Research Paper

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Dose Dependent Impact of Lead Chromate on Selected Haematological and Biochemical Parameters of Swiss Albino Mice

Zoology

KEYWORDS : Haematological parameters, bio-chemical parameters, Urea, Total Protein, Albumin.

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ABSTRACT

After application of Lead chromate (75 mg/kg/bw/day, 100 mg/kg/bw/day and 150 mg/kg/bw/day) on Swiss albino mice, haematological and biochemical parameters were estimated at a time interval of 15 days, 1 m, 2m and 3m. At all doses, Total Erythrocyte Count, Haemoglobin content, Total Protein and Albumin levels decreased while Urea level was increased.

Introduction

Lead chromate (PB.CR-04;Mol. Wt.323.19) is a powder , yellow to orange in colour. It is water-insoluble. It is a suspected human carcinogen of the lung, and can cause chronic lead poisoning (Moore and Meredith,1979; Kazantzis, 1989; Goyer,1992). Lead chromate potentially could pose a double hazard and cause signs and symptoms of chronic lead Intoxication- severe gastrointestinal disturbances, anaemia (Christenson, 1977, Johansson-Sjobech and Larsson, 1979, Hoffman *et al*., 1985, Graziano *et al*., 1991), neuromuscular dysfunction, nephritis (Cramer *et al*., 1974, Goyer et al., 1989, Cardenas *et al*., 1993) and encephalopathy and chromium VI toxicity- sensitization dermatitis, primary irritant dermatitis, ulcerated nasal mucosa and skin and nephropathy. Lead chromate and derived pigments have been tested in rats by sub-cutaneous and intra muscular injection, producing malignant tumors at the site of injection and in one study renal carcinomas (Choie and Richter, 1972). Lead has been found to cross the placenta and cause miscarriage, stillbirths and birth defects. Exposure before birth can cause mental retardation, behavioural disorders and infant death. Lead can also cause reduce sex drive, impotence, sterility and damage the sperm (Cullen *et al*.,1984, Lerda, 1992), increasing the potential for birth defects. Periods in women can also be affected.

While reviewing literature, it was found that the information concerning the oral exposure of experimental animal to Lead chromate is very limited, specifically on the haematological and biochemical parameters,

The present work was undertaken to study the effects of Lead chromate induced haematological and biochemical parameters.

Materials and Methods

Among haematological parameters, Red Blood Cells were counted with standardized Neubauer haemocytometer and Haemoglobin was determined by acid-haematin method (Schalm *et al*,1975). Among biochemical parameters, Urea was determined by Diacetylmonoxime (DAM) method using Urea kit (Wybenga,1971), Total Protein by Biuret method using Total Protein kit (Doumas,1975) and Albumin by BCG method using Albumin kit (Doumas and Watson, 1971).

Results and Discussion

Oral LC- induced changes in the Haematological parameters: -

Table- (1) shows that after exposure TEC declined significantly. Upon 75 mg/kg bw treatment, there was a loss of 0.13%, 1.21%, 7.13% and 9.14%; upon 100 mg treatment **[Table-(2)]** there was 0.98%, 3.51%, 8.2% and 14.52% loss; upon 150 mg treatment **[Table-(3)]** there was 0.74%, 4.87%, 11.39% and 17.07% loss after 15 days, 1m, 2m and 3m respectively.

It is evident from **Table- (1)** that upon 75 mg/kg bw treatment, there was a loss of 0.82%, 4.87%, 11.39% and 17.07% in Hb content after each test period. Upon 100 mg treatment **[Table- (2)]** there was 1.04%, 4.88%, 13.42% and 20.64% loss; upon 150 mg treatment **[Table- (3)]** there was 2.46%, 8.95%, 13.83% and 22.23% loss at the end of 15 days, 1m, 2m and 3m respectively.

Oral LC – induced changes in the Bio-chemical parameters of Blood: -

Tables shows that after an exposure to LC, Urea level elevated significantly. There was a gain of 0.38%, 0.76%, 1.90% and 1.49% upon 75 mg/kg bw treatment **[Table- (1)];** gain of 0.38%, 2.69%, 2.66% and 2.62% upon 100 mg treatment **[Table- (2)]** ; gain of 0.76%, 3.46%, 3.04% and 3.37% upon 150 mg treatment **[Table- (3)]** at the end of 15 days, 1m, 2m and 3m respectively.

Tables shows that there was significant decline in the values of TP at the end of each test period. Upon 75 mg treatment **[Table-(1)]** there was 4.17%, 4.38%, 5.46 % and 8.28% loss; upon 100 mg treatment **[Table- (2)]** there was 6.02%, 6.41%, 7.58% and 9.38 % loss; and upon 150 mg /kg bw treatment**[Table- (3)]** there was 5.33%, 5.60%, 8.09 % and 10.07% loss at the end of 15 days, 1m, 2m and 3m respectively.

Tables shows that Albumin levels were significantly depressed in the treated animals. Upon 75 mg/kg bw treatment **[Table-(1)]** there was 0.35%, 1.37%, 6.27% and 10.15% loss; upon 100 mg/kg bw treatment [**Table- (2)]** there was 0.35%, 1.88%, 6.92% and 11.12% loss; upon 150 mg treatment **[Table- (3)]** there was 5.52%, 2.05%, 7.75% and 12.89% loss at the end of 15 days, 1m, 2m and 3m respectively.

In all species of experimental animals studied, including non-human primates, lead has been shown to cause adverse effects in several organs and organ systems, including the haemopoietic, nervous, renal, cardiovascular reproductive & immune systems. Lead also affects bone and has been shown to be carcinogenic in rats and mice.

Lead has been shown to have effects on many biochemical processes; in particular, effects on haem synthesis have been studied extensively in both adults and children. It affects haemopoietic system at several levels. These include effects on haem & globin synthesis and on erythrocyte formation and function. Lead acts on steps in the synthetic pathway both inside and outside the mitochondrion. It inhibits certain enzymes –ALA dehydratase, ferrochelatase, coproporphyrinogen oxidase. It decreases erythrocyte survival through its inhibition of membrane bound Na-K-ATPase (Rhagavan *et al*, 1981).

Acute expose to lead is known to cause proximal renal tubular damage, characterized by generalized aminoaciduria, hypophosphataemia with relative hyperphosphaturia and glycosuria . Cellular structure changes include nuclear inclusion bodies, mitochondrial changes & cytomegaly of the proximal tubular epithelial cells (Cramer *et al*, 1994).

Lead chromate toxicity elevates urea level, may be due to renal dysfunctioning (Bernard and Becker, 1988). There was a decrease in liver protein content after treatment because Lead chromate inhibits haem oxygenase activity thereby increasing the degradation of haemprotein. This may adversely affect a

number of functions such as respiration and energy production (Maines, 1992).

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Table 1: Changes in the haematological and bio-chemical parameters due to Lead chromate toxicity (75 mg/kg/bw/day)

Parameters Duration		15 days	1m	2m	3m
TEC	Control	8.21±0.2	8.28±0.2	8.42±0.6	8.54±1.0
(10^6/mm^3)	Treated	8.20±0.4*	8.18±0.5	7.82±0.6*	7.76±0.8*
Hb	Control	12.2±1.0	12.2±1.0	12.3±1.4	12.6±1.8
(gm/100ml)	Treated	12.1±0.1*	12.9±0.5*	10.9±1.2*	10.5±1.2
Urea	Control	26.0±0.07	26.3 ±1.3	26.3±0.4	26.7 ±0.5
(gm/100ml)	Treated	26.1±0.04*	26.2±0.2*	26.8±0.8*	27.1±0.9*
TotalProtein	Control	9.823±1.3	9.83±1.2	9.9±1.7	10.03±1.3
(gm/100ml)	Treated	9.414±0.8*	9.4±0.4*	9.36±0.4*	9.2±0.8*
Albumin	Control	5.82±0.05	5.88±0.1	6.07±0.05	6.21±0.8
(gm/100ml)	Treated	5.80±0.03*	5.80±0.3*	5,69±0.8*	5.58±0.05*

*** P<0.05;** P<0.01**

Table 2: Changes in the haematological and bio-chemical parameters due to Lead chromate toxicity (100 mg/kg/bw/day)

Parameters Duration		15 days	1m	2m	3m
TEC	Control	8.21±0.2	8.28±0.2	8.42±0.6	8.54±1.0
(10^6/mm^3)	Treated	8.13±0.6*	7.99±0.4*	7.73±0.5*	7.30±0.8**
Hb	Control	12.2±1.0	12.2±1.0	12.3±1.4	12.6±1.8
(gm/100ml)	Treated	12.0±1.0*	11.7±1.3*	10.5±1.3**	10.0±1.7**
Urea	Control	26.0±0.07	26.3 ±1.3	26.3±0.4	26.7 ±0.5
(gm/100ml)	Treated	26.1±0.04*	26.7±0.8*	27.0±0.7*	27.4±0.8*
TotalProtein	Control	9.823±1.3	9.83±1.2	9.9±1.7	10.03±1.3
(gm/100ml)	Treated	9.232±0.5*	9.2±1.2*	9.15±1.0*	9.09±0.9*
Albumin	Control	5.82±0.05	5.88±0.1	6.07±0.05	6.21±0.8
(gm/100ml)	Treated	5.80±0.03*	5.77±0.08*	5.65±0.03*	6.21±0.8

*** P<0.05;** P<0.01**

Table 3: Changes in the haematological and bio-chemical parameters due to Lead chromate toxicity (150 mg/kg/bw/day)

Parameters Duration		15 days	1m	2m	3m
TEC	Control	8.21±0.2	8.28±0.2	8.42±0.6	8.54±1.0
(10^6/mm^3)	Treated	8.15±0.5*	7.92±0.4*	7.71±0.8*	7.16±1.2*
Hb	Control	12.2±1.0	12.2±1.0	12.3±1.4	12.6±1.8
(gm/100ml)	Treated	11.9±0.8*	7.92±0.4*	7.71±0.8*	7.16±1.2**
Urea	Control	26.0±0.07	26.3 ±1.3	26.3±0.4	26.7 ±0.5
(gm/100ml)	Treated	26.2±0.8*	11.2±1.8*	10.9±1.8*	9.8±1.9**
TotalProtein	Control	9.823±1.3	9.83±1.2	9.9±1.7	10.03±1.3
(gm/100ml)	Treated	9.3±0.4*	9.28±0.5*	9.1±1.2*	9.02±0.8*
Albumin	Control	5.82±0.05	5.88±0.1	6.07±0.05	6.21±0.8
(gm/100ml)	Treated	5.79±0.01*	5.76±0.08**	5.6±0.03	5.4±0.8*

*** P<0.05;** P<0.01**

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