



The Study of Hematological Parameters of Albino Rat after administration of Lead and Zinc Acetate

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ABSTRACT

This study investigates the effects of lead and zinc acetate on haematological parameters in albino rats. Lead, a known environmental toxicant, and zinc, an essential trace element, were administered to examine their impact on blood composition and function. The experiment utilized four groups of rats: a control group, a lead acetate group, a zinc acetate group, and a mixed lead and zinc acetate group. Significant variations in haematological parameters were observed across the groups ($P < 0.05$). The control group exhibited the highest red blood cell (RBC) count and hemoglobin levels, while the mixed lead and zinc acetate group showed the lowest values for these parameters. Hematocrit percentages were significantly higher in the control group compared to the treated groups. Mean Corpuscular Volume (MCV) and Mean Corpuscular Hemoglobin (MCH) showed an inverse trend, with the control group having the lowest values and the mixed group the highest. These findings provide insights into the complex interactions between lead and zinc and their effects on blood parameters, contributing to our understanding of metal-induced haematological alterations and potential health implications of exposure to these metals

KEY WORDS: Haematological parameters, Environmental toxicant, Zn, Pb,

Introduction

Lead and zinc are heavy metals that play significant roles in biological systems, albeit with contrasting effects. Lead, a well-documented environmental toxicant, has been a subject of concern for decades due to its widespread presence in the environment and its detrimental effects on human health. In contrast, zinc is recognized as an essential trace element, crucial for various physiological processes in living organisms. This study aims to investigate the effects of lead and zinc acetate on haematological parameters in albino rats, providing valuable insights into the potential health implications of exposure to these metals. Lead exposure remains a global health concern, particularly in developing countries, due to its persistence in the environment and its ability to accumulate in living organisms over time. The toxicity of lead is well-established, with

numerous studies demonstrating its adverse effects on multiple organ systems, including the nervous, renal, and haematological systems. Lead can interfere with haem synthesis, disrupt erythrocyte membrane stability, and alter the metabolism of other essential elements, leading to various haematological disorders such as anaemia, changes in red blood cell morphology, and alterations in white blood cell counts. On the other hand, zinc plays a crucial role in numerous physiological processes and is essential for proper immune function, wound healing, and DNA synthesis. As a cofactor for over 300 enzymes, zinc is involved in various metabolic pathways and is vital for maintaining cellular homeostasis. In the context of haematology, zinc is important for erythropoiesis, leukocyte function, and platelet aggregation. However, while zinc is generally considered beneficial, excessive intake can lead to adverse effects, including interference with the absorption of other essential minerals and potential toxicity. The haematological system is particularly sensitive to environmental toxicants and nutritional factors, making it an excellent indicator of overall health status. Changes in blood parameters can serve as important indicators of systemic toxicity or nutritional status. Red blood cell count, haemoglobin concentration, hematocrit, and white blood cell differential counts are among the key parameters that can be affected by exposure to heavy metals or alterations in trace element status. These haematological indices provide valuable information about the oxygen-carrying capacity of blood, immune system function, and overall health of an organism. By examining the effects of lead and zinc acetate on haematological parameters, this study seeks to elucidate the potential interactions between these metals and their impact on blood composition and function. The use of albino rats as an experimental model allows for controlled exposure and detailed analysis of haematological changes over time. This approach enables the investigation of dose-dependent effects, potential synergistic or antagonistic interactions between lead and zinc, and the time course of haematological alterations following exposure. Understanding the specific effects of lead and zinc on blood parameters is crucial for several reasons. Firstly, it can provide insights into the mechanisms of toxicity and the potential protective effects of essential elements against heavy metal poisoning. Secondly, it may help in developing more sensitive and specific biomarkers for assessing exposure to these metals in both occupational and environmental settings. Lastly, this knowledge can contribute to the development of targeted interventions or treatments for metal-induced haematological disorders. This research will contribute to the understanding of how lead and zinc affect haematological parameters in a controlled experimental setting. The findings may have implications for assessing and managing health risks associated with environmental or

occupational exposure to these metals, as well as for developing potential interventions or treatments. Moreover, this study may shed light on the complex interactions between toxic and essential elements in biological systems, furthering our understanding of metal homeostasis and its impact on health. In conclusion, this investigation into the effects of lead and zinc acetate on haematological parameters in albino rats represents an important step in elucidating the complex relationships between heavy metal exposure, essential element status, and blood health. The results of this study will not only contribute to the field of toxicology but also have potential implications for public health, occupational safety, and environmental protection.

Material and Methods:

Experimental Animals:

Adult male albino Wistar rats (weighing 180-200 g) were obtained from laboratory. The animals were housed in standard polypropylene cages under controlled conditions (temperature: $22 \pm 2^{\circ}\text{C}$, humidity: $55 \pm 5\%$, 12-hour light/dark cycle) with free access to standard rat pellet diet and water ad libitum. The rats were acclimatized for one week before the experiment.

Experimental Design:

Twelve rats were randomly divided into four groups (n=3 per group):

1. Control group: Received normal saline
2. Lead acetate group: Administered lead acetate (dose in mg/kg body weight)
3. Zinc acetate group: Administered zinc acetate (dose in mg/kg body weight)
4. Lead + Zinc acetate group: Administered both lead and zinc acetate (doses as above)

The treatments were administered daily via oral gavage for 28 days.

Chemical Compounds:

Lead acetate ($\text{Pb}(\text{CH}_3\text{COO})_2 \cdot 3\text{H}_2\text{O}$) and zinc acetate ($\text{Zn}(\text{CH}_3\text{COO})_2 \cdot 2\text{H}_2\text{O}$) of analytical grade were obtained from. Stock solutions were prepared in distilled water and diluted to the required concentrations before administration.

Blood Sample Collection:

At the end of the treatment period, rats were fasted overnight and anesthetized using. Blood samples were collected via cardiac puncture into EDTA-coated tubes for haematological analysis.

Haematological Analysis:

Complete blood count was performed using an automated haematology analyzer. The following parameters were assessed:

- Red blood cell (RBC) count
- Hemoglobin (Hb) concentration
- Hematocrit (HCT)
- Mean corpuscular volume (MCV)
- Mean corpuscular hemoglobin (MCH)

Blood Smear Examination:

Blood smears were prepared, fixed with methanol, and stained with Wright-Giemsa stain. The slides were examined under a light microscope for red blood cell morphology and to confirm the differential leukocyte count.

Results and Discussion:

The results of the hematological parameters shown in table across the four groups showed significant variations ($P < 0.05$). In Group A (Control), the RBC count was the highest at $7.22 \pm 0.13 \times 10^3/\mu\text{l}$, while Group B (Lead acetate group) exhibited a reduced count of $6.03 \pm 0.36 \times 10^3/\mu\text{l}$. Group C (Zinc acetate group) had a slightly higher RBC count ($6.32 \pm 0.13 \times 10^3/\mu\text{l}$) compared to Group B, whereas Group D (Mixed Doses of Lead Acetate and Zinc Acetate) showed the lowest RBC count ($5.89 \pm 0.18 \times 10^3/\mu\text{l}$).

Hemoglobin (Hb) levels were highest in Group A ($12.66 \pm 0.31 \text{ g/dl}$), followed by Group B ($11.93 \pm 0.52 \text{ g/dl}$) and Group C ($11.85 \pm 0.65 \text{ g/dl}$). Group D had the lowest Hb concentration ($10.94 \pm 0.72 \text{ g/dl}$). Similarly, hematocrit percentage was significantly higher in Group A ($41.24 \pm 0.75\%$) compared to the other groups, with Group B ($36.58 \pm 1.42\%$), Group C ($37.74 \pm 1.15\%$), and Group D ($35.48 \pm 1.28\%$) showing progressively lower values.

Mean Corpuscular Volume (MCV) was lowest in Group A ($55.12 \pm 2.96 \mu^3$) and increased in Group B ($61.77 \pm 3.68 \mu^3$), Group C ($62.60 \pm 2.27 \mu^3$), and Group D ($64.80 \pm 2.14 \mu^3$). Mean Corpuscular Hemoglobin (MCH) followed a similar trend, with the highest value in Group A (20.96 ± 0.26 pg), followed by Group B (19.43 ± 0.07 pg), Group C (19.28 ± 0.69 pg), and the lowest in Group D (17.45 ± 0.71 pg).

Overall, Group A (Control) demonstrated the most favorable hematological parameters, while Group D (Mixed Doses) exhibited the most adverse effects. Groups B and C showed intermediate effects, with Group C (Zinc acetate) displaying slightly better outcomes than Group B (Lead acetate). All differences were statistically significant ($P < 0.05$).

Table 1: Effects of Lead Acetate and Zinc Acetate Administration on Haematological parameters of Albino Rat

Parameter	Group A (Control)	Group B (Lead acetate group)	Group C (Zinc acetate group)	Group D (Mixed Doses of Lead Acetate and Zinc Acetate)	Significance
RBC Count ($\times 10^3/\mu\text{l}$)	7.22 ± 0.13	6.03 ± 0.36	6.32 ± 0.13	5.89 ± 0.18	$P < 0.05$
Hb (g/dl)	12.66 ± 0.31	11.93 ± 0.52	11.85 ± 0.65	10.94 ± 0.72	$P < 0.05$
Haematocrit %	41.24 ± 0.75	36.58 ± 1.42	37.74 ± 1.15	35.48 ± 1.28	$P < 0.05$
MCV (μ^3)	55.12 ± 2.96	61.77 ± 3.68	62.60 ± 2.27	64.80 ± 2.14	$P < 0.05$
MCH (pg)	20.96 ± 0.26	19.43 ± 0.07	19.28 ± 0.69	17.45 ± 0.71	$P < 0.05$

The present study evaluated the effects of lead acetate, zinc acetate, and their combination on hematological parameters, revealing significant alterations across all treated groups compared to the control. The findings indicate that lead acetate exposure adversely affects erythrocyte indices, while zinc acetate provides partial mitigation, though not sufficient to fully restore normal values. The combined exposure (lead and zinc) resulted in the most pronounced hematological disruptions, suggesting a potential synergistic or additive toxic effect.

Impact on RBC Count, Hemoglobin, and Hematocrit

The significant reduction in RBC count, hemoglobin (Hb), and hematocrit (HCT) in lead-exposed animals (Group B) aligns with previous studies demonstrating lead-induced anemia (Flora et al., 2012). Lead interferes with heme synthesis by inhibiting δ -aminolevulinic acid dehydratase (ALAD) and ferrochelatase, impairing hemoglobin production (Gurer-Orhan et al., 2004). The observed decrease in Hb and HCT in Group B supports this mechanism, as lead disrupts iron utilization and shortens erythrocyte lifespan (Patrick, 2006).

Zinc acetate supplementation (Group C) slightly improved RBC parameters compared to Group B, likely due to zinc's role in erythropoiesis and antioxidant defense (Prasad, 2013). However, the values remained lower than the control, suggesting that zinc alone cannot fully counteract lead-induced hematotoxicity. Notably, Group D (lead + zinc) exhibited the most severe reductions in RBC count, Hb, and HCT, possibly due to competitive absorption between lead and zinc or oxidative stress exacerbation (Brzóska et al., 2005).

Changes in MCV and MCH

The increase in MCV in lead- and zinc-treated groups (B, C, and D) suggests macrocytic changes, possibly due to impaired erythropoiesis or compensatory mechanisms in response to anemia (Kasper et al., 2015). Elevated MCV in lead toxicity has been linked to disrupted folate and vitamin B12 metabolism (Hoffman et al., 2013). The decline in MCH across all treated groups indicates hypochromic anemia, consistent with lead's interference with hemoglobin synthesis (Wani et al., 2015).

Comparative Analysis of Zinc's Protective Role

While zinc is essential for metallothionein synthesis, which may chelate lead and reduce its toxicity (Roney et al., 2006), our data indicate that zinc acetate alone (Group C) provided only marginal protection. This contrasts with some studies reporting stronger protective effects (El-Sokkary et al., 2005), possibly due to differences in dosage or exposure duration. The worsened hematological profile in Group D suggests that co-exposure may disrupt zinc's beneficial effects, necessitating further investigation into optimal dosing strategies.

Conclusion

Lead acetate exposure significantly impairs hematological parameters, inducing microcytic hypochromic anemia. Zinc acetate offers partial protection but fails to fully reverse lead's effects, while combined exposure exacerbates hematotoxicity. Future studies should explore higher zinc doses or additional antioxidants to enhance protective efficacy.

References

- Brzóska, M. M., et al. (2005). *Toxicology Letters*, 155(2), 175-183.
- El-Sokkary, G. H., et al. (2005). *Toxicology*, 207(1), 1-12.
- Flora, S. J. S., et al. (2012). *Indian Journal of Medical Research*, 136(1), 29-53.
- Gurer-Orhan, H., et al. (2004). *Human & Experimental Toxicology*, 23(9), 465-471.
- Hoffman, R., et al. (2013). *Hematology: Basic Principles and Practice*. Elsevier.
- Kasper, D. L., et al. (2015). *Harrison's Principles of Internal Medicine*. McGraw-Hill.
- Prasad, A. S. (2013). *Current Opinion in Clinical Nutrition & Metabolic Care*, 16(6), 669-674.
- Wani, A. L., et al. (2015). *Frontiers in Pharmacology*, 6, 1-10.