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To feed or to fast? Nutritional triggers in migraine: a narrative review

Daniele Pala,[1,2] Federico Tosto,[3] Antonio Munafò,[4,5] Andrea Burgalassi,[4,5] Marina Romozzi,[6,7] Luigi Francesco Iannone,[8,9] Catello Vollono,[6,7] Paolo Calabresi,[6,7] Francesco Casillo,[10] Cherubino Di Lorenzo[10]

[1]Department of Medical Science and Public Health, University of Cagliari; [2]Department of Biomedical Sciences, University of Cagliari; [3]Department of Neuroscience, Giovanni Paolo II Hospital, Catanzaro; [4]Section of Clinical Pharmacology and Oncology, Department of Health Sciences, University of Florence; [5]Headache Center and Clinical Pharmacology Unit, Careggi University Hospital, Florence; [6]Department of Neurosciences, Università Cattolica del Sacro Cuore, Rome; [7]Neurology, Dipartimento di Neuroscienze, Organi di Senso e Torace, Fondazione Policlinico Universitario Agostino Gemelli IRCCS, Rome; [8]Department of Biomedical, Metabolic and Neural Sciences, University of Modena and Reggio Emilia, Modena; [9]Digital and Predictive Medicine, Pharmacology, Clinical Metabolic Toxicology - Headache Center and Drug Abuse, Laboratory of Clinical Pharmacology and Pharmacogenomics, Policlinico di Modena; [10]Department of Medico-Surgical Sciences and Biotechnologies, Sapienza University of Rome, Polo Pontino, ICOT Istituto Marco Pasquali, Latina, Italy

ABSTRACT

Background: Migraine attacks typically emerge in predisposed individuals after exposure to heterogeneous stimuli that vary widely across patients. A system-level perspective highlights migraine as a disturbance of homeostasis and allostasis: the brain integrates multiple inputs and generates adaptive responses to maintain physiological stability, but sustained physiological or paraphysiological stressors may impose excessive allostatic load. When compensatory mechanisms become energetically costly or insufficient, an "allostatic reset" may occur, clinically manifesting as a migraine attack. Within this framework, the concept of a migraine trigger is better viewed as a threshold modulator rather than a deterministic cause, *i.e.*, a stimulus that lowers the attack threshold in susceptible individuals. Nutrition is among the most frequently implicated domains, including fasting, dehydration, specific foods and additives, and overall dietary patterns.

Methods: This narrative review synthesizes current evidence on dietary exposures as potential migraine precipitants and modulators of disease course.

Results: The most frequently reported food-related triggers include fasting, dehydration, alcohol, coffee and caffeine withdrawal, chocolate, milk and dairy products, processed and cured meats rich in nitrites and nitrates, citrus fruits, tea, onions, tomatoes, ice cream, nuts, spicy foods, and ultra-processed foods. Food additives such as monosodium glutamate, aspartame, sulfites, and other artificial sweeteners have also been repeatedly implicated, alongside dietary histamine and biogenic amines. Importantly, several studies suggest that overall dietary patterns, characterized by high glycemic load, irregular meal timing, excessive sugar and saturated fat intake, or ultra-processed foods, may exert a greater influence on migraine susceptibility than single food items.

Conclusions: Overall, the literature is fragmented and often contradictory, with a frequent mismatch between patient-reported triggers and results from blinded challenge studies, underscoring the roles of recall bias, expectancy effects, and prodromal symptoms (*e.g.*, food cravings) misattributed to causation. Inter- and intra-individual variability – shaped by genetic background, metabolic state, gut-brain axis mechanisms, and comorbidities – suggests that "one-size-fits-all" dietary restrictions are inappropriate. Rather than endorsing broad exclusion lists, current evidence supports personalized trigger identification and prioritization of protective dietary patterns, regular hydration, and consistent meal timing to reduce attack susceptibility and overall disease burden.

Key words: triggers, diet, migraine.

Introduction

The identification of the molecular and neuroanatomical correlates of migraine has returned clear clinical results, such as the commercialization of calcitonin gene-related peptide (CGRP)-targeting therapies. (1) However, our overall understanding of the chain of events that lead to a migraine attack is still unclear. (2) A migraine attack seems to occur, in predisposed patients, in most cases following exposure to various stimuli, which may substantially differ between one individual and another. (3)

A system-level analysis of the migraine phenomenon contributes to revealing its complexity. The homeostasis of a living organism is characterized by the maintenance of functional variables within pre-established physiological limits, a balance maintained through adaptive responses that prevent future disturbances (namely allostasis). (4) The human brain integrates multiple external and internal inputs and produces corrective responses proportionate to the disturbance to be corrected. In this framework, the pressure of physiological or paraphysiological stressors can generate excessive "allostatic load", resulting in the brain expending excessive energy, (5) which ultimately requires an "allostatic reset", generating a migraine attack. (4)

In this context, the concept of migraine trigger could be interpreted differently. A trigger could be defined as "a stimulus that lowers the threshold for a migraine attack in those predisposed to migraine". (6) Indeed, some clinical and neurophysiological studies provide data supporting the threshold hypothesis. (2) Among the most reported triggers are: stress, dehydration, skipping meals/fasting, sleep hours changes, lights/bright sunshine, exercise/physical activity, traveling, sexual activity, odors/certain smells or perfume, intense emotion, much reading, oversleep, hunger, coffee, and dairy products. (2)

Therefore, among the various potential triggers of migraine, nutrition plays a particularly significant role. This includes not only the intake of specific foods (such as chocolate, coffee, cheese, wine, citrus, nuts, and gluten), (7) but also fasting, dehydration, and some specific dietary patterns. (8,9)

This narrative review aims to synthesize current knowledge on this distinctive aspect of migraine management, exploring how fasting, food intake, specific foods and micronutrients, and overall dietary habits could provoke attacks and potentially influence the course of the disease.

Methods

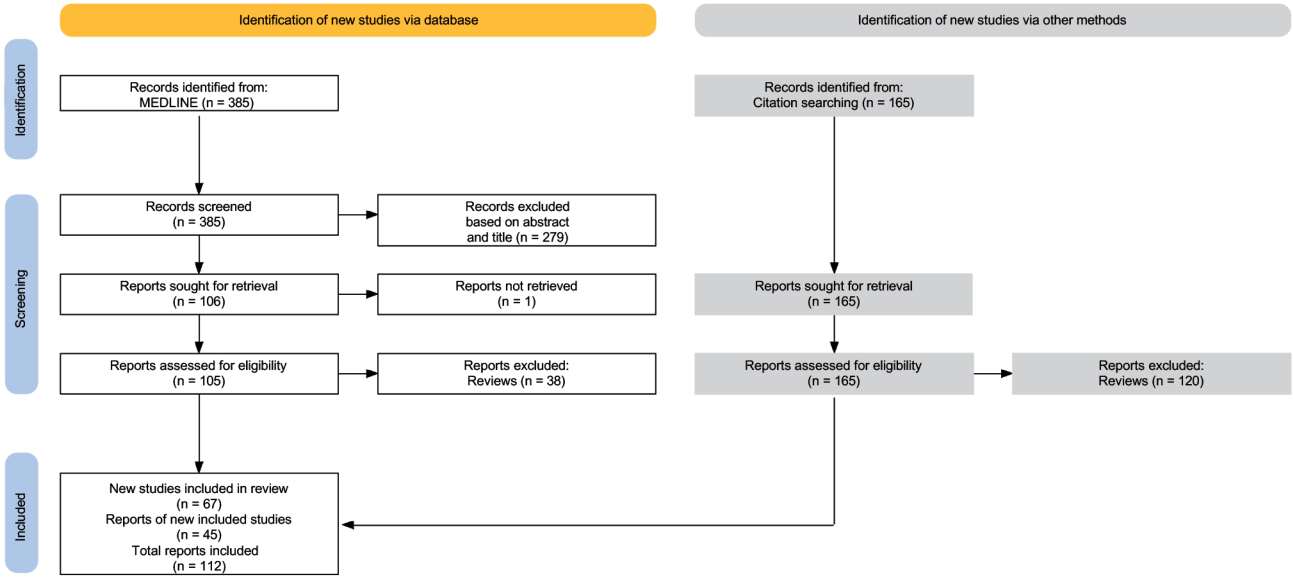
We searched the MEDLINE database for all articles that mentioned food triggers for migraine until July 2025 using the following search string: [(migraine OR headache) AND (food OR diet OR chocolate OR coffee OR cheese OR wine OR citrus OR nuts OR gluten OR fasting OR dehydration) AND (trigger OR triggers)]. We included reviews, randomized controlled trials, observational studies, and case series. Reviews were used to contextualize mechanisms and background evidence, but they were not counted among the original studies included in the flowchart. The results were subsequently screened. Specific migraine triggers were identified from each paper, distinguishing reviews from original articles. Each identified food trigger was then analyzed individually. Subsequently, we added other articles known to us that did not appear in the search string. This occurred for those triggers for which only a few studies were found. The flowchart of the studies included in the present review is summarized in **Figure 1**. The papers discussed in each paragraph are summarized in **Supplementary Tables 1-13**.

Migraine triggers

Fasting. The relationship between fasting and migraine has been extensively documented in the literature, with fasting consistently identified as one of the most common reported headache triggers across multiple populations and study designs. (10) The prevalence varies considerably across studies, with reports ranging from 12% to 60% of migraine patients identifying missed meals or fasting as a precipitating factor. (11) For instance, a large retrospective analysis of 1,207 migraine patients showed that the 57.3% reported "not eating" as a trigger, ranking it as the third most common precipitant after stress and hormonal fluctuations in women. (12) In a Brazilian cohort of 200 migraine patients, fasting was the most frequent dietary precipitant, followed by alcohol and chocolate. (13) Alongside, cohort cross-over studies during religious fasts, such as Ramadan or Yom Kippur, reported a heightened migraine occurrence. (14-17) Paradoxically, while objective data from food diaries of 1,883 patients showed that 67% of 2,313 spontaneous attacks occurred after fasting, only 2-3% of these patients actually perceived fasting as a trigger, suggesting a significant misrecognition of this potential precipitating factor. (18) The main studies on fasting and migraine are summarized in **Supplementary Table 1**.

The precise mechanisms of this phenomenon are currently unclear. From a mechanistic perspective, two complementary models have gained prominence in explaining the fasting-migraine relationship. The glucose-sympathetic model proposes that relative hypoglycemia and rapid glycemic fluctuations activate counter-regulatory catecholamines and alter substrate utilization, potentially lowering the migraine threshold in susceptible patients. This framework helps to explain the observed effectiveness of well-balanced eating patterns with low glycemic variability in migraine prevention. (10,11) In parallel, the neuro-energetic model suggests that prolonged fasting depletes readily mobilizable astrocytic glycogen stores at perisynaptic endfeet, leading to transient impairment in both glutamate and potassium clearance during periods of increased synaptic activity. This impairment may precipitate network depolarization and subsequent activation of trigeminovascular nociceptors, potentially manifesting as migraine attack. (19) These theoretical frameworks align with evidence that sympatho-metabolic counter-regulation, ketone availability, and substrate switching patterns significantly modulate individual risk. Indeed, while fasting generally increases migraine susceptibility, ketogenic dietary regimens that mimic starvation may reduce attack severity in selected patient populations, (20) highlighting how the specific timing, duration, and metabolic context of caloric restriction fundamentally shape clinical outcomes.

Given the high prevalence of fasting as a trigger and its mod-



Adapted from Haddaway NR, Page MJ, Pritchard CC, McGuinness LA. *PRISMA2020*: An R package and Shiny app for producing PRISMA 2020-compliant flow diagrams, with interactivity for optimised digital transparency and Open Synthesis. Campbell Syst Rev 2022;18:e1230.

Figure 1. Flowchart of the studies regarding nutritional triggers in migraine.

ifiable nature, maintaining regular meal schedules and avoiding prolonged periods without food may represent simple yet potentially effective preventive strategies, though the optimal timing and composition of meals to prevent fasting-induced headache requires further investigation.

Dehydration. Dehydration emerges as a condition that can meaningfully influence the susceptibility to migraine attacks, not as a singular or universally determinative trigger but as a physiologic stressor that could lower the threshold at which attacks occur. Some studies showed that even mild hypohydration alters central pain processing. (21,22) Experimental studies using the cold-pressor paradigm demonstrate that dehydration increases pain-evoked activation in central nervous system pain networks and reduces pain thresholds, suggesting that the brain becomes more reactive to nociceptive input when intravascular volume is reduced. (21) Although these data originate from healthy volunteers, the authors note that a lower pain threshold in dehydration may plausibly make the trigeminovascular system more vulnerable to initiating a migraine attack.

Randomized clinical trials in which daily water intake was increased by approximately 1.5 L showed a reduction in headache-hours, number of attacks, and acute medication use, although statistical significance was inconsistent or not statistically significant, and quality-of-life benefits were modest. (23,24) More compelling is a cross-sectional study of 256 women with migraine showing that patients who consumed more water (approximately 2 L/day) experienced lower severity, shorter duration, and decreased frequency of attacks, along with lower disability. (25) These findings support the narrative that hydration influences disease burden even though they do not establish a causal relationship. Fasting studies (see the dedicated paragraph) likewise reinforce this association, with migraine frequency increasing during periods of fluid restriction such as Ramadan, although fluid intake is rarely quantified.

Dehydration frequently acts as an amplifier of ongoing migraine attacks. Nausea and emesis, present in 90% and 70% of migraine attacks, respectively, limit oral intake and can rapidly worsen hypovolemia, thereby intensifying the attack. (26) Despite this, emergency department studies show that intravenous fluids do not improve pain outcomes when added to standard antiemetic therapy, indicating that hydration alone cannot abort established migraine pain. (27,28)

Therefore, the evidence showed dehydration as a state that destabilizes cerebrovascular and autonomic balance, heightens central pain sensitivity, and interacts with established triggers. (29) It does not act as a solitary cause of migraine but instead shifts the physiological milieu toward one in which attacks are more easily initiated or prolonged. Maintaining adequate daily hydration, ≥2 L/day of water for most patients, is a simple, low-risk strategy that may reduce attack frequency and burden, although controlled trials with objective hydration markers are still lacking.

Milk and dairy products. Milk and dairy products, particularly cheese, are among the most frequently reported dietary triggers, though evidence is heterogeneous. These products have been recognized as common migraine triggers in various epidemiological surveys. (30-32) In a Brazilian study, 27.7% of patients with migraine reported cheese as a trigger, (33) as well as in a Saudi Arabian cohort, with 18% of patients. (34) A 2020 review of 43 studies showed that milk and cheese were among the most commonly reported diet-related triggers, though the data were based on cross-sectional studies and patient surveys, with recall bias that limits the quality of evidence. (32)

However, studies pointed out that migraine patients may have an avoidant behavior and consume lower amounts of cheese, possibly reflecting self-imposed avoidance, therefore influencing prevalence. (35) The included studies on milk and dairy products are summarized in **Supplementary Table 2**.

The link between milk, dairy, and migraine may be explained by different pathways. Vasoactive amines such as tyramine and histamine are abundant in aged cheeses and can influence vascular tone and neuronal excitability. (32) Immune-mediated intolerance can play a role, where IgG/IgE responses to milk proteins may act as triggers. (30) It has been hypothesized that dairy proteins and metabolites can promote microbiota-driven inflammation. (36) Moreover, cheese can act synergistically with fasting, stress, or hormonal changes to lower the threshold for attacks. (37) Interestingly, a recent Mendelian randomization analysis reported a protective association of genetically predicted cheese intake with migraine risk (odds ratio [OR]≈0.78; 95% confidence interval [CI]: 0.63-0.95), suggesting that habitual consumption could be beneficial in some patients. (38) Therefore, the evidence is heterogeneous, and the role of milk and dairy products as a trigger for migraine attacks is still unclear.

Chocolate. Chocolate is among the most frequently reported dietary triggers of migraine, although its role remains debated. Cross-sectional surveys conducted in different populations indicate that between 7% and 27% of patients identify chocolate as a precipitant. (31,37,39-41). Other studies have investigated dietary behaviors in patients with migraine, showing that chocolate is often reported as an avoided food, though this may reflect patient beliefs rather than consistent causality. (42) More recently, a prospective lifestyle-oriented study even suggested that chocolate, along with other nutrients such as caffeine, could act as a neutral or protective factor, challenging the traditional view. (43) Furthermore, randomized double-blind provocative studies reported conflicting results. Two controlled cross-over trials using chocolate *vs.* placebo failed to demonstrate a consistent increase in the incidence of headache attacks. (44,45) Conversely, experimental evidence is limited, with one early trial reporting chocolate as a reproducible migraine-provoking agent in susceptible patients. (46) Overall, these findings highlight the discrepancy between self-reported triggers and controlled provocation outcomes. The available studies on chocolate as a migraine trigger are summarized in **Supplementary Table 3**.

Chocolate contains various polyamines whose bioavailability increases significantly after digestion (tyramine, tryptamine), while histamine appears *ex novo*. (47) Many phytochemical components derived from cocoa have potential anti-inflammatory, antioxidant, and analgesic effects. These compounds have neuromodulator activity on the main pain pathways. Particularly, methylxanthines act on the adenosinergic transmission, N-acylethanolamines on the endocannabinoid system (ECS), tryptophan on the serotonergic pathways, and alkaloids on the gamma-aminobutyric acid (GABA)ergic and opioid systems. (48) Chocolate also provides flavonoids capable of altering cerebral blood flow by means of nitric oxide (NO) production. (49) An alternative explanation, supported by prospective data, is that craving or consumption during the premonitory phase may be misinterpreted as a dietary trigger, rather than reflecting an early symptom of the migraine attack itself. (50-52)

From a clinical perspective, although chocolate is one of the most frequently reported triggers, controlled evidence does not confirm a reproducible effect, and in some cases even suggests a neutral or protective role. Individualized assessment with headache diaries remains the most reliable strategy to clarify its clinical relevance in each patient. (51,52)

Meat. The relationship between meat consumption, particularly processed meat products, and migraine pathophysiology represents a complex and evolving area of investigation. Compelling evidence from cross-sectional studies demonstrates that processed meat consumption patterns differ significantly among

migraine populations, with patients experiencing chronic migraine showing substantially higher intake of fried meat, fried chicken, and processed meat products such as luncheon meats and sausages compared to those with episodic migraine. (53) High consumption of processed meat was associated with three times more chronic migraine than episodic migraine, as well as an increase in the frequency of attacks. (53) Moreover, in a randomized, controlled elimination paradigm in adults who identified personal food triggers, the avoidance of specific food items, such as sujuk (a dry fermented sausage) and red meat, led to significant reductions in monthly attack frequency, duration, and intensity at two months, with benefits sustained only in the arm that continued restriction. (54) Sujuk is rich in tyramine, histamine, and nitrates, all of which could have the potential to facilitate a migraine attack. (32)

In addition, large-scale epidemiological data from the Women's Health Study revealed an inverse relationship, showing that patients with migraine with aura had significantly lower intake of processed meats and hot dogs compared to those without aura, suggesting potential avoidance behaviors in response to recognized triggers. (55) The apparent contradiction between higher consumption in chronic populations (suggesting a causal role) and lower consumption in migraine with aura populations (suggesting avoidance behavior) likely reflects different stages of trigger recognition and dietary adaptation. The main evidence regarding meat and processed meat products is summarized in **Supplementary Table 4**.

The mechanistic basis for processed meat as a migraine trigger centers on several key components inherent to these products, including nitrates and nitrites used as preservatives, which can be converted to NO in the body and promote vasodilation that may precipitate migraine attacks. (32) Additionally, processed meats contain elevated levels of tyramine, a biogenic amine derived from tyrosine metabolism that can trigger migraine through effects on neurotransmitter systems and vascular function, particularly in patients with altered tyramine metabolism pathways. (36)

Citrus. Citrus has been reported among potential dietary triggers of migraine, although findings are inconsistent. In a cohort of 360 Iraqi females, 79% consumed citrus regularly and 19.5% reported headache changes following intake, with logistic regression showing a significant association between citrus consumption and headaches lasting 4-72 hours, compared to not having an attack (OR 2.30; 95% CI: 1.25-4.23; p=0.008). (56) In pediatric populations, citrus fruits were identified as triggers in 10% of children with chronic primary headache, and elimination diets targeting the foods identified as potential triggers led to headache freedom in most patients. (57)

From a pathophysiological perspective, citrus fruits contain vitamin C and potassium, which can increase NO production and alter glucose metabolism, thereby lowering the threshold for migraine attacks. (58,59) Grapefruit is rich in naringenin, a flavonoid with hypoglycemic and hypolipidemic properties (60) that can interfere with insulin signaling (61) and CYP450 activity, (62) potentially affecting hormonal and metabolic pathways. (60) Other citrus components, such as nobiletin from the peel, inhibit prostaglandin synthesis and may modulate inflammatory mechanisms. (63)

Observational studies provide additional insights. In a prospective evaluation of dietary habits, migraine patients reported significantly lower citrus consumption compared to controls (p=0.019); 13 patients reported citrus intake the day before at least one attack. (42) Similarly, in a large cross-sectional study, citrus intake did not differ between the migraine group and the non-migraine groups, but patients with daily headache reported significantly lower citrus consumption, supporting either avoidance or increased sensitivity in this subgroup. (55) **Supplementary Table 5** summarizes the evidence from the included studies on citrus.

Tea. In a large Malaysian cohort, tea was identified as a migraine trigger by 4.7% of patients. (31) Elimination studies provide further evidence, with Özön *et al.* noting that tea was a commonly reported trigger (12% of patients). (54) The exclusion of this food from the diet led to a significant reduction in attack frequency, duration, and severity. These positive effects disappeared once dietary restriction was discontinued, suggesting that sustained avoidance is necessary to maintain clinical benefit. (54) In the elderly, similar results were observed with tea as a recurrent trigger. Removal from the diet resulted in reductions in attack frequency, duration, intensity of pain, and the need for acute pain relief. (64) The studies evaluating tea as a potential migraine trigger are summarized in **Supplementary Table 6**.

The action of tea as a migraine trigger could be based on the content of methylxanthines such as caffeine, theophylline, and theobromine. (65) Additionally, the content of caffeine, polyphenols, and tannins can cause changes in cerebral blood flow and can trigger gastrointestinal symptoms, (66) which in turn can contribute to migraine susceptibility. (67) Interindividual variability in caffeine metabolism (*e.g.*, CYP1A2 polymorphisms) may explain why tea represents a trigger only for a minority of patients. (30)

Onions. Onions are an uncommon dietary trigger for migraine. In a large cross-sectional Malaysian cohort, only 0.9% of migraine patients reported onions as a trigger of headache attacks, compared to 0.3% of those with tension-type headache. (31) In a Turkish randomized trial of 50 migraine patients, onions were included among the foods listed in the food sensitivity questionnaire, and elimination of individually reported triggers, including onion, was associated with a significant reduction in monthly attack frequency, duration, and severity after two months compared to baseline. (54) Similarly, in an elderly population, onions were listed in the standardized trigger questionnaire. Exclusion of the listed triggers led to significant improvements in attack frequency, pain duration, severity, and decreased analgesic and triptan use. (64) However, the number of patients identifying onions as a trigger was limited, comprising 3 patients in one study (54) and 6 patients in the other. (64) The available evidence on onions as a migraine trigger is summarized in **Supplementary Table 7**.

Several hypotheses have been proposed to explain how onions might trigger a migraine attack. (68) They are rich in sulphur-containing compounds, such as thiosulfinates and sulfides, and can release volatile organosulfur molecules. (69) Sulfur-containing phytochemicals (from alliaceous plants) activate transient receptor potential (TRP) channels, including TRPA1 and TRPV1, on nociceptors, mediating neurogenic inflammation. (70) The activation of TRPA1 by olfactory or food stimuli (including sulfides) induces the release of CGRP and substance P, explaining trigeminovascular activation and therefore migraine attacks. (6) Onions also contain histamine-releasing agents and have been associated with mast cell activation, which could increase susceptibility in histamine-sensitive patients with migraine. (30) In addition, onions can provoke gastrointestinal irritation and fluctuations in blood glucose due to their mild hypoglycemic effects, two mechanisms involved in migraine attacks. (8)

Tomatoes. Tomatoes are rarely identified as migraine triggers. Tai *et al.* reported that only 0.6% of patients identified tomatoes as a precipitant, with no cases reported among those with tension-type headache. (8) Moreover, in the study by Özön *et al.*, tomatoes were among the foods identified and then removed from the diet of 50 patients with migraine, resulting in an improvement in migraine features. (54) In a similar study con-

ducted in elderly patients, the results were similar. (64) The included studies on tomatoes and migraine are summarized in **Supplementary Table 8**.

From a pathophysiological point of view, the mechanisms potentially involved are various. Tomatoes contain biogenic amines, such as histamine and tyramine, (71) that have been associated with migraine attacks *via* vasodilation, mast cell activation, altered catecholamine release, and trigeminovascular activation. (53) Notably, these molecules also seem to lower the threshold for cortical hyperexcitability. (72) Another possible mechanism involved is the NO metabolism, with tomatoes that could induce an excess of NO, a well-known experimental trigger of migraine. (73) Tomatoes are also a natural source of glutamate, a neurotransmitter linked to cortical spreading depression and central sensitization, key processes in migraine with aura initiation and maintenance. Finally, tomatoes can provoke gastrointestinal intolerance in sensitive patients, and the resulting increase in intestinal permeability and release of pro-inflammatory mediators may contribute to migraine *via* the gut-brain axis. (30)

Ice cream. In a cohort of 684 patients who assessed 25 specific dietary items, ice cream was not among the most common triggers. (31) However, ice cream appears consistently on standardized food sensitivity questionnaires used in controlled elimination studies, such as in the studies by Özön *et al.* and Özön and Karadaş. (54,64) Interestingly, although the data can only be inferred from a few patients, ice cream avoidance was significantly more common among patients with migraine with aura compared to migraine without aura, raising the possibility that ice cream may exert a differential impact depending on migraine subtype. (8)

Ice cream is also a trigger for headache induced by cold stimulus ("ice cream headache") according to the International Classification of Headache Disorders, 3rd Edition (ICHD-3). (74) This type of headache may share important pathophysiological pathways with migraine, including activation of trigeminal afferents and meningeal vasodilation. (75) The cold-induced stimulation of the palate and pharynx during ice cream consumption can lead to transient alterations in cerebral blood flow and increased activity in the trigeminovascular system, lowering the threshold for a migraine attack. (30) In addition, ice cream contains dairy components, including biogenic amines such as tyramine and histamine, with the potential mechanism involved (30) that we described in the section dedicated to dairy. Third, the high sugar content of ice cream may contribute to inducing rapid glycemic fluctuations, which have been implicated in migraine, particularly when combined with reactive hypoglycemia. (8)

Wine and alcohol. Alcohol consumption frequently emerges as a potential trigger for headache, with about one-third of migraine patients reporting it. (76) Recent meta-analyses showed that 22% (95% CI: 17-29) of patients with primary headache, including migraine (with or without aura) and tension-type headache, occasionally attribute their attacks to alcohol intake. (77) Conversely, a 2022 multicenter study involving over 1,500 episodic migraine patients across English-speaking countries found no significant association between daily alcohol consumption and attack onset within 48 hours, challenging retrospective biases and emphasizing multifactorial triggers. (78) Nevertheless, migraine patients tend to consume less alcohol overall than controls, without increased vulnerability to alcohol use disorders, suggesting adaptive avoidance rather than a direct comorbidity. (79) Alcohol affects the central nervous system and body homeostasis through mechanisms such as dehydration, as ethanol causes NO-mediated vasodilation (80,81) and suppresses antidiuretic hormone secretion, leading to systemic dehydration, intracranial hypotension, and mechanical stimulation of meningeal nociceptors. (76) Consequently, almost anyone can develop a headache after alcohol consumption, emphasizing alcohol's broad potential to provoke headache even in those without primary headache diseases. Besides dehydration, ethanol and its metabolites, acetaldehyde and acetate, interact with neurovascular and inflammatory pathways. (82) In animal models, acetaldehyde, produced from ethanol metabolism, induces periorbital mechanical allodynia through activation of the CGRP receptor and the TRPA1 channel in Schwann cells. This process results in oxidative stress and neuronal sensitization, mechanisms relevant to migraine development. (83) Research suggests that people with genetic variants that reduce aldehyde dehydrogenase 2 (ALDH2) activity, resulting in higher levels of acetaldehyde, are more likely to experience alcohol flushing and hangovers, and may be more at risk for alcohol-induced migraine attacks. (84,85) Acetate accumulation further exacerbates headache by increasing adenosine levels, which can cause vasodilation and nociceptive hypersensitivity. (86) Furthermore, sulfites, biogenic amines, congeners, phenolic flavonoids, and serotonin-related compounds, all dissolved in alcoholic beverages, can act as migraine triggers in addition to alcohol itself. (76,87) These complex interactions can trigger neurogenic inflammation in sensitive neural circuits, heightening the risk of short-term headache in response to environmental and physiological triggers. In large-scale cohort studies analyzing web-based questionnaires from 2,197 migraine patients, 35.6% reported alcohol as a trigger, with rapid headache onset (less than 3 hours in one-third and less than 10 hours in nearly 90%), indicating that effects are typically acute rather than delayed. (87) There are multiple ways alcohol can cause a headache, with oxidative stress highlighting conditions like migraine. (88) The evidence on alcohol consumption and migraine occurrence is summarized in **Supplementary Table 9**.

Nonetheless, current evidence suggesting a direct relationship between alcohol intake and migraine initiation is limited, while hangover episodes are often reported in association with attack onset. (89) Probably, it seems to act as a threshold modulator rather than a definitive cause. Indeed, alcohol can increase the bioavailability of some solutes (particularly poorly soluble and hydrophilic drugs) through enhanced solubility and increased intestinal permeability, increasing their trigger effect. (90,91) These insights support personalized trigger tracking and moderation to reduce attacks, which may improve quality of life without requiring complete abstinence.

Coffee and caffeine. Coffee as a migraine trigger is one of the foods for which there are the most clinical studies; the pharmacology of caffeine is also clearer than that of other food components. The use or abuse of coffee is a common variable in the lifestyle habits of a patient with migraine. (8,92) Similar dietary behavior has also been investigated in the pediatric age group. (93-95)

Therefore, the evidence of coffee consumption as a trigger or a protective factor is heterogeneous. (96) Caffeine, the main active ingredient in coffee, is a plant-based methylxanthine class stimulant and acts as a non-selective competitive antagonist at adenosine A_1 and A_{2A} G-protein-coupled receptors. (97) The receptors sometimes have an opposing role: A_1 receptors are generally inhibitory, reduce neuronal excitability, and suppress neurotransmitter release with a final analgesic effect, whereas A_{2A} receptors are excitatory and promote vasodilation. (66,97) In migraine, A_{2A} activation can contribute to the dilation of cerebral vessels, enhancement of CGRP release, and activation of the trigeminovascular system. (66)

The ambiguous role of coffee consumption in migraine pathology should be considered (98) because although many studies suggest its protective role, (38,41,43,99-100) other studies indicate it as a migraine trigger. (101) According to the litera-

ture, caffeine's analgesic effect is dose-dependent and related to a threshold. The intake of up to 130 mg of caffeine acts as an adjuvant in analgesia, while 60 mg or less has no effect; (102) however, more than 225 mg of caffeine does not significantly worsen the outcome. (103) Nevertheless, the pharmacological activity of caffeine is widely recognized as a specific migraine treatment. (66,96,98) Indeed, some authors describe caffeine withdrawal as a clear trigger for attacks, (7) *i.e.*, secondary headache disorders, and in ICHD-3, there is a specific section dedicated to caffeine-withdrawal headache (8.2.1). (74) Some authors suggest that headache following caffeine withdrawal is attributable to low doses of the substance (100 mg per day), while daily use of high doses is itself associated with headache (300 mg per day). (7,104)

Several studies investigated the relationship between coffee consumption and migraine in different populations. (103,105-107) In Korean women of reproductive age, the intake of <3 cups of coffee/day or ≥3 cups of coffee/day was associated with an elevated risk of experiencing migraine. (106) An additional population analysis from the same country found a linear association between coffee consumption and headache or migraine from 0-1 cup of coffee/day to ≥3 cups of coffee/day. (103) In American adults, a positive association between an increasing amount of caffeine and migraine was found, particularly 100 mg/day corresponds to a 5% risk for migraine in males and 7% in females, with a higher incidence with ≥400 mg/day of caffeine. (105) Another analysis from the same population (the National Health and Nutrition Examination Surveys of America) showed a non-linear association between caffeine consumption and migraine, with a dose-effect relationship and threshold effect at 97.5 mg/day. (107) Finally, in other cases, coffee consumption was not associated with an increased risk of migraine. (108) Clinical studies evaluating coffee as migraine trigger are summarized in **Supplementary Table 10**.

Miscellanea. ***Plant foods and fruits.*** Several plant-derived foods have been identified as migraine triggers. In a large Brazilian cohort of 3,935 migraine patients, 40% reported plant foods as triggers, with the most frequently implicated being watermelon (29.5%), followed by passion fruit, orange, pineapple, grape, banana, cucumber, acerola, papaya, mango, durian, and sugarcane. Attacks typically occurred within 90 minutes of ingestion. (109) These foods are rich in biogenic amines, polyphenols, and sugars, which may interact with vascular and metabolic pathways involved in migraine.

Apple, cherry, and apricot. Apple and cherry were less commonly reported in large surveys but were nonetheless identified by a subset of patients in population studies. (109) The suspected mechanisms include polyphenolic compounds, glycemic fluctuations, and fructose load that can modulate gastrointestinal and vascular responses. (30)

Cherries contain polyphenols and anthocyanins with potential anti-inflammatory effects. (110) However, in some patients, cherries have been described as triggers, possibly *via* rapid glucose fluctuations or histamine content in preserved forms. (109)

Apricot has been rarely reported as a trigger. (111) Similar to other stone fruits, apricots contain amines and fermentable sugars, which may provoke migraine attacks in some patients. (109)

Spicy foods. Spicy foods are sometimes reported as migraine triggers, with a low estimated prevalence of 2.8%. (31) Capsaicin, the active compound in chili peppers, activates TRPV1 receptors and trigeminal nociceptors, lowering the threshold for migraine attacks. (112,113)

Eggs and wheat. In elderly migraine patients, diet elimination studies have identified eggs and wheat among the most reported triggers. (64) Removal of these items resulted in significant reductions in attack frequency, pain intensity, and analgesic/triptan use. (54) These findings support the role of immune-mediated intolerance or delayed hypersensitivity mechanisms in a subset of patients.

Olive. Olives and olive-derived products may act as triggers due to tyramine content in fermented olives. However, data are limited, and most literature emphasizes the benefit of their anti-inflammatory action. (30)

Poultry. Certain types of poultry, especially processed or preserved, have been reported as migraine triggers. The mechanism is likely related to preservatives and biogenic amines in cured poultry products rather than fresh meat. (32)

Muesli. Muesli and cereal-based foods can be reported as triggers, potentially due to gluten content or sugar spikes. In some elimination diet trials, cereals were among the foods avoided with subsequent improvement. (64)

Garlic extract. Garlic and garlic-derived extracts are sometimes considered paradoxical: while possessing anti-inflammatory and vasodilatory properties, they may also increase NO bioavailability, potentially provoking attacks in sensitive patients. (114)

Oat. Oats and oat-based cereals have been occasionally reported in elimination diet studies. Their potential trigger role may involve gluten sensitivity or glycemic fluctuations. (64) Evidence is sparse and largely anecdotal.

Sesame. Sesame seeds and oil are rich in tyrosine derivatives and lignans, which in theory may modulate vascular tone. Although not frequently cited in large surveys, case-level reports suggest sesame could act as a trigger in certain patients. (30)

Oily fish. Omega-3 fatty acids from oily fish have been recognized as anti-inflammatory agents. However, some preparations, particularly preserved fish, are rich in histamine and biogenic amines, which can provoke migraine in some patients. (30)

Lime eggs (preserved eggs). Preserved eggs, such as lime eggs, have been noted as triggers in dietary surveys. These foods are rich in amines and additives, which may precipitate migraine in susceptible patients. (32)

Fried food. Fried foods are often associated with migraine in patient surveys. Mechanistic explanations include the ingestion of oxidized fats and lipid peroxidation products, which can enhance oxidative stress and neuronal excitability. (63)

Soft drinks. Soft drinks are consistently reported as triggers. In Saudi cohorts, patients noted associations with sugary and carbonated beverages, likely due to glycemic variability and additive content. (34)

Nuts. Nuts are frequently included in lists of migraine triggers. Their content of tyramine and phenylethylamine provides a plausible biochemical mechanism, although they also contain magnesium and polyunsaturated fats that may be protective in other patients. (30)

Processed foods. Processed foods represent one of the most consistent categories of migraine triggers across cultures. They often combine multiple risk factors, amines, nitrites, sodium, trans fats, and additives, and have been strongly associated with migraine onset in surveys. (30,32)

Sugar. Excessive sugar intake has been associated with migraine in observational cohorts. The main mechanism is based on glycemic oscillation: both hyperglycemia and rebound hypoglycemia can activate cortical hyperexcitability and trigger attacks. (63)

Sucralose. Artificial sweeteners are emerging as potential triggers of migraine attacks. Sucralose, one of the most used of this group, has been linked to migraine in some case reports. Moreover, complete resolution of attacks following sucralose withdrawal has been reported. (115) Proposed mechanisms include alterations in neuronal excitability and excitotoxic action. (116)

Monosodium glutamate. Monosodium glutamate (MSG) is a sodium salt of glutamic acid, commonly used as a flavor enhancer that provides umami taste. It occurs naturally in foods like Parmesan cheese, tomatoes, and mushrooms, and is widely added to processed foods and restaurant meals. (117) The relationship between MSG consumption and migraine attacks has been investigated through some clinical trials, but findings remain highly inconsistent across different study designs. (118)

The association between MSG and headache was first popularized following the description of "Chinese Restaurant Syndrome" in 1968, which linked consumption of Chinese food to various symptoms, including headache. (119) However, scientific inquiry into this connection has yielded mixed results. Several double-blind, placebo-controlled trials have failed to establish a reproducible link between MSG consumption and headaches, particularly when comparing typical dietary consumption with experimental doses, often 5-8 times higher. (120) For instance, one systematic review concluded that when MSG was administered with food, there was generally no significant difference in headache incidence compared to placebo. (121,122) In contrast, studies involving administration of MSG in solution, on an empty stomach, and at high concentrations did report a higher incidence of headaches compared to placebo. (123,124) This suggests that the method of consumption and the presence of other food, which can slow absorption, may be critical factors. However, many of these studies have been criticized for methodological flaws, such as using doses far exceeding average dietary intake and the difficulty of effective blinding due to MSG's distinctive taste at high concentrations. (125) Clinical studies evaluating monosodium glutamate and migraine occurrence are summarized in **Supplementary Table 11**.

Overall, current evidence regarding MSG as a migraine trigger presents a nuanced picture that defies simple categorical statements. While MSG may potentially precipitate headaches in a specific subset of susceptible patients, the inconsistent findings across studies, lack of reproducible dose-response relationships, and absence of effects when MSG is consumed with food as typically occurs in normal dietary contexts indicate that its role in headache pathophysiology remains uncertain and requires accurate dietary assessment to evaluate any potential clinical impact.

Aspartame. Aspartame is an artificial sweetener widely used as a sugar substitute in processed foods and beverages. The first reports of aspartame-induced migraine appeared in the mid-1980s, including a case of headache triggered by soft drinks and reproducible attacks with aspartame solutions. (126) In a headache clinic survey, 8.2% of patients identified aspartame as a precipitant, and among patients with migraine, the prevalence rose to 10.6%. (127) Similar prevalence was reported in cross-sectional cohorts, with 13% of children with chronic headache reporting symptom changes after aspartame exposure, (57) 8.8% of Iraqi females. (56) Randomized double-blind trials, however, produced conflicting results. One study observed no difference in headache incidence between aspartame and placebo in 40 patients with reported sensitivity, (128) whereas another found a significant increase in migraine frequency in 11 patients during aspartame administration. (129) In a crossover study of 32 adults, a higher proportion of headache days was reported with aspartame than with a placebo. (130) Conversely, a 24-week trial with high-dose intake (up to 75 mg/kg/day) in 108 healthy adults documented no increase in headache frequency. (131)

Several mechanisms may explain how aspartame triggers migraine in susceptible patients. Aspartame is metabolized to phenylalanine, aspartic acid, and methanol; the latter is further converted to formaldehyde and formate, which inhibit mitochondrial complex III, generate oxidative stress, and deplete glutathione. (88) Phenylalanine and aspartic acid may enhance excitatory neurotransmission through N-methyl-D-aspartate receptors, lowering the threshold for cortical spreading depression and trigeminovascular activation. Additional evidence points to microglial activation, NO release, and neuroinflammation as potential contributors. (88) The evidence on aspartame exposure and migraine is reported in **Supplementary Table 12**.

From a therapeutic perspective, although population-level evidence remains inconsistent, aspartame appears to act as a reproducible trigger in a minority of migraine patients. Elimination strategies or dietary diaries may help to identify sensitive patients and guide individualized dietary recommendations. (7,50)

Nitrites and nitrates. Compounds containing nitrite and nitrate are preservatives added to food, especially in processed meats, to prevent botulism, to provide coloring, and to add a smoked flavor. The first report of nitrite-induced migraine attacks dates to 1972. (132) These molecules are established dietary and pharmacological triggers of migraine. Their effects are mediated through the production of NO, which produces vasodilation and modulates trigeminovascular activation. Nitrate can induce migraine attacks immediately after ingestion, due to direct NO-mediated vasodilation, or in a delayed manner, involving mediators such as CGRP and glutamate. (133)

Nitrites are among the most cited elements in food triggers in women. (40) In large cohorts, cured meats and nitrate preservatives emerged as principal components shaping the migraine phenotype. (134) In recent years, the oral microbiome has emerged as a potential contributing factor. Gonzalez *et al.* demonstrated that patients with migraine show higher levels of nitrate, nitrite, and NO-reducing bacteria in samples collected from the oral cavity, potentially amplifying endogenous NO production and promoting migraine attacks. (133) Moreover, nitrates may contribute to migraine susceptibility through an increase in oxidative stress. (88) Studies addressing nitrites and nitrates as potential dietary triggers are summarized in **Supplementary Table 13**.

In addition, nitrates have been used as a prototypical trigger in experimental migraine models. Pharmacological nitrates (*e.g.*, nitroglycerin) induce headache in most exposed patients. Some authors suggested that dietary nitrites/nitrates belong to the same category of reproducible triggers. (17) From a therapeutic perspective, in the literature, several authors emphasized that elimination of nitrite-rich foods may benefit selected patients, though evidence remains inconsistent, and individualized approaches are needed. (30,32)

Histamine. The role of histamine and histamine-containing foods as migraine triggers represents a complex interplay of biochemical, genetic, and physiological factors that continues to evolve in our understanding of migraine pathophysiology. (135) Histamine, a biogenic amine derived from the amino acid histidine, is naturally present in many foods, including aged cheeses, fermented products, processed meats, and alcoholic beverages,

with concentrations varying widely, from less than 0.5 mg/kg in fruits to over 500 mg/kg in aged cheeses and non-fresh fish. (136) The hypothesis of its role in the genesis of migraine attacks has been tested several times. (136,137) The supposed mechanism involves the activation of the enzyme NO synthase, the release of NO, and the successive meningeal vasodilation. (137) Indeed, experimental histamine infusion can precipitate migraine attacks that are attenuated by H_1 blockade. (136) Additionally, mast cell degranulation appears to activate meningeal afferents, creating a mechanistic constellation consistent with histamine acting as a trigger in at least a subset of patients. (138) However, attempts to prevent migraine with H_1 or H_2 receptor antagonists have shown limited success and undesirable adverse side effects. (135)

The clinical significance of dietary histamine as a migraine trigger appears most pronounced in patients with histamine intolerance syndrome, a condition characterized by an imbalance between histamine intake and degradation capacity. This syndrome is primarily attributed to a deficiency in diamine oxidase (DAO), the principal enzyme responsible for histamine metabolism in the gastrointestinal tract. (139,140) Notably, DAO deficiency, which may result from genetic polymorphisms in the DAO gene, has been found to occur in a substantial subset of migraine patients and is associated with an increased prevalence and disability of migraine headache. (141,142) However, evidence regarding DAO supplementation in migraine patients with enzyme deficiency remains mixed. While small randomized controlled trials have demonstrated a reduction in headache burden with DAO supplementation in DAO-deficient patients, (143) conflicting reports of paradoxically increased DAO activity in other migraine cohorts have been documented, (144) suggesting heterogeneity in pathogenic mechanisms underlying histamine-related migraine.

A compelling mechanistic bridge between histamine and migraine involves the bidirectional interplay with CGRP. Histamine can drive CGRP release from meningeal afferents, while CGRP reciprocally promotes mast cell activation and histamine release, establishing a feed-forward loop that sensitizes trigeminovascular circuits. (145) This complex interaction extends beyond the central nervous system, encompassing the enteric nervous system, where exogenous dietary histamine may signal *via* gut peptidergic afferents, thereby increasing the probability of migraine initiation.

Overall, available data indicate that low-histamine diets and elimination strategies confer benefits primarily in selected phenotypes rather than across the general migraine population, underscoring the need for personalized identification of patients with true histamine intolerance through targeted biochemical assays and dietary testing.

Discussion

This review synthesizes research on supposed food migraine triggers, providing a framework to understand the mechanistic and clinical complexity of food-related factors. We started from the hypothesis that migraine attacks often follow exposure to various food stimuli, with significant individual differences in triggers and attack thresholds. Still, our results do not provide conclusive evidence in support of this hypothesis.

The complexity of framing the issue. ***Literature.*** The literature review revealed fragmented, sometimes conflicting results. Most studies depend on retrospective self-report or cross-sectional surveys, which significantly limit causal conclusions due to recall bias, expectation effects, and subjective identification of triggers. Randomized, double-masked, challenge studies, although complicated to carry out, often yield results that differ from patients' perceptions, especially for foods such as wheat, citrus, coffee, chocolate, and dairy products. (54) To date, relatively few studies have rigorously investigated the precise mechanisms by which these alleged dietary triggers exert their effects. This discrepancy is striking, especially given the substantial volume of recent reviews addressing the topic, which commonly conclude that the currently available data remains inconclusive and often conflicting. (8,32) This lack of definitive evidence underscores the complexity of diet-migraine interactions, highlighting the need for well-designed research to better elucidate the causal pathways and practical implications for management. The lack of consistent findings across these trials highlights the need for better experimental protocols and greater attention to prodromal phenomena that may confuse trigger identification.

Patients' variability. Clinical practice suggests variability with and within patients, as not everyone responds to the same trigger, and the same person does not always respond consistently when exposed to the same stimulus. (44) This extreme variability could reflect differences in genetic background (for instance, it is well known that genetic variations can lead to different responses to caffeine, [146,147] or histamine [141]). More recently, considerable attention has been conferred to the gut microbiome (GM) and its role in migraine pathogenesis and chronicity, with a particular interest in its relationship with food triggers. (148) GM is a key player in the so-called gut-brain axis (GBA), which encompasses neural, immune, and metabolic connections between the gut and the brain. On the one hand, gut dysbiosis (a qualitative or quantitative alteration of the GM) could increase the negative consequences of certain migraine trigger foods. (149) On the other hand, correlation analyses have shown that proinflammatory bacteria, such as Desulfovibrio and Gemmiger, are positively associated with the consumption of trigger foods, such as cheese, chocolate, and peanuts; (148) while a dietary treatment of histamine intolerance reduces the abundance of some histamine-secreting bacteria, such as the genera *Proteus* and *Raoultella* and the species *Proteus mirabilis*, and increases *Roseburia* spp., a bacterial group frequently related to gut health. (150) Gastrointestinal migraine comorbidities, which can lead to a proinflammatory state and a leaky gut, altering the absorption and activity of trigger foods, represent another critical feature of the GBA. (151) Interestingly, another feature that can lead to subjective responses to different trigger foods is the type of headache they suffer. In fact, in a cross-sectional, observational, and online survey, patients with medication overuse headache have had specific, mostly dopaminergic, trigger foods. In contrast, migraine-specific food triggers were mostly histaminergic and processed foods. (152)

Patients' bias. Overall, many patients with migraine consider dietary triggers as a fundamental driver in the onset and exacerbation of their headaches and identify numerous foods as potential precipitants of migraine attacks. (7) However, there is a significant overlap between patient-reported food triggers and premonitory food cravings, suggesting that what patients perceive as a dietary trigger may actually be an early symptom of the migraine attack itself, rather than a true precipitant. (153) In support of our view, neuroimaging studies have demonstrated hypothalamic and limbic system activation during this phase, which likely underlies these cravings and other homeostatic alterations. (154) Thus, we can suppose that patients frequently misinterpret food cravings during the prodromal phase of migraine as food triggers, leading to a misunderstanding of the causal relationship between foods and migraine attacks. (153) This misattribution can lead to unnecessary dietary restrictions and confusion regarding migraine management, (155) with negative consequences for patients, since a high diet variability is protective against migraine. (156)

Anthropological considerations. Another possible explanation for the patient's overestimation of the impact of food triggers in migraine calls into question anthropological and evolutionary aspects. In fact, throughout history, humans have had to address the risks of consuming dangerous foods, developing, as an adaptive response to potential hazards, a sort of instinctive "food anxiety" expressed through taboos, rituals, and selection practices that have served as defense strategies against such dangers for millennia, building the food identity of peoples and communities. (157-159) Then, the fear of consuming potentially harmful food is a deeply rooted phenomenon in human behavior, closely linked to our evolution and the preservation of the species. Assume people with migraine are more sensitive to food anxiety (or to potentially noxious foods), in that case, they might be at an advantage in scenarios where the risk of poisoning or intoxication is higher, as in the past. Hence, migraine-related trigger food sensitivity may have been a protective factor from an evolutionary perspective.

On the other hand, behind the potential food anxiety, this huge interest also reflects the complexity of the meaning we attribute to food and nutrition, which represents a multidimensional topic, encompassing not only the essential biochemical provision of macro- and micronutrients (that can sustain life or, conversely, pose harm depending on their nature and dose) but also profound symbolic, psychological, and sociocultural implications. (160,161) Biochemically, the quality and quantity of micro- (such as vitamins and minerals) and macronutrients (carbohydrates, fats, and amino acids from proteins) can, in general, promote health or contribute to disease. (161) Beyond biochemistry and physiology, food choices are influenced by individual values, cultural traditions, religious beliefs, and social contexts, shaping dietary patterns and health behaviors. Food also impacts identity, social cohesion, and emotional well-being, serving as expressions of group belonging and as catalysts for personal identity transformation. (162,163) Thus, the implications of eating are not limited to survival or physical health but encompass a complex interplay of symbolic meaning, biochemical effects, and psychological and sociocultural dynamics, necessitating a holistic approach to understanding the complex relationship between nutrition and its role in human life. In this complex scenario, it is difficult to evaluate the actual effect of a single food on an elusive disorder like migraine. For instance, citrus fruits are reported among the foods that most frequently can act as migraine triggers in a few international studies; however, their prominent effect did not emerge in the (to the best of our knowledge) only Italian survey on this issue, (164) although in that country there is a high *per capita* consumption of these fruits. (165) Since Italy is one of the world's leading producers of citrus fruits, and their consumption is widely promoted as a healthy choice, (166) it is possible that Italian participants may be less likely to perceive citrus fruits as harmful or report them as triggers, possibly because of cultural familiarity and positive health-related perceptions.

Trigger variability. However, these unclear results may also reflect the complexity of the topic, where subjective variability interacts with the variability of trigger foods. The nutritional composition of food varies widely due to environmental, agronomic, and genetic factors, including macronutrients, micronutrients, and phytochemicals. Cooking and storage methods also affect vitamin and mineral levels, resulting in variations in losses or increases in bioavailability, depending on the technique employed. These differences affect the bioavailability and overall nutritional value of foods, potentially leading to harmful health effects. (167-170) Another key factor worth noting, which may explain the extreme variability observed, is that we often consume food that has been preserved, cooked, seasoned, and combined with other foods. However, what could potentially be harmful are the bioactive molecules within the food, not necessarily the food itself, and the various steps they undergo from farm to table can alter their bioavailability. (171) For example, drinking coffee and taking caffeine are not the same, (172) particularly because methylxanthines are linked to a lower risk of migraine, while caffeic acid sulfate is associated with a higher risk. (99) The same applies to eating raw oxalate-rich vegetables *vs.* cooking them in plenty of water. (173) Conversely, these farm-to-table processes could also increase the activity of protective compounds in foods. (174) Therefore, the potentially harmful effects of food can be either reduced or amplified by the procedures it undergoes before consumption. The paradox is that the same food can have different effects depending on how it is prepared, as this can enhance some bioactive compounds while suppressing others. A final consideration regarding trigger variability, often underestimated in studies on this topic, is the dose of triggers that subjects assume. As the Swiss physician Paracelsus (1493-1541) stated in 1538: "What is there that is not poison? All things are poison, and nothing is without poison. Solely the dose determines that a thing is not a poison". (175) It means that a compound can be regarded as a neutral food component, a drug, or a poison depending on the dose at which it is consumed. At which dose can a bioactive exert an adverse effect on a specific subject? If we exclude studies on coffee (flawed by the confusion between caffeine and coffee effects), few other studies paid due attention to this crucial point.

Dietary triggers according to aura status. Evidence on whether dietary triggers differ between migraine with aura and migraine without aura remains limited and difficult to interpret, because studies on migraine-related food triggers are highly heterogeneous in design, trigger assessment, and case definition. Aura status is inconsistently reported, and many studies do not perform stratified comparative analyses. Among the few available data, a cross-sectional analysis from the Women's Health Study suggested that dietary patterns may vary according to aura status: women with migraine with aura were more likely to report low intake of several foods commonly considered potential migraine triggers, including chocolate, cheese, ice cream, hot dogs, and processed meats, compared with women with migraine without aura. (55) However, these findings should be interpreted cautiously, as low intake may reflect avoidance behavior due to perceived trigger effects or greater migraine-related disability, rather than a true aura-specific triggering effect of these foods. In contrast, another study found no significant differences in reported food triggers between migraine with aura and migraine without aura, except for strawberry, which differed between groups. (152) Overall, current evidence does not support firm conclusions regarding aura-specific dietary triggers; rather, it highlights the need for prospective studies with standardized trigger assessment, clear aura classification, and analyses designed to distinguish true trigger effects from anticipatory food avoidance.

The multifactorial activity of dietary triggers. A striking result of our literature review is that foods supposedly acting as migraine triggers are very heterogeneous, with varied proposed mechanisms of action. It is not surprising, since migraine pathophysiology is multifactorial and can be figured out as a multistep process, (176) in which triggers can afflict each point.

Epigenetic regulation. Epigenetic regulation (DNA methylation, histone modifications, and microRNAs) is crucial in every disease's pathophysiology, including migraine, (177) and several nutritional factors can influence migraine through epigenetic effects. (178) For instance, processed foods are deficient in

methyl-donor nutrients, which may lead to the dysregulation of genes such as *HDAC4* and *MARK3*, which are linked to migraine chronification and clinical response. (179) Moreover, saturated fats, refined sugars, and alcohol have also been shown to induce adverse epigenetic changes, including altered DNA methylation patterns in adipocytes, which may contribute to the development of metabolic syndrome and related disorders. (180) On the other hand, diet-induced epigenetic modifications can also play a role in combating several diseases; (181) for instance, multimodal epigenetic modifications can explain part of the beneficial effects induced by the ketogenic diet. (182)

Metabolic and neuroendocrine function. There is a close relationship between migraine and metabolism, and dietary interventions in patients with migraine induce metabolic and neuroendocrine changes that may modulate migraine pathophysiology. (183) For instance, it has been well known for many years that there is a relationship between obesity and migraine, (184) with different proposed mechanisms of action. Several mechanisms have been proposed to explain the direct effect of obesity on CGRP expression. (185) Besides, according to other authors, migraine can also be regarded as an early feature of metabolic syndrome (186) or part of the larger disorder underlain by the brain insulin resistance that has at the other end of the spectrum Alzheimer's disease. (187) Interestingly, Mendelian randomization analysis revealed that *AP4E1* and *HSD17B12* are implicated in both migraine and type 2 diabetes. (188) Insulin resistance, other than diabetes mellitus, obesity, Alzheimer's disease, and migraine, was also regarded as a pathogenetic factor in other pathological conditions, including infections, chronic pain syndromes, and chronic inflammatory and autoimmune diseases; moreover, it was also described as a physiological response to various other stimuli. The complex mechanisms of insulin resistance also involve evolutionary factors, leading to the hypothesis that it is a maladaptive genetic condition; (189) a similar hypothesis has also been formulated for migraine. (190) Therefore, high insulinogenic foods, such as refined sugars, high-fat and hypercaloric diets, can act as migraine dietary triggers acting at this stage. On the other hand, weight loss, whether achieved through dietary modification or caloric restriction, is associated with a reduction in headache frequency, likely due to improvements in hypothalamic function and decreased proinflammatory adipokine signaling. (191)

Inflammation and oxidative stress. Migraine can be regarded as a chronic, evolutive inflammatory condition (192) related to oxidative stress. (193) Different foods were proposed as proinflammatory and oxidative triggers. Processed and red meats, ultra-processed foods (such as packaged snacks, industrial baked goods, and fast food), foods rich in saturated and trans fats (including margarine, fried foods, and industrial pastries), added sugars, refined carbohydrates (such as sugary drinks, sweets, and white bread), and alcohol. (194) It is well known that some macronutrients, such as refined carbohydrates and trans fats, increase proinflammatory cytokines and oxidative stress, thereby boosting proinflammatory transcription factors like nuclear factor kappa-light-chain-enhancer of activated B cells, activator protein-1, and early growth response protein 1, which in turn raise reactive oxygen species, leading to higher levels of cytokines such as interleukin (IL)-18, IL-6, and tumor necrosis factor-α. (195)

Excessive salt intake is associated with increased inflammation, neuroinflammation, and oxidative stress in animal models. (196) In contrast, the Dietary Approaches to Stop Hypertension diet, which is low in sodium, is associated with improved migraine symptoms (197) and a reduction in circulating oxidative stress markers. (198) Additionally, refined vegetable oils high in omega-6 fatty acids (such as corn and sunflower oil) may promote proinflammatory pathways and oxidative stress when consumed in excess relative to omega-3 fatty acids. (199)

Neurotransmitters, neuropeptides, hormones, and brain excitability. Neurotransmitters play a crucial role in migraine pathophysiology, as in other neurological disorders. Foods can influence the availability, biosynthesis, and activity of most of them. (200) Serotonin is one of the key neurotransmitters involved in this disorder; its pathways are often dysfunctional, (201) and it plays a role in both the development and treatment of migraine. (202) From a therapeutic standpoint, emphasizing the complexity of proper serotonin modulation in migraine, it is interesting to note that while serotonergic agonists are effective as acute treatments, both agonists and antagonists work as preventive agents. (203) Tryptophan, an essential amino acid that can only be obtained through diet, is the precursor to serotonin, and other nutritional factors influence its absorption, bioavailability, and pathway toward serotonin production. (204) For example, unexpectedly, eating protein-rich meals leads to a decrease, rather than an increase, in brain tryptophan uptake and serotonin synthesis. This occurs because dietary protein provides a higher concentration of competing large neutral amino acids, which compete for transport across the blood-brain barrier *via* the L-type amino acid transporter 1. (204) Additionally, inflammation can affect the absorption and metabolism of tryptophan. As previously mentioned, several food factors can trigger inflammatory states that may suppress serotonin production. At the gut level, inflammatory conditions can alter absorption, while systemically, inflammation can shift tryptophan metabolism toward the kynurenine pathway rather than the serotonin pathway. (205) Finally, the GM may also influence the serotonergic pathway, both positively and negatively. (204) Diet can induce GM modifications that modulate the serotonin pathway. (206)

Other than serotonin, there is a neurobiological connection between migraine and dopaminergic dysfunction, as evidenced by the high psychiatric comorbidity (migraine with aura may be at higher risk of developing psychotic symptoms such as hallucinations and paranoia, and patients with chronic migraine had double the risk of developing psychotic symptoms compared to non-migraine patients), the dopamine hypersensitivity (pain sensitivity, sensory disturbances like photophobia, and nausea), neuroimaging studies (that reveal increased presynaptic dopamine activity), and the response to antidopaminergic drugs (prochlorperazine and metoclopramide alleviate acute migraine symptoms, while atypical antipsychotics have shown potential in reducing migraine frequency). (207) A high-fat diet and excessive sugar consumption may disrupt dopamine function, creating a proinflammatory environment that promotes insulin resistance in neurons, alters reward sensitivity, and contributes to overeating and hedonic feeding, thereby initiating a vicious circle that leads to chronic migraine. (208,209) Like serotonin, dopamine is also derived from an essential amino acid, phenylalanine, and the GM can alter the complex process that leads to the biosynthesis of this neurotransmitter. (210) Still on neurotransmitters, glutamate and GABA are regarded as the other two central players in the genesis of migraine attacks. An imbalance in the brain's main excitatory (glutamate) and inhibitory (GABA) neurotransmitters, characterized by hyperexcitability, contributes to the initiation and progression of migraine attacks. (211) In this case, too, diet also has a significant influence on both neurotransmitters. Glutamate is an amino acid and, as mentioned above, it is suspected to be a primary food trigger. Otherwise, it is a non-essential amino acid; we can produce it if needed, and it is widely present in several foods. Thus, it is difficult to believe that such an abundant compound has no precise regulatory mechanism capable of preventing glutamate-induced

neural toxicity. (125) Indeed, what was experimentally observed is that adhering to unhealthy dietary patterns may be associated with decreased frontal GABA and increased frontal glutamatergic neurotransmission/metabolism, as well as reduced right precentral gyrus grey matter volume, possibly through excitotoxicity due to excitatory/inhibitory imbalance. (212) Moreover, in rats, a high-fat diet has been shown to decrease GABA levels in the frontal cortex and hippocampus. (213) Therefore, these neurochemical and morphological alterations may contribute to the development of migraine. Moreover, it has been observed in animals that the GM can modulate the glutamate/GABA balance. (214) Based on what has been discussed so far, we can infer that shared unhealthy diets may impact various neurotransmitter pathways. Indeed, it has been observed that a high-fat, high-sugar diet alters multiple aspects of glutamate, dopamine, and opioid signaling, (215) supporting our hypothesis.

The ECS is another crucial link between diet and migraine. The ECS contributes to the modulation of pain and neuroinflammation, and its dysfunctions are linked to migraine pathophysiology. Chronic migraine and medication overuse headache patients, compared to healthy individuals, often exhibit lower levels of the endocannabinoid molecules in their plasma and cerebrospinal fluid. (216) A diet characterized by high consumption of palatable, typically energy-dense foods rich in sugar and fat can adversely influence the ECS, promoting conditions such as food addiction (mediated by craving related to reward research) and obesity, (217) both of which are associated with migraine worsening. (184,218)

Histamine is typically regarded as an inflammatory molecule; however, it also functions as a neurotransmitter produced by neurons originating from the tuberomammillary nucleus of the hypothalamus and projecting widely across the brain, including the cortex, hippocampus, and basal ganglia. It is a biogenic monoamine that influences arousal, cognition, and memory. (219) The central nervous system has four histamine receptors (H_1R-H_4R). H_3R acts as both an autoreceptor and a heteroreceptor, thereby negatively modulating the release of histamine and other neurotransmitters, which in turn influences neural plasticity. (219) In small clinical trials, low-dose histamine and the experimental H_3 agonist Nα-methylhistamine have demonstrated migraine prophylactic efficacy, suggesting a dose-dependent bimodal effect for this monoamine, probably mediated through specific H_3R agonism, the potential target for anti-nociception and anti-neurogenic inflammation that may also influence migraine; conversely, the H_3R antagonist pitolisant increases central histamine and causes headache. (135) On the other hand, other pharmacological studies have led us to consider the peripheral effects of histamine on migraine pathogenesis as well. In fact, monoclonal anti-CGRP antibodies inhibit peripheral histamine release, (220) and triptans inhibit histamine-induced dural vessel dilatation. (137) It is a fact that neurogenic inflammation triggers the immune system, inducing the CGRP-mediated release of histamine by dural mast cells, which participates in dural artery vasodilation; (183) this mechanism was also proposed to explain the higher prevalence of migraine in females and the pathogenesis of menstrual migraine, since estrogen induces an increase of dural mast cells and their phenotypic makeup. (221) Moreover, it has been proposed that exogenous food histamine may promote migraine attacks by increasing CGRP levels, (222) prefiguring a sort of vicious circle between CGRP and histamine. The close link between histamine, its precursor histidine (another amino acid), and nutrition was already discussed in the specific paragraph. It is noteworthy that, aside from histamine, no other foods or micronutrients have shown a CGRP-releasing effect, not even cocoa beans, despite their inducing chronic upregulation of NO production. (223)

The hypothalamus is a key brain structure involved in the link between migraine and neurotransmission processes. Several hypothalamic molecules play a role in migraine pathophysiology, including orexins, neuropeptide Y, pituitary adenylate cyclase-activating polypeptide, dopamine, prolactin, melatonin, oxytocin, and vasopressin. (224) It is interesting to note that melatonin is a hormone derived from serotonin, which in turn is derived from the amino acid tryptophan; (204) thus, the same arguments about diet influence apply here as above. Most of these neurotransmitters and peptides are involved in the trigeminovascular-thalamic and thalamo-cortical pathways, crucial players in the genesis of migraine attacks and of any physiological (sleep, wakefulness, food intake, body temperature, heart rate, blood pressure), behavioral (addiction, isolation), cognitive (attention, learning, memory use), and affective (stress, anxiety, depression, anger) non painful migraine-related feature. (225) Interestingly, some hypothalamic molecules implicated in migraine mechanisms are also involved in appetite control, highlighting the shared mechanism between migraine and food intake dyscontrol, through hypothalamic dysfunction. If the hypothalamus can alter appetite, food intake can heavily modify the hypothalamus's functioning and structure, according to the diet composition. (226) In general, unhealthy high-fat diets can induce hypothalamic inflammation, which contributes to the activation of systemic inflammation related to metabolic syndrome, leading to insulin resistance, leading to obesity, cardiovascular, and neurological disorders. (227) The hypothalamus is also the key neurological structure of the so-called GBA, and it is affected by diet. In fact, gut hormones that modulate digestive processes and nutrient absorption peripherally also play a role as neurotransmitters into the brain, controlling food intake, suppressing (peptide YY, pancreatic polypeptide, glucagon-like peptide-1, and oxyntomodulin) or increasing (ghrelin) appetite, at the hypothalamic level. (228) An indirect influence of diet on the hypothalamus by the GBA is related to its impact on the GM that can influence hypothalamic and its circadian rhythmic activities. (229)

Clinical considerations. What we eat is not directly addressed to the brain as it is; in general, it undergoes digestion and absorption, possibly interacting with our microbiome and other nutrients, as well as genetically regulated enzymes, neurotransmitter systems, and the GBA, in a dynamic, dose- and context-dependent manner, before it reaches the brain. Moreover, every meal is different from another apparently similar one, due to the variability in micro- and macronutrients, which depend on the various degrees of maturation, as well as different storage, seasoning, and cooking methods. (167-170) Again, for many triggers, such as histamine and gluten-containing foods, only a subset of patients should be truly sensitive, in relation to defined metabolic or genetic susceptibilities (*e.g.*, DAO deficiency, celiac disease, food allergies). Finally, a personalized approach should also consider drug-food interactions. It is, for instance, the case with caffeine, which is metabolized by CYP1A2, as well as some triptans, such as frovatriptan, rizatriptan, zolmitriptan, and naratriptan. (230) Therefore, the "one-size-fits-all" mechanistic vision sometimes proposed is too simplistic and incorrect for mechanisms linking dietary exposures to migraine attacks. Vasoactive amines (histamine, tyramine), rapidly absorbed sugars, processed food preservatives (nitrites, nitrates), biogenic amines, neuroactive compounds (caffeine, methylxanthines, glutamate), etc., are all questioned as potentially precipitating factors for migraine attacks, according to patients' reports and some literature. However, as we have found in the results of this review, sometimes the most noxious component of the diet for people with migraine is not a specific micronutrient but the dietary pattern, with its peculiar distribution of macronutrients and calories, together with fasting and dehydration.

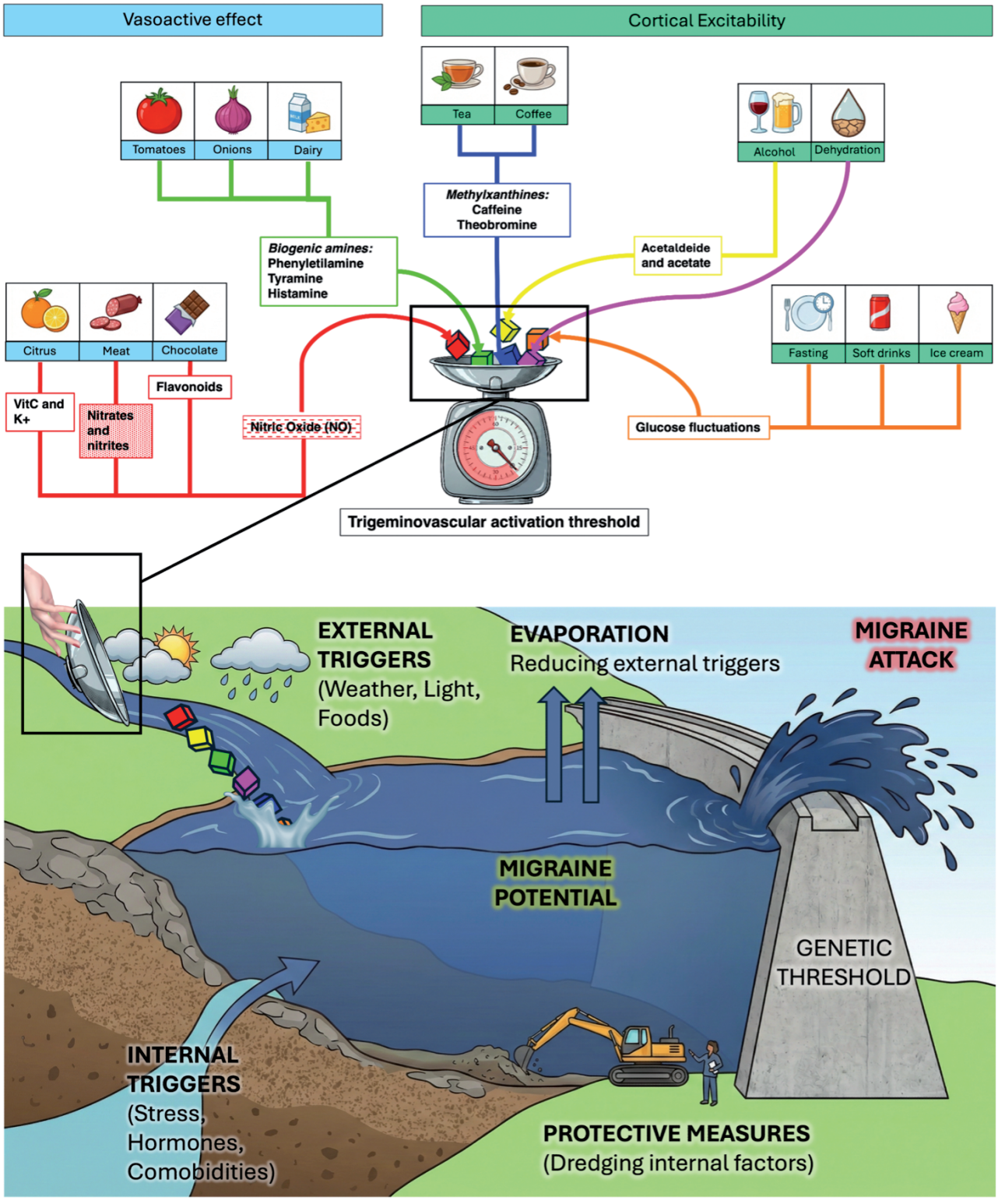


Figure 2. Proposed interaction between dietary triggers and migraine attack threshold. Dietary factors may affect migraine susceptibility through vasoactive mechanisms, changes in cortical excitability, nitric oxide-related pathways, biogenic amines, methylxanthines, alcohol metabolites, dehydration, fasting, and glucose fluctuations. In genetically predisposed individuals, these factors may act together with external and internal triggers to lower the trigeminovascular activation threshold and facilitate migraine attack generation, whereas protective measures may reduce overall trigger burden.

Conversely, the perspective should be reversed. Rather than searching for potential food triggers, we should focus on protective factors – essentially anti-triggers – such as micronutrients that can potentially prevent migraine. (223) Additionally, we should focus on healthy eating patterns that have been shown to benefit this type of headache. (9) Indeed, focusing too much on supposed triggers could reinforce patients' biases, partly because of the symbolic, psychological, and sociocultural roles food plays in humans.

Nonetheless, tempting as it is to speculate about the actual role of triggering foods in migraine, despite all the caution we assume, it could be hypothesized that a model in which a non-dichotomic, partial, and variable trigger effect could be attributed to each supposed noxious food is possible. Above, we have reported a trigger definition: "a stimulus that lowers the threshold for a migraine attack in those predisposed to migraine". (6) This means that what we call a trigger is not the mechanistic factor that directly causes the attack, but rather a predisposing feature that can influence to a various degree the likelihood of the attack occurring when combined with other triggers (food and non-food). Migraine attacks arise from the interaction between a genetically predisposed subject and a hostile environment, characterized by internal and external precipitating factors. (231) A similar scenario can also be hypothesized to explain the chronification process of a previously episodic migraine in an affected subject. Trigger identification should be viewed as a dynamic and analytic process, combining empirical observation with pathophysiologic knowledge and an appreciation for the fluctuating nature of attack initiation.

One model that can describe our view of the phenomenon is the analogy of an overflowing dam. This model features a genetically determined threshold – the maximum amount of water it can hold – along with factors that either raise this volume (external triggers such as foods, weather, light exposure) or lower it (internal triggers like comorbidities, hormonal state, stress, circadian and metabolic factors, and even gut microbiota composition). Protective measures include dredging the bottom (addressing comorbidities and reducing internal triggers) and allowing the water level to decrease through evaporation and soil infiltration (reducing external triggers). When the water exceeds this threshold due to a combination of these factors, a migraine attack occurs. The mission of each case manager in caring for patients with migraine should be to prevent overflow (**Figure 2**).

Limitations of the review. The use of the narrative review approach is a consequence of the observation that most clinical studies analyzing dietary triggers of migraine are heterogeneous, unsystematic, and of limited quality. The number of clinical trials is small, and the evidence that emerges is often inconsistent and contradictory, making it difficult to generalize. For this reason, this review also has methodological limitations. There is a lack of large, randomized studies that can systematically collect consistent data on migraine triggers.

Conclusions

The connection between foods and migraine is recognized as significant yet highly individualized and too complex to be trivialized in a simple reaction to one specific food. The paradigm has evolved from simple exclusions of certain foods to a comprehensive, multi-faceted model that considers genetic factors, metabolic conditions, environmental influences, and psychological aspects affecting both exposure and risk. Future research integrating omics technologies, digital tracking, and personalized nutrition strategies holds promise for developing targeted, individualized interventions to prevent migraine episodes in clinical settings. Thus, dietary advice must be tailored, empowering patients to identify individual triggers without imposing unnecessarily restrictive or stigmatizing recommendations.

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Online supplementary material:

Supplementary Table 1. *Summary of studies assessing fasting as a trigger for migraine attacks.*

Supplementary Table 2. *Summary of studies assessing milk and dairy products as a trigger for migraine attacks.*

Supplementary Table 3. *Summary of studies assessing chocolate as a trigger for migraine attacks.*

Supplementary Table 4. *Summary of studies assessing meat and processed food as a trigger for migraine attacks.*

Supplementary Table 5. *Summary of studies assessing citrus as a trigger for migraine attacks.*

Supplementary Table 6. *Summary of studies assessing tea as a trigger for migraine attacks.*

Supplementary Table 7. *Summary of studies assessing onion as a trigger for migraine attacks.*

Supplementary Table 8. *Summary of studies assessing tomatoes as a trigger for migraine attacks.*

Supplementary Table 9. *Summary of studies assessing wine and alcohol as a trigger for migraine attacks.*

Supplementary Table 10. *Summary of studies assessing coffee and caffeine as a trigger for migraine attacks.*

Supplementary Table 11. *Summary of studies assessing monosodium glutamate as a trigger for migraine attacks.*

Supplementary Table 12. *Summary of studies assessing aspartame as a trigger for migraine attacks.*

Supplementary Table 13. *Summary of studies assessing nitrites and nitrates as a trigger for migraine attacks.*

Correspondence: Daniele Pala, Department of Medical Science and Public Health, University of Cagliari, Strada Provinciale, 8, 09042 Monserrato (CA), Italy.
E-mail: danielepala14@gmail.com

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