Effect of sesame on liver enzymes and lipid profile inrats exposed to oxidative stress induced by Monosodium glutamate

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Abstract: The present research was conducted on twenty-four male mature Wistar rats to study effect of sesame oil on liver enzymes, lipid profile and the protective role against the oxidative stress caused by feeding monosodium glutamate which may be affect the liver. The rates were allocated in four equal groups. The 1st one used as negative control without any treatment. The 2nd group used as positive control feed on monosodium glutamate at dose rate 1.6 mg/gm bodyweight. The 3rd group received monosodium glutamate at dose rate 1.6 mg/gm body weight and treated with sesame oil at doses 4 ml/kg. body weight. The 4th group received monosodium glutamate at dose rate 1.6mg / gm bodyweight and treated with sesame oil at dose rate 8 ml/kg bodyweight.. Results indicated that oral intake of sesame oil at doses 4 and 8 ml/kg. body weight for 14 days significantly improved total cholesterol (TC), triglycerides (TG), lipoprotein fractions, decreased the elevated serum levels of liver enzymes Aminotransferase(AST) and Alanine Aminotransferase(ALT), when compared to the control positive group. Oxidative stress markers Glutathione Peroxidase (GPx), Superoxide dismutase (SOD) and Catalase (CAT) were significantly improved as compared to the control positive group. We can have concluded that the consumption of sesame oil may be have protective effects against the oxidative stress caused by consumption of monosodium glutamate (MSG) and its recommended to intake sesame oil daily for people who consume food with monosodium glutamate (MSG).
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1. Introduction

Sesame oil is a source of vitamin E and polyunsaturated fatty acids which is widely reported to reduce serum cholesterol levels in animals and humans (**Pragya, 2011 and Chandrasekaran *et al*., 2014).** Sesame oil is a source of vitamin E and it is offers better protection against increased blood pressure, hyperlipidemia and lipid peroxidation by increasing enzymatic and nonenzaymatic antioxidant **(Chandrasekaran *et al*., 2014). Hansen (2013)** said that sesame oil attributed to increase the inhibition of intestinal absorption of cholesterol, interference with lipoprotein production which increased expression of hepatic LDL receptor and their protection, leading to an increase in the removal of LDL-c from the blood and its increased degradation and catabolism of cholesterol from the body. All these events either individually or in combination lead to decrease in serum LDL-c levels, which reduced serum total cholesterol level during pretreatment **(Pooja and Priscilla, 2009).**

Several studies suggested that disorders of lipid metabolism, hyperlipidemia and obesity are associated with overproduction of oxygen free radicals **(Rehman *et al*., 2003).** The enhanced accumulation of these free radicals and dysfunction of antioxidant defense system resulted in oxidative stress **(Giao *et al.*, 2008)**. These radicals can bind covalently to the macromolecules and induce peroxidative degradation of the membrane lipids rich in polyunsaturated fatty acids, leads to the formation of lipid peroxides followed by multiple pathological changes **(Shyamala *et al*., 2003)**.

Monosodium glutamate (MSG) is a sodium compound of the amino acid (glutamic acid) which considered as the most abundant naturally non-essential amino acid. MSG contains 78% of glutamic acid, 22% of sodium and water **(Samuels 1999)**. In its pure form, monosodium glutamate is a white crystalline powder described as a “pleasant” taste. It introduced into the food supply in the 1940's **(Staywell, 2013)**. MSG has a long history of use in food as a flavor enhancer **(Geha *et al*., 2000)**. MSG has a characteristic taste called Umami “savory deliciousness” which is considered distinct from the four other basic tastes "sweet, sour, salty, and bitter" **(FSANZ, 2003)**. MSG metabolized in the liver. The liver plays an important role in the metabolism of glutamate to Arginine, some of MSG is metabolized

by conversion it into alanine in the intestinal mucosa **(Garattiini, 2000)**.

The average daily intake of MSG is estimated to be 0.3–1.0 g in industrialized countries, but it can be higher occasional, depending on the MSG content of individual food items and an individual's taste preferences **(Geha *et al.*, 2000)**. MSG is used in both home and restaurant cooking and it is a common component of Asian diets **(Walker and Lupien, 2000)**. According to FDA guidelines, MSG cannot be added to baby food, since it is considered unsafe for that age group **(Staywell, 2013)**. The mean average intake of glutamate in Saudi Arabia is 0.134 ± 0.146 mg for children aged between four and eight years. They considered as the most consumers of snacks containing glutamate **(Hassan and Al-Abbad, 2011)**. Monosodium Glutamate is known to elicit toxic effects such as, impairment in memory retention, damages in the hypothalamic neurons, alterations in mitochondrial lipid peroxidation and antioxidant status in different regions of brain and induce hyperphagia leading to obesity **(Thomas *et al.*, 2009)**. There are a number of repots describing toxic effects in human adult, as named by Chinese restaurant syndrome **(Obaseiki-ebor *et al.*, 2003)**. **(Hsu *et al.*, 2004)** evaluated the effects of parenteral sesame oil on oxidative stress and hepatic disorder induced by lipo polysaccharide. And to determine the defense mechanisms of sesame oil-associated with anti-oxidative effects in rats. Sesame oil (8ml/kg, subcutaneously) was given 3h after lipopolysaccharide, and lipid peroxide levels, hydroxyl radical, superoxide anion, the enzyme activities of superoxide dismutase and catalase as well as the levels of glutathione and nitrite were examined 6h after lipopolysaccharide (LPS). Hepatic function was assessed by determining the activities of serum aspartate aminotransferase and alkaline phosphatase. Results showed that sesame oil reduced lipid peroxidation and hydroxyl radical, but failed to affect superoxide anion. Superoxide dismutase and catalase levels were increased, but glutathione was not affected, and the levels of nitrite were reduced. Sesame oil potently decreased AST and Alkaline phosphatase (ALP) levels compared with the LPS group. The aim of this study was performed to investigate the effect of sesame oil at two doses on liver enzymes and lipid profile in infected rats by oxidative stress induced by Monosodium glutamate (MSG).

2. Material and Methods

Material:

Sesame oil: used in this research was obtained fresh from a local market (Abazeer).

Monosodium Glutamate: was obtained from a local market as white crystals, the commercial name is Ajinomoto.

Animals:

A total number of twenty four (24) adult male albino rats of Wistar strain weighed 150±30 grams were used in this study. The rats were obtained from the experimental Animal Unit of King Fahd Medical Research Center, King Abdul Aziz University, and Jeddah, Saudi Arabia.

Kits for Biochemical Analysis:

Commercial diagnostic kits for estimating serum lipid profile (total cholesterol, triglycerides and lipoprotein fractions) were obtained from Randox Laboratories, U.K. The kits for estimating liver function enzymes Serum aspartate aminotransferase (AST) activity and alanine aminotransferase (ALT) activity were obtained from Diamond Company, Hannover, Germany. Antioxidant enzymes commercial kits were purchased from Roche Diagnostic laboratories, Germany.

Basal Diet:

The basal diet was provided with standard rat chow pellets obtained from Grain Silos and Flour Mills Organization F-1005, Jeddah, Saudi Arabia, the diet consists of the following ingredients: crude protein 20%m crude fat 4.0%, crude fiber 3.5%, vitamin mix 1.0%, mineral mix 3.5%, the remained formula up to 100% cornstarch and it is energy equal 2850 kcal/kg.

Methods:

Preparation of MSG solution:

Monosodium glutamate (MSG) was dissolved in water at a concentration of 240 mg / ml per rat weighed 150 g **(Onyema *et al.*, 2006)**.

Experimental Design and Grouping of Rats:

The experiment was performed on twenty-four male mature Wistar rats. Animals were distributed randomly into four equal groups. Rats were housed in standard plastic cages at a room temperature maintained at 24± 2 °C, with fixed 12 hour lighting system.

All rats were allowed to free access to basal diet and water for one week before starting the experiment for acclimatization. After acclimatization period, the rats were allocated in to the following groups:

Group (1): rats were fed on the basal diet only, kept as a negative control group (Cont. -ve) and received oral gavage of distilled water.

Group (2): rats were fed on the basal diet, and received by gavage MSG at a dose of (**1.6 mg/g** bodyweight) and kept as a positive control group (Cont. +ve) **(Tawfik and Al-badr, 2012)**.

Group (3): rats were fed on the basal diet and received by gavage MSG at a dose of (**1.6 mg/g** body

weight) and receive by gavage sesame oil at a dose of (**4 ml/kg** bodyweight**).**

Group (4): rats were fed on experimental diet and by gavage MSG at a dose of (**1.6 mg/g** bodyweight.), and receive by gavage sesame oil at a dose of (**8 ml/kg** bodyweight)**(Hsu *et al.*, 2004 and Chandrasekaran *et al.*, 2014).**

At the end of the experimental period, all rats were fasted overnight then sacrificed. Blood samples were immediately collected from the retro orbital plexus with capillary tubes under mild ether anesthesia, into clean dried centrifuge tubes. The tubes were then centrifuged at 3000 rpm for 15 minutes. Clear serum samples were carefully separated using Pasteur pipettes, and frozen at - 20° C until biochemical analysis **(Margoni *et al.*, 2011)**. The liver was removed by careful dissection and blotted free of adhering blood immediately after sacrificing the rats.

Serum Analysis:

Determination of Serum Total Cholesterol (TC):

Serum cholesterol was determined according to the method described by **(Allain *et al.*, 1974)**, using a Spectrophotometer (Thermo Fisher Scientific Genesys, USA).

Determination of Serum Triglycerides (TG):

Concentrations of serum triglycerides were determined according to the method described by **(Trinder, 1969)**, using a Spectrophotometer (Thermo Fisher Scientific Genesys, USA).

Determination of High Density Lipoprotein Cholesterol (HDL-c):

Serum high density lipoprotein cholesterol was calorimetrically determined according to the method described by **(Lopes-Virella *et al.*, 1977)**, using a Spectrophotometer (Thermo Fisher Scientific Genesys, USA

Determination of Low Density Lipoprotein Cholesterol (LDL-c):

Serum low density lipoproteins cholesterol was calorimetrically determined according to the method described by **(Fridewald *et al.*, 1972)**. T.

Determination of Very Low Density Lipoprotein Cholesterol (VLDL-c):

Serum very low density lipoproteins cholesterol was calorimetrically determined according to the method described by **(Fridewald *et al.*, 1972).**

Determination of Liver Enzymes Activity:

Serum aspartate aminotransferase (AST) activity and alanine aminotransferase (ALT) activity were estimated enzymatically based on color reaction formation. The developed color was measured according to the method described by **(Bergmeyer *et al.*, 1978)** using a Spectrophotometer (Thermo Fisher Scientific Genesys, USA) adjusted at 505 nm wave length. The concentration was calculated by matching the reading of optical density of concentration of the sample with that of the standard solution.

Determination of Antioxidant Enzyme Activity:

The frozen liver samples were thoroughly homogenized on ice with Tri- HCL buffer solution (pH7.4) to obtain 10 % tissue homogenate. The prepared liver homogenates were used for measurement of activities of antioxidant enzymes. **(Onaolapo *et al.*, 2013)**

Determination of Catalase (CAT):

Catalase activity was measured by monitoring the decomposition of H_2O_2 at 240 nm wave length (extinction coefficient 0.00394 ± 0.0002 mM^{-1} mm^{-1}) according to the method described by **(Sinha 1972)**. CAT enzyme activity was expressed as U of catalase/mg protein (1 unit of catalase is defined as the amount of enzyme required to hydrolyze 1 µmol of H_2O_2 per min).

Determination of Superoxide Dismutase (SOD):

Superoxide dismutase (SOD) activity was assessed using a Xanthine oxidase system to generate superoxide radicals (O_2^-) as described by **(Kakkar *et al.*, 1984)**. The rate of suppression of the reduction of Nitro tetrazolium blue (NTB) by O_2^- was monitored at 550 nm wave length. SOD enzyme activity was expressed as U of SOD/mg of protein (1 unit of SOD is defined as the amount of enzyme required to inhibit the rate of reduction of NTB by 50%.

Determination of Glutathione Peroxidase (GPx):

Glutathione peroxidase(GP_x) activity was assayed by NADPH oxidation at340 nm wave length when GSSG is reduced back by glutathionereductase as described by **Paglia and Valentaine (1967)**, using cumene hydro peroxide (relatively stable organic peroxide, acts as oxidizing agent) as a substrate. Glutathione peroxidase activity was calculated using anextinction coefficient of 6.22 mM^{-1} cm^{-1} and expressedas U of GPx/mg of protein (1 unit of GPx is defined as the amount of enzyme required to convert1 nmol NADPH to $NADP^+$ per min).

Statistical Analysis:

Statistical analyses were carried out using Statistical Package for the Social Sciences (SPSS) for Windows, version 21 (SPSS Inc., Chicago, IL, USA). The obtained data were presented as means ± standard error (SE). Statistical analysis of variance between mean values of different groups was performed using one way ANOVA test followed by the least significant difference (LSD) test to determine the variance between all treatments. Differences were considered significant at $P<0.05$.

3. Results and Discussion

Effect of sesame oil at two doses on Alanine aminotransferase (ALT), Aspartate aminotransferase (AST) in rats exposed oxidative stress induced by

feeding Monosodium glutamate (MSG)are presented in Table(1).

From the same table, the rats oral intake of MSG (positive control group) had a significant (p<0.05) increase in levels of AST and ALT enzymes comparing to normal rats (negative control group) by 93.9% and 43.5 % respectively. On the other hand, oral intake of sesame oil in 8 ml/kg b.wt. had a significant (p<0.05) decreased in AST when compared to rats oral intake of MSG (positive control group) by 39.78 %. Serum Alanine Aminotransferase enzyme (ALT) and Aspartate Aminotransferase (AST) are a sensitive indicators of liver damages. **(Al-Mamary *et al*., 2002).** Therefore, the increase in the serum ALT activity might perhaps be an indication of liver damages. MSG could dissociate easily to release free glutamate. The diminution of GLU produces ammonium ion (NH^{+4}) that could be toxic unless detoxified in the liver via the reactions of the urea cycle. Thus, the possible ammonium ion overload that may occur as a result of an increased level of glutamate following MSG intake could damage the liver, consequently releasing the ALT enzyme that may lead to its observed elevation. This increase could also be explained by free radical production which reacts with polyunsaturated fatty acids of cell membrane leading to impairment of mitochondrial and plasma membranes resulting in enzyme leakage **(Tawfik and Al-badr, 2012and Akanya *et al*., 2015).**

Oral intake of sesame oil along with MSG reduced the signs of hepatocellular injury ALT and AST. Oral intake of sesame oil 4ml along with MSG could not offer effective protection against the oxidative stress induced alteration in the liver tissue membrane integrity, which changed membrane absorptivity resulting in the leakage of intracellular enzymes. The dose of 8ml is more effectively. This may be due to the free radical scavenging property of sesame oil. **(Onyema *et al*., 2006)**.

(Thomas *et al*., 2009) reported increase in plasma transaminases due to oxidative stress which induces alteration in the membrane integrity, thus changing the membrane permeability resulting in leakage of intracellular enzymes.

Vitamin E when intaketogether with MSG reduced the activities of the markers of hepatocellular injury ALT and AST. ALT and AST are also elevated in cases of injury to other organs like kidney, heart and muscles (**Bain, 2003)**. Therefore, the increased activities of ALT and AST in the serum of MSG treated animals might have resulted from the liver injury caused by the MSG-induced oxidative stress **(Onyema *et al*., 2006)**.

Table 1. Effect of sesame oil at two doses on Alanine aminotransferase (ALT), Aspartate aminotransferase (AST) inrats exposed to oxidative stress induced by feeding Monosodium glutamate (MSG)

Groups	AST(IU/L)± S.E	ALT (IU/L) ± S.E
Negative control	14.13± 1.91	9.30± 0.31
Positive control(MSG)	27.40±3.36 [a*]	13.35±0.42 [a*]
Intervention group 1(MSG+4ml sesame oil)	25.41±1.98	12.70± 0.50
Intervention group 2(MSG+8ml sesame oil)	16.50±1.02 [c*]	13.46± 0.92

Results have been represent Mean ± SEM (mean ± standard error of mean) of 6 animals per group.
ANOVA followed by LSD (p< 0.05).
[a:] Significantly different from Negative control group and positive control group.
[b]: Significantly different from positive control group and Treatment group 1 (4ml/kg body weight).
[c]: Significantly different from positive control group and Treatment group 2 (8ml/kg body weight).

Effect of sesame oil at two doses on the serum level of total cholesterol (TC) and triglycerides (TG) in rats exposed to oxidative stress induced by feeding Monosodium glutamate (MSG) are illustrated in Table (2). Results of biochemical analyses revealed that rat oral intake of MSG (positive control group) had a significant (p>0.05) increase in total cholesterol (TC) by 148.1% and triglycerides (TG) by 53.7 % compared to normal rats (negative control group).

Oral intake of sesame oil at the treated doses 4 and 8 ml/kg body weight. Significantly (p<0.05) decreased serum TC by 28.5% and 27.4 % respectively compared to rats oral intake of MSG (positive control group).

Oral intake of sesame oil in treated doses 4 and 8 ml/kg b.wt.to rat oral administration of MSG significantly (p<0.05) reduced serum TG levels by 35.9% and 34.9 % respectively when compared to rats oral intake of MSG (positive control group).

Table 2. Effect of sesame oil at two doses on the serum level of total cholesterol (TC) and triglycerides (TG) in rats exposed to oxidative stress induced by feeding Monosodium glutamate (MSG)

Groups	Total cholesterol (TC) mmol/L	Triglycerides (TG) mmol/L
Negative control	1.10± 0.04	0.67±0.01
Positive control (MSG)	2.73 ± 0.26 [a*]	1.03 ± 0.08 [a*]
Intervention group 1 (MSG+4ml sesame oil)	1.95 ± 0.04 [b*]	0.66 ± 0.006 [b*]
Intervention group 2 (MSG+8ml sesame oil)	1.98 ± 0.03 [c*]	0.67 ± 0.02 [c*]

Results have been represent Mean ± SEM (mean ± standard error of mean) of 6 animals per group.
ANOVA followed by LSD (P < 0.05).
[a]: Significantly different from Negative control group and positive control group.
[b]: Significantly different from positive control group and Treatment group 1 (4ml/kg body weight).
[c]: Significantly different from positive control group and Treatment group 2 (8ml/kg body weight).

Effect of sesame oil at two doses on the serum level of lipoprotein fraction in rats exposed to oxidative stress induced feeding Monosodium glutamate (MSG) are shown in Table (3).

There was a significant decrease ($p< 0.05$) in HDL-c in rats oral intake of MSG (positive control group) when compared to normal rats (negative control group) by 35.89 %. While oral intake of sesame oil in treated doses 4 and 8 ml/kg b.wt.to rats oral intake of MSG significantly ($p<0.05$) increase serum HDL-c levels by 170% and 162 % respectively when compared to rats oral intake of MSG (positive control group),.

There was a significant ($p< 0.05$) increase in LDL-c and VLDL-c in rats oral intake of MSG (positive control group) when compared to normal rats (negative control group) by 181.81% and 54.83 % respectively.

Oral intake of sesame oil in treated doses 4 and 8 ml/kg b.wt.to rat oral intake of MSG significantly ($p<0.05$) decrease LDL-c and VLDL-c by 56.98%. 59.67%, 52.08% and 50 % respectively, when compared to rats oral intake of MSG (positive control group).

In the current study the results revealed significant increase in serum level of total cholesterol (TC), triglycerides (TG), low density lipoprotein cholesterol (LDL-c) and very low density lipoprotein cholesterol (VLDL-c) accompanied with a significant decrease in high density lipoprotein cholesterol (HDL-c) level of control positive group as compared to the negative control group. Our results are in agreement with **(Thomas *et al*., 2009** and **Okediran *et al*., 2015)** who reported that there were an increase in the levels of cholesterol, triglycerides, and free fatty acids in plasma and tissues. MSG increases the synthesis of fatty acids and triglycerides from acetate. This could be due to the transport of acetate into the liver cell, resulting in increased substrate (acetate) availability. MSG intake also increases the synthesis of cholesterol. Another reason, that monosodium glutamate was able to increase the activities of 3-hydroxyl-3-methylglutaryl coenzyme A (HMG Co A) reductase, the rate limiting enzyme in cholesterol biosynthesis resulting in increased synthesis of cholesterol in the MSG treated rats.**(Thomas *et al*., 2009)** reported hyperlipidaemia with significantly elevated levels of serum Triacylglycerol and cholesterol in monosodium glutamate treated rats and proposed that a shift in glucose metabolism towards lipogenesis might account for the hyperlipidaemia. HDL and LDL are two of the four main groups of plasma lipoproteins that are involved in lipid metabolism and the exchange of cholesterol, cholesterol ester and triglycerides between tissues.

Table 3. Effect of sesame oil at two doses on the serum level of lipoprotein fraction in rats exposed to oxidative stress induced by feeding Monosodium glutamate (MSG)

Groups	HDL mmol/L	LDL mmol/L	VLDL mmol/L
Negative control	0.78±0.01	0.66 ± 0.07	0.31 ± 0.00
Positive control(MSG)	0.50±0.03 [a*]	1.86 ± 0.13 [a*]	0.48 ± 0.03 [a*]
Intervention group 1(MSG+4ml sesame oil)	1.35±0.04 [b*]	0.80 ± 0.12 [b*]	0.23 ± 0.00 [b*]
Intervention group 2(MSG+8ml sesame oil)	1.31±0.06 [c*]	0.75 ± 0.11 [c*]	0.24 ± 0.01 [c*]

Results have been represent Mean ± SEM (mean ± standard error of mean) of 6 animals per group.
ANOVA followed by LSD ($p< 0.05$).
[a]: Significantly different from Negative control group and positive control group.
[b]: Significantly different from positive control group and Treatment group 1 (4ml/kg body weight.).
[c]: Significantly different from positive control group and Treatment group 2 (8ml/kg body weight.).

Effect of sesame oil at two doses on liver homogenates levels of catalase, superoxide dismutase and glutathione peroxidase in rats exposed to oxidative stress induced by feeding Monosodium glutamate (MSG) are recorded in Table (4).

There was a significant ($p< 0.05$) decrease in all liver enzymes level CAT, GPX and SOD in rats oral intake of MSG (positive control group) compared to normal rats (negative control group) by 32.3 %, 39.5% and 31.8 % respectively.

Oral intake of sesame oil at treated doses 4 and 8 ml/kg body weight. Significantly ($p<0.05$) increase in liver enzyme level CAT by 23.47% and 36.36% respectively compared to rats oral intake of MSG (positive control group).

Oral intake of sesame oil at treated doses 4 and 8 ml/kg body weight.. Significantly ($p<0.05$) increase in liver enzyme level GP_X 32.04% and 57.39% respectively compared to rats oral intake of MSG (positive control group).

Oral intake of sesame oil at treated doses 4 and 8 ml/kg body weight.. Significantly ($p<0.05$) increase in liver enzyme level SOD by 24.87% and 32.33 %respectively compared to rats oral intake of MSG (positive control group).

Significant alterations (decrease) were observed in SOD activities in hepatic tissues of positive control group. It is affirm that the radical superoxide has important role in the hepatic metabolic shifting induced by MSG administration. SOD catalysis the intake of superoxide radical (O^{2-}) to hydrogen peroxide (H_2O_2) and protects various organs to O^{2-} damage. GSH-Px catalysis the reduction of H_2O_2 to water **(Abuja and Albertini, 2001).** It has been demonstrated that the rate of $O2^-$ and H_2O_2 production by the mitochondria in liver, heart and kidney, was directly related to metabolic rate. In our study it was observed that hepatic SOD antioxidant activity was inversely related to, at least, one of the metabolic changes in hepatic tissue of MSG rats (**Diniz *et al.*, 2004)**.

Sesame oil has a peculiar characteristic due to the presence of the natural antioxidants sesamol, sesamolin and Vitamin E as tocopherol, which gives it high oxidative stability **(Corso *et al.*, 2010)**. Vitamin E when co-administered with MSG significantly reduced lipid peroxidation (LPO) and the activities of superoxide dismutase (SOD) and catalase (CAT) and significantly increased in the tissues. The recycling of vitamin E contributes to glutathione depletion. When vitamin E is depleted due to its oxidation, glutathione reduces the tocopheroxyl radicals to tocopherol, and is itself oxidized. In the presence of an exogenous supply of vitamin E, glutathione is maintained in its reduced state (**Hess, 1993).** It is the most effective chain-breaking antioxidant within the cell membrane, where it protects the membrane fatty acids from LPO **(Singh *et al.*, 2004**).

Table 4. Effect of sesame oil at two doses on liver catalase, superoxide dismutase and glutathione peroxidase in rats exposed to oxidative stress induced by feeding Monosodium glutamate (MSG)

Groups	CAT (U/g tissue)	GP ^{x} (U/g tissue)	SOD (U/g tissue)
Negative control	1.30±.022	49.48±.99	2.95±.072
Positive control (MSG)	0.88±.022 ^{a*}	29.90±.81 ^{a*}	2.01±.047 ^{a*}
Intervention group 1 (MSG+4ml sesame oil)	1.15±.015 ^{b*}	39.48±.75 ^{b*}	2.51±.040 ^{b*}
Intervention group 2 (MSG+8ml sesame oil)	1.20±.017 ^{c*}	47.06±1.23 ^{c*}	2.66±.049 ^{c*}

Results have been represent Mean ± SEM (mean ± standard error of mean) of 6 animals per group.
ANOVA followed by LSD ($P< 0.05$).
[a]: Significantly different from Negative control group and positive control group.
[b]: Significantly different from positive control group and Treatment group 1 (4ml/kg body weight).
[c]: Significantly different from positive control group and Treatment group 2 (8ml/kg body weight).

Recommendations:

The present study recommends the following:

1. People who prefer snacks and food contain MSG should include sesame oil in their daily diet, to alter side effects of monosodium glutamate (MSG) and enhance antioxidant system in the body.
2. Adding sesame oil to salad dressings to increase antioxidants and protect against oxidation damages.
3. Including sesame oil into kid's meals who consume snacks contains MSG to decrease possible damages.
4. Patients suffering from hypercholesterolemia and /or cardiovascular disease (CVD) advised to consume sesame oil because of its health beneficial effect on serum total cholesterol (TC), triglycerides (TG) and LDL-c.

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