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Research Article

Protective Effects of Jobelyn® Against Lead-Induced Hepatorenal Toxicity and Haematological Alterations in Male Mice

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ABSTRACT
Lead exposure remains a major public-health concern due to its oxidative and inflammatory capacity to disrupt hepatic, renal, and haematological function. Jobelyn® (JB), a polyphenol-rich extract from *Sorghum bicolor* leaf sheath with antioxidant and anti-inflammatory properties, has not previously been evaluated in lead-induced systemic toxicity. This study investigated the protective effects of Jobelyn® on lead-induced hepatorenal and haematological alterations in male mice. Twenty male Swiss mice (22–30 g) were randomly assigned into four groups (n = 5) and treated orally for seven days: control (distilled water, 2 mL/kg), lead acetate (100 mg/kg), and lead acetate (100 mg/kg) co-administered with Jobelyn® (50 or 100 mg/kg). Lead exposure significantly increased aspartate aminotransferase (68.8 ± 3.88 U/L, $p < 0.001$), alanine aminotransferase (36.0 ± 0.84 U/L, $p < 0.01$), alkaline phosphatase (99.87 ± 3.74 U/L, $p < 0.01$), urea (39.13 ± 1.33 mg/dL, $p < 0.01$), creatinine (0.57 ± 0.03 mg/dL, $p < 0.01$), white blood cell count (6.05 ± 0.16 × 10^9/L, $p < 0.01$), neutrophils (71.6 ± 3.61%, $p < 0.01$), and lymphocytes (63.8 ± 1.39%, $p < 0.05$), while red blood cell count (4.54 ± 0.18 × 10^{12}/L, $p < 0.01$), packed cell volume (30.8 ± 1.11%, $p < 0.01$), and haemoglobin (9.42 ± 0.29 g/dL, $p < 0.01$) decreased versus control. Co-treatment with Jobelyn® improved these parameters dose-dependently, with the 100 mg/kg dose producing the greatest amelioration ($p < 0.05$ vs lead group). These findings suggest that Jobelyn® exert hepatorenal and haematological protection against lead toxicity in mice.

Keywords: Haematological alterations; Hepatorenal; Jobelyn®**;** Lead acetate; Toxicity

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INTRODUCTION

Lead (Pb) is a ubiquitous, non-biodegradable heavy metal and a significant environmental and occupational toxicant that continues to pose a major global public health concern, particularly in developing nations (Gonzalez-Villalva *et al*., 2025). Human exposure to lead, even at low concentrations, is associated with a wide spectrum of adverse health effects, as there is no known safe blood lead concentration (World Health Organization, 2024). Lead is capable of targeting and inducing deleterious effects across multiple organ systems, including the hematopoietic system, the renal and central nervous systems, and the reproductive system (Flora *et al*., 2012).

The key mechanism underlying lead toxicity is the generation of reactive oxygen species (ROS), leading to oxidative stress, which subsequently induces inflammation and interferes with cellular defense mechanisms (Flora *et al*., 2012; Collin *et al*., 2022). Lead's ability to mimic essential metals like zinc and calcium, disrupting vital cellular functions by replacing them in enzymes and proteins, further contributes to its multi-organ toxicity (Generalova *et al*., 2025).

In the hematopoietic system, lead exposure can inhibit key enzymes involved in heme synthesis, such as delta-aminolevulinic acid dehydratase (ALAD) and ferrochelatase (Kayaaltı *et al*., 2015). This inhibition leads to an accumulation of heme precursors and can cause forms of *anaemia*, like frank and *haemolytic anaemia* due to a combination of reduced haemoglobin synthesis and damage to red blood cell membranes (Sachdeva *et al*., 2018). Furthermore, lead can reduce the lifespan of circulating erythrocytes by increasing membrane fragility (Suwalsky *et al*., 2003).

The liver and kidneys are principal organs for metabolism, detoxification, and excretion, making them primary sites of lead accumulation and high susceptibility to damage following exposure to lead (Nakhaee *et al*., 2019; Ilesanmi *et al*., 2022). Lead acetate intoxication has been shown to cause hepatotoxicity, characterised by increased levels of liver enzymes, indicative of hepatocellular damage, along with oxidative stress, fibrosis, and apoptosis in hepatic cells (Haleagrahara *et al*., 2010; Umar *et al*., 2019; Zidi, 2014). Similarly, nephrotoxicity is a common consequence of lead exposure, manifesting as acute tubular injury and chronic nephropathy, often indicated by elevated serum levels of blood urea and creatinine (Rastogi, 2008; Evans & Elinder, 2010).

The current clinical management for severe lead poisoning involves chelation therapy using agents such as dimercaprol, calcium disodium ethylene diamine tetra-acetic acid ($CaNa_2EDTA$) and succimer. However, these synthetic chelators are often associated with side effects, including the depletion of essential trace metals, prompting the search for safer and more effective therapeutic alternatives (Gracia & Snodgrass, 2007; Balali-Mood et al., 2025).

Jobelyn® is an herbal dietary supplement derived from the leaf sheath of a West African variety of *Sorghum bicolor* (L.) Moench (Olajuwon *et al*., 2018). Traditional and anecdotal reports, as well as emerging scientific evidence, suggest that Jobelyn® possesses potent anti-anaemic, anti-inflammatory, and antioxidant properties (Umukoro *et al*., 2013; Ayuba *et al*., 2014; Okubena *et al*., 2018). Its beneficial effects are primarily attributed to a rich phytochemical profile, particularly high concentrations of polyphenolic compounds like anthocyanins (e.g., apigeninidin), flavonoids (e.g., quercetin, luteolin), and phenolic acids (e.g., gallic acid), which are known for their remarkable free radical scavenging and antioxidant capacities (Oghenekevwe *et al*., 2025). Despite the presence of these potent antioxidants, which suggests a potential protective role of Jobelyn® against toxicities mediated by oxidative stress, such as that induced by heavy metals, there is a paucity of studies on Jobelyn®'s effect in models of heavy-metal toxicity. Hence, this study investigates the protective effects of Jobelyn® on lead acetate-induced hepatorenal toxicity and haematological alterations in mice, with the broader objective of contributing to evidence-based phytotherapeutic strategies for heavy metal toxicity.

MATERIALS AND METHODS

Chemicals and Drug Preparation

Lead acetate was purchased from Sigma-Aldrich (St. Louis, USA). Jobelyn® was obtained from Health Forever Products Ltd., Lagos, Nigeria. Dosing solutions were freshly prepared by dissolving the lead acetate and Jobelyn® in distilled water before administration.

Experimental Animals

Twenty adult male Swiss mice (25-30 g) were used in this study. The animals were obtained from the Animal Care Unit of the Department of Human Physiology, Federal University Wukari, Nigeria. The animals were maintained under standard laboratory conditions with free access to feed and water *ad libitum*. Ethical approval for this study was obtained from the Research and Ethical Committee College of Health Sciences, Federal University Wukari. The study received ethical approval number FUW/CHS/HREC/AUGUST/2025/012. All experimental procedures were carried out in accordance with the National Institutes of Health

Guide for the Care and Use of Laboratory Animals (National Research Council, 2011)

Study Design

Mice were randomly assigned into four experimental groups (n = 5 per group): Group I (Control) received distilled water (2 mL/kg), Group II received lead acetate (100 mg/kg), Group III received lead acetate (100 mg/kg) along with Jobelyn® (JB) (50 mg/kg), and Group IV received lead acetate (100 mg/kg) along with Jobelyn® (100 mg/kg). All doses were administered once daily by oral gavage for seven consecutive days. The lead acetate dose was selected based on prior studies employing 100 mg/kg in acute mouse models (Yu *et al*., 2020), while the Jobelyn® doses were informed by previous pharmacological evidence demonstrating efficacy at 50 and 100 mg/kg in mice (Umukoro *et al*., 2019).

Animal Sacrifice and Tissue Collection

Twenty-four hours after the final dose was administered, the animals were humanely sacrificed under anaesthesia with intraperitoneal injection of ketamine hydrochloride 90 mg/kg and xylazine 10 mg/kg. Blood samples were collected by cardiac puncture, by accessing the heart through a midline incision on the anterior abdominal wall. Blood samples for haematological analyses were collected into EDTA tubes, while blood for biochemical assays was placed in plain tubes, allowed to clot, and then centrifuged at 3,000 rpm for 10 minutes. The serum was stored at −20 °C until analysis.

Haematological Assays

Haematological indices, including red blood cell (RBC) count, white blood cell (WBC) count, haemoglobin (Hb) concentration, packed cell volume (PCV), neutrophils, and lymphocytes, were analysed using a Mindray BC-2800 automated haematology analyser (Mindray Bio-Medical Electronics Co., Ltd., Shenzhen, China).

Biochemical Assays

Serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), and alkaline phosphatase (ALP) activities were measured using Randox assay kits according to the method described by Reitman and Frankel (1957). Serum urea and creatinine concentrations were determined using Agappe diagnostic kits, following the established kit protocols (Patton & Crouch, 1977).

Data Analysis

Data are presented as mean ± SEM. Statistical analysis was performed using one-way ANOVA followed by Newman–Keuls *post hoc* test for multiple comparisons. Statistical tests were performed with GraphPad Prism version 8.0. Values of $p < 0.05$ were considered significant.

RESULTS

Effect of Jobelyn® on Serum Hepatic Enzymes

As illustrated in Figure 1 (A–C), serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), and alkaline phosphatase (ALP) activities were significantly increased in mice treated with lead alone when compared with the control group ($p < 0.001$ for AST; $p < 0.01$ for ALT and ALP). Co-treatment with Jobelyn® significantly reversed these elevations. The 50 mg/kg dose significantly reduced AST ($p < 0.01$), ALT ($p < 0.05$), and ALP ($p < 0.05$) levels compared to the lead group. A similar reduction was observed with the 100 mg/kg dose, which also significantly lowered AST ($p < 0.01$), ALT ($p < 0.05$), and ALP ($p < 0.05$) levels compared to the lead group.

Effect of Jobelyn® on Renal Function Biomarkers

As shown in Figure 2 (A–B), treatment with lead alone significantly elevated serum urea ($p < 0.01$) and creatinine ($p < 0.01$) concentrations compared with the control group. Co-administration of Jobelyn® significantly produced improvement in these renal indices at the 100 mg/kg dose ($p < 0.05$ for both urea and creatinine) when compared to the lead group.

Effect of Jobelyn® on Haematological Parameters

As presented in Table 1, lead administration caused a significant decrease in red blood cell (RBC) count ($p < 0.01$), packed cell volume (PCV; $p < 0.01$), and *haemoglobin* concentration (Hb; $p < 0.01$), alongside significant increases in white blood cell (WBC) count ($p < 0.01$), neutrophil percentage ($p < 0.01$), and lymphocyte percentage ($p < 0.05$) compared to the control group. Co-administration of Jobelyn® ameliorated these haematological alterations in a dose-dependent manner. The 50 mg/kg dose significantly increased RBC, PCV, and Hb levels ($p < 0.05$) and significantly reduced the percentages of neutrophils and lymphocytes ($p < 0.05$) compared to the lead group. At 100 mg/kg, Jobelyn® produced a more significant increase in RBC, PCV, and Hb ($p <$

0.05) and significant reductions in WBC count, neutrophil, and lymphocyte percentages ($p < 0.05$) compared to the lead group.

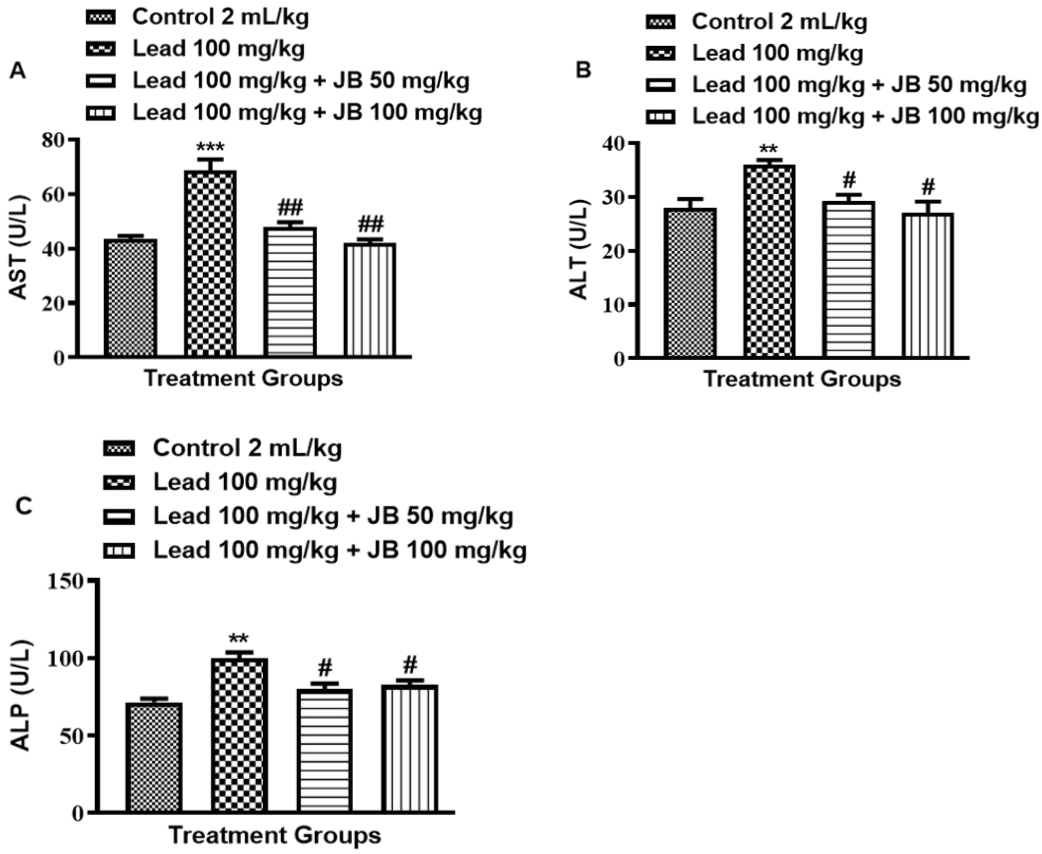

Figure 1 (A–C). Effect of Jobelyn® (JB) on serum hepatic enzyme activities in lead acetate–treated male mice
(A) Aspartate aminotransferase (AST); (B) Alanine aminotransferase (ALT); (C) Alkaline phosphatase (ALP). Data are presented as mean ± SEM (n=5). Data were analysed using One-way ANOVA, followed by Newman-Keuls' comparison *post hoc* test. **p < 0.01, ***p < 0.001 compared with control group with #p < 0.05, ##p < 0.01 compared with lead group.

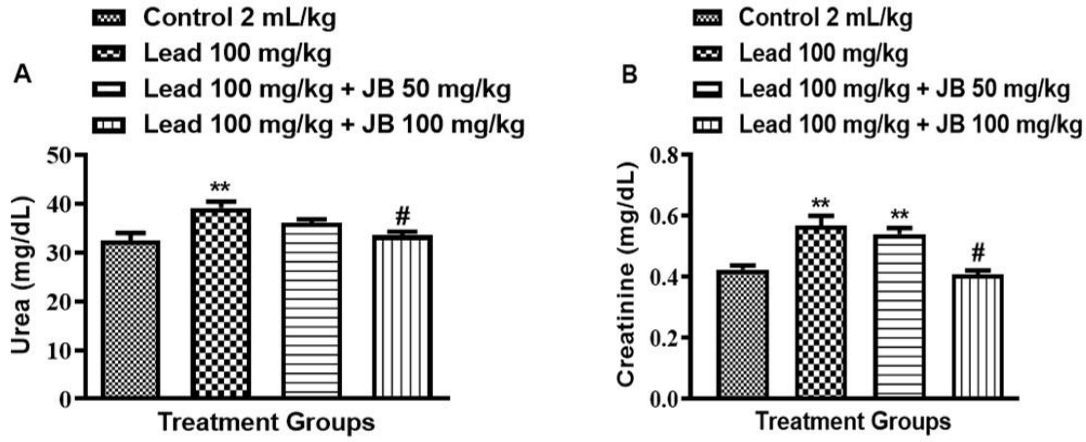

Figure 2 (A–B). Effect of Jobelyn® (JB) on serum renal biomarkers in lead acetate–treated male mice

(A) Urea; (B) Creatinine. Data are presented as mean ± SEM (n=5). Data were analysed using One-way ANOVA, followed by Newman-Keuls' comparison *post hoc* test. **p < 0.01 compared with control group with #p < 0.05, compared with lead group.

Table 1. Effect of Jobelyn® (JB) on haematological parameters

Treatment Groups	RBC(x10^{12}/L)	PCV (%)	Hb (g/dL)	WBC (x 10^9/L)	N (%)	L (%)
Control 2 ml/kg	5.62 ± 0.16	37.60±0.93	12.04±0.35	4.42±0.25	53.20±5.09	45.4 ± 6.21
Lead 100 mg/kg	4.54±0.18**	30.80±1.11**	09.42±0.29**	6.05±0.16**	71.60±3.61**	63.80±1.39*
Lead 100 mg/kg + JB 50 mg/kg	5.80±0.19#	36.20±0.73#	11.06±0.55#	5.62±0.28*	49.20±5.17#	45.60±6.02#
Lead 100 mg/kg+JB 100 mg/kg	5.66±0.15#	37.20±0.58#	11.26±0.34#	4.72±0.34#	47.40±3.56#	40.40±2.60#

Data are presented as mean ± SEM (n=5). Data were analysed using One-way ANOVA, followed by Newman-Keuls' comparison *post hoc* test. *p < 0.05, **p < 0.01 compared with control with #p < 0.05 compared with lead group.

DISCUSSION

This study examined the protective potential of Jobelyn® (JB), a polyphenol-rich extract of *Sorghum bicolor*, against lead acetate-induced hepatic, renal, and haematological alterations in male mice. The results revealed that lead administration significantly elevated blood serum levels of aspartate aminotransferase (AST), alanine aminotransferase (ALT), and alkaline phosphatase (ALP), urea and creatinine, alongside significant haematological alterations including anaemia and leukocytosis, indicating broad systemic toxicity. Co-treatment with Jobelyn® reversed these biochemical and haematological changes in a dose-dependent manner, with greater improvement observed at the 100 mg/kg dose. These findings suggest that Jobelyn® confers protection against lead-induced haematological alterations and hepatorenal dysfunction.

Lead remains a major environmental toxicant with well-documented effects on soft tissues, especially the liver and kidneys, organs central to its metabolism and excretion. Chronic lead exposure disrupts redox balance, impairs mitochondrial respiration, and promotes inflammatory cascades leading to hepatocellular leakage and renal tubular damage (Sharma, 2011; Nakhaee *et al*., 2019; Lakka *et al*., 2023). The elevated activities of AST, ALT, and ALP in the present study signify membrane injury and cytosolic enzyme efflux from damaged hepatocytes, consistent with earlier rodent and human findings linking hepatic enzyme induction to oxidative stress hepatocellular apoptosis (Haleagrahara *et al*., 2010; Kumari *et al*., 2016; Offor *et al*., 2017). Similarly, the increase in serum urea and creatinine levels reflects glomerular and tubular dysfunction typical of lead nephropathy, as observed in both experimental and epidemiological contexts (Ghorbe *et al*., 2001; Moody *et al*., 2018). Together, these biochemical alterations are typical manifestations of lead-induced hepatorenal toxicity, driven by reactive oxygen species (ROS) generation and lipid peroxidation in hepatic and renal tissues (Nakhaee *et al*., 2019).

Co-treatment with Jobelyn® significantly improved these biochemical markers, suggesting protection of hepatic and renal integrity. The decline in AST, ALT, and ALP activities implies stabilization of hepatocyte membranes and suppression of oxidative enzyme leakage. Similarly, reductions in urea and creatinine point to enhanced glomerular function and renal clearance. These findings are consistent with earlier reports showing that polyphenol-based formulations, such as aqueous and hydrophobic extracts of *Sorghum bicolor*, have hepatoprotective and nephroprotective effects (Akande *et al*., 2010; Owumi *et al*., 2023).

The haematological findings in this study further reinforce Jobelyn's systemic protective role. Lead acetate exposure caused a marked reduction in red blood cell (RBC) count, packed cell volume (PCV), and haemoglobin (Hb) concentration, hallmarks of anaemia, along with increased white blood cell (WBC) counts, neutrophils, and lymphocytes, which are indicative of an inflammatory response. These changes align with previous studies showing that lead interferes with heme biosynthesis by inhibiting delta-aminolevulinic acid dehydratase and ferrochelatase, thereby reducing haemoglobin synthesis and accelerating erythrocyte turnover (Kayaaltı *et al*., 2015; Sachdeva *et al*., 2018). The leukocytosis observed in this study may also reflect

the systemic inflammatory response to oxidative tissue injury (Pugin, 2012; Mank *et al*., 2024). Treatment with Jobelyn® ameliorated these haematological disruptions, likely by improving erythropoietic activity and suppressing oxidative stress-driven inflammation. This agrees with the findings of Akande *et al*. (2010), Ayuba *et al*. (2014) and Diaku-Akinwumi *et al*. (2024), who demonstrated that the extract of *Sorghum bicolor* improved haematological indices and reduced oxidative damage.

The protective efficacy observed following Jobelyn® administration is likely rooted in the synergistic actions of its polyphenolic constituents in mitigating the molecular hallmarks of lead toxicity. Lead exposure generates vast quantities of reactive oxygen species (ROS) by disrupting the pro-oxidant/antioxidant cell defence system and interfering with the activity of sulfhydryl-containing enzymes, promoting cellular damage and lipid peroxidation (Lopes *et al*., 2015). Polyphenolic constituents such as quercetin, proanthocyanidins, luteolin, apigenins, gallic acid, naringenins, and 3-deoxyanthocyanidins found in Jobelyn® possess potent radical-scavenging and anti-inflammatory properties (Omorogbe *et al*., 2018; Adebesin *et al*., 2024), which likely contribute to the observed biochemical and haematological restoration by specifically counteracting the molecular mechanisms of lead toxicity. Research indicates that polyphenols, in general, can modulate immune responses and gene expression, including the potential to activate the nuclear factor erythroid 2-related factor 2 (Nrf2) pathway, a master regulator of antioxidant response element (ARE)-driven gene expression, to combat oxidative stress and promote cytoprotecting (Singh *et al*., 2020; Tkaczenko & Kurhaluk, 2025). Furthermore, the anti-inflammatory properties of Jobelyn's constituents, notably luteolin and quercetin, are crucial, these flavonoids can inhibit pro-inflammatory signalling cascades, such as the nuclear factor kappa B (NF-kB) pathway, thereby mitigating the secondary organ damage caused by persistent inflammation following lead-induced cellular injury in the liver and kidneys. By suppressing the phosphorylation and subsequent nuclear transfer of NF-kB, these compounds prevent the transcription of inflammatory genes (Li *et al*., 2016; Lv *et al*., 2025).

In addition to antioxidant signaling, Phenolic compounds can prevent metal-induced reactive oxygen species (ROS) production by binding to and sequestering metal ions like copper and iron (Michalak, 2006). This chelating property may further contribute to the observed protective effects, as less free lead would be available to interact with critical sulfhydryl-containing enzymes in hepatic and renal cells. The present findings indicate that Jobelyn® attenuates lead-induced hepatic, renal, and haematological injury in male mice, likely through antioxidant, anti-inflammatory, metal-chelating and membrane-stabilising mechanisms. While these data reinforce its potential, further studies incorporating oxidative stress markers, histopathological evaluation and gene-expression profiling are needed to define its precise cellular targets and translational relevance.

CONCLUSION

Jobelyn® supplementation attenuated the biochemical and haematological alterations induced by lead acetate in mice, suggesting a protective influence on hepatic and renal function. The observed effects may be attributed to its antioxidant and anti-inflammatory properties. While these findings provide preliminary evidence of Jobelyn®'s potential in mitigating lead-related organ injury, further molecular and translational studies are warranted to confirm these mechanisms and explore clinical relevance.

Conflicts of Interest

The authors declare no conflict of interest.

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Authors' Contributions

Saminu Samaila, Lawrence D. Adedayo and Nanyak Z. Galam conceived and designed the research study, while Esu U. Enene, Joshua M. Komo, Alimonu Enyinnaya, Jerry Z. Joshua and Emmanuel A. Joel contributed to data collection and experimental procedures. Saminu Samaila and Lawrence D. Adedayo analysed the data and interpreted the findings. The manuscript was drafted by Saminu

Samaila and revised critically for intellectual content by all authors, who also approved the final version.

REFERENCES

Adebesin, A., Omogbiya, A. I., Oluwole, O. G., Okubena, O., Asomadu, R. O., Afolabi, M. O. S., Makanjuola, S. B. L., Ajonuma, L. C., Dosunmu, A. O., Otitoloju, O., & Umukoro, S. (2024). An Evidence-based Systematic Review of Pleiotropic Potential Health Benefits of *Sorghum bicolor* Supplement: A Polyphenol-rich Derivative of the Leaf Sheaths of Sorghum Plant. *Journal of Natural Remedies*, 683–702. https://doi.org/10.18311/jnr/2024/33171

Akande, I. S., Oseni, A. A., & Biobaku, O. A. (2010). Effects of aqueous extract of Sorghum bicolor on hepatic, histological and haematological indices in rats. *Journal of Cell and Animal Biology*, *4*(9), 137–142. https://doi.org/10.5897/jcab.9000057

Ayuba, G. I., Jensen, G. S., Benson, K. F., Okubena, A. M., & Okubena, O. (2014). Clinical efficacy of a West African sorghum bicolor-based traditional herbal preparation Jobelyn shows increased *haemoglobin* and CD4+ T-lymphocyte counts in HIV-positive patients. *Journal of alternative and complementary medicine (New York, N.Y.)*, *20*(1), 53–56. https://doi.org/10.1089/acm.2013.0125

Balali-Mood, M., Eizadi-Mood, N., Hassanian-Moghaddam, H., Etemad, L., Moshiri, M., Vahabzadeh, M., & Sadeghi, M. (2025). Recent advances in the clinical management of intoxication by five heavy metals: Mercury, Lead, Chromium, Cadmium and Arsenic. *Heliyon*, *11*(4), e42696. https://doi.org/10.1016/j.heliyon.2025.e42696

Collin, M. S., Venkatraman, S. K., Vijayakumar, N., Kanimozhi, V., Arbaaz, S. M., Stacey, R. G. S., Anusha, J., Choudhary, R., Lvov, V., Tovar, G. I., Senatov, F., Koppala, S., & Swamiappan, S. (2022). Bioaccumulation of lead (Pb) and its effects on human: A review. *Journal of Hazardous Materials Advances*, *7*, 100094. https://doi.org/10.1016/j.hazadv.2022.100094

Diaku-Akinwumi, I. N., Olajide, D., Adekunle, M. B., Adebayo, A. B., Bamiro, S., Ofakunrin, A. O., & Ajonuma, L. C. (2024). Jobelyn, a Sorghum Bicolor Exract-Based Oral Supplement Reduces Oxidative Stress in School Aged Children with Sickle Cell Disease. *Blood*, *144*(Supplement 1), 5362. https://doi.org/10.1182/blood-2024-212305

Evans, M., & Elinder, C. (2010). Chronic renal failure from lead: myth or evidence-based fact? *Kidney International*, *79*(3), 272–279. https://doi.org/10.1038/ki.2010.394

Flora, G., Gupta, D., & Tiwari, A. (2012). Toxicity of lead: A review with recent updates. *Interdisciplinary toxicology*, *5*(2), 47–58. https://doi.org/10.2478/v10102-012-0009-2

Generalova, A., Davidova, S., & Satchanska, G. (2025). The Mechanisms of Lead Toxicity in Living Organisms. *Journal of xenobiotics*, *15*(5), 146. https://doi.org/10.3390/jox15050146

Ghorbe, F., Boujelbene, M., Makni-Ayadi, F., Guermazi, F., Kammoun, A., Murat, J., Croute, F., Soleilhavoup, J. P., & El-Feki, A. (2001). Effect of chronic lead exposure on kidney function in male and female rats: determination of a lead exposure biomarker. *Archives of physiology and biochemistry*, *109*(5), 457–463. https://doi.org/10.1076/apab.109.5.457.8035

Gonzalez-Villalva, A., Marcela, R., Nelly, L., Patricia, B., Guadalupe, M., Brenda, C., Eugenia, C. M., Martha, U., Isabel, G., & Fortoul, T. I. (2025). Lead systemic toxicity: a persistent problem for health. *Toxicology*, *515*, 154163. https://doi.org/10.1016/j.tox.2025.154163

Gracia, R. C., & Snodgrass, W. R. (2007). Lead toxicity and chelation therapy. *American journal of health-system pharmacy : AJHP : official journal of the American Society of Health-System Pharmacists*, *64*(1), 45–53. https://doi.org/10.2146/ajhp060175

Haleagrahara, N., Jackie, T., Chakravarthi, S., Rao, M., & Kulur, A. (2010). Protective effect of *Etlingera elatior* (torch ginger) extract on lead acetate-induced hepatotoxicity in rats. *Journal of Toxicological Sciences*, *35*(5), 663–671.

Ilesanmi, O. B., Adeogun, E. F., Odewale, T. T., & Chikere, B. (2022). Lead exposure-induced changes in hematology and biomarkers of hepatic injury: protective role of TrévoTM supplement. *Environmental analysis, health and toxicology*, *37*(2), e2022007. https://doi.org/10.5620/eaht.2022007

Kayaaltı, Z., Sert, S., Kaya-Akyüzlü, D., Söylemez, E., & Söylemezoğlu, T. (2015). Association between delta-aminolevulinic acid dehydratase polymorphism and placental lead levels. *Environmental Toxicology*

and Pharmacology, *41*, 147–151. https://doi.org/10.1016/j.etap.2015.11.017

Kumari, S. A., Madhusudhanachary, P., Patlolla, A. K., & Tchounwou, P. B. (2016). Hepatotoxicity and Ultrastructural Changes in Wistar Rats treated with Al_2O_3 Nanomaterials. *Trends in cell & molecular biology*, *11*, 77–88.

Lakka, N., Pai, B., Mani, M. S., & Dsouza, H. S. (2023). Potential diagnostic biomarkers for lead-induced hepatotoxicity and the role of synthetic chelators and bioactive compounds. *Toxicology research*, *12*(2), 178–188. https://doi.org/10.1093/toxres/tfad014

Li, Y., Yao, J., Han, C., Yang, J., Chaudhry, M. T., Wang, S., Liu, H., & Yin, Y. (2016). Quercetin, Inflammation and Immunity. *Nutrients*, *8*(3), 167. https://doi.org/10.3390/nu8030167

Lopes, A. C. B. A., Peixe, T. S., Mesas, A. E., & Paoliello, M. M. B. (2015). Lead Exposure and Oxidative Stress: A Systematic review. *Reviews of Environmental Contamination and Toxicology*, *236*, 193–238. https://doi.org/10.1007/978-3-319-20013-2_3

Lv, J., Song, X., Luo, Z., Huang, D., Xiao, L., & Zou, K. (2025). Luteolin: exploring its therapeutic potential and molecular mechanisms in pulmonary diseases. *Frontiers in pharmacology*, *16*, 1535555. https://doi.org/10.3389/fphar.2025.1535555

Mank, V., Azhar, W., & Brown, K. (2024, April 21). Leukocytosis. In *StatPearls*. StatPearls Publishing. Available from: https://www.ncbi.nlm.nih.gov/books/NBK560882/

Michalak, A. (2006). Phenolic compounds and their antioxidant activity in plants growing under heavy metal stress. *Polish Journal of Environmental Studies*, *15*(4), 523–530.

Moody, E. C., Coca, S. G., & Sanders, A. P. (2018). Toxic Metals and Chronic Kidney Disease: a Systematic Review of Recent Literature. *Current environmental health reports*, *5*(4), 453–463. https://doi.org/10.1007/s40572-018-0212-1

Nakhaee, S., Amirabadizadeh, A., Brent, J., & Mehrpour, O. (2019). Impact of chronic lead exposure on liver and kidney function and haematologic parameters. *Basic & Clinical Pharmacology & Toxicology*, *124*(5), 621–628. https://doi.org/10.1111/bcpt.13179

National Research Council (2011). Guide for the care and use of laboratory animals (8th ed.). Washington, DC: The National Academies Press. https://doi.org/10.17226/12910

Offor, S. J., Mbagwu, H. O., & Orisakwe, O. E. (2017). Lead Induced Hepato-renal Damage in Male Albino Rats and Effects of Activated Charcoal. *Frontiers in pharmacology*, *8*, 107. https://doi.org/10.3389/fphar.2017.00107

Oghenekevwe, N. R. M., Olatunbosun, N. S. B., Moshood, N. a. O., & Gloria, N. a. A. (2025). Phytochemical, heavy metal analysis and HPLC profiling of Jobelyn® – an herbal dietary supplement. *Tropical Journal of Phytochemistry and Pharmaceutical Sciences*, *4*(7). https://doi.org/10.26538/tjpps/v4i7.2

Okubena, O., Makanjuola, S., Ajonuma, L. C., Dosunmu, A., Umukoro, S., & Erah, P. O. (2018). The West African Sorghum bicolor leaf sheath extract Jobelyn® and its diverse therapeutic potentials. *MOJ Drug Design, Development, and Therapy*, 2(1), 20-28. https://doi.org/10.15406/mojddt.2018.02.00025

Olajuwon Okubena, Samira Makanjuola, Louis C Ajonuma, Adedoyin Dosunmu, Solomon Umukoro, and Patrick O Erah (2018). The West African Sorghum bicolor leaf sheath extract Jobelyn® and its diverse therapeutic potentials. *MOJ Drug Design Development & Therapy*, 2(1):20–28. https://doi.org/10.15406/mojddt.2018.02.00025

Omorogbe, O., Ajayi, A. M., Ben-Azu, B., Oghwere, E. E., Adebesin, A., Aderibigbe, A. O., Okubena, O., & Umukoro, S. (2018). Jobelyn® attenuates inflammatory responses and neurobehavioural deficits associated with complete Freund-adjuvant-induced arthritis in mice. *Biomedicine & Pharmacotherapy*, *98*, 585–593. https://doi.org/10.1016/j.biopha.2017.12.098

Owumi, S. E., Ajakaiye, B., Akinwunmi, A. O., Nwozo, S. O., & Oyelere, A. K. (2023). The Hydrophobic Extract of *Sorghum bicolor* (L. Moench) Enriched in Apigenin-Protected Rats against Aflatoxin B_1-Associated Hepatorenal Derangement. *Molecules (Basel, Switzerland)*, *28*(7), 3013. https://doi.org/10.3390/molecules28073013

Patton, C. J., & Crouch, S. R. (1977). Spectrophotometric and kinetics investigation of the Berthelot reaction for the determination of ammonia. *Analytical Chemistry*, 49(3), 464–469. https://doi.org/10.1021/ac50011a034

Pugin J. (2012). How tissue injury alarms the immune system and causes a systemic inflammatory response syndrome. *Annals of intensive care*, *2*(1), 27. https://doi.org/10.1186/2110-5820-2-27

Rastogi S. K. (2008). Renal effects of environmental and occupational lead exposure. *Indian journal of occupational and environmental medicine*, *12*(3), 103–106. https://doi.org/10.4103/0019-5278.44689

Reitman, S., & Frankel, S. (1957). A colorimetric method for the determination of serum glutamic oxaloacetic and glutamic pyruvic transaminases. *American Journal of Clinical Pathology*, 28(1), 56–63. https://doi.org/10.1093/ajcp/28.1.56

Sachdeva, C., Thakur, K., Sharma, A., & Sharma, K. K. (2018). Lead: Tiny but Mighty Poison. *Indian Journal of Clinical Biochemistry*, *33*(2), 132–146. https://doi.org/10.1007/s12291-017-0680-3

Sharma, N. S. (2011). Lead toxicity, oxidative damage and health implications. A review. *International Journal for Biotechnology and Molecular Biology Research*, *2*(13). https://doi.org/10.5897/ijbmbrx11.002

Singh, A., Yau, Y. F., Leung, K. S., El-Nezami, H., & Lee, J. C. (2020). Interaction of Polyphenols as Antioxidant and Anti-Inflammatory Compounds in Brain-Liver-Gut Axis. *Antioxidants (Basel, Switzerland)*, *9*(8), 669. https://doi.org/10.3390/antiox9080669

Suwalsky, M., Villena, F., Norris, B., Cuevas, Y., Sotomayor, C., & Zatta, P. (2003). Effects of lead on the human erythrocyte membrane and molecular models. *Journal of Inorganic Biochemistry*, 97(3), 308–313. https://doi.org/10.1016/s0162-0134(03)00292-7

Tkaczenko, H., & Kurhaluk, N. (2025). Antioxidant-Rich functional foods and exercise: unlocking metabolic health through NRF2 and related pathways. International Journal of Molecular Sciences, 26(3), 1098. https://doi.org/10.3390/ijms26031098

Umar, H.O., Adebisi, S.S., Hamman, W.O., Muhammad, A.S., Salisu, R., & Mai-Siyama, I.B. (2019). Histological and Biochemical Effects of Orally administered Lead acetate on the Liver of Adult Wistar Rats of both Sexes. *Journal of Anatomical Sciences*, 10(2), 124-130

Umukoro, S., Oghwere, E. E., Ben-Azu, B., Owoeye, O., Ajayi, A. M., Omorogbe, O., & Okubena, O. (2019). Jobelyn® ameliorates neurological deficits in rats with ischemic stroke through inhibition of release of pro-inflammatory cytokines and NF-κB signaling pathway. *Pathophysiology*, *26*(1), 77–88. https://doi.org/10.1016/j.pathophys.2018.10.002

Umukoro, S., Ugbomah, A., Aderibigbe, A., & Omogbiya, A. (2013). Antioxidant Property of Jobelyn as the Possible Mechanism Underlying its Anti-amnesic Activity in Rodents. *Basic and Clinical Neuroscience*, *4*(1), 42–49.

World Health Organization (2024). Lead poisoning. https://www.who.int/news-room/fact-sheets/detail/lead-poisoning-and-health

Yu, Y., Yu, L., Zhou, X., Qiao, N., Qu, D., Tian, F., Zhao, J., Zhang, H., Zhai, Q., & Chen, W. (2020). Effects of acute oral lead exposure on the levels of essential elements of mice: a metallomics and dose-dependent study. *Journal of Trace Elements in Medicine and Biology*, *62*, 126624. https://doi.org/10.1016/j.jtemb.2020.126624

Zidi, S. A., I. (2014b). Hepatotoxic effects of lead acetate in rats: Histopathological and cytotoxic studies. *Journal of Cytology & Histology*, *05*(05). https://doi.org/10.4172/2157-7099.1000256.