Current Pain and Headache Reports (2024) 28:119–124
https://doi.org/10.1007/s11916-023-01198-z

UNCOMMON AND/OR UNUSUAL HEADACHES AND SYNDROMES (F COHEN, SECTION EDITOR)

# Unraveling the MSG-Headache Controversy: an Updated Literature Review

**Edmond Ahdoot**[1] · **Fred Cohen**[1,2]

Accepted: 30 November 2023 / Published online: 11 December 2023


## Abstract

**Purpose of Review** To review the evidence and role of monosodium glutamate (MSG) as a headache and migraine trigger.

**Recent Findings** MSG is a common food additive, has widely been linked as a trigger of headache, as well as other symptoms. However, the evidence for MSG as a causative agent for headache is debated. Various clinical trials over the past several decades have reported conflicting results, with studies suggesting that MSG does and does not increase the incidence of headache. However, the dosages of MSG exposure are often inconsistent across studies, with many studies administering a dose significantly higher than the average consumption.. Additionally, there are misconceptions about which foods and cuisines have MSG in them.

**Summary** MSG could be a potential trigger for migraine and headaches. It is unclear exactly how MSG plays into the migraine pathophysiology. It's crucial to accurately determine if MSG is present in one's diet to evaluate its potential impact on headaches.

**Keywords** Monosodium · Glutamate · MSG · Headache · Diet · Trigger

Headache and migraine are one of the most common neurological disorders affecting nearly 16% of the US population. [1]. Migraine can be a severely debilitating condition and has been evaluated to be the 2nd highest cause of years lived with disability and 1st among women between the ages of 15 and 49 years old [2]. Headache is persistently one of the top five most common reasons for emergency department visits [3]. Furthermore, the annual direct health care costs for patients with migraine in the USA is estimated to be $22,364 per person, and a greater indirect total cost estimate of over 19 billion dollars [4]. While over the past several decades there has been advancement in understanding the pathophysiology of migraine, there is still much to be understood about this painful and disabling condition.

Management of headache disorders and migraine involves a multipronged approach, and can include lifestyle modifications, medications, procedures, as well as other therapeutic regimens. Common lifestyle modifications may be recommended by healthcare providers that include reducing stress, improving sleep habits, increasing exercise and altering dietary composition [5]. Historically, the relationship between diet and migraine and other headache disorders has been controversial and is frequently debated. Diets including a ketogenic diet, IgG-based elimination diet, high omega-3/low omega-6 diet, low glycemic index diet, and a vegan diet have been shown, through randomized clinical trials (RCTs), to reduce headache/migraine pain and/or severity [6–10]. Several studies have highlighted how the intake of certain foods and drinks may potentially be a trigger for headache disorders (Table 1) [11]. Many of these foods contain histamine, phenylethylamine, and tyramine, which are established headache triggers [12]. Of these reported triggers, Monosodium glutamate (commonly referred to as MSG) has garnered much controversy over evidence and history of its relation as not just a headache trigger, but a cause of numerous ailments such as indigestion, palpitations, chest pain/tightness, flushing, and dizziness [13]. In this paper, we review the history and evidence of MSG as a trigger for headache and migraine disorders, as well as assessing the negative associations of Asian cuisines for its use of MSG.

✉ Fred Cohen
fredcohenmd@gmail.com

[1] Department of Neurology, Icahn School of Medicine at Mount Sinai, Mount Sinai Hospital, New York, NY, USA

[2] Department of Medicine, Icahn School of Medicine at Mount Sinai, Mount Sinai Hospital, New York, NY, USA

**Table 1** Common self-reported dietary triggers of headaches and migraine

| Dietary trigger | Description |
|---|---|
| Caffeine | Common beverages and foods containing caffeine include coffee, tea, chocolate, and certain soft drinks |
| Alcohol | Drinking alcoholic beverages, particularly red wine, beer, and spirits |
| Dairy products | Consumption of milk, yogury, ice cream, and aged cheeses (such as blue cheese, cheddar, feta, and Parmesan) |
| Monosodium glutamate (MSG) | Found in some processed foods, snacks, and used as a flavor enhancer |
| Nitrates and nitrites | Found in processed meats like hot dogs, sausages, and deli meats |
| Citrus fruits | Consumption of citrus fruits like oranges, lemons, limes, and grapefruits |
| Artificial sweeteners | Consumption of artificial sweeteners such as aspartame and surclose, often found in diet sodas |
| Tyramine-rich foods | Consumption of foods high in tyramine, such as aged meats, smoked fish, fermented foods, and certain beans |
| Onions | Consuming onions or foods cooked with onions, green onions, and shallots |
| Nuts | Eating nuts and legumes, such as peanuts, walnuts, almonds, and cashews |
| Fermented foods | Consuming fermented foods like sauerkraut, pickles, kimchi, and kombucha |
| Gluten | Including breads, baked goods, cereals, barley, and rye |

MSG, an umami substance, is a sodium salt of glutamic acid. It is naturally found in certain meats, certain cheeses (Parmesan and Roquefort), and vegetables (mushrooms, tomatoes, and broccoli) [14–16]. MSG was first derived in 1908 by Kikunae Ikeda in Japan and contains ~ 80% glutamic acid, ~ 20% sodium, and ~ 1% of contaminants and water. At room temperature, it exists as an odorless, white powder that is soluble in both alcohol and water. Subsequent to its discovery in Japan, it became the most widely used food additive to provide a unique savory flavor to food products [17]. While MSG may classically be associated as being an additive in Asian cuisine, several other cuisines use it as a flavor enhancer. MSG can be found in popular fast-food dishes, potato chips, frozen meals, and seasoning blends [18]. However, years later, MSG would become widely labeled as a potential trigger for a plethora of symptoms and etiologies including migraines and other headache disorders [19].

## Animal Studies

Numerous pre-clinical studies have been conducted measuring the neurological effects of MSG on mice and other rodents. Onaolapo et al. conducted a study on measuring the effect of low dose MSG on the neuronal morphology and antioxidant effects in mice. Six groups were given one of the following solutions orally: distilled water (control), L-glutamate at 10 mg/kg body, and MSG at 10, 20, 40, and 80 mg/kg of body weight for 28 days. They observed that relative brain weight increased in the mice who were given 40 and 80 mg/kg of MSG compared to distilled water. Histological samples of the mice brains' found neuronal damage in the cerebellum, cerebrum, and hippocampus. Plasma glutamine and glutamine were elevated but no significant difference in total brain glutamine and glutamate levels were observed [20].

Sadek et al. conducted a study giving MSG and/or lycopene subcutaneously. Compared to placebo, those that received MSG were found to have increased levels of brain and serum cholinesterase, creatin phosphokinase, and lactate dehydrogenase, an increase in lipid peroxidation markers, and a decrease in glutathione [21]. Furthermore, msg was found to cause neuronal death via upregulation of pro-apoptotic Bax proteins. Swamy et al. and Shivasharan et al. assessed giving rats 2 g/kg MSG intraperitoneally. Both studies reported decreased glutathione, decreased locomotor activity, and altered hippocampal neuronal histology [22, 23].

## Human Studies

Several studies have been conducted on the relation of MSG and headache in humans, primarily using two approaches with placebo: with and without simultaneous food intake (Table 2). Prawirohardjono et al. conducted a study providing healthy volunteers with either capsules of placebo or MSG (1.5 and 3.0 g/person) during breakfast. No statistically significant difference was seen in the incidence of headache among the treatment and control group [24]. In a similar study, Tarasoff and Kelly conducted a study where placebo and differing levels of MSG were administered before a standardized breakfast over 5 days. They too reported no statistically significant difference in headache incidence among the treatment and placebo groups [25].

Morselli PL and Garattini S, and Zanda et al. conducted studies providing 3 mg of MSG in 150 ml of beef broth with meals. The former reported no statistical difference in headache incidence, while the latter did report a statistically

**Table 2** Studies conducted on the relation of MSG and headache in humans, primarily using two approaches with placebo: with and without simultaneous food intake

| Author (year) | Method of MSG administration | Dose of MSG | Statistical difference |
|---|---|---|---|
| Morselli PL and Garattini, S (1970) [26] | Beef broth | 3 g MSG in 150 ml beef broth | No difference |
| Rosenblum I et al. (1971) [27] | 100 ml tap water or chicken stock | 8 g MSG<br>12 g MSG | No difference |
| Kenney RA et al. (1972) [28] | 150 ml tomato juice | 5 g MSG | No difference |
| Zanda G et al. (1973) [29] | Beef bouillon | 3 g MSG in 150 ml beef bouillon | Only women in the MSG-treated group had a statistically significant higher headache incidence than the control group* |
| Gore ME and Salmon PR (1980) [30] | 150 ml tap water | 1.5 g MSG<br>3 g MSG<br>6 g MSG | No difference |
| Tarasoff L and Kelly MF (1993) [25] | Capsule formulated drinks | 1.5 g MSG (capsule)<br>3.0 g MSG (capsule)<br>3.15 g MSG (drink) | No difference |
| Yang WH et al. (1997) [31] | 200 ml of citrus-tasting beverage 5 g MSG | 1.25 g MSG<br>2.5 g MSG<br>5 g MSG | No difference for 1.25 mg MSG<br>Significant difference* for 2.5 g and 5 g MSG |
| Prawirohardjono et al. (2000) [24] | Capsule | Three possible capsules containing MSG (0.5 g MSG & 0.5 g lactose or 1.0 g MSG) | No difference |
| Geha RS et al. (2000) [32] | 200 ml of citrus-flavored beverage | 5 g MSG | Significant difference† |
| Baad-Hansen L et al. (2010) [33] | 400 ml Sugar-free soda | 75 or 150 mg/kg = 6 or 9 g/60 kg MSG | 75 mg/kg: significant difference*<br>150 mg/kg: no difference |
| Shimada A et al. (2013) [34] | 400 ml Sugar-free lemon soda | 150 mg/kg = 9 g/60 kg MSG | Significant difference* |

*Statistically significant difference was reported between placebo and MSG group ($p < 0.05$)

†Statistically significant difference was reported between placebo and MSG group ($p < 0.01$)

significant higher incidence of headache among women who received the MSG-containing broth [26, 29].

A number of studies have examined the effects of MSG when not combined with a meal; soda was the primary medium used for MSG administration. Comparable to the aforementioned MSG studies on humans, results were mixed. Studies noting statistical significant differences in headache incidence compared to a placebo include Geha et al. (5 g in 200 ml of citrus-flavored beverage), Baad-Hansel et al. (75 mg/kg or 150 mg/kg in 400 ml of sugar-free soda), and Shimada et al. (150 mg/kg in 200 ml of citrus flavored beverage) [32–34]. One of the initial studies examining the effect of MSG on headaches was conducted by Rosenblum et al. Subjects consumed 5 mg of MSG mixed with either tap water or chicken stock, had no significant statistically difference observed when compared to the placebo group [27]. Other studies, which noted no significant statistical difference among the MSG and control groups include studies by Tarasoff et al. (3.15 mg in 300 ml of soda), Gore et al. (1.5–6 g in 150 ml of tap water), and Kenney et al. (1–5 g of tomato juice) [25, 28, 30]. Yang et al. conducted a study providing participants with 1.25–5 g of MSG in 200 ml of a citrus-tasting beverage. While the 1.25 g dose was not found to have a significant statistical difference compared to the placebo, there was a difference with the 2.5 g and 5 g doses [31]. A possible explanation to how MSG could cause headaches includes an overstimulation of glutamate pathways as well as decreased γ-aminobutyric acid (GABA) levels [16].

While some of these aforementioned studies report negative outcomes, it should be noted that the amount of MSG provided is proportionally high compared to what the general public consumes. It has been previously suggested that studies using beverages as a vehicle may not have been properly blinded [35]. Additionally, none of the studies that had MSG administered with a meal reported a statistically significant difference in the incidence of headache. The average intake of food-added MSG in the USA, Europe, the UK is estimated to be 0.6 g/day [32, 36, 37]. MSG consumption in East Asia appears to be higher, with intake rates reported to be 1.1–1.6 g/day in Japan and 1.6–2.3 g/day in South Korea [38, 39]. Consumption of MSG has been linked to hypertension and obesity; however, these studies included participants who consumed higher than-average levels of MSG [40, 41]. Conversely, it is reported that MSGs dietary element may stimulate the intake of foods high in protein

and bolster several physiological activities, such as intestinal motility [42].

For individuals who worried that MSG could be a trigger for headaches or migraines, an elimination diet might serve as an effective diagnostic method. An elimination diet involves the exclusion of a suspected dietary component for a period of 4–6 weeks to analyze how it affects an individual's symptoms [43, 44]. If there's no noticeable improvement in the individual's headache symptoms after the removal of MSG, then it is likely that MSG is not a contributing factor to their headaches. It is paramount for the individual to ensure that their diet is free of MSG, as there might be various foods and/or additives containing it that they are unaware of.

## Chinese Restaurant Syndrome

In 1968, Dr. Ho Man Kwok published a constellation of symptoms occurring after the consumption of Chinese food, dubbed Chinese restaurant syndrome (CRS) [45]. Symptoms included transient facial pressure, burning, chest pain, diaphoresis, palpitations, abdominal pain, headache, and/or nausea. This ill-defined syndrome was later connected with the consumption of MSG. Dr. Kwok, who is of Cantonese descent, would later stress that CRS was in regards specifically to American Chinese food, expressing that he never observed these symptoms prior to coming to the USA [46].

Since then, various publications, tabloids, and medical journals have published the dangers of CRS in the 1970s and 1980s [47]. Kerr et al. described as possible prevalence of CRS to be 1–2% [48]. Several articles describing the condition were published in Nature [49, 50]. Zautcke et al. described CRS as a "benign, self-limited process that has an excellent prognosis for immediate and rapid recovery" [51]. Articles about similar conditions, such as Chinese-Restaurant Asthma, were subsequently published [52]. Several of the aforementioned studies above, such as ones published by Morselli and Gartattini and Gore et al. used the term CRS [26, 30].

MSG was first described as a causative agent of CRS and headaches in 1969 by Schaumburg et al., where they conducted a small double blind experiment ($n = 56$) with soup, with varying amounts of MSG, and measuring the incidence of headaches following consumption [53]. While the relation of MSG and headaches is still debated, there is some evidence for it being a trigger of headache attacks. However, it is important that patients who believe MSG to be a trigger know what foods contain it. As previously mentioned, it is a common misnomer that Chinese and other Asian cuisines are the most common sources of MSG. Many fast-food and chain restaurants frequently contain MSG in their dishes [54]. MSG use in the USA is widespread and often found in frozen vegetables, condiments, breakfast cereals, and even baby food [16]. MSG can also be found in different spices such as Sazón, a popular seasoning used in Hispanic dishes [55]. Various articles and studies have been published on CRS since the late 1960s, even though MSG had historically been used in cooking much earlier [46]. Even as recent as 2023, there are still publications that use the term CRS and give reinforcement that it is still a legitimate condition associated solely with Chinese food [56]. In light of the increase in hate crimes against people of Asian, Asian-American, and Pacific Islander ancestry since the COVID-19 Pandemic, it is vital to debunk myths surrounding certain ethnic cuisines as being a trigger for discomforting symptoms.

## Conclusion

In this narrative review, we reviewed the history of MSG, as well as the studies assessing the effects of MSG on animals and humans. While previously accused of causing various symptoms labeled as CRS, the evidence whether MSG may cause symptoms such as headache is mixed. Yet, there are several studies that demonstrate a statistically significant increase in headaches when compared against placebo. For individuals who are concerned how MSG may be a headache trigger, we recommend completing a thorough elimination diet of MSG. Further studies assessing the effects of MSG and headache are warranted.

**Author Contributions** E.A. and F.C wrote the main manuscript texts and table, and all authors review the manuscript

## Declarations

**Conflict of Interest** F.C. is a section editor for *Current Pain and Headache Reports* and an assistant editor for *Headache: The Journal of Head & Face Pain* and receives honoraria from Springer Nature and Medlink Neurology. E.A. have no conflicts of interest to report.

**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.

## References

1. Burch R, Rizzoli P, Loder E. The prevalence and impact of migraine and severe headache in the United States: updated age, sex, and socioeconomic-specific estimates from government health surveys. Headache. 2021;61:60–8.
2. Steiner TJ, Stovner LJ, Jensen R, Uluduz D, Katsarava Z. Lifting the burden: the global campaign against H. migraine remains second among the world's causes of disability, and first among young women: findings from GBD2019. J Headache Pain. 2020;21:137.
3. Burch R, Rizzoli P, Loder E. The prevalence and impact of migraine and severe headache in the United States: figures and trends from government health studies. Headache. 2018;58:496–505.

4. Yucel A, Thach A, Kumar S, Loden C, Bensink M, Goldfarb N. Estimating the economic burden of migraine on US employers. Am J Manag Care. 2020;26:e403–8.
5. Cohen F, Bobker S. From diet to disasters, lifestyle factors can affect headaches and migraine. Headache. 2023;63:712–3.
6. Evcili G, Utku U, Öğün MN, Özdemir G. Early and long period follow-up results of low glycemic index diet for migraine prophylaxis. Agri. 2018;30:8–11.
7. Bunner AE, Agarwal U, Gonzales JF, Valente F, Barnard ND. Nutrition intervention for migraine: a randomized crossover trial. J Headache Pain. 2014;15:69.
8. Aydinlar EI, Dikmen PY, Tiftikci A, Saruc M, Aksu M, Gunsoy HG, Tozun N. IgG-based elimination diet in migraine plus irritable bowel syndrome. Headache. 2013;53:514–25.
9. Di Lorenzo C, Coppola G, Bracaglia M, et al. Cortical functional correlates of responsiveness to short-lasting preventive intervention with ketogenic diet in migraine: a multimodal evoked potentials study. J Headache Pain. 2016;17:58.
10. Ramsden CE, Zamora D, Faurot KR, et al. Dietary alteration of n-3 and n-6 fatty acids for headache reduction in adults with migraine: randomized controlled trial. BMJ. 2021;374: n1448.
11. Martin VT, Vij B. Diet and headache: part 2. Headache. 2016;56:1553–62.
12. Zaeem Z, Zhou L, Dilli E. Headaches: a review of the role of dietary factors. Curr Neurol Neurosci Rep. 2016;16:101.
13. Freeman M. Reconsidering the effects of monosodium glutamate: a literature review. J Am Acad Nurse Pract. 2006;18:482–6.
14. Stańska K, Krzeski A. The umami taste: from discovery to clinical use. Otolaryngol Pol. 2016;70:10–5.
15. Yamaguchi S, Ninomiya K. Umami and food palatability. J Nutr. 2000;130:921s–6s.
16. Zanfirescu A, Ungurianu A, Tsatsakis AM, et al. A review of the alleged health hazards of monosodium glutamate. Compr Rev Food Sci Food Saf. 2019;18:1111–34.
17. Chakraborty SP. Patho-physiological and toxicological aspects of monosodium glutamate. Toxicol Mech Methods. 2019;29:389–96.
18. Conn H. "Umami": the fifth basic taste. Nutrition & Food Science. 1992;92:21–3.
19. Sauber WJ. What is Chinese restaurant syndrome? Lancet. 1980;1:721–2.
20. Onaolapo OJ, Onaolapo AY, Akanmu MA, Gbola O. Evidence of alterations in brain structure and antioxidant status following "low-dose" monosodium glutamate ingestion. Pathophysiology. 2016;23:147–56.
21. Sadek K, Abouzed T, Nasr S. Lycopene modulates cholinergic dysfunction, Bcl-2/Bax balance, and antioxidant enzymes gene transcripts in monosodium glutamate (E621) induced neurotoxicity in a rat model. Can J Physiol Pharmacol. 2016;94:394–401.
22. Swamy AH, Patel NL, Gadad PC, Koti BC, Patel UM, Thippeswamy AH, Manjula DV. Neuroprotective activity of pongamia pinnata in monosodium glutamate-induced neurotoxicity in rats. Indian J Pharm Sci. 2013;75:657–63.
23. Shivasharan BD, Nagakannan P, Thippeswamy BS, Veerapur VP. Protective effect of Calendula officinalis L. flowers against monosodium glutamate induced oxidative stress and excitotoxic brain damage in rats. Indian J Clin Biochem. 2013;28:292–8.
24. Prawirohardjono W, Dwiprahasto I, Astuti I, Hadiwandowo S, Kristin E, Muhammad M, Kelly MF. The administration to Indonesians of monosodium L-glutamate in Indonesian foods: an assessment of adverse reactions in a randomized double-blind, crossover, placebo-controlled study. J Nutr. 2000;130:1074s–6s.
25. Tarasoff L, Kelly MF. Monosodium L-glutamate: a double-blind study and review. Food Chem Toxicol. 1993;31:1019–35.
26. Morselli PL, Garattini S. Monosodium glutamate and the Chinese restaurant syndrome. Nature. 1970;227:611–2.
27. Rosenblum I, Bradley JD, Coulston F. Single and double blind studies with oral monosodium glutamate in man. Toxicol Appl Pharmacol. 1971;18:367–73.
28. Kenney RA, Tidball CS. Human susceptibility to oral monosodium L-glutamate. Am J Clin Nutr. 1972;25:140–6.
29. Zanda G, Franciosi P, Tognoni G, Rizzo M, Standen SM, Morselli PL, Garattini S. A double blind study on the effects of monosodium glutamate in man. Biomedicine. 1973;19:202–4.
30. Gore ME, Salmon PR. Chinese restaurant syndrome: fact or fiction? Lancet. 1980;1:251–2.
31. Yang WH, Drouin MA, Herbert M, Mao Y, Karsh J. The monosodium glutamate symptom complex: assessment in a double-blind, placebo-controlled, randomized study. J Allergy Clin Immunol. 1997;99:757–62.
32. Geha RS, Beiser A, Ren C, et al. Review of alleged reaction to monosodium glutamate and outcome of a multicenter double-blind placebo-controlled study. J Nutr. 2000;130:1058s–62s.
33. Baad-Hansen L, Cairns B, Ernberg M, Svensson P. Effect of systemic monosodium glutamate (MSG) on headache and pericranial muscle sensitivity. Cephalalgia. 2010;30:68–76.
34. Shimada A, Cairns BE, Vad N, Ulriksen K, Pedersen AM, Svensson P, Baad-Hansen L. Headache and mechanical sensitization of human pericranial muscles after repeated intake of monosodium glutamate (MSG). J Headache Pain. 2013;14:2.
35. Obayashi Y, Nagamura Y. Does monosodium glutamate really cause headache? : a systematic review of human studies. J Headache Pain. 2016;17:54.
36. Beyreuther K, Biesalski HK, Fernstrom JD, et al. Consensus meeting: monosodium glutamate - an update. Eur J Clin Nutr. 2007;61:304–13.
37. Rhodes J, Titherley AC, Norman JA, Wood R, Lord DW. A survey of the monosodium glutamate content of foods and an estimation of the dietary intake of monosodium glutamate. Food Addit Contam. 1991;8:663–72.
38. Maga JA. Flavor potentiators. Crit Rev Food Sci Nutr. 1983;18:231–312.
39. Lee EH, Lee DI. A study on intake level of mono sodium glutamate in Korea. J Environ Health Soc. 1986;12:75–85.
40. Insawang T, Selmi C, Cha'on U, et al. Monosodium glutamate (MSG) intake is associated with the prevalence of metabolic syndrome in a rural Thai population. Nutr Metab (Lond). 2012;9:50.
41. Shi Z, Yuan B, Taylor AW, Dai Y, Pan X, Gill TK, Wittert GA. Monosodium glutamate is related to a higher increase in blood pressure over 5 years: findings from the Jiangsu Nutrition Study of Chinese adults. J Hypertens. 2011;29:846–53.
42. Henry-Unaeze HN. Update on food safety of monosodium l-glutamate (MSG). Pathophysiology. 2017;24:243–9.
43. Meyer R, De Koker C, Dziubak R, et al. The impact of the elimination diet on growth and nutrient intake in children with food protein induced gastrointestinal allergies. Clin Transl Allergy. 2016;6:25.
44. Lozinsky AC, Meyer R, De Koker C, et al. Time to symptom improvement using elimination diets in non-IgE-mediated gastrointestinal food allergies. Pediatr Allergy Immunol. 2015;26:403–8.
45. Kwok RH. Chinese-restaurant syndrome. N Engl J Med. 1968;278:796.
46. Mosby I. 'That won-ton soup headache': the Chinese Restaurant syndrome, MSG and the making of American food, 1968–1980. Social History of Medicine. 2009;22:133–51.
47. van den Berg NWE, Neefs J, Berger WR, et al. Can we spice up our Christmas dinner? : Busting the myth of the "Chinese restaurant syndrome." Neth Heart J. 2017;25:664–8.
48. Kerr GR, Wu-Lee M, El-Lozy M, McGandy R, Stare FJ. Prevalence of the "Chinese restaurant syndrome." J Am Diet Assoc. 1979;75:29–33.

49. Himms-Hagen J. Chinese restaurant syndrome. Nature. 1970;228:97.
50. Reif-Lehrer L. Chinese restaurant syndrome. Nature. 1978;271:712–712.
51. Zautcke JL, Schwartz JA, Mueller EJ. Chinese restaurant syndrome: a review. Ann Emerg Med. 1986;15:1210–3.
52. Allen DH, Baker GJ. Chinese-restaurant asthma. N Engl J Med. 1981;305:1154–5.
53. Schaumburg HH, Byck R, Gerstl R, Mashman JH. Monosodium L-glutamate: its pharmacology and role in the Chinese restaurant syndrome. Science. 1969;163:826–8.
54. Banerjee A, Mukherjee S, Maji BK. Monosodium glutamate causes hepato-cardiac derangement in male rats. Hum Exp Toxicol. 2021;40:S359-s369.
55. Goya. Sazón ingredients. https://www.goya.com/en/products/sazon-with-saffron. In; 2023.
56. Shastri M, Raval DM, Rathod VM. Monosodium glutamate (MSG) symptom complex (Chinese restaurant syndrome): nightmare of Chinese food lovers! J Assoc Physicians India. 2023;71:11–2.