

Effect of sesame oil on feed intake, body weight gain, and histopathological changes in rat liver exposed to oxidative stress of Monosodium glutamate

Maha, A. Hijazi; Amani, A. Alrasheedi and Nahed A. Hareeri

Department of Food and Nutrition, Faculty of Home Economics, King Abdulaziz University, Saudi Arabia.
mhijazi@kau.edu.sa

Abstract: The present research was conducted on twenty-four male mature Wistar rats to study the protective role of sesame oil against the oxidative stress caused by feeding monosodium glutamate. The rats were allocated in four equal groups. The 1st one used as negative control without any treatment. The 2nd group used as positive control feed monosodium glutamate at dose rate 1.6 mg/gm body weight. 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 body weight and treated with sesame oil at dose rate 8 ml/kg body weight. The feed intake (FI), body weight gain (BWG %), feed efficiency ratio and histopathological changes of liver in rats after 14 days were studied. Oral intake of Monosodium glutamate (MSG) at dose 1.6mg/g to rats caused a significant ($p<0.05$) increase in body weight gain (BWG %), Daily feed intake (DFI) and feed efficiency ratio (FER) in all experimental groups compared to control group. Also, the liver relative weights were increased significantly in all experimental groups compared to negative control group. Oral intake of sesame oil in the treated doses 4 and 8 ml/kg body weight caused a significant ($p<0.05$) decreased in BWG%, DFI, FER and liver relative weight when compared to (positive control group). These effects are associated with amelioration of degenerative histopathological changes in liver tissue induced by MSG. The most effective concentration of sesame oil as a hepatoprotective agent (8 ml/kg. body weight).

[Maha, A. Hijazi; Amani, A. Alrasheedi and Nahed A. Hareeri. **Effect of sesame oil on feed intake, body weight gain, and histopathological changes in rat liver exposed to oxidative stress of Monosodium glutamate.** *J Am Sci* 2017;13(2):1-9]. ISSN 1545-1003 (print); ISSN 2375-7264 (online). <http://www.jofamericanscience.org>. 1. doi:[10.7537/marsjas130217.01](https://doi.org/10.7537/marsjas130217.01).

Key words: Sesame oil, Monosodium glutamate, oxidative stress, histopathological changes of liver.

1. Introduction

Sesame oil is derived from the plant species *Sesamum indicum L.*, and contains several antioxidants including sesamin, tocopherol, sesamol and sesaminol which give it high oxidative stability (Corso *et al.*, 2010). Sesame oil enhances hepatic detoxification of chemicals, reduces the incidence of chemically induced mammary tumors, and protects against oxidative stress (Hirose *et al.*, 1992), which is involved in the pathogenesis of endotoxin intoxication (Hsu *et al.*, 2004). Oxidative stress may be caused by reactive oxygen intermediates (ROI). ROI, including singlet oxygen, nitric oxide (NO), hydrogen peroxide, and free radicals, all of which are important mediators of cellular injury and play a putative role in oxidative stress in endotoxin intoxication.

Sesame oil has a peculiar characteristic by the presence of the natural antioxidants sesamol, sesamol and gamma tocopherol, which gives it high oxidative stability (Corso *et al.*, 2010). Sesamin, a major lignin in sesame oil, is known to have many biological activities, especially protective effects against oxidative damage in the liver (Nakai *et al.*, 2003). Sesame oil is a source of vitamin E and it offers better protection against increased blood pressure, hyperlipidemia and lipid peroxidation by

increasing enzymatic and nonenzymatic antioxidant (Chandrasekaran *et al.*, 2014).

Vitamin E is an antioxidant and correlated with lowering cholesterol levels. Sesame oil contains many nutrients, which promote good health. Two of its most important nutrients are vitamin E (an antioxidant) and polyunsaturated fats (Pragya, 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 reports describing toxic effects in human adult, as named by Chinese restaurant syndrome (Obaseiki-ebor *et al.*, 2003). In 1995, the Federation of American Societies for Experimental Biology (FASEB), who commissioned by the United States Food and Drug Administration (FDA) undertake a review on adverse reactions of MSG. The report announced the following symptoms that considered representative of the acute, temporary, and self-limited reactions to oral ingestion of MSG (FASEB 1995), burning sensations in the back of the neck, forearms, chest; facial pressure/tightness; chest pain; headache; nausea; palpitation; numbness in back of neck,

radiating to arms and back. Tingling, warmth, weakness in face, temples, upper back, neck and arms. Bronchospasm (observed in asthmatics only); drowsiness; Weakness (FSANZ 2003).

(Yin *et al.*, 2015) evaluated the effects of dietary glutamate and aspartate supplementations on diquat-induced oxidative stress in piglets.

(AL-Mosaibih, 2013) evaluated the effect of MSG and acrylamide on the liver tissues of adult rats. 40 adult male wistar rats Results showed changes in body weight a highly significant change between mean values of body weight gain in experimental group's specific group 2 which administrated MSG compared to normal rats.

(Okediran *et al.*, 2015) evaluated the effects of MSG on the liver and lipid profile of adult male Wistar rats, blood samples were collected. Results showed significant increase in Serum ALT and AST activity in groups treated with MSG compared to normal group also there were significant increase in the plasma cholesterol and LDL-cholesterol in MSG treated rats compared to normal rats, while there were reduction in both triglyceride and HDL –cholesterol compared to normal rats.

(Onyema *et al.*, 2006) evaluated the protective role of vitamin E against MSG induced hepatotoxicity and oxidative stress in rats. Results showed a significant increase in the liver weight of the rats administrated with MSG which might cause inflammation in liver tissues. Significant increase in the level of serum ALT and AST were shown, which might be a marker of hepatocellular injury. Significant increases in the activities of SOD and Catalase in liver of the experimental animals were shown. MSG generated the reactive oxygen species (ROS) that caused oxidative stress.

The aim of this study was performed to investigate the effect of sesame oil at two doses on feed intake (FI), body weight. gain (BWG %), feed efficiency ratio and histopathological changes in rats exposed to oxidative stress induced by Monosodium glutamate (MSG).

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.

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%, 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 gavages' of distilled water.

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

Group (3): rats were fed on experimental diet and received by gavage MSG at a dose of (1.6 mg/gb. wt.), and receive by gavage sesame oil at a dose of (4 ml/kgb. wt.) (Chu *et al.*, 2012).

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

Biological Evaluation: Determination of Feed Intake (FI), Body Weight Gain Percent % and Feed Efficiency Ratio (FER):

Daily feed intake (FI) per group was calculated throughout the experimental period (14 days). The biological values of different diets were assessed by the determination of body weight gain percent (BWG %) which was calculated at the end of the experimental period as well as feed efficiency ratio (FER) was calculated twice a week, according to the method of (Chapman *et al.*, 1959). Using the following equations:

$$\text{Body weigh gain percent (BWG \%)} = \frac{\text{Final body weight} - \text{Initial body weight}}{\text{Initial body weight}} \times 100.$$

Feed efficiency ratio was calculated as follows:

$$\text{Feed efficiency ratio (FER)} = \frac{\text{Gain in body weight (g)}}{\text{Feed consumed (g)}}$$

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. The liver was washed with cold saline solution and dried between two filter papers then weighed and they saved for the histopathological examination. Calculation of the relative organ weight was done according to the following equation:

$$\text{Organ relative weight} = \frac{\text{Organ weight}}{\text{Animal final bodyweight}} \times 100.$$

Liver was kept in 10% neutral buffered formalin pending for the histopathological examination.

Histopathological Examination:

Specimens from the halves of liver was taken immediately after weighed the organ of the rats and immersed in 10% neutral buffered formalin. The fixed specimens were then trimmed, washed, and dehydrated in ascending grades of alcohol, then cleared in xylene, and stained with Hematoxylin and Eosin (H&E) and examined microscopically according to (Bancroft and Gamble, 2008).

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 \pm 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 feed intake (FI), body weight gain (BWG %), feed efficiency ratio in rats exposed to oxidative stress induced by feeding Monosodium glutamate (MSG) are presented in Table (1). Oral intake of Monosodium glutamate (MSG) at dose 1.6mg/g to rats caused a significant ($p < 0.05$) increase in body weight gain (BWG %), Daily feed intake (DFI) and feed efficiency ratio (FER) in all experimental groups compared to control group. Oral intake of sesame oil in the treated doses 4 and 8 ml/kg body weight caused a significant ($p < 0.05$) decrease in BWG%, DFI, FER compared to (positive control group).

Concerning Feed intake (FI) and body weight gain percent (BWG%) rats group oral administration of MSG only control positive revealed significant increase in final weight and weight gain percent compared to control negative group. These results were in agreement with (Tawfik, Al-badr, 2012; Kumbhare *et al.*, 2015) who confirmed our results.

In the present study, the capability of sesame oil at two doses 4 - 8 ml/kg to decrease body weight gain compared with positive group were investigated. Moreover, the beneficial effect of antioxidant intake against MSG consumption with respect to body weight observed in the present study confirms previous results obtained by (Onyema *et al.*, 2006) who concluded that feeding rats with antioxidants such as Vitamin E could play an important role as a radical scavenger. By scavenging the radicals that contributed to oxidative stress, vitamin E could help in reducing inflammation against the toxic effects of MSG.

Table 1. Effect of sesame oil at two doses on feed intake (FI), body weight gain (BWG %), feed efficiency ratio in rats exposed to oxidative stress induced by feeding Monosodium glutamate (MSG)

| Groups | Body weight gain (%) | Mean of Daily feed intake (DFI) | feed efficiency ratio (FER) |
|---|-----------------------------------|---------------------------------|-----------------------------------|
| Negative control | 27.88 \pm 2.29 | 25.52 | 0.24 \pm 0.016 |
| Positive control (MSG) | 49.97 \pm 2.21 ^{a*} | 58.36 ^{a*} | 0.42 \pm 0.019 ^{a*} |
| Intervention group 1 (MSG+4ml sesame oil) | 39.86 \pm 2.69 ^{b*} | 17.54 ^{b*} | 0.28 \pm 0.014 ^{b*} |
| Intervention group 2 (MSG+8ml sesame oil) | 36.63 \pm 3.60 ^{c*} | 17.62 ^{c*} | 0.26 \pm 0.010 ^{c*} |

Results have been represent Mean \pm SEM (mean \pm 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 relative weight in rats exposed to oxidative stress induced by feeding Monosodium glutamate (MSG) are illustrated in Table (2). The results showed that the rats oral intake of MSG (positive control group) had a significant ($p < 0.05$) increase in the relative liver weight as compared to normal rats (negative control group) by 14.19 % as depicted.

Oral intake of sesame oil in the treated doses 4 and 8 ml/kg body weight caused a significant ($p < 0.05$) decrease in the RLW when compared to rats oral intake of MSG (positive control group) by 17.66% and 16.50 % respectively.

This result was consistent with previous study reported by (Tawfik and Al-badr, 2012) who demonstrated that intake MSG at two doses in rats induced liver inflammatory lead to increase in liver weight. (Onyema *et al.*, 2006) reported that relative liver weight were significantly increased after oral intake MSG by gavage at dose of 0.6 mg/g body weight. The increase in the liver relative weight might as a result of attributed to increase in oxidative damages that could led to inflammation of liver tissues (Park *et al.*, 2000 and Onyema *et al.*, 2006). A significant increase in the liver weight of the rats was observed after intake of MSG. Thus, could be attributed to an increase in activity of inflammatory agents that could have resulted to inflammation of liver tissues which reported by (Park *et al.*, 2000).

Table 2: Effect of sesame oil at two doses on liver relative weight in rats exposed to oxidative stress induced by feeding Monosodium glutamate (MSG)

| Groups | relative Liver weight % |
|---|---------------------------|
| Negative control | 4.51± 0.05 |
| Positive control (MSG) | 5.15± 0.04 ^{a*} |
| Intervention group 1 (MSG+4ml sesame oil) | 4.24 ± 0.05 ^{b*} |
| Intervention group 2 (MSG+8ml sesame oil) | 4.30± 0.07 ^{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).

Histopathological Examination:

The histopathological examination of liver sections of rats in the negative (normal) control group

showed normal histological structure of hepatic lobule as illustrated in (Figure 1).

In the present study section taken from control rat liver and stained by H&E showed it was covered by thin capsule figure 1 (a, b, c, d). The hepatocytes, the liver cells, are arranged in the form of cords or plates radiating from the central veins. The cells are polygonal in shape. They have central rounded nuclei with well-delined nucleoli. The cytoplasm is homogeneously stained and has basophilic spots. Blood sinusoids between the cells have thin walls figure 1 (c). Portal area contain branches of portal vein (PV), Hepatic artery (HA) and Bile duct (BD) which known as portal triad. They are surrounded by scanty connective tissue.

Oral intake of MSG to rat's results in alteration of histology of liver in the form of swollen of hepatocytes that lost the in well defined out lines. The cytoplasm showed marked unstained regions. The nuclei locked small, dark and degenerated blood sinusoids are compressed figure (1). The changes were also observed mean portal region, which showed vascular dilation, congestion and fibrosis figure (2).

Oral intake of sesame oil (SO) 4ml/kg to rats oral intake of MSG resulted in protection of hepatocytes against histological degeneration changes induced by MSG. Hepatocytes looked normal with rounded nuclei, both mean central vein figure (3) or portal region figure (4).

Oral intake of sesame oil (SO) 8 ml/kg to rats oral intake of MSG resulted in significant protection hepatocyte looked normal with normal shape nuclei. Portal components (PV, BD, HA) are also looked similar to control (H& E) stain.

Liver is the largest gland in the mammalian body. The hepatocytes have metabolic functions that deal with very essential processes such as detoxification, deamination, transamination, and removal of ammonia in the form of urea, conversion of carbohydrates and proteins into lipids, oxidation of fatty acids and much other process (Johnson 1995 and Nelson and Cox 2000). The metabolism of xenobiotics largely takes place in the liver. The by-products of such metabolism sometimes become more toxic than the initial substance. This could lead to liver injury and the emergence of liver diseases (Ishak *et al.*, 1991). The liver contains considerable amounts of polyunsaturated fatty acids that are prone to damage by free radicals through oxidative stress (Tawfik and Al-badr (2012).

Results of our study showed that intake of sesame oil by gavage at two doses 4 or 8 ml/kg body weight to hepatotoxic rats showed a significant decrease in relative liver weight compared with negative control group, this finding was in consistent

with that reported by **Lim *et al.*, (2007)**. These results could be explained on the basis that intake of sesame oil had a beneficial effects on MSG damages liver steatosis and damage probably due to its antioxidant nutrient (Vitamin E as tocopherol, sesamin, sesamol and sesaminol) contents and high radical-scavenging capacity.

The dose 8ml /kg was more effective in protecting MSG induced toxicity compared to 4ml/ kg. Vitamin E reduced the MSG-induced increase in the liver weight, and vitamin E was more effective especially with higher intake of MSG, possibly via its action as a radical scavenger. Vitamin E could help in

reducing inflammation (**Onyema *et al.*, 2006**) and (**Tawfik and Al-badr, 2012**). Accumulation of polyunsaturated fatty acids (**PUFAs**) in the bloodstream caused by overinvesting sesame oil may lead to the neutralization of the antioxidative effects of sesame oil. The primary fatty acids in sesame oil are monounsaturated oleic acid and polyunsaturated linoleic acid. Sesame oil contains a high level of PUFAs which are important targets of free radical attack. PUFAs may be attacked by free radicals and oxidized into lipid peroxides that reported by (**Hsu *et al.*, 2008**).

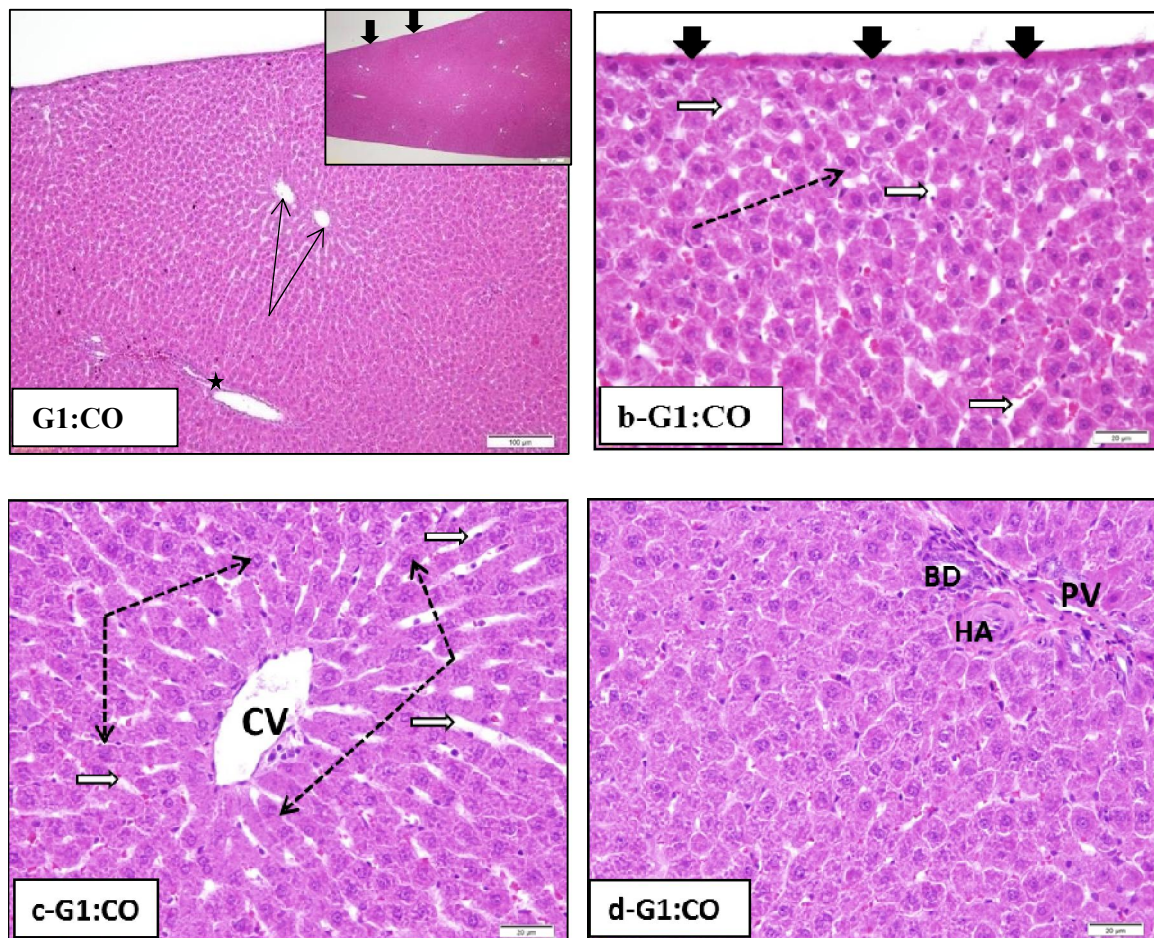


Fig (1) sections from G1. a: control rat liver showing the surface capsule (thick black arrows), central veins (thin arrows).

G1.b: Magnified photo of capsule (thick black arrows) with normal underlying hepatocyte cell cords (dotted arrows), cell cords are separated by thin wall blood sinusoids (white arrows).

G1.c: normal hepatocyte cell cords (dotted arrows) radiating the central vein (CV). Notice also the thin wall blood sinusoids (white arrows).

G1.d: portal region showing branches of portal vein (PV), bile duct (BD) and hepatic artery (HA). Hepatocytes also are of normal appearance.

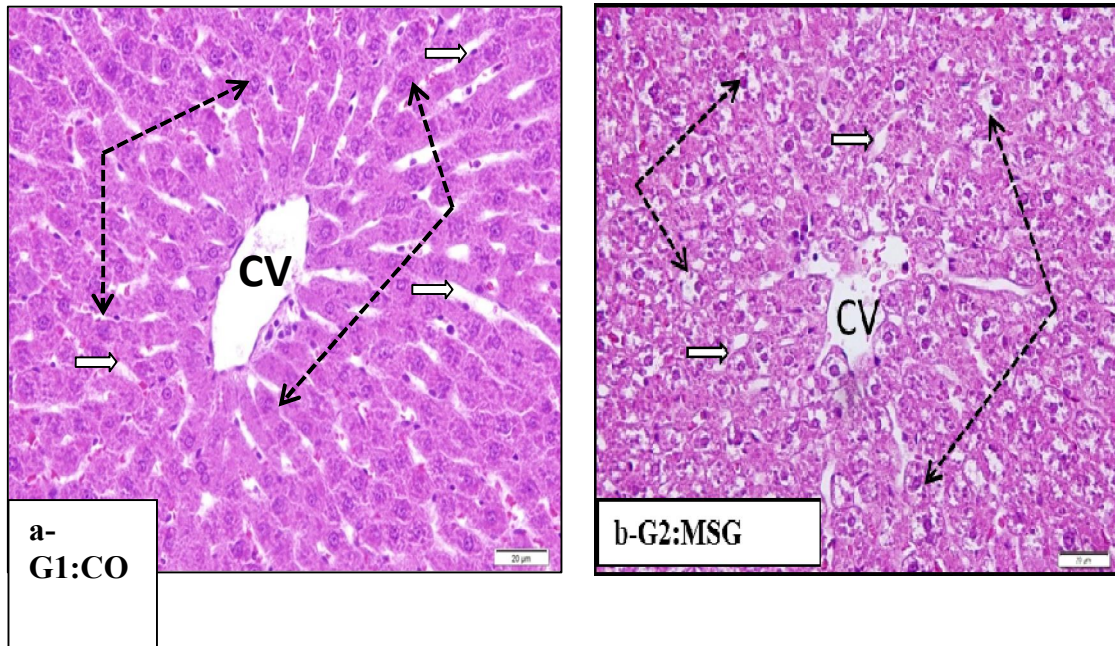


Fig (2) sections from rat liver from **G1: a. control** showing normal structure described in figure 4.18. **b. G2: (MSG)** showing irregular central vein (CV), marked changes in hepatocytes. The cells lost their regular outlines and swollen. The cytoplasm showed unstained regions indicating degeneration. The nuclei are small, dark and degenerated (dotted arrows). Blood sinusoids between cell cords are narrow and compressed by swollen cells (white arrows).

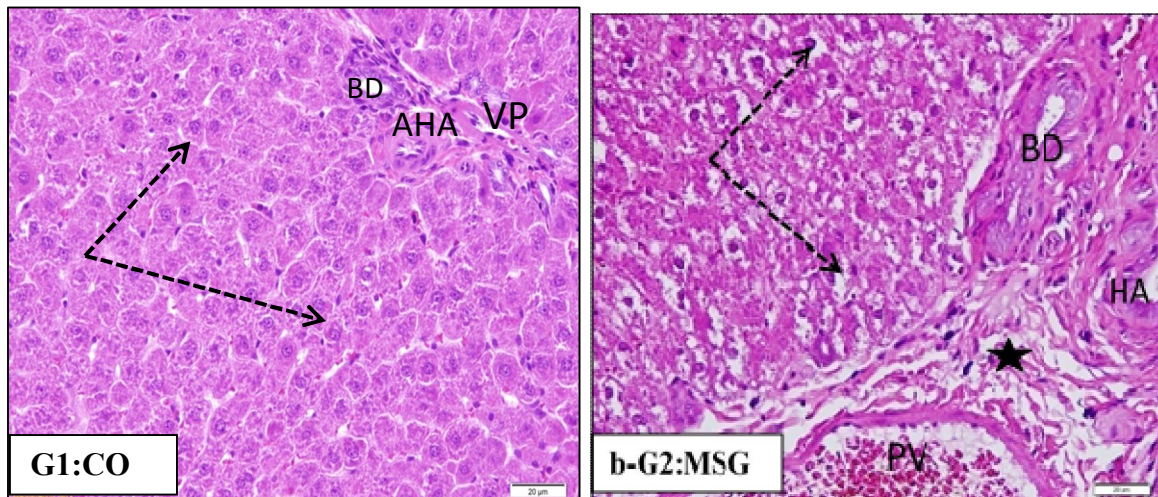


Fig (3) sections from rat liver near portal area showing portal triad, portal vein (PV), bile duct (BD) and hepatic artery (HA) of a. G1: control with normal structure. b. G2: MSG showing congestion of portal vein (PV). fibrosis and inflammatory cells (star). Deformity of bile ducts (BD) hepatocytes showed unstained degenerated cytoplasm (dotted arrows).

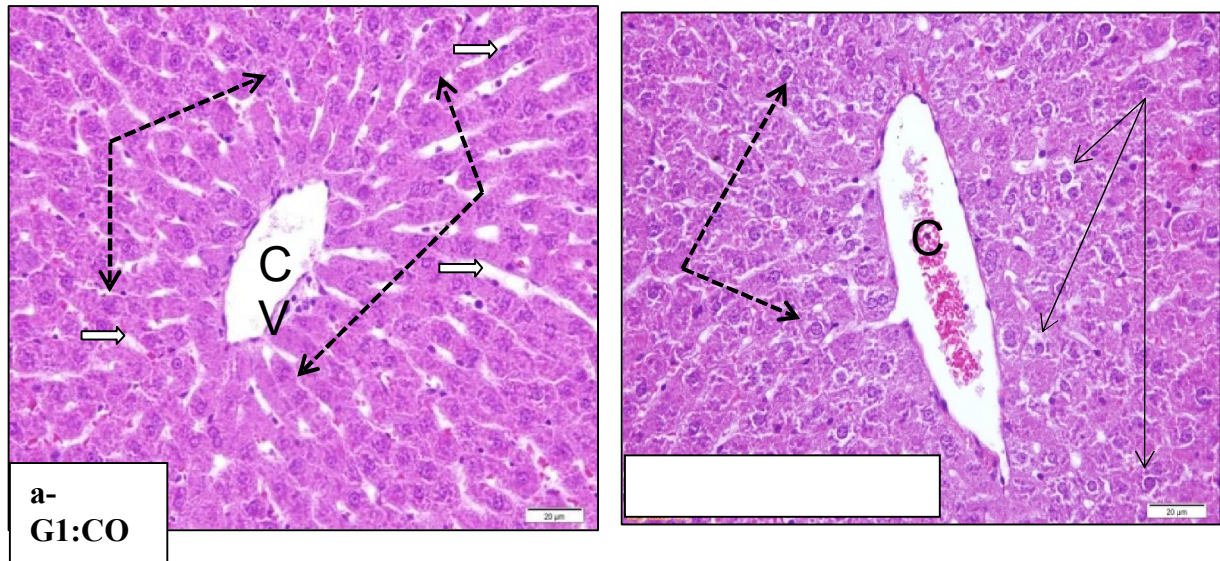


Fig (4) sections from rat liver from a. G1: control showing normal structure described in figure4.20. b. c. G3:(MSG+4ml SO) Moderate protection was observed against changes seen in non treated group. Most hepatocytes looked potentially normal (dotted arrows). Few cells still showed unstained cytoplasm and dark nuclei (thin black arrows).

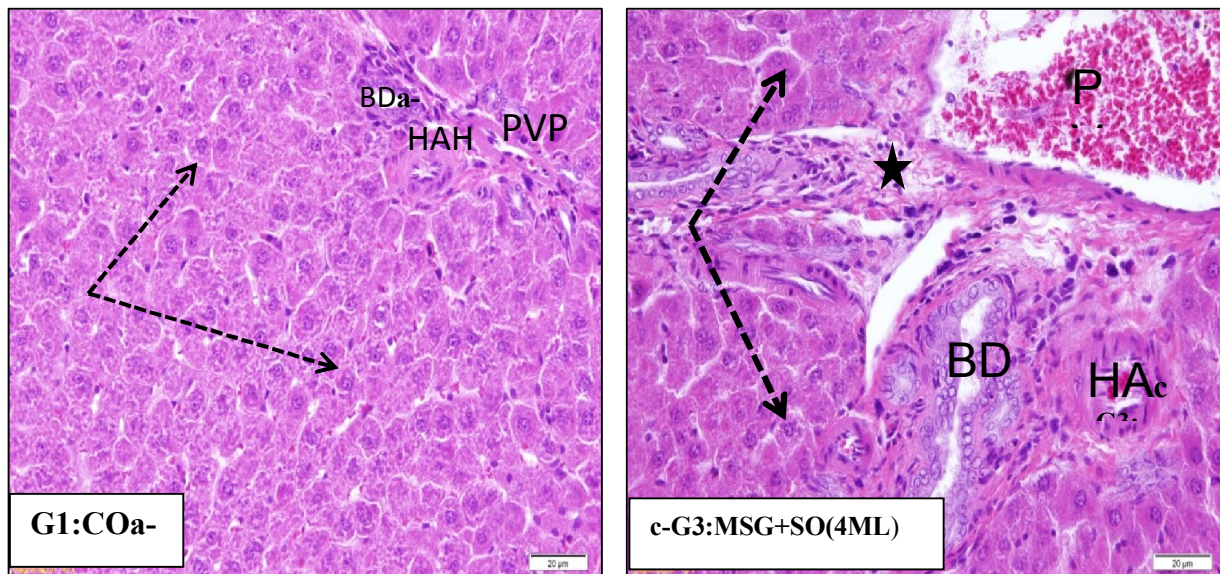


Fig (5) sections from rat liver near portal area showing portal triad, portal vein (PV), bile duct (BD) and hepatic artery (HA) of a. G1: control with normal structure. C. G3. MSG+4ml SO showing protection against MSG induced hepatocyte degeneration. Cells looked normal similar to control. Portal vein still showed congestion and surrounded by scanty fibrosis (star).

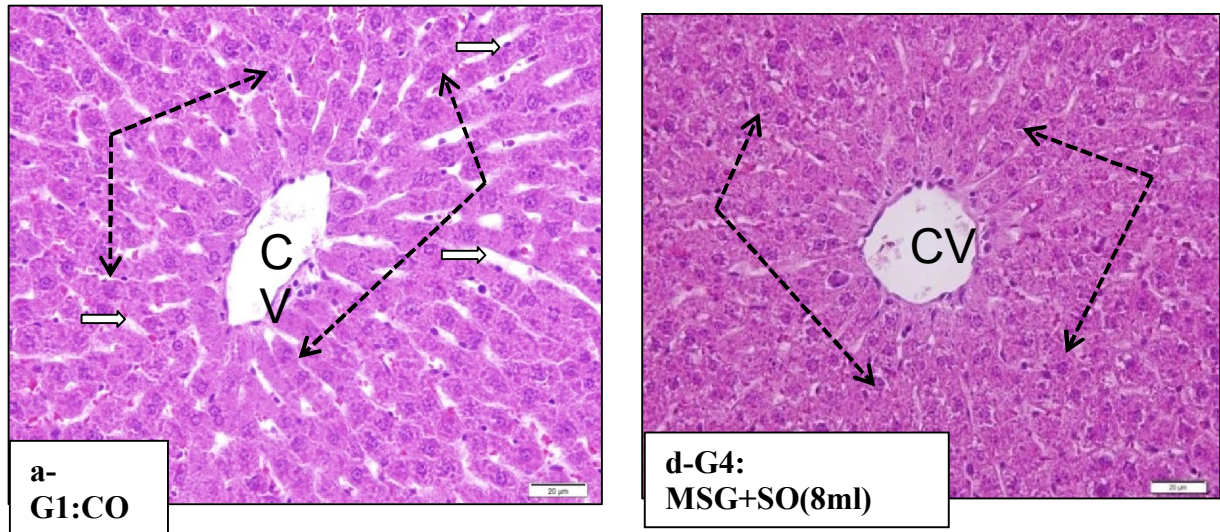


Fig (6) sections from rat liver from a. G1: control showing normal structure described in figure 4. 22. d. G4: (MSG+8ml SO) showing marked protection of hepatic tissue from MSG induced degenerative changes. Hepatocytes and central vein are much similar to those of G1 control (H & E stain).

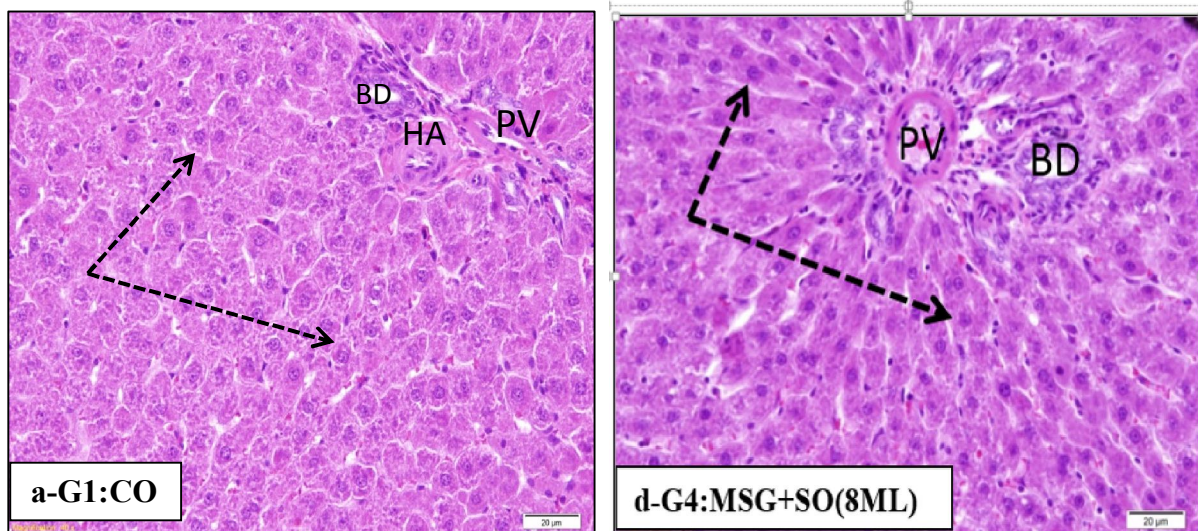


Fig (7) sections from rat liver near portal area showing portal triad, portal vein (PV), bile duct (BD) and hepatic artery (HA) of a. G1: control with normal structure. d. G4. MSG+8ml SO showed significant protection where hepatocytes looked normal with normal shape nuclei (dotted arrows). Portal components (PV, BD, HA) are also looked similar to control (H&E stain).

Conclusion:

The present study concluded that oral intake of MSG with Sesame oil at two concentrations for 14 days to rats improves body weight gain, feed intake, and feed efficiency ratio. These effects are associated with amelioration of degenerative histopathological changes in liver tissue induced by MSG. The most effective concentration of sesame oil as a hepatoprotective agent (8 ml/kg. body weight).

References

1. AL-Mosaibih, M. (2013) Effects of Monosodium glutamate and Acrylamide on the liver tissue of adult Wistar rats. *Life Science Journal*, 10: 35-42.
2. Bancroft, J. and Gamble, M. (2008) *Theory and practice of histological techniques*, Philadelphia: Churchill Livingstone/Elsevier.
3. Chandrasekaran, R. V., Hsu, D. Z. and Liu, M. Y. (2014) Beneficial effect of sesame oil on Heavy Metal Toxicity. *Journal of Parenteral and Enteral Nutrition*, 38: 179-185.

4. Chapman, D., Castillo, R. and Campbell, J. (1959) Evaluation of proteins in foods. *Canadian Journal of Biochemistry and Physiology*, 37: 679-686.
5. Chu, P., Hsu, D., Wang, L. and Liu, M. (2012) Sesame oil protects against Hepatic Injury via the inhibition of Neutrophil activation in Thioacetamide-Treated rats. *Acta Phytopathologica et Entomologica Hungarica*, 47: 203-211.
6. Corso, M. P., Klen, M. F., Silva, E. A., Filho, L. C., Santos, J. N., Freitas, L. S. and Dariva, C. (2010) Extraction of sesame seed (*Sesamum indicum L.*) oil using compressed propane and supercritical carbon dioxide. *The Journal of Supercritical Fluids*, 52: 56–61.
7. FASEB, Federation of American Societies for Experimental Biology. Raiten, D. J., Talbot, J. M. and Fisher, K. D. (Guest eds) (1995) Executive summary from the report: analysis of adverse reactions to monosodium glutamate (MSG). *The Journal of Nutrition*, vol. 125: 2891-2906.
8. FSANZ, Food Standards Australia New Zealand (2003) Monosodium Glutamate. A safety assessment. Technical Report Series No. 20. Canberra: Food Standards Australia New Zealand.
9. Hirose, N., Doi, F., Ueki, T., Akazawa, K., Chijiwa, K., Sugano, M., Akimoto, K., Shimizu, S., Yamada, H. (1992) Suppressive Effect of Sesamin against 7, 12-dimethylbenz Anthracene Induced Rat Mammary Carcinogenesis. *Anticancer Res.*, 12: 1259–1265.
10. Hsu, D. Z., Chiang, P. J., Chien, S. P., Huang, B. M. and Liu, M. Y. (2004) Parenteral sesame oil attenuates oxidative stress after endotoxin intoxication in rats. *Toxicology*, 196: 147-153.
11. Hsu, D. Z., Chien, S. P., Li, Y. H. and Liu, M. Y. (2008) Sesame oil does not show accumulatively enhanced protection against oxidative stress-associated hepatic injury in septic rats. *Journal of parenteral and Enteral Nutrition*, 32: 276-280.
12. Ishak, K. G., Zimmerman, H. J. and Ray, M. B. (1991) Alcoholic liver disease: pathologic, pathogenetic and clinical aspects. *Alcohol. Clin. Exp. Res.*, 15: 45-66.
13. Johnson, P. (1995) *Clinical Biochemistry: Metabolic and Clinical Aspects*, Edited by: Marshall, W. and Bangert, S., The Assessment of Hepatic Function and Investigation of Jaundice, New York: Churchill Livingstone.
14. Kumbhare, V., Gajbe, U., Singh, B. R., Reddy, A. K. and Shukla, S. (2015) Histological and Histochemical changes in liver of adult rats treated with monosodium glutamate: A light microscopic study. *International Journal of Pharmacy and Pharmaceutical Sciences*, 4: 898-911.
15. Lim, J. S., Adachi, Y., Takahashi, Y. and Ide, T. (2007) Comparative analysis of sesame lignans (sesamin and sesamol) in affecting hepatic fatty acid metabolism in rats. *British Journal of Nutrition*, 97: 85–95.
16. Margoni, A., Perrea, D. N., Vlachos, I., Prokopaki, G., Pantopoulou, A., Fotis, L., Kostaki, M. and Papavassiliou, A. (2011) Serum Leptin, Adiponectin and Tumor Necrosis Factor- α in Hyperlipidemic Rats with/without Concomitant Diabetes Mellitus. *The Feinstein Institute for Medical Research*, 17: 36-40.
17. Nelson, D. and Cox, M. (2000) *Lehninger Principles of Biochemistry*, New York: Worth Publishers.
18. Obaseiki-ebor, E., McGhee, E. and Shankel, D. (2003) Improved detection of the genotoxic and mutagenic potentials of a food condiment A-one (monosodium glutamate). Presented at the Fourth International Conference of the Pan-African Environmental mutagen Society (PAEMS) in Dar EL Diafa- Ain Shams University, Cairo, Egypt, 63-63.
19. Okediran, B. S., Olurotimi, A. E., Rahman, S. A., Michael, O. G. and Olukunle, J. O. (2015) Alteration in the lipid profile and liver enzymes of rats treated with monosodium glutamate. *Sokoto Journal of Veterinary Sciences*, 12: 42-46.
20. Onyema, O., Farombi, E., Emerole, G., Ukoha, A. and Onyeze, G. (2006) Effect of Vitamin E on monosodium glutamate induced Hepatotoxicity and oxidative stress in rats. *Indian Journal of Biochemistry and Biophysics*, 43: 20-24.
21. Park, J. S., Choi, M. A., Kim, B. S., Han, I.S., Kurata, T. and Yu, R. (2000) Capsaicin Protects against Ethanol-Induced Oxidative Injury in the Gastric Mucosa of Rats. *Life Science*, vol. 67: 3087-3093.
22. Pragma, T. (2011) Sesame oil Benefits. from: <http://www.buzzle.com>, Access date 2015.
23. Tawfik, M.S. and Al-Badr, N.(2012): Adverse Effects of Monosodium Glutamate on Liver and Kidney Functions in Adult Rats and Potential Protective Effect of Vitamins C and E, *Food and Nutrition Sciences*, 3(5): 651- 659.
24. Thomas, M., Sujatha, K.S. and George, S. (2009) Protective effect of *Piper longum Linn.* on monosodium glutamate induced oxidative stress in rats. *Indian J. Exp. Biol.*, 47: 186-192.
25. Yin, J., Liu, M., Ren, W., Duan, J., Yang, G., Zhao, Y., Fang, R., Chen, L., Li, T. and Yin, Y. (2015) Effects of dietary supplementation with glutamate and aspartate on diquat-induced oxidative stress in piglets. *PLoS One*, 10: e 0122893.