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Dietary *Zingiber officinale* (Ginger) Supplementation Ameliorates Lead Carbonate-Induced Hepato-Renal Toxicity and Inflammation in Female Wistar Rats

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ABSTRACT

In low and middle-income countries, lead contamination remains a significant public health threat due to weak regulatory enforcement. Lead exposure induces oxidative stress and systemic inflammation, leading to liver and kidney dysfunction. This study aimed to investigate the ameliorative effects of dietary ginger (*Zingiber officinale*) supplementation against lead-induced damage in Wistar rats. Female Wistar rats were divided into four groups (n = 6): Control (A); lead carbonate (30 mg/kg/day, oral) (B); lead + 1% ginger diet (C); and lead + 5% ginger diet (D) for 28 days. Serum levels of creatinine, aspartate aminotransferase (AST), alanine aminotransferase (ALT), malondialdehyde (MDA), total antioxidant capacity (TAC), tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), interleukin-10 (IL-10), and liver histology were assessed.
Lead exposure significantly ($p < 0.05$) elevated markers of hepato-renal injury (creatinine, AST, and ALT), increased the oxidative stress marker (MDA), and raised pro-inflammatory cytokines (TNF-α, IL-6). Conversely, it depleted the total antioxidant capacity (TAC) and the anti-inflammatory cytokine IL-10, while inducing severe liver histopathological damage compared to the control group (Group A). Dietary ginger supplementation dose-dependently attenuated these lead-induced alterations. Notably, the 5% ginger diet (Group D) significantly ($p < 0.05$) reduced all markers of organ damage and inflammation, decreased MDA, and increased both TAC and IL-10 levels compared to Group B. In conclusion, dietary ginger, particularly at a 5% supplementation level, demonstrates significant protective effects against lead-induced hepato-renal toxicity, oxidative stress, and inflammation in rats, highlighting its potential as a natural hepato-renal protective agent.

Keywords: *Zingiber officinale*, Ginger, Lead, Inflammation, Hepatotoxicity.

Introduction

Despite the global discontinuation of leaded gasoline, which significantly reduced lead exposure over the years, lead contamination advance a major threat to global health conditions, especially in low- and middle-income countries where regulatory enforcement may be weak.[1,2] Currently, lead exposure remains responsible for a significant global health burden, contributing to an estimated 1.5 million deaths annually.[3,4] Since ancient times, lead has been utilized in various applications, including pottery, window construction, shipbuilding, the arms industry, cosmetics, paint pigments, book printing, and both internal and external medicinal preparations.[5,6] This extensive industrial use has resulted in air and land pollution. Lead toxicity is linked to a range of dysfunctions observed in both laboratory animals and humans,[7] affecting the blood-forming system,[8] liver,[9] and kidneys, [10,5] as well as male and female reproductive organs.[11,12] Despite increasing evidence highlighting the harmful health effects of lead, it continues to be widely employed in consumer products and released into the environment through the burning of coal and oil, waste incineration, and industrial emissions from mining and smelting.[6]

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Lead exposure induces oxidative stress by promoting the formation of Reactive Oxygen Species (ROS) and weakening the body's antioxidant defenses by depleting glutathione, inhibiting enzymes that rely on sulfhydryl groups, or making cells more vulnerable to oxidative damage by altering cell membrane integrity.[13]The liver, as the primary site for xenobiotic metabolism, is particularly sensitive to oxidative stress induced by heavy metals like lead. Sustained exposure can result in elevated hepatic enzyme levels, histological damage, and compromised antioxidant defenses.[14,15] Given the side effects and limited efficacy of conventional chelation therapy, there is increasing scientific interest in alternative interventions particularly those derived from natural dietary sources with antioxidant and hepatoprotective capabilities.[16] Ginger (*Zingiber officinale*), a widely consumed culinary and medicinal plant, has been extensively used in traditional Chinese, Ayurvedic, and Unani medicine to treat various ailments such as respiratory conditions, joint pain, neurological disorders, and inflammation.[17] Beyond its culinary appeal, ginger has gained attention for its wide-range of pharmacological properties, including its capacity to act as an antioxidant, anti-inflammatory, anti-apoptotic, and metal-chelating agent.[18] The bioactive components of ginger, particularly gingerols, shogaols, and paradols have shown promise in experimental settings for reducing oxidative stress and preventing cellular damage caused by toxic exposures.[19] Recent studies have demonstrated ginger's protective effects against tissue injury induced by environmental pollutants and heavy metals.[20-22] Nevertheless, more targeted research is necessary to fully understand its therapeutic value in managing liver damage specifically that caused by lead toxicity. Therefore, this study aimed to investigate the potential ameliorative effect of dietary ginger supplementation on inflammatory markers, liver architecture, and hepato-renal function following lead exposure.

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Materials and Methods

Chemicals and Reagents
Lead (II) carbonate ($PbCO_3$) was a product of Thermo Scientific Chemicals, USA. All other chemicals and reagents used were of analytical grade.

Preparation of Ginger Powder
Fresh ginger (*Zingiber officinale*) rhizomes were obtained from a local market in Osogbo, Osun State, Nigeria. The rhizomes were thoroughly washed with distilled water, sliced, and oven-dried at 39ºC until completely dehydrated. The dried ginger slices were pulverized into a fine powder using a mechanical grinder to ensure consistent particle size. The powder was stored in an airtight containers at 4ºC until its incorporation into the diet.

Experimental Animals and Housing
Healthy female Wistar rats, weighing between 150 g and 170 g, were sourced from a breeding facility in Igbo Sai, Ogbomoso, Oyo State, Nigeria. The animals were transported to the Animal Research Facility of the University of Ilesa, Osun State, Nigeria. Following a two-week acclimatization period, rats were housed in standard polycarbonate cages (25×15×14 inches) under controlled conditions: temperature (22.5 ± 2.5◦C), humidity (50 ± 5%), and a 12-hour light/dark cycle (lights on at 7:00 a.m.). Standard commercial marshed feed and clean drinking water were provided *ad libitum*.

Ethical Approval
All experimental procedures were conducted in strict adherence to the ethical guidelines set by the Faculty of Basic Medical Sciences, University of Ilesa. Ethical approval with the approval reference number BMSUNILESA001/08/2024 was obtained from the ethical committee of the same Faculty. The research complied with the European Council Directive 2010/63/EU for the protection of animals used for scientific purposes.

Experimental Design and Grouping
A total of twenty-four (24) female Wistar rats were randomly allocated into four groups, with six (n = 6) animals per group:
Group A (Control): Received standard diet and distilled water (vehicle) daily via oral gavage.
Group B (Lead-only): Received standard diet and was administered lead carbonate (30 mg/kg body weight) suspended in distilled water daily via oral gavage.
Group C (Low-dose Ginger + Lead): Received a diet supplemented with 1% ginger powder and was administered lead carbonate (30 mg/kg body weight) in distilled water daily via oral gavage.
Group D (High-dose Ginger + Lead): Received a diet supplemented with 5% ginger powder and was administered lead carbonate (30 mg/kg body weight) in distilled water daily via oral gavage.

Diet Preparation and Treatment Administration
The basal diet consisted of standard commercial marshed feed. Ginger-supplemented diets were prepared by incorporating ginger powder into the feed at concentrations of 1% (w/w) for Group C and 5% (w/w) for Group D. These mixtures were thoroughly mixed as previously done by Ojo *et al.* (2025)[23] and Onaolapo *et al.* (2023)[24] Diet and lead carbonate treatments were administered daily for 28 consecutive days.

Sample Collection
On day 29, after an overnight fast, animals were humanely euthanized by cervical dislocation, following the *American Veterinary Medical Association* (AVMA) Guidelines for the Euthanasia of Animals (2020 Edition). Blood samples were collected via intracardiac puncture into heparinized tubes. Plasma was separated by centrifugation (1500×g for 15 minutes at 4ºC) and stored at −80ºC until analysis. Liver tissues were promptly excised, rinsed with ice-cold normal saline to remove blood, blotted dry, and a portion was fixed in 10% neutral buffered formalin for histological assessment.

Biochemical Analyses
Assessment of Liver and kidney Function Markers
Plasma levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), and creatinine were determined using commercially available assay kits [Randox Laboratories, UK] according to the manufacturer's instructions, employing a spectrophotometer.

Determination of the levels of the Inflammatory Cytokines
Plasma concentrations of tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interlukin-10 (IL-10) were quantified using enzyme-linked immunosorbent assay (ELISA) kits (Abcam, Cambridge, UK) as per the manufacturer's protocols. Absorbance was measured using a microplate reader at the wavelength specified by the kit.[25]

Determination of Oxidative Stress Biomarkers
Serum malondialdehyde (MDA) level was evaluated based on the reaction of this compound with thiobarbituric acid (TBA), formation of pink colour complex, and colorimetric assay. The optical density of the complex was measured at a wavelength of 540 nm as previously described by Offiong *et al.* (2024).[26]
Total antioxidant capacity (TAC) in serum was assessed using the ferric reducing antioxidant power (FRAP) method. In this method, TAC was estimated based on the reduction of a ferric tripyridyltriazine (Fe^{3+}-TPTZ) complex to Fe^{2+} form using $FeSO_4.7H_2O$ solution as standard (0.1 – 1 mmol/L).

Histological Examination
Liver tissues fixed in 10% neutral buffered formalin were subjected to standard histological processing. This involved dehydration in graded ethanol series, clearing in xylene, and embedding in paraffin wax. Sections of 4 − 5 µm thickness were obtained using a rotary microtome, mounted on glass slides, deparaffinized, rehydrated, and stained with Hematoxylin and Eosin (H&E). The stained sections were examined under a light microscope for histopathological changes, and representative photomicrographs were captured.

Statistical Analysis
Data were presented as the mean ± standard error of the mean (SEM). Statistical analyses were carried out using GraphPad Prism version 5.0 for Windows (GraphPad Software). Differences between means were assessed using one-way analysis of variance (ANOVA), followed by Tukey's HSD post-hoc test for multiple comparisons. A p-value less than 0.05 was regarded as statistically significant.

Results and Discussion

Lead exposure persists as a significant global health issue, with its pervasive environmental contamination leading to a spectrum of adverse health outcomes, particularly in developing nations.[27] This widespread toxicity underscores the critical need for accessible and effective interventions. As a result of traditional medicinal knowledge and contemporary research highlighting its potent antioxidant and anti-inflammatory properties, ginger (*Zingiber officinale*) was investigated as a potential protective agent against lead-induced liver and renal injuries. This study therefore investigated the potential of dietary ginger in mitigating lead-induced hepato-renal toxicity in Wistar rats, complementing recent findings on ginger's protective effects against lead-induced toxicities in other organ systems.[23,29,30]
The toxicity of lead carbonate was evident in this study, as rats administered lead without intervention (Group B) exhibited multiple pathological changes. Biochemically, these manifested as significant elevations in serum AST and ALT (Figure 1), established indicators of hepatocellular damage due to compromised membrane integrity,[31] alongside increased creatinine, suggesting potential renal dysfunction consistent with lead's known toxic effects,[32] and recent observations of lead-induced nephrotoxicity.[23] The underlying mechanism of this cellular damage was confirmed by a significant state of oxidative stress and inflammation in Group B rats administered lead carbonate compared to the control Group A. This was evidenced by significant ($p < 0.05$) increase in serum malondialdehyde (MDA), a key marker of lipid peroxidation, and a concurrent depletion of Total Antioxidant Capacity (TAC) in the lead-only group (Figure 2).

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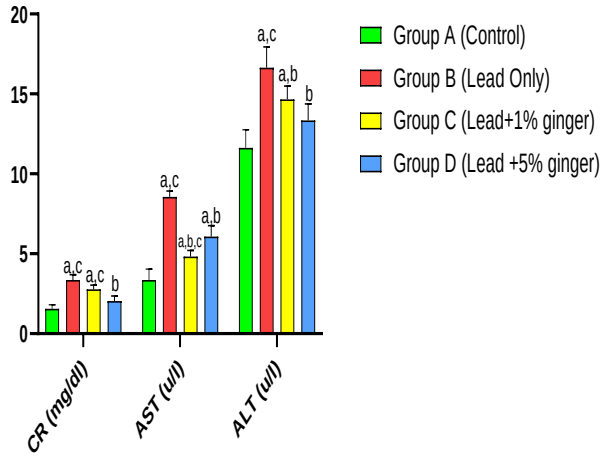

Figure 1: Effect of ginger supplementation on Serum creatinine, AST, and ALT levels in lead-exposed Wistar rats Data represent the mean ± SEM, n = 6; The lower case letters a, b, and c represent significant difference ($p < 0.05$) when compared to the control

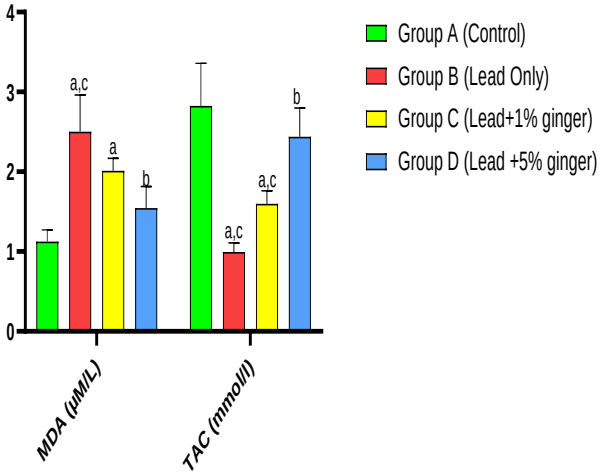

Figure 2: Effect of ginger supplementation on Serum Malonaldehyde (MDA) and Total Antioxidant Capacity (TAC) levels in lead-exposed Wistar rats. Data represent the mean ± SEM, n = 6; The lower case letters a, b, and c represent significant difference ($p < 0.05$) when compared to the control

These findings align with previous works which highlighted the capacity of lead to induce significant oxidative stress by promoting reactive oxygen species (ROS) formation and impairing endogenous antioxidant defenses, resulting in widespread cellular injury.[33] Furthermore, this oxidative assault triggered a severe inflammatory response, evidenced by a significant ($p < 0.05$) increase in the pro-inflammatory cytokines TNF-α and IL-6, and a simultaneous decrease in the anti-inflammatory cytokine IL-10 (Figure 3). This cytokine imbalance indicates a robust, unresolved inflammatory state induced by lead, contributing significantly to the observed tissue pathology.

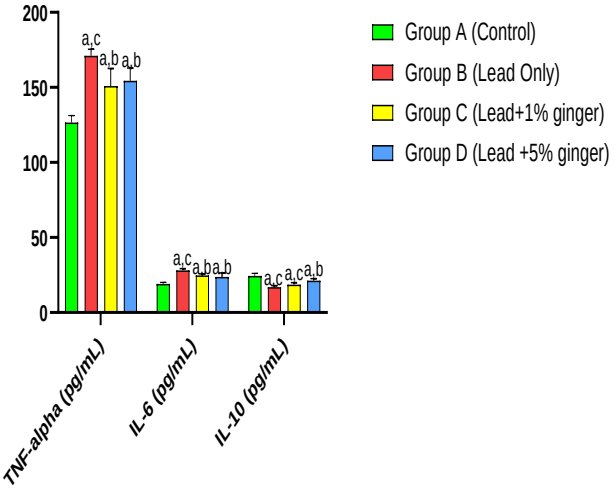

Figure 3: Effect of ginger supplementation on serum TNF-alpha, IL-6, and IL-10 levels in lead-exposed Wistar rats. Data represent the mean ± SEM, n = 6; The lower case letters a, b, and c represent significant difference ($p < 0.05$) when compared to the control

These systemic changes were corroborated at the tissue level, with histological examination of the liver revealing severe architectural disruption, extensive inflammatory cell infiltration, and hepatocellular necrosis (Figure 4B).

In contrast to the severe toxicity induced by lead, dietary supplementation with *Zingiber officinale* demonstrated a dose-dependent protective capacity. In Group C (lead + 1% ginger), there was partial but incomplete mitigation of lead-induced toxicity. Serum levels of AST, ALT, TNF-α, and IL-6 were significantly lower ($p < 0.05$) than those in the lead-only Group B, but remained significantly higher ($p < 0.05$) than that in the control Group A.

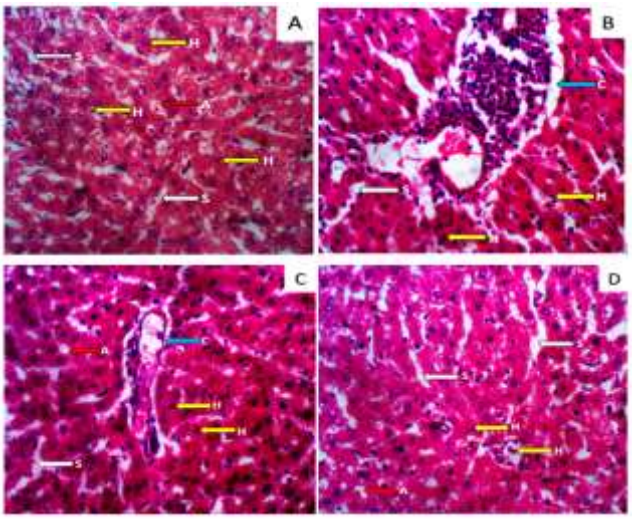

Figure 4: Representative photomicrographs of Hematoxylin and Eosin (H&E) stained liver sections from Wistar rats across the different experimental groups (Magnification 400x).
Key: H = Hepatocytes (Yellow Arrow); S = Sinusoids (White Arrow); A = Arteriole (Red Arrow), C = Central vein/portal vessel (Blue Arrow).

Similarly, while serum MDA was slightly reduced and TAC and IL-10 were slightly improved compared to Group B, these values were not statistically different from the lead-only group and remained significantly impaired compared to the control group (Figures 1, 2 and 3). Histologically, liver sections from Group C exhibited reduced inflammatory cell infiltration and less hepatocellular disarray compared to Group B, indicating moderate protection (Figure 4C). On the other hand, group D (lead + 5% ginger) showed the most substantial protective effects, with significantly lower ($p < 0.05$) serum levels in all measured biochemical markers of organ damage and oxidative stress (creatinine, AST, ALT, MDA) and significantly improved antioxidant status (TAC) compared to the lead-only group (Figures 1 and 2). Notably, serum creatinine, ALT, and Total Antioxidant Capacity in Group D were restored to levels not significantly different from those in the control Group A, suggesting a marked preservation of hepatocellular integrity and renal functional capacity, the latter being consistent with ginger's reno-protective effects against lead.[23] This antioxidant effect was paralleled by a profound modulation of the inflammatory response. On the inflammatory front, Group D showed a significant reduction in pro-inflammatory TNF-α and IL-6 and a significant increase in anti-inflammatory IL-10 when compared to Group B (Figure 3). This restoration of a more balanced cytokine profile points to ginger's ability to not only suppress pro-inflammatory signaling but also attenuate inflammation. However, serum AST, TNF-α, IL-6, and IL-10 levels in Group D, while improved, did not fully return to control levels but remained significantly different ($p < 0.05$) from Group A. This comprehensive biochemical recovery was strongly supported by the histological findings, as the liver sections from Group D rats revealed a well-preserved architecture with minimal inflammatory infiltration or evidence of damage, appearing remarkably similar to the control liver sections (Figure 4). The control group (A) exhibited a normal hepatic architecture, characterized by well-organized hepatocytes (H) arranged in cords, interspersed with clear sinusoids (S), and normal vasculature, including small arteries or arterioles (A) (Figure 4A). In contrast, the lead-only group (B) displayed severe lead-induced hepatotoxicity, evidenced by extensive inflammatory cell infiltration (indicated by the blue arrow, initially labeled 'C', but representing a dense inflammatory focus) within the liver parenchyma, accompanied by significant disarray of hepatocytes (H) and obscured sinusoids (S) (Figure 4B). Treatment with a low dose (1%) of ginger alongside lead exposure (Group C) resulted in partial amelioration of these toxic effects; liver sections showed a marked reduction in inflammation compared to the lead-only group, with more organized hepatocytes (H), discernible sinusoids (S), a normally appearing central vein or portal vessel (C), and an arteriole (A) (Figure 4C). The most substantial protective effect was observed in the high-dose (5%) ginger + lead group (D), where the liver architecture was largely preserved and appeared comparable to the control group. Hepatocytes (H) were well-maintained, sinusoids (S) were clear, and there was minimal evidence of focal disruption (A) or inflammation (Figure 4D), indicating significant mitigation of lead-induced liver damage by the higher concentration of ginger. This restoration to normal liver architecture suggests that ginger effectively counteracted the detrimental effects of lead through a dual mechanism: mitigating direct oxidative injury and dampening the ensuing inflammatory cascade. These protective actions are likely attributable to the complex phytochemical profile of ginger, which contains bioactive compounds like gingerols, shogaols, and paradols, known for their potent antioxidant, anti-inflammatory, and potential metal-chelating properties.[19,20]

The present findings are consistent with existing literature highlighting ginger's efficacy against various chemical-induced organ toxicities,[21,22] largely attributed to its significant antioxidant capabilities. This study provides direct evidence of this by demonstrating ginger's ability to reduce lipid peroxidation (MDA) and enhance total antioxidant status (TAC) in the face of lead toxicity. Indeed, recent studies further affirm ginger's therapeutic potential in reversing lead-induced nephrotoxicity,[23,28] oxidative stress, and cerebral cortex injuries,[29] and ovarian damage [30] in similar experimental models. This study specifically reinforces and extends these observations to the context of lead-induced liver damage, providing clear evidence for ginger as a potent dietary adjunct for mitigating such toxicity. Given the enduring global health burden of lead exposure,[3,2] the translational prospects of ginger warrant careful exploration. In summary, this study demonstrates that dietary ginger, especially at a 5% concentration, offers significant protection against lead-induced hepato-renal injury, oxidative stress, and inflammation in an experimental model, suggesting its utility as a natural agent for mitigating the effects of this pervasive environmental toxicant.

Conclusion

Findings from this study demonstrate that dietary supplementation with ginger, particularly at a 5% concentration, significantly ameliorates lead-induced hepato-renal toxicity and inflammation in Wistar rats. The protective effects are evidenced by the normalization or near-normalization of key biochemical markers and substantial preservation of liver histological architecture. These findings highlight the therapeutic potential of ginger as a natural, accessible, and cost-effective agent to mitigate the adverse health effects of lead exposure, likely through its potent antioxidant and anti-inflammatory properties. Further research is encouraged to fully elucidate its mechanisms and explore its clinical applicability.

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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