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Review

Migraine and dietary factors: a narrative review of current evidence and clinical implications

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Abstract

Background
Dietary factors have been extensively examined as potential triggers or modulators of migraine episodes. Nevertheless, the current body of evidence concerning the influence of diet on the pathophysiology and management of migraines remains varied and occasionally contradictory.

Objective
To critically evaluate the existing evidence concerning the association between dietary factors and migraine, with a focus on potential triggers, dietary patterns, and the gut–brain axis.

Methods
This study employs a narrative review of the literature, concentrating on research that examines dietary triggers, nutritional interventions, and the pathophysiological mechanisms associated with migraine.

Results
Certain dietary components, including caffeine, chocolate, alcohol, and foods containing histamine, have been linked to the onset of migraine attacks in susceptible individuals, although findings remain inconsistent. Various dietary interventions—such as low-fat diets, ketogenic diets, low-glycemic diets, and diets enriched with omega-3 fatty acids—have demonstrated potential benefits in reducing the frequency and severity of migraines.

Conclusions
Dietary factors may affect migraine in a subset of patients through metabolic, inflammatory, and neurovascular pathways. Further well-designed prospective studies are necessary before definitive clinical recommendations can be formulated.

Keywords:
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Introduction

Dietary considerations have long been of interest to headache specialists in the management of migraine patients. Dietary factors have been discussed as potentially contributing to the triggering or exacerbation of migraine attacks. Some evidence in the literature suggests that dietary interventions may offer relief, thereby improving quality of life (1–3). However, the current evidence regarding the interaction between diet and migraine remains controversial. It is well-documented that eliminating certain dietary triggers can have positive effects for some patients, and that weight loss strategies may be beneficial for individuals with comorbid obesity and migraine.

The implementation of a healthy diet is a recognized component of migraine management. There is also increasing interest in the role of nutraceuticals in migraine treatment (1–4). This review will not address treatment with nutraceuticals, as they are covered elsewhere. The association between dietary factors and migraine remains a subject of ongoing scholarly debate. Although numerous studies have investigated potential dietary triggers and nutritional interventions, the evidence available is often heterogeneous, and causal relationships frequently remain indeterminate (4–6). Moreover, clinical recommendations concerning the dietary management of migraine are not consistently aligned across studies. Consequently, a more comprehensive synthesis of the current evidence is warranted.

This narrative review aims to critically evaluate the existing literature on dietary factors associated with migraine, encompassing potential triggers, dietary patterns, and emerging mechanisms such as the gut–brain axis. Furthermore, this review seeks to elucidate the clinical implications of dietary management for migraine patients and to identify areas necessitating further research.

Chocolate and Migraine

Chocolate has been reported as a possible headache trigger in a variable proportion of migraine patients, ranging from 2% to 22% in clinical series (1). Diary-based studies suggest that only a minority of patients identify chocolate as a direct precipitant, although intake is often recorded on days when migraine attacks occur (2). Experimental data are conflicting: one provocation study found that 42% of participants developed migraine after chocolate administration compared with none in the placebo group (3), whereas another double-blind crossover trial showed no difference between chocolate and carob (4). It is worth noting that cravings for chocolate may be part of the premonitory phase of migraine, raising the question of whether chocolate is truly causal or simply consumed because of prodromal symptoms (5,6). Proposed mechanisms include modulation of serotonin and nitric oxide pathways, but the data are inconsistent (7,8). On the other hand, some authors have suggested that chocolate may exert protective effects through its magnesium, riboflavin, and serotonin content, as well as its ability to inhibit nitric oxide and CGRP release (9).

Caffeine and Migraine

Caffeine is widely used as an acute therapy for headache owing to its vasoconstrictive properties, its ability to inhibit prostaglandin and leukotriene synthesis, and its role in enhancing the absorption of analgesics (10). Despite this, long-term consumption may worsen headache disorders, contributing both to withdrawal headaches and to medication-overuse headache (10). Withdrawal occurs because chronic exposure induces adenosine receptor hypersensitivity; abrupt discontinuation leaves these receptors unopposed, facilitating cerebral vasodilation and pain (11). High intake of caffeine may also promote medication-overuse headache by increasing cortical excitability and glutamate release, setting off a cascade that enhances neuropeptide release, particularly CGRP (12).

Dietary Interventions in Migraine

- **Low-fat diet:** An open-label study reported that reducing fat intake was associated with decreases in attack frequency, severity, duration, and the need for rescue therapy (13). These findings were corroborated by a crossover trial showing a significant reduction in migraine frequency and severity with a low-fat regimen (14).
- **High omega-3 and low omega-6 diet:** Omega-3 fatty acids possess anti-inflammatory properties and represent a non-pharmacological option for migraine prevention (15). Supplementation studies have demonstrated reductions in migraine frequency, duration, and severity (16,17). Diets enriched in omega-3 and reduced in omega-6 intake have further been associated with fewer headache days and shorter attack duration (18).
- **Ketogenic diet:** This diet, characterized by high fat, very low carbohydrate, and moderate protein, has been proposed to alleviate migraine severity by increasing GABA, decreasing CGRP synthesis, and limiting cortical spreading depression (19–21). Reports from as early as 1928 and 1930, and more recent studies (2013, 2015, 2016), suggest benefits in both frequency and duration of migraine (22–26).
- **Low-glycemic diet (LGD):** By restricting carbohydrates to 40–60 g with a glycemic index below 50, LGD was shown in a randomized trial to reduce migraine frequency and, after three months, to lessen pain intensity (27,28).
- **High-folate diet:** Supplementation with folate, vitamin B6, and vitamin B12 may benefit patients, particularly those with migraine with aura, through effects on homocysteine metabolism (29). In a randomized trial, supplementation significantly lowered serum homocysteine and reduced migraine-related disability, frequency, and pain severity (30). One trial showed preventive properties with B2 at doses as high as 400 mg a day for 3 months (31).

- **Magnesium in the Diet:** Magnesium supplementation is a topic discussed by many headache specialists. There are many publications around its use in migraine patients. Clinicians believe that magnesium supplementation can be used to prevent migraine attacks, especially in those patients who have identified contraindications to standard medications. The rationale behind its use is that hypomagnesemia can enhance glutamatergic neurotransmission and promote excitotoxicity and consequently lead to oxidative stress causing finally migraine attacks. Since there is about 25–35g of magnesium in the human body, of which about 53% is stored in the bones, 46% in muscles and soft tissues, and only 1% in the blood, measuring intracellular magnesium to demonstrate hypomagnesemia, is a continuous challenge. To date there are no reliable publications confirming the role of magnesium deficiency in the diet as a factor causing migraine attacks, nevertheless, its use in clinical practice is widespread (32).

The evidence concerning chocolate as a migraine trigger remains inconclusive, while caffeine shows a dual role: useful in the acute setting but potentially harmful with chronic use or sudden withdrawal. Dietary approaches—including low-fat, high omega-3/low omega-6, ketogenic, low-glycemic, and high-folate regimens—appear promising in reducing migraine burden, acting through inflammatory, metabolic, and neurotransmitter pathways. Still, methodological limitations highlight the need for larger, prospective, and rigorously controlled trials before clear clinical guidelines can be established.

Migraine and Microbiota

Diet plays a role in the progression of migraine, both preventively and therapeutically. Several dietary factors can trigger migraines in some patients, so a balanced diet should be adopted to reduce the intensity, onset, and duration of pain. The application of phosphorus nuclear magnetic resonance (P-NMR) spectroscopy revealed changes in energy metabolism in the brain of migraineurs. This suggests an important role for the imbalance in brain energy demand and mitochondrial ATP production in migraine. P31 nuclear magnetic resonance studies revealed a brain energy deficit in migraine patients.

The term "gut-brain axis" denotes a bidirectional relationship between the gastrointestinal (GI) system and the central nervous system (CNS). To date, several studies have shown that migraine is associated with several gastrointestinal (GI) disorders, such as Helicobacter pylori (HP) infection, irritable bowel syndrome (IBS), and celiac disease (CD) (33).

Studies suggest that this interaction appears to be influenced by multiple factors, such as inflammatory mediators (IL-1ß, IL-6, IL-8, and TNF-α), the gut microbiota profile, neuropeptides and the serotonin pathway, stress hormones, and nutritional substances. Neuropeptides such as CGRP, SP, VIP, and NPY are thought to have an antimicrobial effect on various gut bacterial strains and are therefore speculated to participate in the bidirectional gut-brain relationship. The microbiota profiles of migraine patients have been found to be distinct compared to healthy controls. Chronic migraine (CM) and medication overuse headache (MOH) are associated with increased intestinal permeability, raising the question that the altered composition of the gut microbiota may be the cause of the intestinal hyperpermeability and inflammation observed in this headache disorder (34).

Based on current findings, the ketogenic diet and the modified Atkins diet are believed to play a role in neuroprotection by improving mitochondrial function and energy metabolism, compensating for serotonergic dysfunction, decreasing calcitonin gene-related peptide (CGRP) levels, and suppressing neuroinflammation. It can also be speculated that prescribing a low-glycemic index diet could be promising for headache/migraine management by attenuating the inflammatory state. Furthermore, obesity and headaches, including migraine, could be mutually attributable to mechanisms such as inflammation and irregular hypothalamic function. Therefore, the application of dietary strategies for weight loss may also improve headache and migraine (35). Despite the findings described above and the brain-gut relationship, the role of the microbiota in the preventive treatment of migraine remains to be resolved.

Alcohol and its Relationship with Headache and Migraine

The IHS International Classification of Headache Disorders (ICHD-III) (36) includes alcohol-induced headache among secondary headache disorders, in group 8 "Headache attributed to substance use or withdrawal," recognizing two subtypes: immediate alcohol headache and delayed alcohol headache.

Although it has been described since ancient times, the relationship between alcohol and other dietary factors as triggers of headache attacks it remains controversial. Several studies have been conducted, but they suffer from methodological errors and biases. Up to 30% of migraine patients and more than 53% of cluster headache patients report alcohol as a trigger for their attacks.

Some hypotheses attempt to explain how alcohol triggers migraines. It is possible that it causes neurogenic inflammation, calcitonin gene-related protein (CGRP) release, or cortical spreading depression. Red wine, and to a much lesser extent white wine, is a powerful releaser of serotonin from platelets.

The question that remains unanswered is whether alcohol itself or another substance present in alcoholic beverages is responsible for triggering the pain. Other substances proposed as responsible include biogenic amines (tyramine, phenylethylamine), flavonoids, and sulfites. A study compared

red wine with vodka in equivalent doses in a population of migraine patients and non-migraine controls, finding that none of the patients who drank vodka and none of the controls developed headaches, while a high percentage of migraine patients who drank wine did.

The current evidence is that some studies show that migraine patients drink lower amounts of alcohol than the general population. It has been found that red wine and almost all forms of alcohol can trigger headaches in sensitive patients. There is a very strong relationship between cluster headaches and alcohol as one of their triggers.

To date, no significant differences have been found between migraine (with and without aura) and tension-type headaches with respect to alcohol triggers. While certain foods and beverages have been identified as potential triggers for migraine attacks in susceptible individuals, the extant evidence often indicates an association rather than a direct causal relationship.

Eliminating these foods from the diet in this subgroup of patients could benefit them.

Approximately less than a third of headache patients attribute their symptoms to food or beverage triggers. Therefore, restricting foods and beverages for all migraine patients is not a recommended practice today.

Histamine and Migraine: Current Evidence and Clinical Implications

Histamine intolerance has been acknowledged as a clinical disorder of relevance for over 60 years, with an estimated prevalence of approximately 1% within the general population (37). As a monoamine, histamine plays significant roles in gastric secretion, immune regulation via mast cells, and acts as a neurotransmitter in the central nervous system (CNS). Its involvement has been noted in various neurological and systemic conditions, including migraines, cluster headaches, chronic pain, fatigue, and attention deficit disorder (38–40). Transitioning to the specific relationship between histamine and migraines, the next section delves into the pathophysiological links that have been established.

Histamine functions as a neurotransmitter within the CNS and is known to induce vasodilation, which is a fundamental aspect of the vascular theory of migraine (38,41,42). During migraine attacks, histamine levels rise concurrently with calcitonin gene-related peptide (CGRP), indicating a reciprocal release between these two mediators in the trigeminovascular system. Despite these observations, the exact mechanisms that govern this interaction remain inadequately understood. Additionally, diets rich in histamine have been correlated with an increase in migraine attacks, likely due to their influence on CGRP levels (43).

Following this examination of histamine's role in migraine, it is essential to explore the mechanisms of histamine metabolism, particularly the function of diamine oxidase (DAO).

Headaches are among the most reported and debilitating symptoms associated with histamine intolerance. A proposed mechanism for this association involves a deficiency in diamine oxidase (DAO), the principal enzyme responsible for degrading histamine at the intestinal epithelium. Reduced DAO activity has been documented in migraine patients, with one study indicating decreased activity in up to 87% of migraine sufferers compared to 44% of healthy controls (cutoff <80 HDU/ml) in a cohort of 198 subjects (137 with migraine, 61 healthy volunteers) (44). Nevertheless, not all studies corroborate a reduction in DAO activity among migraineurs; some research has identified increased DAO activity in nonallergic migraine sufferers, without noting differences in serum histamine levels or DAO gene polymorphisms.

This comprehensive analysis highlights the complexities of histamine's role in migraines and underscores the need for further research to elucidate the underlying mechanisms involved.

Dietary interventions aimed at reducing histamine and gluten intake may alleviate migraine burden, particularly in patients with coexisting non-celiac gluten intolerance (44). Supplementation with exogenous DAO has been suggested to reduce the duration and severity of migraine attacks in patients with DAO deficiency, although its impact on migraine frequency remains unclear (44). Nevertheless, the heterogeneity of migraine underscores the need for further studies to clarify the precise role of histamine metabolism and DAO activity in migraine generation and progression.

To facilitate interpretation of the available evidence, a summary of the main dietary factors associated with migraine and their proposed mechanisms is presented in Table 1.

Table 1. Dietary factors associated with migraine: proposed mechanisms and strength of evidence

Dietary Factor	Proposed Mechanisms	Evidence Summary	Strength of Evidence
Chocolate	Possible modulation of serotonin pathways, nitric oxide release, and CGRP activity; may also be associated with the premonitory phase of migraine	Studies show conflicting results; some patients report chocolate as a trigger, while other studies suggest no causal relationship	Conflicting evidence
Caffeine	Adenosine receptor antagonism, vasoconstriction, enhanced analgesic absorption; withdrawal may increase cerebral vasodilation and neuronal excitability	Beneficial in acute treatment, but chronic use and withdrawal may contribute to headache and medication-overuse headache	Moderate evidence
Alcohol	Neurogenic inflammation, CGRP release, cortical spreading depression; possible role of biogenic amines and sulfites	Observational studies suggest alcohol may trigger attacks in susceptible individuals, particularly red wine	Observational evidence
Histamine-containing foods	Histamine-induced vasodilation, interaction with the trigeminovascular system; possible association with reduced DAO activity	Some studies report increased migraine frequency in patients with histamine intolerance, but findings remain heterogeneous	Emerging evidence
High omega-3 / low omega-6 diet	Anti-inflammatory effects, modulation of lipid mediators involved in pain pathways	Clinical trials suggest reduction in migraine frequency and severity	Moderate evidence
Ketogenic diet	Increased GABA levels, reduction of CGRP synthesis, improved mitochondrial function and energy metabolism	Several clinical studies suggest benefit in migraine prevention	Promising but limited evidence
Low-glycemic diet	Stabilization of glucose metabolism and reduction of inflammatory pathways	Randomized studies indicate potential reduction in migraine frequency and intensity	Moderate evidence
Gut microbiota modulation	Gut–brain axis interaction, inflammatory mediators, serotonin metabolism	Emerging research suggests differences in microbiota profiles among migraine patients	Preliminary evidence

Conclusion

In conclusion, lifestyle-modifiable factors, particularly dietary habits, constitute a significant aspect of migraine management. Nonetheless, the association between specific dietary elements and migraine is intricate and likely varies among individuals. Consequently, dietary recommendations should be personalized and incorporated into a comprehensive clinical strategy.

While several dietary interventions show potential, the current body of evidence is constrained by methodological variability and a predominance of observational studies. Future research should prioritize well-designed prospective trials to elucidate causal relationships and identify patient subgroups that may derive the greatest benefit from specific dietary strategies.

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