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Original Article

Effect of Monosodium Glutamate on Various Lipid Fractions and Certain Antioxidant Enzymes in Arterial Tissue of Chronic Alcoholic Adult Male Mice

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ABSTRACT

Oral ingestion of monosodium glutamate (MSG) to chronic alcoholic adult male mice at dose levels of 4 and 8 mg/g body weight for seven consecutive days caused a significant increase in lipid fractions, lipid peroxidation, xanthine oxidase, whereas the levels of superoxide dismutase, catalase, glutathione, and its metabolizing enzymes like glutathione peroxidase and glutathione reductase were significantly decreased in the arterial tissue. These observations suggested that ingestion of MSG to chronic alcoholic animals had no beneficial effect and thereby, could act as an additional factor for the initiation of atherosclerosis.

Key words: Alcohol, lipid peroxidation, monosodium glutamate, oxidative stress and atherosclerosis

INTRODUCTION

Cardiovascular diseases (CVD) are the most alarming of the many health predictions for the new millennium Worldwide. According to WHO report - 2004,[1] 16.7 million people around the globe die because of CVD each year, that is, one-third of the total deaths. Current projections suggested that by the year 2020, India will have the largest CVD burden in the World. The underlining cause of this disease is atherosclerosis, which is a slowly progressive disease that begin in childhood, manifest middle age and later.

In the Modern World, the entrance of Chinese, Japanese, and ready to serve foods like 2-minute noodles, soups, sauces, etc., all containing monosodium glutamate (MSG), has tremendously increased especially in younger generation because of its palate pleasing favorite flavor. In the present era, MSG, an inducer of oxidative stress[2-8] and alcohol, a well-known factor of atherosclerosis,[9-13] is becoming a part of daily food, especially in younger generation. There are many reports in literature that the age for the establishment of atherosclerosis has become 25 to 30 years in the present, where it was about decade back. It is an alarming situation as number of premature deaths due to coronary heart disease is increasing tremendously. So, the present work was designed to observe the effect of MSG in chronic alcoholic adult male mice to ascertain that whether MSG in the presence of alcohol could act in synergism with alcohol for the initiation of atherosclerosis or not by studying its effect on oxidative stress markers like lipid peroxidation (LPO), antioxidant enzymes like xanthine oxidase (XOD), superoxide dismutase (SOD), catalase (CAT), glutathione (GSH) and its metabolizing enzymes such as glutathione peroxidase (GPx), glutathione reductase (GR) along with various fractions of lipid.

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MATERIALS AND METHODS

Animals: Normal adult male mice (LAKA, US) weighing 25 to 30 g in body weight were procured from the animal house of Panjab University, Chandigarh, India. Animals were maintained on standard pellet diet (Hindustan Lever Ltd., Bombay) with free access to water.

Grouping: Animals were divided into four groups of six mice each and MSG at dose levels of 0, 4, and 8 mg/g body weight was given orally for seven consecutive days (that is from 31st day to 37th day of alcohol ingestion) to chronic alcoholic (30% ethanol/100 g body weight) adult male mice as follows:

Group-I: (Control): 0 mg MSG/g body weight.

Group-II: Alcohol for 37 days (30% ethanol/100 g body weight).

Group-III: Alcohol for 37 days + 4 mg MSG/g body weight (from 31st to 37th day orally).

Group-IV: Alcohol for 37 days + 8 mg MSG/g body weight (from 31st to 37th day orally).

This experimental design was approved by the Animal Experimental Ethics Committee of Panjab University, Chandigarh, and conducted according to Indian National Science Academy Guidelines for the use and care of experimental animals.

Sample preparation: After the dose period (38th day), animals were fasted overnight and sacrificed by decapitation. The arteries were removed, kept on ice, and washed with ice-cold saline. Ten percent homogenate was prepared in potassium phosphate buffer (100 mM, pH - 7.5) containing 0.15M KCl and was centrifuged at 1 000xg for 15 minutes in cold centrifuge (4°C). The supernatant stored was at 4°C and used for various biochemical assays.

Biochemical assays

Determination of Lipid fractions: The levels of total lipids, phospholipids, triglycerides, and free fatty acids were estimated by applying the methods of Frings *et al*., 1972,[14] Fiske and Suba Row, 1925,[15] and McGowan *et al*., 1983,[16] respectively. Total cholesterol levels were assayed by the methods of Zlatkis *et al*., 1953.[17]

Protein assay: The protein contents were estimated by Lowry *et al*.'s methods, 1951[18].

Lipid peroxidation: The LPO levels were assayed by measuring the pink color chromophore formed by the reaction of thiobarbituric acid (TBA) with malondialdehyde (MDA) according to the method of Beuge and Aust, 1978.[19]

Xanthine oxidase (EC 1.2.3.2): The activity of XOD was measured by the method of Fried *et al*. using nitro blue tetrazolium (NBT) which formed farmazan. The increase in the intensity of color with time was measured spectrophotometrically at 540 nm for 10 minutes.[20]

Superoxide dismutase (EC 1.5.1.1): The activity of SOD was assayed by applying the method of Kono.[21] The activity of SOD was measured by monitoring the rate of inhibition of NBT reduction. One unit is defined as the amount of enzyme which caused half-maximal inhibition of NBT reduction.

Catalase (EC 1.11.1.6): The CAT activity was estimated by the method of Luck, 1971,[22] in which decomposition of hydrogen peroxide (H_2O_2) catalyzed by this enzyme was measured by decrease in absorbance at 240 nm, taking 0.0394 $mM^{-1}cm^{-1}$ extinction coefficient and enzyme activity was expressed as nmol of H_2O_2 degraded/min/mg protein.

Reduced glutathione: GSH level was estimated by the method of Beutler *et al*., 1963,[23] using 5-5' dithiobis2-notrobenzoic acid.

Glutathione peroxidase (EC 1.11.1.7): GPx activity was measured by using H_2O_2 as a substrate by applying the method of Rotruck *et al*., 1973.[24]

Glutathione reductase (EC 1.6.4.2): The activity of GR was estimated by measuring the change in absorbance at 340 nm due to NADPH utilization and GR activity was expressed as nmoles NADPH oxidized/min/mg proteins using an extinction coefficient of 6.22 $mM^{-1}cm^{-1}$ by the method of William and Arscott, 1971.[25]

Statistical analysis: Results of biochemical analyses are presented as mean value ± standard deviation (S.D.). The difference between control and test groups was analyzed by using Student "t" test (significant difference at $P<0.05$ confidence level). Correlation between the investigated groups was performed using test ONE-WAY ANOVA (one-way variance analysis).

RESULTS AND DISCUSSION

Ingestion of alcohol significantly increased various lipid fractions that are total lipids by 9.32%, phospholipids by 15.26%, triglycerides by 17.05%, and cholesterol by 20.34% as compared with control mice (Group-1). The maximal increase was observed in cholesterol level [Table 1]. It is well reported that alcohol is oxidized to acetaldehyde by alcohol dehydrogenase containing NAD^+ and producing NADH + H^+. NADH is used to synthesize glycerol and fatty acids and hence producing hyperlipidemia.[26,27] Furthermore, NADH by entering in electron transport

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chain produces ATP.[28] The high energetic state inhibits the normal oxidation of fat in fatty acid spiral and citric acid cycle and could be the reason for elevated level of various lipid fractions in arterial tissue in alcoholic animals. The oral ingestion of MSG at the dose level of 4 mg/g body weight and 8 mg/g body weight along with alcohol further increased the level of various lipid fractions (total lipids by 16.67% and 24.79%, phospholipids by 22.77% and 33.01%, triglycerides by 23.93% and 35.35%, and cholesterols by 28.48% and 43.60%) with respect to control animals in arterial tissue and a significant increase was also found in various lipid fractions in arterial tissue of MSG-ingested alcoholic animals as compared with alcoholic animals not receiving MSG [Table 1]. Previously, we have reported that MSG induced hyperlipidemia and hypertriglyceridemia in normal mice.[2,5,29] A significant increase in various lipid fraction levels suggested that MSG along with alcohol had no beneficial effect and therefore enhances hyperlipidemia.

The activity of XOD, a superoxide-initiating enzyme, was found to be significantly increased by 9.271% in chronic alcohol group (Group-II), 16.55% in 4 mg MSG/g body weight and 21.19% in 8 mg MSG/g body weight with respect to control animals and 6.66% and 18.90% ($P<0.05$) increase was observed in group-III and group-IV, respectively, with respect to chronic alcoholic group-II [Table 2]. XOD, a highly versatile enzyme that is widely distributed from bacteria to human, exists predominantly as NAD^+-dependent xanthine dehydrogenase (XDH) that itself has no role in the start or potentiation of oxidative damage in cells. However, in many pathological conditions, XDH is converted into XOD.[30] XOD catalyses the oxidation of hypoxanthine/xanthine to uric acid and generates superoxide radical ($O_2^{\cdot-}$). H_2O_2 formed from $O_2^{\cdot-}$ could be converted into highly reactive hydroxyl radical ($^{\cdot}OH$) leading to oxidative stress as a result of oxidation of biological molecules. A significant increase in XOD activity in arterial tissue of 4 and 8 mg MSG/g body weight-ingested alcoholic animals could produce a burst of free radicals. Once $O_2^{\cdot-}$ radical is produced, then H_2O_2 and OH are continuously produced by Haber-Weiss reaction and/or Fenton type reaction.[31] Oxygen radicals might cause the LPO of biomembrane through a chain reaction. The first step is the initiation reaction, which begins by taking out hydrogen atom from polyunsaturated fatty acid by oxygen radical. The second is the propagation and the final step is termination. The extent of LPO has often been determined by the TBA test, which has also been

Table 1: Effect of oral ingestion of MSG at different dose levels (0, 4, and 8 mg/g body weight) for consecutive 7 days on various fractions of lipids in arterial tissue of chronic alcoholic adult male mice

Groups	Total lipids (mg/g)	Phospholipids (mg/g)	Triglycerides (mg/g)	Cholesterol (mg/g)
Group I (Control)	21.66 ± 2.06 [a]	11.33 ± 3.02	7.27 ± 2.67	1.72 ± 1.87
Group II (30% alcohol)	23.68 ± 2.1 (+9.32) [b]	13.06 ± 1.9 (+15.26) [b]*	8.51 ± 2.43 (+17.05) [b]*	2.07 ± 2.2 (+20.34) [b]
Group III (30% alc. + 4 mg MSG)	25.27 ± 2.3 (+16.67) [b] * (+6.71) [c]	13.91 ± 2.75* (+22.77) [b]** (+6.51) [c]	9.01 ± 2.72 (+23.93) [b]** (+5.87) [c]	2.21 ± 2.78 (+28.48) [b]** (+6.76) [c]
Group IV (30% alc. + 8 mg MSG)	27.03 ± 2.48 (+24.79) [b]** (+14.14) [c]*	15.07 ± 2.20 (+33.01) [b]*** (+15.39) [c]*	9.84 ± 2.4 (+35.35) [b]*** (+15.27) [c]*	2.47 ± 2.69 (+43.60) [b]*** (+19.32) [c]**

a: Mean ± S.D. of six observations., b: Values in parentheses represent percentage change with respect to control, c: Values in parentheses represent percentage change with respect to alcoholic animals not receiving MSG (Group-II) *$P<0.05$, **$P<0.01$, ***$P<0.001$

Table 2: Effect of oral ingestion of MSG at different dose levels (0, 4, and 8 mg/g body weight) for consecutive 7 days on LPO, XOD, CAT, in arterial tissue of chronic alcoholic adult male mice

Groups	LPO (n moles of MDA/min/mg protein)	XOD (U/mg protein)	SOD (U/mg protein)	CAT (nmol of H_2O_2 degraded/min/mg protein)
Group I (Control)	4.16 ± 0.19	0.015 ± 0.001	2.58 ± 0.017	4.23 ± 0.11
Group II (30% alcohol)	4.94 ± 0.21 (+18.75) [b]*	0.0165 ± 0.002 (+9.271) [b]	2.24 ± 0.019 (-13.17) [b]*	3.44 ± 0.07 (-18.67) [b]*
Group III (30% alc. + 4 mg MSG)	5.25 ± 0.17 (+26.20) [b]** (+6.27) [c]	0.176 ± 0.001 (+16.55) [b]* (+6.66)	2.13 ± 0.016 (-15.50) [b]* (-4.90)	3.16 ± 0.05 (-25.29) [b]** (-8.14)
Group IV (30% alc. + 8 mg MSG)	5.89 ± 0.15 (+41.58) [b]*** (+19.23) [c]*	0.183 ± 0.003 (+21.19) [b]** (+18.90) [c]**	1.92 ± 0.018 (-26.35) [b]** (-14.28) [c]*	2.90 ± 0.09 (-31.44) [b]** (-15.69) [c]*

a: Mean ± S.D. of six observations, b: Values in parentheses represent percentage change with respect to control, c: Values in parentheses represent percentage change with respect to alcoholic animals not receiving MSG (Group-II), *$P<0.05$, **$P<0.01$, ***$P<0.001$

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considered for the detection of MDA. A significant increase in LPO levels from 4.16 ± 0.19 to 5.89 ± 0.15 nmoles of MDA/min/mg protein in all the treated groups [Table 2] that was observed in the present study might lead to susceptibility of the biomembrane, which ultimately leads to tissue injury/damage.

SOD, a superoxide radical scavenging enzyme level was decreased by 13.17% ($P<0.05$) in group-II with respect to Group-I and SOD activity significantly decreased by 15.50% ($P<0.05$) and 26.35% ($P<0.01$) in Group-III and Group-IV as compared with control animals, respectively [Table 2]. A similar trend was found in MSG-ingested alcoholic mice (Group-III and Group-IV) with respect to chronic alcoholic (Group-II) animals [Table 2]. SOD is considered the first line of defense against the deleterious effect of oxygen radicals in the cells and it scavenges reactive oxygen radical species by catalyzing the dismutation of O_2^- radical to H_2O_2 and O_2. In mammals, three isozymes of SOD; CuZn-SOD, Mn-SOD, and extra cellular-SOD[32] CuZn-SOD are located primarily in the cytosol. CuZn-SOD consists of two protein subunits; each has an active site containing one Cu ion and one Zn ion. Cu ion serves as active redox site and Zn ion maintain the protein structure. Mn-SOD is located in mitochondrial matrix.[33] It has four subunits each with Mn ion. EC-SOD is present in plasma, bound to heparin sulfate ion, the surface of endothelial cells. EC-SOD is tetrameric glycoprotein, which contains Cu and Zn ion. The presence of SOD in various compartments of the our body enables it to dismutate O_2^- radicals immediately and protects the cells from oxidative damage. A significant inhibition of SOD activity in arterial tissue of 4 mg MSG/g body weight-ingested alcoholic mice and above may result in an increased flux of O_2^- radical and hence reflects the tissue damage/injury.

The activity of CAT, another potent antioxidant enzyme, especially against the O_2^- radicals and singlet oxygen, was also significantly decreased in arterial tissue by 18.67% in Group-II, by 25.29% in Group-III, and by 31.44% in Group-IV, with respect to control animals [Table 2] and a similar trend in the activity of CAT was also observed in 4 and 8 mg MSG/g body weight-treated alcoholic mice (Group-III and Group-IV) as compared with chronic alcoholic (Group-II) animals [Table 2]. CAT protects cells from the accumulation of H_2O_2 by dismutating it to form H_2O and O_2 or by using it as an oxidant, in which it works as a peroxidase. Therefore, the decrease in the activity of CAT observed in present work could be due to less availability of NADH as MSG favors lipogenesis,[2] and hence MSG along with alcohol had no beneficial effect on CAT activity to reduce lipogenesis/oxidative stress.

GSH, a tripeptide, is maintained in reduced state by an efficient GPx/GR system. GSH, a potent endogenous antioxidant, helps to protect cells from a number of noxious stimuli including oxygen derived free radicals.[34,35] A significant decrease in GSH levels might be accompanied by a significant increase in LPO level. In the present work, the level of GSH is significantly decreased by 19.66% in chronic alcoholic mice (Group-II), 28.15% ($P<0.01$) in 4 mgMSG/g body weight, and 41.99% ($P<0.001$) in 8 mgMSG/g body weight-ingested alcoholic mice with respect to control mice and a significant decrease was also observed by 18.57% ($P<0.05$) and 27.79 ($P<0.01$) in 4 and 8 mgMSG/g body weight-ingested alcoholic mice (Group-III and Group-IV) as compared to alcoholic mice not receiving MSG [Table 3]. Reduced levels of GSH confirm an increased susceptibility to oxidative damage and this observation is an agreement with the reports that inverse relationship exists between LPO and GSH status. GSH depletion of 20 to 30% can impair the cell defense against the toxic action of xenobiotic and may lead to cell injury/death.[4,36]

The activity of GPx, a selenium-containing enzyme, was found to be decreased by 13.52% (from 19.22 ± 0.68 to 16.62 ± 0.52 nmoles of GSH oxidized/min/mg protein, $P<0.05$), by 22.16% (from 19.22 ± 0.68 to 14.96 ± 0.48 nmoles of GSH oxidized/min/mg protein, $P<0.01$),

Table 3: Effect of oral ingestion of MSG at different dose levels (0, 4, and 8 mg/g body weight) for consecutive 7 days on GSH, GR, and GPx in arterial tissue of chronic alcoholic adult male mice

Groups	GSH (n moles of non-protein-SH/g tissue)	GPx (n moles of GSH oxidized/min /mg protein)	GR (n moles moles of NADPH /min/mg protein)
Group I (Control)	4.12 ± 0.21[a]	19.22 ± 0.68	21.78 ± 0.66
Group II (30% alcohol)	3.31 ± 0.32 (-19.66)[b]**	16.62 ± 0.52 (-13.52)[b]*	18.24 ± 0.49 (-16.25)[b]**
Group III (30% alc.+ 4 mg MSG)	2.96 ± 0.21 (-28.15)[b]** (- 18.57)[c]*	14.96 ± 0.48 (-22.16)[b]** (- 9.98)[c]	15.92 ± 0.76 (-26.90)[b]*** (- 12.71)[c]*
Group IV (30% alc.+ 8 mg MSG)	2.39 ± 0.24 (-41.99)[b]*** (-27.79)[c]**	12.08 ± 0.46 (-37.14)[b]*** (-27.31)[c]**	12.72 ± 0.58* (-41.59)[b]*** (-30.26)[c]***

a: Mean ± S.D. of six observations, b: Values in parentheses represent percentage change with respect to control, c: Values in parentheses represent percentage change with respect to alcoholic animals not receiving MSG (Group-II), *$P<0.05$, **$P<0.01$, ***$P<0.001$

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and by 37.14% (from 19.22 ± 0.68 to 12.08 ± 0.46 nmoles of GSH oxidized/min/mg protein, $P<0.001$) in Group-II, Group-III, and Group-IV, respectively, with respect to control mice and a significant inhibition ($P<0.01$) in GPx activity was also found by 9.98% in Group-III and 27.31% ($P<0.01$) in Group-IV as compared with alcoholic group (Group-IV, Table 3). GPx catalyses the reduction of variety of H_2O_2 (ROOH and H_2O_2) using GSH as a substrate, thereby protecting mammalian cells against oxidative stress.[37] It is well reported that low activity of this enzyme may render the tissue more susceptible to LPO damage. Accordingly, in the present work, we observed a significant decrease [Table 3] in GPx activity upon increase in LPO level [Table 2]. This observation is in accordance with the hypothesis that LPO and GPx might play a role in tissue damage.[38-40]

A significant decrease was observed in the levels of GR in different groups from 21.78 ± 0.66 to 12.72 ± 0.58 nmoles of NADPH /min/mg protein upon ingestion of 4 and 8 mg MSG/g body weight to alcoholic animals [Table 3]. The significant inhibition in the activity of GR in arterial tissue attributed to increased oxidation or decreased synthesis of GSH. The less availability of NADPH may also cause a decrease in GR activity.[41]

In conclusion, the aforementioned observations suggested that ingestion of MSG at dose levels of 4 mg/g body weight and above along with alcohol produced hyperlipidemia and increased the oxidative stress by altering the levels of oxidative stress markers like LPO, XOD, SOD, CAT, GSH, GPx, and GR in arterial tissue of adult male mice, thereby MSG along with alcohol had additive effect. Hence, MSG could act as an additional factor for the initiation of coronary artery disease/atherosclerosis.

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