Available online on www.ijpqa.com
International Journal of Pharmaceutical Quality Assurance 2019; 10(2); 272-279
doi: 10.25258/ijpqa.10.2.10

ISSN 0975 9506

Research Article

Physiological and Histological Study to the Effects of Monosodium Glutamate in Laboratory male Rats and the protective role of vitamin E

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Received: 7th Mar, 19; Revised: 28th Apr, 19, Accepted: 3rd Jun, 19; Available Online: 25th Jun, 2019

ABSTRACT

The current study was conducted at the Department of Biology, College of Science, Wasit University to investigate physiological and histological effect monosodium glutamate in laboratory male rats, preventive role of vitamin E. This study was carried out in Laboratories of College of Science, Wasit University, AL- Shaheed Dr. Fairooz Hospitals, from November 2017 to April 2018.The study included twenty-four and divided into four groups (six rats per group). the first group severe as a control group orally dosed with distilled water, and treated the second group (100 mg/kg b.w. Monosodium glutamate for 30 days, and the third group were dosed orally 200 mg/kg of b.w. for 30 days, either The fourth group were dosed with a mixture of Monosodium glutamate 200mg/kg and vitamin E 100 mg/kg of body weight for 30 days. after the trial period has been sacrificing animals for testing and chemical standards physiological and histological. As are result of by exposure to Monosodium glutamate in blood serum are negatively biochemical whole height of the level of serum cholesterol, triglycerides, Low-density lipoprotein, very- low density lipoprotein, liver enzymes, AST, ALT, ALP, creatinine level, urea serum, further more we noticed a decrease in high density lipoprotein. The preventive treatment resulted in vitamin E 100mg/kg b.w. with Monosodium glutamate 200 mg/kg b.w. ($p \leq 0.05$) in body weight and relative weights of organs (liver and kidney). We noticed a higher moral when treatment with vitamin E with Monosodium glutamate 100 mg/kg in high- density lipoprotein, while serum cholesterol level decrease, triglycerides, Low-density lipoprotein, very- low density lipoprotein. And liver and kidney functions have improved by low Enzyme AST, ALT, ALP, creatinine and urea serum level. Histological examination revealed that the liver and kidneys, of rats exposed 100, 200 mg/kg of Monosodium glutamate has been adversely affected by exposure to Monosodium glutamate. Whereas, the histological of the liver of animals treated with vitamin E with Monosodium glutamate natural pictures showed improvement. These results demonstrate that MSG toxic effects on the liver and kidney tissue. The more toxic than salt rate too. The study recommends to avoid using MSG as food additives and food for animals because of the toxic effects of this salt.

Keywords: Monosodium glutamate, kidney, liver, vitamin E, histology.

INTRODUCTION

Monosodium Glutamate (MSG) is one of the world's most extensively used food additives which is ingested as part of commercially processed foods. As a flavor enhancer, MSG increases the rapidity of food (Briks, 2005). Thousands of chemicals are being used recently in our new high tech food ready to eat like, Japanese, Chinese, packaged and tinned food. Most food additives act as either preservatives or flavor enhancers (Dixit et al., 2014). So, Monosodium Glutamate (MSG or E 621) is one of the most widely used food additives all over the world which is a part of many commercial foods like bouillon cubes, frozen food, canned food, snack chips, soups, salad dressing as a flavor enhancer[3]. Studies providing the evidence of MSG toxic effects have raised the increasing interest in MSG intake as flavor enhancer. Neurotoxic effects in brain, obesity and metabolic defects. "Chinese restaurant syndrome" and detrimental effects on sex organs are the most discussed in the connection with MSG intake, specially the effect of MSG in liver, kidney, testis and epididymis which might cause increased oxidative stress in the tissues of animals. The increase in LPO level could be due to increased level of glutamine following MSG administration. Glutamine could initiate the LPO by changing the redox potential of cells to favor the lip genesis. Lipid peroxidation is an autocatalytic mechanism leading to oxidative destruction of cellular membranes. However, the cellular antioxidant system comprises integral antioxidants like GSH and different free radical scavenging antioxidant enzymes against oxidative injury. Among the antioxidant enzymes, SOD and GST are the first line of defiance against oxidative injury. The inhibition of the anti-oxidant system may cause the accumulation of H2O2 or products of its decomposition[4]. SOD is the primary step of the defiance mechanism in the antioxidant system against oxidative stress by catalyzing the demutation of 2 superoxide radicals (O2.) into molecular oxygen (O2) and

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Table 1: The protective role of vitamin E from MSG exposure on male rats in some serum liver enzymes.

Parameter GROUPS	AST IU/I	ALT IU/I	ALP U/I
CONTROL	162.466 ± 3.03 b	60.483 ± 7.77 b	263.500 ± 2.01 b
MSG 100mg/kg	227.666 ± 6.07 ab	89.050 ± 1.31 a	463.166 ± 8.55 a
MSG 200 mg/kg	267.666 ± 8.50 a	84.883 ± 1.15 ac	423.333 ± 5.50 a
MSG 200mg/kg+ vitamin E 100 mg/kg	184.516 ± 1.90 b	75.283 ± 10.00 c	337.666 ±5.15 c
LSD	83.150	13.766	74.166

N=6

The numbers represent the mean ±standard Deviation.

Different letters represent significant difference at ($p \leq 0.05$).

hydrogen peroxide (H2O2.), as it prevents further generation of free radicals[5]. Glutathione plays a critical role in protecting cells from oxidative stress and xenobiotic. It can react non-enzymatically with super-oxide, nitric oxide, hydroxyl radicals, and peroxynitrite[6]. Thus, it functions directly as a free radical scavenger. The physiological role of vitamin E is to react with free radicals in cell membranes and other lipid milieu, thereby preventing polyunsaturated fatty acids (PUFA) from being damaged by lipid peroxidation[7]. This antioxidant activity is important to maintain membrane integrity and takes place in all cells in the body. Vitamin E can influence cellular responses to oxidative stress by modulating signal-transduction pathways because vitamin E is an important antioxidant agent[8]. Vitamin E has also been suggested to improve immune function in elderly and prevent cognitive impairment, but the evidence is inconsistent. Vitamin E (vit E) is a well-known antioxidant and has been shown to protect various tissues against the damage caused by ROS[9]. In addition, many studies have indicated that vit E may be of benefit in chronic diseases, such as cardiovascular disease, cancer and cataract, probably through antioxidant mechanism. Vitamins have indispensable role in almost all biochemical reactions and they are ideal antioxidants able to increase tissue protection from oxidative stress due to their easy, effective and safe dietary administration in a large range of concentrations[10]. Vitamin E (α- Tocopherol [(α-Toc]) is the primary membrane bound, lipid-soluble, chain-breaking antioxidant that protects cell membranes against lipid peroxidation[11].

MATERIALS AND METHODS

Experimental Design

The present study was performed twenty-four adult male rats weighing about 175–250 grams and 10–14 weeks' old were used in the current study. Animals were kept under normal temperature (22-25 °C), and controlled lightening. Animals were randomly divided into four equal groups each group consisted of 6 adult male rats as in the following:

Control group: orally dosed with distilled water.

Group 2: orally dosed with monosodium glutamate 100mg/kg b. w. daily for 30 days.

Group 3: orally dosed with monosodium glutamate 200mg/kg b. w. daily for 30 days.

Groub 4: orally dosed with monosodium glutamate 200mg/kg b. w.+ vitamin E100mg/kg b. w. daily for 30 days.

All animals were weighted at the beginning of the experiment and at the end of experiment, then animals were sacrificed.

Animal Sacrifice and Sample Collection

All the animals of the experiments were sacrificed at the end of the experiments. The rats before sacrifice were first weighed and then anaesthetized by placing them in a closed Becker containing cotton sucked with diethyl ether anesthesia. The abdominal cavity was opened up through a midline abdominal incision to take the samples which include:

Blood sample

Blood sample were collected via cardiac puncture according to the method of[12]. Then the blood sample were drops directly from the heart by using 5 ml disposable syringe. the blood put in plane tube until it was coagulated, then centrifuged (3000 rpm for 15 minutes) to obtain the serum. The serum samples separated into many epndrofe tubes to avoid repeated thawing. All tubes were stored at (-20c) until they were analyzed.

The organs weight

The organs (Liver and Kidney) were immediately removed and separated from the surrounding tissues and lipid, weighed with an electric balance. The two kidney of each male rat were measured and the average value of each of two organs was considered as one measurement. The organs were fixed in 10% formalin for histological examination.

Monosodium Glutamate

Commercial monosodium glutamate Purity 99% (Chinese salt) manufactured by Ajinomoto co.INC. Tokyo, Japan was used in the present study.

Vitamin E

Commercial vitamin E manufactured Germany was used in the present study.

Biochemical analysis

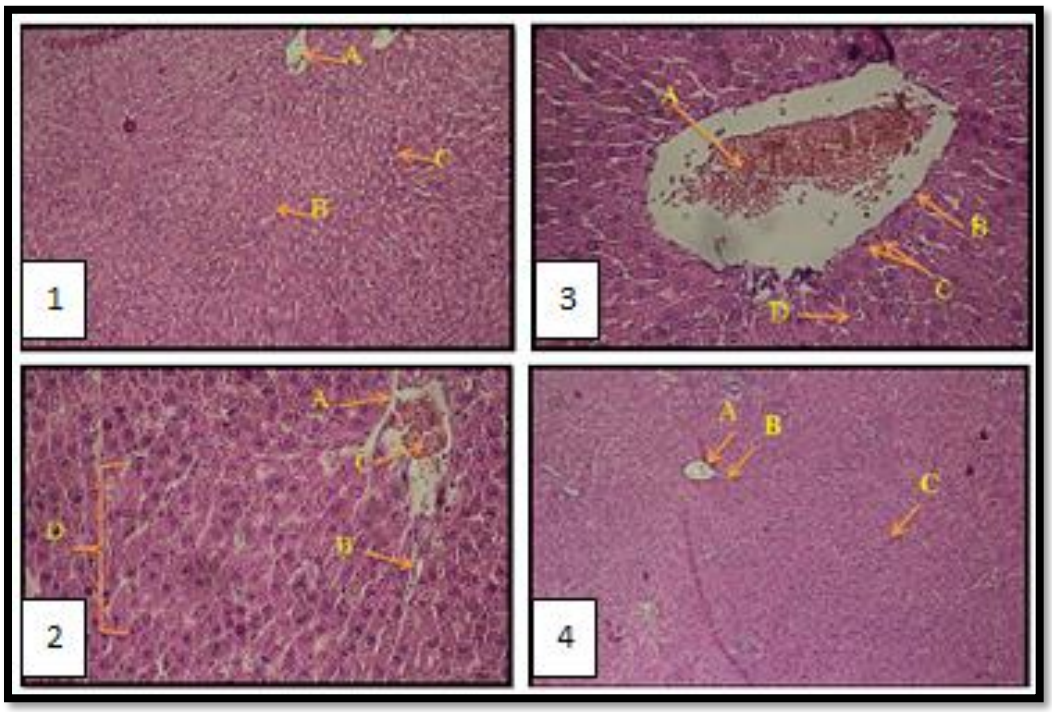


Figure 1: Light micrograph of liver control rat notice central vein (A) liver cords(B) hepatocytes (C). (H&E,4x).
Figure 2: Light micrograph of liver of rat exposed to monosodium glutamate 100mg/kg for 30 days' notice the dilated of central vein(A), disturbance of liver architecture, the necrosis (D),) sinusoidal spaces (C) and degeneration of hepatocytes with verging shapes and sizes (B) (H&E,10X).
Figure 3: Light micrograph of liver of rat exposed to monosodium glutamate 200mg/kg for 30 days' notice increasing change the architecture, the congestion central vein (A) effected endothelium lining(B), highly dilated space of sinusoidal spaces (C)inflammatory cells (D) pycknotic nuclei (D) (H&E 10X).
Figure 4: Light micrograph of liver of rat exposed to monosodium glutamate 200mg/kg and vitamin E 100mg /kg for 30 days' notice central vein (A) liver code (B), sinusoidal space(C) (H&E,4X).

Lipid profile assay: The serum lipid profile (TC, TG, HDL, LDL, and VLDL) was estimated as follow. The serum (TC) level was estimated by kits were supplied by Bio Systems Reagents Instruments, (Spain), (TG) was assay by kit supplied by Linear. (Spain) (LDL) and (VLDL)[13] calculated from the equation): LDL -C = Total cholesterol (TC) – (HDL-C+ VLDL-C. VLDL-C= Triglycerides / 5.

Histopathology study of liver & kidney

The liver and kidney histopathological observations were done according to steps that described by[14]. After rats sacrificed, liver and kidney organ removed and fixed by 10% formalin fluid, processing and staining technique. Then, the liver tissue was sectioned and stained with hematoxylin and eosin (H&E) and examined under high power microscope and photomicrographs were taken.

Statistical Analysis

The analysis of data was expressed as mean ±SD. The comparisons between groups were performed with analysis of variance (ANOVA)by using computer zed SPSS program (Statistical Program for Social Sciences). P≤0.05 was considered to be the lest limit of significance. Least significant different test (LSD)was calculated to test the difference among means for (ANOVA)SPSS (1998). The comparisons within groups were performed with T-test using computerized SPSS program (Statistical Program for Social Sciences). P≤0.05 was considered to be the lest limit of significance.

RESULTS

The results show that table (1) the serum AST is insignificantly increased (p≤0.05) in male rats dosed with MSG 100 mg/kg b. w. as compared with control male rats. It also shows that the serum AST is significantly increased (p≤0.05) in male rats dosed with MSG 200 mg/kg b. w. as compared with control male rats. Protective treatment with doses of vitamin E results in significant decrease (p≤0.05) in serum AST as compared with MSG group. It is obvious that ALT enzyme elevated significantly (p≤0.05) in the group that received monosodium glutamate 100 and 200 mg/kg b. w. as compared with control group. Protective treatment with doses of vitamin E results in significant decrease (p≤0.05) in serum ALT activity as compared with MSG group. Alkaline phosphatase (ALP) enzyme elevated significantly (p≤0.05) in the group that received monosodium glutamate 100 and 200 mg/kg b. w. as compared with control group. Protective treatment with doses of vitamin E results in significant decrease (p≤0.05) in serum ALP activity as compared with MSG group. Table (2) shows that the urea is significantly increased

Table 2: The protective role of vitamin E from MSG exposure on male rats in some serum kidney enzymes.

Parameter Groups	Urea mg/dl	Creatinine mg/dl
Control	37.801 ± 13.18 b	0.351 ± 0. 03 b
MSG 100 mg/kg	55.025 ± 4.51 c	0.466 ± 0.047 c
MSG 200 mg/kg	66.606 ± 9.46 a	0.567 ± 0.05 a
MSG 200 mg/kg +vitamin 10mg/kg	40.061 ± 8.73 b	0.393 ± 0.09 bc
LSD	11.581	0.104

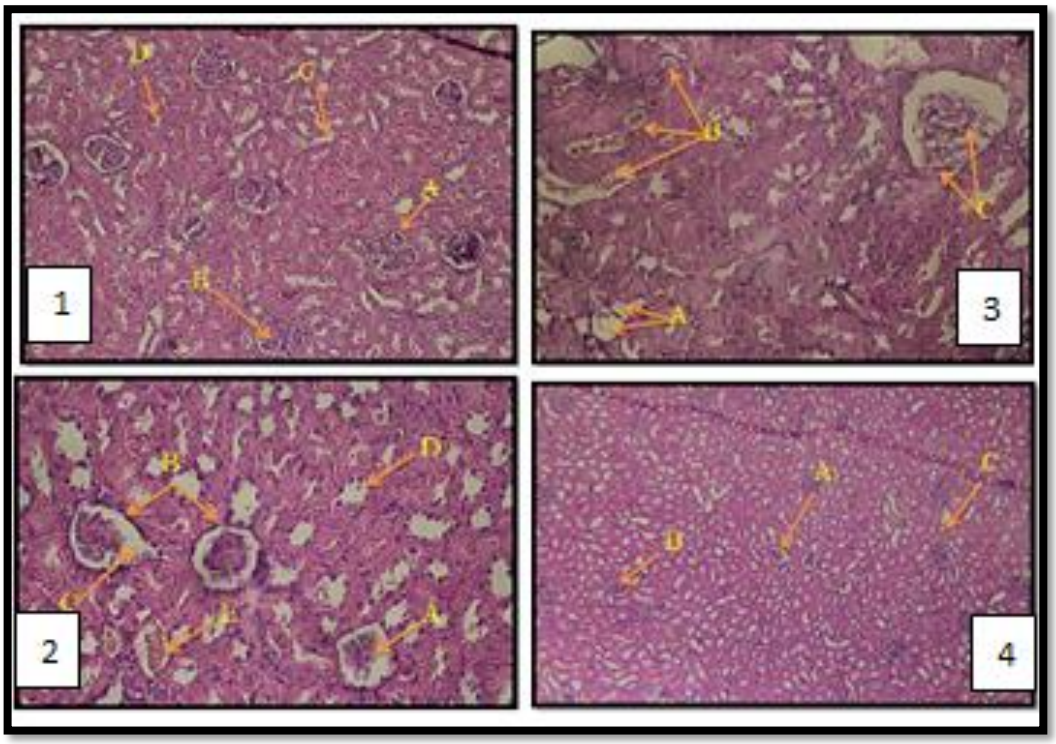
N=6

The numbers represent the mean ±standard Deviation. Different letters represent significant difference at ($p \leq 0.05$).

($p \leq 0.05$) in male rats treated with different doses of MSG 100 and 200 mg/kg b. w. as compared with control male rats. Protective treatment with doses of vitamin E results in significant decrease ($p \leq 0.05$) in serum urea activity as compared with MSG group. Serum creatinine gets higher level significantly ($p \leq 0.05$) with different doses of MSG 100 and 200 mg/kg b. w. as compared with control male rats. Protective treatment with doses of vitamin E results in significant decrease ($p \leq 0.05$) in serum creatinine activity as compared with MSG group. Table (3) shows that exposure to monosodium glutamate (100 and 200 mg/kg b. w.) led to significant increase ($p \leq 0.05$) in serum total cholesterol as compared with control group. The groups that received MSG +vitamin E and protective treatment of (200mg/kgMSG,100 mg /kg b .w. respectively) vitamin E (V.E.) led to significant decrease ($p \leq 0.05$) in serum cholesterol as compared with MSG group and control group. Serum triglycerides increase significantly ($p \leq 0.05$) with MSG exposure 100 and 200 mg/kg b. w. as compared with control groups. Protective treatment with monosodium glutamate and vitamin E 100mg /kg b. w. led to significant decrease in serum triglyceride, but still higher significantly as compared with control group. Serum HDL-C decreases significantly ($p \leq 0.05$) with MSG exposure 100 and 200mg/kg b. w. as compared with control group. Protective treatment with monosodium glutamate 200mg/kg b. w. and vitamin E 100mg /kg b. w. led to significant increase in serum HDL-C, but still higher significantly as compared with control group. Serum LDL-C increases significantly ($p \leq 0.05$) with MSG exposure 100 and 200mg/kg b. w. as compared with control group. Protective treatment with monosodium glutamate 200mg/kg and vitamin E 100mg /kg b. w. led to significant increase in serum LDL-C, but still lower significantly as compared with control group. Serum VLDL-C increases significantly ($p \leq 0.05$) with MSG exposure 100 and 200mg/kg b. w. as compared with control group. Protective treatment with monosodium glutamate 200mg/kg b.w. and vitamin E 100mg /kg b. w. led to significant increase in serum VLDL-C, but still equal to the control group.

Histopathological results

The histological examination of the liver of control group revealed normal rat liver in control group showing central vein, liver cords of hepatocytes radiating from the central vein and separated by blood sinusoid. hepatocytes with rounded nuclei(Fig.1). The histological results indicate that the exposure to monosodium glutamate 100mg/kg b.w shows loss of normal liver architecture, there are areas of necrosis, the dilated of sinusoidal spaces and degeneration of hepatocytes with varying shapes and sizes of nuclei(Fig.2). The histological results indicates that the exposure to monosodium glutamate 200mg/kg b.w shows increasing in disturbance of liver architecture, congested central vein with highly affected endothelial lining which contained hypertrophied nuclei, highly dilated sinusoidal spaces with thickened tunica media of it and pyknotic nuclei .There infiltrated inflammatory cells in the hepatic tissue(fig 4). The protective treatment with vitamin E and monosodium glutamate 100mg/kg b. w. show decrease in the histological changes in liver as compared with the administration of monosodium glutamate in groups (100and 200mg/kg).There is a less dilation of central vein, less dilation of sinusoidal spaces, less congestion in central vein and less infiltration of lymphocytes(fig.6)In the histological examination of the kidney of control group, shapes and sizes of most of the Bowman's capsules and the parts of nephron appear to be normal .(Fig.7). The histological results indicate that the exposure to monosodium glutamate 100mg/kg in male rats distends Bowman's capsule, increase the capsular spaces. There is shrinkage of glomerulus with contraction of the proximal convoluted tubules. The degradation of renal cells and there were not prominent of nuclei with varying shapes and sizes. There is distortion of renal cytoarchitecture and hyper cellularity(Fig.8). By using the 200mg/kg, there is cellular proliferation of mesangial or endothelial cells and infiltration of inflammatory cells (Fig.9) Some of the cells are seen as necrosis or disintegrated and cell debris is found in the lumen of the tubules. The degenerated tubules showed detachment of the cells from the basement membrane and exudation of cellular contents in the lumen with cytoplasmic vacillations. The nuclei of these cells were either pyknotic or karyolysis. The focal hemorrhagic areas were seen in between the renal tubules and there are congestions in the glomerulus and tubules(fig.10). The capillaries existed in between the tubules are also dilated and their basement membrane is thickened. There is an increase in distention of Bowman's capsule and distention of capsular spaces. There is an increase in degeneration of renal cells and nuclei vary in shapes and sizes (fig. 12). The protective treatment with vitamin E 100mg/kg b. w. and monosodium glutamate 200mg/kg b. w. in this research is abnormal in the histological architecture of kidney in rat considerably less than the histological changes with administration of monosodium glutamate in group (100 and 200mg/kg), These changes represented



1-Figure 7: Light micrograph of kidney of rat of control group notice the normal Bowman's capsule (A), glomerulus (B), distal convoluted tubules (C) and proximal convoluted tubules (D). (H&E,10x).
2-Figure 8: Light micrograph of kidney of rat exposed monosodium glutamate 100mg/kg for 30 days' notice shrinkage of glomerulus (A) ,dilation increase of some Bowman's capsule (B), dilated increase of capsular space (C) and degeneration of renal cells (D) congestion (E) ,(H &E 10X).
3-Figure 10: Light micrograph of kidney of rat exposed monosodium glutamate 200mg/kg notice, vacuolation(A), congestion(B) and epithelium cell (C) (H&E40X).
4-Figure 13: Light micrograph of kidney of rat exposed monosodium glutamate and vitamin E notice, normal Bowman's capsule(A) Bowman's capsule(B)and decrease congestion (C), (H&E4X).

with less distances of Bauman's capsule, less capsular spaces ,less number of glomerular shrinkage, are normal in shapes and sizes of nuclei and less of infiltration in lymphocytes(fig. 13).

DISCUSSION

The significantly elevated AST, ALT, ALP in male rats exposed to 100 and 200 mg/kg b. w. of MSG as compared with control group (table 1) are due to the leakage of aminotransferase (AST) enzymes from injured liver cells. The elevated serum AST and ALT values are also reported similarly by[15,16] who suggested that the elevated AST and ALT values could be attributed to acute toxicity of MSG leading to enhancing hepato-cellular activity and it could be reflected to possible treatment-induced damage to the metabolically active organ including the liver. The reduction of ALT as observed in the present study can be explained by the suggestion of[17] who commented that the regular and high consumption of monosodium glutamate could induce enzymatic changes and reduction of liver enzymes activity. The data also reveals that treated rats show highly significant increase in liver enzyme activities (ALT, AST, and ALP). The results of the current study are in agreement with[18,19]. With regard to the increased concentration of ALP in the serum of animals treated with MSG, it can be attributed to the damage to the intestine and gallbladder. In addition to hepatic cells, this enzyme is present in the intestine and gallbladder[20,21]. Treatment with doses of vitamin E in male rats results in significant decrease ($p \leq 0.05$) in serum AST, ALT and ALP enzymes. This result is in agreement with the studies where treatment with vitamin E has been reported to confer protection against such changes in monosodium glutamate induced –hepatotoxicity and oxidative stress in rats[22,23]. Vitamin E when intake together with MSG reduces the activities of the markers of injured hepatocellular ALT and AST. ALT and AST are also elevated
in cases of injury to other organs like kidney, heart and muscles[24]. Vitamin E decreases the elevated serum levels of liver enzymes AST and ALT when compared to the treated MSG group which agrees with[25]. MSG exposure results in significant increase in serum urea and creatinine in male rats as compared with control groups (table 2). The urea and creatinine values indicate dose dependent increase in all treatment groups and indicate remarkable kidney damage in experimental rats. Similar observations of increased creatinine were reported by[26,27] and confirm the findings of present investigation. Exposure to MSG may cause an adverse effect on the renal function which might be due to oxidative stress induced by MSG on the

Table 3: The protective role of vitamin E from MSG exposure on male rats some serum lipids profile.

Parameter Groups	Total Cholesterol mg/dl	T.G mg/dl	HDL mg/dl	LDL mg/dl	VLDL mg/dl
CONTROL	45.085±2.20 b	43.508± 2.33 bc	19.966±1.14 b	13.841±2.85 c	10.413±2.54 ac
MSG 100 mg/kg	56.985±4.71 c	64.058±9.87 ac	17.771±3.03 bc	25.895±6.05 b	13.245±2.35 a
MSG 200 mg/kg	83.613±4.76 a	78.483±39.41 a	16.915 ±2.38 c	50.895±8.00 a	15.631±7.91 a
MSG200 mg/kg +vitamin100 mg/kg	43.398±1.17 b	50.713±10.07 bc	23.218 ±1.94 a	10.021±4.03 c	10.141±2.01 bc
LSD	11.900	27.770	3.051	12.053	5.490

N=6

The numbers represent the mean ±standard Deviation.

Different letters represent significant difference at ($p \leq 0.05$).

renal tissue. This can be explained by a change in threshold of tubular reabsorption and Glomerular filtration rate[28]. Otherwise, it is believed that the significant elevation in urea and creatinine levels is closely related to the impairment of renal function. Increased concentrations of creatinine and total urea in blood during renal diseases or renal damage may be due to high activities of xanthine oxidase, lipid peroxidation, increased triacylglycerol and cholesterol levels, as well as impairment of the urea cycle enzyme activities[29,19]. The significant decrease in serum creatinine with vitamin E 100mg /kg b.w.in male rats shows that protective antioxidants such as Vitamin E which has a modulator effect on MSG-induced serum urea oxidative damage in the liver and kidney of rats. The variation in the level of urea and creatinine are markers of renal dysfunction[30]. The exposure to monosodium glutamate 100 and 200 mg/kg b. w. results in significant elevation in serum total cholesterol, triglycerides, LDL-C, VLDL-C in male rats. With regard to the HDL-C significantly decreases in treated male rats (tables 3) The results are in agreement with[31,32,20,19]. HDL-C concentration shows a reduction in its level when compared with control rats leading to an increase in the synthesis of cholesterol or peroxidation of cell membrane lipids[20]. Increasing in LDL and VLDL levels increases the risk of cardiovascular diseases[33]. The present results are in agreement with previous studies of[23,25]. The present study shows that lipid profile reduced the serum levels of amount of total cholesterol, LDL, VLDL, cholesterol and triacyglycerols, and increased HDL cholesterol. These results are in agreement with the pervious study of[34].

Discussion Histological

The result of the present study is in agreement ion with[35]. who referred to increase of the number of hepatocytes with large nuclei and there are degenerated of hepatocytes with numerous vacuolations in liver of albino rats treated with monosodium glutamate for 30 days. This result is in agreement with[36,37,15,38,39,40] who showed severe abnormal architectural in structure of liver and hepatocyte treated with monosodium glutamate in rats at period for 30 days a lot of inflammatory cell with high doses of monosodium glutamate in liver of rat. The congested hepatic Vein and the dilated or rupture of central vein liver treated with monosodium glutamate. The increasing of hepatocyte and the damage of liver may be due to induce oxidative stress after administration monosodium glutamate[23,41], They mentioned that the metabolism of glutamate leads to liver exacerbation of trans-fat induced fatty liver disease and an increase in the adiposity and change in liver structures. The protective treatment with vitamin E 100mg/kg and monosodium glutamate 200mg/kg b. w. results in moderate improvement liver after vitamin E administration and this may be due to the activity of vitamin E as antioxidant. Al-Attar (2011) suggested that vitamin E might be a useful preventive agent against the effect of the heavy metals at least partly due to its antioxidant properties. can be suggested the administration of vitamin E might alleviate the liver biochemical disturbances and histopathological alterations from the oxidative stress produced by exposure to heavy metals. Magdy et al, (2016) reported that vitamin E act as antioxidants to protect the liver against toxic effects of abamectin. The results carried out by Eweka,(2007), whose study investigated the effects of monosodium glutamate on the kidney of adult Wister rats gave 100mg/kg and 200mg/kg of MSG thoroughly mixed with growers mash for the period of 30 days reduction in the number of renal capsule in the treated groups which was at variance with that of the control group. MSG results in cellular necrosis of the Bowman's capsule and at dose of degeneration and atrophy of the kidney were seen, the high doses and chronic ingestion of MSG results the degenerative and atrophic changes observed in the renal corpuscle, in addition, the progressing renal injury increase with the increase of doses of MSG. Our results suggest that the functions of the kidney could have been adversely affected due to the distortion of the cyto-architecture of the renal cortical structures and cellular necrosis associated with the kidney. These findings come in agreement with several studies[44,2]. The protective treatment with vitamin E 100mg/kg and monosodium

glutamate 200mg/kg b. w. results in moderate improvement kidney in after vitamin E administration and this may be due to the activity of vitamin E as antioxidant (Fig .13)[43]. mention that in male rats treated with 200mg MSG and 100mg vitamin E, the antioxidant action may protect the kidney tissue against the oxidative stress.

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