International Journal of Advanced Research in Biological Sciences
ISSN: 2348-8069
www.ijarbs.com
DOI: 10.22192/ijarbs Coden: IJARQG (USA) Volume 8, Issue 8 -2021

Research Article

DOI: http://dx.doi.org/10.22192/ijarbs.2021.08.08.018

Effect of Cadmium on Liver Glycogen Reserve and its Size in Albino Rats

Kshama Dwivedi

Department of Zoology, Bareilly College, Bareilly – 243005 (U, P), INDIA,
Corresponding Author. E-mail: *kshamadwivedi74@gmail.com*

Abstract

Albino rats were exposed to two different chronic doses of cadmium chloride (2.6 mg/kg.b.wt and 5.2mg/kg.b.wt) in three experimental sets for 15 days, 30 days and 60 days. The present study shows, depletion of liver glycogen in cadmium intoxicated rats on the completion of intoxication duration. The higher dose of cadmium showed more adverse effect as compared to lower dose. Increase in the size of liver was observed in both the groups i.e., animal intoxicated with low or higher dose for 30 days and 60 days. Insignificant increase in liver size was observed in the groups intoxicated for 15 days. Hepatomegaly was characteristic of 60- and 30-days exposed rats. The relative organ weight showed significant ($P<0.05$) increase in size. Observations showed that rats were under stress due to cadmium toxicity. Toxic effects were correlated to dose and time.

Keywords: Liver, Glycogen, Cadmium, Albino rats.

Introduction

Cadmium being a non-essential element present as contaminant in food, water as well as polluted air. It is an important industrial and environmental toxicant with many industrial applications. Cadmium is known for its non-corrosive nature is widely used in paints and dyes, cement, and phosphate fertilizers (Jarrup, 2003). Cadmium occurs naturally in the environment, in insignificant amounts but its release in the recent past is steadily increasing due to human activities causing pollution of soil and aquatic systems. During last century cadmium concentration in atmospheric, aquatic, and terrestrial environment as food chain contamination, has increased the risk of human exposure (ATSDR 2012). The two main routes of human exposure are inhalation and ingestion. It has tendency to get accumulated in various organs and organ system such as brain, liver, lungs, spleen, pancreas, blood system, bone, and testes (Emmanuel *et. al.*, 2003). Cadmium act as active stressor, leading to metabolic alterations similar those observed in starvation conditions.

In humans and other mammals, cadmium exposure can result in variety of adverse effects such as testicular damage, pulmonary edema, renal and hepatic dysfunction, osteomalacia and generalized osteoporosis (Horuguchi *et. al.*, 2010) and type- 2 diabetes (Satarug *et. al.*, 2017). Currently dietary cadmium intake is within FAO/ WHO tolerable level of 58 µg/day for 70 kg person (Soisungwan Satarug, 2018). The aim of this study was to investigate the toxic effect of chronic cadmium exposure on liver glycogen and on its size.

Materials and Methods

Experimental animals

Albino rats of 6 to 10 weeks old weighing approx. 150- 160 grams were purchased from the Laboratory Animal Resource Section, Indian Veterinary Research Institute (IVRI) Izzatnagar Bareilly, U. P. and maintained in experimental animal shed of the division. Animals were kept for a week to be conditioned to the new environment prior to the start of experiments. Animals were kept under conventional condition (6 rats per steel cage, 12 hr. light to dark cycle). The animals were made available to standard rat food and tap water ad libitum. All the chemicals used were from Sigma Chemicals Co., Merk and Qualigens.

Experimental design

The experimental rats were randomly divided into these groups A, B, and C each comprised of 6 rats. Group A was control, Group B was intoxicated with low $CdCl_2$ (2.6mg/kg.b.wt.) and Group C with high dose of $CdCl_2$ (5.2mg/kg.b.wt.). The compound was given in tap water while the control received only plain tap water per os by gavage. In three experimental sets of 15 days, 30 days and 60 days animals were monitored, and their body weight was taken weekly. Mortality rate, food consumption, clinical signs and symptoms and behavioral activities were under observation.

Blood was collected for hematological and biochemical examination after 15 days, 30 days and 60 days from the retro-orbital plexus with the help of capillary tube as described by Sorge and Buckner (1964). Blood was collected in two aliquots. In one aliquot, EDTA (1mg/ml) was added for hematological parameters estimation other aliquot was without any anticoagulant for harvesting serum for biochemical estimation (centrifuged at 3000 rpm for ten minutes). The test samples were stored in air vials at -20^{o}C till used.

Statistical analysis

All data are presented as the mean ±standard error of mean (SEM). The results were analysed for statistical significance by one-way analysis of variance (ANOVA) followed by Dunnett's *post hoc* test of significance. *P* values less than 0.05 ($p \leq 0.05$) were considered as statistically significant.

Results and Discussion

Clinical observations showed that exposed animals were docile and less active than control group. No mortality occurred in control and other groups treated with the different doses of heavy metal. There was time and dose-dependent reduction in the body weight when treated for 15-, 30- and 60-days duration. The effect of treatment of cadmium chloride on liver glycogen reserve of albino rats when intoxicated 15days, 30 days and 60 days is given in Table 1 to 3.

Table1: Effect of cadmium chloride on liver glycogen reserve of albino rats (mg/g of wet tissue)

Groups	15 days	30 days	60days
Group A (control)	1.028±0.063	0.954±0.042	0.949±0.02
Group B (Cd L) **Percentage change**	7.22±0.13 -9.11	0.758±0.063 -20.56	0.620±0.03 -34.68
Group C (Cd H) **Percentage change**	7.03±0.15 -12.67	0.701±0.069 -26.52	0.574±0.02 -39.48

Cd (L)= Low Dose Cd (L)= High Dose

Sharp decline in liver glycogen content in all the groups was observed when animals were intoxicated for 15 days. After 30 days post treatment of heavy metals, maximum fall was observed in group C (26.52%) which was treated with high cadmium dose followed by group C (20.56%).After 60 days post treatment the liver glycogen decreased by almost 39.48% in group C with high cadmium dose followed by group B (34.68%). Decrease in glycogen was directly related with the amount of Cadmium dose and exposure of time of intoxication in three sets of experiment.

The relative liver weight increased significantly in all the groups of 15 days, 30 days and 60 days treated rats indicating hepatomegaly. Relative liver weight increased by 9.59%, and 19.26% in groups B and C respectively in 15 days treated rats and while the relative liver weight increased by 11.87%, and 16.25% in groups B and C respectively in 30 days treated rats when compared to control. In 60 days treated rats, percentage change is very significant in group C (35%) and in group B it is 21.62% compared to control. The increase in relative liver weight seems to correspond to the dose and duration of exposure.

Table 2: Absolute liver weight (g) and percentage change in albino rats after oral administration (gavage) Cadmium chloride.

Groups	15 days	30 days	60 days
Group A (control)	6.34±0.14	7.15±0.42	7.84±0.16
Group B (Cd L)	6.75±0.14	7.76±0.79	8.96±0.23
Percentage Change	(6.47)	(8.53)	(14.30)
Group C (Cd H)	7.26±0.17	7.84±0.58	10.09±0.23
Percentage change	(14.50)	(10.30)	(28.70)

Cd (L)= Low Dose Cd (L)= High Dose

Table 3: Relative liver weight (g/100g body weight)in albino rats after oral administration (gavage) of Cadmium chloride.

Groups	15 days	30 days	60 days
Group A (control)	3.842±0.21	3.65±0.41	3.50±0.15
Group B (Cd L)	4.210±0.31	4.08±0.12	4.33±0.40
Percentage Change	9.59	11.87	21.62
Group C (Cd H)	4.560±0.24	4.24±0.04	4.82±0.40
Percentage change	19.26	16.25	35.90

Cd (L)= Low Dose Cd (L)= High Dose

Cadmium high concentration exposed rats showed decreased liver glycogen while increase in liver size, showing hepatomegaly condition. The liver is one of the primary organs that takes up the cadmium in greatest quantity after exposure. The studies of cadmium induced toxicity shows the acute hepatotoxicity or liver injury showing direct toxic effect of the metal ischemia due to endothelial cell injury. Chapatwala *et.al* (1982) suggested that cadmium alters hepatic and renal gluconeogenesis in female rats which results in increase of serum glucose. Larsson and Haux (1982) reported similar findings. Elevated concentration of glycosylated hemoglobin, an indicator of high blood glucose level, was reported in several Danish workers exposed to arsenic (Jensen and Hansen, 1998).The toxicity of cadmium is attributed to its ability to generate reactive oxygen species that may act as signaling molecules in the induction of gene expression and apoptosis (Waisberg et. al., 2003), deplete endogenous radical scavengers, and damage a variety of transport proteins including the Na^+/ K^+- ATPase. Bedii and Kenan (2005), reported that levels of glycogen reserves in the liver and muscle tissue decreased in fish exposed to sublethal concentration of cadmium compared to control group. Increase in the serum glucose level in fish living under heavy metal stress was reported by Choudhury *et. al.*, (2004). This can be attributed to severe factors and one of them is the decrease in the specific activity of some enzymes like phosphofructokinase, lactate dehydrogenase and citrate kinase that decrease the capacity of glycolysis (Almeida 2001).It also affects the glycogen and lipids levels of the organism. Glycogen level are found to be highest in liver, as it is chief organ of carbohydrate metabolism in animals followed by muscles. Liver glycogen is concern with storage and exports of hexose units for maintenance of blood glucose. Muscle glycogen acts as readily available source of hexose units for glycolysis within the muscles itself. A fall in the liver glycogen level clearly indicates its rapid utilization to meet the enhanced energy demands in the fishes exposed to toxicant through glycolysis or hexose monophosphate pathway. It is assumed that decrease in glycogen content may be due to inhibition of hormone which is contributed to glycogen synthesis. Decrease in liver glycogen levels is correlated with the reports of earlier workers

(Bedii and Kenan, 2005). Some investigations also showed that heavy metals could decrease the glycogen reserve in fish and invertebrates by affecting the activities of enzymes that play a role in carbohydrate metabolism. Serum glucose levels also increased in increasing concentrations of cadmium during intake is indication of liver glycogen depletion. El-Sokkary *et.al.*(2010), reported some histopathological changes such as loss of normal architecture of the parenchymatous tissue, cytoplasmic vacuolization, cellular degeneration and necrosis, congested blood vessels, destructed mitochondria cristae, fat globules, severe glycogen depletion lipofuscin in rats exposed to cadmium for 22 days. Increase in liver weight may be due to loss of appetite also (Sajjad *et. al.*, 2014) or damage of the system glucocorticoid essential hormone in regulation of glucose (Albasha and Azab, 2014).

Conclusion

Thus, present investigation shows that on exposure of albino rats to sub-acute dose of cadmium for 15 days, 30 days and 60 days duration results, in fall of liver glycogen, probably the metal stimulates hormones that accelerate glycogen breakdown or inhibition of those enzymes or intermedins involved in glycogen synthesis. It was observed that there was decrease of absolute liver weight but increase in relative liver weight compared to body weight. This is showing hepatomegaly condition in the rats treated with cadmium chloride, which shows toxicity of cadmium on albino rats.

References

1. Albasha M.O., and El-Saied Azab A. (2014). Effect of cadmium on the liver and amelioration by aqueous extracts of Fenugreek seeds, Rosemary and Cinnamon in guinea pigs: Histological and biochemical study. *Cell Biology* 2(2): 7-17.
2. Almeida J.A., Novelli E.L.B., Dal-Pai Sila M., and Alves-Junior R. (2001). Environmental cadmium exposure and metabolic responses of the Nile tilapia *Oreochromis niloticus*. Environ. Pollute. 114: 169-175.
3. ATSDR (Agency for Toxic Substances and Disease Registry).(2012). Toxicological Profile for Cadmium; Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention: Atlanta, GA, USA.
4. Bedii C. and Kenan E. (2005). The effect of cadmium on levels of glucose in serum and glycogen reserves in the liver and muscle tissues of *Cyprinus carpio* (L., 1758). *Turk J Vet. Animal Sci.*29(1): 113-117.
5. Chapatwala, K.D., Hobson, M., Desaiah, D. and Rajanna, B. (1982). Effect of cadmium on hepatic and renal gluconeogenic enzymes in female rats. *Toxicology. Let*.12**:** 27-34.
6. Chowdhury M.J., Pane E.F., and Wood C.M., (2004). Physiological effects of dietary cadmium acclimation and water born cadmium challenge in rainbow trout: Respiratory, ionoregulatory, and stress parameters. *Comp. Biochem. Physiol. C. Toxicol. Pharmacol.* 139(1-3): 163-173.
7. El-Sokkary G.H., Nafady A.A., and Shabash E.H. (2010). Melatonin administration ameliorates cadmium-induced oxidative stress and morphological changes in the liver of rat. *Ecotoxicol. Environ, Saf.* 73: 465-463.
8. Emmanuel, E., Allison, K., Margaret, M., Carmen, A., Elizabeth, A., Kanesha, L. W., and Bhattacharyya, M. H. (2003). Cadmium pathways during gestation and lactation in control versus metallothionein 1,2- knockout mice. *Toxicol Sci.* Feb;71(2):154-63.
9. Horiguchi, H., Aoshima, K., Oguma, E., Sasaki, S., Miyamoto, K., Hosoi, Y., Katoh, T. and Kayama, F. (2010). Latest status of cadmium accumulation and its effects on kidneys, bone, and erythropoiesis in inhabitants of the formerly cadmium polluted Jinzu river basin in Toyama, Japan, after restoration of rice paddies. *Int. Arch. Occup. Environ. Health*. 83:953-970.
10. Jarrup L. (2003). Hazards of heavy metal contamination *Br. Med. Bull*. 68: 167-182.
11. Jensen, E.G. and Hausen, M.L. (1998). Occupational arsenic exposure and glycosylated hemoglobin. *Analyst.* 123:77-80.
12. Larsson, A. and Haux, C. (1982). Altered carbohydrate metabolism in fish exposed to sublethal level of cadmium. *J. Environ. Biol*.3(2):71-81.
13. Sajjad S., Malik H., Farooq U., Rashid F., Nasim H., Tariq S., and Rehman S. (2014). Cadmium chloride toxicity revisited: Effect on certain andrological, endocrinological, and biochemical parameters of adult male rabbits. *Physiological Research* 63: 505-512.

14. Satarug, S. (2018). Dietary cadmium intake and its effects on kidneys. *Toxics*. 6 (15): 1-23.
15. Satarug, S., Vesey, D. A. and Gobe, G. C. (2017). Current health risk assessment practice for dietary cadmium: Data from different countries. *Food and Chemical Toxicology*. 106: 430-445.
16. Sorg, D. A. and Buckner, B. (1964). A simple method of obtaining venous blood from small laboratory animals. *Proc. Soc. Exp. Bio. Med*. 115: 1131-1132.
17. Waisberg M., Joseph P., Hale B., and Beyersmann D. (2003). Target for toxicity and death due to exposure to cadmium chloride. *Toxicology*. 192(2-3): 95-117.

Access this Article in Online

Website: www.ijarbs.com

Subject: Toxicology

Quick Response Code

DOI:10.22192/ijarbs.2021.08.08.018

How to cite this article:
Kshama Dwivedi. (2021). Effect of Cadmium on Liver Glycogen Reserve and its Size in Albino Rats. Int. J. Adv. Res. Biol. Sci. 8(11): 150-154.
DOI: http://dx.doi.org/10.22192/ijarbs.2021.08.08.018