



EFFECT OF CADMIUM ON GLUCOSE AND GLYCOGEN CONTENT OF GUPPY FISH

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ABSTRACT

Effect of Cadmium on the glycogen and glucose in liver and kidney of Guppy showed drastic changes. Liver and Kidney being the main site of metabolic activity in body and highly active organ of detoxification, was selected for the study purpose. In the present study, the sub-lethal effects of cadmium on various biochemical parameters of Guppy were studied. Liver and Kidney being the main site of metabolic activity in body and highly active in the transport of ions and maintaining equilibrium. There was a drastic decrease in the liver glycogen due to acute hypoxia. The fish was exposed to sub-lethal concentration for 96 hours 0.284 ppm of cadmium was calculated by probit method for acute study and for 15, 30 and 45 days for chronic toxicity studies. . These changes were concentration dependent. The experimental fish were exposed to sub lethal, higher and lower sub lethal concentration (1/3rd & 1/10th of the 96hr LC50 value) of cadmium for 15, 30 and 45 days.

KEYWORDS: Cadmium, Glucose, Glycogen and Sub lethal.

INTRODUCTION

The persistence and ubiquitous nature of Cadmium is coupled with their tendency to accumulate in organisms ultimately produce toxic reactions in aquatic biota, especially in fish. Thus, the deleterious effects of metals on aquatic ecosystems necessitate the continuous monitoring of their accumulation in key species, since it affords indication of temporal and spatial extent of the process and impact on organism's health. As the bioaccumulation of Cadmium in aquatic biota is a serious current issue, it should be closely monitored. The intent of the present review is to briefly address the Cadmium emission sources, uptake and impacts of Cadmium on freshwater fish and bioaccumulation. Mainly, try to emphasis the Cadmium accumulation affinity in freshwater fish tissues; e.g., gills, kidney, liver and the muscle tissue.

This study also gives an overview of differences in the magnitude of Cd residues accumulated in different freshwater -fish tissues in several natural environments of the world. Nevertheless, metal bioaccumulation terms of biodynamic modeling and physiological handling of metals like Cd in freshwater fish, Cadmium that absorbs across the gills or the intestinal walls is distributed via the circulation, bond to transport proteins and distributed, to different tissues of the body.^[9] Once Cd enters into the cells, the metal is made available for the interaction with cytoplasm components such as enzymes causing toxic effects and metallothioneins (MTs).

The aquatic life is constantly threatened by the seepage of Cadmiums and their constituents from agricultural fields, industrial and domestic sewage. Fishes are much vulnerable to toxic substances and their bioaccumulation cause serious risk to life. Such toxic substances enter human body through food chain, as fishes constitute an important part of animal protein in rural and urban areas. Their availability and selectivity to toxic substances are main criteria for selection as an experimental animal. The mechanism by which the Cadmiums exert their toxic action on the arthropods or fishes depends largely on the biochemical processes of animal and the physico-chemical properties of phosphorous compounds. Many toxic substances in use today as control against pests have been known to have accumulative adverse effects on non- target organisms as reported by Omoregie *et al.*^[1]

Cadmiums in aquatic ecosystem may cause reduced productivity and biological activity. It has been reported that fish are extremely sensitive to aquatic pollution and shows severe physiological changes when exposed to sublethal concentrations of it. There is limited knowledge of the effects of daily exposure of fish to sublethal doses of Cadmium and therapeutants. Ufodike and Omoregie^[2] reported reduced growths in *Cyprinus carpio* and *Oreochromis niloticus* when exposed to sublethal concentration of Dipterex (Trichlorphon) and Gammalin 20 and Actelli 25EC. (0, 0-diethyl-0-(3,5,6-trichloro-2-pyridyl)-phosphorothioate) commonly known as Dursban is broad spectrum insecticide. The present

work includes sublethal effects of cadmium on the liver and kidney of *Guppy*, fish. Liver and Kidney being the main site of metabolic activity in body and highly active in detoxifying heavy metal, was selected for the study purpose.

MATERIAL AND METHODS

Guppy (Cyprinodontiform: Poeciliidae) weighing (0.5-1.0 g), length (3.0-4.5 cm) were collected from local pond in Ajmer and acclimatized to laboratory condition. Test chambers were glass aquaria of about 50 liter capacity. The aquaria were aerated with a central system for a period of 48 hours and the fish were exposed to 15, 30 & 45 days conditioning period at room temperature. The fish were fed with commercial pelleted food at least once a day during this period. Acclimatized fish were not fed 24-hr before the start of the experimentation.

For the determination of LC50 concentration, four groups of 10 acclimated fishes were taken in each glass aquarium of capacity, 50 liters. Each group was exposed to 0.284 (Sub lethal), (1/10th of sub lethal) 0.028, and (1/3rd of sub lethal) 0.094 ppm of Cadmium. Mortality of fishes was recorded in each group after 96 hr. The regression equations were established by using probit - mortality and log of concentration of Cadmium and LC50 value was determined. The three concentrations were calculated for experimental studies. Four glass aquaria containing 20 acclimatized fishes in each. The

four groups of fishes were treated for 15, 30 & 45 days as follow.

Group I : Control

Group II : Exposed to 1/10th of LC50 value

Group III : Exposed to 1/3rd of LC50 value

Group IV : Exposed to Sub lethal (LC50) value

The fish were exposed to this concentrations for 15, 30 and 45th days and a control group was maintained at an identical environment. The fish was dissected out from both groups on 15, 30 and 45th days and biochemical analysis was done on the liver and kidney of *Guppy fish*, glycogen by Carrol's method^[3] and glucose by enzymatic method^[4], were estimated quantitatively.

OBSERVATION

Mortality studies showed that the sub lethal level, LC50 of *Guppy* for 96 hr. exposure sub lethal was 0.284 ppm, (1/10th of sub lethal) 0.028, and (1/3rd of sub lethal) 0.094 ppm for Cadmium. The minimum effective doses were calculated for experimental purposes. Biochemical estimations of glycogen, protein and glucose in liver and kidney of control as well as in treated *Guppy* for different periods (15, 30, 45th days) of exposure are given in the tabular form as follows:

Values are expressed mean \pm SD of observations.

Values are significant at ^xP < 0.05, ^yP < 0.01, ^zP < 0.001

Table 1: Changes in Glycogen content in liver and Kidney of *Guppy* during control and post-treatment with three different concentration of Cadmium at different periods (15, 30, 45th days) of exposure.

Days of Exposure	Control	Exposure Concentration		
		1/10 th of LD50	1/3 rd of LD50	LD50
15 Days	2.17 \pm 0.008 ^y	1.48 \pm 0.008 ^y	1.39 \pm 0.005	1.27 \pm 0.008 ^y
30 Days	2.14 \pm 0.01 ^x	1.44 \pm 0.008 ^y	1.37 \pm 0.01 ^x	1.25 \pm 0.008 ^y
45 Days	2.09 \pm 0.008 ^y	1.41 \pm 0.008 ^y	1.30 \pm 0.005 ^y	1.23 \pm 0.008 ^y

Table 2: Changes in Glycogen content in kidney of *Guppy*.

Days of Exposure	Control	Exposure Concentration		
		1/10 th of LD50	1/3 rd of LD50	LD50
15 Days	1.68 \pm 0.012 ^x	1.53 \pm 0.008 ^y	1.48 \pm 0.008 ^y	1.44 \pm 0.008 ^y
30 Days	1.65 \pm 0.012 ^x	1.42 \pm 0.008 ^y	1.39 \pm 0.008 ^y	1.33 \pm 0.008 ^y
45 Days	1.61 \pm 0.012 ^x	1.39 \pm 0.008 ^y	1.36 \pm 0.008 ^y	1.28 \pm 0.008 ^y



Fig: 1 Liver

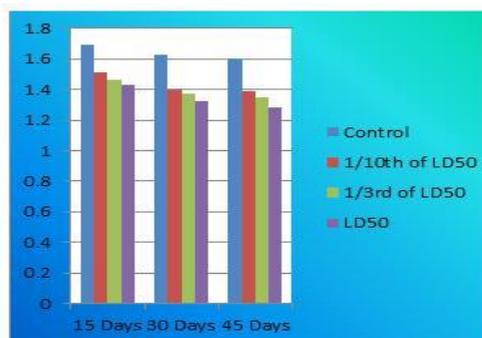


Fig: 2 Kidney

Figure 1&2: Changes in Glycogen content in liver & kidney of *Guppy* during control and post-treatment with three different concentration of Cadmium at different periods (15, 30, 45th days) of exposure

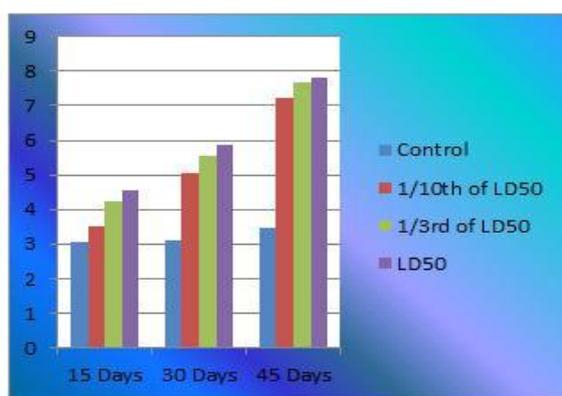
Table 3: Changes in Glucose level in liver of Guppy during control and post-treatment with three different concentration of Cadmium at different periods (15, 30, 45th days) of exposure.

Days of Exposure	Control	Exposure Concentration		
		1/10 th of LD50	1/3 rd of LD50	LD50
15 Days	3.07±0.008 ^y	3.53±0.014 ^x	4.27±0.005	4.55±0.01 ^x
30 Days	3.09±0.01 ^y	5.08±0.008 ^y	5.57±0.005 ^y	5.90±0.008 ^y
45 Days	3.47±0.005 ^y	7.23±0.008 ^y	7.68±0.005 ^y	7.73±0.008 ^y

Table 4: Changes in Glucose level in kidney of Guppy during control and post-treatment with three different concentration of Cadmium at different periods (15, 30, 45th days) of exposure

Days of Exposure	Control	Exposure Concentration		
		Th 1/10 of LD50	Rd 1/3 of LD50	LD 50
15 Days	3.09±0.008 ^y	4.39±0.008 ^y	4.68±0.008 ^y	4.88±0.008 ^y
30 Days	3.11±0.008 ^y	5.18±0.008 ^y	5.54±0.008 ^y	5.74±0.008 ^y
45 Days	3.48±0.008 ^y	8.32±0.009 ^y	8.47±0.005 ^y	8.83±0.005 ^y

Value expressed in (mg/100mg) (Mean ± SD).

**Fig: 3 Liver****Fig: 4 Kidney****Figure 3 and 4: Changes in Glucose level in liver & kidney of Guppy with three different concentration of Cadmium at different periods (15, 30, 45th days) of exposure.**

RESULT AND DISCUSSION

In the present study, it has been observed that the exposure of sub lethal, 1/3rd & 1/10th concentration of cadmium led to the decrease in the level glycogen and an increase in the glucose level leading to lethargy. The amount of glycogen in different tissues was reduced in treated fish than the control fish. This indicates that there is utilization of a major quantity of the glycogen reserves and retention of less amount of glycogen content in the tissues. Liver has two active sites where storage and metabolism of glycogen takes place. A reduction in the glycogen content in the tissues can be attributed to the toxic effects of cadmium on tissue energy reserves. The increase in glucose noticed in the present study could be attributed to difference in respiration and activity as pointed out by Das & Mukherjee.^[5] The progressive accumulation of plasma glucose reported in this investigation revealed the *Clarias gariepinus* exposed to sub lethal concentration of paraquat became hyperglycemic. There was a drastic decrease in the liver glycogen may be due to acute hypoxia.^[6] These perhaps achieved by rapid glycogenolysis and inhibition of glycogenesis through activation of glycogen phosphorylase and depression of glycogen transferase respectively or though stress induced due to increase in

catecholamines.^[7] Khan & Sharma studied that decrease in glycogen and protein content in liver and kidney of fish *Guppy* when exposed to Cadmium.^[8] Murthy and Devi reported decreased level of protein, glycogen and lipid concentration in liver, and increased level in brain of *Channa punctatus* treated with endosulphan.^[9] Sastry and Siddiqui found alterations in levels of plasma protein, glucose, glycogen and lactate concentrations in *Channa punctatus* exposed to sub lethal concentration of quinolphos.^[10] Khan and Jain reported significant reduction in glycogen level of fish *Lebistes reticulatus* when exposed to higher and lower sub-lethal concentration of cadmium chloride for 15, 30 and 45 days.^[11] Sastry and Siddiqui found alterations in levels of plasma protein, glucose, glycogen and lactate concentrations in *Channa punctatus* exposed to sub lethal concentration of quinolphos. The glycogen reserves in the liver and kidney tissues decreased significantly. The decrease levels of liver and kidney glycogen reserves of *Guppy* in this study may be as a result of cadmium stimulating the activities of enzymes that work in glycogenolysis as supported by Cicik and Engin.^[12] Liver glycogen decreased in *Oncorhynchus mylax* as a result of the activation of glycolytic enzymes via catecholamines under lack of food and hypoxic

condition. Vijayan and Moon.^[13] The reduction in protein content of the fish *Guppy* may be due to increased utilization of protein to meet out the energy demand when the fish is under stress condition. The decreased in glycogen level in liver and kidney of *Guppy* may be due to reduction of glucose-6-phosphatase, due to depression of glucokinase activity and disruption of glucose-6-phosphatase when exposed to Cadmium. A significant decrease in glycogen was observed in both tissues under all three concentrations of cadmium. The variation in distribution suggests difference in metabolic calibers of various tissues.

5. CONCLUSION

It has been observed that the exposure of sub lethal concentration of cadmium led to the decrease in the level of glycogen while an increase in the glucose level leading to lethargy the enzyme activity was inhibited. The increase in the glucose level of the tissue while decrement in tissue glycogen in exposed fish makes it clear that the glycogen reserves are being used to meet the stress caused due to cadmium. A significant decrease in glycogen was observed in both tissues under sublethal concentrations of cadmium. The decreased in glycogen level in liver and kidney of *Guppy* may be due to reduction of glucose-6-phosphatase, due to depression of glucokinase activity and disruption of glucose-6-phosphatase when exposed to Cadmium. It is found that Cadmium disturbs the chemical constituents of the fish which leads to cell damages and finally death of fishes.

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