# Heavy Metal Induced Alterations in Haematological Parameters of Rat

Anjali

Visiting Faculty, Dr. G L Gupta Institute of Public Health, Lucknow University, New Campus, Lucknow

#### ABSTRACT

Oral exposure of lead acetate  $(100\mu g \text{ and } 250\mu g / \text{kg} \text{ body weight})$  to rats for 30 and 90 days produced anaemia as evidenced by decrease in erythrocyte count, haemoglobin concentration and haematocrit values. However, exposure to lead resulted in elevation of white blood cell count. The increase in the number of neutrophils and lymphocytes may be a form of defensive process against the toxic effect of lead toxicity. The study suggests that the concentration of lead, which is highly toxic should be carefully controlled.

*Key Words:* Chronic exposure, Lead acetate, Haematological parameters

### **INTRODUCTION**

It is very well known that heavy metals have accumulated in all the parts of our environment (Purves, 1985, Nriagu, 1990 and Flora, 2000). When mammals are exposed to these metals, they usually cause an array of behavioral, haematological and pathological changes in the vital tissues of their body. Of all the changes produced by heavy metals, alterations in the haematological parameters are considered very important as the blood cells are responsible for immune reaction of the body. Immunomodulation by toxic metals has been studied by a number of workers (Mc Cabe 1994, Lawrence and McCabe 1995). Several studies on the effect of heavy metals due to consumption of contaminated vegetables from waste water irrigated area in recent times have also been made (Marques et al 2006, Singh et al 2010 and Chauhan et al 2014). The present study highlights heavy metal the induced alterations in haematological parameters of

rat caused by chronic exposure to lead acetate.

#### **MATERIALS AND METHODS**

For the present immunotoxicity testing, Wistar rats weighing 180-200gm were used as experimental animals. Food and water are provided ad libitum. Upon arrivals to the laboratory, the animals were acclimatized for one week and then distributed at random in the experimental and control groups. The animals were housed in a solid bottom polyethylene cages  $48 \times 27 \times 20$  cm with a stainless steel slotted top and bedding of fine shavings. There was a 12hr light-dark cycle, temperature 22.2 to 23.3<sup>o</sup> C and humidity 45-55%. The rats were inspected daily.

Stock solution of analytical grade lead acetate, obtained from BDH England was prepared by dissolving 1gm inorganic salt in 11itre double distilled water. From the stock solution, measured quantity of toxicant was added separately in the drinking water of each rat. Rats were divided into the following groups of 10 rats in each group and exposure was given daily.

Group 1 : 100µg lead acetate/kg body weight for 30 days after post partum.

Group 2 : 250µg lead acetate/kg body weight for 30 days after post partum.

Group 3 : 100µg lead acetate/kg body weight for 90 days after post partum.

Group 4 : 250µg lead acetate/kg body weight for 90 days after post partum.

Group 5 : 100µg lead acetate/kg body weight for respective days after pp.

Group 6 : 250µg lead acetate/kg body weight for respective days after pp. Group 5 and 6 were considered as control. After completion of exposure the experimental and control rats were processed simultaneously.

For collecting blood samples, 1.0ml of blood drawn from each animal and poured into a K-EDTA tube and mixed for 10 minutes. The blood samples were stored to determine haematological parameters (Wintrobe 1981).All the blood samples were coded to preserve anonymity. Using a blood cell counter, differential white blood cell count was determined. Statistics was applied as when necessary for proper interpretation of the results.

## **RESULTS AND DISCUSSION**

The results of chronic exposure to lead for 30 and 90 days on haematological parameters in experimental animals are summarized in tables 1 and 2.

Parameters	Period of Exposure	Control	Dietary concentration of Lead Acetate	
	(Days)		100µg/kg b.w.	250µg/kg b.w.
RBC(x 10 <sup>6</sup> /µl)	30	06.57	06.50	03.90*
	90	06.89	05.00	02.55**
Hb (g/dl)	30	14.20	14.00	08.70*
	90	13.50	12.00	07.34**
Hct (%)	30	42.80	42.60	30.60*
	90	43.00	40.00	28.10**
MCH	30	21.60	21.50	22.30
	90	19.59	24.00	28.80*
MCHC (%)	30	33.20	32.90	28.43*
	90	31.40	29.90	26.12**
MCV (µm <sup>3</sup> )	30	65.10	65.10	78.50
	90	62.40	80.00*	110.20**
WBC	30	06.90	08.50	11.80*
(x 10 <sup>6</sup> /µl)	90	07.00	10.60*	15.30**

TABLE-1: HAEMATOLOGY PARAMETERS OF RATS TREATED WITH LEAD ACETATE

Values are means of 10 animals /group (\*P < 0.05; \*\*P < 0.01)

TABLE-	2 DIFFERENT	TAL LEUCOCYTE (	COUNTS (	OF RATS TREATED	WITH LEAD ACETATE
	-		~ .		

Parameters	Period of Exposure	Control	Dietary concentration of Lead Acetate	
	(Days)		100µg/kg b.w.	250µg/kg b.w.
Neutrophil (x 10 <sup>3</sup> /µl)	30	1.3	1.7	2.4*
	90	1.0	2.5	5.7**
Lymphocyte (x 10 <sup>3</sup> /µl)	30	5.5	6.5	8.7*
	90	5.9	7.9*	9.0**
Eosinophil x 10 <sup>3</sup> /µl)	30	0.0	0.2	0.4
	90	0.0	0.1	0.2
Monocyte $(x \ 10^3/\mu l)$	30	0.1	0.1	0.3
	90	0.1	0.1	0.4**

Values are means of 10 animals /group (\*P < 0.05; \*\*P < 0.01)

It was observed that hematopoietic system is extremely sensitive to the toxicity of lead. Therefore, small dose could cause variation in the normal haematological parameters as was evidenced in 30 days exposure to 100µg/kg body weight lead. Red blood cell count and hemoglobin percentage were decreased and haematocrit value showed an increase after exposure to 100µg lead/kg body weight for 90 days blood while white cell count and lymphocytes showed an elevation in number. A 250µg/kg body weight dose of lead given daily for 30 and 90 days produced significant decline in the red blood haemoglobin cell count. percentage,

haematocrit and mean cell haemoglobin concentration. However, an increase in neutrophils and white blood cell count were observed. Monocytes number in high dose group was nearly fourfold higher than controls.

General concepts of metal toxicity are developed utilizing data from all the mammals so that result can be extrapolated for man, who is quite different but not entirely separate from other mammals. This general understanding of toxicity provided enlightened consideration of the probable activity of any metal in any given species under a probable set of conditions. Generalization from laboratory animal data is therefore important for other mammals on which there is insufficient information. A comprehensive approach followed in immunotoxicology is induction of toxicity experiments in rodents, as the approach aims to imitate the alteration in man and is feasible for assessing the toxic effect of heavy metals on the immune system.

In this study, oral exposure of lead to 100µg and 250µg/kg body weight produced anaemia as was evidenced by decrease in erythrocyte count. haemoglobin concentration and haematocrit values, while mean cell hemoglobin and mean cell volume showed a slight increase. This deviation could become more and more intense with increasing dose or time interval. Lead seems to have lytic effect like the salts of other metals such as copper and mercury. Metals by binding with red cell membrane alter functional properties of the membrane and intra erythrocyte functions, which may consequently diminish red cell activity, resulting in decreased erythrocyte count. These results confirm the findings of earlier workers in the field (Lia et al 1995, Karmakar et al 1984, Marques et al 2006 and Corsetti et al 2017).

Lead may inhibit haem synthesis in erythropoietic tissues by impairing the incorporation of iron into protoporphyrin in the final step of heam biosynthesis process. These changes might be due to inhibition of haem biosynthesis by lead exposed animals as also reported by Hoffman *et al* 1985 and Horiguchi *et al* 1992.

Exposure to lead also resulted into the elevation in white blood cell count. The number of neutrophils and lymphocytes was markedly increased while the monocyte number showed nearly four- fold increase in high dose group of lead than the controls. The increases in the number of lymphocytes in this study may be a form of defensive process against the toxic element or in response to the necrotic degeneration. The study corroborates the results obtained by Koller and Kovacic 1974, Kundiev 2001, Jadhav 2007 and Corsetti 2017. In brief the study suggests that lead is an important immunotoxin and therefore the concentration of lead in our environment should be carefully controlled. This is recently confirmed by the studies of Chauhan *et al* 2014 in waste water irrigated area of Rewa, India. There is little doubt that haematologicals parameters are seriously affected due to lead accumulation in the body.

## REFERENCES

- Chauhan G and Chauhan UK (2014). Human health risk assessment of heavy metals via dietary intake of vegetables grown in waste water irrigated area of Rewa, India. *Int. J. Of Scientific Res.and Pub.* Vol.4 no.9, pp.1-9.
- Corsetti G, Romano C, Stacchiotti A, Pasini (2017). ER stress and apoptosis trigerred by sub-chronic lead exposure in mice spleen: a histopathological study. *Bio. Trac. Elem. Res.* 25(8):327
- Hoffman DJ ,Heinz GH , Sileo L, Audet DJ,Campbell JK and LeCaptain LJ(2000). *Arch. Environ. Contam. Toxicol.*39 (2):221.
- Horiguchi S, Endo G, Kiyoto I, Teramoto K, Shiragawa K, Waritani F, Konishi Y, Kiyota A, Ota A, Tanaka H and Fukeei M (1992). Serum immunoglobin and complement C3 levels in workers exposed to lead. Osaka City Med. J.38:149-53.
- Jadhav SH, Sarkar SN, Ram GC and Tripahi HC (2007). Immunosuppressive effect of sub-chronic exposure to a mixture of heavy metals, found as ground water contaminants in different areas of India, through drinking water in male rats. *Arch. Environ. Contam. Toxicol.* 53:450-8
- Karmakar N, Saxena R and Anand S (1984). Histopathological changes induced inrat tissues by oral intake of lead acetate . *Environ. Res.* 41(1)23-28
- Koller LD and Kovacic S(1974). Decreased antibody formation in mice exposed to lead. Nature (London) 250:148-150
- Kundiev I(2001). Dependence of immune and biochemical homeostasis mechanisms on lead accumulation in the body (experimental study). Med Jr. Prom. Ehol. 5:11-17
- Lawrence DA and Mc Cabe MA Jr.(1995). Immunomodulation by toxic metals. In:

Metal Toxicology (Eds. RA Goyer, CD Klassen and MP Waalkes Jr.) Academic Press NY pp305-307

- Lia O, Tsai JW and Wang SC(1995). Subacute inhalation toxicity of lead acetate in rats. J. Chinese Soc.Vet. Sci. 21(5);296-307
- Marques M (2006). An assessment of time dependent effects of lead exposure in algerian mice using different methodological approaches. *Biol. Trace Elements Res.* 109(2):75-89
- Mc Cabe MA Jr.(1994). Mechanisms and consequences of immunomodulation bylead. In: Immunotoxicology and Immunopharmacology, 2<sup>nd</sup> ed. Raven Press, NY pp 143-163

- Nriagu JO (1990). Global metal pollution. *Env.32*:7-33
- Singh A, Sharma RK, Agarwal M and Marshall FM (2010). Risk assessment of hevy metal toxicity through contaminated vegetables from waste water irrigated area of Varanasi, India. *Tropical Ecology* vol.51 (2), pp375-387.
- Wintrobe MM (1981). Principles of hematologic examination. In: Wintrobe Ed. Clinical haematology, 8<sup>th</sup> Ed. Philadelphia: Lea and Febiger, 7-91.

How to cite this article: Anjali. Heavy metal induced alterations in haematological parameters of rat. Galore International Journal of Applied Sciences & Humanities. 2018; 2(3): 1-4.

\*\*\*\*\*