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Investigating the Impact of Lead Toxicity on Erythrocyte Indices in Albino Rats

[1] Neetu Sharma *
Department of Zoology
Maharaja Agrasen Mahavidyalaya, Bareilly
&
[2] Chanchal Srivastav
Khandelwal College of Science and Technology Bareilly

Abstract

Lead is a chronic environmental pollutant that is of great health concern to humans and animals. This research examined the hematological consequences of lead acetate exposure in albino rats. Rats were randomly distributed into a control group or a lead acetate-treated group. The lead acetate group had much lower red blood cell (RBC) count, hemoglobin level, packed cell volume (PCV), mean corpuscular volume (MCV), and mean corpuscular hemoglobin (MCH) than the control group. These results show the occurrence of microcytic hypochromic anemia, a frequent hematological presentation of lead toxicity. The pathways for these alterations include interference by lead with heme synthesis through inhibition of critical enzymes, production of reactive oxygen species causing oxidative stress and hemolysis of erythrocytes, and deposition in the bone marrow, interfering with erythropoiesis. The findings underscore the necessity of hematological monitoring in populations exposed to lead and the need for more studies on therapeutic interventions to counteract lead-induced hematotoxicity. This research supplements the large animal literature on lead toxicity, which has repeatedly shown extensive neurobehavioral effects in many species. The results highlight the ongoing risk of environmental lead contamination and the need for public health interventions to reduce exposure and protect susceptible populations.

Keywords: *Lead, Toxicity, Haematological Parameters, Albino Rat*

Introduction:

Contamination of the environment with trace elements as a result of anthropogenic sources shows that the world may already be witnessing a silent epidemic of metal poisoning in the environment. Of the trace elements, lead has been an archetypal environmental and industrial contaminant with toxicological problems traced across human history. Lead's toxicity was known to humans as far back as the second century B.C., and it had severe health problems in lead mining areas even during that time. Lead poisoning from accidental and evil sources has been documented since antiquity, although environmental exposure to lead was an important public health problem in industrialized countries throughout the 20th century (Tchounwou et al., 2012). This is because lead in the environment remains persistent, following its non-biodegradable nature and extensive utilization in modern industries. Some major sources of lead

* Corresponding Author: Neetu Sharma
Email: dr.mehrotraneetu@gmail.com
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contamination in the environment include the emissions from road traffic, paints, pigments, pesticides, food packaging materials, automobile exhausts, agrochemicals, used lead batteries, lead pipes, smelters, residues from gunshot, and lead recycling plants (WHO, 2021). It is approximated that the present world environment holds over 100 times the quantity of lead found in pre-historic times, and it poses a growing hazard to animal and human health (Ericson et al., 2021). Lead poisoning has been widely documented in domestic and farm animals, as well as in many bird species (Martinez-Haro et al., 2019).

Experimental studies of lead poisoning in animal models yield instructive information supplementary to correlative research in people, especially children. Randomized assignment of individuals to treatment regimens and stringent control over exposure to lead facilitate causal inference about lead dosing and neurobehavioral consequence. There exists a vast corpus of animal literature that has routinely shown statistically significant effects on a variety of behavioural endpoints in rats, mice, sheep, dogs, and monkeys at relatively high levels of exposure (Mason et al., 2014). Moreover, lead effects at lower exposure levels have also been reported, testifying to the sensitivity of biological systems to even low levels of lead (Lanphear et al., 2018).

MATERIALS AND METHODS:

Experiments were conducted on male albino rats (Rattus norvegicus) procured from the Laboratory. Before commencing of the experiment, the animals were acclimatized to the new environment for 60 days. During this period, they were examined regularly for haematological studies.

Each animal was marked with a yellow stain on its back, head, tail, head-tail, back-tail, head-back tail and one was left unmarked in order to facilitate daily examination. These experimental animals, (150-300 gm) were divided into 2 groups, viz. A and B each comprising of 6 animals. The albino rats (Rattus norvegicus) of different groups were given the following laboratory treatment for 60 days:

Group- A (Healthy Control) treatment with Sodium acetate (125 mg/body weight)

Group- B Lead intoxication treatment with Lead acetate (125 mg/body weight)

Oral administration of doses was continued for 60 days through tubes. Doses were selected as per method described by Zbinden (1976).

Blood was collected at zero, 30th and 60th day from retro orbital plexus with the help of capillary tube as described by Sorz and Buckner (1964). Blood was collected in two aliquots. In one aliquot, blood was drawn in heparinized vials through clean, sterilized syringes washed with hot nitric acid. The samples were stored in air-tight vials at -20°C till used.

Results and Discussion:

Administration of lead acetate caused drastic changes in hematological indices in albino rats, as indicated by the data. Red blood cell (RBC) count in the lead acetate-treated group (5.03±0.36×103/µl5.03±0.36×103/µl) was found to be drastically lower ($p < 0.05$) than that of the control group (6.70±0.11×103/µl6.70±0.11×103/µl), which reflects a severe decrease in erythrocyte production. The reduction in RBC count is in line with lead-induced anemia, which is a widely reported effect of lead toxicity. Lead disrupts heme biosynthesis by blocking important enzymes like δ-aminolevulinic acid dehydratase (ALAD) and ferrochelatase, which are important for haemoglobin synthesis and red blood cell maturation (Flora et al., 2012). The decrease in heamoglobin level from 12.27±0.07 g/dl12.27±0.07g/dl in the control group to 9.43±0.52 g/dl9.43±0.52g/dl in the lead acetate group further reinforces this mechanism because lead's disruption of heme synthesis directly hampers heamoglobin synthesis.

Packed cell volume (PCV), an indicator of the percentage of blood volume comprised of red blood cells, was also markedly low in the lead acetate group (24.58±1.42%24.58±1.42%) relative to the control group (40.20±0.20%40.20±0.20%). This reduction in PCV indicates a lowering of the total erythrocyte mass, and it further supports the occurrence of anemia. In addition, the mean corpuscular volume (MCV) in the lead acetate-treated group (27.77±3.68 fl/cell27.77±3.68fl/cell) was notably smaller than in the control group (47.77±0.53 fl/cell47.77±0.53fl/cell), indicating the onset of microcytic anemia. Microcytosis, a smaller-than-normal red blood cell, is characteristic of lead toxicity and is commonly seen with deranged iron metabolism and heme synthesis (Wani et al., 2015).

The mean corpuscular haemoglobin (MCH) was also decreased in the lead acetate group (14.43±0.07 pg/dl14.43±0.07pg/dl) versus the control group (16.50±0.20 pg/dl16.50±0.20pg/dl), showing hypochromia, a state of reduced heamoglobin content in red blood cells compared to normal. These data in combination indicate the onset of microcytic hypochromic anemia, a frequent hematological finding in lead toxicity.

The processes responsible for these hematologic alterations are complex. Generation of reactive oxygen species (ROS) by lead is a factor in oxidative stress, which destroys erythrocyte membranes and enhances hemolysis, exacerbating anemia further (Patrick, 2006). Lead also accumulates in the bone marrow, where it interferes with erythroid precursor cell differentiation and maturation, resulting in inhibited erythropoiesis (Wani et al., 2015). The additive effects of compromised heme production, oxidative stress, and bone marrow inhibition lead to the resultant decreases in RBC, heamoglobin content, and PCV.

Overall, the findings confirm that lead acetate exposure causes extensive hematological changes in albino rats, which include microcytic hypochromic anemia. These results are consistent with established mechanisms of lead toxicity, including the interference of lead with heme synthesis and erythropoiesis. The findings emphasize the necessity of surveillance of hematological indices in groups exposed to lead and underscore the need for further investigation into therapeutic interventions to counteract the detrimental effects of lead-induced hematotoxicity.

Table 1: Effects of Lead Acetate Administration on Packed Cell Volume, Red Blood Cell Count, Haemoglobin Concentration, and Erythrocyte Indices

Parameter	Group A (Control)	Group B (Lead Acetate Only)	Significance
RBC Count (x 10^3/µl)	**6.70 ± 0.11**	**5.03 ± 0.36**	**P < 0.05**
Hb (g/dl)	**12.27 ± 0.07**	**9.43 ± 0.52**	**P < 0.05**
PCV (%)	**40.20 ± 0.20**	**24.58 ± 1.42**	**P < 0.05**
MCV (fl/cell)	**47.77 ± 0.53**	**27.77 ± 3.68**	**P < 0.05**
MCH (pg/dl)	**16.50 ± 0.20**	**14.43 ± 0.07**	**P < 0.05**

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