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Effect of monosodium glutamate (MSG) on antioxidant indices in the liver of albino rats

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Abstract
Monosodium glutamate (MSG) is a widely used food additive that enhances the palatability of foods by imparting the umami taste. Despite its extensive consumption, concerns have been raised regarding its potential toxicological effects, particularly when consumed in excessive amounts over prolonged periods. The liver, being the primary organ involved in metabolism and detoxification, is especially vulnerable to oxidative damage induced by xenobiotics.

The present study was designed to evaluate the effect of monosodium glutamate on antioxidant indices in the liver of albino rats. Adult albino rats were administered MSG orally at a specified dose for a defined experimental period. Liver tissues were excised and analyzed for lipid peroxidation and antioxidant parameters, including superoxide dismutase, catalase, glutathione peroxidase, and reduced glutathione using standard biochemical methods.

The results demonstrated a significant increase in lipid peroxidation, as evidenced by elevated malondialdehyde levels, along with a marked decrease in both enzymatic and non-enzymatic antioxidant activities in the MSG-treated group compared to controls. These findings indicate that MSG administration induces oxidative stress in hepatic tissue by disrupting the antioxidant defense system.

In conclusion, prolonged exposure to monosodium glutamate may impair liver antioxidant status and promote oxidative damage in albino rats. The study highlights the potential health risks associated with excessive MSG consumption and emphasizes the need for cautious dietary intake.

Keywords: Monosodium glutamate (MSG), Albino rats, liver, oxidative stress, lipid peroxidation, malondialdehyde (MDA), superoxide dismutase (SOD)

Introduction

Monosodium glutamate (MSG), commonly known as Ajinomoto, is the sodium salt of glutamic acid (Eweka, 2007) [7]. MSG contains approximately 78% glutamic acid, 22% sodium, and water (Samuels, 1999). Glutamate is one of the most abundant amino acids found in nature and is a major constituent of proteins and peptides in most tissues. It is also synthesized endogenously in the body and plays an essential role in human metabolism.

Glutamate is a major component of many protein-rich foods, occurring either in free or bound form. Foods of animal origin such as meat, fish, milk, and cheese, as well as vegetables such as mushrooms and tomatoes, are rich sources of glutamate (IFIC, 1994) [19]. Monosodium glutamate is widely used as a flavor enhancer, particularly in Chinese, Thai, and Japanese cuisines (FDA, 1995) [14]. When added to food, MSG imparts a taste similar to naturally occurring free glutamate, which is distinct from the four basic tastes of sweet, sour, salty, and bitter. This taste quality is referred to as umami.

As a food additive, MSG is often described on food labels as “flavoring” or “hydrolyzed vegetable protein.” Through stimulation of orosensory receptors and improvement in the palatability of food, MSG enhances appetite and may lead to increased food intake and weight gain. Despite its role in taste stimulation and appetite enhancement, several reports have suggested that MSG may exert toxic effects in humans and experimental animals (Biodun & Biodun, 1993).

MSG consumption has been associated with symptoms such as numbness, weakness, flushing, sweating, headache, and palpitations in sensitive individuals. Experimental studies have further indicated that excessive intake of MSG may induce biochemical and physiological alterations in various organs, thereby raising concerns regarding its safety, especially with prolonged consumption.

The liver plays a central role in the metabolism and detoxification of xenobiotics and is therefore highly susceptible to chemical-induced oxidative damage. Oxidative stress occurs when the generation of reactive oxygen species (ROS) exceeds the capacity of the antioxidant defense system, leading to cellular and tissue injury. To counteract oxidative stress, the liver is equipped with a well-coordinated antioxidant system comprising enzymatic antioxidants such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx), as well as non-enzymatic antioxidants including reduced glutathione (GSH).

Albino rats are widely used as experimental models for evaluating hepatotoxic and oxidative effects of food additives due to their physiological similarity to humans and well-characterized antioxidant systems. Investigating the impact of MSG on hepatic antioxidant indices in albino rats provides valuable insight into the potential risks associated with its prolonged intake.

Therefore, the present study aims to evaluate the effect of monosodium glutamate on antioxidant indices in the liver of albino rats by assessing changes in enzymatic and non-enzymatic antioxidants and lipid peroxidation markers. Such findings may contribute to a better understanding of MSG-induced oxidative stress and its implications for liver health.

Review of literature

Ursini *et al*. (1982) worked out purification from pig liver of a protein which liposomes and biomembrane from

peroxidative degradation and exhibits glutathione peroxidase activity on phosphatidyl choline hydroperoxides. Reed and Farris (1984) reported glutathione depletion and susceptibility. Ahluwalia and Malik (1989) observed effect of monosodium glutamate on serum lipids, blood glucose and cholesterol in adult male mice.

Bellisle *et al*. (1991) examined Monosodium Glutamate as a Palatability Enhancer in the European Diet. Machado *et al*. (1993) observed decreased Glucose Transporter (GLUT 4) Content in Insulin-Sensitive Tissues of Obese Aurothioglucose-And Monosodium Glutamate-Treated Mice.

Biodun and Biodun (1993) revealed A Spice or Poison? Is Monosodium Glutamate Safe for Human Consumption? Kight and Fleming (1995) revealed oxidation of Glucose Carbon Entering the TCA Cycle Is Reduced by Glutamine in Small Intestine Epithelial Cells. El-sherbiny *et al*. (1995) observed effects of difubenzuron and chlorafluazuron on the histological and histochemical structures of the ovary of albino rats. Ahluwalia *et al*. (1996) conducted studies on the effect of monosodium glutamate (MSG) on oxidative stress in erythrocyte of adult male mice.

Yang *et al*. (1997) observed the Monosodium Glutamate Symptom Complex: Assessment in a Double-Blind, Placebo-Controlled, and Randomized Study. Yu *et al*. (1997) observed effects of maternal oral administration of monosodium glutamate at a later stage of pregnancy on developing mouse fetal brain. Onakewhor *et al*. (1998) observed chronic administration of monosodium glutamate induces oligozoospermia and glycogen accumulation in Wistar rats testes.

Mitsumari *et al*. (1998) observed relationship between the development of hepatorenal toxicity and cadmium accumulation in arts.

Morris *et al*. (1998) observed reduced BAT Function as a Mechanism for Obesity in the Hypophagic, Neuropeptide Y Deficient Monosodium Glutamate-Treated Rat. Geha *et al*. (1998) observed multicenter multiphase double-blind placebo controlled study to evaluate alleged reactions to monosodium glutamate (MSG). Bergen *et al*. (1998) observed hyperphagia and weight gain after gold-thioglucose and monosodium glutamate: relation to hypothalamic neuropeptide. Y and proopiomelanocortin. Samuels (1999) reported the Toxicity/Safety of MSG: A Study in Suppression of Information, Accountability in Research. Kondo *et al*. (1999) examined scavenging mechanisms of (-)-epigallocatechin gallate and (-)-epicatechin gallate on peroxyl radicals and formation of superoxide during the inhibitory action. Bopanna *et al*. (1999) observed organotropic ultrastructural changes produced by monosodium glutamate in rats on athrogenic diet: Effect of S-Allyl Cysteine sulphoxide. Ajagbonna *et al*. (1999) observed hematological and biochemical changes in rats given extract of Colotropis procera. Adrienne S. (1999) observed the toxicity safety of MSG: a study in suppression of information.

Stoll *et al*. (1999) observed substrate Oxidation by the Portal Drained Viscera of Fed Piglets. Miśkowiak *et al*. (2000) observed longterm effect of neonatal monosodium glutamate (MSG) treatment on reproductive system of the female rat. Meldrum (2000) reported glutamate as a neurotransmitter in the brain: Review of physiology and pathology. Gul *et al*. (2000) reported cellular and clinical implications of glutathione. Brosnan (2000) [6] revealed Glutamate, at the interface between amino acid and carbohydrate metabolism. In: International Symposium on Glutamate. Nakagawa *et al*. (2000) observed effect of Chronic Administration of Sibutramine on Body Weight, Food Intake and Motor Activity in Neonatally Monosodium Glutamate-Treated Obese Female Rats: Relationship of Antiobesity Effect with Monoamines.

Pinterova *et al*. (2001) observed elevated AT1 Receptor Protein but Lower Angiotensin II-Binding in Adipose Tissue of Rats with Monosodium Glutamate-Induced Obesity. Beas-Zarate *et al*. (2001) [4] revealed changes in NMDA-receptor gene expression are associated with neurotoxicity induced neonatally by glutamate in the rat brain. Geha *et al*.2001) give review of Allergic Reaction to Monosodium Glutamate and Outcome of a Multicenter Double Blind Placebo-Controlled Study.

Gobatto *et al*. (2002) observed the monosodium glutamate (MSG) obese rat as a model for the study of exercise in obesity. Beas-Zarate *et al*. (2002) [5] observed neonatal Exposure to Monosodium L-Glutamate Induces Loss of Neurons and Cytoarchitectural Alterations in Hippocampal CA1 Pyramidal Neurons of Adult Rats. Zealand (2003) reported monosodium Glutamate, a Safety Assessment.

Hermanussen and Tresguerres (2003) [17] examined does High Glutamate Intake Cause Obesity? Kanai and Hediger (2003) reported The Glutamate and Neutral Amino Acid Transporter Family: Physiological and Pharmacological Implications. Inkielewicz and Krechniak (2003) [21] worked out fluoride content in soft tissues and urine of rats exposed to sodium fluoride in drinking water. Singh and Ahluwalia (2003) conducted studies on the effect of monosodium glutamate (MSG) administration on some antioxidant enzymes in the arterial tissue of adult male mice. Mozes *et al*. (2004) observed obesity and changes of alkaline phosphatase activity in the small intestine of 40-80-day old subjects to early postnatal overfeeding of monosodium glutamate. Diniz *et al*. (2004) [8] observed toxicity of hyper caloric diet and monosodium glutamate: oxidative stress and metabolic shifting in hepatic tissue.

Materials and Methods

The present investigation has been made on acclimatized specimens of albino rats, Rattus norvegicus (Berkernhout) under the good laboratory conditions.

Collection of Experimental Animals

The colony of albino rats were breed in the animal house of Zoology Department, School of Life Science, khandari Campus, Dr. BR Ambedkar University, Agra. Thirty five healthy male albino rats of equal size and weight 110 ± 12gm and eight week aged were selected for the present investigations. The albino rats were housed in polypropylene cages measuring 45x25x15 cm and maintain controlled temperature (25±2°c), humidity (45±10%) and proper circadian rhythm. The cages were regularly to avoid obnoxious odor and infection. They were fed with green vegetables and tap water. The albino rat were maintain under good laboratory practices (GLP) and guidelines of committee for the purpose of control and supervision of experiments on animal (CPCSEA) were followed

Experimental compound

Monosodium glutamate was selected for the present investigation. The compound is usually available as the monohydrate, a white, odorless, crystalline powder. The solid contains separate sodium cations Na+ andglutamate anions in zwitterionic form, -OOC-CH(NH+3)-(CH2)2-COO-.[37] In solution it dissociates into glutamate and sodium ions.

MSG is freely soluble in water, but it is not hygroscopic and is insoluble in common organic solvents (such as ether). It is generally stable under food-processing conditions. MSG does not break down during cooking and, like other amino acids, will exhibit a Maillard reaction (browning) in the presence of sugars at very high temperatures.

The following are alternative names for MSG:

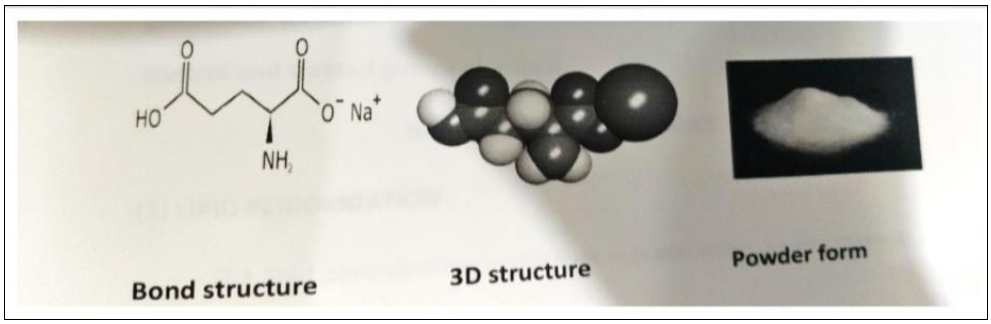
Chemical names and identifiers

- Monosodium glutamate or sodium glutamate
- Sodium 2-aminopentanedioate
- Glutamic acid, monosodium salt, monohydrate
- L-Glutamic acid, monosodium salt, monohydrate
- L-Monosodium glutamate monohydrate
- Monosodium L-glutamate monohydrate
- MSG monohydrate
- Sodium glutamate monohydrate
- UNII-W81N5U6R6U
- Flavour enhancer E621

Trade names

- Accent
- Aji-No-Moto
- Tasting Powder
- Ve-Tsin
- Sazón

Name	
Sodium 2-aminopentanedioate	
Identifiers	
CAS NUMBER	142-47-2
3D model (JSmol)	Interactive image
ChemSpider	76943
EC Number	205-538-1
E number	E621 (flavour enhancer)
Pubmed CID	85314
	DTXSID9020906
Properties	
Chemical formula	C5H8NO4Na
Molar mass	169.111g/mol (anhydrous), 187.127 g/mol (Monohydrate)
Appearance	White crystalline powder
Melting point	232 °C (450°F; 505 K)
Solubility in water	740 g/L
Hazards	
Lethal dose or concentration (LD,LC):	
LD 50 (median dose)	15800 mg/kg (oral, rat)



Dose of Experimental compound

Monosodium glutamate was used as experimental chemical. The compound was prepared in solution form and given to rats orally by gavage tube. The dose of monosodium glutamate was given to rat's 1580mg/kg body weight.

Experimental Protocol

Twenty albino rat of almost equal weight and size were divided in four group and five rats each. The group A was treated as control group and given normal water while group B, C & D were treated with monosodium glutamate for 3,7 & 15 days respectively.

Collection of Tissue

The albino rats were anaesthetized under light chloroform anaesthesia and dissected carefully. The liver was excised out carefully. The tissue was collected after 7, 15 and 30 days of monosodium Glutamate treatment. The samples were analyzed individually for each control and treated groups of animal.

Results

In the present investigations we studied the effect of monosodium glutamate for 7. 15 and 30 days on biochemical parameters *viz*. Lipid Peroxidation; Superoxide Dismutase; Chloramphenicol acetyltransferase: Glutathione-s-transferase and Glutathione reductase in liver.

Lipid Peroxidation (LPO)

a. **Control groups:** The LPO in the control group ranged from 2.48-2.82 with an average of 2.658 nmol/ml (Table-1, Fig. 1).

b. Treated groups: The LPO in the treated groups after 7 days ranged from 2.96-3.14 with an average of 3.052 nmol/ml; after 15 days monosodium glutamate treated group ranged from 3.18-3.34 with an average of 3.282 nmol/ml; after 30 days monosodium glutamate treated group ranged from 3.48-3.67 with an average of 3.568 nmol/ml (Table-1, Fig. 1).

The increased LPO is significant (P<0.05) to highly significant (P<0.01) after 7, 15 and 30 days of monosodium glutamate intoxication.

TABLE-I
Lipid Peroxidation (nmol/ml) in liver of albino rat after monosodium glutamate (MSG) intoxication

Experimental period (in days)	Number of rats	Range	Mean±S.Em.	Significance
Control	5	2.48-2.82	2.658±0.060	-
7 days MSG	5	2.96-3.14	3.052±0.034	*(P<0.05)
15 days MSG	5	3.18-3.34	3.282±0.027	**(P<0.01)
30 days MSG	5	3.48-3.67	3.568±0.035	***(P<0.001)

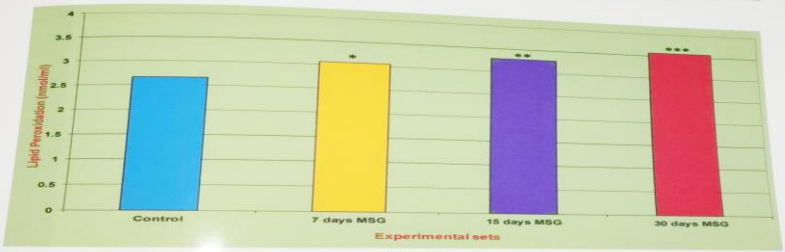


Fig. 1
Lipid Peroxidation (nmol/ml) in liver of albino rat after monosodium glutamate (MSG) intoxication

Superoxide Dismutase (SOD)

a. Control groups: The SOD in the control group ranged from 2.84-3.11 with an average of 2.988 nmol/ml (Table-II, Fig. 2).

b. Treated groups: The SOD in the treated groups after 7 days ranged from 2.61-2.81 with an average of 2.716 nmol/ml; after 15 days monosodium glutamate treated group ranged from 2.23-2.42 with an average of 2.320 nmol/ml; after 30 days monosodium glutamate treated group ranged from 2.04-2.18 with an average of 2.112 nmol/ml (Table-li Fig. 2).

The decreased SOD is significant (P<0.05) to highly significant (P<0.01) after 7, 15 and 30 days of monosodium glutamate intoxication.

TABLE-II
Superoxide Dismutase (nmol/ml) in liver of albino rat after monosodium glutamate (MSG) intoxication

Experimental period (in days)	Number of rats	Range	Mean±S.Em.	Significance
Control	5	2.84-3.11	2.988±0.046	-
7 days MSG	5	2.61-2.81	2.716±0.034	*(P<0.05)
15 days MSG	5	2.23-2.42	2.320±0.033	***(P<0.001)
30 days MSG	5	2.04-2.18	2.112±0.024	***(P<0.001)

Fig. 2
Superoxide Dismutase (nmol/ml) in liver of albino rat after monosodium glutamate (MSG) intoxication

Chloramphenicol Acetyltransferase (CAT)

a. Control groups: The CAT in the control group ranged from 17.89-18.17 with an average of 18.044 nmol/ml (Table-III, Fig. 3).

b. Treated groups: The CAT in the treated groups after 7 days ranged from 17.52-17.73 with an average of 17.624 nmol/ml; after 15 days monosodium glutamate treated group ranged from 17.28-17.45 with an average of 17.362 nmol/ml; after 30 days monosodium glutamate treated group ranged from 17.01-17.16 with an average of 17.062 nmol/ml (Table-III, Fig.3).

The decreased CAT is significant (P<0.05) to highly significant (P<0.01) after 7, 15 and 30 days of monosodium glutamate intoxication.

TABLE-III
Chloramphenicol acetyltransferase (nmol/ml) in liver of albino rat after monosodium glutamate (MSG) intoxication

Experimental period (in days)	Number of rats	Range	Mean±S.Em.	Significance
Control	5	17.89-18.17	18.044±0.046	-
7 days MSG	5	17.52-17.73	17.624±0.037	*(P<0.05)
15 days MSG	5	17.28-17.45	17.362±0.030	**(P<0.01)
30 days MSG	5	17.01-17.16	17.062±0.033	**(P<0.01)

Fig. 3
Chloramphenicol acetyltransferase (nmol/ml) in liver of albino rat after monosodium glutamate (MSG) intoxication

Glutathione-S-Transferase (GST)

a. Control groups: The GST in the control group ranged from 0.38-0.52 with an average of 0.448 nmol/min/ml (Table-IV, Fig. 4).

b. Treated groups: The GST in the treated groups after 7 days ranged from 0.24-0.31 with an average of 0.292 nmol/min/ml; after 15 days monosodium glutamate treated group ranged from 0.13-0.23 with an average of 0.182 nmol/min/ml; after 30 days monosodium glutamate treated group ranged from 0.02-0.11 with an average of 0.058 nmol/min/ml (Table-IV, Fig. 4).

The decreased GST is significant (P<0.05) to highly significant (P<0.01) after 7, 15 and 30 days of monosodium glutamate intoxication.

TABLE-IV
Glutathione-s-transferase (nmol/min/ml) in liver of albino rat after monosodium glutamate (MSG) intoxication

Experimental period (in days)	Number of rats	Range	Mean±S.Em.	Significance
Control	5	0.38-0.52	0.448±0.025	-
7 days MSG	5	0.24-0.31	0.292±0.015	**(P<0.01)
15 days MSG	5	0.13-0.23	0.182±0.017	***(P<0.001)
30 days MSG	5	0.02-0.11	0.058±0.016	***(P<0.001)

Fig. 4
Glutathione-s-transferase (nmol/min/ml) in liver of albino rat after monosodium glutamate (MSG) intoxication

Glutathione Reductase (GR)

a. Control groups: The GR in the control group ranged from 21.24-32.58 with an average of 26.89 nmol/min/ml (Table-V, Fig. 5).

b. Treated groups: The GR in the treated groups after 7 days ranged from 15.32-20.15 with an average of 18.34 nmol/min/ml; after 15 days monosodium glutamate treated group ranged from 10.11-14.12 with an average

of 12.178 nmol/min/ml; after 30 days monosodium glutamate treated group ranged from 05.46-10.01 with an average of 08.07 nmol/min/ml (Table-V, Fig. 5).

The decreased GR is significant (P<0.05) to highly significant (P<0.01) After 7, 15 and 30 days of monosodium glutamate intoxication

TABLE-V

Glutathione reductase (nmol/min/ml) in liver of albino rat after monosodium glutamate (MSG) intoxication

Experimental period (in days)	Number of rats	Range	Mean±S.Em.	Significance
Control	5	21.24-32.58	26.89±1.990	-
7 days MSG	5	15.32-20.15	18.34±0.911	**(P<0.01)
15 days MSG	5	10.11-14.12	12.178±0.068	***(P<0.001)
30 days MSG	5	05.46-10.01	08.07±0.933	***(P<0.001)

Fig. 5

Glutathione reductase (nmol/min/ml) in liver of albino rat after monosodium glutamate (MSG) intoxication

Summary

In the present study, we investigated the ability of MSG to induce oxidative toxicity in the liver of rats. Our data indicate a significant induction of lipid peroxidation in the liver of rats with supported by many studies. Our results indicate a significant alteration in the activities of lipid peroxidation, SOD, catalase, GST, GR in the livers of MSG treated rats. The alterations in enzyme activities could be a response to oxidant treatment. Monosodium glutamate (MSG) is a widely used flavour enhancer with a number of adverse effects. Earlier studies have shown the induction of oxidative stress in some organs of experimental animals after chronic administration of MSG. Some reports have also shown some alterations in hepatic glucose metabolism as a result of MSG administration.

MSG administration was associated with increased hepatic lipid peroxidation, reduced GSH levels, and decreased catalase (CAT) and superoxide dismutase (SOD) activity. Moreover, it was reported that orally administered MSG determined an increase of liver oxidative stress at a dose that extrapolated for humans.

The increase in LPO level was accompanied by a proportional decrease in the level of NPB-SH group (representing the level of glutathione). In the present study, the level of NPB-SH (representing the glutathione level) was significantly decreased in MSG-treated animals. This observation is in agreement with the report that an inverse relationship exists between lipid peroxidation and glutathione status. USG administration significantly decreased the level of glutathione-metabolizing enzymes like GR and GPx. GPx inhibits the peroxidation of membrane phospholipids by converting hydroperoxides groups in the phospholipids into corresponding alcohols.

Catalase, another potent antioxidant enzyme, especially against the superoxide radical and singlet oxygen, was also decreased significantly upon MSG administration. Catalase protects cells from the accumulation of H_2O_2 by dismutating it to form H_2O and 02, or by using it as an oxidant, in which it works as a peroxidase. Therefore, the decrease in the activity of catalase observed in the present study could be due to less availability of NADPH as MSG favors lipogenesis by increasing the level of glutamine

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