

Sublethal Toxicity of Cadmium Chloride on Tissue Glycogen Reserves in *Heteropneustes fossilis*

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Abstract: *This study looked at how cadmium chloride (CdCl₂) affects glycogen reserves in Asian stinging catfish (Heteropneustes fossilis) over two months. Fish were exposed to 75 mg/L of cadmium chloride, and researchers measured glycogen in the muscles, gills, liver, heart, and kidney at 30 and 60 days. The numbers tell a clear story: glycogen dropped in every tissue, and the heart, liver, and gills took the hardest hit. After 60 days, muscles went from 7.81 to 6.87 mg/g (a 12% drop), gills from 5.43 to 3.52 mg/g (down 35%), liver from 16.82 to 10.93 mg/g (down 35%), heart from 3.51 to 2.07 mg/g (down 41%), and kidney from 6.87 to 5.42 mg/g (down 21%). The heart always had the lowest glycogen, which means it's especially vulnerable to the stress from cadmium. This sharp loss of glycogen shows that cadmium messes with how the fish use carbohydrates, ramps up the energy they need to detox, and probably causes some real damage to their organs. In short, even at levels that don't kill them outright, cadmium throws the fish's metabolism out of balance. Different organs react in their own way, but the end result is trouble for the fish's health, growth, and overall chance to thrive—bad news for a species that matters in freshwater fisheries.*

Keywords: - Cadmium chloride, sublethal toxicity, glycogen depletion, tissue-specific response, heavy metal pollution

Introduction

In the list of heavy metals such as lead (Pb), mercury (Hg) and cadmium (Cd) are considered to cause public health hazards. Cd is a naturally occurring non-essential heavy metal present at higher concentrations in association with Cd-rich soils, including shales, oceanic and lacustrine sediments, and phosphorites. However, more than 90% of Cd in the surface environment is the result of industrial and agricultural processes (Pan et al., 2010). Burning of fossil fuels and mineral oil,

smelting, mining, alloy processing and industries that use Cd as a dye (CdS: yellow; CdSeO₃: red) in their manufacturing processes (Swarup et al., 2007) are all potential sources of Cd for farmed ruminants, with exposure decreasing with distance from the pollution source (Vos et al., 1990). Cd may also enter into the atmosphere from zinc, lead or copper smelter. It can enter water through disposal of wastes from households or industries. Further usually, air concentrations of Cd of between

0.01 and 0.35 $\mu\text{g}/\text{m}^3$ have been reported (US Department of Health, Education and Welfare, 1966), with the highest concentrations in industrialized cities. Fertilizers often contain some Cd. Cd is also a pollutant in phosphate fertilizers (Järup, 2003), leading to Cd being added to land through normal farming practice (Roberts et al., 2014). The Cd content in phosphate fertilizers varies considerably, depending on source, ranging from 3.6 to 527 mg/kg (Satarug et al., 2003). Sewage sludge is also recognized as an important source of Cd contamination (Patrick, 2003).

Cd is primarily stored in the liver and kidneys, which account for half of the body's total stores of Cd, rest in bone, pancreas, adrenals and placenta (Pope and Rall, 1995). Cd damages the kidney and cause signs of chronic toxicity, including impaired kidney function, poor reproductive capacity, hypertension, tumors and hepatic dysfunction (Pope and Rall, 1995).

Material and Methods

The present study is intended to investigate the toxicity of Cadmium to a freshwater, air-breathing, stinging catfish, *Heteropneustes fossilis* Bloch (Order: Siluriformes; Family: Heteropneustidae). Fishes were collected from river Gomti and water reservoirs in and around Lucknow, U.P. (India) with the help of local fisherman, brought to laboratory (N-26°51'59'', E- 80°56'17'') and acclimatized to laboratory conditions for 15days before the

experiments. Stock solution of Cadmium chloride ($\text{CdCl}_2 \cdot 2\frac{1}{2} \text{H}_2\text{O}$, M.W. = 228.35AR Grade, manufactured by Thomas baker chemicals ltd. Mumbai, India) was prepared by dissolving weighed amount of salt in double distilled water. For toxicity test six aquaria of 50-liter capacity were taken having 30 liters of dechlorinated tap water (Physico-chemical properties, pH = 7.6 ± 0.2 ; Temp. = $26 \pm 20^\circ\text{C}$; Alkalinity = 65 ± 4.5 mg/L; Total Hardness = 265 ± 2.5 mg/L; D. O. = 7.0 ± 0.2 mg/L). Series of three concentrations of Cadmium chloride viz. 25, 50 and 75 mg/l (Toxic range was predetermined by exploratory tests) was prepared by adding calculated amount of stock solution.

The biochemical constituent Glycogen was estimated by standard procedures in 5 tissues viz., Muscle, Gill, Liver, Heart and Kidney of the healthy fish (Control) and of those from the fish exposed to sub-lethal and lethal concentrations of Cadmium chloride (Merck). One-tenth of the lethal concentration was taken as sub-lethal dose and the fish were exposed to sub-lethal dose for a period of 30 and 60 days before sacrifice for the biochemical analysis. The Glucose and Glycogen in tissues were determined by the method of Kemp et al., (1954)

Result

The glycogen level in muscle, gill, liver, heart & kidney were significantly ($P < 0.05$, $P < 0.001$)

decreased after 30 days and 60 days exposure to CdCl₂. The glycogen level decreased from 7.81mg/gm to 7.12mg/gm in muscle, 5.43mg/gm to 4.26mg/gm in gill, 16.82mg/gm

to 11.79mg/gm in liver, 3.51mg/gm to 2.68 mg/gm in heart and 6.87mg/gm to 5.87mg/gm in kidney after 30 days exposure of at 75mg/gm CdCl₂(Table 1).

TABLE -1: Effect of Cadmium Chloride on glycogen (mg/gm wet tissue) of *Heteropneustes fossilis* after 30 days exposure

ORGAN	CONTROL	25mg/l	50mg/l	75mg/l
MUSCLES	7.81 ± 0.13	7.72 ± 0.12	7.48 ± 0.33	7.12 ± 0.32*
GILLS	5.43 ± 0.44	5.18 ± 0.95	4.83 ± 0.31	4.26 ± 0.33*
LIVER	16.82 ± 1.13	15.91 ± 0.14	13.84 ± 0.14	11.79 ± 0.21*
HEART	3.51 ± 0.42	3.26 ± 0.11	3.03 ± 0.37*	2.68 ± 0.30*
KIDNEY	6.87 ± 0.32	6.69 ± 0.13	6.01 ± 1.11	5.87 ± 0.12*

TABLE -2: Effect of Cadmium Chloride on glycogen (mg/gm wet tissue) of *Heteropneustes fossilis* after 60 days exposure

ORGAN	CONTROL	25mg/l	50mg/l	75mg/l
MUSCLES	7.81 ± 0.13	7.68 ± 0.31	7.23 ± 0.27 *	6.87 ± 0.32**
GILLS	5.43 ± 0.44	5.01 ± 0.51*	3.97 ± 0.41**	3.52 ± 0.30**
LIVER	16.82 ± 1.13	14.93 ± 0.72	12.13 ± 0.84*	10.93 ± 0.31**
HEART	3.51 ± 0.42	3.18 ± 0.33	2.56 ± 0.33*	2.07 ± 0.37**
KIDNEY	6.87 ± 0.32	6.57 ± 0.61	5.93 ± 0.81*	5.42 ± 0.12*

After 60 days exposure at 75mg/l CdCl₂ the glycogen level decreased from 7.81mg/gm to 6.87mg/gm in muscles, 5.43mg/gm to 3.52mg/gm in gill, 16.82mg/gm to 10.93mg/gm

in liver, 3.51mg/gm to 2.07mg/gm in heart and 6.87mg/gm to 5.42mg/gm in kidney (Table 2). The minimum glycogen content were observed

in heart after the 30 and 60 days exposure of 75mg/l CdCl₂.

Discussion:

The biochemical composition in the different fish species has been reported in relation to their age, sex, habitat and seasons (Nazrul Islam and Abdul Razaq Joadder, 2005). Seasonal variations in the biochemical composition of fish were reported in *Mugil cephalus* and in *H. fossilis* which correlates our study. Annual correlative changes were reported in some biochemical contents of testes in the catfish *Clarias batrachus* (Singh and Joy, 1999). Investigations on biochemical composition of different fishes were reported in *Macrornathus aculeatus* (Nabi, 1989) and *Cirrhinus mrigala* (Muslemuddin, 1991). The main source of energy reserves in fish are protein and lipid in three-spined-stickle Back, *Gasterosteus aculeatus* L. (Chellappa, 1988). In rainbow trout, *Onchorhynchus mykiss*, an increased use of hepatic carbohydrates is reported to take place during gonadal maturation (Washburn et al., 1990). Several changes in liver and gonadal metabolism were observed during the onset of testicular recrudescence in *Oreochromis mossambicus* (Soengas et al., 1993).

Seasonal variation in the biochemical composition of Koi fish *Anabas testudineus* (Bloch) were reported by Nargis (2006). In present study, there was decrease in glycogen content of all the tissues at sublethal and lethal

concentrations of Cd in dose-dependent manner. The findings are accordance with several researchers those have shown similar effect due to different pollutants (Maruthi and Rao 2000). Depletion of the glycogen content in the liver and muscle was also observed by other workers in fish *Mystus cavasius* exposed to electroplating industrial effluent (Palanisamy et al., 2011). Since carbohydrates serve as the instant energy source during stress so during acute condition blood glucose level increases due to glycogenolysis but reduction can be correlated to utilization of stored glycogen to meet up the energy require or chronic exposure. In liver, glycogen mobilized to glucose whereas in muscle glycogen/glucose served as readily available source of energy.

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