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WHAT WE KNOW ABOUT MIGRAINE TRIGGERS: EVIDENCE FROM RESEARCH

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ABSTRACT

Aim of the study: The aim of this review was to summarize current knowledge about factors that may trigger migraine attacks and to assess how reliably these triggers are described in the literature.
Materials and methods: The literature was reviewed using medical databases such as PubMed and UpToDate, as well as other relevant sources published over the last 30 years.
Results: The studies showed that people with migraine report many different triggers, but their importance varies widely between individuals. Stress, sleep problems, bright light, strong smells, hormonal changes, and certain eating or drinking habits are most often mentioned, but not all patients react to the same factors. Many papers also highlight that some "triggers" may simply reflect early warning symptoms of an upcoming migraine attack, which makes interpretation difficult. Overall, the evidence suggests that triggers affect a person's general sensitivity rather than directly causing an attack.
Conclusions: Migraine triggers are highly individual, and they should be seen more as factors that raise someone's vulnerability rather than direct causes. For many patients, keeping a headache diary and observing their own patterns can be more useful than trying to avoid long lists of possible triggers. More well-designed studies are needed to better understand how specific factors influence migraine and how this knowledge can improve prevention.

KEYWORDS

Migraine, Trigger Factors In Migraine, Lifestyle Factors And Migraine, Susceptibility Threshold, Migraine Attack Initiation

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Introduction

Migraine is a chronic neurological disorder and one of the most common diseases worldwide [1,2]. According to the GBD 2015 study, migraine is the third leading cause of disability among people under 50 [3]. It is estimated that migraine affects approximately one in ten people globally [4]. In recent decades, both the number of cases and migraine-related DALYs have increased substantially [4,5,6]. Migraine attacks—characterized by pulsating headache, photophobia, phonophobia, and nausea [1,7]—significantly reduce patients' quality of life and generate considerable social and economic burden. Advances in neuroscience and neuroimaging are improving our understanding of migraine's neurobiological mechanisms. This supports more effective treatment and better identification of factors that may precipitate an attack.

In this context, migraine-triggering factors represent an important area of interest. These triggers include both internal (endogenous) and external (exogenous) elements that appear to increase the likelihood of an attack in susceptible people. Identifying these factors may help guide strategies for preventing migraine attacks [8]. However, the literature also highlights substantial variability and methodological limitations in studies on this topic [9,10]. Furthermore, growing evidence suggests it is not a single trigger, but rather the accumulation of several factors—combined with a lowered attack threshold—that may play a crucial role in initiating a migraine episode [2].

The aim of this review is to present the current state of knowledge regarding factors that may trigger migraine attacks.

Definition of migraine

Migraine is classified among primary headache disorders and is defined as a chronic, episodic condition characterized by recurrent headache attacks with specific features and associated symptoms, not caused by another underlying problem [7]. According to the International Classification of Headache Disorders, 3rd Edition (ICHD-3), the main subtypes include migraine without aura, migraine with aura, and chronic migraine—diagnosed when headache occurs on ≥15 days per month for at least 3 months, with at least 8 days per month fulfilling criteria for migraine [7]. Clinically, a migraine attack typically presents as unilateral, pulsating pain of moderate to severe intensity, that could get worse with normal physical activity. Common symptoms that could accompany it include nausea, vomiting, and increased sensitivity to light, sound, or smell [1,7]. In the case of aura, transient focal neurological symptoms—most commonly visual, sensory, or speech disturbances—may occur before or during the headache phase, preceding pain in over 80% of migraine with aura cases [11,12]. Additionally, some patients experience prodromal and postdromal phases, which may include hyperactivity, hypoactivity, low mood, food cravings, repetitive yawning, fatigue, and neck stiffness or pain [7]. These features highlight that the clinical picture of migraine extends beyond the headache phase alone.

Etiology and pathogenesis of migraine

Despite many years of research, the etiology of migraine remains only partially understood. It is currently considered a disorder with a complex genetic and neurobiological basis involving ion channel dysfunction, neuronal mechanisms, and a vascular component [13,14]. Studies indicate that migraine—particularly its rare forms, such as Familial Hemiplegic Migraine (FHM)—can be viewed as a channelopathy associated with mutations in genes such as CACNA1A, ATP1A2, and SCN1A [14]. These abnormalities lead to neuronal hyperexcitability and greater susceptibility to cortical spreading depression (CSD), a key mechanism behind migraine aura [15].

CSD activates the trigeminovascular system, resulting in the release of pain-related neuropeptides, mainly Calcitonin Gene-Related Peptide (CGRP) and Pituitary Adenylate Cyclase-Activating Polypeptide (PACAP), which link the neuronal and vascular components of the migraine attack [16,17].

CGRP

CGRP is a neuropeptide synthesized in sensory neurons of the trigeminal ganglion. Its levels increase during a migraine attack, and CGRP infusion can provoke an attack in susceptible patients [16]. Drugs targeting CGRP or its receptor—monoclonal antibodies and small-molecule antagonists (gepants)—demonstrate high efficacy in migraine prevention and acute treatment [16]. CGRP contributes to migraine pathophysiology through meningeal vasodilation, activation of the trigeminovascular system, and induction of neurogenic inflammation [16].

PACAP

PACAP-38 is another neuropeptide with an important role in migraine pathogenesis. Clinical studies have shown elevated PACAP levels during attacks and the ability of PACAP infusion to induce migraine-like symptoms [17,18]. Its mechanism appears to be partly independent of CGRP, as PACAP-38 can induce an attack even when the CGRP pathway is pharmacologically blocked [17,18]. PACAP is believed to influence not only the pain phase but also prodromal symptoms such as mood changes, photophobia, and sleepiness [17]. It acts mainly through PAC1, VPAC1, and VPAC2 receptors, making it a distinct and promising therapeutic target [17].

Other Neuropeptides and Neuronal Mechanisms

In addition to CGRP and PACAP, other neuropeptides contribute to migraine pain, including substance P and neurokinin A, which are co-released from trigeminal nerve endings and increase vascular permeability and neurogenic inflammation. Vasoactive Intestinal Peptide (VIP), released by parasympathetic nerve fibers, may play a role in autonomic symptoms that occur during migraine attacks, such as tearing or a blocked nose [17]. Recent interest has also focused on amylin and adrenomedullin, peptides from the CGRP family. They show similar vasodilatatory effects and may participate in the broader “neuropeptide axis” of migraine [17].

Neuroimaging studies have demonstrated activation of brainstem structures during attacks—particularly the dorsolateral pons and periaqueductal gray—highlighting the central role of the brain in generating and sustaining migraine pain [15]. Dysfunction of serotonergic and dopaminergic systems also contributes to pathogenesis: serotonin regulates vascular tone (which explains the effectiveness of triptans), while dopamine may be responsible for prodromal symptoms such as sleepiness or nausea [15].

Integrated Model of Migraine Pathophysiology

An integrated model of migraine pathophysiology can therefore be summarized as follows:

genetically driven ion channel dysfunction → neuronal hyperexcitability → CSD → neuropeptide release (CGRP, PACAP, SP, VIP) → trigeminovascular activation → vasodilation and neurogenic inflammation → pain symptoms [15,16,17].

This framework explains the episodic nature of migraine, its diverse clinical manifestations, and the effectiveness of therapies targeting neuropeptide pathways.

Migraine attack triggers - characteristics and prevalence.

Migraine attack triggers are a broad and varied group of factors that patients report as preceding or provoking a headache. The term '*trigger'* does not refer to a specific biological mechanism. Instead, it includes many different environmental, behavioral, physiological, and sensory influences that may influence the sensitivity of a nervous system already predisposed to migraine [19,20,21,22]. What is important is that triggers are highly individual—relevant for some patients while completely irrelevant for others—and they can change over time, reflecting the variable clinical presentation of migraine [19,20].

Early work on migraine triggers, such as the classic study by Kelman (2007), laid the foundation for contemporary descriptions of these factors. In his retrospective analysis of 1,750 migraine patients, as many as 95% could identify at least one factor they believed appeared before their attacks [21]. The most frequently mentioned triggers were stress (79%), hormonal fluctuations related to the menstrual cycle (65%), hunger or skipping meals (57%), fatigue (56%), not enough or too much sleep (50%), bright light (38%), weather or pressure changes (53%), odors (36%), and alcohol (25%). Kelman also pointed out that many patients mentioned several triggers at the same time, which made it hard to identify the one actually linked to the attack. Although these findings served as a reference point for many subsequent analyses, the author emphasized the limitations of retrospective methods and the potential influence of recall bias [21].

A more recent and highly comprehensive study was carried out by van Casteren et al. (2021), including more than 7,000 women and over 1,000 men with migraine. This study provides a modern overview of the most frequently reported triggers and clearly shows differences between women and men. Among women, the most common trigger was menstruation (78%), followed by stress (77%) and bright light (69%). Among men, the leading factors were stress (69%), bright light (63%), and lack of sleep (60%) [19]. The study is especially valuable because of its large sample size and detailed analysis of different trigger categories.

Other cross-sectional studies focusing on specific populations also offer important insights. Athar et al. (2022) analysed 393 migraine patients and found high rates of lifestyle-related triggers. The most frequently reported were sleep problems (70.5%), stress (66.7%), fatigue (64.4%), as well as triggers typical of modern life, such as excessive screen time (61.1%) and loud noise (58.8%) [20]. The strong influence of screen exposure is particularly interesting in the context of rapid technological changes, suggesting that in younger adults, it may be becoming one of the leading triggers.

Rustom et al. (2022) obtained similar results in a student population, where the most common triggers were insufficient sleep (74.7%), stress (58%), and skipping meals (57.3%) [22]. These findings highlight that, among young people, factors such as irregular lifestyles, study-related stress, and disrupted daily rhythms play a key role.

The importance of current data in understanding triggers

Studies from the past decade show that some triggers remain consistent over time—stress, sleep disturbances, bright light, and fatigue are reported most often across populations [19,20,22]. At the same time, new triggers are becoming increasingly relevant, especially those linked to modern technology and lifestyle changes, such as prolonged screen use [20].

Recent data also highlight notable sex differences, particularly regarding hormonal factors, as well as differences between age groups. It is also important to keep in mind that some reported “triggers” may in fact be early symptoms of the prodromal phase of a migraine attack, which makes interpreting such reports more complicated [9,23].

Methodological challenges – why proving cause and effect is difficult

Research on migraine triggers faces several methodological difficulties that make it hard to determine whether a trigger truly causes an attack. Most available studies rely on retrospective reports and self-completed questionnaires, which are highly vulnerable to recall bias. Patients may overestimate the importance of well-known triggers, such as stress or weather changes, because these factors match their expectations or are widely discussed in society [9]. This can also lead to confirmation bias, in which both patients and clinicians unintentionally reinforce the role of commonly accepted triggers [9,24]. Another challenge is the overlap of multiple factors. For example, stress can affect sleep, appetite, and muscle tension, making it difficult to determine which of these actually plays the key role [9,25]. A further issue is confusing prodromal symptoms with triggers. Symptoms such as tiredness, neck stiffness, mood changes, or concentration problems can appear several hours before the headache starts. Many patients, however, interpret them as outside triggers, even though they are actually early signs of the migraine itself. In the classic study by Giffin et al. (2003), patients recognized an approaching attack from these early symptoms in roughly 72% of cases [23]. More recent analyses using electronic diaries confirm that the perception of environmental stimuli (e.g., light, smells) can be influenced by increased neuronal hyperexcitability that begins even before the onset of pain [9,24,26].

1. ENVIRONMENTAL TRIGGERS

1.1 Weather Conditions

The relationship between weather conditions (such as temperature, atmospheric pressure, humidity, and wind) and the frequency of migraine attacks has been the subject of numerous studies. In a retrospective analysis of headache diaries collected over one year from 20 patients, attacks were found to occur more frequently during periods of lower temperature and higher humidity. In a subgroup of patients, a statistically significant association was observed between changes in meteorological variables and migraine attacks [27]. However, the authors highlighted substantial individual variability and limitations related to the small sample size. A broader review and meta-analysis including 31 studies reported that weather conditions may represent an important factor modulating migraine occurrence—particularly temperature and atmospheric pressure—whereas findings concerning humidity were inconsistent [28]. Furthermore, studies suggest that the effect of weather conditions may be indirect, for example, through disturbances in sleep, changes in physical activity, or mood deterioration, all of which can themselves facilitate migraine attacks. For this reason, atmospheric factors are considered relevant for some patients; however, their overall clinical significance appears to be moderate and dependent on individual sensitivity [27].

1.2 Light

Light is one of the triggers most commonly reported by patients who feel that it can either precede or provoke a migraine attack. In survey studies, people with migraine often indicate that exposure to bright sunlight, glare, reflections, or flickering artificial light tends to occur shortly before their headache begins [29]. Similar reports concern high-contrast visual stimuli or repetitive geometric patterns, which is consistent with research on so-called pattern glare—a phenomenon observed more frequently in individuals with migraine than in healthy controls [29].

The importance of flickering visual stimuli is supported by controlled psychophysical studies. These studies show that people with migraine have lower contrast tolerance and experience more discomfort when exposed to flickering light across different frequencies [30]. This suggests that light flicker—found, for example, in certain fluorescent lamps or low-refresh-rate screens—may be a particularly unpleasant type of visual stimulus, often mistaken by patients for a direct trigger.

However, patients' beliefs about the role of light are not always backed up by careful prospective or provocation studies. In one controlled experiment, people with migraine with aura were shown stimuli they believed could trigger an attack, but only a small number actually experienced a full migraine episode in the laboratory [31]. This indicates that self-reported environmental triggers do not consistently induce attacks, highlighting the gap between perceived causality and objective evidence.

The interpretation of such findings is further complicated by the fact that, in some patients, light sensitivity appears already in the premonitory phase of the attack. Research shows that photophobia may occur hours before headache onset, functioning as part of the developing attack rather than its cause [32]. In these cases, patients may mistakenly attribute light as a trigger even though their reaction is actually an early prodromal symptom.

Survey data must therefore be interpreted with caution. Studies comparing retrospective patient reports with prospective headache diaries reveal considerable discrepancies: patients identify light as a trigger far more often in retrospective questionnaires than is reflected in their real-time diary entries [33]. This suggests that some reports may arise from recall bias, misattribution, or overlap between the premonitory phase and environmental stimuli.

Understanding the mechanisms underlying light sensitivity helps explain why people with migraine may react strongly even to moderate illumination. Current models suggest that migraine-related photophobia results from a complex interaction between retinal and central nervous system processes[34]. Neurophysiological studies show that people with migraine have increased excitability of the visual cortex, leading them to respond more strongly to flickering light and high-contrast patterns. At the same time, accumulating evidence indicates that intrinsically photosensitive retinal ganglion cells (ipRGCs), which react to light and transmit signals to deep brain structures, including those involved in pain modulation, may contribute to migraine. Their activation could increase the excitability of the trigeminal system, which plays a key role in generating migraine pain. Light may also influence brainstem nuclei that integrate sensory and pain information, thereby amplifying pain perception when the nervous system is already in a state of heightened sensitivity. These mechanisms, described in one of the recent reviews, show that light sensitivity in migraine is not only a symptom of an attack but also a result of the unique way the nervous system functions in this condition [34].

1.3 Noise

Loud noise or sound with rapidly fluctuating intensity is a frequent external trigger reported by people with migraine, serving as a factor that exacerbates symptoms. In a quantitative study assessing the sound aversion threshold (SAT), patients with migraine showed clearly lower thresholds than healthy controls, both between attacks and during attacks [35]. These findings suggest the presence of auditory hypersensitivity in migraine, which could explain why noise may be perceived as a trigger or act as an exacerbating factor in susceptible individuals.

Analogous to light, auditory stimuli such as noise may function both as an environmental factor and as one of the prodromal symptoms of a migraine attack [35,36].

1.4 Odors

In a study by Silva-Néto et al., individuals with migraine most commonly identified perfumes, paint, gasoline, and cleaning agents as smells that can provoke an attack [37]. Another study demonstrated that interictal hypersensitivity to odors (“osmophobia”) is associated with longer disease duration and greater clinical burden [38]. Odors can therefore be considered a credible trigger in certain patients, although their role—similar to that of other sensory stimuli—is modulated by the individual's sensitivity of the nervous system [37,38].

2. DIETARY TRIGGERS

2.1 Food triggers

Survey studies show that people with migraine most often mention foods like chocolate, aged cheeses, citrus fruits, tomatoes, nuts, products with monosodium glutamate (MSG), nitrates, and artificial sweeteners (e.g., aspartame) as possible triggers [39,40]. A systematic review that included 43 surveys, cross-sectional, and interventional studies found that patients identified a wide range of dietary triggers. However, the reviewers noted that most of the included studies (around 68%) were observational or questionnaire-based, which limits the strength of the evidence. Overall, chocolate, cheese, alcohol, and caffeine were among the most frequently mentioned dietary factors [39]. Another narrative review also listed aged cheese, chocolate, tyramine, histamine, nitrates, and aspartame as potential migraine triggers, while pointing out the methodological limitations of many studies [40].

The mechanisms by which foods might play a role in triggering migraine attacks are complex and not yet fully understood. Biogenic amines such as tyramine, histamine, and phenylethylamine occur in fermented products, aged cheeses, chocolate, and wine. These compounds may affect the release of neurotransmitters, such as serotonin, which can potentially affect vascular tone and possibly contribute to an attack [40].

MSG is another substance suggested to increase neuronal sensitivity. However, intervention studies have shown mixed results. When MSG is added to regular foods, it rarely leads to headaches, while studies using MSG in solution suggest a possible increase in migraine episodes. Still, many of these studies lacked proper blinding, making their results less reliable [41].

Dietary sources of nitrates may increase nitric oxide (NO) production. This leads to vasodilation and activation of the trigeminal system. Experimental studies using nitric oxide (NO) donors, such as nitroglycerin, have shown that these substances can trigger migraine-like headaches with a delay in people who are sensitive to them [42,43,44]. It is also possible that some of the dietary effects reported by patients are influenced by their expectations (placebo/nocebo effects) rather than by the foods' actual biological actions [39]. In addition, the body's metabolic state at the time of eating may interact with a potential food trigger. States such as dehydration, skipping meals, hunger, or fluctuations in blood glucose may increase the likelihood of an attack [39,40].

2.2 Dehydration

In one survey study involving 95 people with migraine, 34 participants (36%) identified insufficient fluid intake as a factor that tended to precede an attack, especially in situations that increase water loss, such as physical exertion or high temperatures [45]. In a pilot study, increasing daily water intake by about 1.5 liters for 2 weeks in individuals with recurrent headaches, including migraine, was associated with fewer total headache hours and improved overall well-being, although not all results reached statistical significance [46]. A larger randomized trial conducted by the same research team showed that raising daily water intake led to a meaningful improvement in headache-related quality of life (MSQOL) and more frequent reports of subjective improvement. However, the impact on the number of days with moderate headache was small [47].

Reviews of the literature highlight that evidence for a direct link between dehydration and migraine is still limited—most available studies are observational or pilot-level, and randomized clinical trials involve relatively small patient samples [48,49,50]. At the same time, because proper hydration is safe, inexpensive, and potentially helpful, maintaining adequate fluid intake may serve as a useful component of behavioral migraine prevention.

2.3 Caffeine

Caffeine has a complex effect on migraine attacks. On the one hand, it may ease pain during an attack or enhance the effect of pain-relieving medications. On the other hand, both high habitual intake and sudden withdrawal in regular users can increase the risk of a migraine attack [51,52].

Review studies show that the percentage of patients reporting caffeine as a trigger varies across publications, with figures ranging from several to over a dozen percent (e.g., 6–14% in selected studies) [51,53]. Headache following caffeine withdrawal has also been described [53]. In a small clinical study on abrupt caffeine discontinuation, most participants (7 out of 9 individuals) experienced a migraine attack, suggesting that suddenly stopping regular intake may act as a trigger— although the low participant count means the results should be interpreted with caution [52]. A prospective diary study found that consuming three or more caffeine-containing drinks in a single day was linked to increased risk of migraine either that same day or the following day, whereas intake of one or two servings was generally not linked to a higher risk [55].

In clinical practice, monitoring each patient's individual response is recommended, for example, by keeping a record of caffeine intake and migraine attacks. It may also be helpful to limit intake to about ≤200 mg of caffeine per day or to reduce it gradually if the patient notices a connection between caffeine use and attacks. Because available studies are heterogeneous—with many relying on retrospective or survey-based designs and relatively few randomized controlled trials—therapeutic decisions should be individualized [51,52].

2.4 Alcohol

Alcohol consumption is commonly reported by people with migraine as a possible trigger for attacks, although the available evidence is variable and of limited quality. In a large survey including 2,197 migraine patients, 35.6% identified alcohol as a trigger, and more than 25% of those who had stopped drinking or never consumed alcohol did so out of concern that it might provoke an attack. In this group, red wine was most often reported as a potential trigger (77.8%), although only 8.8% of participants stated that it consistently provoked an attack. For about one-third of patients, a migraine appeared within three hours of drinking, and for almost 90%, it started within ten hours. This pattern suggests that the mechanism underlying these attacks differs from the typical "hangover" headache [56].

Mechanistically, it is assumed that attacks may be triggered not only by ethanol itself but also by other components present in alcoholic beverages, such as histamine, tyramine, tannins, sulfites, prostaglandins, congeners, and phenolic compounds [57,58]. Many analyses emphasize that the type of alcohol matters—red wine is reported much more frequently than spirits such as vodka or whisky [58].

Literature reviews indicate that about one-third of migraine patients report alcohol as a potential trigger, but only around 10% consider it a regular or consistent trigger [58,59]. Moreover, the reported frequency varies significantly across populations (ranging from 1.4% to 6.1% in some cohorts), suggesting that individual, genetic, and environmental factors play a role [58].

Interesting insights come from a prospective cohort study in which 487 individuals with episodic migraine were monitored for at least 90 days. Interesting insights come from a prospective cohort study in which 487 individuals with episodic migraine were monitored for at least 90 days. The results did not show a clear increase in the chance of a migraine on the day following alcohol consumption. Moreover, alcohol intake two days earlier was linked to a slightly lower chance of an attack [60]. These findings suggest that alcohol does not affect migraines in the same way for everyone, and the connection is not simple.

2.5 Hunger/skipping meals

A classic experiment from 1997 showed that a 19-hour fast significantly increased headache frequency in people with frequent migraines or tension-type headaches compared with a control group [61]. This finding suggests that hunger may function as an independent trigger of headache attacks, although it should be noted that the study included mixed patient populations and that other factors, such as stress, could also influence the onset of headaches. In a study of people fasting during Ramadan, migraine attacks were more common in the first few days of fasting, then gradually decreased as participants' bodies adapted [62]. This indicates that fasting can trigger attacks, but the body may develop some tolerance to the energy shortage over time.

Analyses of glucose levels during and between migraine attacks have shown dynamic fluctuations. In many cases, blood glucose rises during an attack, which may represent a compensatory response to an earlier energy deficit. However, the authors emphasize that higher glucose levels may also reflect a stress response to pain rather than a mechanism specific to migraine [63]. It is still unclear whether changes in glucose levels are a cause of the attack or simply a consequence of the stress and physiological changes associated with pain.

Contemporary research suggests that migraine may, at least in part, involve an energy-processing dysfunction in the brain. According to the neuroenergetic hypothesis, neurons in people with migraine are less efficient at using glucose and generating ATP [64]. When glucose levels drop (for example, during fasting), their energy reserves run out more quickly. This may lead to membrane depolarization, activate pain pathways, and ultimately trigger a migraine attack. It should be noted, however, that this remains a theoretical model and has not yet been fully confirmed in clinical studies.

Some patients also experience reactive hypoglycemia after meals, which could explain attacks occurring not only during hunger but also following a rapid rise and fall in blood sugar [65]. Supporting this idea are studies indicating that stabilizing blood glucose—through low-glycemic or ketogenic diets—may reduce the frequency of migraine attacks [66]. Nevertheless, most of the available research is observational or based on small patient groups, so larger randomized intervention studies are still needed.

3. HORMONAL FACTORS

Women experience migraine much more often than men, and the risk increases during periods of significant hormonal fluctuations, such as the menstrual cycle, pregnancy, and menopause (67). Variations in estrogen, progesterone, prolactin, and neurosteroid levels modulate the excitability of neurons within the trigeminal system, potentially lowering the threshold for migraine attacks. Particular attention has been given to the interactions of estrogen and oxytocin with CGRP — a key neuropeptide responsible for nociceptive activation and the transmission of migraine pain. Literature reviews suggest that hormonal modulation of CGRP may help explain sex-related differences in the prevalence and severity of migraine (68).

3.1 The "estrogen withdrawal" mechanism

The most well-established hormonal mechanism triggering migraine attacks is the rapid drop in estrogen levels that occurs during the perimenstrual period. A randomized study showed that perimenstrual estradiol administration reduces the number of migraine days, while its abrupt discontinuation leads to an increased frequency of attacks. This suggests that stable estrogen concentrations have a protective effect, whereas a sudden decline can initiate a migraine episode (69).

An animal study has shown that sudden estrogen withdrawal heightens vulnerability to cortical spreading depression (CSD), the electrophysiological substrate of migraine aura (70). Clinical and population-based data further support the hypothesis that the "estrogen withdrawal" mechanism is a principal factor underlying menstrual migraine (71).

3.2 Neurosteroids and progesterone

Neurosteroids such as allopregnanolone — a metabolite of progesterone — modulate neuronal excitability through GABA_A receptors. A cross-sectional study found that women with perimenstrual migraine have lower allopregnanolone levels compared with healthy controls, which may indicate reduced GABAergic inhibition and increased nociceptive susceptibility (72). Another study confirmed abnormal neurosteroid patterns in patients with both migraine and cluster headache, suggesting a broader dysregulation of the steroid system (73).

3.3 Prolactin and trigeminal nerve sensitivity

Prolactin may increase the excitability of trigeminal neurons. In an animal model of endometriosis, elevated prolactin levels were shown to enhance the sensitivity of trigeminal nociceptors, while pharmacological blockade of prolactin reduced this hypersensitivity (74). Preclinical studies suggest that prolactin levels may increase during migraine attacks in some patients, pointing to its possible use as a biomarker (75). An experimental study has also shown that long-term exposure to prolactin increases CGRP expression and makes trigeminal neurons more sensitive, further supporting prolactin's proposed role in modulating migraine pain (76).

3.4 Oxytocin

Oxytocin has pain-relieving effects and influences the trigeminal system. Research suggests that it may inhibit CGRP release and modify nociceptive processes associated with migraine (68). Because of these features, it may serve as a potential therapeutic target.

3.5. Androgens and migraine

Although migraine occurs more often in women, androgens also influence pain-system excitability and may affect migraine risk. Men with chronic migraine have been reported to have lower testosterone levels compared with the general population (77). Findings from a pilot study suggest a possible protective effect of androgens; testosterone implants in women were associated with reduced frequency and severity of migraine attacks (78). Although preliminary, these data suggest a potential modulatory influence of testosterone.

4. LIFESTYLE TRIGGERS

4.1 Stress

Psychological stress has long been one of the most frequently reported factors preceding migraine attacks, and both prospective and experimental studies have helped to clarify the nature of this relationship. One of the best-documented phenomena is the so-called let-down headache, meaning an increased risk of an attack after a sudden drop in stress levels. In a prospective study by Lipton et al., in which patients reported their daily stress levels, day-to-day decreases in stress were linked to a higher likelihood of an attack within 6, 12, and 18 hours. After accounting for individual differences, the absolute stress level was no longer a significant factor, suggesting that the body's physiological response to the shift from tension to relaxation may activate mechanisms that lead to an attack [79].

A link between day-to-day stress changes and migraine attacks has also been shown in research using electronic headache diaries. In one study, stress was reported as a leading pre-attack factor and appeared in 27.6% of headache days, suggesting that this pattern is highly consistent among patients who record their symptoms daily [25]. Prospective observations further indicate that stress reported before an attack is common, although it is still uncertain how much of this reflects a true triggering effect versus an early sign of the prodromal phase.

Important insights into the differences between objective and subjective stress come from the study by Schoonman et al., which measured perceived stress and biological markers of the stress response (salivary cortisol, heart rate variability) in the days preceding spontaneous attacks. Although some patients reported increased psychological tension before an attack, no significant changes were detected in the objective stress markers [80]. This suggests that the stress they report may stem from early prodromal symptoms rather than a genuine rise in the body's biological stress response.

A study using Ecological Momentary Assessment (EMA), which allows repeated measurements throughout the day, paints a more mixed picture. One analysis found that stressful events on attack days were not more frequent than on pain-free days [81]. This result also suggests that patients may recall stress after the

fact and link it to their attack, even though its actual role as a trigger may be smaller than what subjective reports imply [80,81]

Additional insights come from recent research on stress mechanisms, particularly regarding the hypothalamic–pituitary–adrenal (HPA) axis. One study suggested that during periods of high stress, increased cortisol levels may have a protective effect, whereas the later drop—restoring 'pain vulnerability'—might help trigger an attack [79]. The idea of neuroendocrine changes after stress is partly supported by experimental studies, though primarily in animal models. In a recent investigation using a mouse model, researchers manipulated HPA axis activity after stress exposure. Interventions targeting the hormonal response - such as inhibiting corticosterone production with metyrapone - were found to reduce later pain hypersensitivity resembling migraine [82]. These results suggest a potential role for HPA axis alterations during the "recovery from stress" period, although the evidence remains preclinical and requires confirmation in human studies. Overall, the findings support the idea that not only stress itself, but also the body's transition out of a stress state, may influence vulnerability to an attack [79,82].

Bringing together findings from prospective studies, EMA research, ambulatory monitoring, and experimental models, it becomes evident that stress plays a significant yet multifaceted role in triggering migraine attacks. Key factors seem to include both subjective fluctuations in psychological tension and neuroendocrine changes following stress, rather than stress alone.

4.2 Sleep disturbances

One key study on sleep in migraine patients is the 2013 polysomnographic work by Engstrøm et al. The authors found more frequent awakenings, shorter total sleep time, and altered NREM stages in people with migraine. Interestingly, these patients do not always report poorer subjective sleep quality. The study also found reduced pain thresholds and signs of sleep dysregulation already present in the pre-attack phase [83]. This suggests sleep disturbances may act as triggers and reflect early trigeminovascular activation. Taken together, these findings strongly support the view that sleep is an integral component of migraine pathophysiology rather than merely a consequence of it.

The mechanisms through which sleep disruption may increase susceptibility to migraine attacks are illustrated by an experimental study by Mykland et al. (2022). Under controlled sleep-restriction conditions, the authors assessed cortical excitability using transcranial magnetic stimulation (TMS). They found that short-term sleep reduction weakens GABAergic inhibition, especially in patients with migraine with aura [84]. This reveals a mechanism of increased neuronal excitability—consistent with the concept of migraine as a state of cortical hypersensitivity—that promotes the initiation of phenomena such as cortical spreading depression. The study provides experimental evidence supporting a causal/mechanistic link: insufficient sleep disrupts the balance between excitation and inhibition, potentially increasing the risk of attack onset.

Although sleep loss is a classic trigger, growing evidence shows that circadian irregularity may be just as important. A 2018 study reported that people with migraine often exhibit delayed melatonin onset (DLMO) and a mismatch between their internal biological clock and actual sleep Times [85]. This "circadian misalignment" was significantly associated with a higher number of migraine days. A common example is social jet lag—the difference in sleep timing between workdays and weekends. Sudden shifts in sleep schedules can disrupt the circadian system, thereby increasing the risk of attacks.

A cohort study published in 2017 showed that patients with migraine more often display a "rigid" circadian rhythm, meaning they have reduced ability to adapt to changes in sleep or activity timing. Moreover, both morning-type and evening-type chronotypes were associated with greater vulnerability to circadian disruption and, consequently, a higher risk of attacks [86]. These findings suggest that migraine may involve limited flexibility of the biological clock, making even small shifts in sleep schedules—such as those caused by travel, changes in work hours, or academic demands—potential triggers.

4.3 Physical exertion

Physical exertion has long been considered a potential trigger for migraine attacks, although its role remains unclear [87,88,89,92]. Patients fairly often report that intense or sudden effort precedes the onset of head pain [87]. However, both epidemiological data and clinical observations suggest that this relationship depends on many factors [87,88,92]. In one clinical study involving 103 people with migraine, 38% reported having experienced an attack directly after physical activity at least once. These individuals also showed a somewhat different clinical profile: for example, they more frequently reported neck pain early in the attack

and were more likely to avoid sports activities out of fear of provoking pain. This points to the possible influence of behavioral factors [87].

In a large population-based study, the link between activity level and migraine frequency appeared less clear. No meaningful association was found between physical activity and headache occurrence, although less active individuals reported headaches more often [88]. These results should be interpreted with caution because reverse causality is possible—people with frequent attacks may limit their activity, which can distort the observed relationship.

Provocation studies provide additional insight, showing that exercise can trigger a migraine attack in controlled conditions. In one such study, participants who identified physical effort as a trigger completed an exercise test to the point of exhaustion. Some of them developed a migraine within several hours afterward [89]. This suggests that very intense exertion may provoke attacks in susceptible individuals, although the reaction was inconsistent across participants. This pattern indicates that additional factors may be required and highlights the importance of exercise intensity—attacks were most often observed after very strenuous activity and less often after moderate effort.

Interestingly, results from intervention-based research highlight a different perspective on the effects of regular physical activity. A randomized study showed that performing aerobic exercise three times per week reduced the frequency of migraine attacks to a degree comparable to that of relaxation training and similar to the efficacy of topiramate in primary endpoint evaluations [90]. Likewise, a meta-analysis of aerobic exercise trials suggested a moderate reduction in migraine days among individuals who adhered to structured training programs [91]. Overall, these findings indicate that the impact of physical activity depends on both its type and consistency, suggesting that a single episode of vigorous exertion may produce effects distinct from those of regular, sustained activity.

Several physiological mechanisms have been proposed to explain this complex phenomenon. Sudden hemodynamic changes during intense exercise may activate vascular pathways involved in migraine pathophysiology [92]. Another suggested mechanism is the rise in lactate that occurs during heavy muscle effort, which, in people with lower metabolic tolerance, may promote the activation of neurons sensitive to migraine [92]. Some literature also discusses the possibility that physical effort influences the release of neuropeptides such as CGRP [92], which plays a key role in migraine. Psychological factors—including pain expectation and stress responses—may also contribute [8,9]. These mechanisms can interact, leading to varied responses to exercise among individual patients.

Summary

Although many internal and external factors have been suggested as possible migraine triggers, current evidence indicates that their role is highly individualized and depends on the specific situation. Instead of acting as direct causes, many triggers seem to affect a changing threshold of susceptibility, interacting with the patient's baseline neurobiological state. This view helps explain why study results are often inconsistent and why people report triggers so differently.

Looking at migraine through this threshold-based approach emphasizes the value of personalized assessment, prospective monitoring, and balanced lifestyle habits rather than strict avoidance of certain stimuli. Future research—especially using objective, real-time methods—will be crucial for clarifying how different factors contribute to the start of an attack and how this knowledge can lead to more effective, individualized prevention.

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