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Research Article

STUDIES ON THE INFLUENCE OF LEAD ACETATE ON BODY WEIGHT, ORGAN WEIGHT AND LIPID PEROXIDATION IN MICE HEART AND GASTROCNEMIUS MUSCLE

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A B S T R A C T

The present study was designed to determine the effects of lead acetate [Pb(C2H3O2)2] on body weight of mice along with changes in organ weight and malondialdehyde (MDA) levels, a product of cellular lipid peroxidation in tissues. The experiment was carried out on swiss albino mice weighing 20-25g and the mice were divided into three groups. Group I was designated as control whereas group II and group III received lead acetate having doses 10 mg/kg body weight of lead acetate, daily and 150 mg/kg body weight of lead acetate, weekly respectively. Study was performed at 40 and 80 days stages. Mice given lead acetate exposure for 1 day period maintained almost constant body and organ mass whereas daily and weekly doses of lead acetate caused a significant decrease in the average body and organ weight as compared to control. Significantly higher concentration of MDA was measured in tissues which confirm that lipid peroxidation is one of the molecular mechanisms of cell injury in lead acetate poisoning.

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INTRODUCTION

Lead poisoning is one of the oldest and most widely studied occupational and environmental hazards (Flora *et al*., 2006). Lead is known to induce a broad range of physiological, biochemical and behavioral dysfunctions in laboratory animals and humans, including central and peripheral nervous system, haemopietic system, cardiovascular system, kidneys, liver, male and female reproductive system (Lancranjan and Popescu, 1975; Sharma and Street, 1980; Khalil-Manesh *et al*., 1993; Goyer, 1996; Ruff *et al.*, 1996; Bressler *et al*., 1999; Lanphear *et al*., 2000; Damek-Poprawa and Sawicka-Kapusta, 2004; Leonard *et al.*, 2004; Flora *et al*., 2006).

Lead is toxic in most of its chemical forms, whether it is inhaled or ingested via water or feed. The extent to which orally administered lead absorption is small, however due to its slow rate of elimination, harmful levels of lead can accumulate in tissues after prolonged exposure in low quantities (Demichele, 1984; Ercal *et al*., 2001; Madhvi *et al*., 2007). Lead acetate given in high dose (640mg/kg/day) resulted decrease in body weight as compared to control males during developing period of pups (Garu *et al*., 2011). Goyer *et al* (1970), revealed that the exposure of lead acetate for 15 or 45 days produced a significant decrease in mice body mass, but after 90 days of exposure mass reversed to initial values suggesting a remarkable lead poisoning resistance in mice. Similar changes were observed by Graca *et al*., (2004).

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Lipid peroxidation can be described generally as a process under which oxidants such as free radicals attack lipids containing carbon-carbon double bonds, especially polyunsaturated fatty acids that involve hydrogen abstraction from a carbon with oxygen insertion resulting in lipid peroxyl radicals and hydroperoxides (Yin *et al.*, 2011). Under low lipid peroxidation rates the cell stimulates their maintenance and survival through consecutive antioxidant defense systems that unregulated antioxidant proteins resulting in an adaptive stress response. By contrast under medium or high lipid peroxidation the extent of oxidative damage overwhelms repair capacity and the cell induce apoptosis or necrosis, which facilitates development of various pathological states. Main primary product of lipid peroxiation is lipid hydroperoxides (LOOH). MDA has been widely used as a convenient biomarker for lipid peroxidation of omega-3 fatty acids because of its facile reaction with thiobarbituric acid (Pryor, 1989; Esterbauer and Cheeseman, 1990). MDA is believed to originate under stress conditions and has high capability of reaction with multiple biomolecules such as protein or DNA that leads to the formation of adducts (Luczaj and Skrzydlewska, 2003; Blair, 2008; Zarkovic *et al*., 2013).

MATERIALS AND METHODS

All the experimental procedure was conducted after the approval of Institutional Animal Ethics Committee (IAEC/Bio/6-2011) of Himachal Pradesh University, Shimla, India. Present study was conducted on heart and gastrocnemius muscle of adult sexually mature Swiss albino mice weighing 20-30g. They were maintained in polypropylene cages under hygienic conditions. Animals were allowed to acclimatize to

the laboratory conditions for 10 days. They were fed upon Hindustan lever pellets diet and water *ad libitum.*

Chemicals: All reagents used were of highest grade. Lead acetate used for this study was obtained from Sigma Chemicals, St. Louis, MO, USA.

Grouping of Animals and Dose Administration

Normal healthy looking mice showing no sign of morbidity were divided into three groups:-

- Group I served as control
- Group II received oral administration of lead acetate (10 mg/kg body weight) daily
- Group III administered lead acetate (150 mg/kg body weight), weekly

Lead acetate was given for 40 days and mice were sacrificed at 1, 40 and 80 days period by cervical dislocation.

Body weight & organ weight: Body weight along with heart and gastrocnemius muscle weight of normal and lead acetate administered mice were recorded at various stages of investigation.

Lipid peroxidation: Levels of malonialdehyde index of lipid peroxidation was estimated according to Dhindsa *et al*., (1981) using barbituric acid and the MDA content was calculated in n moles / ml.

RESULTS

Body and organ weight of mice after one day autopsy interval shows almost similar values in control and treated ones with a little progression. At 40 days stage the body weight along with organ weight showed decrease in their values in comparison to control. At 80 days stage after the withdrawal of lead acetate at 40 days stage, organs as well as wholes body mass tried to come towards the normal weight however a slight decrease was recorded in comparison to control. Mice muscle and heart witnessed increase in MDA value in comparison to control hence showed the progressive increase in normal and treated mice tissues al all stages.

Table & Fig I Changes in body weight (g) of control and lead acetate treated mice during 1-80 days period. Values are mean ± SEM; (P* < 0.05)

Table I

Groups	Days		
	1	40	80
Control	22.17±0.80	25.83±1.58	28.61±2.17
Lead acetate (10 mg/kg body weight)	22.20±2.43	23.49±0.64*	28.13±0.43
% increase or decrease	0.13%	-9.06%	-1.68%
Lead acetate (150 mg/kg body weight)	22.22±0.99	22.07±1.47*	27.48±1.52
% increase or decrease	0.22%	-14.56%	-3.95%

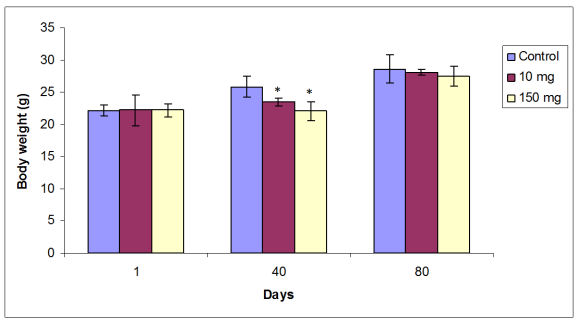

Fig. I

Table & Fig II Change in heart weight (mg) of normal and lead acetate treated mice after 1-80 days period. Values are mean ± SEM; n = 3 (P* < 0.05)

Table II

Groups	Days		
	1	40	80
Control	131.31±1.13	132.12±1.50	133.61±1.13
Lead acetate (10 mg/kg body weight)	132.34±1.93	129.37±2.54*	132.24±0.91*
% increase or decrease	0.78%	-2.08%	-1.02%
Lead acetate (150 mg/kg body weight)	133.65±1.37*	128.64±1.90*	131.97±1.54
% increase or decrease	1.78%	-2.63%	-1.23%

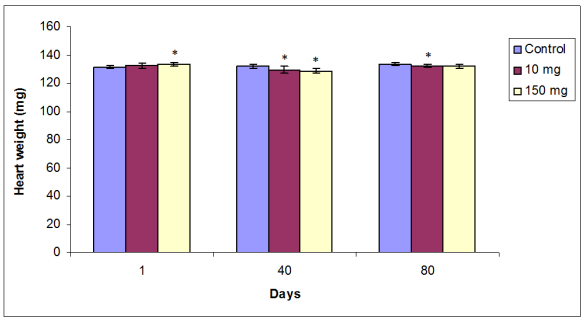

Fig II

Table & Fig III Change in gastrocnemius muscle weight (mg) of normal and lead acetate treated mice 1-80 days period. Values are mean ± SEM; n = 3 (P* < 0.05)

Table III

Groups	Days		
	1	40	80
Control	196.7±1.68	229.2±2.98	234.6±1.96
Lead acetate (10 mg/kg body weight)	197.2±2.11	221.6±1.67*	227.7±1.82*
% increase or decrease	0.25%	-3.23%	-4.26%
Lead acetate (150 mg/kg body weight)	195.8±3.0	219.3±1.9*	224.6±1.86*
% increase or decrease	0.46%	-4.26%	-2.26%

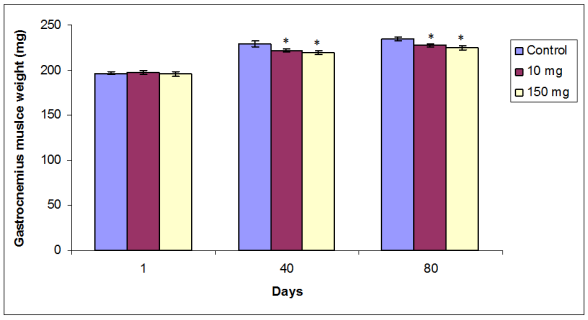

Fig III

Table & Fig IV Lipid peroxides (n moles of TBARS formed/g of fresh tissue weight) in heart of normal and lead acetate treated mice during 1-80 days period. Values are mean ± SEM; n = 3 (P* < 0.05)

Table IV

Groups	Days		
	1	40	80
Control	9.63±1.13	11.90±0.78	12.94±1.66
Lead acetate (10 mg/kg body weight)	12.13±0.31*	19.36±1.31*	21.68±1.40*
% increase or decrease	25.96%	172.24%	67.54%
Lead acetate (150 mg/kg body weight)	15.34±0.88*	22.91±1.53*	25.79±1.57*
% increase or decrease	59.29%	103.82%	99.30%

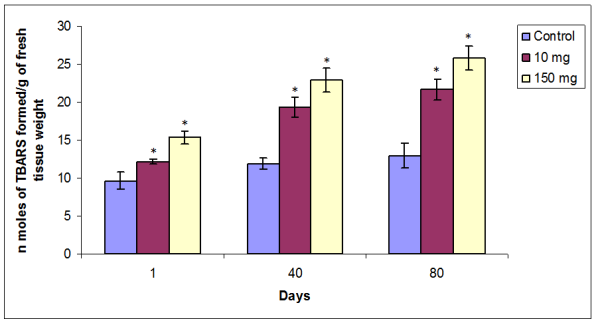

Fig IV

Table & FigV Lipid peroxides (n moles of TBARS formed/g of fresh tissue weight) in gastrocnemius muscle of normal and lead acetate treated mice during 1-80 days period. Values are mean ± SEM; n = 3 (P* < 0.05)

Table V

Groups	Days		
	1	40	80
Control	4.87±0.15	5.12±0.49	5.99±0.29
Lead acetate (10 mg/kg body weight)	5.29±0.99	9.68±1.51*	12.63±1.48*
% increase or decrease	8.62%	89.06%	110.85%
Lead acetate (150 mg/kg body weight)	6.02±0.75*	10.24±0.71*	13.39±1.37*
% increase or decrease	23.61%	100%	123.53%

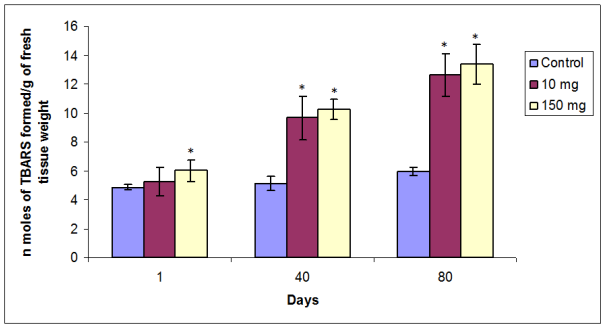

Fig. V

DISCUSSION

The objective of this study was to evaluate the effect of lead acetate on body weight and organ weight changes. Changes were also noticed in MDA content, a product of cellular lipid peroxidation in tissues. In present study, mice were exposed to different doses of intoxication. i.e. 10 mg/kg body weight, daily and 150 mg/kg body weight, weekly. Mice given lead acetate exposure for 1 day period maintained almost constant body mass. Daily and weekly doses of lead acetate caused a significant decrease in the average body and organ weight. Several other investigations confirm these findings. Yokoyama and Araki (1992), noted 15% decrease in final body weight where lead acetate was administered via drinking water to wistar rats. Banu *et al*., (2007), reported that lead acetate given in low (160 mg/kg/day) and high (320 mg/kg/day) doses caused dose dependant significant decrease in body weight as compared to control animals. Later Dieter *et al.*, (1993), reported 14-20 % decrease in body weight of male.

Lead exposed animals showed increased lipid peroxidation or decrease in antioxidant defense mechanism (Adegbesan and Adenuga, 2007; Bokara *et al*., 2008). Similar results were shown in present study where increase in lipid peroxidation was observed at all stages of investigation. Increased lipid hydroperoxides could be explained by lead-induced inhibition of free radical scavenging enzymes, leading to accumulation of reactive oxygen species and caused increased oxidation. The reason for increased lipid hydroperoxide increase could also be due to the combined inhibitory effects of various antioxidant enzymes (SOD, CAT, GPx).

CONCLUSION

The present study confirmed that exposure to lead acetate produced significant alterations in body weight, organ weight and lipid peroxidation in mice and it was found that lipid peroxidation is one of the molecular mechanisms of cell injury in heavy metal poisoning.

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