

Reproductive fertility, infant mortality and haematological response of female Wistar rats intoxicated with lead acetate

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Abstract

Forty (40) adult female wistar rats were used for this study. They were procured from the Veterinary Teaching Hospital, University of Nigeria Nsukka. The wistar rats were three weeks old and had an average weight of 25g. They were divided into two groups (group A and B) replicated four times with 5 rats per replicate. The treated (group A) were dosed with 60mg/kg while the untreated (group B) were not intoxicated with lead acetate (control). The experiment lasted for four months during which the body weights and the haematological parameters of the rats were recorded on monthly basis. At the end of the experiment, the gonads of the wistar rats were decapitated for histological examination. The data collected were subjected to T-test using SPSS version 21. The probability level of $P < 0.05$ was used to determine the level of significant. The results showed that there were no significant change(s) on the body weights of the female wistar rats. In the haematological indices, the Hb, PCV and RBC had significant ($p < 0.05$) reduction almost below the normal values while the WBC tend to increase significantly above the control group. The histological examination of the ovary showed that group A (untreated group) had two corpora lutea (CL) which is an evidence of two successful ovulations. While the ovarian tissue of group B (treated group) showed one corpus luteum (CL) which is evidence of one successful ovulation and two atretic follicles which is evidence of arrested follicular development. In reproductive performance of the female wistar rats, there were significant ($p < 0.05$) reduction in the values of Progesterone (pg/ml) where the treated group had (10.29 ± 0.92) and untreated group (17.54 ± 3.18) and in Estradiol (ng/ml), the treated group had (357.74 ± 5.30) and untreated group (382.63 ± 11.87). This study therefore, revealed that lead acetate has some detrimental effects on haematology and reproductive physiology of female wistar rats. It calls for the need to intensify actions or proffer solutions on measures to avoid constant exposure to lead acetate in Africa and the world at large in order to avert its negative consequences.

Keywords: Lead acetate; Haematology; Wistar rats; Reproductive fertility; Histology

1. Introduction

The recent hike in infertility cases when compared with the rate in the days of our forefathers could be attributed greatly to environmental pollution, urbanization and industrial emissions which are exceedingly high in our time. We are currently in constant exposure to numerous environmental pollutants either through touch, inhalation or direct consumption. Most times, some of the environmental pollutants come in form of heavy metals such as lead acetate, mercury, nickel and cadmium, etc. These metals are ubiquitous thus, they are found everywhere; in the air, soil and water (WHO, 2019). Daily activities of man result in multiplication of these heavy metals in our environment. More so,

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human exposure to lead compounds such as lead acetate has increased geometrically due to its wide range of applications in industries, cosmetics and medicine (Mayo, 2019). Other major source of lead in the environment are from gasoline used in the cars, industrial emission, lead pipelines used for transporting drinking water, petroleum refining, emission during construction and bullets of gun (Dignam et al., 2019; Stroud and Hunt 2009). It is reported that the manipulation of lead for these uses has led to contamination of air, dust and soil (Kiran et al., 2008). Report has it that constant exposure to these heavy metals could lead to serious reproductive failures resulting in infertility (Kumar, 2018). However, information on impact of environmental pollutants on female reproductive systems and how these affect female reproduction is almost obscured. Adverse effects like decreased spermatogenesis, increased sperm degenerations and testicular dysfunction due to lead have been studied in male wistar rats (Ibrahim et al., 2012) but such studies on reproductive toxicology of female wistar rats have been limited thus, dearth of information on the effects of lead poisoning and environmental pollutants on infertility, premature delivery, and infant mortality in female wistar rats. It is to this backdrop that we decided to investigate the reproductive/haematological implications of lead acetate commonly found in our environment.

The objectives of the study are to;

- Evaluate the effect of lead acetate on gonadal histology of female wistar rats intoxicated with lead acetate.
- Determine the effect of lead acetate on infant mortality rate of wistar rats intoxicated with lead acetate.
- Evaluate the haematological profiles of wistar rats intoxicated with lead acetate.

2. Material and methods

2.1. Histopathology

Specimens from ovaries were collected from all experimental and control groups and fixed in 10% neutral buffered formalin, dehydrated in ascending concentrations of ethyl alcohol (70–100%) and then prepared using standard procedures for Hematoxylin and Eosin stain (Bancroft, *et al.*, 1996).

Progesterone (P₄) levels in plasma samples of different groups of rats were determined using commercially available ELISA kit (Labserv, Fisher Scientific, India).

2.2. 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.

2.3. 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 were prepared using the method as described by Baker *et al.* (2001).

2.4. Total White Blood Cell Count (WBC)

The method of WBC 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. The acid destroys the cell envelope so that the red cells are not seen on count. The acid also makes the white cells more prominent; the dye colours the nuclei. The contents of the pipette were mixed thoroughly by rocking on the palm. Four drop of mixture was blown out to eliminate the diluting fluid in the capillary stem of the pipette. Thereafter, Neubauer chamber was charged accordingly. 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 each 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

2.5. 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 Schalm, *et al.* (1975). 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 micro hematocrit reader and the result will be expressed in percentage as:

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

2.6. Haemoglobin Concentration (Hb)

It was determined using the method described by Drabkin and Austin 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 haemoglobin cyanide. 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:

$$\text{Hb in g/dl} = \frac{\text{Absorbance of test sample}}{\text{Absorbance of standard}} \times \frac{\text{Dilution factor}}{100}$$

2.7. 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:

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

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

$$\text{DF} = \text{Dilution factor} = 1: 200$$

$$\text{Volume counted} = 0.02\text{mm}^3$$

2.8. 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

2.9. 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 *et al.* (2001). The formula is given below;

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

2.10. 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 *et al.* (2001). The formula is given as:

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

2.11. 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, *et al.* (2001). The formula is therefore given as:

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

Experimental design: The data collected from the treated and untreated wistar rats were compared using T-test with two treatment groups, replicated five times with four wistar rats per replicate. The test statistical probability level of $P < 0.05$ was used to determine the level of significance.

3. Results

The Histological sections of the ovary of wistar rats intoxicated with lead acetate

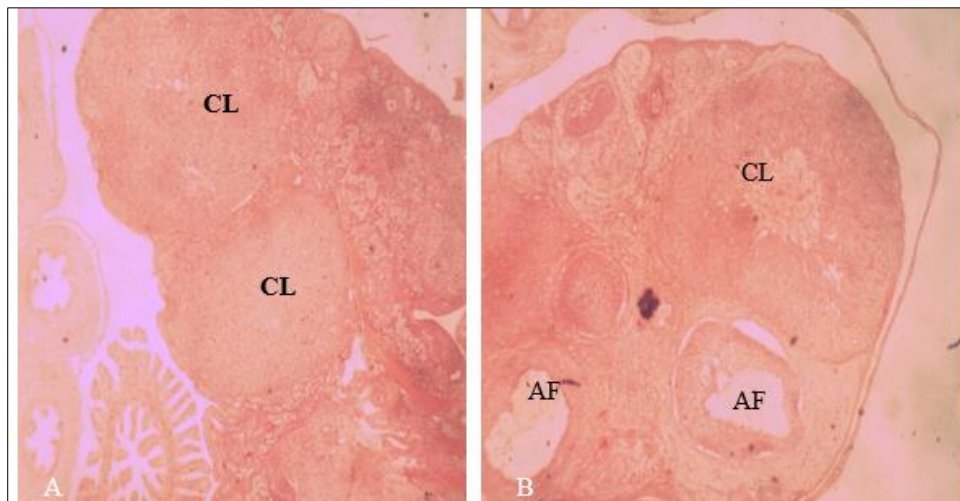


Figure 2 A) Microscopic cross section of the ovary from group 1 showing two corpora lutea (CL) which is evidence of two successful ovulation. B) Ovarian tissue of group 2 showing one corpus luteum (CL) which is evidence of one successful ovulation and two atretic follicles (AF) which is evidence of arrested follicular development. Hematoxylin and eosin staining (Magnification = $\times 100$)

Table 1 Effect of Lead Acetate on Female Reproductive Hormones of Wistar Rats

Means \pm Standard error		
Parameters	Treated	Untreated
Progesterone (pg/ml)	10.29 ^b \pm 0.92	17.54 ^a \pm 3.18
Estradiol (ng/ml)	357.74 ^b \pm 5.30	382.63 ^a \pm 11.87
FSH (miu/l)	6.34 \pm 0.01	7.10 \pm 0.84

FSH: Follicle Stimulating Hormones

Table 2 Haematological indices of Female Albino Rats Exposed to Lead Acetate

Month One		
Means ± standard error		
Parameters	Treated	Untreated
PCV (%)	39.41 ± 0.13	39.93 ± 0.02
RBC (mm ³)	7.58 ± 0.08	7.30 ± 0.04
WBC (mm ³)	10.44a ± 0.22	8.60b ± 0.07
Hb (g/dl)	13.12 ± 0.04	13.15 ± 0.07
MCV (µm ³)	51.79 ± 0.28	54.70 ± 0.32
MCH	17.21 ± 0.08	18.02 ± 0.07
MCHC (mg/dl)	33.29 ± 0.14	33.13 ± 0.17
L (%)	83.00a ± 2.24	76.0b ± 0.45
N (%)	15.0 ± 2.24	23.60 ± 0.68
M (%)	1.40 ± 0.24	0.00 ± 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 Haemoglobin; MCHC = Mean Corpuscular Hemoglobin Concentration.

In table 7 above, there were significant difference only on white blood cell (WBC) and lymphocyte (L). The other parameters were not significantly affected by the treatment (lead acetate toxicity). In WBC, the treated group had the value of 10.44 ± 0.22 mm³ which was significantly higher ($p < 0.05$) than the value of 8.60 ± 0.07mm³ obtained from the untreated group. However, the lymphocyte followed similar trend as white blood cell in which the treated group had higher value of 83.0 ± 2.24(%) which was significantly ($p < 0.05$) higher than the value of 76.0 ± 0.45 reported on untreated group.

Table 3 Haematological indices of Female Albino Rats Exposed to Lead Acetate

Month Two		
Means ± Standard error		
Parameters	Treated	Untreated
PCV (%)	36.03 ± 0.15	38.69 ± 0.74
RBC (mm ³)	7.0 ± 0.02	7.29 ± 0.08
WBC (mm ³)	10.90 ^a ± 0.63	8.65 ^b ± 0.07
Hb (g/dl)	12.0 ± 0.05	12.89 ± 0.25
MCV (µm ³)	51.36 ± 0.09	53.01 ± 0.42
MCH	17.14 ± 0.02	17.66 ± 0.14
MCHC (mg/dl)	33.29 ± 0.01	33.32 ± 0.02
L (%)	81.00 ± 0.32	82.20 ± 0.91
N (%)	18.60 ± 0.51	16.40 ± 1.50
M (%)	0.40 ± 0.24	1.00 ± 0.45

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 Haemoglobin; MCHC = Mean Corpuscular Hemoglobin Concentration.

Table 3 above, showed that there were significant different on white blood cell (WBC) only. The other parameters were not significantly affected by the treatment (lead acetate toxicity) at this level of concentration given. However, in WBC the treated group had the higher significant ($p < 0.05$) value of $10.90^a \pm 0.63 \text{ mm}^3$ as against the value of $8.65 \pm 0.07 \text{ mm}^3$ reported on the untreated group.

Table 4 Haematological indices of Female Albino Rats Exposed to Lead Acetate

Month Three		
Means \pm Standard error		
Parameters	Treated	Untreated
PCV (%)	$35.0^b \pm 0.31$	$40.67^a \pm 0.32$
RBC (mm^3)	7.53 ± 0.08	8.51 ± 0.04
WBC (mm^3)	$11.90^a \pm 0.04$	$8.07^b \pm 0.03$
Hb (g/dl)	12.23 ± 0.11	13.54 ± 0.12
MCV (μm^3)	52.99 ± 1.02	53.87 ± 0.27
MCH	17.74 ± 0.26	18.02 ± 0.16
MCHC (mg/dl)	33.38 ± 0.32	33.30 ± 0.02
L (%)	$86.00^a \pm 1.22$	$79.20^b \pm 1.71$
N (%)	$18.00^a \pm 2.44$	$12.60^b \pm 0.97$
M (%)	1.60 ± 0.24	1.40 ± 0.24
B (%)	1.20 ± 0.49	0.00 ± 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 Haemoglobin; MCHC = Mean Corpuscular Hemoglobin Concentration.

In the table 4 above, there were no significant different in all the haematological parameters evaluated except in PCV, WBC, Lymphocyte and Neutrophil. The treated group had the PCV value of $35.0 \pm 0.31\%$ which was significantly ($p < 0.05$) lower than the value of $40.67 \pm 0.32\%$ reported on untreated group. In WBC, the treated group had the higher value of $11.90 \pm 0.04 \text{ mm}^3$ which was significantly different from the value of $8.07 \pm 0.03 \text{ mm}^3$ reported from the untreated group. More so, in the lymphocyte values, the treated group ($86.0 \pm 1.22\%$) was significantly ($p < 0.05$) higher than the untreated group ($79.20 \pm 1.71\%$). While in the report of Neutrophil, the value of $18.00 \pm 2.44\%$ obtained from the treated group was significantly higher than the value of $12.60 \pm 0.97\%$ reported on the untreated group.

Table 5 Haematological indices of Female Albino Rats Exposed to Lead Acetate

Month Four		
Means \pm Standard error		
Parameters	Treated	Untreated
PCV (%)	$37.25^b \pm 0.23$	$39.46^a \pm 0.57$
RBC (mm^3)	7.25 ± 0.02	7.40 ± 0.06
WBC (mm^3)	$12.82^a \pm 0.38$	$9.22^b \pm 0.07$
Hb (g/dl)	12.05 ± 0.07	13.15 ± 0.19
MCV (μm^3)	52.75 ± 0.16	53.66 ± 0.29
MCH	17.42 ± 0.07	17.84 ± 0.13
MCHC (mg/dl)	33.33 ± 0.03	33.33 ± 0.01
L (%)	$81.60^a \pm 2.69$	$74.60^b \pm 2.61$

N (%)	18.60 ^a ± 2.68	14.0 ^b ± 2.44
M (%)	0.00 ± 0.00	1.0 ± 0.00
B (%)	0.4 ± 0.24	0.00 ± 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 Haemoglobin; MCHC = Mean Corpuscular Hemoglobin Concentration

In the table 5 above, there were significant differences in the haematological parameters evaluated, the PCV, WBC, Lymphocyte (L) and Neutrophil (N). The treated group had the PCV value of 37.25 ± 0.23 which was significantly lower than the value of $39.46^a \pm 0.57$ %. Thus, in WBC, the value of 12.82 ± 0.38 obtained was significantly ($P < 0.05$) higher than the value of 9.22 ± 0.07 from the untreated group. While the Lymphocyte value of 81.60 ± 2.69 obtained from the treated group was significantly ($p < 0.05$) higher than the value of 74.60 ± 2.61 reported on the untreated group. Finally, the value of 18.60 ± 2.68 reported on the treated group was significantly ($p < 0.05$) higher than the value of 14.0 ± 2.44 obtained from the untreated group.

Table 6 Effects of lead acetate on infant mortality of wistar rats

Replicates	Group 1 (Treated)	Mortality at 2 weeks after Parturition	Group 2 (Untreated)	Mortality at 2 weeks after Parturition
R1 (5 rats)	22 Liters	12 Liters	20 Liters	0 Liter
R2 (5 rats)	20 Liters	8 Liters	24 Liters	1 Liter
R3 (5 rats)	24 Liters	14 Liters	20 Liters	0 Liter
R4 (5 rats)	18 Liters	11 Liters	22 Liters	0 Liter
Total	84 Liters	45 Liters	86 Liters	1 Liter

Key: R = Replicate.

The table above revealed that the litter size of the wistar rats were not affected by the treatment but there was an observed high rate of post parturition mortality (forty five wistar rats died) in the treated group as against low post parturition mortality in the untreated group (only one wistar rat died).

4. Discussion

4.1. Reproductive Performance of Female Wistar Rats Exposed to Lead Acetate

Reports had it that toxic chemicals such as lead acetate, mercury and cadmium could negatively influence fertility and reproduction in animal. Thus, there is a close correlation between infertility and environmental toxicity associated with heavy metals (Sikka and Wang (2008). The findings from this work is in consonance with the earlier work of Sharma *et al.*, 2012 who reported reduced number of primordial follicle and increased number of atretic follicles in the ovaries of mice which is an evidence of arrested follicular development in wistar rats.

In the present study, were wistar rats were dosed with 60mg/kg body weight witnessed significant ($p < 0.05$) reduction in progesterone and follicle stimulating hormones of female hormones. This work is in total agreement with the work of Mokhtari and Zanoori (2011) who reported significant ($p < 0.05$) decrease in hormonal level of wistar rats exposed to 50 and 100mg/kg body weight of lead acetate when compared with the control group. More so, Hamed *et al.* (2012) reported significant reduction in the values of luteinizing hormones and follicle stimulating hormones of wistar rats intoxicated with lead acetate at 50mg/kg body weight. Consequently, report from this present study tallies with the work of Dearth *et al.*, 2002 who observed that prepubertal female rats exposed to lead exhibited reduced circulating levels of estradiol-17 β . Similarly, Franks *et al.*, 1989 reported decreased plasma progesterone concentration in wistar rats exposed to lead. These works are in tandem with the findings from this present work where the treated group had lower progesterone and estradiol concentrations than the untreated group. It is possible that lead acetate may have negatively influenced the normal functioning of steroidogenic enzyme resulting in lower progesterone and estradiol levels.

Haematological Profiles of Female Rats Exposed to Lead Acetate

The effects of Lead acetate on female haematological indices followed similar trend with that of their male counterparts. The results showed that there were slight reductions in the value of RBC, Hb and PCV of the treated groups against the control group. However, Piomelli (2002) reported that one of the main effects of lead acetate in the hematopoietic system is the reduction in Hb synthesis pathway through disrupted expression of genes encoding. Thus, 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 thus, resulting in anaemia. (Baranowska-Bosiacka *et al.*, 2012).

The results from this work showed that the female PCV, RBC and WBC were significantly similar at the first month of the experiment but as the experiment progressed there were significant ($p < 0.05$) difference in some of the parameters in which the untreated or control group became better than the treated group. In this current study, the female PCV value of the treated group decreased significantly (Treated: 36.03 ± 0.15) and (Control: 38.69 ± 0.74) when compared with the control group. 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 current study. In the third month of the experiment, the PCV followed the same trend in which the treated group had the significant higher value of (Treated: 35.00 ± 0.31) and (Control: 40.67 ± 0.32). The RBC, though not significantly affected but the treated group had reduced numerical values from the first month to the fourth month of administration over the control group. This work is in consonance with 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 on 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 a similar vein, the treated group had lower haemoglobin concentration (Hb) all through the period the experiment in both male and female wistar rats. However, 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 tantamount with the work of Kilikdar *et al.* (2013) who reported that when rats were treated with LA IP for 7 consecutive days; the Hb content of blood significantly decreased by 25% in comparison to the value observed in the control rats. For the results of WBC, there was significant ($p < 0.05$) increase in the value of treated groups as against the untreated groups from the first month after administration to the fourth month. It could be that the system was secreting more WBC to help counter the effects of the invading antibody (lead acetate). This findings tallies with the earlier work of Ibrahim *et al.*, (2012) who also 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 also 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).

5. Conclusion

- The study revealed that lead acetate has a significant negative impact on the hematological profiles of rats, leading to reduced PCV, Hb, and RBC values.
- Histological examinations showed that treated rats had one corpus luteum (CL), indicating a single successful ovulation, and two atretic follicles (AF), indicating halted follicular development, while untreated rats had two corpora lutea (CL) indicating two successful ovulations.
- The treated wistar rats exhibited significant decrease in Progesterone and Estradiol levels compared to the untreated group
- High infant mortality was equally observed in the treated group within two weeks after parturition, unlike the untreated group.
- These findings underscore the importance of taking immediate measures to reduce exposure to lead acetate in order to prevent its adverse consequences.

Recommendation

I recommend that similar work be done using other heavy metals such as mercury, cadmium, nickel etc. to find out their effects on health and fertility of wistar rats and to proffer possible remedial solutions to ever-increasing heavy metals in our environment.

Compliance with ethical standards

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Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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