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The role of *Anthocleista djalonensis* in mitigating mercury chloride-induced hematological alterations

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Abstract

Background: Mercury chloride is a well-established toxicant known to induce hematological alterations, including oxidative hemolysis, anemia, and thrombocytopenia. Medicinal plants with antioxidant properties have been investigated as potential protective agents against toxicant-induced damage. *Anthocleista djalonensis* (*A. djalonensis*), which is widely used in traditional medicine, has been reported to contain bioactive compounds with therapeutic potential. This study aimed to investigate the role of *A. djalonensis* in mitigating mercury chloride-induced hematological alterations.

Methods: This experimental randomized controlled animal study was conducted using thirty-six adult Wistar rats, which were randomly assigned to six groups (n=6 each). Group A served as the control; Group B received only 2 mg/kg of mercury chloride. Groups C and D received mercury chloride together with 150 mg/kg and 300 mg/kg of *A. djalonensis* extract, respectively, while Groups E and F received only *A. djalonensis* at doses of 150 mg/kg and 300 mg/kg, respectively. The extract was prepared by freeze-drying, and hematological parameters were assessed using an automated hematology analyzer. Statistical analysis was performed using one-way ANOVA, with the significance level set at $p < 0.05$.

Results: Mercury chloride exposure significantly reduced red blood cell count, hemoglobin concentration, and platelet levels, indicating hematotoxicity. Co-administration with *A. djalonensis*, particularly at 150 mg/kg, significantly improved these hematological indices, suggesting a protective effect. The plant extract also enhanced mean corpuscular hemoglobin and mean corpuscular hemoglobin concentration, indicating potential erythropoietic support.

Conclusion: The findings suggest that *A. djalonensis* mitigates mercury chloride-induced hematotoxicity, likely through antioxidative mechanisms. These results highlight its potential therapeutic role in the management of hematological disorders associated with toxicant exposure.

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Introduction

Mercury chloride ($HgCl_2$) is a widely recognized environmental and industrial toxicant that poses significant health risks to humans and animals. It is used in various industrial processes, laboratory reagents, and chemical synthesis; however, it has no known metabolic or physiological role in living organisms, and its presence in biological systems reflects contamination and exposure (1,2). Among the toxic effects of mercury chloride, hematological alterations are prominent, including oxidative hemolysis, reduced hemoglobin concentration, anemia, leukocyte changes, and thrombocytopenia (3). Moreover, mercury intoxication has also been associated with the induction of oxidative stress in tissues (4). Oxidative stress has been linked to the excessive production of reactive oxygen species (ROS), which may induce enzymatic inactivation and peroxidation of cellular constituents (5).

Mercury toxicity has been strongly associated with hematological and biochemical abnormalities in both experimental animals and humans. Laboratory studies have clearly demonstrated that mercury chloride induces hematotoxicity. Administration of mercury chloride to Wistar rats has been shown to markedly reduce red blood cell (RBC) count, hemoglobin concentration (Hb), and packed cell volume (PCV), while also causing leukocytosis and abnormalities in differential white blood cell counts. Following exposure, dose-related anemia and increased levels of malondialdehyde (MDA), a marker of lipid peroxidation, have also been reported. Collectively, these findings indicate that mercury chloride causes hematological dysfunction primarily through oxidative damage to erythrocyte membranes and impairment of antioxidant defense mechanisms (3).

Engwa et al. (6) reported the involvement of free radicals and endogenous antioxidant defenses in metal intoxication of animal blood. Erythrocytes are particularly susceptible to oxidative damage because of their high polyunsaturated fatty acid content and continuous exposure to oxygen (7). Although RBCs possess endogenous antioxidant mechanisms, including superoxide dismutase, catalase, and glutathione peroxidase, excessive mercury-induced free radical generation can overwhelm these defenses, resulting in membrane fragility, hemolysis, and reduced hematological function (6,8).

Medicinal plants have long been recognized as indispensable sources for drug discovery and have been used by humans for various purposes since the beginning of human history (9). Medicinal plants contain organic compounds that produce specific physiological effects in the human body, and their bioactive constituents include tannins, alkaloids, carbohydrates, terpenoids, steroids, and flavonoids (10). Medicinal plants are beneficial for the well-being of both individuals and communities (10). Consequently, the economic value of medicinal plants has attracted the attention of many international organizations, particularly the World Health Organization (WHO) (11). Since the WHO has supported and encouraged the integration of traditional medicinal resources into global health systems, the use of medicinal plants has increased rapidly (12). In this context, ethnobotanical research facilitates the development of new treatments and medications and also supports plant conservation (13). Many ethnobotanical studies worldwide have reported the use of herbal plants for healing practices that have been maintained for several generations in their respective societies (14). *A. djalonensis* A. Chev, commonly known as the cabbage tree, called “Kwari” in Hausa, “Ewe” by the Yoruba people of South-West Nigeria (15), and “Oyinwin’ wi-uwu” by the Binis of South-South

Nigeria (16), belongs to the family Loganiaceae. It is a medium-sized tree of West tropical Africa, 30-45 feet high, with blunt spines on the branches, a pale grey trunk, and a wide-spreading crown (15). The stem, root, bark, and leaves of *A. djalonensis* are used to treat malaria, jaundice, diabetes, and abscesses (17). Phytochemical screening of *A. djalonensis* has revealed the presence of flavonoids, terpenoids, and phenolic compounds (18), which are associated with antioxidant and cytoprotective activities. However, evidence regarding its potential to mitigate heavy metal-induced hematotoxicity remains limited. Therefore, this study aimed to investigate the protective role of *A. djalonensis* in mitigating mercury chloride-induced hematological alterations in Wistar rats.

Methods

This study was designed as an experimental randomized, controlled laboratory animal study.

Plant collection and identification

The stem bark of *A. djalonensis* used in this study was collected from a farm around Benin City. Plant collection was performed in accordance with the guidelines established by the Edo State Forestry Commission for the collection of wild plants. The specimen was identified and authenticated by a plant taxonomist in the Department of Plant Biology and Biotechnology, Faculty of Life Sciences, University of Benin, Benin City, Edo State, Nigeria, and was assigned the herbarium number UBH-A594.

Extract preparation

The plant was extracted using an aqueous freeze-drying method. The stem bark of *A. djalonensis* was rinsed with tap water, shade-dried, and crushed into smaller pieces. The powdered material was placed in distilled water and left in a separating funnel for 24 hours, with intermittent shaking. The solution was then filtered. The filtrate was allowed to settle and was subsequently decanted. It was then frozen, dried using a freeze-drying machine, and stored in a refrigerator at -6 °C.

Ethical approval

The guidelines of the Research Ethics Committee for the handling and care of animals at the College of Medical Sciences, University of Benin, were strictly followed. Ethical approval was granted with approval number CMS/REC/2023/340.

Experimental design

Thirty-six (36) adult Wistar rats were randomly assigned into six (6) groups, Groups A - F, with six rats per group.

The experimental animals were randomly assigned into six groups: Group I served as the control and received no treatment; Group II received mercury chloride at a dose of 2 mg/kg; Group III received mercury chloride (2 mg/kg) co-administered with low-dose *A. djalonensis* extract (150 mg/kg); Group IV received mercury chloride (2 mg/kg) co-administered with high-dose *A. djalonensis* extract (300 mg/kg); Group V received *A. djalonensis* extract alone at 150 mg/kg; and Group VI received *A. djalonensis* extract alone at 300 mg/kg.

All administrations were performed orally using an oral gavage attached to a calibrated syringe for 28 consecutive days.

Determination of hematological parameters

Hematological parameters were analyzed using a Mindray BC-2800 automated hematology analyzer (Mindray Bio-Medical Electronics Co., Shenzhen, China), based on the electrical impedance method.

This method uses three detector blocks and two types of reagents for blood analysis. The white blood cell (WBC) detector block measures WBC count using the DC detection method (19). The RBC detector block counts RBCs and platelets using the DC detection method (20). The Hb detector block uses the non-cyanide hemoglobin technique to determine hemoglobin concentration (21). Most electronic blood cell analyzers count blood cells based on impedance.

The normal reference ranges for adult Wistar rats were considered as follows: RBC (6.0-8.5 $\times 10^{12}$/L), hemoglobin (12-17 g/dL), packed cell volume (36-50%), WBC (6-18 $\times 10^{9}$/L), and platelet count (500-1300 $\times 10^{9}$/L). These reference values were used for comparison when assessing mercury chloride-induced hematological alterations.

Principle of impedance analyzer

Blood cells are diluted using a buffered electrolyte solution. A measured volume of the sample is passed through an aperture tube between two electrodes. Interruption of the current by non-conducting blood cells modifies the electrical charge, resulting in a pulse. The amplitude of each pulse is proportional to the volume of the cell that generated it. A threshold circuit ensures that only pulses exceeding predefined threshold levels are counted. The cell count is calculated using the number of pulses collected from a given volume of blood (22).

Statistical analysis

The data were statistically analyzed using GraphPad Prism version 9.0, and relevant statistical values were determined. Data were analyzed using one-way ANOVA and reported as mean ± SEM. Tukey's post hoc test was used. P-values of <0.05 were considered statistically significant. The statistical data obtained were transformed into graphical representations in the form of bar charts.

Results

The hematological indices of experimental animals after 28 days of administration of mercury chloride and *A. djalonensis* are presented in Table 1. The study found that mercury chloride exposure reduced WBC count, although the decrease was not statistically significant. The percentage of lymphocytes and mean inhibitory dilution also showed no significant changes across groups. However, granulocyte percentage was significantly affected, with mercury chloride reducing its levels, whereas 300 mg/kg of *A. djalonensis* increased them.

Table 1. Hematological indices of experimental animals after 28 days of administration with mercury chloride and *A. djalonensis*

Groups/Test	Control	2mg/kg Mercury chloride only	2mg/kg Mercury chloride + 150mg/kg of *A. djalonensis*	2mg/kg Mercury chloride + 300mg/kg of *A. djalonensis*	150mg/kg of *A. djalonensis* only	300mg/kg of *A. djalonensis* only	P-Value
White blood cell (uL)	4.50±0.75	4.28±1.21	5.93±0.41	7.55±0.75	6.20±1.14	7.15±0.97	0.1056
Lymphocyte (%)	91.50±0.81	89.10±3.69	91.10±0.95	90.20±1.44	93.30±0.54	88.30±1.54	0.4693
Mean inhibitory dilution (%)	5.53±0.87	4.83±0.69	5.90±0.85	7.35±1.09	4.68±0.56	8.33±1.36	0.0796
Granulocyte (%)	3.00±0.20	2.35±0.17	3.05±0.21	3.15±0.31	2.08±0.28	3.40±0.18	0.0050
Red blood cell (uL)	7.66±0.28	6.87±0.07*	6.36±0.25	7.17±0.17	7.16±0.13	6.81±0.16*	0.0039
Hemoglobin (g/dL)	14.50±0.68	12.80±0.29	13.70±0.68	16.10±0.33#	13.80±0.40	14.70±0.40	0.0042
Hematocrit (%)	37.60±1.09	35.20±0.68	32.70±1.22	31.00±5.71	36.70±1.01	35.40±1.12	0.4492
Mean corpuscular volume (fL)	49.30±0.75	50.30±1.63	51.40±0.77	50.20±0.54	51.40±0.71	50.20±0.71	0.5537
Mean corpuscular hemoglobin (pg)	18.80±0.21	17.50±1.47	21.50±0.35#	22.40±0.71#	19.20±0.23	21.70±0.33*	0.0005
Mean corpuscular hemoglobin concentration (g/dL)	38.40±0.96	34.90±2.18	42.00±1.33#	44.80±1.88#	37.50±0.60	43.10±1.18	0.0010
Standard deviation of red-cell distribution width (fl)	31.00±10.8	32.60±2.69	28.30±1.03	27.30±0.55	35.80±1.36	26.70±0.64	0.0017

Table 1 (Continued)

Coefficient of variation of red-cell distribution width (%)	14.70±0.34	15.10±0.88	14.70±0.34	14.50±0.33	16.40±0.43	14.20±0.30	0.0679
Platelet (uL)	536±33.60	365±52.80	707±87.70#	445±43.70	385±36.20	619±56.00	0.0017
Mean Platelet Volume (fl)	7.33±0.16	7.98±0.43	6.80±0.21	9.03±0.39	7.85±0.40	7.28±0.13	0.0017
Platelet Distribution width (%)	8.50±0.41	11.90±0.41*	8.13±0.08#	9.20±0.50#	10.00±0.73	7.98±0.08	<0.0001
Plateletcrit (%)	1.24±0.07	0.28±0.11*	0.45±0.04	1.40±0.01#	0.33±0.04*	0.43±0.05*	<0.0001

Values are expressed as mean ± Standard Error of Mean (SEM): * The mean difference is significant at P<0.05 compared with control. # The mean difference is significant at P<0.05 compared with 2mg/kg of mercury chloride only.

RBC count was significantly reduced by mercury chloride exposure compared with the control group. Co-administration with 150 mg/kg of *A. djalonensis* did not restore RBC count, whereas the 300 mg/kg co-treatment group showed RBC values closer to those of the control group. Similarly, mercury chloride significantly decreased hemoglobin concentration, whereas co-administration with 300 mg/kg of *A. djalonensis* significantly increased hemoglobin concentration compared with the mercury-treated group.

Hematocrit levels did not differ significantly among the groups. Mean corpuscular volume (MCV) remained unchanged, whereas mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC) were significantly altered. Mercury chloride reduced MCH and MCHC, whereas *A. djalonensis* treatment significantly increased them.

The study also observed significant changes in red cell distribution width (RDW), with the 300 mg/kg *A. djalonensis* group showing the lowest RDW values. However, the coefficient of variation of RDW remained statistically unchanged.

Platelet count was significantly reduced by mercury chloride, whereas *A. djalonensis* at 150 mg/kg significantly increased it. Mercury chloride also increased mean platelet volume (MPV), while *A. djalonensis* showed variable effects on this parameter. Platelet distribution width (PDW) was significantly elevated by mercury chloride, whereas *A. djalonensis* reversed this effect.

Finally, mercury chloride markedly reduced plateletcrit levels, while 150 mg/kg of *A. djalonensis* significantly improved them. Overall, mercury chloride reduced several erythrocyte- and platelet-related hematological parameters, whereas co-administration of *A. djalonensis* attenuated some of these changes.

Discussion

In the current investigation, exposure to mercury chloride appeared to cause evident hematological alterations. Specifically, declines in RBC count, Hb levels, and PCV were observed, along with evidence of thrombocytopenia (Table 1). These observations are consistent with earlier studies suggesting that inorganic mercury can induce hematotoxicity in laboratory animals (3,23). Mercury's effects on blood do not appear to be limited to oxidative hemolysis; they may also involve disruption of heme production.

The reductions in RBC and Hb counts observed in mercury-treated rats are likely attributable to oxidative damage to erythrocyte membranes and suppression of erythropoiesis, probably through mitochondrial dysfunction and bone marrow suppression (24-26). These findings are consistent with reports indicating that mercury chloride disrupts erythro-megakaryopoiesis and bone marrow progenitor activity, leading to impaired hematopoiesis (27). Similarly, mercury has been shown to induce hemoglobin oxidation and suppress erythrocyte antioxidant defenses (28). Wistar rats exposed to mercury chloride also showed increased oxidative stress markers and reduced hematological values (27,28). A similar pattern was observed in the present study, with reductions in RBC and Hb levels in the mercury-treated group, probably due to a combination of membrane oxidative injury and impaired erythropoiesis associated with mitochondrial dysfunction and bone marrow suppression. Wistar rats treated with mercury chloride presented stronger signs of oxidative stress and reduced hematological values (29,30).

Treatment with *A. djalonensis* extract exerted a protective effect on several hematological parameters. Co-administration improved hemoglobin concentration, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, and platelet-related indices, although the reduction in RBC count was not fully reversed (Table 1), indicating protective potential, possibly because of the plant's relatively high concentration of antioxidant phytochemicals, such as flavonoids and phenolic compounds. These substances may act as free radical scavengers by neutralizing reactive oxygen species (ROS), thereby helping protect mitochondria and erythroid precursor cells from mercury-induced injury. If mitochondrial enzymes are preserved and bone marrow activity is supported, erythropoiesis and thrombopoiesis are also likely to improve, thereby reducing the hematotoxic effects caused by mercury chloride. This outcome is also consistent with other studies in which natural plant antioxidants counteracted heavy metal-induced suppression of hematopoiesis (31,32).

The capacity of *A. djalonensis* to restore hematological parameters suggests that it has both cytoprotective and hematopoietic properties, making it a potential candidate for reducing heavy metal-induced hematotoxicity. However, the exact bioactive compound or compounds responsible for these effects were not analyzed in this study, and further research is needed to determine the precise molecular pathway or pathways involved. In addition, oxidative stress biomarkers, inflammatory cytokines, and bone marrow histology were not assessed in this research; evaluation of these parameters would provide a clearer understanding of how the plant preserves tissue integrity.

Despite these limitations, the present study suggests that *A. djalonensis* can reduce mercury-related hematological damage. This effect appears to occur through antioxidant activity and indirect improvement of bone marrow function. Overall, these results add to the broader body of evidence supporting the potential role of medicinal plants as alternative interventions for heavy metal-induced blood disorders, while also highlighting the need for further pharmacological and clinical research.

Conclusion

This study found that *A. djalonensis* attenuated mercury chloride-induced hematotoxicity, as demonstrated by improvements in hemoglobin concentration, erythrocyte indices, and platelet-related parameters. The observed restoration of hematological parameters indicates that the plant's antioxidant phytochemicals not only protect erythrocyte membranes from oxidative damage but may also promote hemoglobin synthesis and bone marrow proliferative activity. These two mechanisms, antioxidative protection and hematopoietic stimulation, provide a plausible explanation for recovery from mercury-induced hematological suppression. Although the findings are promising, further research is needed to identify the exact bioactive compounds involved, establish their roles in erythropoiesis and thrombopoiesis, and assess long-term safety and efficacy. Overall, *A. djalonensis* appears to be a promising natural medicinal agent for the treatment of hematological disorders caused by heavy metal toxicity.

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Ethical Statement

All experimental procedures involving animals were conducted in accordance with the guidelines for the care and use of laboratory animals established by the Research Ethics Committee of the College of Medical Sciences, University of Benin. Ethical approval for the study was obtained with approval number CMS/REC/2023/340.

Conflicts of Interest

The authors declare that there was no conflict of interest in the study.

Author Contributions

Joseph Raymond Enoghase: Investigation, resources, data curation, writing, review and editing, and visualization. Silvanus Olu Innih: Methodology, supervision, and project administration.

Data Availability Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Use of Artificial Intelligence

The author(s) hereby declare that no generative Artificial Intelligence (AI) technologies-including large language models (e.g., ChatGPT, Copilot, XLNet, and ERNIE 3.0 Titan) or text-to-image generators were used in the writing or editing of this manuscript.

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