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Camel Milk Attenuates Hepatic Inflammation in Monosodium Glutamate-Exposed Rats

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ABSTRACT

Monosodium glutamate (MSG) is a white crystalline food additive that is widely used, and the accumulating evidence suggests that it is an inducible of hepatic inflammation. Camel milk is a natural diet product containing antimicrobial, antioxidant and immunomodulatory qualities. This paper was able to assess the protective properties of camel milk against MSG-induced hepatic inflammation in the rats. The sample consisted of 24 male Wistar rats (average weight 195g) that were separated into four groups. Group A was treated with distilled water (1 ml/kg), Group B was treated with MSG (60mg/kg), Group C was treated with camel milk (5 ml/kg) 15 minutes after MSG treatment, and Group D was used as a control group and monitored within 21 days after the MSG treatment. Measures of hepatocytic inflammatory markers, myeloperoxidase (MPO), nitric oxide (NO), C-reactive protein (CRP), tumor necrosis factor-a (TNF-a), interleukin-1b (IL-1b), and nuclear factor-kB (NF-kB), and histological examination were done. Serum inflammatory markers greatly increased and there was significant histological changes in exposure to MSG as opposed to the control group. Treatment with camel milk significantly decreased these indices of inflammation and led to liver microarchitecture. Despite the recovery group recording some inflammatory markers reduction, the effect was not as high as in the camel-milk-treated animals. The results demonstrate great anti-inflammatory properties of camel milk on hepatic inflammatory markers of MSG-induced hepatic inflammation. Such results improve the knowledge of the harmful effect of MSG and emphasize the possibilities of camel milk as a natural medication of liver inflammation.

Keywords: Monosodium glutamate, camel milk, inflammatory markers, liver histology.

Introduction

Inflammation is the biological reaction of a living tissue in response to injury or infection, which may be a significant process of healing, or as part of an immune response. It is (Sherman *et al.*, 2015) that includes changed blood circulation, augmented vascular leaksage and migration of leukocytes to the location of harm (Pahwa *et al.*, 2024). The liver inflammation is usually known as hepatitis. Knowles E. Merriam. (2022). As WHO (2015) says, 1.34 million deaths in 2015 were caused by hepatitis and can be acute or chronic depending on the course of the disease and circumstances. The MPO, NO, CRP, TNF-a, IL-1b, and NF-kB are some molecules as they increase rapidly in hepatic inflammation and are used clinically to diagnose, monitor, and assess responses to treatment (Mullaicharam *et al.*, 2014). Some medical conditions or viruses, heavy alcohol use, toxins, and some medications may cause hepatic inflammation (Younossi *et al.*, 2018). A high concentration of these

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substances may be taken to indicate an inflammation which is undergoing. Hence, they are employed in the diagnosis of the inflammatory damage, tracking them, and determining the efficiency of the anti-inflammatory treatment (Mullaicharam, 2014). Fibrosis may result due to unregulated or persistent liver inflammation (Menzel *et al.*, 2021).

The least produced is the camel milk which constitutes 0.5 percent of the global production of dairy milk. It has much more immunoprotective factors including lactoferrin and lysozyme (Mansour *et al.*, 2017; Konuspayeva *et al.*, 2020). Its vitamins and bioactive elements have been associated with antihypertensive, antidiabetic, anticancer, antioxidant and immunomodulatory effects, which were traditionally used to treat such diseases as dropsy, asthma, anemia and diabetes (Abdelrahman *et al.*, 2020).

Monosodium glutamate (MSG), a flavouring substance commonly incorporated into food has been investigated to cause the inflammation of the liver. Besides the MSG use as a food additive, it is naturally present in most foods like tomatoes, vegetables, and cheeses, and it is therefore unavoidable in foods (Kesherwani *et al.*, 2024). In the meantime, recent findings indicate that even the dosages of 100mg/kg of MSG may trigger inflammation (Kesherwani *et al.*, 2024). The high prevalence of MSG as a food additive to enhance the flavor of food has been of concern because it could cause inflammation of the liver and accompanying health complications. It is also known that MSG causes liver damage and hepatic inflammation, yet natural interventions that can reverse these effects are poorly studied. Camel milk has anti-inflammatory effects, and this could be protective, but it is not clearly known whether it will have any effect on hepatic inflammatory markers in MSG-exposed helpless male Wistar rats. This experiment examines the capacity of camel milk in decreasing hepatic inflammation resulting out of MSG in male Wistar rats.

Literature Review

Conceptual and Theoretical Review

Monosodium glutamate (MSG) is a sodium salt of L-glutamic acid used extensively as a flavour enhancer since it is able to stimulate umami receptors and enhance the palatability of food. Biochemically, MSG is quickly broken down to sodium and free glutamate when ingested, thus raising levels of glutamate in the system which are not regulated by proteins (Walker and Lupien, 2000). Despite glutamate being a central metabolite as well as a neurotransmitter, its excessive levels as a free amino acid have long been the recognised dietary protein-derived glutamate has been recognised to be biologically active in a vastly different way. This difference is conceptually the foundation of experimental studies of MSG toxicity especially at high-dose or repeated dose which overwhelms normal metabolic buffering systems (Olney, 1969; Fernstrom, 2013). Studies show that the strongest evidence showing MSG-induced systemic toxicity has been in animal models, where repeatable effects have been shown in the areas of neurological, metabolic and hepatic systems. Initial research findings determined that exposure of fetuses to neonatal MSG causes lesions in the arcuate nucleus of the hypothalamus that results in lifelong disregulation of the appetite, energy expenditure and endocrine signalling (Olney, 1969; Hermanussen *et al.*, 2006). These key changes are closely interconnected to peripheral effects of metabolism such as hyperinsulinaemia, dyslipidaemia, and obesity that have a secondary impact on the hepatic functionality. This has however been contradicted by further experiments in adult rodents as to MSG being able to cause hepatic injury without developmental hypothalamic injury, meaning that the liver is also a direct toxicological target (Onyema *et al.*, 2006; Farombi & Onyema, 2006).

Liver damage associated with MSG exposure is always marked with increased serum levels of transaminases, histology, and oxidative stress biomarkers. Rat studies have shown severe improvements in ALT and AST and hepatocellular degeneration, sinusoidal

swellings and inflammatory infiltration following a repetitive dosage of MSG (Onyema *et al.*, 2006; Eweka and Om'Iniabohs, 2007). These results indicate that membrane integrity and leakage of enzymes have been compromised, and not isolated membrane metabolic adaptation. Notably, the effects of such changes are evident even in comparatively short exposure periods, which confirms the idea that MSG triggers early cellular stress responses instead of being a trigger to metabolic dysfunction (Farombi & Onyema, 2006).

Empirical review

The strongest argument-backed mechanistic pathway in MSG-induced hepatotoxicity is oxidative stress. In several studies, the exposure to MSG enhances the products of lipid peroxidation (malondialdehyde (MDA)) levels but also reduces the levels of endogenous antioxidants, including reduced glutathione (GSH), superoxide dismutase (SOD), and catalase (Onyema *et al.*, 2006; Farombi & Onyema, 2006). The sensitivity of the liver to such redox imbalance is due in part to the fact that liver has a high density of mitochondria and is the key site of amino acid oxidation, which increases ROS production in the case of glutamate homeostasis disequilibrium. These oxidative alterations do not follow definite histological alterations but come before them, meaning that they are causative, rather than consequential, to hepatic injury (Hashem *et al.*, 2012). Furthermore, it is shown that mitochondrial dysfunction constitutes one of the most crucial downstream responses of the oxidative stress induced by MSG. The surplus glutamate in the cells disrupts the mitochondrial electron transport chain resulting to the inhibition of the ATP production and the excess leakage of electrons, which even further enhance the generation of ROS (Fonnum, 1984; Farombi & Onyema, 2006). The ultrastructural observation shows that hepatocytes exhibit swollen mitochondria and broken cristae after exposure to MSG, a morphological confirmation of impaired functions (Eweka and Om'Iniabohs, 2007). This mitochondrial susceptibility forms a feed-forward mechanism, where oxidative stress and bioenergetic deficiency supports each other, eventually reducing the point of cell death.

Inflammatory signalling pathways also enhance hepatic damage by MSG, and nuclear factor-kB (NF-kB) turns out to be a key control node. The use of MSG triggers the NF-kB, which leads to an augmentation in the expression of pro-inflammatory cytokines in the liver, including TNF-a, IL-6, and IL-1b (Hashem *et al.*, 2012; Eweka and Om'Iniabohs, 2007). These cytokines recruit immune cells but also impair insulin signalling and mitochondrial function hence the association of metabolic and oxidative injury to inflammation. Direct evidence of inflammasomes activity is not found in MSG models; however, convergence of ROS productions, mitochondrial injury, and cytokines release is consistent with known triggers of NLRP3 inflammasome signalling in hepatic inflammation (Kubes & Mehal, 2012). Thus, the results of cell death pathways are the end results of long-term oxidative and inflammatory stress in the liver of MSG. Others show the augmentation of caspase-3 action as well as the ratios of Bax/Bcl-2 indicating the activation of the intrinsic apoptotic way (Hashem *et al.*, 2012; Onyema *et al.*, 2006). The excess oxidative damage is beyond the capability of apoptotic pathways, which leads to necrotic cell death, which is a cause of the collapse of the architecture and the loss of functional mass in the liver. A combination of both the apoptotic and necrotic changes highlights the degree of cellular stress caused by MSG and places its hepatotoxicity in the context of the more generalized models of toxicant-induced liver damage (Jaeschke *et al.*, 2012).

Understanding dose-response relationships and exposure pathways is crucial when interpreting MSG toxicity. Most animal experiments employ doses that are significantly higher than those typically consumed by people, typically either orally or intraperitoneally at 2–8 g/kg body mass (Walker & Lupien, 2000). Due to developmental sensitivity and non-physiological pathways of exposure, mechanistically informative models in neonatology have low translational

applicability. However, recurrent MSG dose can cause hepatic oxidative and inflammatory reactions without the confounding effect of neurodevelopmental change, according to adult oral exposure models. This is biologically reasonable but necessitates careful extrapolation (Fernstrom, 2013). Hence, in this context of toxicology camel milk has gained more and more interest as a functional food possessing hepatoprotective properties. The macronutrient content of the camel milk is significantly different as compared to bovine milk because it has low fat and lactose and high Vitamin C and protective proteins (Konuspayeva *et al.*, 2009). The protein fraction is defined by the lack of b-lactoglobulin and the increase of immunoglobulins, lactoferrin and lysozyme, which are bioactive factors that can be used in the modulation of inflammation and oxidative stress (El-Agamy, 2007). The antioxidant potential of the camel milk is especially applicable in MSG induced hepatic injury. In a number of experimental models of hepatic and systemic toxicity, camel milk supplementation has been found to decrease lipid peroxidation and recover antioxidant enzyme activity (Korish *et al.*, 2013; Al-Asmari *et al.*, 2015). It has high vitamin C, which is a direct scavenger of ROS and endogenous antioxidant enzyme systems are supported by trace elements like zinc and selenium. A product of these effects is that camel milk is not only an exogenous antioxidant, but is also a regulator of cellular redox homeostasis (Konuspayeva *et al.*, 2009).

The anti-inflammatory effects are also provided to reinforce the argument that camel milk is a viable protective factor against MSG-induced liver inflammation. Lactoferrin, which is the most significant protein in camel milk, is reported to inhibit the activity of NF-kB, thereby preventing the production of pro-inflammatory cytokines in the hepatic and immune cells (Actor *et al.*, 2009). Immunoglobulins and bioactive peptides also have the ability to neutralize the amplification of inflammation by regulating the innate immune responses. Considering that NF-kB and cytokines are the key contributors of MSG hepatotoxicity, these characteristics offer a consistent mechanistic correlation between the intake of camel milk and diminished hepatic injury (Korish & Arafah, 2013).

Research Gaps

Limited empirical studies that directly looked at camel milk in MSG-exposed rats demonstrate hepatoprotective effects that are consistently observed. They are normalisation of the ALT and AST levels, decrease of MDA levels, recovery of GSH, SOD, and catalase activity, and maintenance of hepatic histoarchitecture (Al-Asmari *et al.*, 2015). Molecular studies also reveal that pro-inflammatory cytokine down-regulation and apoptotic markers attenuation occur. It is important to note that these effects are greatest when camel milk is used prophylactically or concomitantly, which implies that its major clinically meaningful effect is interruptive of initial oxidative and inflammatory cascades and not reversive of tissue injury. Similarly, the dose and timing seem to be crucial factors that determine the camel milk efficacy. The biochemical and histological protection is greater in studies using continuous or pre-exposure supplementation compared to those where treatment commences after the injury has occurred with MSG (Korish & Arafah, 2013). Nonetheless, there are limited studies that carry out a formal dose-response study or even compare various modes of processing, e.g. raw versus pasteurised milk. This methodological weakness restricts the definition of minimum effective doses or a standardisation of interventions across studies (Konuspayeva *et al.*, 2009).

Furthermore, the protective effects of camel milk seem to meet mechanistically on interdependent antioxidant, antiinflammatory and antiapoptotic pathways. The emerging evidence may indicate the possible stimulation of the Nrf2 antioxidant response pathway, but it is not confirmed directly in MSG models (Al-Asmari *et al.*, 2015). Simultaneous inhibition of NF-kB signalling can offer a reasonable explanation of lower cytokine release and inflammatory infiltration. Mitochondrial stability and regulation of the apoptotic equilibrium also help provide a multi-targeted protective profile in harmony with the intricate nutritional interventions

(Actor *et al.*, 2009). Although the current evidence base of animals shows promising results, it is limited. The majority of the researches use limited sample sizes, are not blinded, and undercharacterise the composition of camel milk which has been shown to differ depending on location, diet, stage of lactation, and processing (Konuspayeva *et al.*, 2009). The lack of dose-ranging and mechanistic specificity restricts causes and reproducibility. Furthermore, not many studies replicate their results in other independent laboratories, which is a necessary aspect of translational confidence (Jaeschke *et al.*, 2012). Translational relevance should be taken at face value, that is. Exposure of human MSG is hardly close to experimental doses of exposure in animal experiments, and there is a wide variation in patterns of camel milk consumption. Still, the hepatic injury, caused by MSG, has a mechanistic overlap with more general aetiologies, including non-alcoholic fatty liver disease, especially in terms of oxidative stress and inflammatory signalling (Kubes & Mehal, 2012). In this light, the camel milk can be used as more of an antidote to MSG but as a model of functional food in terms of regulating the hepatic redox and inflammatory systems.

Materials and Methods

Chemicals and Animal Procurement

To guarantee experimental dependability and repeatability, all substances employed in the investigation were of analytical grade and purchased from reputable commercial suppliers. Before processing, the crystals of monosodium glutamate (MSG), which were bought from Mich Mikedenson, were verified to be free of any obvious impurities. Fresh camel milk was purchased locally in Nigeria from reputable suppliers, such as Simply Precious Enterprise and Nigeria Enterprises Limited in Ilorin, Kwara State, and Badagry, Lagos State. The use of locally acquired camel milk is consistent with earlier experimental research that used fresh, unprocessed milk to maintain bioactive components and mimics real-world consumption patterns (Bastaki *et al.*, 2016). Before being administered, milk samples were transported in a sanitary manner and kept at 4 degrees Celsius to prevent microbial growth and the deterioration of components that are sensitive to heat or light.

Drug Preparation

A new MSG stock solution was prepared by dissolving 60g of MSG crystals in 1,000ml of distilled water, yielding a final concentration of 60 mg/ml. This solution was given orally at a dosage of 1 ml/kg body weight, or 60 mg/kg, in compliance with established protocols that have been demonstrated to cause consistent biochemical and histological changes in the liver without causing significant mortality (Khan *et al.*, 2022). Oral camel milk was given at a dose of 5 ml/kg body weight, which was within a physiologically acceptable volume range for rats and in line with previously confirmed hepatoprotective investigations (Bastaki *et al.*, 2016). Oral gavage was used for all treatments to guarantee precise dosage and reduce inter-animal variability.

Animals

Twenty-four adult, healthy male Wistar rats weighing between 150 and 200 g were used in the experiment. The College of Medicine at Ekiti State University in Ado-Ekiti provided the animals. In order to prevent hormonal fluctuations linked to the oestrous cycle, which could affect oxidative stress and inflammatory reactions, male rats were chosen. Before being utilised in experiments, the animals were given a five-day acclimatisation period and kept in well-ventilated cages under typical laboratory settings. Rats were given regular rodent food (Top Feed Nigeria Limited) and unlimited access to potable water during this time.

Ethical Approval

Strict adherence to ethical guidelines for the care of laboratory animals was maintained throughout all experimental procedures. The Ekiti State University Department of Physiology Research Ethics Committee granted ethical approval (Approval No. EKSU/P100/2024/002), and all protocols adhered

to the principles of reduction, refinement, and replacement as well as the National Institutes of Health's guidelines for the care and use of laboratory animals.

Experimental Design

The rats were assigned randomly to four groups (n=6 each):
Group A: Control - distilled water (1 ml/kg) in 21 days.
Group B: MSG (60 mg/kg) for 21 days
Group C: MSG (60mg/kg) + camel milk (5ml/kg) after 15mins.
Group D: MSG 60 mg/kg 21 days followed by 21 days of no treatment no mortality was observed all through the research period.

Sample Collection and Biochemical Assay

Rats were sacrificed and weighed 24 hours after the last treatment through intraperitoneal injection of ketamine-xylazine (40/4mg/kg) (Hassan *et al.*, 2021). Biochemical analysis of liver tissues was done by harvesting, weighing and homogenizing liver tissues in phosphate buffer (pH 7.4). Biochemical analysis of the inflammatory markers was performed by assays of levels of MPO, CRP, TNF-a, IL-1b, and NF-kB using rat-specific ELISA kits (MyBioSource). and storing the homogenate at 800C. To evaluate the clinical necessity of the liver tissue, it was fixed in an adequate level of 10 percent neutral buffer formalin and kept at room temperature to assess it historically.

Histopathological Analysis

Following the sacrifice of rats, the liver was cut and sliced into small pieces and preserved in 10% formalin and further dehydrated in the various increasing alcoholic concentrations. (Omodanisi *et al.*, 2017) and sliced at 5 um thickness (Elhag *et al.*, 2017). The staining of slides using hematoxylin and eosin was done and assessed by a histopathologist. Photo-micrographed stained slides were checked and viewed by a specialist in the field.

Statistical Analysis

GraphPad Prism version 9.0, was used to statistically analyse the experimental data. To represent central tendency and variability within each experimental group, all quantitative results were presented as mean ± standard deviation (SD). One-way analysis of variance (ANOVA), which is suitable for comparing several independent groups under a single experimental factor, was used to assess differences between groups. Tukey's post hoc multiple comparison test was used to find particular group-wise differences while adjusting for type I error when the ANOVA showed a significant overall effect. Statistical significance was defined as a probability value (P) of less than 0.05, which meant that observed differences were unlikely to have happened by accident.

Results

Table 1: indicates the camel milk (CM) effect on Body weight and liver weight of MSG exposed rats. mean ± SD was used as values, *P < 0.05 vs control, +P < 0.05 vs MSG-exposed, and P < 0.05 vs MSG-exposed+CM were initial body weight, final body weight, body weight change, and liver weight, respectively. Figure 2 expressed the results of hepatic IL-1b, TNF- a, and NF-kb. Both hepatic IL-1b and TNF-a results (Figures 1A and 1B) demonstrated a significant ($p<0.05$) increase in all other populations of rats compared to the control rats and significant ($p<0.05$) decrease in either the group C and group D rats compared to the group B rats. The outcome of hepatic NF-kb (Figure 1C) showed that there is a significant ($p<0.05$) high level of group B and D rats as compared to the control rats and a significant ($p<0.05$) low level of group C and D rats as compared to group B rats.

The photomicrograph (Figure 2(A)) illustrates the liver tissue which is mainly constituted of the hepatic parenchyma and portal space. The portal region (circle), which consists of the branches of hepatic portal vessels and bile duct (BD) looks normal, and it suggests that the liver tissue has a normal histomorphology. Figure 2 (B), which is a

photomicrograph, depicts the liver tissue consisting of the hepatic parenchymal and portal regions. The portal area (circle), which is a combination of the branches of the hepatic portal vessels (HPV), bile duct (BD) and exhibited vascular congestion (star) and slight inflammatory cell infiltration (arrowhead), suggesting periportal hepatitis of the liver tissue. Figure 2(C) is a photomicrograph that demonstrates the liver tissue that consists of hepatic parenchyma and portal predominantly. The Portal Region (Circle) which is made up of branches of the hepatic portal vessels and bile duct (BD) looks normal, thus suggesting normal histomorphology. Figure 2(D) depicts the liver tissue which is mainly composed of the hepatic parenchymal tissue and the portal tissue. The portal region (circle), which is made up of branches of the hepatic portal vessels (HPV), bile duct (BD) displayed slight inflammatory cell invasion (arrowhead) suggesting a hepatic cellular response to injury.

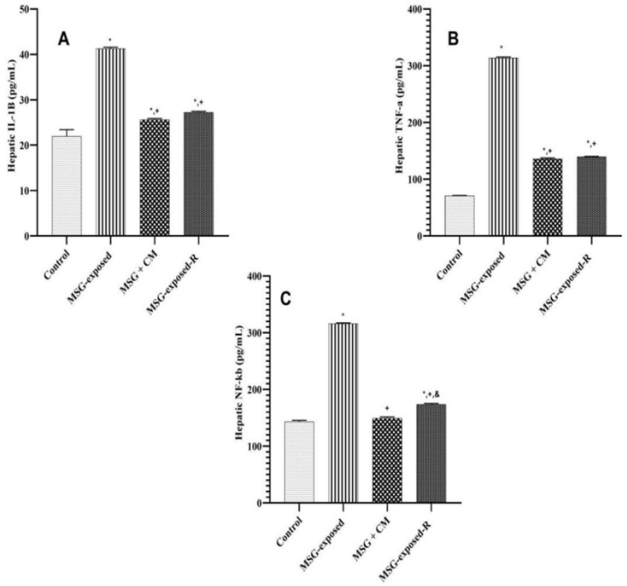

Figure 1: Graph showing the effect of Camel milk on Hepatic IL-1β, TNF-α, and NF-kβ in MSG-exposed rats

P< 0.05 vs MSG-exposed+CM. Figure 1 represented the results of hepatic IL-1b, TNF-a, and NF-kb. Figure 1A and Figure 1B results of hepatic IL-1b and TNF-a indicated that all other groups of rats increased significantly ($p<0.05$) compared to the control rats and that group C and D rats reduced significantly ($p<0.05$)

Table 1: Impact of Camel milk (CM) on Body weight and liver weight of rats subjected to MSG

	Groups			
	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+and
LW (g)	8.87± 0.06	6.47± 0.06*	7.33± 0.12*+	7.87 ± 0.15*=and

The values of 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 LW were initial body weight, final body weight, change in body weight and liver weight, respectively.

compared to group B rats.

Histopathology

Photomicrograph of liver section stained with H&E (Mg x400)
Where H=Hepatocytes, N=Nucleus, S=Sinusoids, Circle= Portal region, BD= Bile duct, HPV= Hepatic portal vessel, Star= Vascular congestion, Arrow head= inflammatory cell infiltration.

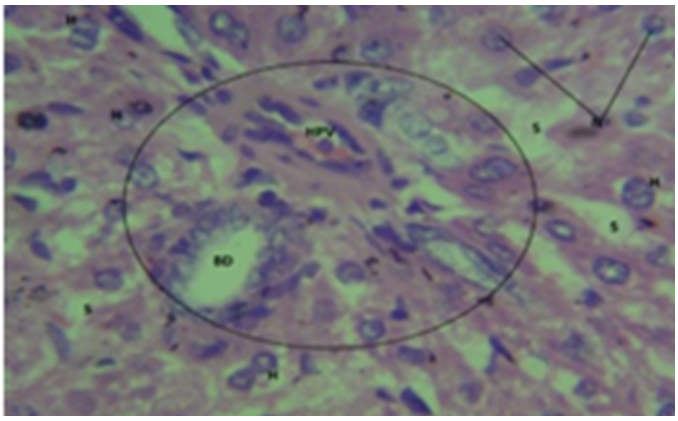

Figure 2a. Photomicrograph of liver tissue with normal histomorphology

Photomicrograph (Figure 2a) shows the liver tissue composed predominantly of the hepatic parenchyma and portal regions. The portal region (circle), composed of branches of the hepatic portal vessels, and bile duct (BD) appears normal implying normal histomorphology of the liver tissue.

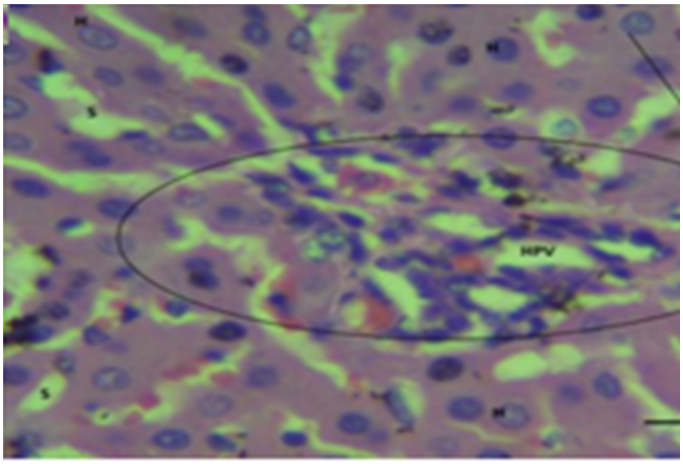

Figure 2b. Photomicrograph of liver tissue with vascular congestion implying periportal hepatitis in rats exposed to MSG

Photomicrograph (Figure 2(B)) shows the liver tissue composed predominantly of the hepatic parenchymal and portal regions. The portal region (circle), composed of branches of the hepatic portal vessels (HPV), bile duct (BD) and showed vascular congestion (Star) and mild inflammatory cell infiltration (arrowhead) implying periportal hepatitis of the liver tissue.

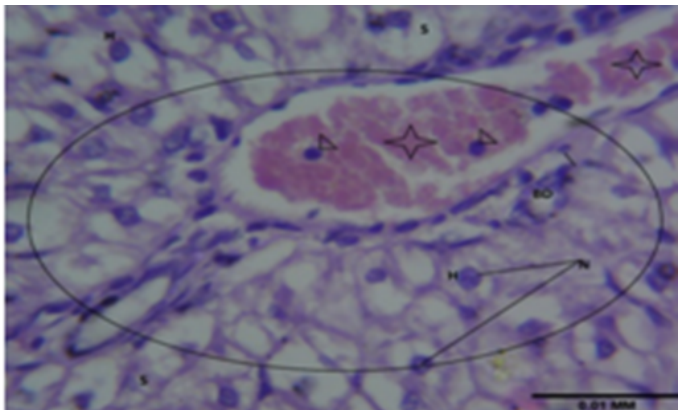

Figure 2c. Photomicrograph of liver tissue with normal hepatic portal vessels and bile duct in MSG exposed rats treated with camel milk

Photomicrograph (Figure 2c) shows the liver tissue composed predominantly of the hepatic parenchyma and portal regions. The Portal Region (Circle), composed of branches of the Hepatic portal vessels, and bile duct (BD) appear normal implying normal histomorphology

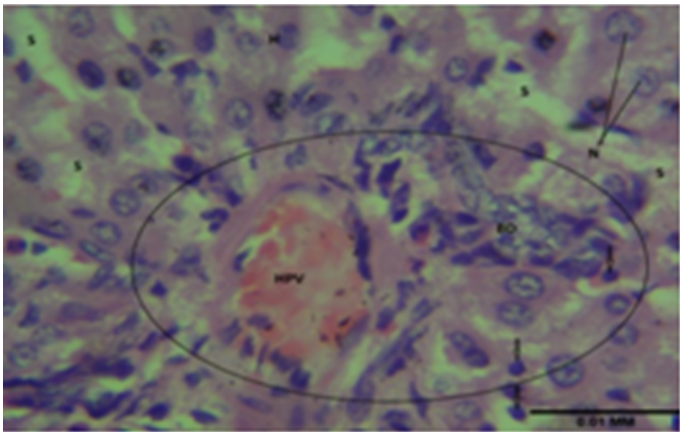

Figure 2d. Photomicrograph of liver tissue with mild inflammatory infiltration implying hepatic cellular injury.

Photomicrograph (Figure 2d) shows the liver tissue composed predominantly of the hepatic parenchymal and portal regions. The portal region (circle), composed of branches of

the hepatic portal vessels (HPV), bile duct (BD) and showed mild inflammatory cell infiltration (arrowhead) implying hepatic cellular reaction to injury.

Discussion

In line with the existing literature (Khan *et al*., 2022; Asejeje *et al*., 2023), MSG was a hepatotoxic agent, which caused a significant rise in inflammatory markers, as well as the disruption of liver histoarchitecture. It is, however, an unavoidable popular flavour enhancer that is present in many kinds of food. Another organ that is highly affected by MSG is the liver, which is the first organ to metabolise anything. Mohammed *et al*., (2022). Thus, nutritional measures to counteract MSG hepatic inflammation are deemed more appealing, less costly, and the amendable to regular rations, and they enhance health conditions. The present research examined the impact of camel milk on hepatic proinflammatory cytokines in MSG-stimulated male Wistar rats. A perfect and sensitive indicator of toxicity is the overall change of the body weight. BWC and LW of MSG-exposed rats were reduced significantly relative to the control, and this indicates possible metabolic disturbances. Interestingly, this tendency was not only stopped by the use of Camel milk supplementation (Group C), but this group also had the best BWC. It might mean that CM might positively influence the general metabolism and development, potentially by preventing metabolic changes caused by MSG. In this study, one dose of the MSG (60 mg/kg) BW placed in rats led to significant increases in the levels of the hepatic inflammatory markers (MPO, NO, and CRP) as compared to the control group to confirm the pro-inflammatory effects of the MSG on the liver. This is in line with those who found that MSG treated rats had higher markers of oxidative stress and inflammation (Asejeje). Mohammed *et al.*, 2022). There was also the levels of pro-inflammatory cytokines that were found to be significantly high such as TNF-a and IL-1b that indicated a potential underlying mechanism through which MSG induced the hepatic inflammation. Asejeje *et al*., 2023.NF-kB was highly up regulated in MSG-exposed rats compared to the control group. The high concentration of the pro-inflammatory cytokines in MSG induced hepatic inflammation could have stimulated the NF-kB transcription signalling pathway that is central to the up-regulation of cytokines. Banerjee *et al*., 2020. The histological findings are in support of the biochemical findings.

The exposure of the mice to MSG also had some distinct liver damage symptoms such as vascular congestion and minor inflammatory cell infiltration suggesting periportal hepatitis. On the other hand, the camel milk-based treatment reduced substantially the elevated concentration of the hepatic inflammatory markers (as presented in Figures 1 and 2). The findings are supported by the fact that the anti-inflammatory effects of CM are explained by its high lactoferrin content and other bioactive substances, which have been reported to regulate immune reactions and mitigate the oxidative stress. Abdelrahman *et al*., 2020. The capacity of camel milk to reduce NF-kB indicates that the compound could have an action at one of the basic stages of the inflammatory pathway and this could be this reaction by preventing the activation or nuclear translocation of NF-kB. The administration of camel milk was found to normalize the liver histology in which tissues were similar to the control group. The present research has revealed the anti-inflammatory nature of CM against MSG-induced liver damage in rats in the first instance.

The recovery group had an inhibited level of inflammatory markers (Figure 1 and Figure 2) than the MSG-exposed group. Certain inflammatory marker concentration (NO, NF-kB, and IL-1b) was increased. This is to imply that, the liver is able to regenerate some of the damage caused by MSG with time, but with some more assistance or more time, it might heal fully. This has been observed to be in line with NIDDK (2017) who reported that liver recovery in hepatic injury caused by toxin was time-dependent. Figure 1 and Figure 2 have shown that camel milk resolved the hepatic inflammatory markers faster than the recovery group. This states that the camel milk may provide a dependable and prompt method of treating the MSG-induced inflammation.

Conclusion and Recommendation

The normalisation of inflammatory biomarkers and the preservation of liver histoarchitecture demonstrated that camel milk significantly reduced MSG-induced hepatic inflammation. Through oxidative stress-driven activation of inflammatory signalling pathways, MSG exposure is known to compromise hepatocellular integrity, resulting in increased pro-inflammatory cytokines and hepatic tissue structural deterioration. The demonstrated healing benefits of camel milk point to a complex mechanism that includes stabilising hepatocyte membranes, boosting endogenous antioxidant defences, and suppressing inflammatory mediators. Its hepatoprotective potential is further supported by improvements in liver histology, such as decreased necrosis, decreased cellular infiltration, and preservation of sinusoidal architecture. All of these results point to camel milk as a potentially useful natural therapeutic agent for reducing toxin-induced liver inflammation, which may have implications for dietary or supplemental treatments aimed at inflammatory liver diseases.

Considering the encouraging results of the anti-inflammatory properties of camel milk on the liver markers in MSG-exposed male Wistar rats, it is recommended that more studies be done to perfect dosing schedules and evaluation of long term effectiveness. It is also important that future research work should focus on the possibility of using camel milk in the clinical practice of people, especially those who are at a risk of liver diseases. Next, the research on the impact of camel milk on other organ systems will give a deeper insight into the potential of camel milk as a therapeutic option. Inclusion of camel milk in dietary intervention may provide a natural, available source of alleviating inflammation of the liver and enhancing overall health outcomes.

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Conflict of Interest

The authors declare that they have no financial or personal relationships which may have inappropriately influenced them in writing this paper.

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