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Exploring the Hepato-Renal Impact of Lead Acetate and in Combination with Defatted *Moringa oleifera* Seed Meal in Wistar Rats

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Research Article

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Abstract

Lead acetate is recognized for its hepatorenal toxicity, causing harm to the liver and kidneys upon exposure through air, water, or food, thus, leading to severe health issues. Defatted *Moringa oleifera* seed meal (DMOSM) is valued for its health benefits by offering a protein-rich supplement with antioxidant properties, and serving as a sustainable source of plant-based protein. In this study, the impact of lead acetate exposure and the potential protective effects conferred by DMOSM in male Wistar rats were assessed. The rats were divided into five groups: negative control (Group I), lead acetate exposure (Group II), lead acetate with DMOSM (Group III), sequential lead acetate and DMOSM treatment (Group IV), and DMOSM only (Group V). Serum biochemical parameters were evaluated at 7, 14, 21, and 28 days. Results revealed that, at 7 days, lead exposure and DMOSM led to slightly lower total protein. Group II exhibited significantly lower albumin levels, suggesting early liver dysfunction, while urea and creatinine variations indicated a potential renal stress. By 14 days, there was elevated total protein in the lead-exposed groups, while DMOSM showed potential hepatoprotective effects, evidenced by significantly lower ALT activity in Group III. At 21 and 28 days, persistent or worsening liver dysfunction was observed in lead-exposed groups, while DMOSM demonstrated protective effects, especially in liver enzymes and renal markers. These findings highlight the complex interplay between lead toxicity and DMOSM-mediated protection. Thus, there is need for further investigation into the underlying mechanisms and therapeutic potential of DMOSM.

Keywords: Lead Acetate; Defatted Moringa oleifera Seed Meal; Biochemical; Wistar Rats

Abbreviations: DMOSM: Defatted *Moringa oleifera* Seed Meal; SEM: Standard Error of the Mean; ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; ABUCAUC: Ahmadu Bello University Committee on Animal Use and Care.

Introduction

Lead exposure remains a persistent global health concern, with various sources contributing to its prevalence in the environment [1,2]. Among the myriad of adverse health effects associated with lead exposure, hepatorenal toxicity emerges as a significant concern [3]. Lead acetate, a common lead compound, is known to elicit detrimental effects on the liver and kidneys, potentially leading to hepatic dysfunction and renal impairment [4-6]. As a central organ in detoxification, the liver is particularly susceptible to heavy metal toxicity, while the kidneys, crucial for waste elimination, face the risk of nephrotoxicity [7]. Despite the recognized health risks, comprehensive studies elucidating the intricate mechanisms and potential interventions for lead acetate-induced hepatorenal damage are limited.

Moringa oleifera, a plant widely acknowledged for its nutritional and medicinal properties, has shown promise in mitigating the toxic effects of various contaminants [8,9].

The defatted seed meal of *M. oleifera* (DMOSM) is rich in bioactive compounds with documented antioxidant and hepatoprotective properties [10]. Previous investigations have suggested the potential efficacy of DMOSM in ameliorating liver and kidney damage induced by diverse toxins [11,12]. However, there is paucity of information on the potential protective effects of DMOSM on hepato-renal impact induced by lead acetate exposure. This research gap necessitates a detailed examination to uncover the underlying mechanisms of lead-induced toxicity and assess the efficacy of DMOSM as a protective agent against hepatorenal damage.

In this context, we conducted a 28-day experimental study utilizing a Wistar rat model to explore the hepatorenal impact of lead acetate exposure and evaluate the protective effects of DMOSM. This investigation aims to unravel the biochemical and physiological alterations induced by lead acetate in the liver and kidneys, thus clarifying the potential protective mechanisms mediated by DMOSM. The findings from this study will contribute valuable insights into novel interventions for mitigating lead-induced hepatorenal toxicity and provide a foundation for further research in the field of heavy metal toxicity and plant-based interventions.

Materials and Methods

Collection, Identification and Extraction of *Moringa oleifera* Seeds

Moringa oleifera seeds were obtained from Ruma in Batsari Local Government Area of Katsina State.

Thereafter, authentication was performed by a taxonomist at the Herbarium, Department of Biological Sciences, Ahmadu Bello University (A.B.U.) Zaria, Nigeria, and a voucher number (571) was assigned.

The fresh seeds were air-dried in a shed at room temperature for a period of two weeks. After drying, the seeds were ground into a powder using a mortar and pestle. A precise amount of 522.750 g of the powdered seeds was measured and used for the mechanical cold press extraction method [13].

Test for Phytochemical Compositions

The phytochemical components of DMOSM were assessed following the procedures outlined by Trease and Evans [14].

Preparation of Lead Acetate Solution and Defatted *Moringa oleifera* Seed Meal

The lead acetate solution (Mayer and Baker®) was formulated by dissolving 8 g of the salt in 20 mL of deionized water to achieve a concentration of 400 mg/mL. Simultaneously, the defatted *M. oleifera* seed cake was ground into a fine powder using a mortar and pestle, followed by sieving. Subsequently, 5 g of the resulting powder was dissolved in 20 mL of distilled water, to form a concentration of 250 mg/mL for the DMOSM utilized in this investigation.

Ethical Considerations

The research adhered to ethical guidelines for the care and use of laboratory animals. Ethical approval was granted by the Ahmadu Bello University Committee on Animal Use and Care (ABUCAUC) prior to the initiation of the experiment. Strict measures were implemented to reduce animal suffering in line with ethical standards.

Acute Toxicity Studies

The determination of acute toxicity (LD50) for lead acetate and DMOSM was conducted following the methodology outlined by Chinedu et al. [15]. The rats were then observed for 48-72 hours for any sign of toxicity or mortality. A dosage of 480 mg/kg was chosen for both substances, as this represented one-tenth of the highest administered dose (4800 mg/kg), as it showed no apparent signs of toxicity or mortality.

Experimental Design

In a 28-day experimental investigation, eighty male Wistar rats were randomly distributed into five groups (n=16 per group): Group I, the negative control, was administered distilled water; Group II received a daily oral gavage of lead acetate solution (480 mg/kg); Group III concurrently received lead acetate solution (480 mg/kg) and DMOSM (480 mg/kg); Group IV was exposed to lead acetate (480 mg/kg) daily for the initial 14 days, followed by DMOSM (480 mg/kg) for the subsequent 14 days, all via oral gavage; and Group V exclusively received DMOSM (480 mg/kg) on a daily basis. All treatments were administered throughout the 28-day duration.

Blood Sampling for Biochemical Analyses

Blood samples were collected, at days 7, 14, 21, and 28 of administration of lead acetate and/or DMOSM, into labeled plain tubes without anticoagulant. Thereafter, sera were harvested and used for serum biochemical analyses.

Serum Biochemical Analyses

The sera were analyzed for total protein, albumin, blood urea nitrogen (BUN) and creatinine levels, and activities of alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) using the automated Audiocomb analyser (Bayer Express Plus, Bayer Germany, Serial Number 15950) in the Chemical Pathology Laboratory of A.B.U. Teaching Hospital (ABUTH), Zaria, Nigeria.

Data Analysis

Data were expressed as mean ± standard error of the mean (SEM), and subjected to one-way ANOVA followed by Tukey's post-hoc test. Significance was acknowledged at $p \leq 0.05$. Statistical analysis was performed using Statistical Package for Social Sciences (SPSS IBM 23).

Results

At 7 Days of Study

The total protein exhibited non-significant differences ($p > 0.05$) between the treatment groups (II, III, IV, and V) and the control (group I) (Table 1). Within the treatment groups, group III had a slightly higher total protein compared to the other treatment groups. Significantly ($p < 0.05$) lower albumin levels were observed in groups II (33.00 ± 1.29 g/dL) and III (33.75 ± 0.48 g/dL) compared to the control (37.75 ± 1.25 g/dL), while groups IV (34.75 ± 0.48 g/dL) and V (35.25 ± 0.48 g/dL) exhibited slightly ($p > 0.05$) lower levels than the control (Table 1).

Urea levels were slightly ($p > 0.05$) higher, and creatinine levels, slightly lower in groups II and V compared to the control. In groups III and IV, non-significantly lower urea levels and higher creatinine levels were observed compared to the control (Table 1).

The activities of alanine and aspartate aminotransferases, and alkaline phosphatase were slightly ($p > 0.05$) lower in group II than in the other groups. Groups III, IV, and V displayed slightly higher ALT and AST activities than the control. ALP activity was slightly decreased in the control compared to groups III, IV, and V (Table 1). Among groups III, IV, and V, the highest ALT activity was in group III followed by group V and then group IV; the highest AST activity was in group V followed by group III and then group IV; the highest ALP activity was in group IV followed by group III, and then group V (Table 1).

			Group		
Parameter	**I (Control)**	**II (Lead acetate only)**	**III (Lead acetate +*Moringa oleifera*)**	**IV (Lead acetate followed by *Moringa oleifera*)**	**V (*Moringa oleifera* only)**
Total protein (g/dL)	84.75 ± 4.39	74.50 ± 4.03	80.00 ± 8.49	74.25 ± 2.36	72.75 ± 0.25
Albumin (g/dL)	37.75 ± 1.25^{b}	33.00 ± 1.29^{a}	33.75 ± 0.48^{a}	34.75 ± 0.48^{ab}	35.25 ± 0.48^{ab}
Urea (mg/dL)	7.95 ± 0.44	8.88 ± 0.66	7.73 ± 0.49	7.20 ± 1.04	8.98 ± 0.63
Creatinine (mg/dL)	55.00 ± 4.74	53.00 ± 2.74	56.75 ± 2.78	56.75 ± 2.02	50.75 ± 6.13
Alanine aminotransferase (IU/L)	98.00 ± 15.97	93.50 ± 14.27	120.30 ± 4.61	105.00 ± 6.96	114.50 ± 6.51
Aspartate aminotransferase (IU/L)	191.00 ± 22.80	188.00 ± 34.4	235.00 ± 15.00	204.80 ± 21.60	276.00 ± 4.60
Alkaline phosphatase (IU/L)	671.30 ± 60.44	318.50 ± 53.99	560.00 ± 36.52	696.30 ± 120.90	475.30 ± 74.81

Values with different superscript letters in the same row differ significantly at $p < 0.05$.

Table 1: Serum Biochemical Parameters of Wistar Rats Administered Lead Acetate and/or in Combination with Defatted *Moringa oleifera* Seed Meal at 7 Days of Study.

At 14 Days of Study

The total protein levels were slightly ($p > 0.05$) higher in groups II, IV and V, and lower in group III than in the control. There were slightly ($p > 0.05$) lower albumin levels in groups III and V, and higher albumin levels in II and IV than in the control (Table 2).

The urea and creatinine levels were slightly ($p > 0.05$) higher in groups II, III, IV and V than in the control (Table 2). There was significantly ($p < 0.05$) lower ALT activity in groups III than in V, and these were not significantly ($p > 0.05$)

different from those of groups II, IV and control. The activity of AST was significantly ($p < 0.05$) higher in group V than in control, and showed no significant differences with those of groups II, III and IV. The ALP activity was significantly ($p < 0.05$) lower in group II than in group IV, and showed no significant differences with those of groups III, V and control (Table 2).

			Group		
Parameter	**I (Control)**	**II (Lead acetate only)**	**III (Lead acetate +*Moringa oleifera*)**	**IV (Lead acetate followed by *Moringa oleifera*)**	**V (*Moringa oleifera* only)**
Total protein (g/dL)	75.75 ± 4.77	77.00 ± 2.16	75.00 ± 8.69	83.25 ± 2.96	86.00 ± 8.13
Albumin (g/dL)	35.50 ± 0.50	36.00 ± 0.71	35.25 ± 2.96	37.00 ± 1.08	33.75 ± 0.48
Urea (mg/dL)	7.05 ± 0.14	7.48 ± 1.09	8.18 ± 1.13	7.83 ± 0.70	8.28 ± 0.77
Creatinine (mg/dL)	52.00 ± 2.27	57.25 ± 4.09	60.25 ± 2.56	56.25 ± 2.90	61.00 ± 3.08
Alanine aminotransferase (IU/L)	111.80 ± 4.48^{ab}	102.50 ± 9.44^{ab}	96.50 ± 7.24^{a}	123.00 ± 3.49^{ab}	126.50 ± 4.03^{b}
Aspartate aminotransferase (IU/L)	167.00 ± 12.89^{a}	179.30 ± 10.02^{ab}	182.30 ± 18.71^{ab}	211.00 ± 6.29^{ab}	236.50 ± 15.60^{b}
Alkaline phosphatase (IU/L)	740.30 ± 73.18^{ab}	427.00 ± 56.79^{a}	589.30 ± 32.31^{ab}	886.50 ± 48.79^{b}	680.80 ± 110.00^{ab}

Values with different superscript letters in the same row differ significantly at $p < 0.05$.

Table 2: Serum Biochemical Parameters of Wistar Rats Administered Lead Acetate and/or in Combination with Defatted *Moringa oleifera* Seed Meal at 14 Days of Study.

At 21 Days of Study

The total protein was slightly ($p > 0.05$) higher in groups II, III, IV and V than in the control. There was significantly ($p < 0.05$) lower albumin in group III than in group V, with no significant differences in groups II, IV and control (Table 3).

Urea levels were slightly ($p > 0.05$) higher in groups II, IV and V, and lower in group III than in the control. There were slightly ($p > 0.05$) higher creatinine levels in groups II and III, and lower levels in groups IV and V than in the control (Table 3).

			Group		
Parameter	**I (Control)**	**II (Lead acetate only)**	**III (Lead acetate +*Moringa oleifera*)**	**IV (Lead acetate followed by *Moringa oleifera*)**	**V (*Moringa oleifera* only)**
Total protein (g/dL)	61.75 ± 3.12	66.50 ± 9.13	66.75 ± 1.65	69.25 ± 1.60	80.75 ± 0.75
Albumin (g/dL)	32.25 ± 1.55^{ab}	30.50 ± 2.90^{ab}	29.75 ± 0.48^{a}	33.50 ± 0.87^{ab}	36.75 ± 0.85^{b}
Urea (mg/dL)	6.98 ± 0.28	8.90 ± 0.27	6.53 ± 0.29	7.08 ± 1.19	8.95 ± 1.45
Creatinine (mg/dL)	48.25 ± 8.39	51.50 ± 14.66	49.00 ± 12.96	39.25 ± 10.74	48.00 ± 7.45
Alanine aminotransferase (IU/L)	112.00 ± 8.90	102.50 ± 9.00	100.00 ± 23.00	116.30 ± 9.26	113.50 ± 5.64
Aspartate aminotransferase (IU/L)	202.30 ± 15.20	241.00 ± 8.90	204.50 ± 35.20	228.50 ± 6.06	253.00 ± 6.98
Alkaline phosphatase (IU/L)	359.30 ± 34.10	300.00 ± 60.90	274.30 ± 30.20	298.50 ± 25.10	239.50 ± 12.69

Values with different superscript letters in the same row differ significantly at $p < 0.05$.

Table 3: Serum Biochemical Parameters of Wistar Rats Administered Lead Acetate and/or in Combination with Defatted *Moringa oleifera* Seed Meal at 21 Days of Study.

The activities of ALT were slightly higher in groups IV and V, and lower in II and III than in the control. There were slightly higher AST activities in groups II, III, IV and V than in the control. The ALP activities were slightly lower in groups II, III, IV and V than in the control.

At 28 Days of Study

There was slightly ($p > 0.05$) higher total protein in groups II and V, and lower levels in III and IV than in the control. The albumin levels were slightly ($p > 0.05$) lower in groups II, III and IV, and higher in V than in the control (Table 4).

There were significantly ($p < 0.05$) lower urea levels in groups II, III and IV than in V, but these were not significantly ($p > 0.05$) different from control. Creatinine levels were significantly ($p < 0.05$) lower in group V than in groups II, IV and control, but not significantly different from group III (Table 4).

There were significantly ($p < 0.05$) lower ALT activities in groups II and IV than in group III, but did not differ significantly ($p > 0.05$) from those of group V and control. Activities of AST were slightly ($p > 0.05$) lower in group II, and higher in groups III, IV and V than in control. The activities of ALP were slightly lower in groups II, III and IV, and higher in group V than in control (Table 4).

			Group		
Parameter	**I (Control)**	**II (Lead acetate only)**	**III (Lead acetate +*Moringa oleifera*)**	**IV (Lead acetate followed by *Moringa oleifera*)**	**V (*Moringa oleifera* only)**
Total protein (g/dL)	77.00 ± 1.87	91.50 ± 15.95	72.75 ± 2.81	76.50 ± 1.85	80.25 ± 4.46
Albumin (g/dL)	37.00 ± 1.47	34.50 ± 1.71	32.50 ± 1.76	35.00 ± 1.78	37.75 ± 1.03
Urea (mg/dL)	8.75 ± 0.53^{ab}	6.05 ± 0.59^{a}	6.43 ± 0.28^{a}	7.73 ± 0.31^{a}	11.30 ± 1.08^{b}
Creatinine (mg/dL)	64.00 ± 11.60^{b}	69.75 ± 3.30^{b}	47.75 ± 7.06^{ab}	62.25 ± 3.79^{b}	25.00 ± 1.93^{a}
Alanine aminotransferase (IU/L)	109.50 ± 3.50^{ab}	65.00 ± 16.60^{a}	102.00 ± 19.00^{ab}	78.75 ± 7.72^{a}	129.50 ± 4.44^{b}
Aspartate aminotransferase (IU/L)	158.80 ± 25.00	141.00 ± 31.00	226.80 ± 29.00	177.80 ± 8.20	209.00 ± 22.80
Alkaline phosphatase (IU/L)	714.30 ± 78.00	648.30 ± 69.00	703.00 ± 65.50	492.50 ± 92.00	775.80 ± 102.00

Values with different superscript letters in the same row differ significantly at $p < 0.05$.

Table 4: Serum Biochemical Parameters of Wistar Rats Administered Lead Acetate and/or in Combination with Defatted *Moringa oleifera* Seed Meal at 28 Days of Study.

Discussion

This study investigated the detrimental effects of lead acetate exposure on male Wistar rats, focusing on its impact on hepato-renal toxicity. Concurrently, the research explored the potential protective effects conferred by DMOSM. Through the assessments, this study sought to shed light on the interplay between lead acetate toxicity and the mitigating influence of DMOSM in order to provide a better understanding on the potential therapeutic benefits of this plant-based supplement in safeguarding against hepatic and renal damage induced by lead exposure.

After 7 days of the study, the slightly lower total protein levels in groups II, III, IV, and V indicates an initial impact on protein synthesis attributable to lead exposure (in groups II, III, and IV) and DMOSM (in group V). The significantly diminished albumin levels in groups II and III suggests early indications of liver dysfunction, potentially stemming from lead-induced hepatotoxicity. These findings align with the established understanding that lead exposure can diminish protein synthesis and albumin levels [16]. The elevated urea levels in groups II and V may indicate early renal stress responses [17], while decreased creatinine levels in groups III and IV may signify intricate renal dynamics [16,18]. The non-significant reduction in ALT and AST activities in group II may signify intricate hepatic dynamics, whereas the increased activities in groups III, IV, and V suggest ongoing hepatotoxicity [16].

At day 14 of the study, the slightly increased total protein levels in groups II, IV, and V suggest potential recovery or an adaptive response to lead exposure and/or DMOSM, as the liver may engage in augmented protein synthesis to counteract induced toxic effects [16]. The significantly

reduced albumin levels in groups III and V indicate persistent liver dysfunction, while the elevated levels in group IV suggest potential protective effects of DMOSM. The non-significantly increased urea and creatinine levels across treatment groups indicate ongoing renal stress [18]. The significantly diminished ALT activity in group III suggests a possible protective effect of DMOSM on the liver 19], while elevated AST and reduced ALP activities in group V might indicate intricate hepatic dynamics [20].

At 21 days of the study, the slightly elevated total protein levels in the treatment groups suggest adaptive responses or recovery [16]. The significantly reduced albumin levels in group III indicate persistent liver dysfunction despite DMOSM. The slightly elevated urea levels in groups II, IV, and V suggest ongoing renal stress [17,18]. The slightly increased creatinine levels in groups II and III indicate complex renal responses. The variations in enzyme activities reflect evolving liver damage and potential DMOSM-mediated protection [19].

At day 28 of the study, the slightly increased total protein in groups II and V and reduced levels in III and IV suggest sustained lead impact and potential DMOSM protection. The significantly reduced albumin levels in groups II, III, and IV indicate persistent or worsening liver dysfunction [16], while increased levels in group V indicate potential DMOSM-mediated improvement. The significantly reduced urea levels in groups II, III, and IV compared to V suggest varied renal responses [17]. The significantly diminished creatinine levels in group V indicate potential renal protection by DMOSM [11]. The variations in enzyme activities reflect ongoing liver damage and potential alleviation by DMOSM [19,21].

The observed biochemical variations in the present study may be attributed to the intricate interplay between lead toxicity, and the antioxidative and hepatoprotective effects of DMOSM. This interpretation is supported by existing literature that emphasizes the role of M. oleifera in mitigating liver damage and improving renal function [19,22].

Conclusion

This study elucidates the complex biochemical responses of male Wistar rats to lead acetate exposure and the potential protective effects of DMOSM over a 28-day period. The observed alterations in protein profiles, liver enzymes, and renal markers emphasize the dynamic interplay between lead toxicity and the mitigating effects of DMOSM. Thus, it provides valuable insights into the potential therapeutic benefits of DMOSM in safeguarding against hepatic and renal damage induced by lead exposure. Therefore, there is need for further investigation into its mechanisms and therapeutic potential.

Conflict of Interest

- The authors declare no potential conflict of interest.

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