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HEADACHE (RB HALKER, SECTION EDITOR)

Headaches: a Review of the Role of Dietary Factors

Zoya Zaeem[1] · **Lily Zhou**[1] · **Esma Dilli**[1]

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Abstract Dietary triggers are commonly reported by patients with a variety of headaches, particularly those with migraines. The presence of any specific dietary trigger in migraine patients varies from 10 to 64 % depending on study population and methodology. Some foods trigger headache within an hour while others develop within 12 h post ingestion. Alcohol (especially red wine and beer), chocolate, caffeine, dairy products such as aged cheese, food preservatives with nitrates and nitrites, monosodium glutamate (MSG), and artificial sweeteners such as aspartame have all been studied as migraine triggers in the past. This review focuses the evidence linking these compounds to headache and examines the prevalence of these triggers from prior population-based studies. Recent literature surrounding headache related to fasting and weight loss as well as elimination diets based on serum food antibody testing will also be summarized to help physicians recommend low-risk, non-pharmacological adjunctive therapies for patients with debilitating headaches.

Keywords Headaches · Migraines · Dietary triggers · Caffeine · Elimination diets · Biogenic amines

Introduction

Headache is a common presenting complaint to primary care providers, emergency room physicians, and neurologists alike with a lifetime prevalence of above 90 % [1]. Although less prevalent than tension-type headaches, migraines are associated with significantly higher morbidity. It has a prevalence of 15 % and is the seventh highest cause of disability in the world [2]. In the USA, it was associated with a healthcare spending exceeding $11 billion dollars in the year 2004 alone [3].

While there are many established pharmacological treatments for common primary headaches, both for headache prevention (prophylactic therapy) and headache termination (abortive therapy), these are all associated with a variety of side effects. Additionally, the overuse of abortive medications can increase headache frequency. Because of these reasons, it is important to explore dietary factors for headache prevention.

The role of dietary supplements (also known as nutraceuticals) has been reviewed recently and found to show promise in the treatment of migraines [4, 5••]. Currently, the American Academy of Neurology concludes that there is moderate (level B) evidence for the use of riboflavin, feverfew, and magnesium and some lower quality evidence (level C) for the use of coenzyme Q10 in migraine prevention [5••].

Through this review, we hope to consolidate the current literature regarding food, food additives, and dietary patterns that trigger headaches, with a focus on migraines, and the implications of this for clinical practice.

Dietary Triggers

The definition of what qualifies as a food trigger is variable between studies and often based on patient self-report. A popular definition proposed previously included foods or beverages that precede an attack by less than 48 h [6]. Estimates of the prevalence of specific dietary triggers among migraine patients vary widely between 10 and 64 % in recent studies (Table 1).

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✉ Esma Dilli
esma.dilli@vch.ca

[1] Division of Neurology, University of British Columbia, 8219-2775 Laurel Street, Vancouver, BC V5Z 1M9, Canada

Table 1 Dietary effects among migraine patients

	Paper	Prevalence (%)
Any specific dietary trigger	Rasmussen [7] (Denmark, $n = 119$, not specified)	10
	Rasmussen [8] (Denmark, $n = 96$, not specified)	10.4
	Kelman [9] (USA, $n = 1009$, with and without aura)	10.6
	Zivadinov [10] (Croatia, $n = 720$, with and without aura)	12.5
	Cologno [11] (Italy, $n = 77$, with aura)	15.6
	Andress-Rothrock [12] (USA, $n = 200$, not specified)	18
	Turner [13] (USA/Mexican Americans, $n = 132$, with and without aura)	21.2
	Kelman 2007 (USA, $n = 1750$, with and without aura)	26.9
	Robbins [14] (USA, $n = 494$, with and without aura)	30
	Deniz [15] (Turkey, $n = 185$, with and without aura)	32.9
	Constantinides [16] (Greece, $n = 51$, with and without aura)	33.3
	Karli [17] (Turkey = 56, with and without aura)	33.9
	Chabriat [18] (France, $n = 385$, not specified)	36
	Mollaoğlu [19] (Turkey, $n = 126$, with and without aura)	43.6
	Van den Bergh 1987 (Belgium, $n = 217$, not specified)	44.7
	Ierusalimschy [20] (Brazil, $n = 100$, without aura)	46
	Spierings [21] (USA, $n = 38$, not specified)	58
	Fukui 2008 (Brazil, $n = 200$, not specified)	64
Hunger/fasting	Takeshima 2004 (Japan, $n = 244$, with and without aura)	0.8
	Kelman 2006 (USA, $n = 1009$, with and without aura)	27.2
	Chakravarty [22] (India, $n = 200$, without aura, pediatric)	31.5
	Andress-Rothrock 2010 (USA, $n = 200$, not specified)	39
	Robbins 1994 (USA, $n = 494$, with and without aura)	40
	Deniz 2004 (Turkey, $n = 185$, with and without aura)	41.6
	Scharff 1995 (USA, $n = 172$, without aura)	44.9
	Yadav [23] (India, $n = 182$, without aura)	46.3
	Ierusalimschy 2002 (Brazil, $n = 100$, without aura)	48
	Bánk 2000 (Hungary, $n = 78$, with and without aura)	52
	Mollaoğlu 2013 (Turkey, $n = 126$, with and without aura)	53.9
	Turner 1995 (USA/Mexican Americans, $n = 132$, with and without aura)	54.5
	Kelman 2007 (USA, $n = 1750$, with and without aura)	57.3
	Fukui 2008 (Brazil, $n = 200$, not specified)	63.5
	Karli 2005 (Turkey = 56, with and without aura)	73.2
	Spierings 2001 (USA, $n = 38$, not specified)	82

The International Classification of Headache Disorders III (ICHD III-beta) also set out specific criteria for related secondary headaches induced by food and/or additives [24]. These definitions are more stringent and describe headaches related to foods or additives containing one or more specific substances, which may not be identified but is capable of causing headache in a sensitive patient. By ICHD III criteria, the headache must have developed within 12 h of ingestion, resolve within 72 h after ingestion, and have one of the following features: bilateral, mild to moderate, pulsatile, and/or aggravated by physical activity.

Chocolate

Chocolate is a processed food that originates from the cultivation of cacao plants in Mesoamerica and contains vasoactive compounds including biogenic amines, flavonoid phenols, and caffeine (to be discussed separately below) [25].

It is commonly found to be a trigger among survey data from migraine patients in Caucasian and Hispanics populations though frequencies vary widely, with 1.4 [26] to 22.5 % [27] of patients being affected (Table 2). Conversely, not a single patient among a Japanese survey including more

Table 2 Food triggers in migraine patients

Triggers	Paper	Prevalence (%)
Wine	Takeshima 2004 (Japan, $n = 244$, with and without aura)	1.4
Alcohol	Karli 2005 (Turkey = 56, with and without aura)	3.6
	Mollaoğlu 2013 (Turkey, $n = 126$, with and without aura)	3.9
	Ulrich [28] (Denmark, $n = 169$, with aura)	9
	Rasmussen 1992 (Denmark, $n = 96$, not specified)	19.8
	Kelman 2006 (USA, $n = 1009$, with and without aura)	20
	Rasmussen 1993 (Denmark, $n = 119$, not specified)	20.2
	Andress-Rothrock 2010 (USA, $n = 200$, not specified)	20.5
	Henry [29] (France, $n = 880$, with and without aura)	23
	Constantinides 2015 (Greece, $n = 51$, 49 responses, with and without aura)	26.5
	Ierusalimschy 2002 (Brazil, $n = 100$, without aura)	28
	Peatfield 1995 (UK, $n = 429$, not specified)	28[a]
	Peatfield [30] (UK, $n = 500$, with and without aura)	29
	Bánk 2000 (Hungary, $n = 78$, with and without aura)	30
	Fukui 2008 (Brazil, $n = 200$, not specified)	34[b]
	Scharff 1995 (USA, $n = 172$, without aura)	35.3
	Kelman 2007 (USA, $n = 1207$, with and without aura)	37.8
	Spierings 2001 (USA, $n = 38$, not specified)	42
	Van den Bergh 1987 (Belgium, $n = 217$, not specified)	51.6
Beer or spirits	Russell [31] (Denmark, $n = 333$, with and without aura)	9.3
Red wine or cheese	Russell 1996 (Denmark, $n = 333$, with and without aura)	26.1
Cheese	Yadav 2010 (India, $n = 182$, without aura)	0
	Bánk 2000 (Hungary, $n = 78$, with and without aura)	0
	Constantinides 2015 (Greece, $n = 51$, 49 responses, with and without aura)	4.1
	Fukui 2008 (Brazil, $n = 200$, not specified)	8.5
	Scharff 1995 (USA, $n = 172$, without aura)	9.1
	Peatfield 1995 (UK, $n = 429$, not specified)	16.5
	Peatfield 1984 (UK, $n = 500$, with and without aura)	18.2
Cheese and dairy	Van den Bergh 1987 (Belgium, $n = 217$, not specified)	18.5
Milk and cheese	Mollaoğlu 2013 (Turkey, $n = 126$, with and without aura)	10.3
Milk	Van den Bergh 1987 (Belgium, $n = 217$, not specified)	2.3
	Fukui 2008 (Brazil, $n = 200$, not specified)	2.5
Ice cream	Takeshima 2004 (Japan, $n = 244$, with and without aura)	1.2
	Fukui 2008 (Brazil, $n = 200$, not specified)	3.0
	Van den Bergh 1987 (Belgium, $n = 217$, not specified)	4.6
Chocolate	Yadav 2010 (India, $n = 182$, without aura)	0.0
	Takeshima 2004 (Japan, $n = 244$, with and without aura)	0.0
	Bánk 2000 (Hungary, $n = 78$, with and without aura)	1.4
	Chakravarty 2009 (India, $n = 200$, without aura, pediatric)	1.5[c]
	Russell 1996 (Denmark, $n = 333$, with and without aura)	6.9
	Constantinides 2015 (Greece, $n = 51$, 49 responses, with and without aura)	10.2
	Peatfield 1995 (UK, $n = 429$, not specified)	16.5
	Mollaoğlu 2013 (Turkey, $n = 126$, with and without aura)	18.3
	Peatfield 1984 (UK, $n = 500$, with and without aura)	19.2
	Fukui 2008 (Brazil, $n = 200$, not specified)	20.5
	Scharff 1995 (USA, $n = 172$, without aura)	22.1
	Van den Bergh 1987 (Belgium, $n = 217$, not specified)	22.5

Table 2 (continued)

Triggers	Paper	Prevalence (%)
Coffee	Yadav 2010 (India, $n = 182$, without aura)	0.0
	Mollaoğlu 2013 (Turkey, $n = 126$, with and without aura)	6.3
	Peatfield 1984 (UK, $n = 500$, with and without aura)	7
	Fukui 2008 (Brazil, $n = 200$, not specified)	14.5
Caffeinated drinks/ caffeine	Van den Bergh 1987 (Belgium, $n = 217$, not specified)	6.4
	Andress-Rothrock 2010 (USA, $n = 200$, not specified)	8
	Scharff 1995 (USA, $n = 172$, without aura)	10.6
Citrus	Yadav 2010 (India, $n = 182$, without aura)	0.0
	Fukui 2008 (Brazil, $n = 200$, not specified)	4
	Peatfield 1984 (UK, $n = 500$, with and without aura)	11.1
Aspartame	Fukui 2008 (Brazil, $n = 200$, not specified)	8.5
	Scharff 1995 (USA, $n = 172$, without aura)	9.4
MSG	Yadav 2010 (India, $n = 182$, without aura)	0.0
	Fukui 2008 (Brazil, $n = 200$, not specified)	2.5
	Scharff 1995 (USA, $n = 172$, without aura)	12.9

[a] 18.4 % to all alcoholic drinks, 11.8 % to red wine but not to white; 28 % for beer

[b] Red wine: 19.5 %[a]; white wine: 10.5 %

[c] "Bitter chocolate"

than 240 migraine patients identified it as a trigger [32] suggesting possible geographic variations in frequency.

Evidence from small double-blind studies among patients who identified chocolate as a consistent migraine trigger have yielded mixed results with two studies finding no difference between chocolate and placebo as a provocative factor for headaches [33, 34] and the other finding chocolate causing more headaches than placebo (5/12 vs. 0/8) in patients with self-reported chocolate-triggered migraines [25].

Caffeine

Caffeine is a plant-based methylxanthine class stimulant. It is estimated that 87 % of Americans consume caffeine on a daily basis, with a mean estimated average dietary intake of 193 mg/day per consumer [35]. It is even more popular in Scandinavian countries where the average daily exceeding 400 mg per person [36].

Caffeine acts as a competitive antagonist at adenosine A1 and A2A G-protein-coupled receptor subtypes [36]. Purines including adenosine are linked to cerebral vessel vasodilation. It is known that plasma adenosine increases during migraine attacks [37] and that migraine can be triggered by exogenous adenosine administration [38]. Additionally, central nervous system adenosine levels are elevated during extended periods of wakefulness and decrease with sleep, an effective migraine remedy for many patients [39]. Because of this reason, caffeine is a common additive to abortive medications for both tension- and migraine-type headaches with good evidence of improved efficacy [40].

Caffeine is also a well-studied therapy for the treatment of post-dural puncture headaches (PDPH). A 2015 Cochrane meta-analysis concluded that compare to placebo "caffeine shows a significant decrease in the proportion of participants with post-dural puncture headache persistence and in those needing supplementary interventions" [41]. The meta-analysis reviewed five randomized controlled trials (RCTs) involving the use of caffeine in this context [42–46]. Two of these trials compared caffeine directly to placebo in the treatment of PDPH, and both oral [39] and IV [40] administrations of caffeine were found to be effective. There is mixed evidence that caffeine may also be effective for PDPH prophylaxis with one trial finding benefit with intravenous administration [47] during surgeries under spinal anesthesia and another two studies showing no benefit with oral caffeine alone [48] or in conjunction with acetaminophen [49].

Caffeine and caffeine-containing analgesics are also current first-line treatments in the termination of hypnic headaches—the so called "alarm-clock" headaches that wake patients from a night's sleep at a consistent time, usually in the early mornings [50]. There are several case series to support the use of caffeine in the treatment of hypnic headaches [51–54], and due to the rarity of the condition, there is unlikely to be higher quality evidence.

Tonic caffeine exposure is known to result in adenosine receptor up-regulation [55, 56] but the clinical impact of chronic regular caffeine intake on headache is less well understood. Certainly, caffeine intake may exacerbate primary

headaches by triggering secondary headaches recognized by the ICHD III-beta criteria as two related disorders: caffeine withdrawal headaches and medication overuse headaches [24]. The observed higher frequency of migraines during the weekends compared to weekdays is felt by some to be related to caffeine withdrawal [57]. The prevalence of caffeine withdrawal headaches varies widely in survey studies ranging from 0.04 [58] to 56 % [59] and in experimental studies between 9 and 100 % [59].

The prevalence of coffee as a trigger for migraine in the reported literature ranges from 6.3 [19] to 14.5 % [60] (Table 2). In the Daisen study, the odds ratio associated with daily coffee or tea intake and the prevalence of migraines was 2.4 (CI 0.6–1.9, $P < 0.0001$) compared to those with occasional coffee or tea intake but it is difficult to attribute this to a causal relationship as opposed to increased use of caffeine for headache alleviation among migraine sufferers [32]. The Head-Hunt study, which analyzed cross-sectional data from 50,483 Norwegian individuals over 20 years, also found increased headache prevalence, including migraines, in those with the highest amount of daily caffeine intake (>540 mg/day) compared with those from the lowest group (0–240 mg/day) (odds ratio (OR) = 1.13, 95 % CI 1.07–1.20) [61]. However, the bulk of the effects are seen to be driven by non-migraine headaches (OR = 1.14, 95 % CI 1.07–1.21) rather than migraine headaches (OR = 1.10, 95 % CI 1.01–1.20).

Interestingly, in Head-Hunt study, chronic headaches (>14 days/month) were more prevalent among individuals with low caffeine intake compared to those with moderate or high intake. This is in contrast to other studies showing patients with chronic migraines and chronic daily headaches are more likely to have higher caffeine consumption [62, 63], with one study finding the high caffeine intake predating the development of a chronic daily headache [63].

Alcohol

The recreational use of ethanol is ubiquitous across cultures and likely dates as far back as 10,000 BC [64]. Because of its popularity, its relationship to migraines has been well documented (Table 2). However, due to the heterogeneity of alcoholic beverages available, these results are not easy to interpret. Wine, beer, and whiskey are all known to contain vasoactive bioactive amines (discussed below) where as other liquors like gin and vodka generally do not in appreciable quantities [65]. Furthermore, wine and beer can contain other compound such as sulphites and phenolic flavonoids, which may further confuse interpretation.

Alcohol-related headaches have been documented since antiquity [66], and the ICHD III-beta lists two secondary headaches directly associated with alcohol: immediate and delayed alcohol-induced headaches [24]. Immediate alcohol-induced headaches develop within 3 h of alcohol ingestion and resolve within 72 h post alcohol cessation, and the headache is either pulsatile, bilateral, or worsen with physical activity [24]. Delayed alcohol-induced headaches, one of the most common types of secondary headaches, have similar features to immediate alcohol-induced headache except the headache develops within 5–12 h.

With regards to its effect on migraine, within the Head-Hunt study, migraine prevalence was found to be lower among both consumers of wine (0.8 %; 95 % CI 0.7–0.8) and liquor (0.8 %; 95 % CI 0.7–0.9) compared with those who abstain, and prevalence was found to decrease with increased units of alcohol consumption [67]. A causal relationship should not be inferred as migraine patient may self-restrict alcohol use as alcohol is known to be a dietary trigger for many migraine patients with 1.4 to 51.6 % of patient affected (Table 2). Those with cluster headache also tend to consume less alcohol than the general population [68].

Whether this effect is driven purely by bioactive amine contained within alcoholic beverages is uncertain. One study demonstrated that headache was provoked by 300 ml of red wine but not a vodka and lime mixture of equal ethanol content [69]. There is still little certainty on what the culprit mechanism leading to alcohol-triggered migraines is though it is likely multifactorial involving histamine, tyramine, sulphites, flavonoids, and 5-HT release [70].

Dairy

As with alcohol, studies looking at the relationship of dairy and migraines are difficult to interpret due to the heterogeneity of dairy products being studied. In particular, aged cheeses contain more vasoactive compounds such as tyramine when compared to milk or fresh cheeses like cottage cream and cream cheese. When the dietary intake of common foods was reviewed within the Women's Health Study, a cohort of American women with and without headaches, a reduction in reported total dairy product, aged cheese, sour cream, and milk consumption was seen in those with migraines compared to those without headaches [71••]. The authors interpreted this as a possible indication of self-restriction of a self-identified migraine trigger.

In general, processed dairy products seem to be reported more often than milk as a trigger among migraine patients (Table 2). One study tracking dietary migraine triggers among patients in Brazil found that while 8.5 % identified cheese as a trigger, only 2.5 % identified milk [60]. A Belgium study reported that 4.6 % thought ice cream a migraine trigger while only 2.3 % identified milk as a trigger [27].

Tyramine

Tyramine is a naturally occurring biogenic monoamine derived from the amino acid tyrosine. It has been proposed that migraine patients with certain food sensitivity may have a genetic deficiency in the enzyme responsible for the sulfate conjugation of tyramine [72]. Tyramine may underlie the associate of chocolate, alcohol, and dairy to migraines. One study was able to demonstrate precipitation of migraines in food-triggered migraineurs after 100 mg of tyramine but not with a 100 mg placebo of lactose while patients with no history of dietary triggers and controls were not affected by the tyramine [73]. A study by Peatfield looking at food triggers discovered an association between sensitivity to cheese/chocolate, red wine, and beer ($P < 0.001$) within migraine patients further supporting a shared factor in these food sensitivities [74].

However, a review conducted in 2003 on dietary intolerance of biogenic amines reviewed 12 available studies looking at tyramine and migraines where subjects were given an oral challenge of tyramine including Peatfield's study [75]. Six of these 12 studies were from E. Hanington and had considerable overlap in data. The 12 studies reviewed had mixed results with all of the Hanington studies and one other study finding a relationship between tyramine ingestion and headaches while the other five studies found no such relationship. The review identified methodological flaws with all but two of the studies, both of which concluded there was no elevated incidence of headaches after tyramine ingestion among patients with migraine.

Histamine

Histamine is also a naturally occurring biogenic amine derived from the amino acid histidine. Some authors have proposed that defects in diamine oxidase leading to inadequate histamine degradation may be found in a portion of individuals with food- and wine-triggered headaches [76]. Individuals who suffer from this disorder can also experience sneezing, diarrhea, pruritis, and shortness of breath. Wine intolerance is a manifestation because alcohol competitively inhibits the diamine oxidase enzyme. This theory of wine-triggered migraines was challenged by a study showing no difference between histamine antagonists vs. placebo for alleviation of wine-induced headaches among migraine patients [77].

Phenylethylamine

Phenylethylamine is another biogenic monoamine, which is synthesized from the amino acid phenylalanine. It is found in a variety of foods, including animal products, wine, and chocolate. Because phenylethylamine (PEA) is metabolized by monoamine oxidase, it is thought that reduced monoamine oxidase activity may be the underlying mechanism behind PEA-triggered migraines [78].

There is not a great deal of evidence supporting this theory. One non-randomized PEA oral challenge study in those with self-reported chocolate sensitivity found PEA capsule triggered more migraines when compared with lactose capsules [78]. Another study involving 66 women with a history of chronic headaches randomized to either 60 g of chocolate (with 1.9 μg/g of phenylethylamine) or 60 g of carob (with 0.4 μg/g of phenylethylamine) after a restrictive 2-week diet found no difference in headache frequency between the groups [79].

Aspartame

Aspartame is a dipeptide, which consists of the methyl ester of phenylalanine and aspartic acid. It is eventually converted into methanol and oxidized to formaldehyde and formic acid. Since its approval as a sweetener by the FDA in 1980s, numerous complaints have been made to the FDA regarding adverse side effects including headache (45 %), dizziness (39 %), confusion/memory loss (29 %), and insomnia (14 %) [80].

Evidence from five randomized double-blind studies exploring aspartame-triggered headaches yield mixed results with three studies involving 40, 48, and 108 patients, respectively, revealing no difference in headache frequency between those exposed to placebo vs. those exposed to aspartame [81–83] and two other studies, which demonstrated higher frequency of headaches among certain subjects' exposure to aspartame compared to placebo [84, 85].

Case reports exist of migraines triggered after ingesting aspartame-containing rizatriptan [86] and chewing gum [87], and aspartame has been identified by 9 % migraine patients as a trigger in two studies [60, 88].

Nitrites

In 1972, Henderson and Raskin published a case of nitrite-sensitive headache, which later became known as the "hot-dog headache" [89] as nitrates are often used as preservatives for meats. While previously thought to be an inert degradation productive of nitric oxide, it is now known that dietary nitrites and nitrates serve to help regulate nitric oxide hemostasis [90]. The reduction of nitrite to nitric acid can lead to vasodilation via activation of soluble guanylyl cyclase in smooth muscle [91]. Nitric oxide is also produced and released during cortical spreading depression and may be directly involved in pain modulation within the central trigeminal pathway [92].

Plasma nitrite levels of migraine patients have been shown to increase during attacks compared to when in periods of remission [93]. However, a recent review of four RCTs and an open-labeled clinical trial concluded that non-selective NOS inhibitors likely are ineffective in migraine treatment and carry significant cardiovascular side effects [94]. Work on neuronal nitric oxide synthesis inhibition in headache treatment remains an area of ongoing research.

Monosodium Glutamate

Monosodium glutamate is the sodium salt of glutamic acid, a naturally occurring nonessential amino acid, which enhances the savory ("umami") taste of many foods. It was first identified by the Japanese chemist Kikunae Ikeda in 1908 [95] and is now a common food additive and flavor enhancer. With its increasing prevalence, especially among packaged fast food and Chinese food, there have been many subjective reports of adverse reactions. Headaches due to ingestion of monosodium glutamate (MSG) are typically pressing/tightening or burning in quality; however, in patients with migraines (Table 2), it may be pulsatile. Flushing of the face; pressure in the face and chest; burning sensations in the neck, shoulders, and/or chest, dizziness; and abdominal discomfort may accompany the headache in response to MSG [24]. According to ICHD III-beta criteria, MSG-induced headache must have developed within 1 h of MSG ingestion and resolve within 72 h after ingestion.

The authors of a recent review concluded that the available evidence suggests that due to its distinctive taste, beverages containing 1.3 % MSG (2 g/150 ml) or more should be distinguishable by taste from placebo, which affects blinding within RCTs [96]. The review identified five papers describing six RCTs studying the effects of MSG in food. There was no difference in headache found between placebo and administrations of 1.5 and 3.0 g of MSG in capsule form prior to food, 3.15 g of MSG in 300 ml of beverage, 3.0 g of MSG in boiled rice with pork, and 3.0 g of MSG in 150 ml beef broth. The only significant difference was within female subjects administered 3.0 g MSG in 150 ml (2.0 %) of beef bouillon but not in male subjects. No MSG effect of headache was found within the three studies that were felt to be adequately blinded by the authors of the review [97–99].

Elimination Diets Based on Serum Food Antibody Testing

Recent efforts for more objective identification of food hypersensitivities among migraine patients have led to research involving serological testing of antibodies against various food antigens.

An early study found no difference in serum titers of total IgE or IgG antibodies directed against cheese, milk, and chocolate in migraine patients with food triggers compared to those without headaches [100]. However, since then, there has been an association found between atopic conditions such as allergic rhinitis, eczema, and asthma to migraine and elevated total serum IgE antibody titers in migraine patients compared to controls [101]. Additionally, higher IgE titers were found in patients with more severe migraines [102] and during migraine periods than in remission [103]. However, a prospective study looking at IgE skin prick testing for food trigger identification in migraine patients failed to trigger any attacks after oral challenges with foods positive on IgE skin testing [104].

There has also been revitalized interest in IgG antibodies after promising research for IgG-guided elimination diets among patients with irritable bowel syndrome (IBS) [105]. A study involving 56 migraine patients from Mexico found that the patients had elevated IgG titers to many more food triggers compared to controls and that 43 out of 65 patients showed a complete remission of their migraine after 1 month of an IgG-guided elimination diet [106]. Since then, there have been two small blinded cross-over studies within migraine patients showing a reduction in headache days and number of attacks on IgG-guided food elimination diets [107, 108]. A larger-scale RCT involving 167 subjects with self-endorsed migraines showed a small decrease in the number of migraine-like headaches over 12 weeks between those randomized to an IgG-guided elimination diet compared to a sham elimination diet, though the result did not reach statistical significance [109].

However, there are some who believe that elevated titers of food-directed IgG in many migraine patients represent an epiphenomenon of increased baseline inflammation rather than true allergic reaction [110]. As in IBS, the use of IgG-guided elimination diets for migraine control remains an area of contention and is not yet standard of care until more evidence is available regarding its efficacy.

The Impact of Dieting

An association between migraine headaches and an elevated body mass index (BMI) has been found in numerous large population studies, particularly among those of reproductive age (<50 years old) or younger [111]. One of these studies reported an 1.5-fold (OR 1.48, 95 % CI 1.12–1.96) increase in odds of migraines for those with class I obesity (BMI 30–34.9), a 2-fold (OR 2.07, 95 % CI 1.27–3.39) increase in those with class II obesity (BMI 35–39.9), and an almost 3-fold (OR 2.75, 95 % CI 1.60–4.70) increase in those with class III obesity (BMI >40) [112].

So far, no difference has been found in the number of migraine days in obese migraine patients and those with normal BMI [113]. However, obese individuals with episodic headache have a greater risk of transformation to chronic headaches than those with episodic headache who are not obese [111]. Additionally, two case series involving bariatric surgery in overweight migraineurs have demonstrated significant improvements in frequency and duration of the attacks ($P = 0.02$) as well as lower medication use for migraine attacks after surgery [114, 115]. All this would suggest that there may be significant benefits to healthy diets promoting weight loss among migraine patients.

Care should be taken during this process as hunger or skipped meals are among one of the most consistently identified dietary triggers (Table 1). Furthermore, headache attributed to fasting is also a well-described secondary headache with its own specific diagnostic criteria in the ICHD III-beta [24]. A study looking at patients during Yom Kippur found that 39 % (82 patients) developed headache as compared to 7 % (9 patients) in the non-fasting control group [116]. Studies looking at patients fasting for Ramadan found similar results [117, 118] with perhaps a tripling of migraine days during Ramadan compared to a control month.

It is difficult to tease out the contribution of caffeine withdrawal and hydration in fasting headaches. Recent case reports have noted that water deprivation can trigger migraines [119], and a very small study looking at 18 migraineurs showed an increase of one liter per day of water intake was associated with an average reduction of headaches by 21 h in 2 weeks as well as a reduction in subjective headache intensity as rated by a visual analog scale [120].

In terms of a diet that may be beneficial, the Dietary Approaches to Stop Hypertension (DASH)-sodium RCT found that reduced sodium intake was associated with a significantly reduced risk of headaches [121]. This may be secondary to blood pressure benefits associated with the DASH diet.

The effect of dietary fat on migraine is more nuanced. One population study identified a higher lipid intake among migraine patients compared to the rest of the population [25], and certain patients find fatty food to be a migraine trigger [27]. Several studies have reported that lower dietary fat significantly decreases the number and intensity of acute migraine attacks [122, 123]. However, the ketogenic diet, which shifts the majority of caloric intake from carbohydrates to lipid sources, has also been extensively studied as a method of improving migraines since as early as 1928 [124, 125]. Most recently, one study involving 96 overweight female migraine patient randomized to either 1 month of low-calorie ketogenic diet followed by a standard low-calorie diet for 5 or 6 months of the standard low-calorie diet found that while headache frequency and total headache days decreased in both the groups, the decrease occurred much more rapidly in those randomized to a period of ketogenic diet first [126].

Conclusion

The impact of diet on headache is clearly variable between patients. Studies attempting to quantify the frequency of triggers among migraine patients yield widely discrepant information (Tables 1 and 2). One potential source of variation may be from patient selection as some of these studies recruited from headache clinics whereas others were cross-sectional population studies. Most studies in Tables 1 and 2 were retrospective studies using self-reporting, which is vulnerable to small variations within the data collection method. For example, one study showed that while only 75.9 % of patients initially endorsed having migraine triggers when asked, this number rose to 94.6 % when the patients were questioned regarding specific triggers [127]. Intuitively, prospective studies either using either oral challenges or food diaries would likely yield information that is less sensitive to bias but these study designs are less feasible for studying larger populations.

There is clearly still much to discover in the relationship between diet and headache. However, given the prevalence of dietary triggers, clinicians should encourage the use of a food diary for headache patients to identify these triggers. A trial of eliminating the identified triggers from the diet is a relatively benign intervention that may significantly lessen morbidity. Other strategies like ensuring regular meals to avoid hunger and dehydration, a sodium-restricted diet, and calorie-restricted diets for overweight migraine patients are other promising adjunctive strategies to traditional headache management.

Compliance with Ethical Standards

Conflict of Interest Zoya Zaeem, Lily Zhou, and Esma Dilli declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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