (RVM), and norepinephrine (NE) neurons of the dorsolateral pontomesenchephalic tegmentum (DLPT).[31] At least some of the pain relieving effects of opioid analgesics, 5-HT agonists, and NE agonists occur via this circuit.[31] The PAG appears particularly influential in migraine.[32] For example, the placement of electrodes into the PAG can induce migraine-like pain[33] and neuroimaging studies show that activity within the PAG and other brainstem structures is associated with migraine pain.[34]

This PAG-RVM-DLPT circuit receives input from multiple forebrain regions involved with psychological processes—particularly the limbic system. For example, the amygdala, an area known to be important for emotion,[29] can activate this circuit and appears critical for modulation of pain by cognitive-emotional factors.[35–40] Additionally, the anterior cingulate cortex, orbitofrontal cortex, insula, and hippocampus are implicated in pain modulation resulting from attention, placebo, expectation, perceptions of controllability, and/or anxiety.[41–48] These findings support the utility of a biopsychosocial view of headache.[49,50] Indeed, the interconnectivity between psychological and biological systems can explain how transient, experimentally-induced states can lead to headache,[51,52] and how long term changes (through neuroplasticity/sensitization) in these systems may influence headache chronification and lead to comorbid headache and mood/cognitive disorders.[53,54]

In summary, psychological factors can influence headache pain via various CNS pathways. However, it is important to identify specific factors that are influential. The next sections review specific cognitive and affective factors that influence the development, course, and consequences of headache.

HEADACHE BELIEFS AND COGNITIONS

Cognitive processes encompass the thoughts, beliefs, attributions, and attitudes people utilize when negotiating their environment. As it relates to headache, cognitions influence whether a patient engages in behaviors that lessen the likelihood of having a headache attack, adheres to medication regimen, and how the patient copes with the headache attack (thus influencing headache-related disability). Although numerous cognitive processes can influence headache and disability, 2 types of cognitions are particularly influential: locus of control (LOC) beliefs and self-efficacy (SE) beliefs.

Locus of Control (LOC)

LOC beliefs concern the degree to which an individual perceives that an event is under his/her personal control. These beliefs range from a completely internal LOC (where the individual perceives the event as totally under his/her control), to a completely external LOC (where the event is perceived as totally outside the individual's realm of influence). Individuals typically experience LOC about an event that falls somewhere between these 2 extremes. In most situations, having less internal LOC (ie, more external LOC) negatively influences affective (eg, increased dysphoric feelings), behavioral (eg, less likely to use active coping),[55–57] and physiological (eg, NE depletion and increased 5-HT sensitization)[58,59] responses to an event.[56–58] If perceived internal LOC remains low for an extended period then the individual may perceive that the situation is "hopeless" and thus "gives up," resulting in more pronounced affective, behavioral, and physiologic problems.

As it pertains to headache, 3 loci have been identified that determine who the individual views as controlling the onset, course, and consequences of headache.[9,60] These 3, internal LOC ("I am the key factor in controlling my headaches"), external-chance LOC ("nothing predicts my headaches"), and external-health care professionals LOC ("only my doctor and prescription medication can control my headaches") are present to varying degrees for all individuals. There may also be variability in an individual's LOC as it relates to different phases of a headache attack and its consequences. Research has shown that a high internal LOC is associated with better headache treatment outcome[61] and less headache-related disability.[62] Conversely, patients with low internal LOC are less likely to engage in behaviors that reduce the likelihood of a headache attack (eg, managing headache triggers), reduce/eliminate headache pain (eg, not seeing themselves as responsible for taking medication as prescribed), and minimize headache-related disability. As it pertains to external LOC, patients who believe their headaches are controlled primarily by chance factors report higher levels of depression, are

less likely to engage in adaptive coping strategies, and experience more headache-related disability.[63] Patients who believe that health care professionals are the primary influence on headaches report higher levels of medication use,[63] which may put these individuals at greater risk for medication overuse headache. Thus, a higher internal LOC and lower external LOC is typically desirable for optimal outcome.

Self-Efficacy (SE)

SE refers to an individual's belief that he/she can successfully engage in a course of action to produce a desired outcome.[64,65] SE beliefs exist for any potential behavior in which the individual may engage and are situation specific. SE beliefs vary across people and situations and can be modified over time as a result of successful or unsuccessful experiences.[64] For example, individuals possess SE beliefs about their cognitive, behavioral, and affective responses to a stressor.[66]

The influence of SE on headache has received considerable attention. The majority of this attention has focused on the role of SE in predicting headache treatment response, particularly as a potential mediator or moderator of treatment outcome.[67–71] Seminal work concerning what mediates patient improvement during biofeedback training indicated that changes in SE were the mechanism of change.[72–75] Baseline self-efficacy predicts differential response to combined pharmacologic and behavioral treatment[76] and changes in SE correlate with changes in headache frequency.[77,78]

SE also influences how one manages headache and disability. Individuals possess SE beliefs for managing modifiable triggers, adhering to treatment regimens, and coping with pain.[79] High levels of SE are associated with less dysphoric and anxious feelings[80] and moderate the influence of perceived stress on headache frequency.[81] Low SE for coping with a stressor increases autonomic arousal[82] thus making one more susceptible to experiencing a headache.[12,83–85] SE has also been proposed as a predictor of headache-related disability and preliminary findings appear to support this hypothesis.[86]

LOC and SE are related, yet independent concepts. Together they represent the manner whereby an individual determines whether or not to engage in behaviors that lessen the likelihood of a headache attack, whether to adhere to pharmacologic treatment recommendations, and/or whether to cope with headache in a manner that reduces headache-related disability. Thus, patients' LOC and SE beliefs are relevant issues to consider in treatment formulation.[87]

NEGATIVE AFFECT AND EMOTIONAL STATES

Pain involves both a sensory and an affective component.[18] Pain-related affect is almost always aversive and involves "negative affect" (NA). NA is a construct consisting of a "triumvirate" of negative emotions: anxiety, depression, and anger.[88,89] NA emotions influence the course and impact of headache within the normal range of affective experience, not simply when an Axis I disorder is present. These emotions can influence the likelihood an individual will experience a headache attack,[90–95] the intensity of headache pain,[96–98] and headache-related disability.[6,9,99–104] NA comprises a basic unpleasant-defensive motivation system involving a complex neural circuit that can provoke varying degrees of neural and physiological activation that can potentially onset or exacerbate a headache.[105] At a central level, it has recently been hypothesized that anxious/stressful feelings may trigger activation in the PAG and paraventricular hypothalamic nucleus.[106] This activates a series of events in the superior salivatory nucleus (SSN) and trigemenovascular system that results in migraine pain.[106] As noted earlier, the amygdala and septo-limbic system play a role in the experience of emotion and the cognitive-emotional modulation of pain. At a peripheral level, NA can instigate adrenal

release, change blood lipid levels, infuse sugar into the blood stream, increase heart rate, respiration, and muscle tension. Any one or combination of these processes could trigger a headache attack.[107]

Potential neurochemical links between NA and headache include 5-HT and γ-aminobutyric acid (GABA) dysregulation, which both have been implicated in NA emotions and headache. Dysregulated GABA is implicated in anxiety[108–110] and medications that enhance GABA-ergic function are useful in preventing migraine headache, perhaps by reducing cortical excitability.[12,111,112] Dysregulated 5-HT may be responsible for headache, anxiety, and depressed mood.[113,114] The mechanisms whereby anger and anger expression influence headache activity are unknown; however, its influence likely comes from limbic system activation.[115–117] Although anxiety, depression, and anger together create NA, each appear to uniquely influence headache and thus warrant independent review.

Anxiety

Anxiety is an aversive state of worry, fear, uneasiness, or apprehension "resulting from feelings of being unable to predict, control or obtain desired outcomes"[118,119] in regards to a specific situation (eg, an upcoming evaluation at work, having enough money to make credit card and utility payments for the month) but can also be more nebulous (eg, career, family, finances). Patients often use the term "anxiety" and "stress" interchangeably, as the concepts are highly correlated.[120] It is likely that an individual who responds anxiously when exposed to a stressor will become stressed about that response, which then heightens anxiety, and thus creates a vicious anxiety/stress cycle.

Although much attention has been focused on the relationship between depressive symptoms and headache, anxious feelings may be even more prevalent among headache sufferers.[85,121–125] Anxious/stressful feelings are one of the most common headache triggers and individuals with headache are more anxious than persons without pain.[91,126–128] Heightened anxiety can increase headache pain intensity[129,130] and reduced anxiety is associated with decreased headache frequency over time.[91]

Perhaps the greatest influence anxious feelings have is on headache-related disability. Increased anxiety among headache sufferers is associated with greater disability, poorer quality of life, and increased cost of care.[93,122,127,131–134] Interestingly, one recent study found less anxious/worried feelings after 6 months was a stronger predictor of lower headache impact than changes in headache frequency or changes in medication.[76]

Another way whereby anxiety can precipitate the likelihood of having a headache attack, exacerbate pain intensity, and make headaches more disabling is through a concept termed "Anxiety Sensitivity" (AS).[107,135–138] AS is a dispositional construct wherein individuals higher on AS react fearfully to unusual bodily sensations or physiological symptoms of anxiety or fear. For example, a pounding heart might be interpreted by someone high on AS as evidence of cardiac problems or an impending heart attack. This catastrophic interpretation may then lead to sympathetic activation and (via a negative feedback loop) further increased heart rate. The intensification of the symptoms (eg, heart rate) strengthens the belief in the negative interpretation (eg, cardiac problems), leading to greater autonomic activation and further catastrophic beliefs, culminating through a vicious cycle into a panic attack. Similar observations of symptom misinterpretation have been described in hypochondriasis[139] and chronic musculoskeletal pain.[140]

Although the study of AS in headache is in its infancy, 3 possible relationships exist. First, and most likely, is that AS increases headache-related disability. Individuals high in AS may misinterpret innocuous sensations as evidence of possible headache onset, thus making them

more likely to exit and subsequently avoid situations or activities around which those symptoms occurred.[136] Second, AS may amplify headache pain in much the same way that high AS amplifies a panic attack. If early headache symptoms are interpreted negatively and provoke anxiety and fear, sympathetic nervous system activation could lead to physiological increases in heart and respiration rates, blood sugar and lipid levels, and general muscle tension that might, in turn, intensify headache pain. Third, it is plausible that high AS could trigger a headache attack through a similar mechanism; that is, the misinterpretation of innocuous sensations may cause anxiety and sympathetic arousal which, in turn, may provoke a headache attack. One interesting study reported that heightened AS may reduce the likelihood that rescue medications will be taken as directed.[141] However, before any conclusions can be made about the relationship between AS and headache, more empirical investigation is needed to establish the parameters of this relationship.

Depression

Depression as a clinical syndrome is commonly described by feelings of sadness, despair, emptiness, or loss of interest or pleasure in activities occurring nearly every day for more than a 2-week period.[142] Although depression is a clinical disorder, all individuals experience transitory dysphoric feelings of sadness, despair, emptiness, or loss of interest or pleasure in activities in the course of their lives.

Dysphoric feelings are higher among those with headache than those without.[100,101,143–145] Heightened dysphoria increases the likelihood that stress will trigger a headache, increases headache pain severity, is a negative prognosticator for response to treatment,[16,93,94,146,147] and negatively influences a patient's level of satisfaction with his/her care.[122] In addition, hopelessness (a cognitive perception characterized by low internal LOC, low SE, and heightened pessimism) often accompanies dysphoric feelings[148] and increased headache-related disability.[93,101,103,143,149–151]

Anger

Anger is a state of displeasure ranging in intensity from mild irritation to intense fury in response to a perceived wrong that threatens the well-being of an individual or others with whom the individual identifies.[152–154] There is a great deal of inter- and intra-individual variability in the level of emotional intensity and physiological arousal one experiences when angry.[152–156] Although the extent whereby one becomes angry can influence the course and outcome of diseases,[157–159] how the individual expresses/manages their anger has a far greater impact on disease course and impact.[159,160] When considering how anger is expressed, researchers have identified 2 distinct styles of anger expression: anger-in and anger-out. Anger-in is when an individual does not outwardly express their anger, but experiences increased internal arousal[161,162] whereas anger-out involves physical acts (eg, slamming doors) or verbal expression (eg, sarcastic remarks[162,163]). Being able to express anger appears to lessen the negative impact of anger on emotional and physical function.[163–165] However, expressing anger can have negative consequences such as being "socially unacceptable," creating unpleasantness ("if you can't say anything nice, don't say anything at all"), and instigating perceived negative consequences.[152,155,166]

Individuals with headache are more likely to hold their anger in than persons without headaches.[100,104,144,145,167] Individuals who hold anger in experience increased pain severity[98] and failure to express anger leads to more disability.[96,98,101,124,168,169] However, being extremely high on anger-out increases pain sensitivity and disability.[170,171] Overall, either too much expression or inhibition of anger appears to have deleterious effects. Failure to adequately express one's anger also negatively influences patient-provider

communication.[123] This lessens the likelihood that headaches will be diagnosed adequately[123] and is a poor prognosticator of treatment outcome.[160]

Overall, inadequately managed NA increases the individual's risk for experiencing more headache attacks, more intense headache pain, and more headache-related disability via central, neurochemical, and peripheral routes.

CLINICAL IMPLICATIONS

The current paper has argued that viewing headache as a biopsychosocially influenced disease is more appropriate than considering biological factors alone. The paper has focused on how specific psychological factors (LOC, SE beliefs, and NA) can influence the development, course, and consequences of headache. Taking a biopsychosocial view of headache presents both a challenge and an opportunity to the practicing physician. The challenge is that these factors interact to influence the development, course, and consequences of headache in ways heretofore not fully elucidated. On the other hand, taking such a view allows the physician to utilize their clinical acumen by considering all aspects of the individual (biological, psychological, and social) when establishing treatment recommendations. Listed below are examples of how psychological factors can influence treatment recommendations and treatment outcome along with suggestions for how to address psychological factors.

1. Recognizing and managing triggers is recommended to prevent headache attacks and to prevent episodic headaches from becoming chronic.[172,173] However, if a patient believes they have little or no influence on whether they experience a headache attack (low internal LOC) and low SE for managing headache triggers, then it is unlikely they will follow this recommendation. Educating the patient about how triggers influence a headache attack, and how managing triggers can reduce the number of headache attacks, increases internal LOC. Having patients keep regular diaries of potential headache triggers allows the patient to see how triggers are related to headache.[174] Similarly, teaching skills for self-managing triggers will increase SE. Also, an individual with catastrophic fear about being exposed to a potential trigger will need to become aware that triggers can set the stage for headache, but exposure to a trigger does not mean they will experience a debilitating headache. The sage physician will elicit examples from the patient (or talk about other similar patients' experiences) in which exposure to the triggers did not result in a severe, debilitating headache, or in which the individual took medication (eg, triptan) that lessened/aborted the headache.

2. Taking headache medication as prescribed is imperative for maximum efficacy. However, a patient with low SE for taking medication will be less adherent to protocol. Having a medical staff member (eg, nurse) teach the individual (or model for them) how/when/why to take medication (eg, taking a triptan at the appropriate time) and then following up with the patient to reinforce adherence or discuss barriers to adherence will raise SE and increase adherence.

3. Current conceptualizations of headache, especially migraine, suggest that a "hyper excitable" brain is an important factor for experiencing headache.[84,175,176] Thus, interventions that seek to reduce the brain's excitability are theoretically sound. Patients whose stress, anxiety, and/or anger are not well managed will have heightened arousal and thus be at greater risk for experiencing a headache attack because of their influence on the brain's chemistry and excitability. The physician who normalizes the patient's need for managing stress ("A lot of patients find that doing something to take the edge off helps them deal better with life") and encourages

the patient to use evidence-based relaxation and stress management strategies will make it more likely the patient will adopt such strategies.

As the examples above have noted, there are clinically sound strategies for addressing psychological factors in headache. Although no validated treatment algorithms exist for knowing how and when to address psychological factors, there are certain strategies that likely enhance the efficacy of ongoing pharmacologic intervention. An efficacious and easily administered strategy is relaxation/stress-management.[177–179] Although most physicians appreciate the potential benefits of managing stress, many are surprised that these strategies can also improve SE for managing headache (and likely create a more internal LOC). Education strategies (eg, recognizing and managing triggers, taking medication as prescribed, understanding the "migraine" brain) can also improve patient outcomes and increase SE.[76, 78,180]

One of the difficulties of using these strategies has been integrating them into ongoing pharmacologic care and/or connecting the patient with a behavioral specialist. However, using a self-management model whereby the patient and physician partner together to maximize treatment benefits by allowing the patient a more active role in managing their disease has been used successfully in other chronic diseases and would likely work in headache as well.[87]

Addressing psychological factors is a low priority for certain headache sufferers. This includes patients who experience headache once a month or less, whose acute medications are fully efficacious at treating their headaches, and who experience no more than mild headache-related disability. There is also a subset of patients whose presentation make it highly unlikely that addressing psychological needs alone will have significant benefits until other issues are addressed.[181] This includes patients with continuous or near-continuous headaches, high levels of medication overuse, and those with severe depression. However, relatively few headache sufferers presenting for treatment fall into these categories and thus, almost all headache patients presenting for treatment will benefit from considering psychological risk factors in headache and addressing them as appropriate.

Acknowledgements

The authors would like to thank Ms. Megan Rochey for her assistance in preparing the manuscript, and Drs. Donald Penzien and Jeanetta Raino for their guidance in the direction of the manuscript. Supported by grant 1K23NS048288-01A2 from the National Institutes of Health.

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