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Camel Milk Attenuates Cardiotoxicity Induced by Monosodium Glutamate via Inhibition of Oxidative Stress and Inflammation in Rats

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ABSTRACT

Monosodium glutamate (MSG), a widely used food additive, has been implicated in cardiotoxic effects through mechanisms involving oxidative stress, inflammation, and apoptosis. Camel milk (CM), known for its antioxidant and anti-inflammatory properties, may offer protective effects against such toxicity. This study evaluated the cardioprotective potential of CM against MSG-induced cardiac damage in rats**.** Forty male Wistar rats (185-205 g) were randomized into four groups (n = 10): Control (distilled water), MSG (6 g/kg), MSG + CM (MSG 6 g/kg + CM 5 ml/kg), and Recovery (MSG 6 g/kg followed by 21 days without CM treatment). All treatments were administered orally for 21 days. Body and heart weights were recorded. Cardiac tissues were assayed for myeloperoxidase (MPO), nitric oxide (NO), C-reactive protein (CRP), interleukin-1β (IL-1β), tumor necrosis factor-α (TNF-α), NF-κB, malondialdehyde (MDA), catalase (CAT), superoxide dismutase (SOD), reduced glutathione (GSH), glutathione-S-transferase (GST), glutathione peroxidase (GPx), caspase-3 activity, and DNA fragmentation index. Histopathological evaluation of the myocardium was performed. Data were analyzed by one-way ANOVA with Tukey's post hoc test ($p < 0.05$). MSG significantly increased inflammatory (MPO, NO, CRP, IL-1β, TNF-α, NF-κB), oxidative (MDA), apoptotic (caspase-3), and DNA damage markers, while reducing antioxidant enzyme activities (CAT, SOD, GSH, GST, GPx) and heart weight ($p < 0.05$). CM restored antioxidant defenses, lowered inflammatory and apoptotic indices, reduced DNA fragmentation, and preserved myocardial architecture. The recovery group exhibited partial but less pronounced improvements. Camel milk effectively mitigates MSG-induced cardiotoxicity, highlighting its importance as a natural cardioprotective dietary intervention.

Keywords: Camel milk; Monosodium glutamate; Cardiotoxicity; Oxidative stress; Inflammation.

Introduction

Cardiotoxicity, defined as the occurrence of heart muscle damage due to exposure to toxic substances, remains a major concern in both clinical and environmental health. It often manifests through oxidative stress, inflammation, and structural alterations of cardiac tissue, ultimately compromising cardiovascular function.[1] Monosodium glutamate (MSG), a common food additive used to enhance flavor, has come under scrutiny due to its potential adverse health effects when consumed excessively.[2] Initially introduced in the 1960s as an inexpensive substitute for natural seasonings,[3] MSG has been linked to various toxicological effects, especially on the cardiovascular system. Evidence suggests that MSG promotes oxidative stress by stimulating the overproduction of reactive oxygen species (ROS), leading to lipid peroxidation and disruption of antioxidant defense systems.[4] Oxidative stress can contribute to the development of cardiomyocyte dysfunction and remodeling, which can lead to decreased cardiac function.[5]

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Moreover, MSG-induced cardiac damage involves the activation of apoptotic pathways. Excessive calcium influx via overactivated glutamate receptors enhances mitochondrial ROS generation, ultimately initiating caspase-mediated cardiomyocyte apoptosis.[6] At the cellular level, oxidative stress, inflammation, and apoptosis contribute synergistically to the disruption of genomic integrity within cardiac tissue. Oxidative stress-induced damage, along with pro-inflammatory signaling and apoptotic activation, compromises the stability of the DNA and the transcriptional regulation of genes essential for cardiomyocyte function. This genomic instability impairs the structural and functional capacity of cardiac cells, leading to diminished contractility, disrupted electrical conductivity, and progressive deterioration of myocardial performance.[7] Although several studies have identified the oxidative and apoptotic effects of MSG in various tissues, limited research has characterized its role in promoting cardiac DNA fragmentation. Furthermore, there is a paucity of studies evaluating natural protective agents that could mitigate these deleterious effects, particularly within the context of MSG-induced cardiotoxicity. Camel milk (CM), known for its rich nutritional profile and therapeutic benefits, has attracted attention for its anti-inflammatory, antioxidant, and anti-apoptotic properties.[8,9] Its immunomodulatory actions are facilitated by bioactive substances, including lactoferrin and immunoglobulins, which have demonstrated promise in reducing tissue damage in a variety of experimental paradigms. Despite its cardioprotective potential, CM's ability to prevent MSG-induced heart injury has not been thoroughly studied. Therefore, the goal of this study is to assess camel milk's cardioprotective potential in reducing oxidative stress, apoptosis, and DNA fragmentation in cardiac tissue caused by MSG.

Materials and Methods

Chemicals

All chemicals utilized in this study were of high-quality analytical grade. Fresh pasteurized camel milk was purchased from Simply Precious Enterprise (Badagry, Lagos State, Nigeria). The product was supplied in sealed 1-liter bottles, stored at 4 °C, and each bottle was used within five days of opening as instructed by the manufacturer. All animals were treated with milk from the same batch to minimize variability. Monosodium glutamate (MSG) was purchased from Mich Mikedenson Nigeria Enterprises Limited, Ilorin, Nigeria. Ethanol, distilled water, and phosphate buffer were acquired from the laboratory at the Physiology Department at Ekiti State University (EKSU), Ado-Ekiti, Nigeria.

Animals

The Central Animal House at EKSU served as the study's location. From the same Central Animal House, 40 male Wistar rats weighing between 185 and 205 g were acquired. The animals were housed in plastic cages with aluminum coverings and sawdust bedding, and they were maintained under typical laboratory settings. They were fed high-quality rat pellets (Tripods Feed Limited, Nigeria) and tap water on an as-needed basis.

Ethics approval

Ekiti State University's Department of Physiology's Research Ethics Committee approved the research protocol, which was assigned the ethical number EKSU/P100/2024/01/002.

Stock Solution Preparation

The MSG was administered at a dosage of 6 g/kg body weight, and the stock solution was constituted by dissolving 0.6 g of MSG in 1 ml of distilled water, with a concentration of 600 mg/ml.[10] MSG was administered orally using rat cannulas according to the research protocol. The dosage of camel milk was 5 ml/kg body weight.[11] The camel milk was also administered orally using rat cannulas according to the research protocol

Experimental Protocol

After acclimatizing the animals to the laboratory environment for 14 days, the 40 male Wistar rats were randomly grouped into 4 groups of 10 animals per group (A, B, C, D). Administration was done daily through oral routes for 21 days, and the animals were treated accordingly using oral cannulas as shown in Table 1.

Table 1: Experimental Grouping and Dosage

GROUPINGS	DOSAGE
Group A (Negative Control)	Distilled water was given at a dosage of 10ml/1kg body weight
Group B (positive Control)	They were given MSG only at a dosage of 6/kg or 0.6g/100g body weight
Group C (Treatment Group)	They were given MSG and Camel Milk only at a dosage of 5ml/kg or 0.5ml/100g body weight
Group D (Recovery Group)	They were given MSG only at a dosage of 6g/kg or 0.6g/100g body weight and left for another 21 days after administration

Specimen/Tissue Collection

As previously described,[12] the rats were weighed and culled using ketamine-xylazine (40 mg/kg – 4 mg/kg) administered intraperitoneally twenty-four (24) hours following the last injection on day 22. As previously reported, the hearts were extracted, weighed, and recorded. To extract the serum from the clot, cardiac blood samples were extracted and centrifuged for 15 minutes at 3000 rpm. Using a laboratory mortar and pestle and a freshly made phosphate buffer solution (pH 7.4), approximately 1g of the left ventricular tissue was homogenized. The homogenate was then refrigerated at -80°C for the biochemical test. For histological analysis, the remaining heart tissue was kept in a vial with 10% neutral buffered formalin.[13]

Biochemical Evaluation

Myeloperoxidase (MPO) Activity

As previously reported by Varthya,[14] myeloperoxidase activity was assessed. At 4°C, the colonic homogenate was centrifuged for 20 minutes at 20,000g. The resulting solution was combined with hydrogen peroxide and 0.167% O-dianisidine hydrochloride. Lastly, the absorbance was measured for four minutes at 460 nm. The quantity of MPO used to convert 1 mM of H_2O_2 to water in one minute is known as one unit of myeloperoxidase activity.

Determination of Nitric Oxide

According to Yucel,[15] nitrite levels were measured using the Griess reagent in order to gauge nitric oxide generation. Following a four-fold dilution with distilled water, plasma samples were deproteinized by adding zinc sulfate until they reached a final concentration of 15 g/L. Following centrifugation (10,000 g for 5 minutes or 1,000 g for 15 minutes), 100 µL of the supernatant and 100 µL of Griess reagent (1 g/L sulfanilamide, 25 g/L phosphoric acid, and 0.1 g/L N-(1-naphthyl)ethylenediamine) were added to a microtiter plate well. A microplate reader was used to detect absorbance at 540 nm after the mixture was incubated for 10 minutes at room temperature. To obtain background values, 25 g/L of phosphoric acid was used. In distilled water, calibration curves were created using potassium and sodium nitrate.

Analysis of C Reactive Protein (CRP)

C-reactive protein (CRP) was measured using a Quantikine ELISA kit (R&D Systems Europe, UK) according to the manufacturer's instructions. The samples were thawed and the reagents were brought to room temperature (21 °C). To perform the test, add 100 µL of 'Assay Diluent' to each well of a 96-well plate, then 100 µL of standard or sample. The plate was incubated for two hours at 21 degrees Celsius before being aspirated and cleaned four times. After adding 200 µL of conjugate, the plate was incubated for a further 2 hours. Following the aspiration and cleaning processes, 200 µL of substrate solution was added and incubated for 20 minutes in the dark. A 50 µL stop solution was added, and absorbance was measured at 450 nm (with a correction filter at 570 nm). A calibration curve was constructed from the standards, allowing for the determination of CRP concentrations in the samples.[16]

Determination of Tumor Necrosis Factor Alpha (TNF-α)

Serum TNF-α levels were tested using a commercial ELISA kit (Boster: EK0525) according to the manufacturer's procedure. Precoated 96-well plates with a monoclonal antibody specific to TNF-α. Standards and test samples were introduced, followed by a biotinylated detection antibody. After washing with PBS or TBS buffer, an avidin-biotin-peroxidase complex was introduced. The reaction was seen using the HRP substrate TMB, which produced a blue hue that became yellow when an acidic stop solution was added. It was found that the yellow color density in samples is proportional to the levels of TNF-α.[17]

Determination of Interleukin-1 Beta (Il-1β)

Invitrogen ELISA kits (catalog number KHC0011) were used to detect serum IL-1β levels, following manufacturer instructions. Microtiter wells were coated with monoclonal antibodies that target IL-1β. Samples and standards were added, allowing IL-1β to attach to the immobilized antibodies. After washing, a biotinylated monoclonal antibody was applied, followed by streptavidin-peroxidase. After a second incubation and washing, the substrate solution was added, and the absorbance was measured at 450 nm after adding the stop solution. The intensity of the color produced is exactly related to the levels of IL-1β.[18]

Determination of Nuclear Factor Kappa Beta (NF-κB)

NF-κB activity was measured with a Trans-AM ELISA kit (Active Motif). Nuclear extracts were produced and attached to a consensus oligonucleotide immobilized on a 96-well plate. The p65 subunit of NF-κB was identified with a particular primary antibody and a horseradish peroxidase-conjugated secondary antibody. Spectrophotometric data were represented as the ratio of absorbance for each experimental condition compared to that of control cells exposed to vehicle alone.[19]

Lipid peroxidation assay
MDA content was determined using the thiobarbituric acid (BA) test.[20] After incubating the obtained cardiac tissue with TBA at 95°C, MDA reacts to generate a colorful complex. The MDA content was determined by measuring absorbance at 532 nm using a spectrophotometer. The concentration is reported as nmol/mg protein.

Measurement of Glutathione (GSH) Levels
Glutathione (GSH) levels were measured in serum and cardiac tissue samples using Ellman's reagent technique. This assay was based on a reaction between GSH's sulfhydryl group and Ellman's reagent (5,5'-dithiobis-(2-nitrobenzoic acid), which resulted in a yellow product. This product's absorbance was measured spectrophotometrically at 412 nm, and GSH concentrations were determined using a standard curve prepared with known GSH values.[21]

Measurement of superoxide dismutase (SOD)
SOD activity was evaluated using the pyrogallol auto-oxidation test at 440 nm for 3 minutes. One unit of SOD activity was defined as the amount of protein that inhibited 50% pyrogallol auto-oxidation. A blank without homogenate served as a control for non-enzymatic pyrogallol oxidation in Tris-EDTA buffer (50 mM Tris, 10 mM EDTA, pH 8.2). SOD activity is measured as U/mg protein.

Measurement of catalase (CAT)
By measuring the hydrolysis of H_2O_2 and the resulting drop in absorbance at 240 nm over the course of three minutes at 25 °C, CAT activity was ascertained. Samples were diluted 1:9 with 1% (v/v) Triton X-100 before CAT activity measurement. The CAT activity is expressed as mmol/mg protein.[22]

Measurement of glutathione peroxidase (GPx)
H_2O_2 was used as a substrate in accordance with the procedure to test GPx activity. For three minutes, the oxidation rate of NADPH at 240 nm served as an indirect indicator of the process. When hydrogen peroxide was added to 0.1 M Tris buffer (pH 8.0), a blank without homogenate was utilized as a control for the non-enzymatic oxidation of NADPH. The unit of measurement for enzyme activity was nmol/mg protein.[23]

Glutathione S-Transferase (GST) Activity Assay
The activity of the detoxifying enzyme glutathione S-transferase (GST) in heart tissue homogenates was determined by measuring the conjugation of the substrate 1-chloro-2, 4-dinitrobenzene (CDNB) with reduced glutathione (GSH). This reaction produces a dinitrophenyl thioether that can be monitored spectrophotometrically at 340 nm. The rate of increase in absorbance is directly proportional to the GST activity in the sample.[24]

Analysis of Caspase-3
A colorimetric test using a particular caspase-3 activity assay kit was used to measure the activity of caspase-3.[25] In order to investigate apoptotic activity, the heart tissue was homogenized in ice-cold lysis solution. The supernatant was then obtained by centrifugation and used for the test. A microplate reader was used to measure the absorbance at 405 nm with the intent to quantify caspase-3 activity.

Diphenylamine (DPA) colorimetric assay
A modified diphenylamine (DPA) colorimetric test was used to quantify cardiac DNA fragmentation and ascertain the ratio of intact to fragmented DNA in cardiac tissue. In short, samples from the left ventricle were mixed in lysis buffer and centrifuged to separate the intact genomic DNA (pellet) from the fragmented DNA (supernatant). To create a blue-green chromogen, both fractions were precipitated with cold trichloroacetic acid, cleaned, and then reacted with DPA reagent for 16 hours at 37°C in an acidic environment. The ratio of fragmented DNA absorbance to total DNA absorbance, represented as a percentage, was used to compute the DNA fragmentation index after absorbance was measured at 600 nm.[26]

Histopathological Analysis
The heart collected were fixed in Bouin's solution, dehydrated with ascending ethanol series, and then cleared with toluene. The heart were embedded at room temperature and blocked in paraffin wax incubated in a 60° C incubator overnight. Then, hematoxylin and eosin (H&E) stain was applied to about 5 µm thick paraffin sections of the heart. The stained slides were examined under a light microscope, and photomicrographs were taken at 100x and 400x magnification.[27]

Statistical Analysis
Data analysis was conducted using GraphPad Prism software (version 9.0, GraphPad Software, Inc.). Mean values with standard deviation (mean ± SD) are used to report all data. For multiple comparisons between groups, Tukey's post hoc test was used after one-way analysis of variance (ANOVA). The threshold for statistical significance was set at $P < 0.05$.

Results and Discussion

Effect of Camel Milk on Body Weight and Heart Weight in MSG-Exposed Rats
As shown in Table 2, there was no significant difference in initial and final body weights across all groups; however, body weight change (BWC) revealed marked alterations. MSG exposure (Group B) led to a significant drop in BWC compared with control rats, suggesting that MSG may impair overall growth or metabolic regulation. This agrees with earlier studies linking chronic MSG consumption to metabolic dysfunction and abnormal weight regulation. In contrast, rats receiving camel milk (Group C) showed a significant improvement in BWC compared with Group B, indicating that camel milk exerts restorative effects on weight. This could be attributed to its rich composition of bioavailable vitamins, minerals, insulin-like proteins, and antioxidant peptides that enhance nutrient utilization and metabolism. Interestingly, Group D (recovery) rats exhibited partial restoration, though BWC remained lower than in Group C, suggesting that concurrent supplementation during MSG exposure may be more beneficial than post-exposure recovery. Heart weight (HW) also declined significantly in MSG-treated rats, pointing toward possible cardiomyocyte atrophy or tissue loss, a finding consistent with previous studies. Camel milk restored HW closer to control levels, particularly in Group C, suggesting protective or regenerative effects against MSG-induced cardiac deterioration.

Table 2: Effect of CM on Body weight and heart weight in rats exposed to MSG

	Group A (Control)	**Group B (MSG-exposed)**	**Group C (MSG-exposed+CM)**	**Group D (MSG-exposed-R)**
IBW (g)	193.00 ± 10.58	199.70 ± 7.57	196.70 ± 4.73	193.00 ± 3.61
FBW (g)	216.70 ± 11.93	210.30 ± 7.64	230.30 ± 5.51	215.00 ± 4.58
BWC (g)	23.67 ± 1.53	10.67 ± 1.53*	33.67 ± 1.53*+	22.00 ± 1.00+&
HW (g)	1.27 ± 0.12	0.83 ±0.06*	1.17 ± 0.06^{+}	1.07 ± 0.06*$^{+}$

*Values are mean ± SD of 3 replicates, where *P < 0.05 vs control, $^{+}$P < 0.05 vs MSG-exposed, and $^{\&}$P < 0.05 vs MSG-exposed+CM, and IBW, FBW, BWC, and HW were initial body weight, final body weight, body weight change, and heart weight, respectively.*

Effect of Camel Milk on Inflammatory Markers (MPO, NO, CRP, IL-1β, TNF-α, NF-κB)
Biochemical assays demonstrated that MSG exposure significantly increased cardiac MPO, NO, and CRP (Figure 1). Elevated MPO indicates oxidative stress and neutrophil infiltration, while higher NO and CRP reflect nitroative stress and systemic inflammation. These changes align with the pro-inflammatory actions of MSG and its association with cardiovascular dysfunction. Camel milk

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supplementation markedly reduced these markers in Group C, suggesting strong anti-inflammatory activity. Group D also showed improvements, though levels remained higher than Group C, highlighting the advantage of prophylactic supplementation. Similarly, pro-inflammatory cytokines (IL-1β, TNF-α) and transcription factor NF-κB (Figure 2) were elevated in MSG-exposed rats, reflecting heightened myocardial inflammation and remodeling. Camel milk significantly downregulated these mediators, likely through inhibition of NF-κB activation. Group D rats showed partial reductions compared with MSG-only rats, but values were slightly elevated compared with Group C, reinforcing the benefit of concurrent treatment.

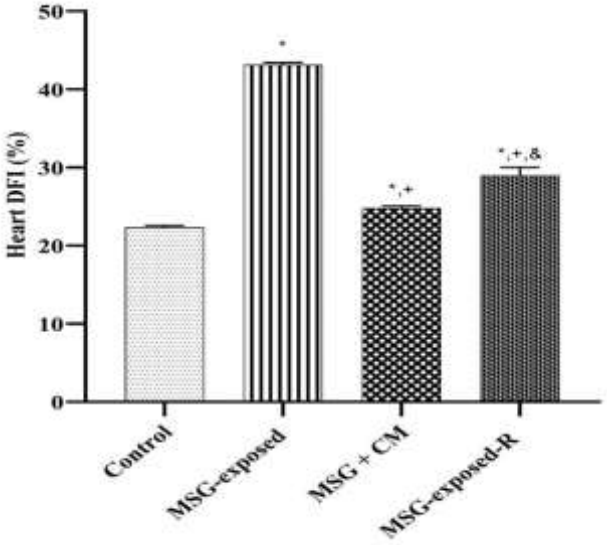

Figure 1: Effect of CM on Cardiac MPO, NO, and CRP in MSG exposed rats. Values are expressed as mean ± SD of 3 replicates, where *P < 0.05 vs control, + P < 0.05 vs MSG-exposed, and & P < 0.05 vs MSG-exposed+CM

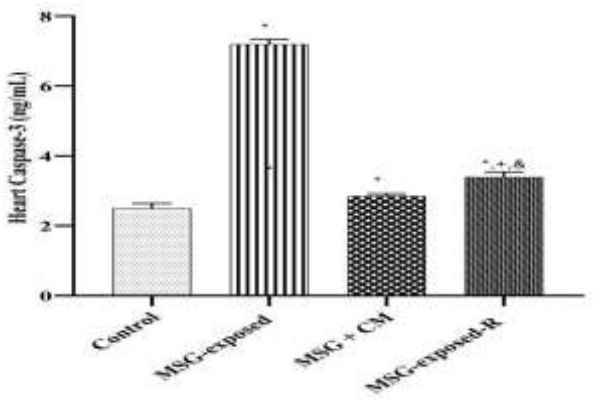

Figure 2: Effect of CM on Cardiac IL-1β, TNF-α, and NF-kβ in MSG exposed rats. Values are mean ± SD of 3 replicates, where *P < 0.05 vs control, + P < 0.05 vs MSG-exposed, and & P < 0.05 vs MSG-exposed+CM.

Effect of Camel Milk on Oxidative Stress (MDA, CAT, SOD, GSH, GST, GPx)

MSG exposure disrupted redox balance, as shown by increased malondialdehyde (MDA, Figure 3A) and reduced antioxidant enzyme activities (CAT and SOD, Figures 3B–C). Elevated MDA indicates lipid peroxidation, while the depletion of CAT and SOD reflects weakened antioxidant defenses. These alterations confirm that MSG induces significant oxidative stress in cardiac tissue. Camel milk supplementation (Group C) effectively lowered MDA and improved antioxidant enzyme activity, consistent with its richness in antioxidant peptides, vitamins A and C, glutathione, and zinc. Group D rats exhibited partial but incomplete restoration, indicating some reversibility after MSG insult but less efficacy compared with concurrent administration. Likewise, cardiac reduced glutathione (GSH), glutathione-S-transferase (GST), and glutathione peroxidase (GPx) (Figure 4) were all depleted by MSG exposure, impairing detoxification pathways. Camel milk significantly restored these markers in Groups C and D, though Group D remained slightly below control levels. This underscores camel milk's role in enhancing glutathione metabolism and re-establishing redox balance.

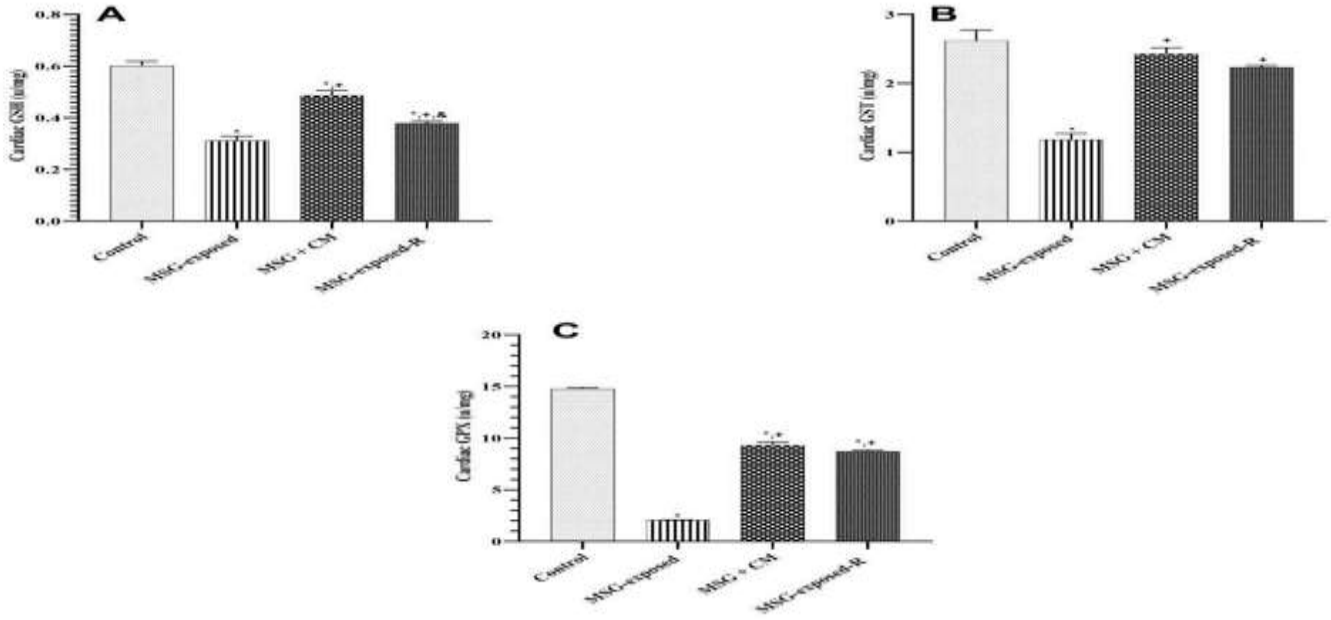

Figure 3: Effect of CM on Cardiac MDA, CAT, and SOD in MSG exposed rats. Values are mean ± SD of 3 replicates, where *P < 0.05 vs control, + P < 0.05 vs MSG-exposed, and & P < 0.05 vs MSG-exposed+CM.

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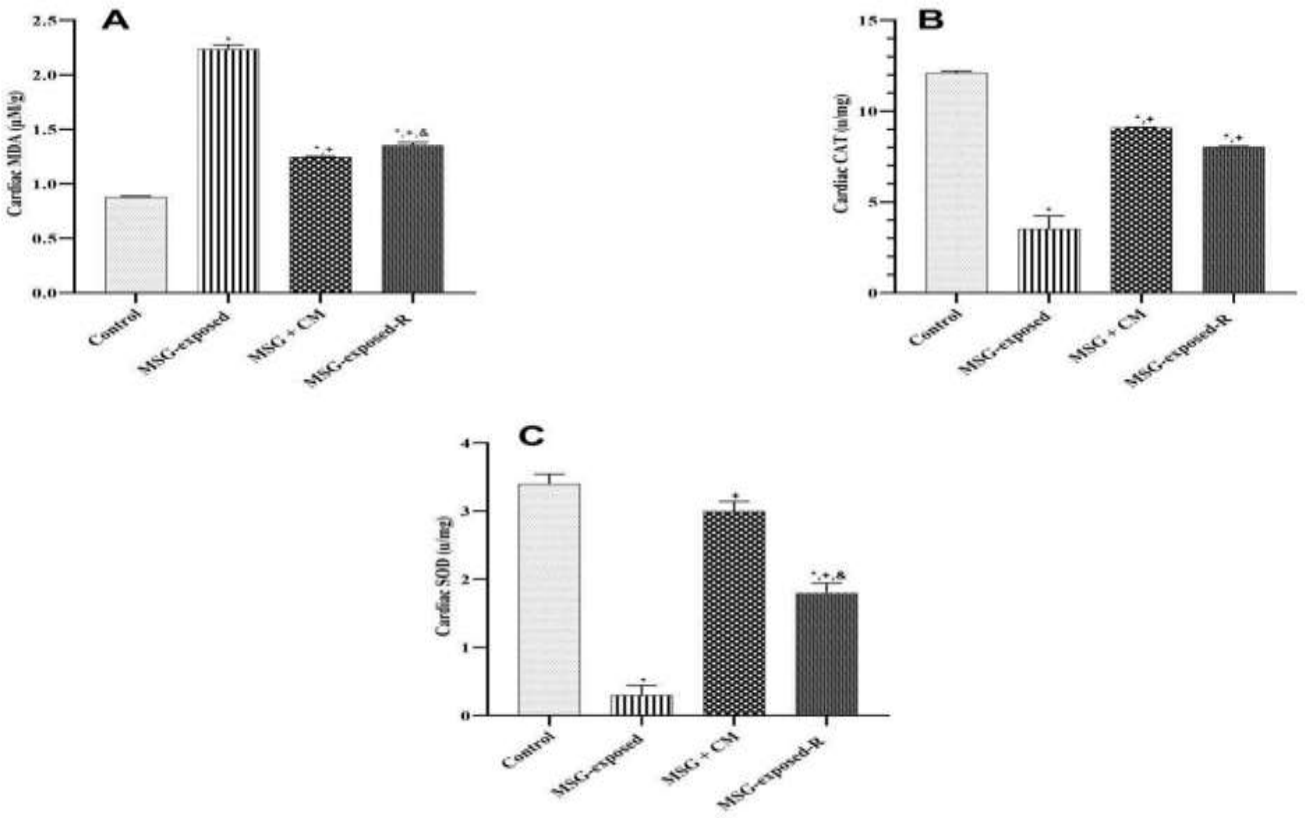

Figure 4: Effect of CM on Cardiac GSH, GST, and GPx in MSG exposed rats. Values are mean ± SD of 3 replicates, where *P < 0.05 vs control, + P < 0.05 vs MSG-exposed, and & P < 0.05 vs MSG-exposed+CM.

Effect of Camel Milk on Apoptosis and DNA Integrity

Apoptotic markers were notably affected by MSG exposure. Caspase-3 and DNA fragmentation index (DFI) (Figures 5 and 6) were significantly elevated in MSG-only rats, indicating activation of apoptosis and genotoxic damage. Camel milk supplementation significantly reduced both markers, suggesting anti-apoptotic and DNA-protective effects. Group D rats showed reduced levels compared to Group B, but values were higher than Group C, highlighting again the greater effectiveness of concurrent administration.

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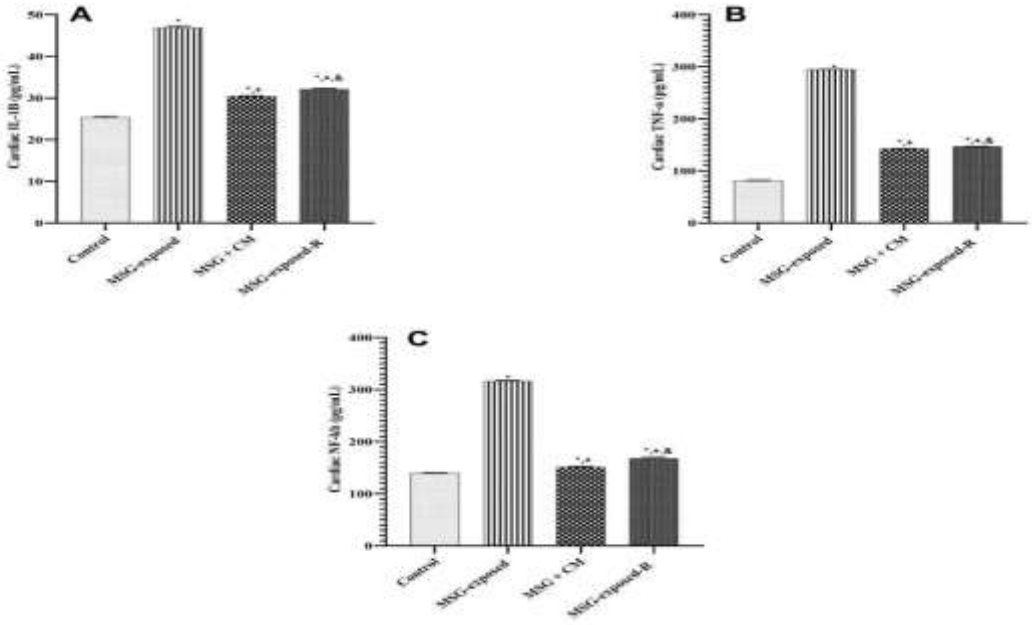

Figure 5: Effect of CM on Heart Caspase-3 in MSG exposed rats. Values are mean ± SD of 3 replicates, where *P < 0.05 vs control, + P < 0.05 vs MSG-exposed, and & P < 0.05 vs MSG exposed + CM.

Histopathological Findings

Histological assessment of the myocardium (Figure 7) corroborated biochemical results. Control rats exhibited intact myofibrils, clear interstitial spaces, and normal coronary vasculature. MSG-treated rats showed vascular congestion, disrupted myofibrillar arrangement, and inflammatory cell infiltration. In contrast, camel milk-treated rats (Group C) demonstrated preserved myocardial organization, reduced congestion, and intact fibers, confirming structural cardioprotection. Group D rats showed partial histological recovery, with minor inflammatory infiltration but largely preserved architecture.

exposed+CM.

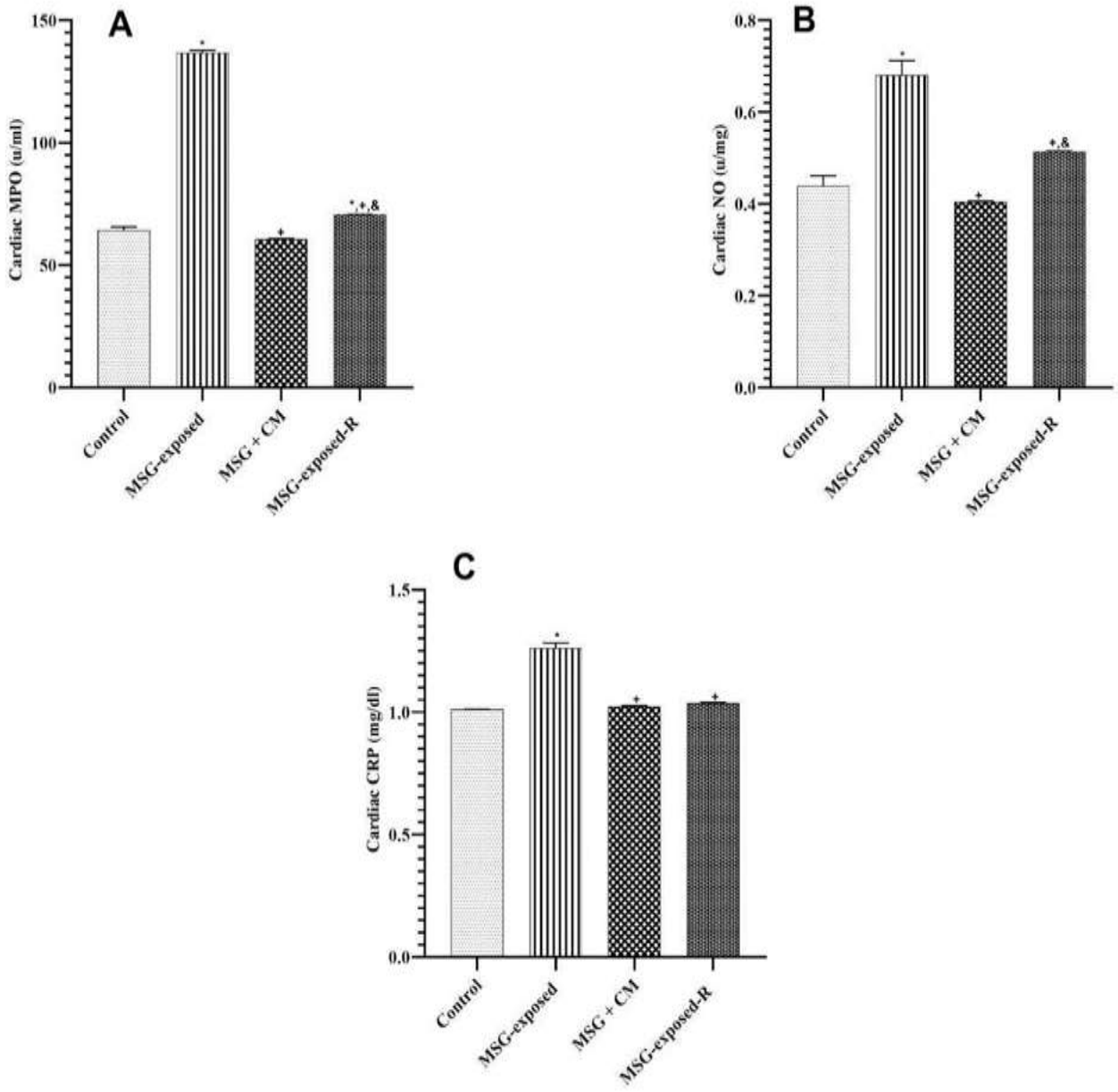

Figure 6: Effect of CM on Heart DFI in MSG exposed rats. Values are mean ± SD of 3 replicates, where *P < 0.05 vs control, + P < 0.05 vs MSG-exposed, and & P < 0.05 vs MSG-

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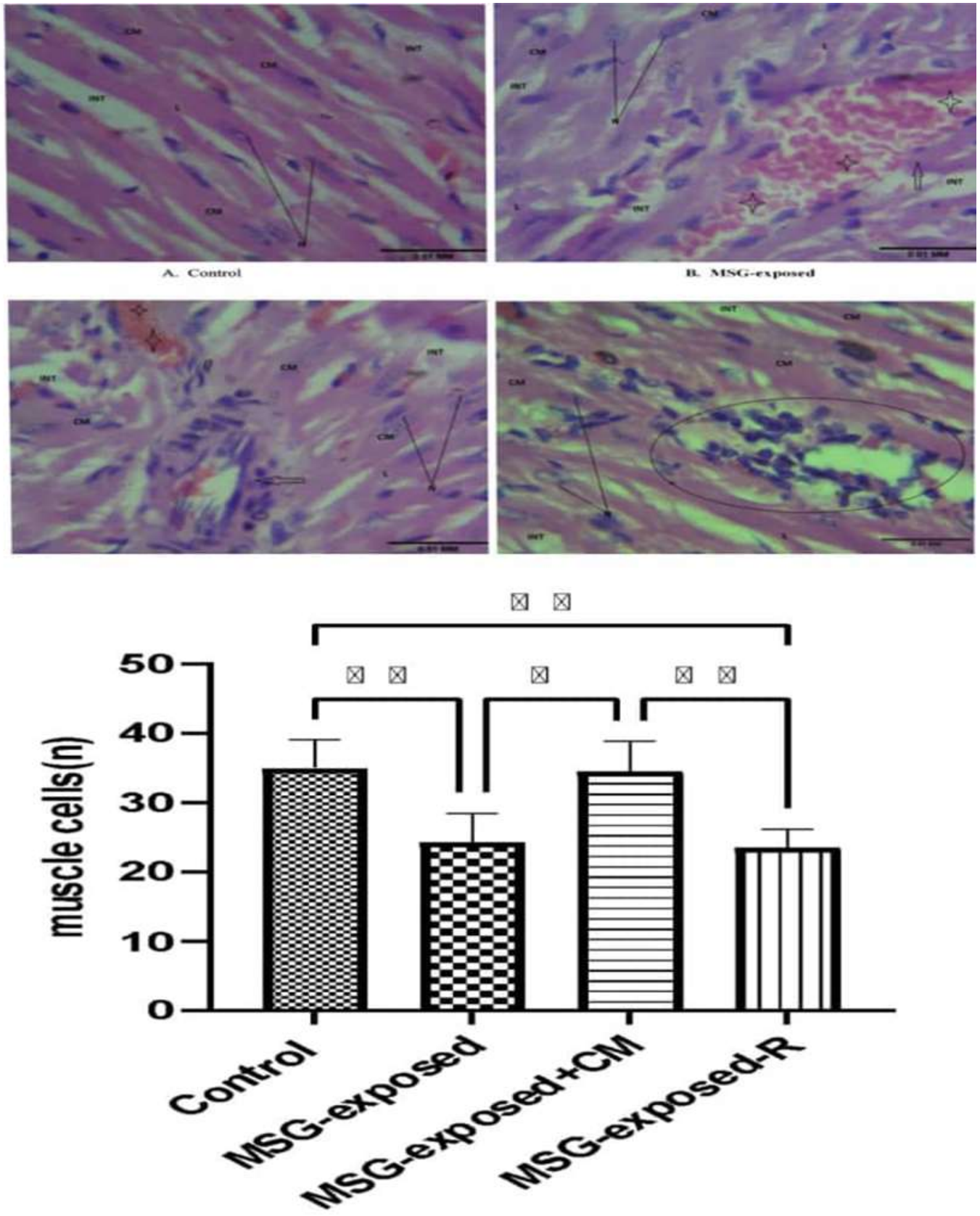

Figure 7: Photomicrograph of cardiac sections stained by H & E (Mg ×400) and image quantification of cardiac cell. Where CM = cardiac muscles; L = Lateral branch; N = Nucleus; INT = Interstitiun; STAR = Congested coronary vessels; Circle = Inflammatory cells

Conclusion

Improvements in body and heart weights, decreases in oxidative stress and inflammatory markers, restoration of antioxidant enzyme activity, and preservation of myocardial structure are all indicators of the substantial cardioprotective effects of camel milk in MSG-induced cardiac toxicity. Its potential significance in avoiding and reducing cardiovascular damage is highlighted by the fact that early and continuous administration of CM is more beneficial than post-exposure therapy. These results offer a strong justification for more research on the clinical suitability of camel milk as a dietary supplement or medicinal agent in the treatment of inflammatory and oxidative heart diseases.

Conflict of Interest

The authors declare no conflict of interest.

Authors' Declaration

The authors hereby declare that the work presented in this article is original and that any liability for claims relating to the content of this article will be borne by them.

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