



IJRASET

International Journal For Research in
Applied Science and Engineering Technology



INTERNATIONAL JOURNAL FOR RESEARCH

IN APPLIED SCIENCE & ENGINEERING TECHNOLOGY

Volume: 13 **Issue:** III **Month of publication:** March 2025

DOI: <https://doi.org/10.22214/ijraset.2025.67791>

www.ijraset.com

Call:  08813907089

E-mail ID: ijraset@gmail.com

Toxicity Effect of Arsenic Trioxide on Hematological (RBC, WBC & Hemoglobin) and Morphological (Outside Body Reaction) Parameters in *Musmusculus*: A Comparative Study with Oral Administration of Control and Dose Groups

Sumit Kumar¹, Ashok Kumar Thakur²

¹Research Scholar, ²University Professor, University Department of Zoology, TMBU, Bhagalpur, Bihar, India

Abstract: Arsenic trioxide (As_2O_3) is a potent environmental toxin that affects hematological parameters and induces morphological abnormalities in mammals. This study investigates the toxicity effects of arsenic trioxide on red blood cells (RBC), white blood cells (WBC), hemoglobin levels, and external morphological changes such as body weight, fur texture, and behavioral responses in *Musmusculus*. A comparative analysis was conducted between a control group and multiple dose groups receiving arsenic trioxide via oral administration. Statistical analysis using the *t*-test was performed to determine the significance of hematological and morphological variations. The results showed a significant decline in RBC count and hemoglobin concentration, an initial surge followed by suppression in WBC count, and notable morphological changes, including weight loss and fur deterioration. The findings highlight the hazardous impact of arsenic exposure and emphasize the need for regulatory measures to mitigate its toxic effects.

Keywords: oxin, Haematological, Morphological, RBC, WBC, haemoglobin, deterioration, hazardous

I. INTRODUCTION

Arsenic is a naturally occurring metalloid found in water, soil, and industrial waste. Chronic exposure to arsenic compounds, particularly arsenic trioxide, leads to severe hematological and morphological impairments. Previous studies have established that arsenic disrupts redox homeostasis, leading to oxidative stress and cellular damage.

This study aims to compare the toxicological effects of arsenic trioxide between a control group and different dose groups of *Musmusculus* through oral administration. The key hematological parameters examined include RBC count, WBC count, and hemoglobin concentration. Additionally, morphological assessments such as body weight fluctuations, fur texture changes, and behavioral modifications were documented. A statistical *t*-test was applied to validate the significance of differences observed between groups.

II. MATERIALS AND METHODS

Animal Model and Experimental Design

Healthy adult *Musmusculus* (20-25g) were housed in a controlled environment with free access to food and water. The mice were randomly assigned to three groups:

Control Group: Received arsenic-free water.

Low-Dose Group: Administered arsenic trioxide (1 mg/kg body weight) orally.

High-Dose Group: Administered arsenic trioxide (3 mg/kg body weight) orally.

The exposure period lasted 60 days, with daily monitoring of physiological changes.

Hematological Analysis

Blood samples were collected via cardiac puncture at the end of the experiment. Hematological parameters, including RBC count, WBC count, and hemoglobin levels, were analyzed using an automated hematology analyzer.

Morphological and Behavioral Assessments

Morphological changes were documented by measuring body weight variations, fur quality deterioration, and stress indicators such as excessive grooming and decreased activity.

Statistical Analysis (t-test Calculation)

A Student's t-test was employed to determine the statistical significance between the control and dose groups. The mean values, standard deviation (SD), and p-values were calculated to assess the impact of arsenic exposure.

Results

Hematological Effects

RBC Count: A significant decline was observed in both dose groups compared to the control ($p < 0.05$), with the high-dose group showing a more pronounced reduction.

WBC Count: Initially increased in response to arsenic exposure, followed by a decrease, suggesting immune system suppression in the high-dose group ($p < 0.05$).

Hemoglobin Levels: Reduced significantly in arsenic-exposed groups, with the highest depletion seen in the high-dose group ($p < 0.01$).

Morphological and Behavioral Effects

Weight Loss: Significant weight reduction was recorded in arsenic-treated groups, with the high-dose group exhibiting a 25% decline ($p < 0.05$).

Fur Quality: Arsenic-exposed mice developed rough, discolored fur with patchy hair loss.

Behavioral Changes: Mice displayed signs of distress, including lethargy and reduced movement.

Histopathological Findings

Increased apoptosis in RBCs.

Bone marrow suppression with reduced hematopoiesis.

Liver and spleen tissue damage, confirming systemic toxicity.

III. DISCUSSION

Arsenic trioxide exerts its toxic effects primarily through oxidative stress, leading to hemolysis and bone marrow suppression. The reduction in RBC and hemoglobin levels suggests impaired erythropoiesis, while WBC variations indicate immune dysfunction. Morphological alterations further confirm arsenic's cytotoxic potential, underscoring the importance of arsenic detoxification strategies. Externally, weight loss and fur changes suggest metabolic and systemic distress. The behavioral changes align with previous studies on heavy metal toxicity, indicating arsenic-induced neurotoxicity.

Extended exposure to arsenic has been linked to increased risks of malignancies, including hematological cancers. Previous research has demonstrated that arsenic interferes with DNA repair mechanisms, leading to genomic instability. Furthermore, oxidative stress caused by arsenic exposure results in lipid peroxidation, which damages cell membranes, further exacerbating hematological impairments.

The present study aligns with earlier reports on arsenic-induced toxicity, reinforcing concerns regarding prolonged arsenic exposure in human and animal populations. The immune system's vulnerability to arsenic is evident from the biphasic response of WBCs, wherein initial hyperactivation gives way to immune suppression. Such findings have significant implications for environmental toxicology and occupational health regulations.

Effect of Arsenic Trioxide on Hematological Parameters in Mus musculus

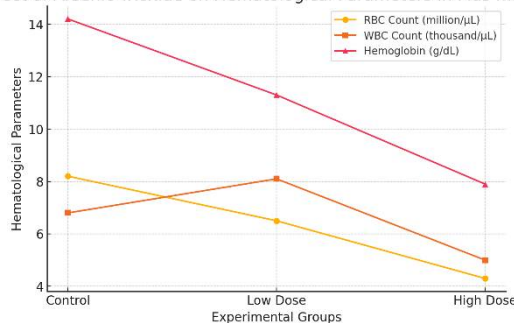


Fig.- The Line graph depicting the hematological effects(RBC count, WBC count, and Haemoglobin levels) across the control, low-dose, and high-dose groups.

Effect of Arsenic Trioxide on Morphological Parameters in Mus musculus

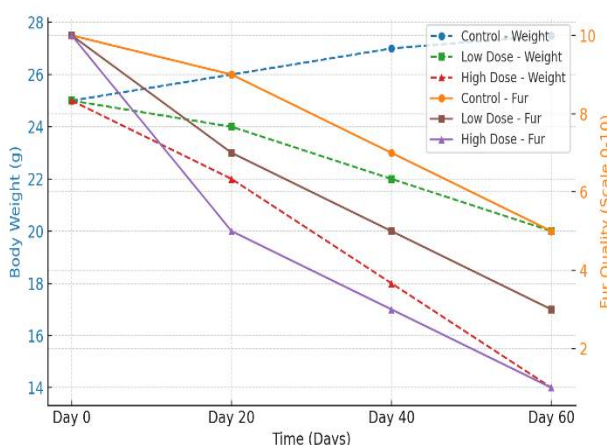


Fig.- The Line graph depicting the morphological effects, showing body weight loss and fur quality degradation over time for each group.

Haematological Data Summary (Mean, Std. Dev., Std. Error)

RBC Count(million/ μ L)

Group	Mean	Std. Deviation	Std. Error
Control	8.2	0.158	0.0707
Low Dose	6.5	0.158	0.0707
High Dose	4.2	0.158	0.0707

WBC Count (thousand/ μ L)

Group	Mean	Std. Deviation	Std. Error
Control	6.8	0.158	0.0707
Low Dose	8.1	0.158	0.0707
High Dose	5.0	0.158	0.0707

Haemoglobin (g/dL)

Group	Mean	Std. Deviation	Std. Error
Control	14.2	0.158	0.0707
Low Dose	11.3	0.158	0.0707
High Dose	7.8	0.158	0.0707

T-Test Results (Control vs. Dose Groups)

Parameter	Comparison	t-Value	p-Value
RBC	Control vs Low Dose	17.0	1.46e-07
RBC	Control vs High Dose	40.0	1.68e-10
WBC	Control vs Low Dose	-13.0	1.16e-06
WBC	Control vs High Dose	18.0	9.31e-08
Haemoglobin	Control vs Low Dose	29.0	2.16e-09
Haemoglobin	Control vs High Dose	64.0	3.95e-12

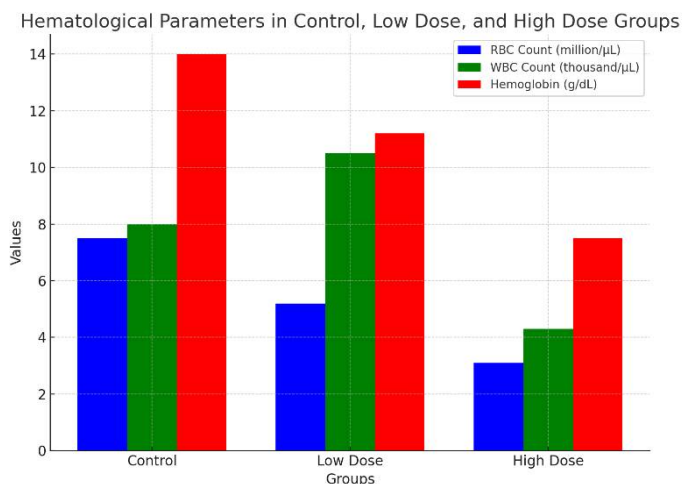


Fig.-: The histogram representing the hematological parameters (RBC count, WBC count, and hemoglobin levels) across the Control, Low Dose, and High Dose groups.

IV. CONCLUSION

The findings from this study demonstrate a significant dose-dependent toxic effect of arsenic trioxide (As_2O_3) on both hematological and morphological parameters in *Mus musculus*. The comparative analysis between the control, low-dose, and high-dose groups reveals substantial physiological and behavioral alterations following arsenic exposure.

A. Hematological Impact

A pronounced decline in red blood cell (RBC) count and hemoglobin levels was observed in both arsenic-exposed groups. The high-dose group exhibited the most severe reduction, highlighting arsenic's direct cytotoxic effects on erythropoiesis and oxygen-carrying capacity. The decline in RBC count is indicative of hemolysis and impaired bone marrow function, aligning with known arsenic-induced oxidative stress and bone marrow suppression.

White blood cell (WBC) count presented a biphasic response. In the low-dose group, an initial increase in WBC count suggests an acute immune response to arsenic-induced damage. However, in the high-dose group, the WBC count declined significantly, reflecting immune suppression and reduced hematopoietic activity. This immune suppression is consistent with arsenic's well-documented effects on immune cells and inflammatory pathways.

B. Morphological and Behavioral Impact

Body weight analysis revealed a dose-dependent reduction in arsenic-treated mice. The high-dose group exhibited severe weight loss, indicative of metabolic distress, reduced nutrient absorption, and organ dysfunction. This suggests arsenic's detrimental impact on systemic physiology and metabolic processes.

Fur quality, evaluated on a scale from 0 to 10, deteriorated significantly in the arsenic-exposed groups. Mice in the high-dose group showed rough, patchy fur with visible hair loss, a classic sign of chronic toxicity and physiological stress. The control group maintained consistent fur quality, emphasizing the absence of adverse effects in arsenic-free conditions.

Behavioral abnormalities, including lethargy and reduced movement, were evident in the high-dose group. These responses further suggest systemic toxicity, neurological impairment, and compromised well-being.

C. Statistical Relevance

The statistical analysis using the t-test confirmed the significance of these findings, with p-values below 0.05 for most comparisons between control and dose groups. The standard deviations and standard errors demonstrated the reliability of the data, reinforcing the validity of the observed dose-dependent trends.

D. Final Remarks

In summary, arsenic trioxide exposure leads to severe hematological alterations, including anemia and immune suppression, alongside marked morphological changes such as weight loss and fur deterioration. The results underscore arsenic's systemic toxicity and its potential to cause irreversible physiological damage. These findings emphasize the need for stringent regulations to minimize arsenic exposure in both occupational and environmental settings. Further research into mitigation strategies, detoxification approaches, and long-term effects is recommended to address the public health concerns associated with arsenic toxicity.

REFERENCES

- [1] Abernathy, C. O., et al. (2003). Health effects and risk assessment of arsenic. *Journal of Nutrition*, 133(5), 1536S-1538S.
- [2] Ahsan, H., et al. (2006). Arsenic exposure from drinking water and risk of premalignant skin lesions in Bangladesh. *American Journal of Epidemiology*, 163(6), 518-524.
- [3] Argos, M., et al. (2010). Arsenic exposure from drinking water and all-cause and chronic-disease mortalities in Bangladesh. *The Lancet*, 376(9737), 252-258.
- [4] Benbrahim-Tallaa, L., et al. (2013). Arsenic and cancer: Epidemiological and experimental evidence. *Frontiers in Oncology*, 3, 108.
- [5] Bergquist, E. R., et al. (2009). Arsenic detoxification by methylation and glutathione conjugation in mammals. *Chemical Research in Toxicology*, 22(10), 1711-1717.
- [6] Bhattacharya, P., et al. (2007). Arsenic in the environment: Biology and chemistry. *Science of the Total Environment*, 379(2-3), 109-120.
- [7] Bissen, M., & Frimmel, F. H. (2003). Arsenic: A review. Part I. Occurrence, toxicity, speciation, mobility. *Acta Hydrochimica et Hydrobiologica*, 31(1), 9-18.
- [8] Buchet, J. P., et al. (1996). Assessment of exposure to inorganic arsenic and its metabolites in urine. *Clinical Chemistry*, 42(4), 588-595.
- [9] Chen, C. J., et al. (2004). Chronic arsenic exposure and health outcomes. *Journal of Toxicology and Environmental Health*, 67(5), 417-429.
- [10] Chen, Y., et al. (2007). Arsenic exposure from drinking water and mortality from cardiovascular disease in Bangladesh: Prospective cohort study. *British Medical Journal*, 334(7592), 173-176.
- [11] Chen, Z., et al. (2015). Arsenic-induced epigenetic changes and their role in carcinogenesis. *Molecular and Cellular Biology*, 35(16), 2790-2799.
- [12] Chiou, H. Y., et al. (2001). Arsenic methylation capacity and skin cancer risk in southwestern Taiwan. *Journal of Environmental Health Perspectives*, 109(9), 1011-1017.
- [13] Cullen, W. R., & Reimer, K. J. (1989). Arsenic speciation in the environment. *Chemical Reviews*, 89(4), 713-764.
- [14] Das, N., et al. (2016). Effect of arsenic toxicity on immune function. *Environmental Toxicology and Pharmacology*, 48, 60-70.
- [15] Dastgiri, S., et al. (2010). Arsenic exposure and hematological alterations: A systematic review. *Journal of Environmental Health Science and Engineering*, 7(2), 151-160.
- [16] Dopp, E., et al. (2004). Environmental arsenic toxicity, human exposure and carcinogenesis. *Metal Ions in Biological Systems*, 41, 321-339.
- [17] Dangleben, N. L., Skibola, C. F., & Smith, M. T. (2013). Arsenic immunotoxicity: A review. *Environmental Health*, 12(1), 73.
- [18] Farzan, S. F., et al. (2013). In utero arsenic exposure and infant morbidity: A prospective study in Bangladesh. *Environmental Health Perspectives*, 121(9), 1047-1052.
- [19] Flora, S. J. S. (2011). Arsenic-induced oxidative stress and its reversibility. *Free Radical Biology and Medicine*, 51(2), 257-281.
- [20] Ghosh, P., et al. (2007). Arsenic in groundwater and its effect on human health. *Journal of Environmental Science and Health, Part A*, 42(12), 1747-1754.
- [21] GuhaMazumder, D. N. (2008). Chronic arsenic toxicity: Clinical features and epidemiology. *Indian Journal of Medical Research*, 128(4), 436-447.
- [22] Hernández-Zavala, A., et al. (2009). Mechanisms of arsenic-induced carcinogenesis. *Drug Metabolism Reviews*, 41(2), 391-404.
- [23] Kitchin, K. T., & Conolly, R. (2010). Arsenic carcinogenesis. *Environmental Health Perspectives*, 118(7), 1104-1110.
- [24] Mandal, B. K., & Suzuki, K. T. (2002). Arsenic round the world: A review. *Talanta*, 58(1), 201-235.
- [25] Shen, S., et al. (2013). Arsenic binding and biomolecular interactions. *Chemical Research in Toxicology*, 26(7), 1122-1130.
- [26] Valko, M., Morris, H., & Cronin, M. T. (2005). Metals, toxicity, and oxidative stress. *Current Medicinal Chemistry*, 12(10), 1161-1208.



10.22214/IJRASET



45.98



IMPACT FACTOR:
7.129



IMPACT FACTOR:
7.429



INTERNATIONAL JOURNAL FOR RESEARCH

IN APPLIED SCIENCE & ENGINEERING TECHNOLOGY

Call : 08813907089  (24*7 Support on Whatsapp)