

Impact of Cadmium Chloride (CdCl₂) Exposure on Glucose Levels in Various Tissues of *Heteropneustes fossilis*

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Abstract:

The present study examines the implications of acute and sub-acute exposure to cadmium chloride (CdCl₂) on tissue specific glucose metabolism in muscle, gill, liver, heart, and kidney, after 30 and 60 days. Data represent significant increase in tissue glucose levels, corresponding to the metabolic response caused by cadmium-induced stress. We found out the glucose level after that 24 hour in the liver where the liver had the highest glucose levels, that the liver was going through its detox phase. Our results support a model in which there are systemic metabolic adaptations to cadmium toxicity having broader implications for how the metabolic response to exposure of an environmental toxicant is seen.

Keywords: *Cadmium Chloride, Various Tissues, Glucose, Heteropneustes fossilis*

I. INTRODUCTION

Various natural and anthropogenic activities led to release of heavy metals in aquatic ecosystem creating serious problems to its flora and fauna. Heavy metal pollution is among five major pollution problems of aquatic bodies. Heavy metals have long been recognized as serious pollutant of the aquatic life. Pollution by heavy metals has become a serious environmental as well as public health hazard concern because the construction related into the environment from industrial processes often exceed permissible levels. Due to their bio-accumulative and non-biodegradable properties heavy metals constitute a scare group of aquatic pollutants also. These chemicals accumulate in the tissues of aquatic organisms at concentrations many times higher than concentrations in water may be biomagnified in the food chain to levels the cause physiological impairment of higher trophic levels in human consumers. Cd is non-biodegradable element with still not known biological functions and reported to be a major

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containment of aquatic ecosystems causing adverse effects on aquatic organisms (Hollis et al., 1999, McRae et al., 2018). It is entering into aquatic ecosystem through diverse sources including both natural and anthropogenic activities. The major sources of Cd contamination include electroplating paper PVC plastic pigment & ceramic industries battery, mining and moldering units and many other modern industries (Gupta et al., 2003). Seasonal changes and annual variation in the protein, glycogen and cholesterol content in the ovary and testes were studied in *H. fossilis* by (Hunge and Baile, 2003). Though a lot of work has been carried out on relation to biochemical aspects of ovary, testes and liver, in fishes this study suggests the effects of salinity and correlates the biochemical composition with reproductive cycle.

Material and Methods: The biochemical constituent Glucose 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.

Estimation of the Glucose

The Glucose in tissues were determined by the method of Kemp et al., (1954)

Reagents:

KOH 30%, Anthrone Reagent, Con: Sulphuric Acid, Ethyl Alcohol, Standard glucose (40mg/ml)

Result and Discussion:

Results show that the tissue glucose content increased significantly after 30 and 60 days CdCl₂ exposure in muscle, gill, liver, heart, and kidney tissues. This observation was statistically significant, and p-values < 0.05 and 0.001 represent a high chance that the changes observed were actually due to the exposure as opposed to random fluctuation.

Glucose levels of the muscle, first, changed in the range of 9.16 mg/gm to 12.32 mg/gm after 30 days of exposure to different sub-lethal concentration of CdCl₂. The glucose content in gills ranged from 2.84 to 5.26 mg/gm. Glucose levels of liver 11.34 mg/gm to 13.64 mg/gm Glucose levels in the heart ranged from 5.97 mg/gm to 8.25 mg/gm. The last one, in the kidney, reflected glucose levels from 7.51 to 9.42 mg/g.

Table 1 and Figure 1 provide an overview of all these changes, respectively.

For 60 days of exposure, overall, a 40% glucose intolerance caused by fishing line influx was noted in all tissues with blood sugar levels significantly higher. Glucose content in muscle increased from 9.16 to 14.34 mg/gm. In the gills, it was increased from 2.84 mg/gm to 7.21 mg/gm. The liver had a higher increase with the glucose level going from 11.34mg/gm to 15.04mg/gm at 60 days this increase was 5.95 to 7.87 mg/gm in the liver, 5.97 to 9.11 mg/gm in the heart, and 7.50 to 10.36 mg/gm in the kidney after exposure to 75 mg/l of CdCl₂. The latter are shown in Table 2 and Figure 2. As seen, the liver had the highest glucose levels at both 30 and 60 days of exposure, in a concentration-dependent way, to up to 13.64 mg/gm and 15.04 mg/gm respectively at maximum concentration, CdCl₂.

Increase glucose levels in different tissues on CdCl₂ exposure may be a manifestation of metabolic stress due to cadmium toxicity. Heavy metal such as cadmium can induce instigate redox imbalance, disrupt cellular redox and eventually lead to the development of oxidative stress. The first thing that happens to almost all organisms under this stress is that their glucose metabolism changes. High glucose levels in the blood show up as more of an energy demand to fight the toxic effects of cadmium and to repair injured tissue.

An increase over 60 days from 9.16 mg/gm to 14.34 mg/gm of glucose in muscle tissue indicates that the animal made a substantial metabolic adjustment to ensure that its muscle tissues could function and repair. Muscles are some of the most metabolically active tissues in the human body, and are thus reliant on a constant flow of glucose to keep them functioning at their optimal capacity. The increased glucose levels indicate an increased glycolytic activity in order to keep up with the higher energy demands associated to the stress exerted by cadmium.

Gill is an important osmoregulatory and respiratory organ and we observed significant rise in glucose content from 2.84 mg/gm to 7.21 mg/gm in gill which is an indicative of modulation of ions. The gills are in direct contact with the external environment, representing the first-line protection against waterborne pollutants as CdCl₂. Levels of glucose were found to increase, which can be viewed as a metabolic reaction ensuring adequate energy provision to maintain ion balance and structural stability in response to the cadmium-induced stress.

They found that glucose levels were most prominently increased in the liver tissue, from 3.37 to 15.04 mg/gm after 60 days, functioning at the hub of the detoxification pathways. The liver functions as a metabolic laboratory, detoxifying environmental toxins and synthesizing essential biomolecules. This also means the liver would be working hard metabolically to detoxify cadmium and poorly cope with the

effects of the cadmium when blood glucose is high. This increased glucose content reflects the importance of a liver response for glucose supply and underscores the role of the liver in retaining and mobilizing glucose as necessary for the host's response to toxic stress.

The increase in heart glucose (5.97 mg/gm) to 9.11 mg/gm may allow the heart to maintain some function, apparently an adaptive response to the cadmium stress. The heart needs lots of energy to keep beating, so it relies on a very steady supply to keep ticking over. Increased levels of glucose are indicative of a high glycolytic flux due to the energy demands of the heart under toxic conditions.

The glucose assay also revealed that glucose level increased in kidney, which involved in excretion and maintaining fluid and electrolyte balance from 7.50 mg/gm to 10.36 mg/gm. They are responsible for filtering blood and this brings them in direct contact with all kinds of toxins in the blood stream, including cadmium. The increase in glucose content in the kidney suggests a metabolic adjustment to fuel the augmented energy needs of detoxification and excretion under cadmium stress. This study demonstrated that exposure to cadmium chloride causes the significant changes in glucose metabolism in different tissues. The trend in glucose levels is consistent with a systemic metabolic response by the host to relieve the toxic effects caused by cadmium. This is achieved through increased glycolytic pathway by increased glucose utilization to support the greater demand for energy in stressed and or metabolically active tissues.

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

ORGAN	CONTROL	25mg/l	50mg/l	75mg/l
MUSCLES	9.16 ±0.54	9.30±0.52	10.10 ±0.31*	12.32 ±0.77**
GILLS	2.84 ±0.54	3.21 ±0.51	4.19 ± 0.14*	5.26 ±0.12**
LIVER	11.34 ±0.44	12.04 ±0.43	12.41 ±0.58*	13.64 ±0.51**
HEART	5.97 ±0.38	6.45 ±0.30	7.64 ±0.72*	8.25 ±0.35**
KIDNEY	7.51 ±0.32	7.92 ±0.31	8.13 ±0.18*	9.42 ±0.31**

*Significant at P<0.05level, ** Significant at P<0.001 level

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

ORGAN	CONTROL	25mg/l	50mg/l	75mg/l
MUSCLES	9.16 ± 0.54	9.83 ± 0.54	11.44 ± 1.84*	14.34 ± 1.17**
GILLS	2.84 ± 0.54	3.67 ± 0.31	4.55 ± 0.81*	7.21 ± 0.88**
LIVER	11.34 ± 0.44	12.62 ± 0.64	13.51 ± 0.62*	15.04 ± 0.86**
HEART	5.97 ± 0.38	7.05 ± 0.32	8.09 ± 0.82*	9.11 ± 0.35**
KIDNEY	7.51 ± 0.32	8.53 ± 0.42	9.12 ± 0.91*	10.36 ± 0.36**

*Significant at P<0.05 level, ** Significant at P<0.001 level

Data shown in Tables 1 and 2 together provide an extensive understanding about CdCl₂-exposure-induced alterations in glucose level in different tissues. Of all the glucose values altered, as the main organ of detoxification, the liver attributed the greatest difference in glucose levels increased, demonstrating a pivotal role of the liver, for the control of cadmium toxicity. Changes in the glucose level of muscle, gills, heart and kidney also implied their consistent metabolic adaptations to the stress induced by cadmium. Freshwater fish have high food value and also are economically important. Biochemical studies on fish tissues such as muscles, gills, liver, heart and kidney have drawn the attention of several researchers, because tissues are the major source of glucose, glycogen, protein, lipid and cholesterol, have a high calorific value (Joshi et al., 1979).

In present study we have noted the increased glucose levels in muscles, gills, liver, heart and kidney tissues by the doses of CdCl₂. The varying levels of blood glucose are indicative of abnormal carbohydrate metabolism and possibly the result of impaired hormonal control (Andersson et al., 1988). Heavy metal exposure alters carbohydrate metabolism in fish (*Cyprinion watsoni*) and thus resulted in the depletion of energy. This depletion of energy might be the cause of behavioral impairments as we noted in our experimental fish, *H. fossilis*. Unquestionably the generally results of the present study are in conformity with the findings of the above researchers.

Finally, this study shows that cadmium chloride exposure leads to marked changes in the glucose metabolism of different tissues. The elevation of glucose suggests a metabolic change to meet the energy demands of cadmium toxicity. Our findings provide evidence that glucose metabolism might play essential roles in the physiological response to heavy metal stress and raise new questions on possible

connection between environmental pollutants and metabolic health.

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