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Dietary Strategies for Migraine Management: A Comprehensive Review of Mechanisms, Evidence and Clinical Applications

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Abstract— Migraine is a multifactorial neurological disorder influenced by a wide range of biological and environmental factors, including diet. This review aims to synthesize current knowledge on how dietary factors affect migraine pathophysiology and its clinical management. A comprehensive literature search was conducted using PubMed, ResearchGate, and Google Scholar, analyzing observational, clinical, and mechanistic studies focused on dietary triggers, beneficial dietary patterns, and the mechanisms linking nutrition to migraine. Current evidence indicates that diet may influence migraine through multiple pathways, including neurovascular reactivity, inflammation, gut microbiota modulation, metabolic regulation, and genetic susceptibility. Certain foods and food components, such as alcohol, caffeine, nitrates, and tyramine, have been identified as potential migraine triggers, although individual responses vary considerably. At the same time, specific dietary patterns, including the Mediterranean, ketogenic, and low–glycemic index diets, show promise in reducing migraine frequency and severity, although definitive clinical evidence remains limited. Personalized, patient-centered approaches, including dietary diary tracking and individualized nutritional interventions, appear to be particularly important in clinical practice. Further research is needed to establish evidence-based dietary recommendations for migraine. An integrated and personalized strategy combining dietary modification, lifestyle changes, and pharmacological treatment offers the greatest potential to improve migraine management and patients' quality of life.

Keywords— migraine; headache; dietary triggers; gut-brain axis; nutrients

1. INTRODUCTION

Migraine is a neurological disorder affecting over 1 billion people globally, ranking as the second-leading cause of years lived with disability worldwide among the general population and the first among women [1][2]. Characterized by recurrent episodes of disabling head pain with multitude of different sensory and motor disturbances such as photophobia, nausea or sensory hypersensitivity, migraines impose a profound socioeconomic burden due to lost productivity and healthcare expenditures [3]. While pharmacological therapies such as triptans, CGRP inhibitors, and prophylactic agents remain the basis of treatment, their efficacy is often inconsistent, and side effects limit long-term utility [4][5]. This has prompted interest in non-pharmacological strategies, particularly dietary interventions, which offer a low-risk approach to reducing migraine frequency and severity.

While the precise mechanisms underlying migraine development remain incompletely understood, current evidence points to multiple interconnected factors including gut-brain axis interactions, with emerging research underscoring the intricate interplay between dietary influences and migraine pathophysiology [6]. Specific foods and nutrients can act as triggers by modulating neurovascular pathways, oxidative stress, and inflammatory cascades. For instance, tyramine in aged cheeses, nitrates in processed meats, and aspartame in diet sodas are well-documented precipitants of attacks [7][8][9][10]. Conversely, certain dietary patterns, such as ketogenic or high omega-3 regimens, may exert prophylactic effects by stabilizing neuronal excitability and mitigating neuroinflammation. The gut-brain axis further complicates this relationship, as dysbiosis and intestinal permeability can amplify systemic inflammation and trigger migraine episodes through immune and vagal signaling [9].

Despite promising evidence, dietary management of migraines remains underutilized in clinical practice, hindered by a lack of consensus on optimal strategies and individual variability in trigger susceptibility [11]. This review synthesizes contemporary evidence on the role of diet in migraine prevention and management, evaluating the efficacy of elimination diets, nutritional supplements, and therapeutic eating patterns. It also explores mechanistic links between nutrition, gut microbiota, and migraine pathways, while addressing challenges in personalization and adherence. By bridging gaps between research and practice, this article aims to provide actionable insights to integrate dietary interventions into holistic migraine care.

2. MATERIALS AND METHODS

This review was conducted through an extensive search of scientific literature from databases such as PubMed, Scopus, and Google Scholar. Keywords included “migraine,” “headache,” “diet,” "dietary triggers,” “dietary patterns,” “gut-brain axis,” “nutraceuticals” and related terms. The included studies encompassed observational research, randomized controlled trials, systematic reviews, and mechanistic studies published up to 2025. The selected articles were critically analyzed to synthesize current insights on dietary triggers, potential benefits of various diets, underlying biological pathways, and clinical strategies. The review also considered emerging technologies and personalized approaches for dietary intervention, aiming to provide a comprehensive overview of the field’s current state and future directions.

3. MECHANISMS LINKING DIET AND MIGRAINES

Migraine pathophysiology is intricately connected to dietary influences through neurovascular, inflammatory, metabolic, and gut-brain axis mechanisms, which collectively shape susceptibility to attacks. At the neurovascular level, cortical spreading depression (CSD), a wave of neuronal depolarization linked to migraine aura, and dysregulated neurotransmitter activity are hypothesized to be central to migraine pathophysiology [12][13]. CSD is believed to activate afferent fibers of the trigeminal ganglion that innervate the meninges. This activation stimulates the release of neuropeptides such as substance P, neurokinin A, and calcitonin gene-related peptide (CGRP). These peptides subsequently promote endothelial and platelet activation, increasing nitric oxide (NO) synthesis and triggering vasodilation, which contributes to the clinical onset of migraine symptoms [14]. Certain dietary triggers exacerbate these processes by directly influencing neurotransmitter dynamics, however some of those mechanisms are not fully confirmed and still being researched. For example, diets rich in tryptophan, found in poultry, eggs, and tofu, support serotonin synthesis, a neurotransmitter critical for stabilizing mood and vascular tone. Alcohol triggers neurogenic inflammation in the trigeminovascular system and stimulates the dilation of meningeal blood vessels by prompting the release of CGRP from sensory nerve terminals surrounding these vessels. Nitrates in cured meats and certain vegetables convert to nitric oxide (NO), a vasodilator implicated in migraine aura, while tyramine in aged cheeses and fermented foods induces norepinephrine release, triggering rebound vasodilation [15][16].

Inflammation and oxidative stress further bridge diet and migraine pathology. Pro-inflammatory diets high in omega-6 fatty acids (e.g., corn oil, fried snacks) promote the production of inflammatory eicosanoids, whereas omega-3-rich foods like fatty fish and walnuts generate anti-inflammatory resolvins. Deficiencies in magnesium, riboflavin (B2), and coenzyme Q10, nutrients vital for mitochondrial energy production and antioxidant defense, correlate with increased migraine frequency, highlighting the importance of dietary micronutrients in mitigating oxidative damage [17][18][19].

The gut-brain axis adds another layer of complexity. Dysbiosis, driven by low-fiber, high-processed-food diets, disrupts gut microbiota balance, elevating circulating lipopolysaccharides (LPS) and pro-inflammatory cytokines that cross the blood-brain barrier. This systemic inflammation activates trigeminal nociceptors, a key pathway in migraine pain. Short-chain fatty acids (SCFAs), produced by fiber-fermenting gut bacteria, counteract inflammation and strengthen the blood-brain barrier, yet their production dwindles in fiber-deficient diets. Histamine intolerance, common in migraineurs with impaired diamine oxidase (DAO) enzyme activity, exacerbates symptoms when histamine-rich foods like citrus or fermented products are consumed, underscoring the gut's role as both a trigger and therapeutic target [20][21][22][23]. Despite growing evidence implicating the gut-brain axis in migraine pathophysiology, the underlying mechanisms have yet to be fully characterized.

Metabolic and hormonal imbalances further intertwine with dietary habits. Hypoglycemia from skipped meals or fasting elevates plasma free fatty acids, disrupts neuronal ion homeostasis, and alters dopamine and serotonin levels- metabolic shifts that may collectively contribute to migraine pathogenesis [24]. Genetic factors, such as *MTHFR* gene polymorphisms, compound dietary risks by impairing folate metabolism and elevating homocysteine, a known migraine trigger [25].

4. Dietary Triggers And Elimination Strategies

Dietary triggers are a well-documented yet highly individualized component of migraine management. According to research, about one-third of migraine sufferers link their headaches to food triggers [26][27]. Identifying and mitigating these triggers through elimination strategies can significantly reduce attack frequency and severity for many patients. However, it is important to mention that Despite the common belief among patients that diet plays a significant role in migraines, a 2016 Austrian study assessing lifestyle and migraine triggers found notable discrepancies [28]. Notably, patient-reported dietary triggers showed limited accuracy in predicting actual triggers identified through empirical data (predictive value: 0.22–0.36). However, such studies often lack robust methodologies (e.g., double-blind designs) and are prone to recall bias, weakening the reliability of these findings [11].

4.1. Common Dietary Triggers

Migraineurs frequently identify specific foods and beverages as triggers, though individual susceptibility varies due to genetic, metabolic, and environmental factors. Among the most consistently reported triggers is **alcohol**, particularly red wine, beer, and spirits, which contain histamine, tyramine, and sulfites. These compounds promote vasodilation and neuroinflammation, while ethanol's diuretic effect exacerbates dehydration- a known migraine precipitant. Studies suggest up to 35% of migraineurs experience alcohol-induced attacks, with red wine cited most frequently [15][27][29][30][32][31][33][34][35][36][37]. Alcohol's potential to provoke migraines differs among migraine subtypes, with studies reporting inconsistent outcomes, underscoring variability in its triggering effects [38][39].

Caffeine's role in migraine involves multiple mechanisms: it blocks adenosine A2A receptors (linked to CGRP-mediated pain) [40], induces urinary magnesium loss [41][42], and causes dehydration via diuretic effects [43][44]. It also elevates glutamate by inhibiting excitatory amino acid transporters, increasing nociceptor sensitization. Withdrawal may trigger migraines through increased cerebral blood flow. Caffeine presents a paradox: while low doses (50–100 mg) may alleviate acute migraines via vasoconstriction, excessive intake (>200 mg/day) or abrupt withdrawal disrupts adenosine receptor signaling, triggering rebound headaches. Meta-analyses indicate caffeine withdrawal contributes to migraine recurrence in over a third of cases [45][46][47]. However, randomized trials show 100–200 mg

of caffeine combined with analgesics effectively relieves migraines and tension-type headaches (TTHs), with 200 mg alone aiding TTHs [48][49]. Caffeine is also a first-line treatment for hypnic headaches [50]. These findings highlight caffeine's complex role, necessitating tailored approaches in headache therapy. Current evidence is insufficient to establish universal guidelines for caffeine consumption in managing headache disorders. A pragmatic approach, informed by existing research, involves limiting dietary caffeine to moderate quantities consumed consistently before midday and infrequent use of caffeine analgesics for mild headaches [51][52].

Aged cheeses and fermented foods, such as blue cheese, soy sauce, and sauerkraut, are rich in tyramine, a monoamine that stimulates norepinephrine release. This leads to transient vasoconstriction followed by rebound vasodilation, a process implicated in migraine aura and pain [15][53][54][55]. However, the evidence of the role of tyramine in migraine pathogenesis is inconclusive. Current estimates suggest that sensitivity to tyramine affects approximately 5% of individuals with migraine [8][56][57]. Additionally, phenylethylamine (PEA), a biogenic amine derived from phenylalanine, is also found in potential migraine-triggering foods like chocolate, wine, and certain cheeses, as well as in aspartame. It acts as an excitatory neuromodulator, enhancing dopamine release and motor activity, while also inhibiting reuptake of norepinephrine and dopamine. Despite its proposed role in migraine via neurotransmitter modulation, evidence linking PEA to migraine attacks remains inconclusive, with no definitive confirmation as a trigger [15].

Processed meats like bacon and salami contain nitrates and nitrites, which convert to nitric oxide, a potent vasodilator linked to cortical spreading depression. Mitochondria, along with enzymes like xanthine oxidase and proteins such as myoglobin, convert nitrite into nitric oxide (NO), primarily under low-oxygen conditions to promote vasodilation, reduce metabolic rate, and protect against free radicals [58]. While endogenous and dietary nitrite serves vital antioxidant roles, xanthine oxidase can paradoxically generate superoxide under certain conditions, converting NO into peroxynitrite, a potent oxidant linked to cellular damage [59]. An additional mechanism that may play a role in this process is formation of methemoglobin created in an oxidative reaction of nitrites with hemoglobin [60]. Although nitrates (abundant in vegetables and the Mediterranean diet) are likely benign, processed meats may pose risks due to nitrosamines- toxic byproducts of nitrate-protein reactions associated with brain cancer in animal studies, though human epidemiological findings remain inconsistent [61][62]. A 1972 case report described a patient who developed moderately severe, non-throbbing headaches within minutes to hours of consuming nitrite-containing foods like cured meats [7]. Later studies on migraineurs found that nitrate ingestion can provoke headaches in two distinct patterns: rapid onset (mild-moderate within 1 hour) or delayed (severe, migraine-like symptoms after 3–6 hours) [63][64]. Daily diaries from migraine patients attributed approximately 5% of attacks to nitrate consumption [10]. It is also noteworthy that nitrate-based medications, frequently prescribed for cardiovascular disorders, provoke severe headaches in over 80% of users, leading to therapy discontinuation in approximately 10% of cases due to headache severity [65].

Artificial sweeteners, notably aspartame in diet sodas, may lower serotonin, noradrenaline and dopamine levels and heighten glutamate-mediated cortical excitability, increasing oxidative stress and inflammation in the brain [66][67][68]. Small trials linked aspartame to headaches in 15–30% of migraineurs, however evidence remains mixed [69][70]. Sucralose has also been linked to headache episodes in some cases, though current evidence remains limited to isolated case reports [71].

Monosodium glutamate (MSG), common in savory snacks and restaurant foods, is controversially tied to "Chinese Restaurant Syndrome" (headache, flushing, palpitations, sweating), though evidence

remains inconsistent [72][73][74]. The association MSG and migraines may arise from its influence on glutamatergic neurotransmission via activation of peripheral *N*-methyl-D-aspartate (NMDA) receptors and subsequent sensitization of nociceptors (pain-sensing neurons), potentially amplifying migraine-related pain signaling [75][76][77].

Citrus fruits (e.g., oranges, lemons) and **chocolate** are also debated triggers. Citrus fruits release histamine, which can provoke inflammation in those with impaired histamine clearance [20]. Chocolate's role in migraines involves multiple mechanisms: sulfotransferase inhibitors elevate migraine-linked dopamine [78][79]; flavanols stimulate nitric oxide (via eNOS), causing vasodilation; caffeine (contained in cocoa beans) through caffeine-related processes [80], serotonin modulation [69], and phenylethylamine (increasing cerebral blood flow in animal models [81][82]) may contribute. Conversely, components like magnesium, riboflavin, and interactions with CGRP/gut microbiota suggest potential therapeutic benefits [80]. The hypothesis that chocolate acts as a migraine trigger remains unresolved, with extant literature presenting heterogeneous conclusions [83][84][85][86][10]. Chocolate's role is also complicated by its frequent emergence as a pre-migraine craving rather than a direct trigger, blurring causal links [87].

Tu et al. (2025) in their review on dietary pattern and migraine proposed a few hypothesis to explain the inconsistent findings across studies linking diet to migraines: variability in food components (e.g., caffeine levels in coffee), cultural dietary patterns affecting sample comparability, regional food types and preparation methods altering ingredient profiles, genetic/metabolic differences influencing individual responses, and methodological limitations, such as recall bias in observational studies versus the logistical challenges of controlled trials [16].

IgG Positive Foods

Testing for IgG antibodies to foods has been explored to identify migraine triggers, with three randomized controlled trials (RCTs) assessing dietary exclusion of IgG-reactive foods. Alpay et al. (30 participants) reported reduced headache frequency with IgG-guided elimination diets compared to controls ($P < 0.001$) [88]. Mitchell et al. (167 participants) found short-term reductions (1 day at 4 weeks) but no sustained benefit by 12 weeks, possibly due to poor dietary compliance [89]. Aydinlar et al. (21 patients with migraine and IBS) observed significant decreases in attack frequency, duration, and severity during elimination phases ($P < 0.001$ for frequency) [90]. While two of three RCTs suggest modest preventive benefits, methodological limitations (e.g., compliance challenges, small cohorts) and mixed results underscore the need for further research to confirm efficacy.

4.1. ELIMINATION DIET

Elimination diets offer a valuable tool for personalized migraine management, particularly in patients with identifiable food triggers or comorbid gastrointestinal conditions. However, their success hinges on systematic implementation, patient education, and multidisciplinary support. Future research should prioritize long-term outcomes and biomarkers to refine dietary recommendations, ensuring they complement, rather than replace, holistic migraine care strategies.

A 2010 Turkish study compared migraine management in two groups: one receiving standard medication (metoprolol, riboflavin, naproxen) and the other adding a structured dietary plan (with strict guidelines across food categories). After one year, the dietary group exhibited lower pain scores, reduced attack frequency, and decreased analgesic use compared to medication alone [91].

Elimination diets involve the systematic identification and removal of dietary components suspected of triggering migraines, followed by controlled reintroduction to confirm causality. This approach often begins with self-observation, where individuals note correlations between specific foods and migraine attacks, subsequently avoiding implicated items. However, this personal method is prone to bias, as subjective interpretation may overemphasize coincidental associations or overlook subtle triggers [9]. A critical distinction lies in differentiating food allergies from intolerances or migraine-specific triggers. Food allergies involve immune-mediated reactions, marked by antibody activation and symptoms ranging from mild rashes to anaphylaxis, identifiable through serological testing. In contrast, migraine triggers, such as aged cheeses or processed meats, typically act through non-immunological mechanisms like vasodilation or neuroinflammatory pathways, with intolerances reflecting digestive or metabolic inefficiencies rather than immune responses [92]. Accurate trigger identification often relies on food diaries, where patients log dietary intake and migraine occurrences for clinical review [53]. While these records offer practical insights, their reliability is hampered by recall inaccuracies and the variable latency between trigger exposure and symptom onset, which can span hours to days. Furthermore, migraines arise from a complex, non-linear interplay of multiple factors, stress, hormonal fluctuations, dehydration, and environmental changes, that may amplify or mask dietary effects. For instance, a food trigger might only provoke an attack when combined with sleep deprivation or weather shifts, complicating the isolation of individual culprits [93]. To address these challenges, researchers propose empirical thresholds for defining dietary triggers, such as classifying a food as provocative if headaches follow ingestion in ≥50% of instances within 24 hours. Yet even this criterion struggles with real-world applicability, as meals often contain multiple ingredients (e.g., processed foods with additives, preservatives, and natural compounds), obscuring which component drives symptoms. Emerging tools like electronic dietary apps leverage statistical algorithms to detect patterns across larger datasets, potentially enhancing objectivity by correlating migraine frequency with specific foods or meals [94].

While elimination diets can aid migraine management, their prolonged or excessively restrictive application poses significant risks. Nutritional deficiencies, such as inadequate protein, vitamins, or caloric intake, stemming from unnecessary avoidance of nutrient-rich foods may heighten susceptibility to infections, metabolic dysregulation, and mental health challenges [88]. For instance, studies highlight increased risks of micronutrient deficiencies (e.g., iron, B vitamins) and psychological stress in individuals adhering to strict elimination protocols without medical oversight [95][96]. Clinicians must therefore balance trigger avoidance with nutritional adequacy, ensuring diets remain sustainable and tailored to individual needs.

In summary, while elimination diets offer a structured framework for migraine management, their success hinges on recognizing the non-linear nature of triggers, mitigating observer bias through technology, and safeguarding against nutritional deficits. Future strategies should integrate multidisciplinary support to navigate these complexities, optimizing dietary interventions within holistic migraine care.

5. DIETARY HABITS

Dietary habits, beyond individual food triggers, significantly influence migraine risk. Systematic reviews indicate that fasting, whether for health, cultural, or religious reasons, can exacerbate migraines. For instance, during Ramadan, daily fasting correlates with a marked rise in migraine frequency and severity compared to non-fasting periods. This phenomenon may stem from dehydration and hypoglycemia linked to prolonged fasting. Water deprivation or insufficient water intake alone can act as a

trigger as well. These metabolic stressors are hypothesized to trigger migraines via mechanisms including elevated vasopressin secretion, sympathetic nervous system activation, and serotonergic system dysregulation [43][97][98][99][100][101]. Emerging evidence suggests migraine is linked to dietary habits, including irregular meal patterns, meal skipping, and consumption of processed foods, while adherence to nutrient-dense diets rich in fruits, vegetables, and whole grains may reduce attack frequency and severity. Iranian studies found female migraineurs less likely to eat regular meals (37.6% vs. 23.5%; p=0.046) and more likely to consume <3 meals/day (29.4% vs. 9.4%; p=0.001) [102]. Swedish data linked skipped breakfast to higher migraine prevalence [103], while U.S. analysis noted 40% lower headache odds with nighttime snacking in migraineurs (HR=0.60; p=0.013) [104], though observational studies caution that correlation does not imply causation.

Diet quality also correlates with migraine. U.S. cross-sectional data revealed normal-weight non-migraine women had healthier diets (HEI-2005: 52.5 vs. 45.9; p<0.0001), particularly in fruit/vegetable intake and reduced fats/sugars [105]. Iranian and Italian studies tied "healthy" diets (rich in vegetables, whole grains) to reduced migraine frequency (OR=1.09; p=0.04) [106] and lower attack rates with whole-grain consumption (p=0.004) [107].

6. Gluten Free Diet

The gluten-free diet (GFD), traditionally used for celiac disease, has emerged as a potential therapeutic strategy for migraineurs, particularly those with comorbid gluten-related disorders. Gluten, a protein in wheat, barley, and rye, may exacerbate migraines through immune-mediated inflammation, gut dysbiosis, and neurovascular mechanisms. In celiac patients, gluten triggers autoimmune intestinal damage, releasing pro-inflammatory cytokines (e.g., IL-6, TNF-α) that activate trigeminal pain pathways, while non-celiac gluten sensitivity (NCGS) may increase gut permeability and systemic inflammation, disrupting the gut-brain axis and promoting neuroinflammation [23][108][109]. Clinical evidence shows migraines affect 21–30% of celiac patients, with a GFD reducing headache frequency likely through resolving intestinal inflammation [110][111][112][113]. Implementation requires screening via serological tests (anti-tTG IgA) and biopsy for celiac disease, followed by strict avoidance of gluten-containing foods and cross-contamination risks, supported by dietitian-guided nutritional monitoring. Conclusively, the GFD is a targeted, evidence-based option for gluten-sensitive migraineurs but requires rigorous screening and personalization, emphasizing future research on biomarkers and long-term outcomes to optimize its role in holistic migraine care [9][23].

7. Therapeutic Diets For Migraines Prevention

Specific dietary approaches for migraine management have emerged in parallel with elimination strategies, with various regimens proposed to reduce attack frequency or severity. While many lack robust mechanistic or empirical foundations, proponents suggest they modulate pathways such as serotonin signaling, neuronal hyperexcitability, and levels of migraine-relevant mediators, including CGRP, nitric oxide, adiponectin, and leptin, along with mitochondrial efficiency, neuroinflammatory processes, hypothalamic regulation, and platelet activity [114]. Dietary recommendations should also account for comorbidities (e.g., obesity, gut dysfunction) and migraine subtypes (e.g., aura-associated migraine), as these factors may influence treatment responsiveness. Combining evidence-supported nutritional strategies or employing multimodal dietary approaches may enhance therapeutic outcomes compared to single-intervention regimens.

7.1. Ketogenic Diet

The ketogenic diet (KD), a high-fat, low-carbohydrate regimen that induces ketosis, has garnered attention as a potential therapeutic strategy for migraine management [114]. By shifting the body’s

primary energy source from glucose to ketones, the KD is hypothesized to stabilize neuronal excitability and enhance mitochondrial efficiency, addressing energy metabolism deficits implicated in migraine pathophysiology. Ketones, such as β-hydroxybutyrate, may suppress neuroinflammation, reduce oxidative stress, and modulate neurotransmitters like glutamate and GABA, which are linked to cortical hyperexcitability and pain signaling [115]. Clinical studies suggest promising outcomes: a 2018 randomized controlled trial demonstrated a significant reduction in migraine frequency among chronic migraineurs after three months on a modified KD, while observational data associate the diet with decreased attack severity and duration [116][117]. The KD's effects may also extend to calcitonin gene-related peptide (CGRP) pathways, with preclinical models indicating ketone-mediated inhibition of CGRP release. However, adherence challenges, including gastrointestinal side effects, nutrient deficiencies, and restricted food choices, limit its practicality for many patients [118][199]. Current evidence supports its use as an adjunct therapy for refractory or chronic migraine, ideally under medical supervision to mitigate risks and tailor macronutrient ratios. While mechanistic and clinical findings are encouraging, larger long-term studies are needed to define its role in mainstream migraine care [120].

7.2. DASH DIET

The Dietary Approaches to Stop Hypertension (DASH) diet, originally designed to reduce blood pressure, has shown promise as a dietary strategy for migraine management. Rich in fruits, vegetables, whole grains, low-fat dairy, nuts, and legumes while limiting sodium, red meat, and added sugars, the DASH diet emphasizes nutrients such as magnesium, potassium, calcium, and fiber, many of which are implicated in migraine pathophysiology. Magnesium, for example, plays a role in modulating cortical excitability and serotonin signaling, while potassium and calcium contribute to vascular stability and neurotransmitter regulation. Observational studies suggest that adherence to the DASH diet correlates with reduced migraine frequency and severity, potentially through its anti-inflammatory and antioxidant properties, which mitigate oxidative stress and endothelial dysfunction linked to migraine attacks. Additionally, the diet's low sodium content may help stabilize blood pressure fluctuations, a known migraine trigger in susceptible individuals. While rigorous clinical trials are limited, preliminary evidence positions the DASH diet as a balanced, sustainable option for migraine prevention, particularly in patients with comorbid hypertension or metabolic syndrome. Its focus on whole foods and nutrient density further supports overall cardiovascular and neurological health, making it a practical component of a holistic migraine management plan [121][122]123][124][125].

7.3. LOW FAT VEGAN DIET

A low-fat vegan diet, which eliminates animal products and minimizes fat intake, has been explored for its potential benefits in migraine management. Four research studies examined how low-fat diets affect migraine prevention. They showed that cutting dietary fat intake for three months resulted in reductions in headache severity, frequency, and the consumption of abortive medications. In another trial, adults with chronic migraine were randomly assigned to either a diet rich in omega-3 and low in omega-6 or a diet low in omega-6 alone. Those following the high omega-3/low omega-6 regimen experienced more significant improvements in their headache symptoms compared to the low omega-6 group. The observed benefits were linked to several factors: the balance between omega-6 and omega-3 pathways, which plays a role in controlling inflammation; the role of omega-6 fatty acids in promoting vasodilation; and the tendency of high-fat diets to increase blood coagulability [23]. A low-fat vegan diet may also lower estrogen activity by increasing sex-hormone binding globulin (SHBG) levels, which binds to and neutralizes circulating estrogens [126][127]. This mechanism parallels the well-documented association between estrogen fluctuations (e.g., during menstrual cycles) and migraine susceptibility, suggesting a potential indirect pathway for dietary interventions to modulate migraine risk [128]. Additionally, the diet's exclusion of common migraine triggers like aged cheeses and processed meats may further contribute to symptom

relief. However, challenges such as nutrient deficiencies (e.g., vitamin B12, iron) and the restrictive nature of veganism may limit long-term adherence for some patients. While promising, current evidence remains sparse, necessitating larger randomized trials to validate efficacy and delineate optimal dietary protocols. For motivated individuals, a well-planned low-fat vegan diet offers a plant-based, nutrient-dense approach to migraine management, particularly when combined with supplementation and professional nutritional guidance [129].

7.4. MEDITERRANEAN DIET

The Mediterranean diet emphasizes the consumption of legumes, fish, whole grains, olive oil, vegetables, fruits, and nuts- foods rich in B vitamins and magnesium. Prior research has validated the benefits of this dietary pattern, showing notable links between regular intake of Mediterranean diet components and reductions in the frequency, duration, and severity of migraines [130][131]. The effectiveness of a Mediterranean-style diet in alleviating migraines remains to be confirmed through future interventional research.

7.5. EPIGENETIC DIET

A dietary modification approach has been proposed, suggesting that incorporating specific nutritional compounds with targeted mechanisms of action modulate gene expression to influence disease pathways [132], such as in cancer [133]. These diets focus on particular cellular components, like mitochondria, and molecules, such as DNA. In line with this approach, Hardy and Tollefsbol coined the term “epigenetic diet” in 2011 to describe how environmental factors, including dietary elements, can influence the epigenetic patterns of individuals, potentially aiding in disease prevention [134]. By targeting epigenetic mechanisms such as DNA methylation, histone modification, and non-coding RNA regulation, these diets aim to downregulate pro-inflammatory genes, enhance antioxidant defenses, and stabilize neuronal excitability implicated in migraines [135]. Key bioactive compounds include folate (abundant in leafy greens), which supports DNA methylation to regulate genes like *MTHFR*, a polymorphism linked to migraine risk (by increasing homocysteine plasma level), and polyphenols (e.g., resveratrol in grapes, curcumin in turmeric), which inhibit histone deacetylases (HDACs) to suppress neuroinflammatory mediators such as NF-κB. Sulforaphane from cruciferous vegetables activates Nrf2 pathways, boosting endogenous antioxidants to counteract oxidative stress, while omega-3 fatty acids from fatty fish modulate DNA methylation of pain-related genes [136][137][138]. Several genes have been reported to show methylation patterns potentially linked to migraine, including SH2D5 (SH2 domain-containing 5) [139], COMT (catechol-O-methyltransferase), ZNF234 (zinc finger protein 234), SOCS1 (suppressor of cytokine signaling 1) [140], as well as SLC2A9, SLC38A4, and SLC6A5 (members of the solute carrier family 2, 38A, and 6A), DGKG (diacylglycerol kinase gamma), KIF26A (kinesin family member 26A), DOCK6 (dedicator of cytokinesis 6), CFD (complement factor D) [141], RAMP1 (receptor activity modifying protein 1) [142], and CGRP [143]. The synthesis of homocysteine depends on the availability of folate and vitamins B6 and B12. Deficiencies in these nutrients can lead to DNA hypomethylation, which has been proposed as a potential trigger for migraines [144]. Overall, folate plays a crucial role in DNA methylation, and dietary intake of this vitamin has been associated with beneficial effects on migraine. However, it is still unclear whether these benefits are directly due to changes in DNA methylation or other epigenetic modifications. As a result, researchers have cautioned against broadly applying the term “epigenetic diet” or categorizing certain diets, such as those fortified with folate, as “epigenetic diets,” despite their possible advantages for migraine management [96][145]. Challenges include individual genetic variability, requiring personalized nutrient profiling, and the need for long-term adherence to sustain epigenetic changes. While promising, this emerging field necessitates further research to define optimal dietary components, dosages, and synergistic effects with conventional therapies, positioning epigenetic diets as a future frontier in precision nutrition for migraine prevention.

7.6. Effects of Weight Loss on Migraine Headaches

Weight loss, particularly in individuals with obesity, has been associated with significant improvements in migraine frequency, severity, and disability. Obesity is a recognized risk factor for migraine chronification, likely mediated by systemic inflammation, insulin resistance, and dysregulation of adipokines—proteins secreted by adipose tissue, such as leptin and adiponectin, which influence neuroinflammation and pain pathways. Excess body fat promotes the release of pro-inflammatory cytokines (e.g., TNF-α, IL-6) and oxidative stress, exacerbating trigeminovascular activation and central sensitization [146]. Emerging research suggests that the connection between obesity and migraine may also stem from common pathophysiological pathways, with elevated plasma CGRP levels, a biomarker implicated in migraine, also observed in individuals with obesity [147]. Clinical studies demonstrate that intentional weight loss through lifestyle modifications (diet, exercise), pharmacotherapy, or bariatric surgery correlates with reduced migraine days and intensity [148][149]. While confounding factors like improved diet quality or physical activity may independently contribute, weight loss remains a promising adjunctive strategy for migraine management, underscoring the importance of addressing obesity in holistic care plans [150]. Further research is needed to refine optimal weight loss targets and tailor interventions to individual migraine phenotypes.

8. Nutritional Supplements

Nutritional supplements have emerged as a complementary strategy in migraine management, targeting pathways involved in neuronal excitability, mitochondrial function, and oxidative stress. Magnesium, one of the most studied supplements, plays a critical role in regulating neurotransmitter release and vasodilation; oral magnesium glycinate or citrate (400–600 mg/day) has been shown to significantly reduce migraine frequency [151]. Riboflavin (vitamin B_2), at doses of 400 mg/day, enhances mitochondrial energy production, with randomized trials demonstrating a 50% reduction in attack frequency after 3 months [152]. Coenzyme Q10 (CoQ10), a mitochondrial antioxidant, similarly reduces migraine frequency, duration and severity of attacks at 100–300 mg/day, likely by mitigating oxidative stress and stabilizing neuronal membranes. Feverfew, a herbal supplement, exhibits mixed efficacy but may inhibit prostaglandin synthesis and serotonin release, while butterbur (Petasites hybridus) showed promise in early studies before safety concerns arose due to hepatotoxicity. Emerging evidence supports omega-3 fatty acids for their anti-inflammatory effects, and vitamin D supplementation in deficient patients, linked to reduced headache severity through immunomodulation. However, variability in individual responses, bioavailability issues (e.g., magnesium oxide's poor absorption), and inconsistent dosing protocols underscore the need for personalized approaches guided by biomarkers and clinical evaluation. While these supplements are generally well-tolerated, professional oversight is crucial to avoid interactions and optimize therapeutic synergy with conventional treatments [16][153][154][155][156]. Recently, primary care physicians have also introduced best-practice guidelines known as SEEDS (Sleep, Exercise, Eat, Diary, and Stress) to assist migraine patients in lifestyle modifications [157]. These approaches are especially attractive to patients with chronic conditions who wish to avoid the challenges associated with long-term medication use. In individuals with migraines, the use of such non-pharmacological therapies is increasing and is likely to be significantly undervalued, although further research is definitely much needed [158].

9. HYDRATION

Adequate hydration and electrolyte equilibrium are foundational yet often overlooked components of migraine management. Dehydration reduces cerebral blood flow and elevates blood viscosity, triggering cortical spreading depression and activating trigeminovascular pathways implicated in migraine pain. Even mild dehydration can provoke headaches in susceptible individuals, while chronic low fluid intake correlates with increased migraine frequency and duration [99][159][160][161][162]. Personalized hydration plans, tailored to activity levels and climate, offer a low-risk, accessible adjunct to conventional therapies, emphasizing prevention through consistent fluid and electrolyte monitoring.

10. PREBIOTICS AND PROBIOTICS

The gut-brain axis, a bidirectional communication network linking the gastrointestinal tract and central nervous system, has emerged as a critical player in migraine pathogenesis. Dysbiosis, an imbalance in gut microbiota, may exacerbate migraines potentially due to an increased presence of bacteria that produce nitric oxide (NO) in migraine sufferers' guts. Elevated NO levels may raise CGRP levels, which could trigger migraine attacks [163]. Gut microbes also influence serotonin synthesis, with over 90% of the body's serotonin produced in the gut, and modulate calcitonin gene-related peptide (CGRP), a key mediator of migraine pain. Additionally, bacterial metabolites such as short-chain fatty acids (SCFAs) regulate neuroinflammation and blood-brain barrier integrity, while histamine-producing strains (e.g., *Lactobacillus reuteri*) may directly trigger migraines in sensitive individuals. Probiotics, particularly strains like *Lactobacillus casei*, *Bifidobacterium infantis*, and *Lactobacillus rhamnosus GG*, show therapeutic potential by restoring microbial balance, reducing gut permeability, and dampening systemic inflammation. Clinical studies suggest probiotic supplementation correlates with decreased migraine frequency and severity, possibly through suppressing pro-inflammatory cytokines (e.g., IL-6, TNF-α) and enhancing serotonin availability [23]. Prebiotics, found in certain foods, serve as nourishment for the human microbiome, while probiotics are live beneficial microorganisms that can be consumed through food or supplements. Several studies, including a systematic review, have examined whether probiotics, such as Bifidobacterium, Lactobacillus, Lactococcus, and Streptococcus, can help prevent migraines. However, due to differences in study methods, there is no clear consensus on their effectiveness. While probiotics might reduce both the frequency and severity of migraines, more research is necessary to confirm their role in migraine management [164][165][166].

11. FUTURE DIRECTIONS

The future of dietary approaches in migraine management centers on precision nutrition, utilizing advances in sciences to customize diets based on individual genetic, metabolic, and microbial profiles. Emerging technologies, such as AI-driven apps and wearable sensors, will facilitate real-time monitoring of dietary triggers, symptom patterns, and biomarkers like CGRP and inflammatory cytokines, allowing for dynamic, personalized dietary modifications. Research is expected to focus on identifying epigenetic modulators, nutrients like sulforaphane and omega-3s that influence pain-related gene expression and on optimizing probiotic strains to restore balance within the gut-brain axis in patients with dysbiosis. Combining these approaches with biologic treatments, like CGRP inhibitors, or neuromodulation techniques could produce synergistic benefits. Public health efforts aimed at improving food access and education, along with personalized advice for patients. Ultimately, a shift toward interdisciplinary care-bringing together neurologists, dietitians, and microbiome researchers, will foster evidence-based, holistic dietary strategies that focus not only on migraine prevention but also on enhancing overall metabolic health.

12. CONCLUSION

This comprehensive review underscores the intricate and multifactorial relationship between diet and migraine, highlighting both the potential triggers and therapeutic avenues. While specific foods such as alcohol, processed meats, aged cheeses, and certain additives are well-documented triggers for some individuals, the variability in individual susceptibility, shaped by genetic, metabolic, and gut-brain axis factors, warrants personalized dietary strategies. Emerging evidence suggests that dietary patterns like the Mediterranean, ketogenic, and high omega-3 regimens may offer prophylactic benefits by modulating neurovascular, inflammatory, and gut-related pathways involved in migraine pathophysiology. However, current research is often limited by methodological constraints, and additional well-designed, controlled research is crucial to develop solid, evidence-based guidelines. Understanding the mechanistic links between nutrition, gut microbiota, and migraine pathways opens new therapeutic possibilities, emphasizing the importance of holistic, personalized care. Clinically, patient-centered strategies, such as encouraging increased consumption of fruits and vegetables, reducing processed meats and fast foods, maintaining regular meal times, and avoiding over- or undereating are key. Incorporating dietary modifications alongside lifestyle factors such as physical activity and sleep hygiene, and integrating pharmacological treatments when necessary, can optimize migraine management. Future research should focus on refining personalized dietary interventions, elucidating gut-brain interactions, and developing practical tools for clinicians and patients to effectively implement these strategies. Ultimately, a multidisciplinary approach rooted in emerging scientific insights holds the promise of improving quality of life for individuals suffering from this debilitating disorder.

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