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Tyramine Ingestion and Migraine Attack: A Systematic Review

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Abstract

AIM: This systematic review aimed to clarify the association between tyramine ingestion (food/capsule) with the occurrence of a migraine attack.

METHODS: Based on a comprehensive search of PubMed, PMC, ProQuest, and EBSCOhost, we included all studies that observed migraine headaches after tyramine ingestion. Association was evaluated with the control (placebo) group as a comparison. We further assessed the abstracts and full texts of selected papers. We included articles fulfilling the inclusion criteria in this systematic review.

RESULTS: Seven non-randomized studies with 322 eligible subjects (168 women, 63 men, and 91 unknown genders) aged 4 to 62 years old were included in the study. A high to moderate (17.2–50%) occurrence of headache after tyramine ingestion was observed. Similar results were observed in the control (placebo) group (0–42.1%). Quality appraisal on all seven non-randomized studies using ROBINS-I showed that six studies had a moderate risk of bias, and one study had a serious risk of bias.

CONCLUSION: The relationship between tyramine-containing food and migraine remains unclear.

Introduction

Headache is one of the most prevalent neurological disorders and the most frequent symptom seen in general practice [1]. The two most common primary headache subtypes are tension-type headache (TTH) and migraine [2]. More than 90% of the general population worldwide reported a lifetime history of headaches [3]. Migraine is a disabling chronic disease that influences an individual physical, mental, and social function. The active global prevalence of migraine is 10–15%. It is commonly found between the ages 25–55 years, which is a productive age, meaning that it will cause a significant economic impact if left untreated. Migraine is also 3 times more common in females [4].

Many factors contributed to the onset, frequency, duration, and severity of migraine attacks, including nutrition as a predisposing factor or even as a trigger [5]. For years, tyramine has been associated with migraine attacks. Tyramine is a trace monoamine produced in foods from the natural breakdown of the amino acid tyrosine. Monoamines (such as dopamine, norepinephrine, and serotonin) are molecules with an amine group separated from an aromatic ring by a two-carbon chain. It is naturally found in food, plants, and animals, but the levels will increase naturally when they are aged, fermented, stored for long periods, or when they are no longer fresh [6]. Tyramine is degraded by several enzymes, like decarboxylase enzyme [7]. Tyramine has long been shown to cause cardiovascular effects when consumed in large amounts or combined with monoamine oxidase inhibitors (MAOIs) [7]. It acts as a vasoactive amine that leads to cerebral vasoconstriction and subsequent rebound vasodilatation that causes a migraine attack in susceptible person [8].

Direct administration of tyramine displaces norepinephrine, epinephrine, and dopamine from pre-synaptic storage vesicles [9]. The release of these neurotransmitters, particularly norepinephrine, causes vasoconstriction, increased heart rate, and increased blood pressure. Tyramine causes the release of endogenous presynaptic neurotransmitters and disrupts the monoamine activity, further limiting the breakdown of monoamine neurotransmitters [10].

In this review, we aim to elucidate the role of tyramine as a risk factor for the development of migraine. Our study could provide additional insight into preventing migraine, primarily through nutrition.

Methods

For this systematic review, a structured literature search was conducted from inception until September 19, 2022, to identify published articles on the effect of tyramine ingestion on a migraine attack. We use the Preferred Reporting Items for Systematic Reviews and Meta-analysis (PRISMA) statement guideline, with a pre-determined search strategy, in which we identified potential studies, screened titles and abstracts, assessed full-text articles, and determined relevant studies.

Search strategy and selection criteria

We did a systematic literature search on PubMed, PMC, ProQuest, and EBSCOhost. The following search terms were used: ("tyramine" OR "4-hydroxyphenethylamine" OR "4 hydroxyphenethylamine") AND ("migraine" OR "headache"). Table 1 provides our complete search terms strategy and findings. The search terms, especially for headaches, were kept broad to encompass all possibilities for applicable studies. There were no restrictions on the publication date and the articles' language. After eliminating duplicates, the authors reviewed all titles and abstracts and excluded those articles with full text that failed to be retrieved. Any disagreement will be resolved with consensus.

Table 1: Search terms strategy

Source	Search term	Number of studies
PubMed	("Tyramine"[MeSH Terms] OR "Tyramine"[All Fields] OR "4 - Hydroxyphenethylamine"[All Fields] OR "4 Hydroxyphenethylamine"[All Fields]) AND ("Migraine"[MeSH Terms] OR "Migraine"[All Fields] OR "Headache"[All Fields])	194 studies
PMC	("Tyramine"[MeSH Terms] OR "Tyramine"[All Fields] OR "4-Hydroxyphenethylamine"[All Fields] OR "4 Hydroxyphenethylamine"[All Fields]) AND ("Migraine"[MeSH Terms] OR "Migraine"[All Fields] OR "Headache"[All Fields])	698 studies
ProQuest	(ti ("Tyramine") OR ti ("4-Hydroxyphenethylamine") OR ti ("4 Hydroxyphenethylamine") OR ab ("Tyramine") OR ab ("4-Hydroxyphenethylamine") OR ab ("4 Hydroxyphenethylamine")) AND (ti ("Migraine") OR ti ("Headache") OR ab ("Migraine") OR ab ("Headache"))	29 studies
EBSCOhost	(TI Migraine OR TI Headache OR AB Migraine OR AB Headache) AND (TI Tyramine OR TI "4 hydroxyphenethylamine" OR TI "4 - hydroxyphenethylamine" OR AB Tyramine OR AB "4 hydroxyphenethylamine" OR AB "4 - hydroxyphenethylamine")	151 studies

Inclusion and exclusion criteria

The inclusion criteria were the following: (a) clinical studies in humans, (b) diagnosis of migraine was clearly defined, (c) migraine was evaluated after ingestion of tyramine in chocolate/tyramine capsule, and (d) patients treated with placebo and/or any supplemental or non-invasive therapy as a comparison. The exclusion criteria were the following: (a) other types of studies, including case–control, cohort, cross-sectional, case report, case series, literature review, book sections, conference papers, preliminary report, and correspondences, (b) studies in animal subjects, (c) absence of a comparison group/placebo, (d) prior consumption of medication that affects platelet function/ monoamine oxidase (MAO) activity, and (e) subjects with comorbidities.

Data extraction and analysis

We reviewed the final articles to extract the information from each: first author, date published; study design; study population characteristics; the number of samples; intervention; outcome measurement; and the result. The specific outcome measures recorded for the systematic review were the frequency of headache/ migraine attacks ≤48 h after the ingestion of tyramine. The information was extracted by two independent authors (HS and OD) to ensure the accuracy of all the data. We resolved all conflicting data with consensus among all the authors.

Quality assessment

Articles selected for the final analysis were graded by two authors (HS and OD) using Risk of Bias in Non-randomized Studies of Interventions (ROBINS-I) [11] for the quality assessment. We will resolve any disagreement with consensus. ROBINS-I is a tool for evaluating the risk of bias in estimates of the comparative effectiveness of interventions from studies that did not use randomization to allocate units to comparison groups [11]. This tool was deemed suitable as all five studies included in this study are non-randomized. All researchers independently evaluate the quality of each study, with any discrepancies resolved through consensus.

Result

Study selection

We identified 1077 published papers, and seven studies were included in this systematic review [12], [13], [14], [15], [16], [17], [18]. We imported results into Endnote X9 (Trial Version). From the initial search, a total of 199 articles were identified from PubMed, 698 from PMC database, 151 from EBSCOhost database, and 29 from ProQuest database. One hundred and ninety-three articles were excluded as duplicates, leaving 884 articles to be reviewed. The abstract of these articles was reviewed based on the following criteria: relevant independent and dependent variables, direct ingestion of tyramine or food-containing tyramine, and trial studies. Fourteen full texts were reviewed of which seven articles were excluded. Two of these excluded articles measure tyramine level in the case and control groups, one article measure tyramine

in the food, one article measure tyramine excreted from the urine, one article uses blood pressure as the dependent variable, one article is only available in German language, and the last article only had a single subject. The search strategy and selection methods of the present study following PRISMA guidelines are illustrated in Figure 1.

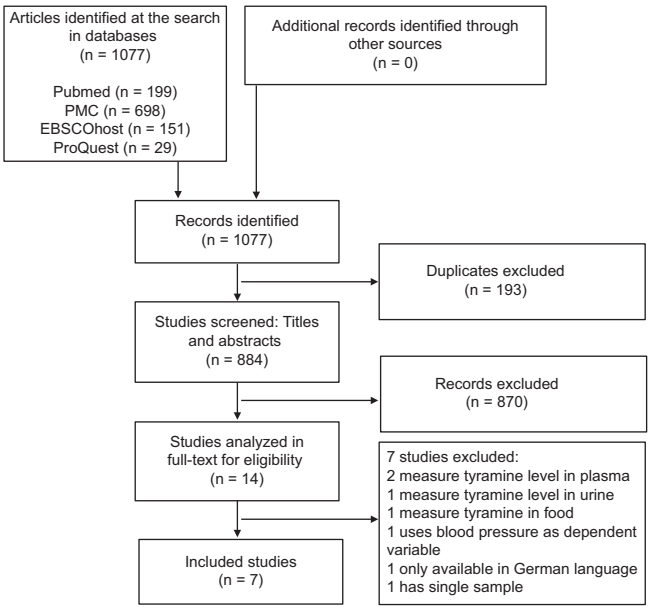

Figure 1: Study selection process

We finally selected seven articles for the systematic review with the following criteria: clinical trial studies in humans, intervention in the form of tyramine supplementation, and headache/migraine attacks frequency are recorded completely. The study type, sample size, mean/median age, and male: female distribution of various studies are shown in Table 2. The group, intervention, measurement, and outcome are shown in Table 3.

Study characteristics and demographics

Table 2 shows the summary of all seven studies' characteristics. All studies were considered old because they were done before 2000. A total of 322 subjects were collected; of all 231 subjects with identified gender, most were female (168/231; 72.7%). The study population age varies from 4 to 62 years old. The range is considered wide because one study by Forsythe *et al.* [15] was conducted in children with an average age of 10.5 years (phase 1) and 9.6 years (phase 2), while the other four studies [13], [14], [17], [18] were conducted in adults. Two studies [12], [16] did not specify their study population characteristics.

Table 4 summarizes the intervention and the outcome of all studies. The intervention of tyramine ingestion was given in chocolate or capsule form. Three studies [13], [17], [18] use chocolate as a tyramine source compared to non-cocoa chocolate as a placebo. Four studies [12], [14], [15], [16] directly use tyramine powder in capsule form. The tyramine-containing chocolates used across all studies using chocolate were manufactured either by the American Cocoa Research Institute or Cadbury Ltd., Bourneville, Birmingham. The dose of tyramine in tyramine-containing chocolates was not specified in any of the studies, but in the capsule form, the tyramine was given as much as 100 mg up to 250 mg daily.

Out of the ten experimental phases from seven studies, migraine attack from tyramine digestion (capsules/tyramine-containing chocolate) was low to moderate (17.2–50%), similar to placebo (0–42.1%). The definition of migraine varied across all studies, depending on the guidelines used then. All studies measured headaches <48 h after ingestion. Most post-ingestion headaches in these studies are identified as a migraine as it was reported to be similar to the patient's usual migraine headache as described earlier by the patient questionnaire, except for one study by Moffet *et al.* in 1972 that described reported atypical headache compared to the patient's usual migraine headaches.

Quality appraisal

Quality appraisal on all seven studies was done using the Risk of Bias in Non-randomized Studies of Interventions (ROBINS-I) tool. Most of these studies showed moderate confounding bias as they are non-randomized and lacked patient dietary control before the intervention. This is an essential factor in this study observing the association between tyramine-containing food and migraine attack. The study by Forsythe

Table 2: The sample size of selected studies and their characteristics

First Author, Publication year	Country of origin	Study type	Group	Sample size (n)	Age, mean/median	Male (n; %)
Hanington *et al*., 1967[12]	England	Double-blind placebo-controlled trial	Tyramine (capsule)	12*	N/A	N/A
			Control (placebo)	12*		
Moffett *et al*., 1974[13]	England	Two separate double-blind placebo-controlled trial	Tyramine (chocolate)	25*	49	*n* = 2; 8
			Control (placebo)	25*		
Moffett *et al*., 1972[14]	England	Double-blind placebo-controlled trial	Tyramine (capsule)	25*	35.7	0
			Control (placebo)	25*		
Forsythe *et al*., 1974[15]	Ireland	Two separate double-blind placebo-controlled trial	Tyramine (capsule)	97*	10.5 (phase - 1) and 9.6 (phase - 2)	n = 58; 59.8
			Control (placebo)	97*		
Ryan (phase-1)[16]	USA	Two separate double-blind study	Tyramine (capsule)	79*	N/A	N/A
			Control (placebo)	79*		
Gibb, *et al*., 1991[17]	England	Double-blind parallel study	Tyramine (chocolate)	12	37	*n* = 1; 8.3
			Control	8	42	*n* = 2; 25
Marcus, *et al*., 1997[18]	USA	Double-blind study	Tyramine (chocolate)	64*	28.3	0
			Control	64*		

*Same subjects on intervention and control group, but different period of time (1–14 days apart). N/A: Not available.

Table 3: Summary of risk of bias in nonrandomized studies of interventions - I assessment

Author	Confounding bias	Selection bias	Measurement of intervention	Deviation from intended intervention	Missing data	Measurement of outcomes	Reported result	Overall
Hanington, (1967)[12]	High	Moderate	Low	Low	Low	Moderate	Low	Moderate
Moffett *et al*., (1974)[13]	Moderate	Low	Low	Low	Low	Moderate	Low	Moderate
Moffett *et al*., (1972)[14]	Moderate	Low	Low	Low	Low	Moderate	Low	Moderate
Forsythe *et al*., (1974)[15]	Moderate	Low	Low	Low	Low	Moderate	Low	Moderate
Ryan (1974)[16]	High	Moderate	Low	Low	Moderate	Moderate	Low	Serious
Gibb *et al*., (1991)[17]	Moderate	Low	Low	Low	Low	Moderate	Low	Moderate
Marcus *et al*., (1997)[18]	Low	Low	Low	Low	Low	Moderate	Low	Moderate

et al. [15] also told the parents about the contents of the capsule; therefore, this might affect the outcome psychologically. One study by Marcus *et al*. [18] was considered a low confounding bias because they restricted vasoactive amine consumption 2 weeks before the intervention, and subjects were asked to fill in food diaries for diet monitoring. Two studies by Hanington *et al.* [12] and Ryan *et al.* [16] did not specify their study population characteristics/demographics (e.g., age, female-to-male ratio, and recent drug use); therefore, the author considered this a risk for confounding and selection bias.

The measurement of the outcomes of all studies was considered prone to bias because they are based on subjective feelings of headache and thus cannot be scientifically proven. However, the authors of this study believe that migraine attacks are subjective bodily symptoms. Thus, they are difficult to measure objectively.

Discussion

Many of the subjects in these studies (72.7%) were female. The reason is that migraine is a predominantly female disorder and most reports of foods triggering headaches also came from women [19]. The age of the included studies ranged from 4 to 62 years. Migraine is a common disease in childhood, with its highest prevalence in adolescence and early adulthood

Table 4: Interventions and measured outcomes

First author, (year)	Group	Intervention	Assessment	Outcome
Hanington (1967)[12]	Tyramine (capsule)	12 subjects were given a capsule containing 100 mg of tyramine	Headache after ingestion of a capsule in 24 h	6 subjects (50%) had headache after tyramine consumption
	Control (placebo)	12 of the same subjects were given a capsule containing 100 mg of lactose as placebo		0 subjects (0%) had headache after placebo consumption
Moffett A, *et al*. (1974)[13] Phase-1	Tyramine (chocolate)	25 subjects were given 44–62 g of chocolate containing tyramine	Headache after ingestion of a chocolate sample in 48 h	9 subjects (36%) had headache after tyramine chocolate consumption
	Control (placebo)	25 of the same subjects were given 44–62 g of placebo (noncocoa and coberine-containing chocolate), 14 days after chocolate ingestion		6 subjects (40%) had headache after placebo consumption
Moffett A *et al*. (1974)[13] Phase-2[a]	Tyramine (chocolate)	15 subjects from the first trial were given 44–62 g of chocolate containing tyramine	Headache after ingestion of a chocolate sample in 48 h	6 subjects (40%) had headache after tyramine chocolate consumption
	Control (placebo)	15 subjects from the first trial were given 44–62 g of placebo (noncocoa and combine-containing chocolate), 14 days after chocolate ingestion		4 subjects (26.7%) had headache after placebo consumption
Moffett A *et al*. (1972)[14]	Tyramine (capsule)	25 subjects were given a capsule containing 125mg of tyramine	Headache after ingestion of a capsule in 48 h	6 subjects (24%) had headache after tyramine consumption
	Control (placebo)	25 of the same subjects were given a capsule containing 125 mg of lactose placebo, 4–7 days after chocolate ingestion		6 subjects (24%) had headache after placebo consumption
Forsythe WI *et al*. (1974)[15] Phase-1	Tyramine (capsule)	59 subjects were given a capsule containing 100 mg of tyramine	Headache after ingestion of a capsule in 48 h	16 subjects (27.1%) had headache after tyramine consumption
	Control (placebo)	59 subjects were given a capsule containing 100 mg of lactose placebo, 2 days after chocolate ingestion		14 subjects (23.7%) had headache after placebo consumption
Forsythe WI *et al*. (1974)[15] Phase-2[b]	Tyramine (capsule)	38 subjects were given a capsule containing 100 mg of tyramine	Headache after ingestion of a capsule in 48 h	9 subjects (23.7%) had headache after tyramine consumption (5 after tyramine only, 4 after both tyramine and placebo)
	Control (placebo)	38 of the same subjects were given a capsule containing 100 mg of lactose placebo, 2 days after chocolate ingestion		16 subjects (42.1%) had headache after placebo consumption
Ryan (1994)[16] Phase-1	Tyramine (capsule)	79 subjects were given a capsule containing 125 mg of tyramine	Headache after ingestion of a capsule in 24 h	31 subjects (39.2%) had headache after tyramine consumption
	Control (placebo)	79 of the same subjects were given a capsule containing unspecified placebo, 24 h after chocolate ingestion		33 subjects (41.8%) had headache after placebo consumption
Ryan (1994)[16] Phase-2[a]	Tyramine (capsule)	50 subjects were given a capsule containing 125 mg of tyramine	Headache after ingestion of a capsule in 24 h	17 subjects (34%) had headache after tyramine consumption
	Higher dose Tyramine (capsule)	23 subjects were given a capsule containing 250 mg of tyramine		5 subjects (21.7%) had headache after higher dose tyramine consumption
	Control (placebo)	73 subjects were given a capsule containing unspecified placebo, 24 h after chocolate ingestion		26 subjects (35.6%) had headache after placebo consumption
Gibb CM *et al*., (1991)[17]	Tyramine (chocolate)	12 subjects were given 40 g of bar chocolate	Headache after ingestion of a chocolate sample in 32 h	5 subjects (41.7%) had headache after chocolate consumption
	Control (placebo)	8 subjects were given 40 g of placebo (noncocoa and coberine-containing chocolate)		0 subjects (0%) had headache after placebo consumption
Marcus *et al*., (1997)[18]	Tyramine (chocolate)	64 subjects were given 60 g of bar chocolate	Headache after ingestion of a chocolate sample in 12 h	11 subjects (17.2%) had headache after chocolate consumption
	Control (placebo)	64 of the same subjects were given 60g of placebo (noncocoa and coberine-containing chocolate)		26 subjects (40.6%) had headache after placebo consumption

[a]Subjects participation gin phase-2 trials who were kept from phase-1 trial, bCompletely new subjects who have not participated in phase-1 trial.

(late teens and early twenties). In children, headaches are considered rare, especially in children below the age of 4, because the data collection in this population was challenging [20], [21]. Migraine was common in late adolescence and early adulthood, with the prevalence peaking in late teens and early 20s [22].

The association between chocolate and migraine attack is rather ambiguous. The original evidence of an association between chocolate and migraine attack was based on the assumption of allergy mechanism. However, epidemiologic surveys showed controversial results. A study by Peatfield *et al.* [23] reported that migraine attributed to chocolate in 19% of subjects, higher compared to other non-tyramine food (e.g., citrus 11%; $p < 0.001$), but still lower than that of alcohol (29%; $p < 0.001$) and oral contraceptives in females (31%; $p < 0.001$). A study by Van den Bergh *et al.* on 217 migraineurs showed that chocolate caused migraine attacks in 22.5% of cases, still lower than alcoholic beverages (51.6%) but higher than cheese and dairy products (18.5%). In comparison, several other studies reported that the headache was attributable to chocolate in only 1.4 to 1.7% of cases [24], [25], [26], [27], this number was far lower than what was reported previously. A study by Yadav *et al.* [28] in 182 patients reported that no migraine attacks could likely be attributable to chocolate.

Hanington first postulated the potential association between tyramine-containing food and migraine attacks in 1967 [12]. A remarkable proportion of migraine sufferers had attributed their headaches to the consumption of certain types of food including cheese, citrus, chocolate, and alcohol. Many agreed that alcoholic beverages were most likely to trigger migraine attacks, followed by chocolate, cheese, and dairy products in order [29], [30], [31]. The difference in tyramine dose across food and beverages may explain this result because tyramine acts on the adipose tissues in a dose-dependent antilipolytic effect. A dose of 10 mg tyramine has been associated with a migraine attack, with a lower dose of 6 mg if monoamine oxidase inhibitor (MAOI) was consumed before tyramine ingestion. A high-performance liquid-chromatography study showed most beers contain roughly 0–3.16 mg/L of tyramine [32], which is far higher than chocolate (0.1–2.8 mcg/g of tyramine) [33], [34], and cheese (depending on the type of cheese but in general 17.5 ± 6.1 mcg/g of tyramine) [35]. Besides, Littlewood *et al.* [36] also showed that some alcoholic beverages like red wines might contain a migraine-provoking agent that is neither alcohol nor tyramine.

Many individual and dietary factors along with various mechanisms may contribute to the development of migraine, from neuropeptides, neuroreceptors, ion channels, sympathetic nervous system, cerebral glucose metabolism, inflammation pathway, nitric oxide release, etc. There was a belief that tyramine only induced migraine in previously migrainous patients, postulated a low concentration of gut monoamine oxidase in this population. A study by Smith *et al*. [37] observed tyramine excretion in urine after tyramine ingestion opposed this hypothesis. The result showed a similar level of free p-hydroxy-phenylacetic-acid (HPAA) in the urine of both normal and migrainous patients, meaning both populations have similar monoamine oxidase concentrations and aldehyde oxidase in the liver, kidney, and gut in.

The limitations of this study are: (1) All of the studies recorded are considered old studies, and the author could not identify additional studies that could conclusively elucidate this hypothesis. More research is needed in a larger controlled sample size. (2) Many factors could contribute to migraine attacks. Thus, it is difficult for the researcher to control all confounding variables that might play a role. Most studies can only control the ingestion of medication that affects monoamine oxidase enzyme activity in their sample. (3) The inability to objectively measure migraine headaches as a subjective bodily symptom may affect the data recording.

Conclusion

The relationship between tyramine-containing food and migraine remains unclear. Patients with migraine should not completely avoid all of the food described in this study unless they obtain a clear association and consistent migraine attacks after certain food ingestion. Many individual and dietary factors, along with various mechanisms, may contribute to the development of migraine. The effects of dietary triggers also depended on dosage, the timing of exposure, and genetic factors. Further controlled large-population clinical trial studies with randomization are needed to draw a more conclusive result.

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