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EFFECT OF EXCESSIVE MONOSODIUM GLUTAMATE (MSG) CONSUMPTION ON BRAIN FUNCTION AND STRUCTURE: A SYSTEMATIC REVIEW IN ANIMAL MODELS

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ABSTRACT
Monosodium glutamate (MSG) is widely used as a flavor enhancer, but excessive consumption has been linked to neurotoxicity. Poor dietary intake affects neurotransmitter balance, oxidative stress levels, and long-term neuronal health. Objective: To evaluate the effects of excessive MSG consumption on brain function and structure using animal models. Method: A systematic review was conducted using PubMed, Google Scholar, and ResearchGate (2014–2024). From 1,692 initially identified articles, 8 were selected using the PICO method, focusing on MSG exposure, neurotoxicity, and structural or functional brain changes in animal models. Results: Studies indicate that MSG exposure contributes to neurotransmitter imbalances, increased oxidative stress, and neuronal degeneration, leading to cognitive impairment and behavioral alterations. Additionally, elevated tau phosphorylation and neuroinflammation were observed, potentially increasing the risk of neurodegenerative conditions such as Alzheimer's disease. The route of administration (oral vs. intraperitoneal) and duration of exposure influenced the severity of effects. Conclusion: Excessive MSG consumption negatively affects brain function and structure through mechanisms such as excitotoxicity, oxidative stress, and neuroinflammation. Further research is needed to standardize dosage thresholds, explore protective interventions, and assess long-term effects in mammalian models.

Keywords: animal models; monosodium glutamate; neurotoxicity; neurotransmitter imbalance; oxidative stress

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INTRODUCTION

The use of monosodium glutamate (MSG) as a food flavor enhancer is increasing worldwide, despite its known neurotoxic effects (Atef, Fattah, Mahmoud, Abdel-Rahman, & Salem, 2021). MSG is a sodium compound derived from glutamic acid, which serves as a crucial excitatory neurotransmitter for maintaining brain function (Zhou & Danbolt, 2014). In appropriate doses, glutamate supports cognitive function; however, excessive glutamate can lead to neuronal damage and cell death (Abdelhamid et al., 2024). Studies indicate that high doses of MSG can act as a neurotoxic or excitotoxic agent for neurons in the central nervous system (Razali, Zulkarnain, & Asrizal, n.d.). MSG excessively stimulates nerve cells to the point of damage or death, subsequently triggering the release of inflammatory mediators and increased oxidative stress (Husarova & Ostatnikova, 2013). Free glutamic acid poses potential risks due to its ability to interact with multiple receptors in brain tissues and its capacity to cross the blood-brain barrier in certain regions, such as the hypothalamus (Sriram Bs & Ravichandra V, 2019). Excessive glutamate concentrations in the brain affect cognitive function, particularly in the cerebral cortex, dentate gyrus of the hippocampus, and striatum, highlighting the essential role of this amino acid in memory formation and as an excitatory neurotransmitter (Prastiwi, Djunaidi, & Partadiredja, 2015).

Ongoing research suggests that glutamate plays a role beyond generating postsynaptic excitatory currents; it is also involved in brain development, neuronal migration and differentiation, axonal growth, neuronal survival, and neuroplasticity (Mattson, 2009). These functions are closely related to memory, cognitive abilities, and neuronal death in several diseases, such as Huntington's, Alzheimer's, and amyotrophic lateral sclerosis (Onaolapo, 2015). The toxic effects of MSG were first discovered by Olney, who observed neuronal necrosis following MSG administration in neonatal rats (Kayode, Bello, Oguntola, Kayode, & Olukoya, 2023). Necrosis and nerve damage occurred due to elevated glutamate levels in various brain regions, including the hippocampus, after high-dose MSG exposure (Lewerenz & Maher, 2015). Consequently, glutamate is considered a "double-edged sword," transitioning from a neurotransmitter to a neurotoxin (Hazzaa, Abdelaziz, Eldaim, Abdel-Daim, & Elgarawany, 2020). The accumulation of glutamate in the synaptic cleft can lead to excitotoxicity, a pathological condition in which nerve cells are damaged due to excessive stimulation by excitatory neurotransmitters (Belov Kirdajova, Kriska, Tureckova, & Anderova, 2020). Glutamate binding to its receptors allows excessive calcium ion (Ca2+) influx, triggering apoptosis and neuronal degeneration (Onaolapo, Onaolapo, Akanmu, & Olayiwola, 2016). This systematic review aims to evaluate the effects of MSG on brain function and structural damage in animal models. Specifically, this review seeks to synthesize existing literature on MSG-induced neurotoxicity, including its impact on neuronal survival, excitotoxicity mechanisms, oxidative stress, and inflammatory responses. By analyzing these effects, this study intends to provide a comprehensive understanding of MSG's potential risks to brain health and its broader implications in neurological disorders

METHOD

Literature Search and Screening

A systematic review was conducted following the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. The electronic search was performed across three databases: PubMed, Google Scholar, and ResearchGate. Relevant keywords were constructed using the PICO framework and adapted to Medical Subject Headings (MeSH) terms, including "Monosodium Glutamate OR MSG," "Neurotoxicity," "Neurotransmitter Imbalance," "Oxidative Stress," and "Animal Models." The Boolean operators AND and OR were applied to refine the search strategy, ensuring comprehensive retrieval of relevant studies. The literature search focused on studies published between 2014 and 2025, ensuring the inclusion of the most recent and relevant research on MSG-induced neurotoxicity in animal models. From the initial search, 1,692 articles were identified: 192 from PubMed, 100 from ResearchGate, and 1,400 from Google Scholar. The screening process began with duplicate removal, eliminating 56 redundant articles, leaving 1,636 articles for further assessment. Articles were screened in three stages: (1) title and abstract review to exclude irrelevant studies, (2) full-text assessment based on inclusion criteria—original research on MSG neurotoxicity in animal models, and (3) quality evaluation using SYRCLE's Risk of Bias (RoB) tool.

Selection

The selection process involved applying predefined inclusion and exclusion criteria to ensure the relevance of the studies. Articles were filtered based on the following criteria:
Inclusion Criteria:

1. Experimental studies conducted on animal models (albino rats and mice).
2. Studies investigating the effects of MSG on brain function and structure.
3. Research published in English between 2014 and 2024.

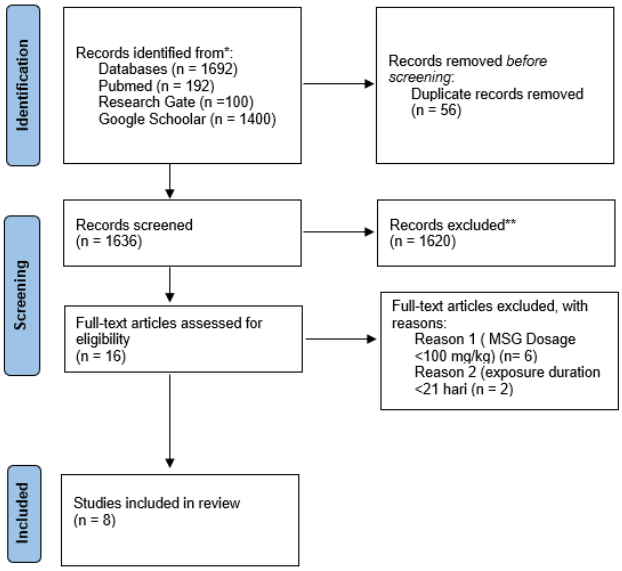
Exclusion Criteria:
1. Studies focusing on MSG effects on organs other than the brain.
2. Epidemiological studies on humans without experimental validation in animal models.
3. Reviews, conference abstracts, or studies with insufficient methodological details.

After this filtering process, 1,620 articles were excluded due to irrelevance, leaving 16 articles for further data extraction. Following an in-depth review, only 8 studies met all inclusion criteria, focusing on MSG dosage, exposure duration, and its neurotoxic effects.

Data Extraction

The data extraction process was conducted systematically to ensure consistency and accuracy. The extracted variables included:

1. Animal Model Details: Species, weight (30–50 g), and age (8–12 weeks).
2. MSG Exposure Parameters: Dose range (100–500 mg/kg to 2–6 g/kg), duration (21–120 days), and administration method (oral vs. intraperitoneal injection).
3. Cognitive and Behavioral Assessments: Tests such as T-maze, Y-maze, and water maze to evaluate memory and learning abilities.
4. Molecular and Biochemical Analysis: Immunohistochemistry and ELISA to measure IL-6 and Brain-Derived Neurotrophic Factor (BDNF); Histological Analysis using Hematoxylin & Eosin (H&E) staining and morphometric evaluations; Neurochemical Assessments for oxidative stress markers and acetylcholinesterase activity.



Picture 1. PRISMA flow diagram

RESULT

Table 1.
Summary of Studies on the Effects of Excessive MSG Consumption on Brain Function and Structure in Animal Models

Author	Dose	Duration	Route	Findings
(Sreejesh & Sreekumaran, 2018)	100 mg/kg 400 mg/kg 2 gr/kg 4 gr/kg	60 days 120 days	Oral	Oral MSG consumption reduces acetylcholinesterase levels in the cortex and hippocampus, impairing synaptic transmission and contributing to cognitive dysfunction. Higher doses and longer exposure periods lead to more severe effects.
Hazzaa et al., 2020)	4gr/kg	30 days	IP	MSG exposure increases lipid peroxidation, leading to malondialdehyde (MDA) accumulation and reduced antioxidant enzyme activity (SOD, CAT) in the brain, contributing to oxidative stress and potential neuronal

Author	Dose	Duration	Route	Findings
				damage.
(Umukoro et al., 2015)	100 mg/kg 250 mg/kg 500 mg/kg	21 days	Oral	MSG significantly increases MDA levels while decreasing reduced glutathione (GSH) levels, indicating enhanced oxidative stress. Additionally, catalase and superoxide dismutase (SOD) enzyme activity increases, suggesting an adaptive response to oxidative stress.
(Çetin Kardeşler & Başkale, 2017)	100 mg/kg 200 mg/kg	28 days	IP	MSG affects neurochemical parameters in the brain, altering dopamine and serotonin levels. Histological analysis shows neuronal degeneration in the cortex and hippocampus, along with reduced locomotor activity in experimental animals.
(Liang et al., 2024)	2 gr/kg 4 gr/kg	60 days	IP	MSG causes cellular edema and hippocampal damage, as observed in histological staining. There is an increased expression of inflammatory markers (IL-1β, TNF-α), indicating neuroinflammation.
Ajibade & Akinola (2016)	2 gr/kg 4 gr/kg 6 gr/kg	29 days	Oral	MSG induces neuronal degeneration, characterized by dendritic atrophy in hippocampal neurons. Functionally, there is a significant decline in motor skills, suggesting impaired motor control.
(Hassaan et al., 2019)	2 gr/kg	60 days	IP	MSG significantly increases tau protein phosphorylation, associated with neurofibrillary aggregate formation similar to that seen in Alzheimer's disease. Additionally, memory deficits are observed in the Morris water maze test.
Sriram & Ravichandra (2019)	500 mg/kg	60 days 120 days	Oral	MSG consumption increases interleukin-6 (IL-6) levels and decreases brain-derived neurotrophic factor (BDNF) levels, contributing to depression-like symptoms. More severe effects are observed with prolonged exposure (120 days).

DISCUSSION

Neurotoxicity of MSG and Its Mechanisms of Neural Damage

Monosodium glutamate (MSG) is a widely used food additive, but excessive consumption has been linked to neurotoxic effects through mechanisms such as excitotoxicity, oxidative stress, and neuroinflammation (Kayode, Rotimi, Kayode, Olaolu, & Adeyemi, 2020). Excitotoxicity occurs when MSG overstimulates glutamate receptors, such as NMDA and AMPA, leading to excessive Ca^{2+} influx into neurons, triggering apoptosis and neuronal degeneration (Mark et al., 2001).A study by (Hazzaa et al., 2020) demonstrated that administration of MSG at 4 g/kg for 30 days increased malondialdehyde (MDA) levels and decreased antioxidant enzyme activity (SOD and CAT) in the brain, contributing to neuronal damage due to oxidative stress. This finding aligns with (Umukoro, Oluwole, Olamijowon, Omogbiya, & Eduviere, 2015), who reported that MSG significantly increased MDA levels while reducing reduced glutathione (GSH), leading to redox imbalance in brain tissue.Additionally, research by (Onaolapo et al., 2016) indicated that long-term MSG consumption resulted in morphological changes in neurons, particularly in the hippocampus and prefrontal cortex. These findings support earlier work by Olney (1969), who reported that MSG caused neuronal necrosis in the hypothalamus of neonatal rats (Miller & Mirsky, n.d.).

Neurotransmitter Imbalance and Its Impact on Brain Function

Neurotransmitters play a crucial role in regulating cognitive function and behavior. High doses of MSG can disrupt neurotransmitter balance, leading to cognitive deficits and behavioral changes (Kouzuki et al., 2019). (Sreejesh & Sreekumaran, 2018) found that MSG administration at doses ranging from 100 mg/kg to 4 g/kg over 60–120 days decreased acetylcholinesterase levels in the cortex and hippocampus, correlating with impaired memory

and learning. Acetylcholine plays a fundamental role in cognitive processing, and any dysregulation in its levels can lead to deficits in attention, working memory, and executive function (Hasselmo, 2009). This finding is supported by (Çetin Kardeşler & Başkale, 2017), who reported that MSG affected dopamine and serotonin levels, impacting locomotor behavior and stress response. Dopamine dysregulation has been associated with motor dysfunction, reduced motivation, and impaired decision-making, while altered serotonin levels contribute to mood disorders, anxiety, and depression-like symptoms (Belujon & Grace, 2017). Additionally, (Sriram Bs & Ravichandra V, 2019) demonstrated that chronic MSG consumption also plays a significant role in neuroinflammation, further promoting neurodegeneration. Elevated glutamate levels from MSG exposure induce activation of microglia and astrocytes, leading to the excessive production of pro-inflammatory cytokines such as TNF-α, IL-6, and IL-1β. MSG consumption increased interleukin-6 (IL-6) levels while decreasing brain-derived neurotrophic factor (BDNF), contributing to depression-like symptoms (Haddad, Esmail, & Khazali, 2021). Other supporting studies include: (Hassaan et al., 2019) reported that MSG consumption caused long-term memory impairment by reducing synaptic plasticity. (Onaolapo, 2015) found that MSG inhibited GABA receptor function, leading to hyperactivity and increased anxiety in rodents exposed to high MSG doses. (Magdaleno Roman & Chapa González, 2024) showed that elevated glutamate levels due to MSG consumption caused neurotransmitter dysregulation, contributing to neurodegenerative disorders.

Neurodegeneration and Potential Risk of Alzheimer's Disease

MSG has been associated with an increased risk of neurodegeneration, including Alzheimer's and Parkinson's disease. The mechanisms underlying this association involve a combination of excitotoxicity, oxidative stress, neuroinflammation, and disrupted protein homeostasis, all of which contribute to progressive neuronal damage and cognitive decline (Ambrogini et al., 2019). (Hassaan et al., 2019) found that MSG increased tau protein phosphorylation, a hallmark of Alzheimer's disease. Hyperphosphorylated tau aggregates into neurofibrillary tangles (NFTs), disrupting microtubule stability and impairing axonal transport, which ultimately leads to synaptic dysfunction and neuronal death (Liu et al., 2021). This finding was reinforced by (Liang et al., 2024), who demonstrated that MSG caused cellular edema and neuronal degeneration in the hippocampus. These findings are consistent with (Razali et al., n.d.), who reported that long-term MSG consumption led to beta-amyloid accumulation in the rat brain, potentially accelerating Alzheimer's progression. Amyloid plaques are another defining characteristic of Alzheimer's disease, contributing to synaptic toxicity, mitochondrial dysfunction, and widespread neuroinflammation (Brokowski C, 2019). Furthermore, (Zanfirescu, Cristea, Nitulescu, Velescu, & Gradinaru, 2018) found that high-dose MSG administration disrupted calcium homeostasis and exacerbated neuroinflammation, contributing to progressive brain damage, additionally the excessive glutamate exposure due to MSG consumption exacerbates this process by promoting calcium dysregulation, leading to mitochondrial overload and neuronal apoptosis.

Route of Administration and Intensity of Neurotoxic Effects

The neurotoxic effects of MSG also depend on the route of administration and exposure duration. This discrepancy is largely attributed to differences in absorption, metabolism, systemic distribution, and bioavailability of MSG (Yang et al., 2023). A study by Ajibade & Akinola (2016) found that oral MSG administration at doses of 2–6 g/kg over 29 days caused more severe neuronal degeneration in the hippocampus and cortex, likely due to prolonged glutamate exposure, compared to intraperitoneal injection (IP). Studies show that dietary MSG consumption increases markers of oxidative stress, such as malondialdehyde (MDA), while reducing antioxidant defenses like glutathione (GSH) and superoxide dismutase (SOD)

(Boonnate et al., 2015). However, (Liang et al., 2024) reported that high-dose IP administration of MSG (2–4 g/kg) over 60 days resulted in significant hippocampal damage, as evidenced by neuronal shrinkage, nuclear fragmentation, and increased neuroinflammatory markers (IL-6, TNF-α). Another study by (Boonnate et al., 2015) demonstrated that systemic oxidative stress levels are lower in IP administration compared to oral intake. This could be due to the absence of gastrointestinal metabolism, which prevents prolonged elevation of glutamate in the blood. However, acute cellular edema and neuronal apoptosis are more pronounced in IP models, reinforcing the idea that IP administration induces a more localized but severe neurotoxic response.

CONCLUSION

The findings of this systematic review indicate that excessive consumption of monosodium glutamate (MSG) negatively affects brain function and structure in experimental animals. MSG exposure can lead to neurotransmitter imbalances, increased oxidative stress, and neuronal degeneration, contributing to cognitive impairment and behavioral changes. Additionally, increased tau protein phosphorylation and neuroinflammation observed in various studies suggest a potential elevated risk of neurodegenerative diseases such as Alzheimer's. The severity of MSG's neurotoxic effects depends on dosage, exposure duration, and the route of administration. Studies have shown that both oral and intraperitoneal administration of MSG can induce adverse effects on the brain, with variations in intensity depending on these factors. Further research is needed to establish safe dosage thresholds, explore protective interventions against neurotoxic effects, and assess the long-term consequences of MSG consumption in more complex mammalian models.

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