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RESEARCH ARTICLE

EFFECT OF LEAD ACETATE ON SOME BIOCHEMICAL FACTORS IN BLOOD OF RATS

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ABSTRACT

Lead is the one of the most ubiquitous biochemical parameters heavy metals and has been detected in virtually all areas of the environments [air, water, and soil] and in biological systems. Lead in the environment occurs both naturally and as a consequence of human activities. The experiments investigated the effect of lead acetate on some blood serum factors in rats. Twenty rates divided to two equal groups of 10 rats, the control and treated group, the control and treated group. The control group drenched drinking Water Without lead acetate, while treated group drenched drinking Water with 300 ppm lead acetate for 45 days. At the end of study blood obtained through heart puncher and tested for effects on biochemical parameters. Total serum protein, albumin, serum globulin, cholesterol and alkaline phosphatase had been estimated. The treatment of Rates with lead acetate cause low levels of blood protein levels, albumin and increase level in globins' concentrations and cholesterol. Alkaline phosphatase enzyme linked with liver function also undergoes increased levels. It can be concluded that the exposure to lead acetate can affect different body organs systems and physiological processes.

INTRODUCTION

Due to the industrial processes and smokes from petrol vehicle (Aykin-Burns *et al*., 2003) lead is considered as one of the major environmental pollutants (Upasani and Balaraman, 2001). Although lead is eliminated from petrol in many countries, but it may have other origins such as industrial pollution (Upasani and Balaraman, 2001; Blazovic *et al*., 2001). Occupational lead exposure may occur during the manufacture of batteries, painting, printing, pottery glazing, and lead smelting processes. Exposure may also occur during the construction of tank linings, piping and other equipments that carries corrosive gases and liquids, superconductors, and fiber optic technologies (Blazovic *et al*., 2001; Heidari *et al*., 2002; Slobozhanina *et al*., 2005), during magnetic resonance imaging, and nuclear medicine (Slobozhanina *et al*., 2005). All sources of lead contribute to an increased in permissible exposure limit for metallic lead, lead oxide, and lead salts and soaps that has set by WHO and other health organizations (Heidari *et al*., 2002; Hertz-Picciotto, 2000). There are evidences, which show that lead is a toxic agent with multiple target organs such as hematopoietic system, immune system, kidneys, and nervous system (Slobozhanina *et al*., 2005). There are some controversies over the influence of lead on hematological parameters. Lead is absorbed through digestive and respiratory tracts, and skin. After absorption into the blood, 99% of lead is bound to erythrocytes and the remaining 1 percentage stay in plasma to be carried to other tissues. Serum lead half- life is around 25 days (Pabello *et al*., 2005). In a study performed on young dogs, basophilic stippling of the RBCs, nucleated RBCs, and proteinuria were observed (Markowitz, 2000). Also development of anemia, leukocytosis, monocytopenia, polychromato- philia, glycosuria, increased serum urobilinogen, and hematuria has been reported (Markowitz, 2000). In more advanced cases of lead toxicity, absolute neutrophilia, leukocytosis [with shifting to left], eosinopenia, and monocytopenia have been reported (Coles, 2000). In another study, lead was shown to induce microcytic hypochromic anemia that was due to interference with iron and copper metabolism (Tietz, 1982). Investigation of the toxicity of triethyl lead on some hematological indices has revealed a significant decrease in the MCH, MCV and RBC count and an increase in monocyte count, and platelets in comparison with the control group (Richmond, 1973)Administration of high doses of lead in female rats has caused mild anemia, reduced MCH, MCV and MCHC; low ALAD enzyme activity in erythrocytes and development of stippled RBCs, all of which disappeared when acute intoxication resolved (Sipos *et al*., 2003).

Since lead does not undergo detoxifying metabolism in the body, but rather redistributes among several compartments in response to a number of factors prior to elimination, lead is continue to present in the body after exposure ceases, particularly that in the bone, will continue to present a risk when physiological conditions dictate its systemic release (Aykin-Burns *et al*., 2003). Water solubility of lead compounds does not appear to be a good predictor of

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absorption from ingestion of lead (Upasani *et al*., 2001). The following table shows selected lead compounds, molecular weight, molecular formula and solubility in water (Blazovic *et al*., 2001).

Table 1.1. Some Lead Compounds, Their Molecular Weights, Formula and Their Solubility in Water

Chemical	Mol. wt.	Formula	Solubility in Water
Lead	207.2	Pb	Insoluble
Lead acetate	225.3	$C_4H_6O_4Pb$	Very soluble
Lead carbonate	276.2	CO_3Pb	Insoluble
Lead chloride	278.1	$PbCl_2$	Slightly soluble
Lead oxide	223.2	PbO	Insoluble
Lead nitrate	331.2	N_2O_6Pb	Very soluble
Lead phosphate	811.5	$O_8P_2Pb_3$	In soluble
Lead subacetate	566.5	$C_4H_8O_6Pb_2$	Very soluble
Lead sulfide	239.3	PbS	Insoluble

In feeding studies, lead acetate and lead oxide in feed are more readily absorbed (Heidari *et al*., 2002). Some experiments have suggested that gastrointestinal absorption of lead compounds is saturable process, i.e. the exposure to 1 mg/kg body weight of lead will result in 42% absorption, while exposure to 100 mg/kg body weight will result in 2% absorption of lead (Heidari *et al*., 2002). Once lead in the blood, it is distributed and accumulated among three compartments: the blood; soft tissues [kidney, liver, and brain]; and the mineral tissues [bones and teeth] (Slobozhanina *et al*., 2005).

The plasma fraction of lead is known to produce effects in various organ systems (Aykin-Burns *et al*., 2003) and cause changes in characteristics of lipids and proteins of cell membrane of liver and decrease the activity of enzyme linked to liver function in rats (Heidari *et al*., 2002). This reduces liver ability of detoxification (Blazovic *et al*., 2001). The toxicity of lead may largely be explained by interference with different enzymes by binding to SH groups of its proteins or by displacing other essential metal ions. Anemia considered to be the oldest symptoms combined with lead tonicity due to its effect on enzymes responsible for hemoglobin biosynthesis and reduces erythrocytes life span due to increase their fragility (Upasani *et al*., 2001), lead also has been shown to modulate various function of immune system In addition to direct toxicity on different tissues, lead had suppressive effect on the immune system and the individual exposed to lead had high susceptibility to the infections (Slobozhanina *et al*., 2005); exposure may either suppress or enhance immune responses depending on treatment and duration of exposure, due to the industrial processes and smokes from petrol vehicles (Pabello and Bolivar, 2005) lead considered as one of major environmental pollutants (Markowitz, 2000).

MATERIAL AND METHODS

Animals

Twenty animals of four weels age were kept in plastic cages in animal house at 22 – 25 c with light duration of 14 hrs. The animals divided to two equal groups. The first group considered the negative control group While the second group drenched drinking water with 300 ppm lead acetate for 45 days and considered positive control. At the end of the experiments blood samples were collected to perform the tests on this samples.

Study Parameters

Hemoglobin estimation

The concentration of hemoglobin estimation using Eyanmethemoglobin described by (Coles, 2000).

Total serum protein estimation

Protein concentration was calculated depending Biuret method (Tietz, 1982), using [Ramdox UK].

Albumin estimation

The albumin concentration determined depending on Bromocresol green method (Rodkey, 1965) using [Randox UK].

Serum globulin estimation

Serum globulins was calculated according the equation:
Globulin conc. [mg/ 100 ml] = Total protein conc. [mg /100 ml] – Albumin conc. [mg/ml].

Cholesterol estimation

Cholesterol was estimated using Kit of [Biomerieux France] by the method used by (Richmond, 1973).

Alkaline phosphatase activity estimation

Alkaline phosphatase activity was estimated using [Biomerieux France].

RESULTS AND DISCUSSION

The treatment with lead acetate cause significant decrease in total protein concentrations which reveal functional abnormalities (Belfeld and Goldberg, 1973). Studies showed that the exposure to lead environmentally and experimentally cause damages to liver cells by affecting their membrane permeability (Sipos *et al*., 2003). The study also Showed significant increase in globulin in blood that indication of immune response to lead exposure. This may be result from albumin decrease (Al-Joudy and Wahab, 2004) in the onset of liver damage symptoms.

Table 1. parameters affected by lead exposure

Parameter	Control	Treatment
Total protein [mg/100ml]	6.53± 0.28	5.89± 0.24
Albumin [g/100ml]	4.33± 0.17	3.11± 0.04
Globulin [g/100ml]	2.19±0.1	2.78± 0.25
Cholesterol [mg/100ml]	61.93± 1.6	78.81± 1.6
ALP [IU/L]	28.88 ±0.61	32.94± 0.61

In table (Aykin-Burns *et al*., 2003) the results showed increase in cholesterol concentration in blood serum which may as reason of its direct effect on in activation of several enzymes in biosynthesis pathway of cholesterol in liver cells (Mudipalli, 2007), Enzymes like 3-Hydroxy farnesyl Diphosphate synthetase and 3-methyl Glutaryl Co A Reductase inhibited

due to the rule of lead in reducing cholesterol catabolism indirectly by inhibition of cholesterol -7-α-hydroxylase causing cholesterol concentration increase (Pillai and Gupta, 2005). Results showed an increase in Alkaline Phosphatase Which shows increase in treated group. Because of the wide spread of ALP in body tissues in levels higher than those in blood plasma, its presence in blood with High concentration reveal the damages in tissues (Hainaut *et al*., 1990). Studies showed that ALP most affected enzyme and its activity increases in the cases of liver damage which contain the higher concentration among other tissues (O'Flaherty, 1991). The toxicity of lead may largely be explained by interference with different enzymes by binding to SH groups of its proteins or by displacing other essential metal ions (Pillai and Gupta, 2005).

Treatment with lead acetate cause decrease in total serum protein and albumin which may reveal different functional abnormalities. Studies showed that the exposure to lead cause damages to liver cells due to affecting cell membrane permeability (Hainaut *et al*., 1990), combined with abnormalities in kidneys functions which lead to release of amino acids with urine that limits liver capability to produce proteins (O'Flaherty, 1991). Results showed that globulin increase in serum that may be as a result of immune response to lead exposure, and this response may caused due to decrease of serum albumin as mentioned by (Hainaut *et al*., 1990) which revealed that serum globulins increased after serum albumin decreased in onset of most disease cases with liver damage. Furthermore, decreased hema- tocrit and hemoglobin levels might arise from reduction in serum copper as well as reduced iron metabolism and consumption induced by lead (Slobozhanina *et al*., 2005; Tietz, 1982). Lead suppresses bone marrow hematopoiesis, probably through its interaction with the enteric iron absorption (Tietz, 1982; Rodkey, 1965).

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