



Accumulation of lead and cadmium in the organs and tissues of albino rat

Josthna P. *, Geetharathan T., Sujatha P. and Deepika G.

Department of Biotechnology, Sri Padmavati Mahila Visvavidhyalayam, Tirupati, (A.P.) - India

Abstract

Lead and cadmium are trace metals which accumulate in the body and are extremely toxic in living organisms. The objective of the present study is to determine the accumulation of heavy metals Lead (Pb) and Cadmium (Cd) in different organs and tissues of Albino rat. The experimental animal was exposed to Pb and Cd at sub lethal concentration. The elements of Pb, and Cd were assayed by using shimadzu AA 6300 atomic adsorption spectrophotometry and the results were given as $\mu\text{g/g}$ dry wt. The high level heavy metal accumulation was found to be in liver. The order of heavy metal accumulation in liver was $\text{Cd} > \text{Pb}$, and in kidney and tissues $\text{Pb} > \text{Cd}$. The results were statistically significant at $p < 0.001$. The accumulation of lead and cadmium was significantly high in tissues.

Key-Words: Heavy metals, Accumulation, Liver, Kidney, Tissues

Introduction

Lead and cadmium are trace elements which, because they serve no known useful purpose in the body of any living organism, have serious and varied adverse effects. As a result, lead and cadmium may accumulate in particular organs of the body. In order to prevent harmful exposure, awareness of sources and uses, modes of entry into the body, toxic effects and safe limits must be established. Special attention is often given to exposure in children, because it may result in developmental problems. The lead that is absorbed is most often bioconcentrated, gathered in one particular organ or tissue, and is also bioaccumulated, which means that increasing amounts build up in the body¹⁻². Cadmium affects various organs such as bones, brain, kidney and nervous system. Research on the long term toxic effects and accumulation patterns of cadmium in fish³⁻⁴ also lend support to the effects on humans. Lead and cadmium are the two most abundant toxic metals in the environment. The common sources of lead and cadmium are diverse in nature including natural and anthropogenic processes such as combustion of coal and mineral oil, smelters, mining and alloy processing units, paint industries, and so forth. The quantity of lead used in the present decade far exceeds the total amount consumed in all previous eras⁵. The anthropogenic activities and vehicular emissions contribute to the entry of toxic metals to humans and other animal's food chains

Cadmium is an important environmental pollutant present in soil, water, air and food. Anthropogenic sources add 3–10 times more cadmium to the atmosphere than natural sources⁶. Major occupational exposure occurs from nonferrous smelters during production and processing of cadmium, its alloys, and compounds, and the exposure is increasingly common during recycling of electronic waste. Lead and cadmium do not have any detectable beneficial biological roles. On the contrary, their detrimental effects on physiological, biochemical, and behavioral dysfunctions have been documented in animals and humans by several investigators⁷⁻⁸. The higher levels affect the central and peripheral nervous systems⁹, haemopoietic system¹⁰, cardiovascular system¹¹, kidneys¹², liver¹³ and reproductive systems¹⁴⁻¹⁵. Cadmium is more toxic than lead and causes renal and hepatic damage in exposed animals¹²⁻¹³. Cadmium is a well-recognized environmental pollutant with numerous adverse health effects. It principally affects lung, liver, kidney, and testes following acute intoxication, and nephrotoxicity, immunotoxicity, osteotoxicity and tumors on prolonged exposures. In an effort to better understand, research was done to investigate which organ was the major accumulator of lead and cadmium in Rat. The methods and results will be discussed.

* Corresponding Author

E.mail: geetharathan7@gmail.com

Material and Methods

Maintenance of Experimental Animals

Healthy rats of Wistar strain were purchased from authorized vendor (M/S Raghavendra Enterprises, Bangalore, India). All rats were housed in polypropylene cages (18" 10"x 8") lined with sterilized paddy husk, and provided filtered tap water and rat food ad libitum in an air-conditioned environment (25±2°C) with a 12-h light and 12-in dark cycle. The experiments were carried out in accordance with the guidelines of the Committee for the purpose of Control and Supervision on Experiments on Animals.

Chemicals

Analytical grade: Lead nitrate (PbNO₃) and Cadmium chloride (CdCl₂)

Procedure

Rats (110±10 days weighting 190±12 g) were selected for experimentation and acclimatized to laboratory conditions for a week. Thirty rats were used for the experiment, and were starved for 24 hr prior to experimentation. After the adaptation period, rats were randomly assigned to one of two groups, rats in group I (controls) II and III (experimental) received the drinking water and drinking water supplemented with 25 mg Pb/L, 5 mg Cd/L, respectively. The consumption of feed and water, and body weight gains were evaluated during the whole experimental period. The rats were killed by immersion in gaseous carbon dioxide. Activities were measured in the liver, kidneys, and in the tissues. Before measurements, the organs and tissues were dried with filter paper and weighed.

Metal Analysis

Metal levels in liver, kidney and tissues were estimated according to the method of Ballentine and Barford¹⁶(1957). To 100mg of the tissues, 1ml of concentrated nitric acid was added, followed by 1ml of perchloric acid. The sample was then digested over a sand bath until the solution becomes clear and yellow in color. If the color of the digest was brown, more nitric acid and perchloric acid were added and the digestion was repeated. The digest was made up to known volume with deionized water. Aliquots of this were used to estimate the metals by atomic absorption spectrophotometer. Results of accumulated heavy metals were measured as (mean ± SD) were expressed as the percentage of the total dose. Statistical evaluations were based on 10 rats per group. Mean values were compared by Student's t-test; values which differed at P< 0.001 were considered statistically significant.

Results and Discussion

Guideline values recommended for lead, cadmium in drinking water are 0.05 mg/L, 0.005 mg/L and

respectively¹⁷. In our experiment the exposure of rats to lead and cadmium involved much higher doses of these metals but below concentrations which are thought to be chronically toxic to rats when these metals are given separately¹⁸. The doses of 25 mg Pb/L, 5 mg Cd/L were selected because they are reported to resemble the ratio of 5 to 1 among the two toxic metals in drinking water¹⁹ results showing a marked decrease in body weight gain within the experimental period may, to some extent, support the observations reported by Mahaffey *et al.* who showed that cadmium and lead administered may depress weight gain. Although there are reports indicating that cadmium may depress gastrointestinal uptake of concomitant doses of lead and cadmium. The heavy metals are known to affect organ/body weight ratio when given at toxic doses²⁰. Average daily food and drinking water intake decreased in the rats intoxicated with cadmium and lead. However, differences were not statistically significant. Rats intoxicated with the metals showed a markedly decreased body weight gain in comparison to that in the controls (Table 1). Gross examinations did not reveal any significant morphological alterations and no statistically significant differences were found in relative organ to body weights. The heavy metals like lead (Pb) and cadmium (Cd) were analyzed in different organs like liver, kidney and tissues (Table 2) of both the control and experimental animals. Results showed that the accumulation of lead and cadmium was high in tissues. The order of heavy metal accumulation in liver was Cd > Pb (Fig 1), in kidney (Fig 2) and tissues (Fig 3) Pb > Cd.

Acknowledgement

Authors thank Sri Padmavati Mahilavisvidyalayam, Tirupati, for providing lab facilities to carry out research work.

References

1. Sures, B., H. Taraschewski, and E. Jackwerth. (1994). "Comparative Study of Lead Accumulation in Different Organs of Perch (*Percafluviatilis*) and Its Intestinal Parasite *Acanthocephalus lucii*," Bulletin of Environmental Contamination and Toxicology, Vol. 52, pp 269-273.
2. Coughlan, David J., Steven P. Gloss, and Joe Kubota, (1986). "Acute and Sub-Chronic Toxicity of Lead to the Early Life Stages of Smallmouth Bass (*Micropterus dolomieu*)," Water, Air, and Soil Pollution, Vol. 28, pp 265-275.
3. Suresh, A., B. Sivaramakrishna, and K. Radhakrishnaiah. (1993). "Patterns of Cadmium Accumulation in the Organs of Fry

- and Fingerlings of Freshwater Fish (Cyprinus Carpio) Following Cadmium Exposure,” *Chemosphere*, Vol. 26, No.5, pp 945-953.
4. Nriagu, J.O. and J.B. Sprague. (1987). “Effects of Cadmium on Freshwater Fish from Cadmium in the Aquatic Environment,” John Wiley and Sons, New York, NY, pp 139-169.
 5. Phillips C, Gyori Z, Kovács B. (2003). The effect of adding cadmium and lead alone or in combination to the diet of pigs on their growth, carcase composition and reproduction. *Journal of the Science of Food and Agriculture*. 83(13):1357–1365.
 6. Okada IA, Sakuma AM, Maid FD, Dovidemskas S, Zenebon O. (1997). Evaluation of lead and cadmium in milk due to environmental contamination in Paraiba valley region of South Estern Brazil. *Raissade-Saude-Publica*. 31:140–143.
 7. Goyer RA, Cherian MG. (1979). Ascorbic acid and EDTA treatment of lead toxicity in rats. *Life Sciences*. 24(5):433–438.
 8. Ruff HA, Markowitz ME, Bijur PE, and Rosen JF. (1996). Relationships among blood lead levels, iron deficiency, and cognitive development in two-year-old children. *Environmental Health Perspectives*. 104(2):180–185.
 9. Dressier J, Kim KA, Chakraborti T, Goldstein G. (1999). Molecular mechanisms of lead neurotoxicity. *Neurochemical Research*. 24(4):595–600.
 10. De Silva PE (1981). Determination of lead in plasma and studies on its relationship to lead in erythrocytes. *Brazilian Journal of Indigenous Medicine*. 38:209–217.
 11. Khalil-Manesh F, Gonick HC, Weiler EWJ, Prins B, Weber MA, Purdy RE. (1993). Lead-induced hypertension: possible role of endothelial factors. *American Journal of Hypertension*. 6(9):723–729.
 12. Humphreys DJ. (1991) Effects of exposure to excessive quantities of lead on animals. *British Veterinary Journal*. 147(1):18–30.
 13. Sharma RP, Street JC. (1980). Public health aspects of toxic heavy metals in animal feeds. *Journal of the American Veterinary Medical Association*. 177(2):149–153.
 14. Rom WN. (1980). Effects of lead on reproduction. In: Infante PF, Legator MS, editors. *In: Proceedings of the Workshop on Methodology for Assessing Reproductive Hazards in the Workplace*; Washington, DC, USA. pp. 33–42.
 15. Lancranjan I, Popescu HI, GAvanescu O, Klepsch I, Serbănescu M. Reproductive ability of workmen occupationally exposed to lead. *Archives of Environmental Health*. 1975; 30(8):396–401.
 16. Ballantine, R. and Barford, D. D. (1957). Determination of metals. In: *Methods in Enzymology*. Eds. S. P. Colowick and N. O. Kaplan. 3, pp. 1002. Academic Press. Inc. N. Y.
 17. WHO. (1984) Guidelines for drinking-water quality.
 18. Bowen H.J.M (1979). *Environmental Chemistry of the Elements*. Academic Press, NY.
 19. Van Vleet J.F., Boon D., Ferrans V.J. (1981). Induction of lesions of selenium-vitamin E deficiency in weanling swine fed silver, cobalt, tellurium, zinc, cadmium, and vanadium. *Am. J. Vet. Res.*, 42, 789-799.
 20. Mahaffey K.R., Capar S.G., Gladen B.C. Fowler B.A. (1981). Concurrent exposure to lead, cadmium, and arsenic. Effects on toxicity and tissue metal concentrations in the rat. *J. Lab. Clin. Med.*, 98, 463-481.

Table 1: Body weight gain (in gram) in control and lead, and cadmium exposed rats

| Body weight | Group I | Group II | Group III |
|----------------|----------|----------|-----------|
| Initial weight | 225 ± 13 | 232 ± 17 | 218 ± 14 |
| Final weight | 445 ± 47 | 385 ± 43 | 348 ± 35 |

Group I – control rats, Group II rats exposed to lead (25 mg/L), Group III rats exposed to cadmium (5 mg/L)

Table 2: Heavy metal analysis in the different organs of Female Rat (µg/g.d.wt)

| Heavy Metals | Liver | Kidney | Tissue |
|--------------|---------------|---------------|---------------|
| Pb | 2.000 ± 0.015 | 2.000 ± 0.017 | 1.900 ± 0.020 |
| Cd | 2.400 ± 0.020 | 1.166 ± 0.015 | 0.646 ± 0.025 |

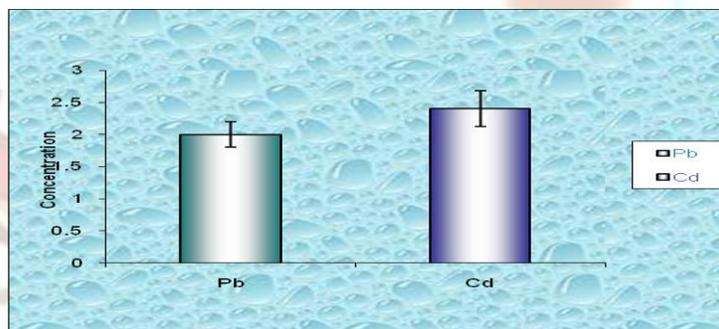


Fig. 1: Accumulation of Lead and Cadmium in Liver

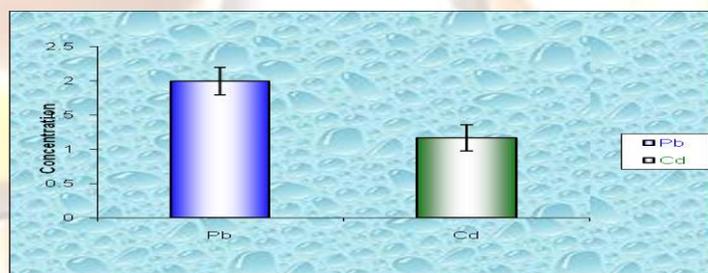


Fig. 2: Accumulation of Lead and Cadmium in Kidney

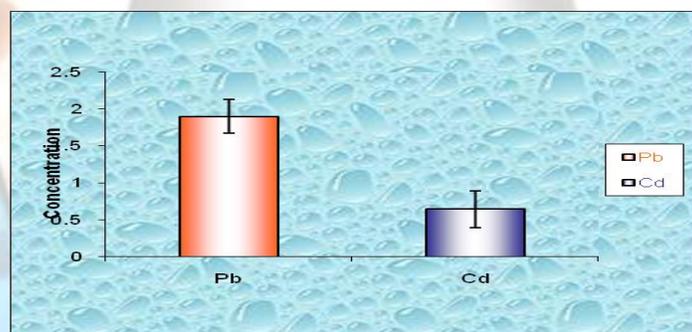


Fig. 3: Accumulation of Lead and Cadmium in Tissues