



ISSN: 0976-3031

Available Online at <http://www.recentscientific.com>

International Journal of Recent Scientific Research
Vol. 6, Issue, 5, pp.3999-4004, May, 2015

**International Journal
of Recent Scientific
Research**

RESEARCH ARTICLE

HAEMATO-BIOCHEMICAL CHANGES INDUCED BY LEAD INTOXICATION IN MALE AND FEMALE ALBINO MICE

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ARTICLE INFO

Article History:

Received 2nd, April, 2015
Received in revised form 10th,
April, 2015
Accepted 4th, May, 2015
Published online 28th,
May, 2015

Key words:

lead, toxicity, haematology,
biochemical, mice

ABSTRACT

Lead is one of the main environmental contaminants which can threaten the living organisms in many ways. Lead toxicity may affect multiple organs of human body and is associated with a number of physiological, biochemical, and morphological alterations. The study aimed to investigate the risk which may result from exposure to different doses of lead acetate on body and organs weight, haematological indices, and the function of liver and kidney. The experiment was performed on 80 mice aged 3 month. They maintained on standard healthy laboratory conditions and had free access to food and drinking water *ad libitum*. The animals were divided into four equal groups. The first group represented the healthy control animals, while group II, III, and IV were given sub-lethal doses of lead acetate (0.4, 0.8, and 1.2 mg/kg bd. wt.) in drinking water for 12 weeks. At the end of the experimental period, blood was collected and used for haematological and biochemical analysis. The results indicated that mice treated with lead acetate showed significant reduction in TEC, PCV, Hb, MCV, MCH, and MCHC compared to the healthy control ones. While there were significant elevation in TLC and platelets. The results also showed significant increase in the activities of ALT, AST, GGT, AKP, and LDH indicating liver dysfunction. In addition, the serum level of BUN and creatinine was also increased compared to the control indicating renal deficiency.

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INTRODUCTION

Lead is a widespread natural element in the environment. It is considered as one of the main persistent and common environmental pollutants (Joworaski, 1968). Lead is used in production of various manufactured products such as paints, printing, gasoline, batteries, water pipes, cosmetic products, pottery glazing, tank linings, brass faucets, toys, and many others (Harbison, 1998). Due to its toxic cumulative action in the environment, lead can affect all biological system via exposure from different sources including air, water, and food. Lead can translocate through the food chain and cause harmful effects to human and other living organisms. It is one of the poisonous metals in the environment and has deleterious impact to most organs of the human body (Duruibe *et al.*, 2007). Lead enters into the body through three main routes including digestive and respiratory tracts, and skin. When it is absorbed into the blood, some of it is bound to erythrocytes and the remaining stay in plasma to be distributed to other tissues (Howard *et al.*, 1973).

There are many evidences which report that lead is a poisonous factor which targets numerous organs such as kidneys, liver, nervous system, immune system, and haematopoietic system. Lead toxicity is associated with a number of physiological, morphological, and biochemical alterations such as liver

dysfunction (Atsdr, 1993; Elayat and Bakheetf, 2010), haematological disorders (Mugahi *et al.*, 2007), impairment of renal system functions (Suradkar *et al.*, 2009), glucose metabolism abnormality (Ahrens, 1993; Yokoyama *et al.*, 2000), and nervous system disturbances (Pitot, 1996).

Accumulation of lead in the body could lead to destructive impacts in haematic, gastrointestinal, and renal system (Correia *et al.*, 2000). Lead toxicity has been associated with multiple forms of cancer, cardiovascular disorders, nephrotoxicity, and distraction of nervous system. Lead poisoning is related to sex, age, exposure duration, exposure route, absorption rate, frequency of intake, solubility, and retention percentage (Pitot, 1996). Exposure to excessive amount of lead has been shown to elevate blood pressure and cardiovascular disorders in adults, and to decrease the cognitive development and intellectual performance in children (CEC, 2002).

Exposure to lead has been shown to increase production of reactive oxygen species (ROS) and consequently induce lipid peroxidation and alteration of antioxidant defense systems in mice (Demirezen and Kadiriye, 2006) resulting in oxidative stress (Xienia *et al.*, 2000). ROS are the by products of numerous degenerative reactions in various tissues, which affect the regular metabolism by damaging the cellular components (Foyer and Noctor, 2002). Decreasing the

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possibility of lead interacting with critical bio-molecules and stimulating oxidative damage, or bolstering the cell's antioxidant defense might be attributed to beneficial role of antioxidant nutrients through exogenous supplementation of antioxidant molecules (Marija *et al.*, 2004). Binding of lead to phosphatidylcholine in the cell membrane of red blood cells, lead to reduction of phospholipid levels. Lipid peroxidation has also been determined in tissue from different parts of the brain of lead-intoxicated rats. Lead exposure may cause hypochromic and normochromic anemia which results from reactive oxygen species production and subsequent erythrocyte hemolysis (Patrick, 2006). Therefore, the present study was designed to investigate the risk which may result from exposure to different doses of lead acetate on body weight, haematological indices, and the function of liver and kidney.

MATERIALS AND METHODS

Experimental animals

The study was conducted on forty male and forty female white wool albino mice, *Mus musculus*, aged three month. The animals were kept in standard compartmented rectangular and well-ventilated cages. They maintained on standard healthy laboratory conditions at temperature of 18-24°C and twelve hours light and darkness. Animals were adapted to the new environment for fourteen days prior to study start. All mice had free access to drinking water and food, *ad libitum*, during the experimental period. They were fed with standard pellet diet (LabDiet, Missouri, USA) consisting of 60% starch, 20% casein, 10% cotton seed oil, 4% salt mixture, 5% cellulose, and 1% vitamin mixture.

Lead dosage

The animals were divided into four equal groups. Each group comprised of ten male and ten female separately and was marked as group I, II, III, and IV. The first group represented the healthy control animals, while the second, third and fourth groups were given 0.4, 0.8, and 1.2 mg/kg body weight of sub-lethal doses of lead acetate (Sigma-Aldrich Ltd., UK) respectively in their daily supply of drinking water for twelve weeks. Each mouse was weighed every week and its daily water intake was determined.

Blood collection and analysis

For haematological and biochemical investigations, blood was collected from each mouse individually. The animals were fasted for twelve hours prior to blood collection. All animals were anesthetized by chloroform and blood samples were collected immediately from their heart using heart puncture technique with the aid of disposable sterile syringe and needle (Sigma). Blood sample of each mouse was then transferred to a sterile capped tube containing anticoagulant EDTA (Greiner Bio-One, Frickenhausen, Germany) for haematological estimation. Some of blood was transferred to other sterile anticoagulant-free tube and centrifuged at 3000 rpm for about 10 min using centrifuge 5418 R (Eppendorf, Ontario, Canada) to obtain the serum for biochemical examination.

Blood cell counter URIT-2900 automated hematology analyser (Dhanwantari medical systems, DMS, India) was used to determine haematological indices including total erythrocyte count (TEC), total leukocyte count (TLC), Packed cell volume (PCV), Haemoglobin (Hb) concentration, mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), mean corpuscular haemoglobin concentration (MCHC), and the amount of platelets.

Serum biochemical parameters were analysed using auto serum analyser, Selectra ProS (Merck Ltd., Germany) and Ecoline kits (Merck Ltd.) in accordance to manufacturer's instructions. These parameters included aspartate aminotransferase (AST) or glutamic oxaloacetic transaminase (GOT), alanine aminotransferase (ALT) or glutamic-pyruvic transaminase (GPT), bilirubin concentration, gamma glutamyl transferase (GGT), lactate dehydrogenase (LDH), alkaline phosphatase (AKP), the concentration of blood urea nitrogen (BUN), and serum creatinine concentration.

Statistical analysis

The data provided in this study were analysed using statistical based methods, analysis of variance and student's t-test to compare the difference between parameters. Results were expressed as mean values \pm standard error. All statements of significance were based on probability of less than 0.05 ($P < 0.05$).

RESULTS AND DISCUSSION

Effects on body and organs weight

The results of this investigation revealed that the mean body weight of the experimental animals was significantly decreased ($P < 0.05$) in all treated groups in both genders after twelve weeks of treatment with lead acetate (table 1). They were reduced to 80%, 77%, and 71% in male while 82%, 78%, and 70% in female when compared to the healthy normal control. The harmful effect of lead acetate on the body weight was significantly increased with the increasing of its dose.

These observations are in accordance with the result of previous studies which reported that lead caused reduction in growth rate in experimental animals when fed lead (Ali *et al.*, 2010; Seddik *et al.*, 2010). It has been observed reduction of body weight in lead induce toxicity in rats (Aseth *et al.*, 1995; Teijon *et al.*, 2006). The body weight gain was decreased after treatment with lead in a dose of 400 mg/kg of the fodder (Szymezak *et al.*, 1983). The body weight loss might be resulting from the interruption of lead acetate in absorption and metabolism of feed nutrients essential for health (Marija *et al.*, 2004).

At the end of the experimental period, there was a significant increase in some organs weight including liver, kidney, and heart of both male and female under the influence of lead acetate compared to the normal group (table 1). The detected increased in organs weight under the effect of lead might be due to the necrosis and apoptosis which accompanied by the accumulation of lipids in tested organs. Accumulation of lipids

in kidney cells of intoxicated rats after treatment with lead has previously been reported (Hwang and Wang, 2001).

and MCV were found following exposure of rats to lead acetate (Helmy et al., 2000; Teijon et al., 2006).

Table 1 Lead toxicity on the body and organs weight of the experimental animals after 12 weeks of treatment

Lead acetate dose	Sex	Body weight (g)	Liver weight (g)	Kidney weight (g)	Heart weight (g)
Group I (control)		60.17±2.23	3.54±0.06	1.01±0.06	0.75±0.05
0.0 mg/kg b. wt.		61.18±1.56	3.78±0.25	1.00±0.10	0.73±0.09
Group II		48.79±2.92*	4.12±0.99*	1.10±0.09*	0.82±0.08*
0.4 mg/kg b. wt.		48.82±2.68*	4.16±0.42*	1.08±0.12*	0.79±0.11*
Group III		46.97±2.36*	4.23±0.03*	1.19±0.11*	0.94±0.10*
0.8 mg/kg b. wt.		47.56±3.01*	4.27±0.55*	1.17±0.04*	0.91±0.06*
Group IV		42.45±1.74*	4.31±0.22*	1.18±0.03	0.96±0.02*
1.2 mg/kg b. wt.		43.61±2.08*	4.33±0.37*	1.19±0.07	0.98±0.12*

Data are represented as mean±SE, n=10, *P<0.05

Table 2 Haematological values alteration in blood serum of the experimental animals after 12 weeks of treatment with lead.

Lead acetate dose	Group I (control) 0.0 mg/kg b. wt.		Group II 0.4 mg/kg b. wt.		Group III 0.8 mg/kg b. wt.		Group IV 1.2 mg/kg b. wt.	
Gender								
TEC (× 10 ³ / μl)	6.52±0.71	6.31±0.63	6.12±0.44*	5.99±0.09*	5.71±0.48*	5.68±0.78*	5.2±0.05*	5.31±0.93*
TLC (× 10 ³ / μl)	7.12±0.32	7.00±0.09	7.80±0.85	7.74±0.39	8.00±0.73*	8.22±0.26*	8.50±0.81*	8.90±0.02*
PLT (× 10 ³ / μl)	253±4.63	255±3.98	340*±5.26	349*±4.35	370±3.88*	368±5.61*	382±3.72*	379±4.11*
HGB (g/dl)	13.91±1.55	14.2±1.08	11.75±1.92*	12.03±1.02*	10.82±1.42*	11.6±1.59*	10.1±1.30*	9.34±1.05*
MCV (fl)	62.3±1.95	61.8±2.33	60.24±2.70	60.61±1.06	58.80±1.25*	58.01±2.91*	58.1±2.22*	57.95±1.08*
MCH (pg)	19.74±1.82	19.51±1.04	15.62±1.22*	16.33±1.73*	13.81±1.90*	14.03±1.41*	12.4±1.00*	12.71±1.37*
MCHC (%)	31.81±2.02	32.00±1.46	25.80±1.88	25.68±2.50	23.53±1.09*	22.84±2.39*	21.4±1.58*	21.12±2.04*
PCV (%)	46.52±2.77	46.03±2.00	45.33±1.71*	46.00±1.49*	34.98±1.80*	32.63±1.13*	31.09±2.08*	30.87±1.12*

Data are represented as mean±SE, n=10, *P<0.05

Table 3 Lead toxicity on the functions of liver and kidney of the experimental animals after 12 weeks of treatment

Lead acetate dose	Group I (control) 0.0 mg/kg b. wt.		Group II 0.4 mg/kg b. wt.		Group III 0.8 mg/kg b. wt.		Group IV 1.2 mg/kg b. wt.	
Gender								
AST (U/L)	22.8 ± 1.2	26.7 ± 0.62	36.5 ± 1.86*	35.4 ± 2.98*	41.9 ± 2.04*	64.6 ± 2.86*	63.8 ± 2.52*	68.2 ± 1.48*
ALT (U/L)	18.9 ± 1.8	21.0 ± 1.90	33.5 ± 1.72*	44.3 ± 2.04*	44.9 ± 2.12*	64.2 ± 1.96*	61.1 ± 2.04*	77.3 ± 1.80*
AKP (U/L)	74.3 ± 3.28	69.9 ± 1.56	85.8 ± 1.82*	72.9 ± 2.5*	91.4 ± 1.34*	79.3 ± 1.78*	115.3 ± 3.74*	112.6 ± 4.10*
LDH (U/L)	101.2 ± 5.34	95.2 ± 2.54	111.5 ± 5.10*	99.5 ± 3.24*	121.2 ± 3.4*	135.7 ± 2.20*	138.4 ± 3.88*	145.2 ± 4.74*
GGT (U/L)	33.4 ± 1.14	38.3 ± 2.14	43.2 ± 3.04*	42.4 ± 1.56*	46.4 ± 2.28*	65.5 ± 3.20*	61.4 ± 4.14*	80.3 ± 2.70*
Bilirubin (mg/dl)	0.72 ± 0.13	0.83 ± 0.30	3.02 ± 0.23*	2.97 ± 0.42*	2.25 ± 0.16*	3.10 ± 0.82*	3.18 ± 0.19*	3.51 ± 0.75*
Creatinine (mg/dl)	0.93 ± 0.03	0.89 ± 0.09	1.06 ± 0.27*	1.27 ± 0.29*	1.62 ± 0.42*	2.71 ± 0.05*	2.74 ± 0.08*	3.03 ± 0.61*
BUN (mg/dl)	16.87 ± 1.06	16.69 ± 1.11	17.54 ± 1.32*	19.11 ± 1.68*	22.72 ± 1.01*	21.02 ± 1.54*	26.45 ± 1.48*	28.03 ± 1.07*

Data are represented as mean±SE, n=10, *P<0.05

Effects on blood indices

The results in table 2 indicated significant reduction (P<0.05) in the total erythrocyte count (TEC) following exposure of lead acetate in group II, III, and IV in comparison with the control group in both sexes. It was also observed marked decrease in the levels of haemoglobin (Hb) and packed cell volume (PCV). MCV, MCH, and MCHC were also significantly reduced in treated mice relative to the healthy ones.

The reduction of TEC and decreased level of other indices are other concordant haematological alteration were observed in the groups where lead acetate was administrated (Falke and Zwennis, 1990; Yagminas et al., 1990) and showed microcytic hypochromic anemia (Mugahi et al., 2007; Suradkar et al., 2009). Similarly progressive decrease of TEC count, PCV, Hb,

THESE haematological changes might be attributed to the TOXIC effect of lead on cell metabolism, interaction with some reactions where calcium is their secondary mediator, and inhibition of some enzymatic activities such as aminolevulinic acid dehydratase which play key role of heme biosynthesis (Klassen, 2001), and other erythrocyte enzymes e.g GA3PD and G6PD (Calderon-Salinas et al., 1993).

Continuous exposure to lead might adversely affect the heme biosynthesis in the body due to the inhibition of cytoplasmic and mitochondrial enzymes (Atsdr, 1993). The depressing effects of lead acetate on the activity of the major enzymes in heme biosynthesis process might be refer to imperfection of iron metabolism (Chmielnika et al., 1994; Yagminas et al., 1990). The inhibitory effect of lead acetate on conversion of coproporphyrinogen III to protoporphyrin IX resulting in

shortening erythrocyte life span and decrease the production of haemoglobin (Klassen, 2001). The reduction of haematological values might be attributed to binding of lead to red blood cells which increase membrane fragility and RBCs destruction (Rous, 2000).

Total leukocyte count (TLC) was also significantly increased in all groups which were administrated lead acetate relative to the control in both genders. It has been reported that lead induced inflammation which lead to increasing in white blood cells (Yagminas *et al.*, 1990) which concur this study. Platelets count revealed considerable increase in intoxicated animals compared to the control. This may be due to thrombocytopenia after lead intoxication (Sudakova *et al.*, 1983) followed by thrombocytosis (Sudakova *et al.*, 1983; Yagminas *et al.*, 1990).

Effects on biochemical parameters

To assess the effect of lead on liver function, the activities of serum AST and ALT were investigated. AST is widely used to evaluate the liver function. ALT is a cytoplasmic enzyme while AST is found in both mitochondria and cytoplasm. The findings of this study indicated significant increase ($P < 0.05$) in the enzymatic activities of ALT and AST in male and female of the intoxicated animals relative to the healthy control ones (table 3). The activities of these enzymes were elevated paralleled with the increase of lead acetate doses. This elevation might be due to increasing of cell membrane permeability or cell membrane damage of hypatocytes under the influence of lead.

These results concur with previous studies reported an elevation in AST and ALT levels after treatment with lead due to acute hepatitis, jaundice, and liver cirrhosis (Mehta *et al.*, 2002; Patil *et al.*, 2007). Lead has hepatotoxic effect resulting in liver cell damage which causes increasing serum levels of AST and ALT (Abdou *et al.*, 2007). It has been observed that lead has toxicological effects on rat liver leading to liberation of AST and ALT (Shalan *et al.*, 2005). The high activities of plasma AST and ALT are attached by high liver microsomal membrane fluidity, production of free radicals, and alteration in the liver cells when animals were treated with lead acetate (Ibrahim *et al.*, 2012). Increase in ALT and AST enzymatic activities might be resulting from lead acetate toxicity which causes increased cellular basal metabolic rate, irritability and destructive alteration of liver (Elayat and Bakheetf, 2010; Ghorbe *et al.*, 2001). The elevated level of serum bilirubin following exposure to lead may be due to induction of heme oxygenase which play an important role in heme catabolism and can convert heme to bilirubin (Murrey *et al.*, 2006; Seddik *et al.*, 2010).

The results in table 3 also showed that the elevation of lactate dehydrogenase (LDH) was gradually increased with the increasing lead acetate dose in both male and female of all treated animals. Simillar findings were achieved in rats dosed with lead acetate (Ibrahim *et al.*, 2012; Seddik *et al.*, 2010) who found graduall stimulation in the activity of LDH in intoxicated rats. The present study also investigated the changes in serum level of alkaline phosphatase (AKP). The activity of AKP was significantly increased ($P < 0.05$) in group

II, III, and IV compared to the control group. The effect was elevated paralleled with the increase of lead acetate dose. It has been found an increasing in serum AKP activity might be resulting from liver, kidney, and bone damage leading to releasing of AKP (Kaplan and Reghetti 1970). These results are accorded with other findings (Shalan *et al.*, 2005) in which stimulation of AKP had been noted in rats under the effect of lead. The results of gamma-glutamyle transferase (GGT) showed that the stimulation of serum GGT under the effect of lead acetate was also increased with the increasing lead acetate dose in both sexes. This elevation of serum GGT is an indication of hepatotoxicity and oxidative damage in liver cells (Tatjana *et al.*, 2003).

In the case of kidney function, the concentrations of blood urea nitrogen (BUN) and creatinine were examind to check how well kidney work in intoxicated mice compared to the healthy ones. Significant increase in blood concentration of both BUN and creatinine was detected in male and female (table 3). This elevation might be due to kidney dysfunction and considered as functional evidence of lead induced nephrotoxicity (Patterson, 1965; Zook, 1972). Similar results were found after oral administration of lead in rats (Elayat and Bakheetf, 2010; Mugahi *et al.*, 2003), goat (Haneef *et al.*, 1998; Swarup and Dwivedi, 1992) and sheep (Ahmed and Shalaby, 1991).

To conclude, treatment with lead acetate at low doses has harmful effects on experimental animals and induced haematological and biochemical alterations. Therefore, this work advises people to prevent any exposure to this toxic metal to avoid its hazardouse impacts on health.

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How to cite this article:

Saeed A. Alwaleedi., Haemato-Biochemical Changes Induced by Lead Intoxication In Male and Female Albino Mice. *International Journal of Recent Scientific Research* Vol. 6, Issue, 5, pp.3999-4004, May, 2015
