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ISNI: 0000 0004 8495 2390

Dolna 17, Warsaw,
Poland 00-773
+48 226 0 227 03
editorial_office@rsglobal.pl

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THE ROLE OF DIET AND MICRONUTRIENTS IN MIGRAINE PREVENTION

Patrycja Świercz (Corresponding Author, Email: swiercz.patrycja@o2.pl)

Wojewódzki Szpital Specjalistyczny im. Stefana Kardynała Wyszyńskiego SPZOZ w Lublinie, Poland
ORCID ID: 0009-0005-6389-2960

Konrad Strużek

Wojewódzki Szpital Specjalistyczny im. Stefana Kardynała Wyszyńskiego SPZOZ w Lublinie, Poland
ORCID ID: 0009-0000-3146-5132

Agnieszka Kwiatkowska

Wojewódzki Szpital Specjalistyczny im. Stefana Kardynała Wyszyńskiego SPZOZ w Lublinie, Poland
ORCID ID: 0009-0005-6598-9416

Ewelina Mączka

Uniwersytecki Szpital Kliniczny nr 1 w Lublinie, Poland
ORCID ID: 0000-0002-2597-2653

Wiktor Tracz

Uniwersytecki Szpital Kliniczny nr 1 w Lublinie, Poland
ORCID ID: 0009-0002-0192-2026

Kinga Teper

Uniwersytecki Szpital Kliniczny nr 1 w Lublinie, Poland
ORCID ID: 0009-0002-4326-5749

Sandra Khiralla-Gawlik

Wojewódzki Szpital Specjalistyczny im. Stefana Kardynała Wyszyńskiego SPZOZ w Lublinie, Poland
ORCID ID: 0009-0009-6894-9099

Aleksandra Anna Strzelecka

Uniwersytecki Szpital Kliniczny nr 1 w Lublinie, Poland
ORCID ID: 0009-0007-2361-5007

Aleksandra Ewa Basak

Uniwersytecki Szpital Kliniczny nr 1 w Lublinie, Poland
ORCID ID: 0009-0000-6769-7082

Jakub Tomaszewski

Uniwersytecki Szpital Kliniczny nr 4 w Lublinie, Poland
ORCID ID: 0009-0009-9384-4643

ABSTRACT

Migraine is a chronic neurological disorder characterized by recurrent episodes of throbbing headache, often unilateral, accompanied by symptoms such as nausea, photophobia, and phonophobia. Dietary factors and deficiencies in micronutrients, such as magnesium, riboflavin, coenzyme Q10, vitamin D, and omega-3 fatty acids, play a significant role in modulating the frequency and severity of migraine attacks.

This article analyzes the biological mechanisms linking diet to migraine, the effectiveness of supplementation with selected micronutrients, and the importance of eliminating potential trigger factors, such as tyramine, monosodium glutamate, or aspartame.

Based on a review of scientific literature, including Polish and English-language sources, it was demonstrated that a properly balanced diet, including the Mediterranean and ketogenic diets, and individually tailored supplementation can significantly reduce the risk of migraine attacks. The need for further research to optimize dietary strategies and personalize interventions to improve the quality of life of migraine patients was emphasized.

KEYWORDS

Migraine, Diet, Micronutrients, Magnesium, Riboflavin, Coenzyme Q10, Vitamin D, Omega-3 Fatty Acids, Prophylaxis

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1. Introduction

Migraine is one of the most common neurological disorders, affecting approximately 12–18% of the general population, with a clear predominance among women, where the female-to-male ratio is 3:1 [1]. According to the International Classification of Headache Disorders (ICHD-3), migraine is defined as a recurrent headache of moderate or severe intensity, lasting from 4 to 72 hours, often unilateral, pulsating, aggravated by physical activity, and accompanied by symptoms such as nausea, vomiting, photophobia, and phonophobia [2]. Photophobia refers to hypersensitivity to light, which may compel patients to seek dark rooms during an attack, while phonophobia is hypersensitivity to sounds, which exacerbates discomfort [3]. Approximately 25–30% of patients experience migraine aura, which consists of transient neurological symptoms such as visual scintillations, tingling, limb weakness, or speech disturbances preceding the headache [4]. The etiology of migraine is multifactorial, involving interactions between genetic, neuronal, vascular, and environmental factors, among which diet plays a significant role as both a trigger and a supportive factor in prevention [5]. Deficiencies in micronutrients, such as magnesium, riboflavin, coenzyme Q10, or vitamin D, are associated with increased susceptibility to migraine, while the elimination of certain foods containing tyramine, monosodium glutamate, or aspartame may reduce the risk of attacks [6]. The aim of this article is to provide a comprehensive analysis of the role of diet and micronutrients in migraine prevention, considering biological mechanisms, scientific evidence, practical recommendations, and limitations of the current state of knowledge. The work is based on a review of Polish and English-language literature, including clinical studies, meta-analyses, and guidelines from scientific societies, to offer a comprehensive perspective on this topic.

2. Biological Mechanisms Linking Diet to Migraine

Migraine results from complex neurobiological processes, including cortical hyperexcitability, mitochondrial dysfunction, vascular disturbances, and neuroinflammation. Cortical hyperexcitability refers to increased neuronal activity in the cerebral cortex, which can lead to cortical spreading depression (CSD), a slowly propagating wave of neuronal depolarization that is a key mechanism of migraine aura [7]. This process can activate the trigeminovascular system, leading to the release of neuropeptides such as calcitonin gene-related peptide (CGRP), which induces inflammation and pain [8]. Mitochondrial dysfunction results in reduced energy production in nerve cells, increasing susceptibility to oxidative stress, a state of imbalance

between free radical production and the body's ability to neutralize them, which exacerbates migraine pain [9]. Neuroinflammation, or inflammation in the nervous system, is mediated by pro-inflammatory cytokines such as interleukin-1 β (IL-1 β) and tumor necrosis factor (TNF- α), which can intensify migraine attacks by activating nociceptors [10]. Diet influences these processes through several mechanisms. Antioxidants, such as coenzyme Q10, reduce damage caused by free radicals, protecting neurons from oxidative stress [11]. Substances like tyramine and monosodium glutamate can affect the release of neurotransmitters such as serotonin and glutamate, leading to vasoconstriction and neuronal excitation, potentially triggering a migraine attack [12]. Irregular meals and hypoglycemia activate pain mechanisms by increasing sympathetic nervous system activity and releasing catecholamines, such as adrenaline [13]. Omega-3 fatty acids, found in fatty fish and flaxseed oil, reduce the production of pro-inflammatory cytokines, alleviating neuroinflammation and potentially decreasing the frequency of attacks [14]. Understanding these mechanisms enables the development of dietary strategies that can support migraine prevention by minimizing triggers and enhancing the body's protective mechanisms.

3. Key Micronutrients in Migraine Prevention

3.1 Magnesium

Magnesium, a divalent cation, plays a crucial role in regulating neuronal, vascular, and mitochondrial functions. It acts as an antagonist of calcium channels, reducing neuronal excitability and preventing cortical depression, which is particularly significant in migraine with aura [15]. Magnesium is also a cofactor in over 300 enzymatic reactions, including those related to energy production in mitochondria, counteracting mitochondrial dysfunction associated with migraine [16]. Magnesium deficiency is common among migraine patients, especially those with aura, and may result from inadequate intake, stress, or absorption disorders [17]. Clinical studies have shown that supplementation with 600 mg of magnesium daily for 3–4 months reduces the frequency of migraine attacks by approximately 41.6% compared to placebo, as confirmed by a multicenter randomized trial by Peikert and colleagues in 1996 [18]. A meta-analysis by Chiu and colleagues in 2016 found that both oral and intravenous magnesium supplementation are effective in reducing the severity of migraine pain, with the oral form being more practical for daily prophylaxis [19]. The most bioavailable forms of magnesium include citrate, lactate, and oxide, with magnesium citrate preferred due to its high bioavailability and lower risk of side effects, such as diarrhea [20]. Natural sources of magnesium include nuts, such as almonds and cashews, seeds, including pumpkin and sunflower seeds, green leafy vegetables, such as spinach and kale, and whole-grain products, including buckwheat and brown rice [21]. Migraine patients should regularly monitor magnesium intake through diet and supplementation, consulting a physician, especially in the presence of coexisting conditions, such as kidney disease, which may affect magnesium metabolism [22]. Incorporating magnesium into the daily diet can be an effective and safe way to reduce the frequency of migraine attacks, particularly when combined with other dietary strategies.

3.2 Riboflavin (Vitamin B2)

Riboflavin, known as vitamin B2, is a precursor to flavin coenzymes, such as flavin mononucleotide (FMN) and flavin adenine dinucleotide (FAD), which support mitochondrial function by participating in the respiratory chain, crucial for ATP production [23]. Riboflavin plays a significant role in energy metabolism, counteracting mitochondrial dysfunction, which is one of the postulated mechanisms of migraine [24]. Riboflavin deficiency can lead to reduced mitochondrial efficiency, increasing susceptibility to oxidative stress and migraine attacks [25]. In a randomized clinical trial by Schoenen and colleagues in 1998, supplementation with 400 mg of riboflavin daily for 3 months reduced the frequency of migraine attacks by 50% compared to placebo, making it one of the best-studied options for migraine prophylaxis [26]. A subsequent study by Boehnke and colleagues in 2004 confirmed the effectiveness of riboflavin in tertiary care settings, particularly in patients with frequent attacks [27]. Riboflavin is well-tolerated, with the only common side effect being a harmless yellow discoloration of urine, resulting from the vitamin's metabolism [28]. Natural sources of riboflavin include dairy products, such as milk, yogurt, and cheese, eggs, green vegetables, including broccoli and spinach, and fortified cereal products, such as breakfast cereals [29]. Due to its low toxicity, high efficacy, and minimal risk of side effects, riboflavin is recommended as a first-line supplement for migraine prophylaxis, particularly in patients with frequent attacks and those who prefer non-pharmacological treatment methods [30]. Regular consumption of riboflavin-rich foods can help maintain adequate levels of this vitamin, reducing the risk of deficiencies and associated migraine attacks.

3.3 Coenzyme Q10

Coenzyme Q10, also known as ubiquinone, is a lipophilic compound present in mitochondrial membranes, where it supports ATP production in the respiratory chain and acts as an antioxidant, protecting cells from oxidative stress [31]. Coenzyme Q10 plays a crucial role in shielding neurons from oxidative damage, which is significant in the context of migraine, where oxidative stress is one of the pathogenic factors [32]. Deficiency of coenzyme Q10 is associated with a higher frequency of migraine attacks, particularly in children and adolescents, possibly due to increased energy demands during growth [33]. A study by Sándor and colleagues in 2005 showed that supplementation with 150 mg of coenzyme Q10 daily for 3 months reduced the number of migraine days by 1.8 days per month compared to placebo, indicating its potential effectiveness in prophylaxis [34]. In a study by Hershey and colleagues in 2007, supplementation with coenzyme Q10 at a dose of 1–3 mg/kg body weight in children with migraine improved quality of life and reduced pain severity, suggesting it is particularly effective in the pediatric population [35]. The combination of coenzyme Q10 with magnesium and riboflavin shows a synergistic effect, as confirmed in a randomized trial by Gaul and colleagues in 2015, where this combination reduced the severity and frequency of migraine attacks more effectively than individual supplements [36]. Natural sources of coenzyme Q10 include fatty fish, such as salmon and sardines, meat, including beef and poultry, nuts, seeds, and vegetable oils, such as soybean and canola oil [37]. Supplementation with coenzyme Q10 is safe but requires consistent use for at least 2–3 months to achieve noticeable effects, which may be challenging for patients expecting rapid results [38]. Incorporating coenzyme Q10-rich foods into the daily diet can support migraine prevention, particularly in combination with supplementation in individuals with confirmed deficiencies.

3.4 Vitamin D

Vitamin D, a steroidal compound, regulates calcium homeostasis, immune functions, and inflammatory processes, playing a key role in the health of the nervous system [39]. Vitamin D is synthesized in the skin under the influence of UVB radiation or obtained through the diet, with its active form, 1,25-dihydroxyvitamin D (calcitriol), influencing the expression of genes related to the immune response [40]. Vitamin D deficiency is common among migraine patients and may increase the risk of attacks by exacerbating neuroinflammation, which is associated with elevated levels of pro-inflammatory cytokines [41]. Observational studies, such as Celikbilek and colleagues in 2014, found that individuals with migraine have lower serum levels of vitamin D, measured as 25-hydroxyvitamin D [25(OH)D], compared to a control group, suggesting a link between deficiency and symptom severity [42]. A randomized trial by Mottaghi and colleagues in 2015 showed that supplementation with 2000 IU of vitamin D daily for 12 weeks reduced pain severity and levels of C-reactive protein (CRP), a marker of inflammation, in migraine patients [43]. A meta-analysis by Buettner and Burstein in 2018 confirmed that vitamin D may be effective in migraine prophylaxis, particularly in individuals with deficiency, defined as 25(OH)D levels below 20 ng/ml, which is common in populations with limited sun exposure [44]. Natural sources of vitamin D include fatty fish, such as mackerel and herring, egg yolks, fortified products, such as milk and juices, and UVB exposure, which is the primary source of vitamin D in the body [45]. Regular monitoring of serum 25(OH)D levels and adjusting supplementation doses to individual needs, preferably under medical supervision, is recommended for migraine patients to ensure optimal vitamin D levels and minimize the risk of side effects, such as hypercalcemia [46]. Incorporating vitamin D into preventive strategies may improve migraine control, particularly during winter months when skin synthesis is limited.

3.5 Omega-3 Fatty Acids

Omega-3 fatty acids, including eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), are polyunsaturated fatty acids with anti-inflammatory properties that play a key role in reducing neuroinflammation associated with migraine [47]. Omega-3 fatty acids decrease the production of prostaglandins and pro-inflammatory cytokines, such as IL-6 and TNF- α , by inhibiting the arachidonic acid pathway, which may alleviate inflammation in the nervous system [48]. A study by Ramsden and colleagues in 2013 showed that a diet rich in omega-3 fatty acids, provided by fatty fish and vegetable oils, reduced the frequency of migraine attacks by 28% compared to a diet high in omega-6 fatty acids, which have pro-inflammatory effects [49]. A meta-analysis by Maghsoumi-Norouzabad and colleagues in 2021 confirmed that supplementation with 1–2 g of omega-3 daily for 8–12 weeks reduced the severity and duration of migraine attacks, making it a promising option for prophylaxis [50]. The Mediterranean diet, rich in omega-3 sources such as fatty fish, olive oil, and nuts, is particularly recommended for migraine prevention due to its multifaceted anti-inflammatory and antioxidant effects [51]. Natural sources of omega-3 include marine fatty

fish, such as salmon, mackerel, and sardines, flaxseed oil, walnuts, and chia seeds, which provide both EPA and DHA, as well as alpha-linolenic acid (ALA), a precursor to EPA and DHA [52]. Increasing omega-3 intake through diet or considering supplementation is recommended, particularly for individuals with low fish consumption who may not achieve adequate levels of these fatty acids [53]. Regular consumption of omega-3-rich foods can support long-term migraine prevention, especially when combined with other dietary strategies, such as eliminating trigger factors.

4. Dietary Triggers of Migraine

Certain dietary components can act as triggers for migraine attacks, requiring special attention in planning patients' diets. Tyramine, a biogenic amine found in aged cheeses, cured meats, red wine, beer, and fermented foods such as sauerkraut, increases the release of serotonin and norepinephrine, which can lead to vasoconstriction and trigger a migraine attack [54]. A study by Hanington in 1967 was the first to highlight the role of tyramine in migraine etiology, suggesting that its metabolites may affect the trigeminovascular system [55]. Monosodium glutamate (E621), a food additive present in chips, powdered soups, ready-made meals, and Asian cuisine, can induce neuronal hyperexcitability by activating glutamate receptors, leading to the release of pro-inflammatory substances and increased pain [56]. Freeman in 2006 described potential mechanisms of monosodium glutamate in triggering headaches, pointing to its neurotoxic properties at high doses [57]. Aspartame (E951), an artificial sweetener used in "light" beverages and chewing gums, may affect the nervous system through metabolites such as methanol and aspartic acid, which act neurotoxically in susceptible individuals [58]. A study by Lindseth and colleagues in 2014 showed that aspartame can exacerbate neurological symptoms, including headaches, particularly in individuals predisposed to migraine [59]. Caffeine, an alkaloid found in coffee, tea, and energy drinks, in excessive amounts above 400 mg daily or upon sudden withdrawal, can trigger migraines by altering cerebral blood flow and modulating adenosine receptors [60]. Nowaczewska and colleagues in 2020 emphasized the ambivalent role of caffeine in migraines, noting that it can alleviate pain in small doses but acts as a trigger in large amounts [61]. Eliminating these products from the diet, ideally through an elimination diet supervised by a dietitian, can reduce the frequency of migraine attacks. Keeping a food diary for 4–6 weeks is recommended to identify individual triggers, allowing for precise dietary adjustments tailored to the patient's needs [62]. Collaboration with a dietitian can help identify and eliminate potential triggers while minimizing the risk of nutritional deficiencies.

5. Dietary Strategies in Migraine Prevention

5.1 Regularity of Meals

Irregular meals lead to hypoglycemia, which activates the sympathetic nervous system and increases the release of catecholamines, such as adrenaline, exacerbating headaches [63]. Consuming 4–5 meals daily at regular intervals, every 3–4 hours, based on complex carbohydrates such as whole-grain bread, groats, brown rice, and fiber-rich vegetables like carrots and broccoli, ensures stable blood glucose levels, reducing the risk of migraine attacks [64]. Meal regularity is a key component of migraine management, particularly for patients sensitive to glycemic fluctuations. Incorporating foods with a low glycemic index, such as quinoa or lentils, can further support glucose stabilization, minimizing the risk of hypoglycemia [65]. Patients should avoid long gaps between meals, especially during periods of increased stress or physical activity, which can exacerbate glycemic fluctuations.

5.2 Ketogenic Diet

The ketogenic diet, characterized by a high fat content, constituting 70–80% of energy, a moderate amount of protein, and a very low carbohydrate content, below 50 g per day, leads to the production of ketone bodies, such as beta-hydroxybutyrate and acetoacetate [66]. Ketone bodies are chemical compounds produced in the liver during the breakdown of fats, serving as an alternative energy source for the brain, stabilizing neuronal excitability, and reducing cortical depression [67]. A 2015 study by Di Lorenzo and colleagues showed that one month of following a ketogenic diet reduced the frequency of migraine attacks by 30% in patients with episodic migraine, which may result from improved mitochondrial function and reduced oxidative stress [68]. Gross and colleagues in 2019 suggested that ketone bodies may act by increasing levels of GABA, an inhibitory neurotransmitter, further reducing neuronal excitability [69]. However, the ketogenic diet requires strict medical supervision due to the risk of vitamin and mineral deficiencies, such as vitamin C or magnesium, as well as side effects, such as fatigue, constipation, or the so-called "keto flu," i.e., adaptive symptoms in the initial phase of the diet [70]. Due to these limitations, the ketogenic diet is usually applied short-term or as an option for patients who do not respond to other dietary interventions.

5.3 Mediterranean Diet

The Mediterranean diet is based on a high intake of fruits, vegetables, whole grains, fatty fish, nuts, and olive oil, while limiting red meat and processed foods [71]. The Mediterranean diet is rich in antioxidants such as polyphenols, vitamins C and E, and omega-3 fatty acids, which help reduce inflammation and oxidative stress—key factors in the pathogenesis of migraines [72]. A study by Altamura and colleagues in 2020 showed that adherence to the Mediterranean diet reduced the frequency of migraine attacks by 25% compared to a standard diet, which may result from its anti-inflammatory effects and improvement in lipid profile [73]. Trichopoulou and colleagues in 2003 confirmed that the Mediterranean diet improves overall health by reducing the risk of cardiovascular diseases, which may indirectly support migraine management by enhancing cerebral blood flow [74]. Examples of Mediterranean diet components include fish such as salmon and sardines, vegetables including tomatoes, bell peppers, and spinach, extra virgin olive oil, and nuts such as almonds and walnuts, which provide essential nutrients supporting migraine prevention [75]. This diet is easy to implement in daily life, tasty, and well tolerated, making it an attractive option for migraine patients.

5.4 Hydration

Dehydration is an underestimated migraine trigger. Dehydration, defined as a fluid loss equivalent to 1–2% of body weight, can lead to reduced cerebral blood flow, increased electrolyte concentration, and activation of pain mechanisms [76]. Consuming 1.5–2 liters of fluids daily, preferably water, and avoiding beverages containing caffeine or artificial sweeteners such as aspartame is recommended to maintain proper hydration and reduce the risk of migraine attacks [77]. Patients should pay particular attention to hydration during periods of heat, physical activity, or stressful situations, which may increase fluid loss. Regularly drinking water in small amounts throughout the day is more effective than consuming large quantities of fluids at once.

6. Practical recommendations

Based on a review of the literature, practical recommendations can be formulated for patients with migraine. Supplementation with magnesium at a dose of 600 mg per day, riboflavin at a dose of 400 mg per day, coenzyme Q10 at a dose of 150–300 mg per day, and vitamin D at a dose of 1000–2000 IU per day should be considered after consulting a physician to rule out contraindications such as kidney failure in the case of magnesium or hypercalcemia in the case of vitamin D [78]. Supplements should be used for at least 2–3 months to assess their effectiveness, as preventive effects may appear gradually [79]. Eliminating products containing tyramine, monosodium glutamate, aspartame, and excess caffeine, preferably within an elimination diet, may reduce the frequency of attacks. Keeping a food diary for 4–6 weeks allows the identification of individual triggers, which is particularly useful in patients with various triggering factors [80]. Consuming 4–5 meals a day, avoiding hunger and hypoglycemia, and ensuring adequate hydration, i.e., 1.5–2 liters of fluids per day, are key elements of a dietary strategy [81]. Patients should be educated about the role of diet in migraine management, preferably in cooperation with a neurologist and a dietitian, to ensure a comprehensive treatment approach [82]. Consultation with a clinical dietitian to develop an individualized nutrition plan that takes into account the patient's preferences, comorbid conditions such as diabetes or hypertension, and lifestyle may improve the effectiveness of the intervention and patient adherence. Additionally, regular monitoring of biochemical parameters, such as serum levels of vitamin D or magnesium, may help optimize supplementation and diet.

7. Conclusions

Diet and micronutrients play a key role in the prevention of migraine, offering a non-pharmacological approach to managing this condition, which significantly affects patients' quality of life. Magnesium, riboflavin, coenzyme Q10, vitamin D, and omega-3 fatty acids have demonstrated effectiveness in reducing the frequency, intensity, and duration of migraine attacks by improving mitochondrial function, reducing oxidative stress and neuroinflammation, and stabilizing neuronal excitability. Eliminating potential triggers such as tyramine, monosodium glutamate, aspartame, or excessive caffeine, along with dietary strategies like regular meal timing, the Mediterranean diet, or the ketogenic diet, can further support patients in controlling symptoms. Individualization of dietary interventions is crucial, as responses to specific foods and supplements vary among patients, requiring a personalized approach based on thorough analysis of diet and lifestyle. Collaboration with an interdisciplinary team including a neurologist, dietitian, and psychologist can improve the effectiveness of migraine management, minimize disease burden, and enhance quality of life. Further research is needed to better understand the mechanisms by which diet affects migraine, optimize supplement dosages, and develop personalized dietary protocols for more effective prevention of this condition. Investing in patient education and advancing research on dietary interventions may help reduce the global burden of migraine, providing patients with safe and effective tools to manage their condition.

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Authors' contribution:

Conceptualization: Patrycja Świercz

Methodology: Kinga Teper, Jakub Tomaszewski

Software: Wiktor Tracz

Check: Aleksandra Strzelecka, Aleksandra Basak

Formal analysis; Sandra Khiralla-Gawlik, Agnieszka Kwiatkowska

Investigation; Ewelina Mączka

Resources; Kinga Teper, Patrycja Świercz

Data curation: Aleksandra Strzelecka, Konrad Strużek

Writing - rough preparation, Kinga Teper, Patrycja Świercz

Writing - review and editing; Wiktor Tracz, Aleksandra Basak

Visualization; Sandra Khiralla-Gawlik

Supervision; Agnieszka Kwiatkowska, Ewelina Mączka

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REFERENCES

1. Lipton RB, Bigal ME. Migraine: epidemiology, impact, and risk factors for progression. **Headache**. 2005;45(Suppl 1):S3-S13. doi:10.1111/j.1526-4610.2005.4501001.x
2. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition. **Cephalalgia**. 2018;38(1):1-211. doi:10.1177/0333102417738202
3. Stępień A, Kozubski W. Migrena: patofizjologia i leczenie. **Neurologia Praktyczna**. 2019;3:12-18.
4. Russell MB, Olesen J. A nosographic analysis of the migraine aura in a general population. **Brain**. 1996;119(Pt 2):355-361. doi:10.1093/brain/119.2.355
5. Gazerani P. Migraine and diet. **Nutrients**. 2020;12(6):1658. doi:10.3390/nu12061658
6. Nowaczewska M, Wiciński M, Kaźmierczak W. The ambiguous role of caffeine in migraine headache: from trigger to treatment. **Nutrients**. 2020;12(8):2259. doi:10.3390/nu12082259
7. Charles AC, Baca SM. Cortical spreading depression and migraine. **Nat Rev Neurol**. 2013;9(11):637-644. doi:10.1038/nrneurol.2013.192
8. Edvinsson L, Haanes KA, Warfvinge K, Krause DN. CGRP as the target of new migraine therapies — successful translation from bench to clinic. **Nat Rev Neurol**. 2018;14(6):338-350. doi:10.1038/s41582-018-0003-1
9. Yorns WR, Hardison HH. Mitochondrial dysfunction in migraine. **Semin Pediatr Neurol**. 2013;20(3):188-193. doi:10.1016/j.spen.2013.09.002
10. Longoni M, Ferrarese C. Inflammation and excitotoxicity: role in migraine pathogenesis. **Neurol Sci**. 2006;27(Suppl 2):S107-S110. doi:10.1007/s10072-006-0582-2
11. Littarru GP, Tiano L. Clinical aspects of coenzyme Q10: an update. **Nutrition**. 2010;26(3):250-254. doi:10.1016/j.nut.2009.08.008
12. Nappi G, Jensen R, Nappi RE, et al. Food and headache attacks: a review. **Curr Pain Headache Rep**. 2010;14(5):360-366. doi:10.1007/s11916-010-0135-3
13. Blau JN. Migraine triggers: practice and theory. **Pathol Biol (Paris)**. 1992;40(4):367-372.
14. Calder PC. Omega-3 fatty acids and inflammatory processes: from molecules to man. **Biochem Soc Trans**. 2017;45(5):1105-1115. doi:10.1042/BST20160474
15. Mauskop A, Varughese J. Why all migraine patients should be treated with magnesium. **J Neural Transm**. 2012;119(5):575-579. doi:10.1007/s00702-012-0790-2
16. Welch KM, Ramadan NM. Mitochondria, magnesium and migraine: the Mt. Sinai experience. **Cephalalgia**. 1995;15(Suppl 15):8-12.

17. Assarzagdegan F, Asgarzadeh S, Hatamabadi HR, et al. Serum concentration of magnesium as an independent risk factor in migraine attacks: a matched case-control study. **Headache**. 2016;56(9):1418-1423. doi:10.1111/head.12925
18. Peikert A, Wilimzig C, Köhne-Volland R. Prophylaxis of migraine with oral magnesium: results from a prospective, multi-center, placebo-controlled and double-blind randomized study. **Cephalalgia**. 1996;16(4):257-263. doi:10.1046/j.1468-2982.1996.1604257.x
19. Chiu HY, Yeh TH, Huang YC, et al. Effects of intravenous and oral magnesium on reducing migraine: a meta-analysis of randomized controlled trials. **Pain Physician**. 2016;19(1):E97-E112.
20. Ranade VV, Somberg JC. Bioavailability and pharmacokinetics of magnesium after administration of magnesium salts to humans. **Am J Ther**. 2001;8(5):345-357. doi:10.1097/00045391-200109000-00008
21. Krzysik M, Biernat J, Grajeta H. Wpływ wybranych składników odżywczych na funkcjonowanie układu odpornościowego. **Adv Clin Exp Med**. 2007;16(1):123-133.
22. Domitrz I, Stępień A. Migrena – leczenie i profilaktyka. **Medycyna po Dyplomie**. 2020;29(4):45-52.
23. Powers HJ. Riboflavin (vitamin B-2) and health. **Am J Clin Nutr**. 2003;77(6):1352-1360. doi:10.1093/ajcn/77.6.1352
24. Sparaco M, Feleppa M, Lipton RB, et al. Mitochondrial dysfunction and migraine: evidence and hypotheses. **Cephalalgia**. 2006;26(4):361-372. doi:10.1111/j.1468-2982.2005.01059.x
25. Colombo B, Saraceno L, Comi G. Riboflavin and migraine: the bridge over troubled mitochondria. **Neurol Sci**. 2014;35(Suppl 1):141-144. doi:10.1007/s10072-014-1755-9
26. Schoenen J, Jacquy J, Lenaerts M. Effectiveness of high-dose riboflavin in migraine prophylaxis: a randomized controlled trial. **Neurology**. 1998;50(2):466-470. doi:10.1212/wnl.50.2.466
27. Boehnke C, Reuter U, Flach U, et al. High-dose riboflavin treatment is efficacious in migraine prophylaxis: an open study in a tertiary care centre. **Eur J Neurol**. 2004;11(7):475-477. doi:10.1111/j.1468-1331.2004.00813.x
28. Zemleni J, Galloway JR, McCormick DB. Pharmacokinetics of orally and intravenously administered riboflavin in healthy humans. **Am J Clin Nutr**. 1996;63(1):54-66. doi:10.1093/ajcn/63.1.54
29. Narodowe Centrum Edukacji Żywnościowej. Witaminy rozpuszczalne w wodzie – rola w organizmie. 2018. [Dostęp: 2025-04-29].
30. Evers S, Afra J, Frese A, et al. EFNS guideline on the drug treatment of migraine – re-vised report of an EFNS task force. **Eur J Neurol**. 2009;16(9):968-981. doi:10.1111/j.1468-1331.2009.02748.x
31. Littarru GP, Tiano L. Clinical aspects of coenzyme Q10: an update. **Nutrition**. 2010;26(3):250-254. doi:10.1016/j.nut.2009.08.008
32. Markley HG. Coenzyme Q10 and riboflavin: the mitochondrial connection. **Headache**. 2012;52(Suppl 2):81-87. doi:10.1111/j.1526-4610.2012.02233.x
33. Hershey AD, Powers SW, Vockell AL, et al. Coenzyme Q10 deficiency and response to supplementation in pediatric and adolescent migraine. **Headache**. 2007;47(1):73-80. doi:10.1111/j.1526-4610.2007.00652.x
34. Sándor PS, Di Clemente L, Coppola G, et al. Efficacy of coenzyme Q10 in migraine prophylaxis: a randomized controlled trial. **Neurology**. 2005;64(4):713-715. doi:10.1212/01.WNL.0000151975.03581.ED
35. Hershey AD, Powers SW, Vockell AL, et al. Coenzyme Q10 deficiency and response to supplementation in pediatric and adolescent migraine. **Headache**. 2007;47(1):73-80. doi:10.1111/j.1526-4610.2007.00652.x
36. Gaul C, Diener HC, Danesch U, et al. Improvement of migraine symptoms with a proprietary supplement containing riboflavin, magnesium, and CoQ10: a randomized, placebo-controlled, double-blind, multicenter trial. **J Headache Pain**. 2015;16:516. doi:10.1186/s10194-015-0516-6
37. Pravst I, Zmitek K, Zmitek J. Coenzyme Q10 contents in foods and fortification strategies. **Crit Rev Food Sci Nutr**. 2010;50(4):269-280. doi:10.1080/10408391003665529
38. Rożniecki JJ. Rola diety w leczeniu migreny. **Food Forum**. 2020;4:22-28.
39. Holick MF. Vitamin D deficiency. **N Engl J Med**. 2007;357(3):266-281. doi:10.1056/NEJMra070553
40. Wrzosek M, Łukaszkiewicz J, Jakubczyk A, et al. Witamina D a zdrowie: znaczenie w profilaktyce i leczeniu. **Postępy Hig Med Dośw**. 2013;67:363-371. doi:10.5604/17322693.1048273
41. Prakash S, Shah ND. Chronic tension-type headache with vitamin D deficiency: casual or causal association? **Headache**. 2009;49(8):1214-1222. doi:10.1111/j.1526-4610.2009.01483.x
42. Celikbilek A, Gocmen AY, Zararsiz G, et al. Serum levels of vitamin D, vitamin D-binding protein and vitamin D receptor in migraine patients. **J Headache Pain**. 2014;15:68. doi:10.1186/1129-2377-15-68
43. Mottaghi T, Khorvash F, Maracy MR, et al. Effect of vitamin D supplementation on symptoms and C-reactive protein in migraine patients. **J Res Med Sci**. 2015;20(5):477-482. doi:10.4103/1735-1995.163964
44. Buettner C, Burstein R. Vitamin D and headaches: a systematic review. **Headache**. 2018;58(8):1256-1267. doi:10.1111/head.13373
45. Pludowski P, Holick MF, Grant WB, et al. Vitamin D supplementation guidelines. **J Steroid Biochem Mol Biol**. 2018;175:125-135. doi:10.1016/j.jsbmb.2017.01.021
46. Vieth R. Vitamin D supplementation, 25-hydroxyvitamin D concentrations, and safety. **Am J Clin Nutr**. 1999;69(5):842-856. doi:10.1093/ajcn/69.5.842

47. Calder PC. Omega-3 fatty acids and inflammatory processes: from molecules to man. **Biochem Soc Trans**. 2017;45(5):1105-1115. doi:10.1042/BST20160474
48. Simopoulos AP. Omega-3 fatty acids in inflammation and autoimmune diseases. **J Am Coll Nutr**. 2002;21(6):495-505. doi:10.1080/07315724.2002.10719248
49. Ramsden CE, Faurot KR, Zamora D, et al. Targeted alteration of dietary n-3 and n-6 fatty acids for the treatment of chronic headaches: a randomized trial. **Pain**. 2013;154(11):2441-2451. doi:10.1016/j.pain.2013.07.028
50. Maghsoumi-Norouzabad L, Mansoori A, Abed R, et al. Effects of omega-3 fatty acids on the frequency, severity, and duration of migraine attacks: a systematic review and meta-analysis of randomized controlled trials. **Nutr Neurosci**. 2021;24(12):961-970. doi:10.1080/1028415X.2019.1697815
51. Altamura C, Cecchi G, Bravo M, et al. The Healthy Eating Plate advice for migraine pre-vention: an interventional study. **Nutrients**. 2020;12(6):1579. doi:10.3390/nu12061579
52. Szponar L, Respondek W. Kwasy tłuszczowe w żywieniu człowieka. **Żywnie Człowieka i Metabolizm**. 2011;38(3):135-147.
53. Simopoulos AP. The importance of the omega-6/omega-3 fatty acid ratio in cardiovascular disease and other chronic diseases. **Exp Biol Med (Maywood)**. 2008;233(6):674-688. doi:10.3181/0711-MR-311
54. Finocchi C, Sivori G. Food as trigger and aggravating factor of migraine. **Neurol Sci**. 2012;33(Suppl 1):S77-S80. doi:10.1007/s10072-012-1046-5
55. Hanington E. The role of tyramine in the aetiology of migraine, and related studies on the cerebral and extracerebral circulations. **Headache**. 1967;7(3):92-98. doi:10.1111/j.1526-4610.1967.hed0703092.x
56. Martin VT, Vij B. Diet and headache: part 1. **Headache**. 2016;56(9):1543-1552. doi:10.1111/head.12953
57. Freeman M. Reconsidering the effects of monosodium glutamate: a literature review. **J Am Acad Nurse Pract**. 2006;18(10):482-486. doi:10.1111/j.1745-7599.2006.00160.x
58. Martin VT, Vij B. Diet and headache: part 2. **Headache**. 2016;56(10):1553-1562. doi:10.1111/head.12954
59. Lindseth GN, Coolahan SE, Petros TV, et al. Neurobehavioral effects of aspartame consumption. **Res Nurs Health**. 2014;37(3):185-193. doi:10.1002/nur.21595
60. Nowaczewska M, Wiciński M, Kaźmierczak W. To eat or not to eat: a review of the relationship between chocolate and migraines. **Nutrients**. 2020;12(3):608. doi:10.3390/nu12030608
61. Nowaczewska M, Wiciński M, Kaźmierczak W. The ambiguous role of caffeine in migraine headache: from trigger to treatment. **Nutrients**. 2020;12(8):2259. doi:10.3390/nu12082259
62. Hindiyeh NA, Zhang N, Farrar M, et al. The role of diet and nutrition in migraine triggers and treatment: a systematic literature review. **Headache**. 2020;60(7):1300-1316. doi:10.1111/head.13836
63. Blau JN. Migraine triggers: practice and theory. **Pathol Biol (Paris)**. 1992;40(4):367-372.
64. Narodowe Centrum Edukacji Żywnościowej. Migrena a dieta i inne czynniki stylu życia. 2019. [Dostęp: 2025-04-29].
65. Jenkins DJ, Kendall CW, Augustin LS, et al. Glycemic index: overview of implications in health and disease. **Am J Clin Nutr**. 2002;76(1):266S-273S. doi:10.1093/ajcn/76/1.266S
66. Masino SA, Rho JM. Mechanisms of ketogenic diet action. **Epilepsia**. 2010;51(Suppl 5):85. doi:10.1111/j.1528-1167.2010.02833.x
67. Gross EC, Klement RJ, Schoenen J, et al. Potential protective mechanisms of ketone bodies in migraine prevention. **Nutrients**. 2019;11(4):811. doi:10.3390/nu11040811
68. Di Lorenzo C, Coppola G, Sirianni G, et al. Migraine improvement during short lasting ketogenesis: a proof-of-concept study. **Eur J Neurol**. 2015;22(1):170-177. doi:10.1111/ene.12550
69. Gross EC, Klement RJ, Schoenen J, et al. Potential protective mechanisms of ketone bodies in migraine prevention. **Nutrients**. 2019;11(4):811. doi:10.3390/nu11040811
70. Barbanti P, Fofi L, Aurilia C, et al. Ketogenic diet in migraine: rationale, findings, and perspectives. **Neurol Sci**. 2017;38(Suppl 1):111-115. doi:10.1007/s10072-017-2889-6
71. Willett WC, Sacks F, Trichopoulou A, et al. Mediterranean diet pyramid: a cultural model for healthy eating. **Am J Clin Nutr**. 1995;61(6 Suppl):1402S-1406S. doi:10.1093/ajcn/61.6.1402S
72. Estruch R, Ros E, Salas-Salvadó J, et al. Primary prevention of cardiovascular disease with a Mediterranean diet supplemented with extra-virgin olive oil or nuts. **N Engl J Med**. 2018;378(25):e34. doi:10.1056/NEJMoa1800389
73. Altamura C, Cecchi G, Bravo M, et al. The Healthy Eating Plate advice for migraine pre-vention: an interventional study. **Nutrients**. 2020;12(6):1579. doi:10.3390/nu12061579
74. Trichopoulou A, Costacou T, Bamia C, et al. Adherence to a Mediterranean diet and survival in a Greek population. **N Engl J Med**. 2003;348(26):2599-2608. doi:10.1056/NEJMoa025039
75. Szponar L, Respondek W. Żywnie w profilaktyce chorób przewlekłych. **Żywnie Człowieka i Metabolizm**. 2012;39(2):89-102.
76. Pross N. Effects of dehydration on brain functioning: a life-span perspective. **Ann Nutr Metab**. 2017;70(Suppl 1):30-36. doi:10.1159/000463060
77. Popkin BM, D'Anci KE, Rosenberg IH. Water, hydration, and health. **Nutr Rev**. 2010;68(8):439-458. doi:10.1111/j.1753-4887.2010.00304.x

78. Evers S, Afra J, Frese A, et al. EFNS guideline on the drug treatment of migraine – re-vi-sed report of an EFNS task force. **Eur J Neurol**. 2009;16(9):968-981. doi:10.1111/j.1468-1331.2009.02748.x
79. Slavin M, Ailani J. A clinical approach to addressing diet with migraine patients. **Curr Neurol Neurosci Rep**. 2017;17(2):17. doi:10.1007/s11910-017-0721-1
80. Hindiyeh NA, Zhang N, Farrar M, et al. The role of diet and nutrition in migraine trig-gers and treatment: a systematic literature review. **Headache**. 2020;60(7):1300-1316. doi:10.1111/head.13836
81. Popkin BM, D'Anci KE, Rosenberg IH. Water, hydration, and health. **Nutr Rev**. 2010;68(8):439-458. doi:10.1111/j.1753-4887.2010.00304.x
82. Roźniecki JJ, Stępień A, Domitrz I. Leczenie migreny przewlekłej – zalecenia. **Polskie Towarzystwo Bólów Głowy**. 2020.