

Growth Performance, Haematological And Physiological Responses Of Male Wistar Rats Exposed To Lead Acetate

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Abstract

Human exposure to lead poses serious public health problems owing to lead toxicity. It has been alleged that frequent intake of lead over can be detrimental to human health. This underscores why this study was carried out to evaluate the effects of lead acetate on the haematology and histology of male wistar rats at low dosage over a long period of time. Forty (40) adult wistar rats procured and housed at the Department of Veterinary Physiology and Pharmacology Teaching and Research Farm, University of Nigeria Nsukka. They were divided into 2 groups and replicated four times with 5 rats per replicate. The treated (group A) were dosed with 60mg/kg while the untreated (group B) were not exposed to lead acetate. The experiment lasted for four months during which the body weights and the haematological parameters of the wistar rats were recorded on monthly basis. At the end of the experiment, the gonads were decapitated for histological examination. The data collected were subjected to analysis of variance (ANOVA) using SPSS version 21. The probability level of $P < 0.05$ was used to determine the level of significance. The results showed that there were no significant change(s) on the body weights of the wistar rats. The Hb, PCV and RBC of the treated group had significant ($p < 0.05$) reduction almost below the normal values of the wistar rats of their age and size while the WBC tend to increase significantly above the control group. The histological examination of the gonad showed normal robust seminiferous tubules in the organs of wistar rats from the control groups while the treated groups had wide intertubular spaces with shrunk seminiferous tubules. Significant reduction in number of sperm cells of the treated groups as against the control groups was also observed. This study therefore revealed that lead acetate has some detrimental effects on haematology and reproductive physiology even at a very low dose of 60mg/kg.

Keywords: lead acetate, haematology, albino rats, histology, reproductive physiology

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I. INTRODUCTION

Endocrine disruptors are chemicals that, at certain doses, can interfere with endocrine (or hormone) systems. Body systems are controlled by hormones and these hormones can be derailed by endocrine disruptors. They interfere with the synthesis, secretion, transport, binding, neurodevelopment, reproductive organs, fertility, behaviour, and maintenance of homeostasis (Virgolini and Aschner, 2021). However, one of these endocrine disrupting chemicals of interest is lead acetate (Pb). Lead is a naturally occurring bluish-gray heavy metal found in abundance and an essential environmental pollutant of anthropogenic sources (Jaishankar *et al.*, 2014). It is one of the most abundant heavy metal and has been used globally for centuries. Lead is considered as one of the most hazardous pollutants and toxins that are found in our environment (Patra *et al.*, 2011) thus, is a carcinogenic (cancer-causing) element. It is ambiguous and can be found in all parts of our environment – the air, the soil, the water, and even inside our homes (WHO, 2019).

Studies in cells and laboratory animals have shown that lead acetate can cause adverse biological effects in animals (Sunil, 2018). Lead acetate in the environment may contribute to both infertility and reproductive failures in wildlife and bans and restrictions on the use of these chemical is associated with reduction in health problems as well as the recovery of wildlife populations (Pain *et al.*, 2019). Thus, there is limited information on low-level exposure of lead acetate on male wistar rats over a long period of time. It is on this backdrop that this study was design to investigate the effect of low concentration of lead acetate on haematological and histological responses of male wistar rats.

Objectives

The objectives of this present study are;

1. To determine the effect of lead acetate on growth performance of wistar rats.
2. To evaluate the long-term effects of lead acetate on haematological profile of male wistar rats.
3. To determine the effect of lead acetate on histological examinations of male wistar rats.

II. MATERIALS AND METHODS

Study Area

The work was carried out in the department of Veterinary Physiology and Pharmacology laboratory, University of Nigeria, Nsukka. Nsukka is located in South-East Nigeria in Enugu State at latitude 6.85783 and longitude 7.39577

Research Procedure

The procedure used for the work includes:

Reagents and Chemicals

The reagent and chemical used were lead 11 acetate trihydrate, distilled water, normal saline, normal saline, red blood cell diluting fluid, white blood cell diluting fluid, phosphate buffer, immersion oil, blood sample, and semen.

Instruments, Materials and Glass Wares

Haemocytometer (Improved Neubauer chamber), Light microscope, Analytical weighing balance, Test tubes and racks, Surgical blades, ceramics mortar and pestle, Beakers, Cover slips, Vita grower feeds, aluminium cages, albino rats, 1ml syringe, intubation cannula, capillary tubes, red blood cell diluting pipette, white blood cell diluting pipette, microscope slides, leishman stain, micropipette, nylon sieve

Procurement and Acclimatization of the Animals

Forty healthy sexually matured Albino rats (*Rattus norvegicus*) comprising of forty (40) males weighing between 200 and 250g were sourced from the Faculty of Veterinary Medicine. The rats were kept in clean stainless steel wire mesh aluminium cages and housed in the animal house unit of the Department of Veterinary Physiology/ Pharmacology, University of Nigeria, Nsukka. The rats had no history of drug consumption (i.e. they have not been used for any investigation). Standard commercial pelleted feed (Vita Grower feeds, Nigeria) and clean drinking water were given to the rats *ad libitum*. Prior to the commencement of the experiment, the rats are allowed a space of two weeks (14 days) for acclimatization. They were allowed access to food and water. The faecal droppings were removed daily.

Experimental Design

The design of the experiment was Completely Randomized design with two treatments and five replicates. The T-test was used to compare the two treatment groups. And the probability level of $P < 0.05$ was used to determine the level of significant.

A total number of forty (40) male wistar rats were divided into two groups (A and B) of twenty per group. The experimental group A were the (treated group) while group B were (untreated group) or control group. Group A were given the same concentration of lead acetate at weekly interval based on their body weight following oral route of administration while Group B were not given lead acetate.

Statistical Analysis: The data collected were subjected to analysis of variance (ANOVA) using Statistical Package for Social Sciences (SPSS) version 21 while the statistically different means were separated using Duncan's option as found in the statistical package/software (Duncan, 1955). The test statistical probability level of $P < 0.05$ was used to determine the level of significant.

Determination of Body Weight

The body weights of the rats were determined once in every 28 days using Metler Electronic Weighing balance

Preparation of Reagent

Lead acetate trihydrate was obtained from Oxford Lab. Co., India (CAS: 6080-56-4). Lead acetate was dissolved in distilled water at concentration of 60 mg/kg body weight and administered to rats by gavage tube.

Collection of Blood Samples

Blood samples of the rats were collected once every twenty eight (28) days for five (5) months from each group to determine various haematological parameters.

Haematological Profile:

Haematological determination of Red Blood Cell (RBC), White Blood Cell (WBC), Haemoglobin (Hb), Packed Cell Volume (PCV), Mean Cellular Haemoglobin Concentration (MCHC), and Mean Cellular Haemoglobin (MCH) were assessed monthly while the differential Leucocyte count will be prepared using the method as described by Barbara (1975).

Total White Blood Cell Count (WBC)

The method of RBC count was according to Sood (2006). The blood was properly mixed for approximately one minute. Using the aspirator and white cell pipette, the blood was drawn up to the 0.5 mark in the pipette. It was then diluted up to the 11 mark with a 1.5% solution of acetic acid in water, tinted with methyl violet. Using the low power count (x10 objective), the white cells seen over the whole of the ruled area were counted, that is nine squares, each of 1sqmm in area. If the cells are satisfactorily and uniformly distributed, the figures for total white cells in each square millimeter should not differ from each other by more than eight to ten cells. Since the depth of fluid over the 9sqmm, the total number of which cells counted within the 9sqmm is the number present in 0.9c.mm. Since the original dilution of the blood was 1:20,

$$\text{White Blood Cell Count} = \frac{\text{No of cell counted} \times \text{Dilution factor (DF)}}{\text{Volume counted in mm}^3}$$

$$\text{WBC/mm}^3 = \text{cell counted} \times 50$$

Where N = no of cell counted

DF = Dilution factor = 1: 200

Volume counted = 0.02mm³

Packed Cell Volume (PCV)

The packed cell volume is the percentage of erythrocytes in whole blood. The PCV was determined by the hematocrit method as described by Dacie and Lewis (1991). The capillary tube was filled with blood. One end of the capillary tube was sealed with plasticine and was placed in a cell compartment of the centrifuge, spun/centrifuged at 3000rpm for 5 minutes to ensure maximum packing of cells. The PCV was determined by measuring the height of the red cell column and expressing this as a ratio of the height of the total blood column. The PCVs were read using a microhematocrit reader and the result will be expressed in percentage as:

$$\text{PCV (\%)} = \frac{\text{Height of red cell (mm)} \times 100}{\text{Total height (mm)}}$$

Haemoglobin Concentration (Hb)

It was determined using the method described by Drabkin 1932. Five millilitre of drabkin's solution was added in a test tube and mixed with 20microliter of blood. The mixture was allowed to stand for five minutes for transformation of haemoglobin to haemoglobincyanide. The mixture was poured into a cuvette and the absorbance was read in a spectrophotometer at 540 nanometres. The haemoglobin in g/dl was calculated using the formular: Hb in g/dl = $\frac{\text{Absorbance of test sample}}{\text{Absorbance of standard}} \times \frac{\text{Dilution factor}}{100}$

Red Blood Cell (RBC)

The method of RBC count was according to Sood (2006). The blood was sucked slowly and carefully up to 0.5mark in the red blood cell diluting pipette. The pipette was plunged into the diluting fluid and sucked up to the 101 mark. Then, the ends of the pipette was gripped between the finger and the thumb, and mixed thoroughly for about three minutes. The Neubauer counting chamber was charged with one drop of the diluted blood sample and allowed to settle for two minutes. The RBCs in the five groups of 16 small squares in the central area of the Neubauer chamber was counted using a light microscope at a high magnification of x 40 objective. The number of cells counted for each sample was calculated using the formular:

$$\text{Red Blood Cell Count} = \frac{\text{No of cell counted} \times \text{Dilution factor}}{\text{Volume counted in mm}^3}$$

$$\text{Then RBC (mm)}^3 = \frac{\text{No of cell counted} \times 200}{0.02 \text{ mm}^3}$$

DF = Dilution factor = 1: 200

Volume counted = 0.02mm³

Estimation of Red Blood Cell Indices

The red blood cell indices are used to define the size and hemoglobin content of the red blood cell. They consist of the MCV (mean corpuscular volume), MCH (mean corpuscular hemoglobin), and MCHC (mean corpuscular haemoglobin concentration). The red blood cell indices are used as an aid in differentiating anemia; when these are used together with an examination of the red cells on the stained smear, a clear picture of red cell morphology may be ascertained

Estimation of Mean Cell Volume (MCV)

The MCV indicates the average volume of red blood cells. It was determined using the method described by Baker, Silverton & Pallister (2001). The formula is given below;

$$\text{MCV} = \frac{\text{Haematocrit} \times 10}{\text{Red blood count in millions}}$$

Estimation of Mean Cell Haemoglobin (MCH)

MCH indicates the average weight of haemoglobin in the red blood cell. It was determined using the method described by Baker, Silverton & Pallister (2001). The formula is given as:

$$\text{MCH} = \frac{\text{Haemoglobin g/dl} \times 10}{\text{Red blood count in millions}}$$

Estimation of Mean Cell Haemoglobin (MCHC)

MCHC is an expression of the average concentration of hemoglobin in the red blood cells. It gives the ratio of the weight of hemoglobin to the volume of the red blood cell. It was determined using the method described by Baker, Silverton and Pallister (2001). The formula is therefore given as:

$$\text{MCHC} = \frac{\text{Haemoglobin} \frac{\text{g}}{\text{dl}} \times 100\%}{\text{Haematocrit}}$$

Hormone Assay

The blood samples collected were centrifuged at 2500rpm for 5min using Wispertuge model 1384 centrifuge (Tamson, Holland) at 10-25°C to obtain the serum sample which was analysed for testosterone, follicle stimulating hormone (FSH), luteinizing hormone (LH) and estradiol hormone level using enzyme linked immunoassay (ELISA) technique; using analytical grade reagents (Syntron Bioresearch Inc., USA).

III. RESULTS

The result of the monthly weight gain of male wistar rats showed that there were no significant ($p > 0.05$) different among the treatment variables, however there were numerical differences where by the untreated group were slightly higher than their treated counterpart as the experiment progressed.

The results of the haematological parameters showed that there were significant ($p < 0.05$) difference only on white blood cell (WBC) and lymphocyte (L). The other parameters were not significantly affected by the treatment (lead acetate). In WBC, the treated group had the value of $9.60 \pm 0.06 \text{ mm}^3$ which was significantly higher ($p < 0.05$) than the value of $8.42 \pm 0.04 \text{ mm}^3$ obtained from the untreated group. While the lymphocyte follow similar trend as white blood cell in which the treated group had higher value than the untreated value.

In the second month, the haematological parameters showed that there were significant ($p < 0.05$) difference in the PCV, RBC, Hb and Lymphocyte. In PCV, the untreated group had higher PCV percentage of $40.23 \pm 0.25 \%$ which was significantly ($p < 0.05$) higher than the value of $35.85 \pm 0.01\%$ obtained from the treated group. In RBC, the treated group had lower RBC value of $6.97 \pm 0.00 \text{ mm}^3$ which was significantly ($p < 0.05$) different from the value of $7.42 \pm 0.01 \text{ mm}^3$ obtained from the untreated group. In haemoglobin concentration (Hb), the treated group had lower value of $11.92 \pm 0.01 \text{ g/dl}$ which differed ($p < 0.05$) significantly from the value of $13.49 \pm 0.11 \text{ g/dl}$ obtained from the untreated group while in lymphocyte, the treated group had $85.0 \pm 1.28\%$ which was significantly ($p < 0.05$) higher than the untreated group which had lower value of $83.20 \pm 0.70 \%$.

However, in the third month, the PCV, RBC, WBC, Hb, MCV, MCH, Lymphocyte, and Neutrophil were significantly affected by the treatment (lead acetate). In PCV, untreated group had the highest value of 45.05% which was significantly ($p < 0.05$) different from the value of 39.84% obtained from the treated group. In RBC, the untreated group had the value of $8.01 \pm 0.02 \text{ mm}^3$ which differed significantly from the value of $7.41 \pm 0.22 \text{ mm}^3$ obtained from the treated group. On the contrary, the results obtained from WBC, shows that the treated group had the higher value of $10.0 \pm 0.21 \text{ mm}^3$ which were statistically ($p < 0.05$) different from the value of $8.12 \pm 0.03 \text{ mm}^3$ obtained from untreated group. Thus, in hemoglobin concentration (Hb) the untreated group had the value of $14.05 \pm 0.24 \text{ g/dl}$ which was significantly ($p < 0.05$) higher than the value of $11.22 \pm$

0.06g/dl obtained from the treated group. In MCV and MCH followed similar trend with that of hemoglobin concentration (Hb), MCH, Lymphocyte and the Neutrophil.

In the fourth month, the result of the haematological parameters showed that there were significant ($p < 0.05$) differences in PCV, WBC, MCH, Lymphocyte and Neutrophil. Thus, in PCV, the untreated group had the value of 41.61 ± 0.48 which was significantly higher than the value of 39.64 ± 0.41 obtained from the treated group. In WBC, the treated group had the higher value of 11.98 ± 0.47 which was significantly ($p < 0.05$) higher than the value of 7.97 ± 0.47 obtained from the untreated group. While in Lymphocyte, the treated group had the higher value of 86.50 ± 0.82 which was significantly ($p < 0.05$) different from the value of 81.40 ± 0.24 obtained from the untreated group. In Neutrophil, the treated group had the higher value of 18.0 ± 1.10 which differed ($p < 0.05$) significantly from the value of 15.80 ± 0.71 obtained from the untreated group.

In all, the packed cell volumes (PCV) of the untreated groups were progressively higher than the values obtained from treated groups. A critical look at the tables also revealed that month three had the highest PCV value of 45.05% which was above the rest of the values obtained. Interestingly, this result has shown that lead acetate negatively influenced the values of PCV, Hb and RBC of wistar rats that low dosage.

In reproductive hormones of the wistar rats, only the luteinizing hormone (LH) was significantly affected by the lead acetate. The treated group had the value of 5.89 ± 0.10 mui/l which was significantly ($p < 0.05$) lower than the value of 6.56 ± 0.20 mui/l reported on the untreated group. In testosterone and follicle stimulating hormones, the treated groups had lower numerical values though they were not significant.

IV. DISCUSSION

Body Weight Gain

Reports had it that the body weight of an animal could be altered following its exposure to lead acetate (Ekeh *et al.*, 2015). Thus, its significant changes could be observed depending on the dosage, duration of the exposure and age of the animal (Yara *et al.*, 2008). In line with the above, this experiment has revealed that there were no significant ($p > 0.05$) difference in weight of treated and untreated or control groups of male wistar rats exposed to lead acetate. However, this result is in consonance with the earlier report of Kumar and Asha (2018) who reported similar weight gains in which there were no significant ($p > 0.05$) difference but observed numerical reduction in weight after exposure to lead acetate as the experiment progressed. More so, this work is in total agreement with the earlier work of Haouas *et al.* (2015) who reported that there were no significant ($p > 0.05$) difference in body weights of wistar rats exposed to lead acetate. Though, there was slight reduction in weight of the treated group over the control group. In contrast with the above report, this work disagrees with the earlier work of Hammond, (1994) who reported significant ($p < 0.05$) reduction in body weight of albino rats treated with a dose of 17.5mg/l of lead acetate for 10 days. Conversely, Handal-Silva *et al.* (2016) opined that lead did not in any way affect the body weight of albino rats when exposed to 0.6g/l concentration of lead. This could be attributed to low concentration of lead acetate, as reports had it that at very low concentration, the detrimental effects of might not reflect on the body weight of wistar rats (Kumar and Asha (2018) but could accumulate in some organs of the body such as the kidney, liver, bones and in the blood. This result is in tandem with the above submission emphasizing that at low concentration of lead acetate the body weight of wistar rats remains unaffected while the organs such as Heart Kidney and liver) could be impaired or affected (Muhammad *et al.*, 2011). This was attributed to the fact that lead acetate first accumulates in the bone and other organs (Fei Yu *et al.*, 2008) when ingested, before it manifests in the other parts of the body. Thus, the work of Suradkar *et al.* (2010) corresponds with this work as they reported similar weight changes in which there were no significant ($p > 0.05$) different in body weights of wistar rats exposed to lead acetate at (100, 1000ppm) for 28 days.

Haematological Parameters of Male Albino Rats Exposed to Lead Acetate.

Lead has toxic effects in a wide variety of organs, causing impairments in the nervous, hematopoietic, renal and cardiovascular following ingestion, inhalation or skin absorption (Baranowska-Bosiacka *et al.*, 2012). One of the main effects of lead toxicity in the hematopoietic system involves impairment of the Hb synthesis pathway through disrupted expression of genes encoding thus, leading to reduction in haemoglobin concentration (Piomelli, 2002). The hematopoietic system would be directly affected by lead by restraining the hemoglobin (Hb) synthesis through prevention of several key enzymes, which are parts of the pathway of heme synthesis. Yet, it decreases the circulating erythrocytes life span via raising the cell membranes fragility. Hence, anemia would be the result of the joint outcome of these two processes (Baranowska-Bosiacka *et al.*, 2012).

Thus, the result from this work shows that PCV, RBC and WBC were significantly similar at the first month of the experiment but as the experiment progressed there were significant difference in all the parameters in which the untreated group became better than the treated group. In this current study, the PCV value of the treated group (39.84 ± 0.19) decreased significantly when compared with the control group (45.05 ± 0.72). However, the result tallies with the work of Nabil *et al.* (2011) who reported that lead toxicity could affect the

hematopoietic cells resulting in impaired packed cell volume as observed in this present study. The RBC were also affected by the lead acetate as there were significant reduction in RBC values obtained in this study. This is tantamount to the work of Piomelli (2002) who reported that stressor such as lead could decrease the circulating erythrocytes life span via raising the cell membranes fragility thus resulting in reduction in RBC values of the treated group. Erythrocytes are considered as the most vulnerable cells toward oxidative stress from lead as they have very limited reservoirs of antioxidant enzymes to counter the effect of reactive oxygen species (ROS) and however results in decreased RBC. In haemoglobin concentration Hb, the results from the present study corresponds with the work of Baranowska-Bosiacka *et al.* (2012) who reported that lead could restrain the hemoglobin (Hb) synthesis through prevention of several key enzymes, which are parts of the pathway of heme synthesis resulting in decrease in haemoglobin concentration. However, this work is in consonant with the work of Kilikdar *et al.* (2011) who reported that when rats were treated with LAIP for 7 consecutive days; the Hb content of blood significantly decreased by 25% in comparison to the value observed in the control rats. Ibrahim *et al.*, (2012) in his findings reported that lead acetate when given orally to albino rats at a dose of 10 mg/kg body weight (BW) revealed significant ($p < 0.05$) increase in WBC count and decrease in Hb concentration, mean corpuscular Hb concentration, RBC count, and packed cell volume. The above report corresponds with the results obtained from this very study. The reason could be attributed to inability of the animal to replenish antioxidant enzymes because of lack of rough endoplasmic reticulum thus, becoming much prone to the damage by reactive oxygen species (ROS). However, the above resulted in significant ($p < 0.05$) increase in WBC count as it is mobilized to counter the detrimental effects of ROS.

Histology of the Male Wistar Rat Gonadal

The result of histological examination carried out, showed that there were reductions in seminiferous tubules as it shrank when compared with the control group. It is equally reported that the seminiferous tubule of the control group was robust as against the treated group. Thus, lead acetate may have affected the spermatogenic layer in the seminiferous tubules leading to observed emaciation. This finding is in agreement with the work of Ekeh *et al.* (2015) who dosed wistar rats with lead acetate and found out that the testes of the treated wistar rats were altered resulting in organ defects of the wistar rats (Elgawish and Abdelrazek, 2014). More so, Ekeh *et al.*, 2015 also reported that the toxic effects of lead resulted in degeneration of seminiferous tubules as well as thickening the basement membrane. This coincides with the results obtained from this study where the seminiferous tubules tend to shrink resulting in weight loss as well as organ dysfunction. This could be attributed to the facts that lead acetate acts as spermicidal agent especially when administered in high dosage. This work corresponds with the work of Sokol *et al.* (2002) who reported that 200mg/kg of lead acetate administered to wistar rats resulted in alteration in the metabolism of the testis, epididymis and vas deference.

Table 1: Monthly Weights of Male Albino Rats Exposed to Lead Acetate

GROUPS	TREATED	UNTREATED
ONE MAA (g)	157.50 ± 5.6	155.75 ± 8.2
TWO MAA (g)	182.13 ± 7.47	188.06 ± 9.65
THREE MAA (g)	200.25 ± 6.30	206.25 ± 13.44
FOUR MAA (g)	219.06 ± 8.04	221.13 ± 15.58
FIVE MAA (g)	230.83 ± 6.41	233.50 ± 15.49

KEY: MAA : Months After Administration

Haematological Indices of Male Wistar Rats Exposed to Lead Acetate.

Table 2: Month One

Parameters	Treated	Untreated
PCV (%)	40.0 ± 0.14	40.43 ± 0.10
RBC (mm ³)	7.35 ± 0.07	7.27 ± 0.03
WBC (mm ³)	9.60 ^a ± 0.06	8.42 ^b ± 0.04
Hb (g/dl)	13.30 ± 0.04	13.45 ± 0.06
MCV (µm ³)	54.34 ± 0.04	55.66 ± 0.53
MCH	18.09 ± 0.10	18.61 ± 0.13
MCHC (mg/dl)	33.10 ± 0.01	33.44 ± 0.10
L (%)	84.0 ^a ± 1.68	81.40 ^b ± 0.64
N (%)	15.0 ± 1.68	15.80 ± 0.36

Hb = Haemoglobin Concentration; PCV = Packed Cell Volume; RBC = Red Blood Cell; WBC = White Blood Cell; L = Lymphocyte; N =Neutrophil; M = Monocyte; PLT = Blood Platelet; MCV = Mean Corpuscular Volume; MCH = Mean Corpuscular Hemoglobin; MCHC = Mean Corpuscular Hemoglobin Concentration.

**Table 3: Haematological Indices of the Male Wistar Rats Exposed to Lead acetate
Month Two**

Parameters	Treated	Untreated
PCV (%)	35.85 ^b ± 0.01	40.23 ^a ± 0.25
RBC (mm ³)	6.97 ^b ± 0.00	7.42 ^a ± 0.01
WBC (mm ³)	9.32 ± 0.05	8.76 ± 0.19
Hb (g/dl)	11.92 ^b ± 0.01	13.49 ^a ± 0.11
MCV (µm ³)	52.61 ± 1.09	53.33 ± 0.9
MCH	17.10 ± 0.01	17.10 ± 0.01
MCHC (mg/dl)	33.27 ± 0.03	33.27 ± 0.03
L (%)	85.0 ^a ± 1.28	83.20 ^b ± 0.70
N (%)	12.20 ± 0.88	12.80 ± 0.70
M (%)	1.40 ± 0.23	2.20 ± 0.20

Hb = Haemoglobin Concentration; PCV = Packed Cell Volume; RBC = Red Blood Cell; WBC = White Blood Cell; L = lymphocyte; N =Neutrophil; M = Monocyte; PLT = Blood Platelet; MCV = Mean Corpuscular Volume; MCH = Mean Corpuscular Hemoglobin; MCHC = Mean Corpuscular Hemoglobin Concentration.

Haematological Indices of the Male Wistar Rats Exposed to Lead acetate

Table 4: Month Three

Parameters	Treated	Untreated
PCV (%)	39.84 ^b ± 0.19	45.05 ^a ± 0.72
RBC (mm ³)	7.41 ^b ± 0.22	8.01 ^a ± 0.02
WBC (mm ³)	10.0 ^a ± 0.21a	8.12 ^b ± 0.03
Hb (g/dl)	11.22 ^b ± 0.06	14.05 ^a ± 0.24
MCV (µm ³)	52.88 ^b ± 0.14	56.35 ^a ± 1.15
MCH	17.85 ^b ± 0.03	18.78 ^a ± 0.42
MCHC (mg/dl)	33.22 ± 0.21	33.34 ± 0.71
L (%)	85.40 ^a ± 0.90	80.0 ^b ± 1.35
N (%)	18.80 ^a ± 0.35	12.40 ^b ± 1.42
M (%)	0.1.60 ± 0.23	0.60 ± 0.24

Hb = Haemoglobin Concentration; PCV = Packed Cell Volume; RBC = Red Blood Cell; WBC = White Blood Cell; L = lymphocyte; N =Neutrophil; M = Monocyte; PLT = Blood Platelet; MCV = Mean Corpuscular Volume; MCH = Mean Corpuscular Hemoglobin; MCHC = Mean Corpuscular Hemoglobin Concentration.

Haematological Indices of the Male Wistar Rats Exposed to Lead acetate

Table 5: Month Four

Parameters	Treated	Untreated
PCV (%)	39.64 ^b ± 0.41	41.61 ^a ± 0.48
RBC (mm ³)	7.40 ± 0.04	7.63 ± 0.04
WBC (mm ³)	11.98 ^a ± 0.47	7.97 ^b ± 0.47
Hb (g/dl)	13.25 ± 0.13	13.88 ± 0.15
MCV (µm ³)	53.49 ± 0.23	54.55 ± 0.41
MCH	17.74 ± 0.07	18.19 ± 0.19
MCHC (mg/dl)	33.35 ± 0.02	33.42 ± 0.02
L (%)	86.50 ^a ± 0.82	81.40 ^b ± 0.24
N (%)	18.0 ^a ± 1.10	15.80 ^b ± 0.71
M (%)	0.00 ± 0.00	1.0 ± 0.00

Hb = Haemoglobin Concentration; PCV = Packed Cell Volume; RBC = Red Blood Cell; WBC = White Blood Cell; L = lymphocyte; N =Neutrophil; M = Monocyte; PLT = Blood Platelet; MCV = Mean Corpuscular Volume; MCH = Mean Corpuscular Hemoglobin; MCHC = Mean Corpuscular Hemoglobin Concentration.

Table 6: Effects of Lead Acetate on Male Reproductive Hormones of Wistar Rats

Means ± Standard error		
Parameters	Treated	Untreated
Testosterone (ng/ml)	1.21 ± 0.10	1.57 ± 0.24
Leutenizing Hormones (mIU/l)	5.89 ^b ± 0.10	6.56 ^a ± 0.20
FSH (mIU/l)	5.99 ± 0.83	6.42 ± 0.20

FSH: Follicle stimulating hormones.

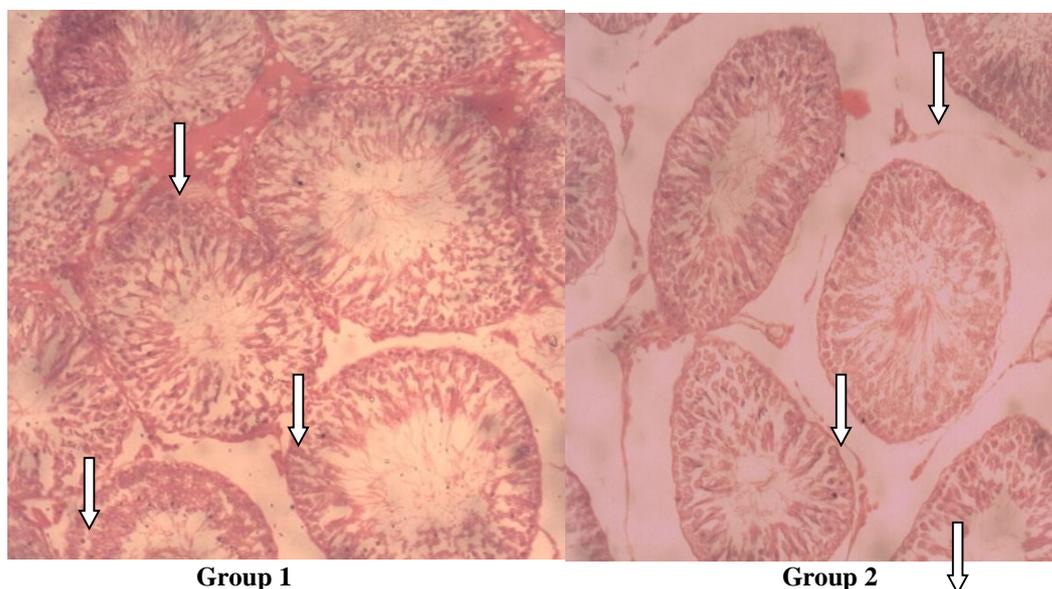
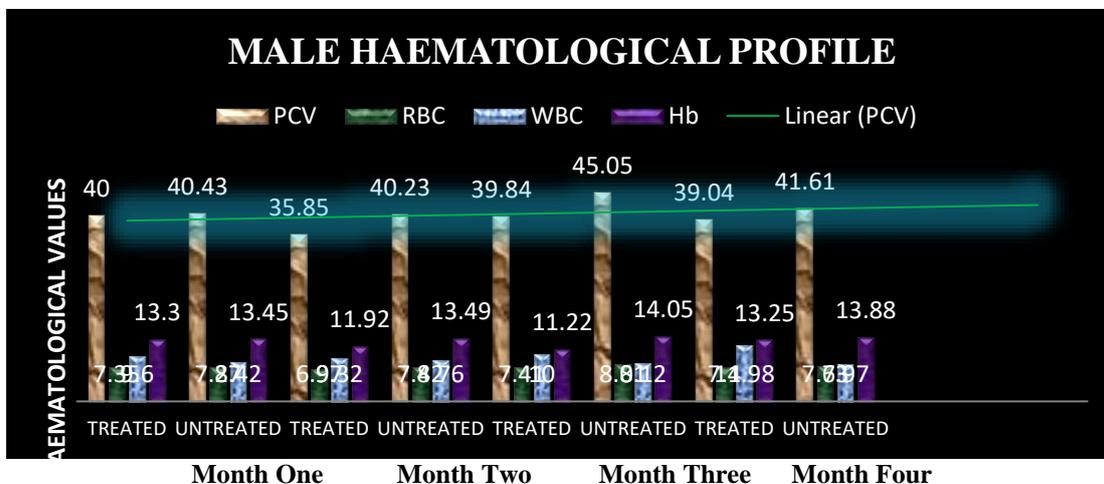


Plate 1: Cross section of the testes showing their intertubular spaces and seminiferous tubules.

(A) Microscopic cross section of the testes from group 1 showing normal intertubular spaces (arrow) showing normal robust seminiferous tubules.

(B) Testicular tissue of group 2 showing wide intertubular spaces (arrow) which is evidence of shrinking of the seminiferous tubules. Hematoxylin and eosin staining (Magnification = ×100).

V. CONCLUSION

It is has been revealed that lead is toxic to health as well as reproductive system of male wistar rats even when exposed at low dosage. Though, at low intake of lead acetate, the toxic effects might not be visible but the long term effect of low intake might be detrimental to health. This is because lead accumulates in the body organs and blood where its effect starts early before reflecting on the body weight of animals. Thus, campaign against lead exposure should be globally intensified.

VI. RECOMMENDATIONS

Strategic research could also be developed for treatment of lead toxicity so as to reduce lead infertility-based cases. Further work should be done to proffer solutions on possible ways the lead acetate associated with our environment could be curbed or ameliorated. Government should as a matter of urgency promulgate laws against environmental lead pollution.

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