Biodiversity Research Journal, 2025, volume 3, issue 1, 154-165.

Vitamin c attenuates monosodium glutamate-induced hepatotoxicity in albino rats

AUTHORS

Sherif Rabea Mohamed, and
Mona Abd El-Rhman Ibrahim

AFFILLIATIONS

Department of Zoology and Entomology, Faculty of Science, Helwan University, Cairo, Egypt

CORRESPONDENCE

sherifrabea@science.helwan.edu.eg

SUBMISSION HISTORY

RECIVED: March 1, 2025

ACCEPTED: April 25, 2025

PUBLISHED: June 30, 2025

CITATION

Mohamed S R, Ibrahim M A. *Vitamin C Attenuates Monosodium Glutamate-Induced Hepatotoxicity in Albino Rats*. ***Biodiversity Research Journal***, 2025. 3, (**1**); 154-165.

ABSTRACT

The overconsumption of monosodium glutamate (MSG) may cause liver injury by promoting oxidative stress, along with activating inflammatory processes within the organ. This research was intended to evaluate the hepatoprotective potential of vitamin C in a murine model of MSG-triggered liver injury. Four groups of 24 adult albino male rats were arbitrarily assigned: a group for control, an MSG group (given 60 mg/kg MSG daily for 30 days), a vitamin C group (given 100 mg/kg vitamin C daily for 30 days), and a co-treatment group receiving vitamin C followed by MSG two hours later for 30 days. Biochemical assessments were performed to measure liver antioxidant markers, such as the activity of superoxide dismutase or SOD, catalase or CAT, and glutathione, or GSH, as well as lipid peroxidation markers such as malondialdehyde (MDA) and nitric oxide (NO) levels. Measurements of interleukin-1 beta (IL-1β) along with tumor necrosis factor-alpha (TNF-α) were employed to assess the inflammatory status, while serum alanine aminotransferase (ALT) along with aspartate aminotransferase (AST) activities were employed to assess liver function. Histopathological examination of liver tissues was also conducted. MSG treatment significantly impaired antioxidant defenses and increased both oxidative and inflammatory markers, leading to hepatocellular injury. Conversely, vitamin C administration effectively restored antioxidant enzyme activities, reduced oxidative and inflammatory mediators, and retained normal liver architecture. These results indicate that vitamin C represents a viable therapeutic approach for counteracting MSG-induced hepatic toxicity by attenuating oxidative stress and inflammation.

KEYWORDS: Vitamin C, monosodium glutamate, Inflammation, and Liver.

INTRODUCTION

MSG, or monosodium glutamate, has emerged as a common taste enhancer found in a lot of manufactured meals, snacks, and restaurant meals worldwide. Its popularity stems from its function as an umami taste enhancer, making it a common additive in Asian cuisines, canned soups, instant noodles, and fast food (Shi et al., 2012). Although regulatory bodies like the U.S. FDA consider MSG to be 'generally recognized as safe, growing research indicates that long-term or excessive intake could harm multiple biological systems (Ogunmokunwa and Ibitoye, 2025).

Some studies associate MSG with adverse effects such as metabolic syndrome, neurotoxicity, and hepatotoxicity, though findings remain controversial (Niaz et al., 2018, and Zanfirescu et al., 2019). Notably, in regions with high dietary MSG intake, such as East Asia, concerns have been raised about its contribution to liver dysfunction, prompting further investigation (He et al., 2021). The liver, a primary organ for detoxification and metabolism, is particularly vulnerable to substances that promote oxidative stress and inflammation. Research indicates that high doses of MSG can contribute to oxidative stress, trigger inflammatory responses, and lead to hepatocellular damage (Eweka et al., 2011).

An important mechanism by which MSG causes injury which is attributable to a surplus of reactive oxygen species (ROS). Elevated levels of ROS can lead to the impairment of cellular integrity and peroxidation of lipids, and hepatocyte apoptosis, thereby impairing liver function (Singh and Ahluwalia, 2012). A prior study found that MSG boosted the expression of pro-inflammatory mediators as TNF-α along with IL-6 (Asejeje et al., 2023). MSG consumption promotes ROS production, which triggers lipid, protein, and DNA damage induced by reactive radical species. The peroxidation of lipids degrades polyunsaturated lipids in cell membranes, culminating in apoptotic cell death (Sahin et al., 2023).

Vitamin C (Vit. C), generally called ascorbic acid, is a vital nutrient noted for its strong antioxidant capabilities. It is essential for eliminating free radicals, strengthening the body's inherent antioxidant defense mechanisms, and also modifying inflammatory pathways (Rizvi et al., 2014). According to scientific data, vitamin C supplementation can mitigate oxidative stress, restore antioxidant enzyme activity, and support liver function in various models of toxin-induced hepatotoxicity (El-Meghawry El-Kenawy et al., 2013). Due to its protective effects, vitamin C is proposed as a potential therapeutic agent for counteracting MSG-induced liver damage.

This current work aims to investigate the Vitamin C's capacity to protect the liver in counteracting MSG-triggered liver injury. By assessing oxidative stress biomarkers, inflammatory mediators, and histopathological changes, this research seeks to provide critical insights into whether vitamin C can serve as a preventive or therapeutic strategy against MSG-associated liver toxicity. Given the global prevalence of MSG consumption and the ongoing safety debates, public health can benefit greatly from this study, especially in areas where dietary exposition to MSG is substantial.

MATERIALS AND METHODS

Chemicals

Morgan Pharma Co provided the pure monosodium glutamate (a concentration of 99%) in Egypt, and C-Retard (500 mg of vitamin C) was sourced from Hikma Company in Egypt. Oxidative stress indicator kits were supplied by Biodiagnostics in Cairo, while enzyme-linked immunosorbent assay kits for detecting inflammatory markers were provided by Thermo Fisher Scientific in the USA. Additionally, 10% formalin and high-purity analytical-grade stains were acquired from El-Gomhoryia Chemical Company in Cairo.

Experimental design and treatment protocol

The Medical Research Institute of Ain-Shams in Cairo, Egypt, provided twenty-four mature male rats weighing 150–180 grams per each. The work was carried out under veterinary supervision, adhering to the animal housing regulations of the Ain-Shams Research Institute. Additionally, all experimental procedures followed the guidelines of ethical research established by Ain Shams University Animal Research Unit assigned the approval number [RE (189)22]. All rats were maintained under standard laboratory conditions. After a seven-day acclimatization period, using a random number table, the rats were randomly assigned to one of four groups (n=6/group) in a randomized design. The groups were as follows:

Group 1 (Control group): Administered the 0.9% saline vehicle by daily oral gavage for 30 days.

Group 2 (MSG group): Received MSG at a dose of 60 mg/kg daily via oral gavage for 30 days. This dose reflects established hepatotoxic levels from prior research without causing acute mortality (Hamza & Al-Harbi, 2014).

Group 3 (Vit. C group): Given 100 mg/kg of vitamin C orally each day for 30 days. This dosage was chosen in accordance with previous research showing its efficacy in mitigating oxidative stress in rodent models (Wahdan & Shareef, 2016).

Group 4 (Vit. C + MSG) was administered vitamin C (100 mg/kg) orally for 30 days, after this, MSG administration (60 mg/kg) two hours later each day for 30 days. The co-administration protocol was designed to assess the potential of vitamin C to mitigate toxicity triggered by MSG.

Following euthanasia (xylazine-ketamine combination, i.p.), retro-orbital blood was taken and centrifuged to extract serum for liver function testing (storage at -20°C). Livers were removed and split, one portion was homogenized in Tris-HCl buffer (0.1 M, pH 7.4), centrifuged, and the supernatant was stored at -20°C for subsequent analysis of oxidative stress, antioxidant, and inflammatory parameters. The remaining piece was formalin-fixed for histopathology.

Biochemical Analysis

Oxidative stress markers: Hepatic GSH levels were quantified using a colorimetric assay with Ellman's reagent, which serves as an indicator of nonenzymatic antioxidant capacity (Ellman, 1959). The activity of SOD was assayed by measuring the reduction inhibition of nitroblue tetrazolium (Sun et al., 1988). The activity of CAT was quantified based on the rate of H_2O_2 breakdown (Aebi, 1984). Nitric oxide or (NO) concentrations in liver tissue were evaluated using a commercial kit (Biodiagnostics, Cairo, Egypt). In this method, NO is converted to nitrous acid, measured by azo dye formation using the Griess reagents (sulfanilamide and N-(1-naphthyl) ethylenediamine), absorbance of the dye can be determined at 540 nm (Bryan and Grisham, 2007). The peroxidation of lipids was assessed via quantifying MDA levels (Ohkawa et al.,1979).

Inflammatory markers: Employing particular commercial ELISA kits (Scientific Thermo Fisher, USA; Cat. No. BMS607-3 for TNF-α plus BMS6002 for IL-1β), serum concentrations of TNF-α plus IL-1β measured in accordance with the manufacturer's instructions.

Liver function test: Using commercial colorimetric assay kits, the levels of AST plus ALT in sera were measured enzymatically (Biodiagnostic Co., Giza, Egypt) (Reitman and Frankel, 1957).

Histopathological examination

Tissue samples were fixed in formalin, cleared in xylene, and progressively dehydrated using ascending concentrations of ethanol before having paraffin wax infused in it. After cutting sections at 5 μm thickness, they were stained through hematoxylin and eosin to do microscopic evaluation (Slaoui & Fiette, 2011).

Statistical Analysis

The mean ± SD serves to express the data. GraphPad Prism (v.8.0) was adopted to examine group differences via a one-way ANOVA with Tukey's post-hoc test for multiple comparisons; $p < 0.05$ was deemed statistically significant.

RESULT

Oxidative Stress plus Antioxidant Status

MSG exposure significantly (*P<0.05*) reduces intracellular glutathione (GSH), an essential nonenzymatic antioxidant that neutralizes reactive oxygen species, whereas vitamin C supplementation replenishes GSH levels (Figure 1a). Additionally, catalase (CAT) activity, crucial for breaking down hydrogen peroxide, is substantially impaired by MSG treatment; however, vitamin C markedly boosts CAT activity ($P < 0.05$) (Figure 1b). Similarly, MSG leads to a considerable decline in superoxide dismutase (SOD) activity, which is essential for converting superoxide radicals into hydrogen peroxide ($P < 0.05$). At the same time, vitamin C helps maintain or even enhance SOD function (Figure 1c). Furthermore, the MSG-treated group shows higher malondialdehyde (MDA) levels, a sign of peroxidation of lipid and membrane damage, but co-treatment with vitamin C significantly reduces these levels ($P < 0.05$) (Figure 1d). Finally, MSG increases nitric oxide (NO) production, suggesting heightened nitrosative stress, which is effectively *lowered* ($P < 0.05$) by vitamin C supplementation (Figure 1e). Overall, these findings suggest that vitamin C can counteract the oxidative stress induced by MSG by restoring the power of antioxidant enzymes in addition reducing markers of peroxidation of lipid and nitrosative stress (Table 1 and Figure 1).

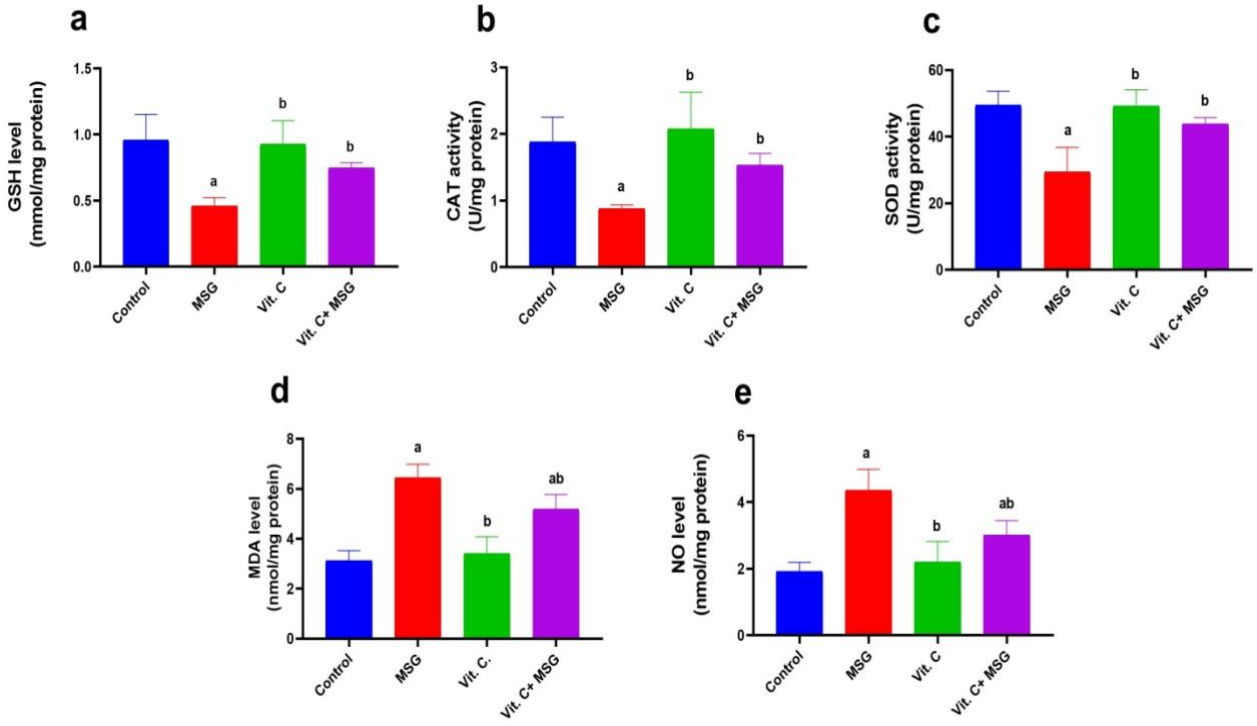

Figure 1. Vitamin C effects on hepatic oxidative stress markers in MSG-exposed rats: (a) glutathione (GSH), (b) catalase (CAT), (c) superoxide dismutase (SOD), (d) malondialdehyde (MDA), (e) nitric oxide (NO). Data shown as mean ± SD (n=6). Distinct letters (a, b) denote significant differences ($p < 0.05$) from the control and MSG groups, respectively.

Table (1): Statistical comparison of biochemical parameters across groups.

Parameters \ Groups	Control	MSG	Vit. C	Vit. C + MSG
GSH: mmol/mg prot.	0.96 ± 0.19	0.46 ± 0.06 ^{a}	0.93 ± 0.18 ^{b}	0.75 ± 0.04 ^{b}
CAT: U/mg prot.	1.9 ± 0.37	0.88 ± 0.06 ^{a}	2.1 ± 0.55 ^{b}	1.5 ± 0.18 ^{b}
SOD: U/mg prot.	50 ± 4.1	29 ± 7.4 ^{a}	49 ± 4.9 ^{b}	44 ± 2.0 ^{b}
MDA: nmol/mg prot.	3.1 ± 0.41	6.4 ± 0.55 ^{a}	3.4 ± 0.69 ^{b}	5.2 ± 0.6 ^{ab}
NO: nmol/mg prot.	1.9 ± 0.28	4.4 ± 0.63 ^{a}	2.2 ± 0.62 ^{b}	3.0 ± 0.44 ^{ab}
TNF: Pg/mg prot.	41 ± 5.4	98 ± 9.6 ^{a}	42 ± 7.2 ^{b}	73 ± 11 ^{ab}
IL1B: Pg/mg prot.	38 ± 5.6	89 ± 5.9 ^{a}	39 ± 6.3 ^{b}	60 ± 5.5 ^{ab}
ALT: U/L	42 ± 6.4	80 ± 7.2 ^{a}	42 ± 6.7 ^{b}	65 ± 8.6 ^{ab}
AST: U/L	42 ± 4.9	89 ± 7.0 ^{a}	43 ± 5.9 ^{b}	66 ± 9.1 ^{ab}

Data shown as mean ± SD (n=6). Distinct letters (a, b) denote significant differences ($p < 0.05$) from the control and MSG groups, respectively.

Inflammatory markers:

The MSG group exhibits a substantial ($P < 0.05$) increasing in TNF-α as contrasted to the control, indicating a heightened inflammatory response. By contrast, vitamin C alone maintains TNF-α near control levels, and Supplementation with vitamin C alongside MSG substantially ($P<0.05$) lowers TNF-α compared to MSG-only treatment (Figure 2, left panel). Similar to TNF-α, MSG treatment significantly ($P < 0.05$) elevates IL-1β, indicating an intensified pro-inflammatory state. Meanwhile, vitamin C alone does not differ significantly from the control, but its combination with MSG notably ($P < 0.05$) reduces IL-1β compared to MSG alone (Figure 2, right panel). Overall, these data suggest that MSG induces an inflammatory response characterized by elevated TNF-α and IL-1β, whereas vitamin C supplementation mitigates this effect, highlighting its potential anti-inflammatory role. Different letters (a, b, ab) above the bars denote statistical significance at $P<0.05$ (Table 1, Figure 2).

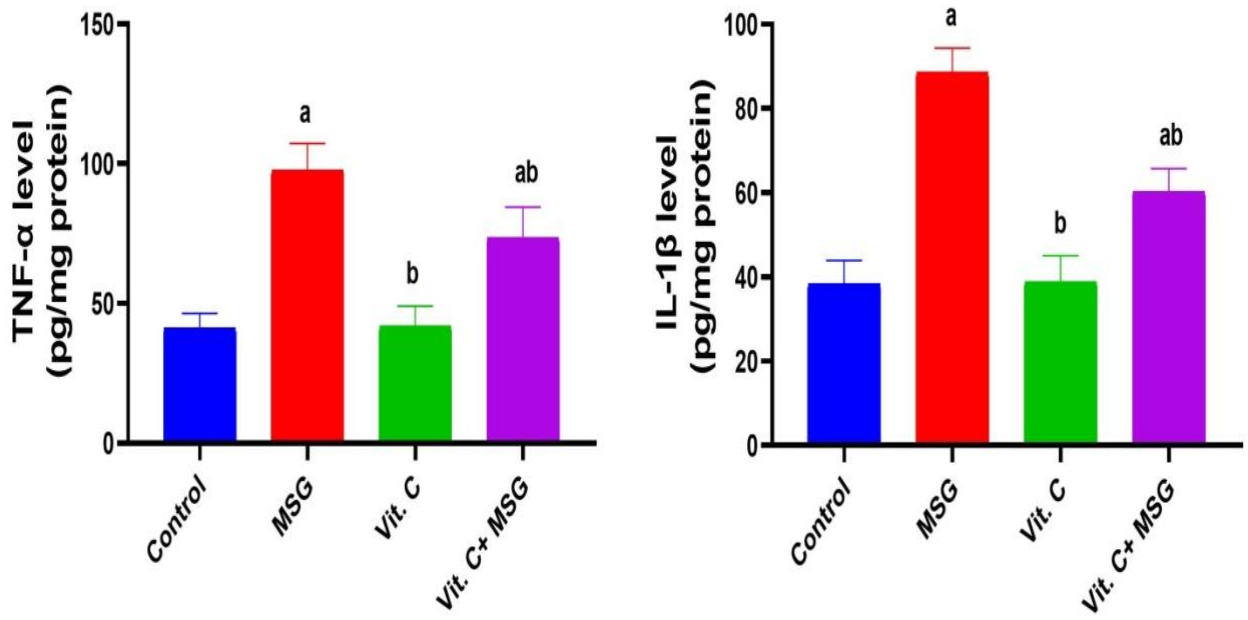

Figure 2. The effect of vitamin C on inflammatory indicators, such as TNF-α plus IL-1β levels, in the liver tissues of male rats recived MSG. Distinct letters (a, b) denote significant differences ($p < 0.05$) from the control and MSG groups, respectively.

Liver function markers:

Figure 3 shows serum ALT and AST levels across treatment groups. The MSG-only group exhibits a pronounced ($P < 0.05$) increasing in both ALT plus AST as contrasted to the control, suggesting potential liver injury. By contrast, vitamin C alone keeps these enzymes at levels similar to those of the control, reflecting minimal hepatic stress. When vitamin C is administered alongside MSG, there is a notable ($P < 0.05$) reduction in ALT in addition to AST activities as contrasted to the MSG group, indicating that vitamin C exerts a preventive effect against MSG-triggered hepatocellular damage. Bars marked with different letters (a, b, ab) are significantly different ($p < 0.05$) (Table 1, Figure 3).

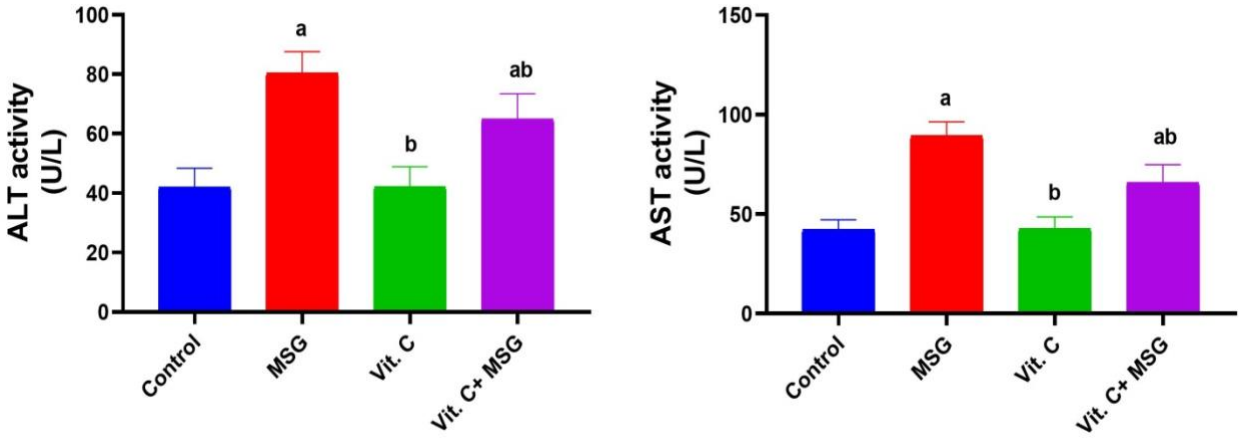

Figure 3. The effect of Vit. C on MSG-triggered abnormalities in ALT activity & AST activity in rat serum. Data shown as mean ± SD (n=6). Distinct letters (a, b) denote significant differences ($p < 0.05$) from the control and MSG groups, respectively.

Liver histopathology:

Hematoxylin and eosin (H&E) stained liver tissue photomicrographs are presented in Figure 4 at a 400× magnification (scale bar of 100 μm). The control group exhibits a normal hepatic architecture, characterized by orderly hepatocyte plates radiating from the central vein and distinct sinusoidal spaces (Figure 4a). In contrast, the MSG-treated group exhibits a marked disruption of the typical liver structure, including focal inflammatory infiltrates and numerous degenerated hepatocytes concentrated in the central region (Figure 4b). The vitamin C group maintains a histological appearance similar to that of the control, with no apparent abnormalities (Figure 4c). Finally, the co-treated group receiving vitamin C and MSG demonstrates minimal or no visible lesions, suggesting a preventive role for vitamin C toward MSG-triggered hepatic damage (Figure 4d).

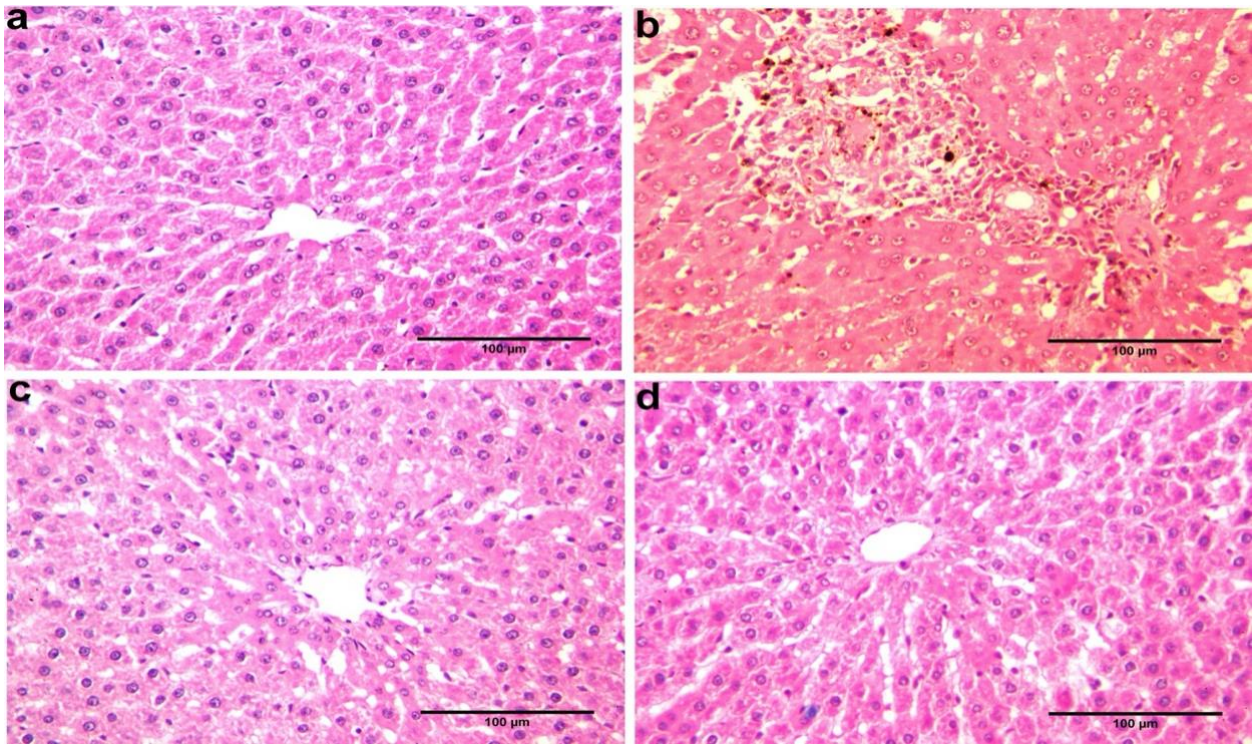

Figure 4. Representative liver histology (H&E staining). (a) Control group: typical hepatic architecture with intact hepatocyte plates emanating from the central vein and well-defined sinusoids. (b) The MSG group exhibited loss of normal histoarchitecture, characterized by marked focal inflammation and numerous degenerated hepatocytes in the center. (c) Vit. The C group showed normal liver histoarchitecture (d) Vit. C +MSG group showed no visible lesions. (Scale bar = 100µm)

DISCUSSION

The current study reveals that contact with monosodium glutamate (MSG) generates considerable oxidative stress plus inflammation in the liver, resulted in hepatocellular damage. Specifically, MSG administration triggered a substantial reduction in intracellular GSH concentration levels and impaired the potential of key antioxidant enzymes, including CAT plus SOD. This decline in antioxidant defences correlated with elevated levels of MDA, a marker of fats peroxidation, and elevated nitric oxide (NO) production, indicating heightened oxidative and nitrosative stress. These biochemical disturbances align with previous reports suggesting that MSG can promote the genesis of reactive radical species and trigger oxidative damage in liver tissues (Henry-Unaeze, 2017).

Farombi and Onyema (2006) demonstrated that vitamin C reduces oxidative damage triggered by monosodium glutamate. The antioxidant action of vitamin C in our study is indeed line with Mohammed et al. (2024) findings, vitamin C significantly reduced amoxicillin/clavulanic acid (AC)-induced hepatotoxicity. The considerable reduction of blood liver enzymes, MDA reduction, and GSH elevation in the vitamin C-treated group demonstrated that vitamin C protects against AC-induced hepatotoxicity (Mohammed et al., 2024). Our results corroborate existing evidence that vitamin C acts as a potent inhibitor of lipid peroxidation (Soylu et al.,

2006). Vitamin C acts as an indirect antioxidant by inhibiting NF-κB activation, which participates in the production of ROS (Moore & Khanna, 2023). Vitamin C, as an electron donor, may diminish ROS directly, including metabolic byproducts such as superoxide plus hydroxyl radicals (Cimmino et al., 2018).

Furthermore, vitamin C promotes redox balance by decreasing oxidized forms of vitamin E plus glutathione (Parker et al., 2012). Vitamin C serves as an indirect antioxidant by blocking NF-κB, which triggered the genesis of ROS (Carr & Maggini, 2017). Tan et al. (2005) mentioned that vitamin C reduces oxidative stress in human dendritic cells via decreasing NF-κB activation. Beyond its antioxidant actions, vitamin C may neutralize hazardous nitrogen-based molecules as carcinogenic N-nitrosamines and also nitrosamides (Sauberlich, 1994).

In parallel, the study found that MSG administration substantially raised TNF-α plus IL-1β levels, indicating an intensified inflammatory response. This finding supports the notion that oxidative stress and inflammation are closely interrelated processes, where increased free radical formation can activate inflammatory pathways, further exacerbating tissue injury. The reduction in these cytokines observed with vitamin C supplementation underscores the potential anti-inflammatory properties of vitamin C, which may be attributed to its capacity to stop pro-inflammatory signaling cascades and scavenge ROS (Choudhary & Tran, 2012).

The impact of MSG on liver function was further evidenced by marked raising in serum ALT plus AST levels. These enzymes are well-known indicators of hepatocellular damage, and their increased levels reflect the compromised integrity of liver cells following exposure to MSG. Conversely, vitamin C alone maintained ALT and AST activities close to control levels, and its co-administration with MSG substantially reduced these enzyme levels, suggesting that vitamin C exerts a hepatoprotective effect.

Histopathological evaluation provided additional confirmation of MSG-induced liver damage. The control group exhibited normal hepatic architecture, characterized by well-organized hepatocyte plates and distinct sinusoidal spaces. In contrast, liver sections from the MSG-treated group showed pronounced disruptions in tissue organization, including focal inflammatory infiltrates and degenerative changes in hepatocytes. Notably, the liver tissues from the vitamin C-treated group were comparable to those of the controls. In contrast, co-treatment with vitamin C and MSG resulted in markedly fewer lesions, demonstrating the preventive role of vitamin C toward MSG-elicited histological damage. This finding is consistent with previous studies that have documented the deleterious actions of MSG on liver function through oxidative and inflammatory mechanisms (Henry-Unaeze, 2017; Choudhary & Tran, 2012). Furthermore, the preventive effects of vitamin C observed in the present study align with earlier research highlighting its capacity to restore antioxidant enzyme activities and reduce lipid peroxidation and inflammatory cytokine production (Carr & Maggini, 2017; Al-Ghafari, 2021). However, some discrepancies in the literature regarding the magnitude of MSG-induced damage may arise from variations in experimental protocols, including differences in MSG dosage, exposure duration, and the specific animal models used.

Limitations and Future Directions

Despite the robust findings, this study has several limitations. The short duration of MSG exposure and the exclusive utilize of rats with male gender may restrict the broader applicability of these findings to other populations, including female subjects and humans. Future studies should consider more extended treatment periods and a more diverse range of animal models or clinical populations to validate these findings. Additionally, a better comprehension of the molecular processes that underlie vitamin C's protective effects could provide valuable insights into its therapeutic potential in preventing MSG-induced hepatic injury.

CONCLUSION

In summary, the results indicate that MSG triggered significant inflammation, oxidative stress, and liver damage, as evidenced by altered biochemical and histopathological markers. Vitamin C supplementation effectively counteracts these effects by restoring antioxidant defences, reducing pro-inflammatory cytokine levels, and preserving liver tissue architecture. These results imply that vitamin C might present a viable tactic for mitigating MSG-induced hepatic toxicity; Future research is required to assess the clinical relevance of these results.

CONFLICT OF INTERESET STATEMENT

We declare that we have no conflict of interest.

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