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# STRESS, PERSONALITY AND MIGRAINE. A REVIEW OF PSYCHOSOCIAL AND NEUROPHYSIOLOGICAL RISK FACTORS

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## ABSTRACT

Migraine is a complex neurological and psychological condition that causes repeated headache attacks, sensitivity to light and sound, and major effects on quality of life. Modern research shows that migraine develops from the interaction between biological sensitivity, stress, and personality traits. This review combines findings from neuroscience, psychology, and clinical studies to explain how stress reactivity, emotional regulation, and coping style affect migraine onset and management. Understanding these biological and psychosocial links can improve treatment and support more effective combined approaches that use both medication and psychotherapy.

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## KEYWORDS

Migraine, Stress, Neurophysiology, Personality, Alexithymia, Coping, CBT, Biopsychosocial Model

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### 1. Introduction

Migraine is one of the most common and disabling neurological conditions in the world, affecting about 14% of the global population (GBD, 2021). The World Health Organization ranks migraine as the sixth leading cause of disability and the top cause among young women (Steiner et al., 2020).

In the past, migraine was viewed mainly as a vascular or neurological problem. Today, it is seen as a **biopsychosocial disorder**, where biological factors interact with stress, emotions, and the social environment (Engel, 1977; Buse et al., 2020). Many patients report that stress, emotional tension, and exhaustion are their main triggers. Psychological issues such as anxiety, depression, and alexithymia are also common among migraine sufferers (Sauro & Becker, 2009; Minen et al., 2016).

Certain personality traits- like high neuroticism, perfectionism, and emotional reactivity- are often linked to more frequent or intense migraine attacks (Dindo et al., 2014). These traits make individuals more reactive to stress and less flexible in coping. Because of this, stress does not only trigger migraine attacks but can also increase their frequency and duration (Savi et al., 2005).

This perspective shows that migraine should not be seen only as a neurological disorder, but as a condition influenced by how people experience emotions and manage stress. Understanding these interactions can help improve both prevention and treatment.

### 2. Migraine Overview and Definition

According to the *International Classification of Headache Disorders, 3rd edition (ICHD-3, 2018)*, migraine is a recurring headache disorder with attacks lasting from 4 to 72 hours. The pain is usually on one side of the head, pulsating, and moderate to severe in intensity. It is often accompanied by nausea, vomiting, and sensitivity to light and sound.

There are two main forms of migraine:

- **Migraine without aura** - the most common type, involving headache and related symptoms without neurological signs.

- **Migraine with aura** - includes additional sensory or visual disturbances that appear before or during the headache phase (ICHD-3, 2018).

Many patients also experience **prodromal and postdromal phases**, which can include irritability, tiredness, food cravings, or difficulty concentrating (Goadsby et al., 2017). These phases show that migraine is more than just pain; it is a disorder of the brain's regulation systems that involves both biological and psychological processes.

### 3. Neurophysiological Mechanisms of Migraine

Neurophysiological studies show that migraine involves problems in the brain's natural pain-control systems, especially those that depend on serotonin and endogenous opioids (Goadsby et al., 2017). Early researchers suggested that changes in serotonin levels play a key role in how migraines start and progress (Levin, 2009). These findings formed the basis of the "serotonin hypothesis."

Modern research confirms this idea. Drugs that lower serotonin or affect serotonin receptors can trigger migraine attacks, while medicines that activate serotonin receptors can stop them. This is why triptans, which act as serotonin 5-HT<sub>1</sub> receptor agonists, remain among the most effective treatments for stopping an attack (Goadsby et al., 2002). Preventive treatments often target the 5-HT<sub>2</sub> receptor system because serotonin influences both blood vessel tone and pain transmission (Bublij et al., 2013).

Brain imaging studies show that migraine aura is linked to cortical spreading depression (CSD) - a slow wave of neural activity across the cortex (Charles & Brennan, 2009). CSD activates the trigeminovascular system and causes the release of calcitonin gene-related peptide (CGRP), leading to inflammation and pain sensitization (Goadsby et al., 2017).

Genetic research suggests that some people inherit differences in serotonin or opioid metabolism that make their brains more excitable and their pain thresholds lower (Huang et al., 2019). Family studies also show a strong genetic component, with up to 70–80% heritability when both parents have migraine (Buse et al., 2020; Steiner et al., 2020).

However, genetics alone do not explain why migraine attacks vary so much between people. Stressful events, emotional strain, and fatigue are among the most common triggers (Sauro & Becker, 2009; Bigal & Lipton, 2008). Around 70% of patients report stress as their main or secondary trigger (Buse et al., 2012).

From a biological perspective, stress activates the hypothalamic-pituitary-adrenal (HPA) axis, increasing cortisol levels and changing how the brain processes pain (Huang et al., 2019). Long-term stress can reduce the effectiveness of serotonin and the brain's natural opioids, making migraine attacks more likely (Levin, 2009).

The link between stress and migraine works both ways. Repeated attacks create more stress, anxiety, and avoidance behaviors, forming a vicious cycle (Nicholson et al., 2007). According to Engel's biopsychosocial model (1977), this ongoing interaction between body, mind, and environment explains how migraines can become chronic.

Neuroimaging studies also show that stress changes the brain's emotional circuits -especially the amygdala, hypothalamus, and prefrontal cortex -which are involved in regulating both mood and pain (Minen et al., 2016). These findings confirm that emotional reactivity and stress management play key roles in migraine development and persistence.

### 4. Personality Traits and Emotional Vulnerability

#### 4.1 Neuroticism and Emotional Reactivity

People high in neuroticism are more likely to experience anxiety, worry, and negative emotions. Studies show that neuroticism is linked to more frequent and severe migraine attacks (Dindo et al., 2014). Such individuals often react strongly to stress and tend to focus excessively on their pain. This can increase sympathetic nervous activity and make them more vulnerable to headaches (Goadsby et al., 2017).

#### 4.2 Alexithymia and Emotional Awareness

Another common trait in migraine patients is alexithymia-difficulty recognizing and expressing emotions. Research shows that alexithymic patients often experience stronger pain and greater disability (Caputi et al., 2017). Because they cannot express emotions verbally, their tension may instead appear as physical symptoms like headaches. Brain imaging also shows reduced activity in the anterior cingulate cortex, a key area that connects emotions and pain (Belot et al., 2021).

#### 4.3 Perfectionism, Rigidity, and Harm Avoidance

Many people with migraine describe themselves as perfectionists or highly responsible individuals who find it hard to relax. While these traits can lead to success, they also cause constant self-pressure and internal stress (Savi et al., 2005). The temperament trait of harm avoidance-feeling anxious, cautious, and easily tired-has also been linked to more frequent migraines.

#### 4.4 Personality, Depression, and Anxiety

Long-term pain can lead to emotional exhaustion, depression, and sleep problems (Levin, 2009). Depression is about twice as common among migraine patients compared with the general population (Welch et al., 2006). This comorbidity worsens pain perception and makes treatment more difficult. Gender differences

are also observed: women with migraine often report more emotional instability, while men tend to suppress emotions and show higher muscle tension (Gasbarri et al., 2008).

#### *4.5 Psychosomatic Integration*

Classic psychosomatic theories, such as those by Alexander (1950) and Engel (1977), describe migraine as a physical expression of emotional conflict. Modern psychology supports this view, emphasizing that emotional regulation and stress response systems directly influence pain perception (Minen et al., 2016). Recognizing these emotional factors helps in creating personalized treatment plans. For example, patients who are highly reactive may benefit from cognitive-behavioral therapy (CBT), while alexithymic individuals may respond better to mindfulness or body-focused approaches.

### **5. Coping Strategies and Psychotherapeutic Interventions**

#### *5.1 Cognitive-Behavioral Therapy (CBT)*

CBT is one of the best-studied psychological treatments for migraine. It combines stress-management, relaxation, and cognitive restructuring. Studies show that CBT can reduce headache frequency by 30-50%, improve quality of life, and help patients reduce medication use (Nestoriuc & Martin, 2007). It teaches individuals to recognize negative thoughts and behaviors - such as catastrophizing - that make migraine worse (Penzien et al., 2003).

#### *5.2 Mindfulness and Acceptance-Based Therapies*

Mindfulness-based stress reduction (MBSR) teaches awareness of the present moment and helps reduce emotional reactivity. Clinical trials show that it can lower headache severity and improve coping confidence (Morone et al., 2008). Acceptance and Commitment Therapy (ACT) encourages flexibility and acceptance of pain, helping patients live meaningful lives even with migraine (Schmidt et al., 2014).

#### *5.3 Biofeedback and Relaxation Training*

Biofeedback provides real-time information about body functions such as muscle tension or skin temperature. With training, patients can learn to control these functions and reduce stress. Combining biofeedback with relaxation techniques helps normalize nervous system activity and reduces migraine frequency (Nestoriuc & Martin, 2007).

#### *5.4 Integrative and Multimodal Approaches*

The best outcomes come from combining psychotherapy, exercise, lifestyle changes, and medication (Buse et al., 2013). Tailoring psychological methods to each patient's personality and emotional profile improves adherence and prevents migraines from becoming chronic.

### **6. Discussion**

This review highlights how biological, psychological, and social factors interact in migraine. Neurological dysfunction- such as problems with serotonin and HPA axis regulation-works together with emotional and cognitive patterns like anxiety, perfectionism, and poor coping.

Personality traits such as neuroticism or alexithymia make individuals more reactive to stress, leading to stronger and more frequent headaches. These findings support the biopsychosocial model, which explains migraine as the result of both brain function and emotional experience.

While medication is essential for treating and preventing attacks, psychological therapies -especially CBT, mindfulness, and biofeedback-help reduce stress, improve emotion regulation, and break the cycle between stress and pain.

### **7. Conclusions**

Migraine is a complex disorder that involves both brain dysfunction and psychological stress. Stress acts as both a trigger and a result of migraine, forming a feedback loop that can make attacks chronic. Personality traits and emotional patterns affect how people experience and manage migraine.

Combining pharmacological and psychological approaches - such as CBT, mindfulness, and biofeedback - can improve outcomes and quality of life. A holistic, biopsychosocial approach that considers the connection between brain, mind, and environment is key to effective migraine management.

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All authors approved the final version of the manuscript and agreed to its submission to Journal of Innovative Technologies in Social Science.

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