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ORIGINAL ARTICLE

EFFECT OF ARSENIC ON GLYCOGEN AND GLUCOSE LEVEL IN ARIOUS TISSUES OF FRESH WATER FISH, LABEO ROHITA

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ABSTRACT

The present study is aimed to investigate the effect of arsenic in gill, liver and kidney tissues of fresh water fish, Labeo rohita. The fishes were exposed to sublethal concentration of arsenic on glycogen and glucose content in fresh water fish, Labeo rohita. The present stydy shows the level of glycogen was decreased and glucose was decreased due to arsenic

Keywords: Arsenic ,Glycogen, Glucose, *Labeo rohita*, Gill, Liver Kidney

1.INTRODUCTION

The global population increase and industrial development have led to an increase in the contamination of the environment by metal (Franca *et al*., 2005). This has increased the concerns about the accumulation of metals, in biota and ultimately humans (Gibbs and Miskewicz, 1995). Metals tend to accumulate. In biota (Forstner and Wittmann, 1979; Daka *et al*., 2003), but may be released under certain physiological conditions, moving up through the food chain [Bryan and Langston, 1992]. Toxicological and environmental studies have prompted interest in the toxic metals in the food. The ingestion of food is an obvious means of exposure to metals, not only because many metals are natural components of food stuffs but also because of environmental contamination and contamination during processing. The toxic nature of certain metals and the major contribution mate to the total body bur ten of metal by food consumption are well documented (Hellou *et al*, 1992; Sharif *et al*., 1993.

The heavy metal contamination of aquatic system investigators both in the developed an developing countries of the world [Faromobi *et al*., 2007]. Many industrial and agricultural processes have contributed to the contamination of fresh water system there by causing water system there by causing adverse effects on aquatic biota and human health (Wang, 2002; Dautremepuits *et al*., 2004). Heavy metals can accumulated in the tissues of aquatic animals and as such tissue concentration of heavy metal can he of public health concern to animals (Kalay *et al*., 1999; Asharf, 2005).The accumulation of heavy metal in an aquatic environment has direct consequence to man and ecosystem [Merian, 1991]. Howerer, it is of interest to note that metals are toxic when supplied in trace levels. This heavy metal contamination in aquatic environment is of critical concern due to the toxicity of metal and their accumulation aquatic habitats. [Tam and Wong, 1995].

Arsenic is being a potent environmental toxic agent, leads to development of various hazardous effects on human health. Arsenic is considered as a human carcinogen [Wang and Huang, 1994]. Arsenic contamination. In natural water is a world wide problem and has become a challenge for world scientist. It has been reported in recent years form several parts of world [Chappell *et al*., 1997; Frost *et al*., 1993; Moncure *et al*., 1992]

Fishes are sensitive to contamination of waters and the pollutants may damage certain physiological and biochemical processes when they enter the organs of fishes [Tulasi *et al*., 1992]. Fishes are being at the higher level of the food chain accumulate large quantities of thesis semeiotics and the accumulation depends on the intake and the elimination from the body [Karadede *et al*., 2004].Fishes are being used for the assessment of the quality environment and as such can serve as bio-indicator of environmental pollution [Lopes *et al*., 2001]. Fish is used extensively for environmental monitoring [Lanfranchi *et al*., 2006], because they uptake contaminates directly form water. Generally the ability of fish to metabolize toxicants is moderate; there fore, contaminant

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loading in fish is well reflective of the state pollution in surrounding environments [Fisk *et al*., 1998].

The gills are the first target organs in the heavy metal accumulation because they are directly in contact with water [Dubale and Shah, 1979]. The gills, which serve as the primary uptake site in the fish for trace metals, represent the most important targets when exposed to elevated levels of ambient metals [New man and Jogoe, 1997]. Liver is one of the most multi faceted and active organ in higher animals. In a vertebrate body, the liver is most important target organ as it is the chief metabolic and detoxification center [Bhattacharya and Mukherjee, 1976]. The kidney is the most sensitive organ with respect to overt toxicity following exposure to heavy metal. The kidney is the main excretory organ, is mainly concerned with removar of waste materials [Lawrence and Mc Cabe, 2002].

2.MATERIALS AND METHODS

Procurement of experimental animal

The fresh water fish, *Labeo rohita* were collected from the fish farm located in Puthur,Nagai District, 15 Km away from the Uinversity campus. This fishes were brought to the laboratory and transferred to the rectangular fibre glass tanks (100X175cm) of 500liters capacity containing chlorine free aerated well water.

Acclimatization of animals

The fresh water fish, *Labeo rohita* were acclimatized for a minimum period of 15 days in the laboratory conditions at room temperature (28±1ºC) before subjecting them for screening test. These fingerlings were fed with artificial food pellets on alternative days and the water renewed every 24 hours. The tanks were rinsed with potassium permanganate or acroflavine (2mg/l) to prevent fungal attack. The fresh water fish, *Labeo rohita* were critically screened for the signs of disease, stress,physical damage and mortality. The injured, severely diseased, abnormal and dead fishes were discarded. The feeding was discontinued 24 hours before the beginning of the experiment to reduce the excretory products in the test trough as suggested by Arrora et al., (1972). During the acclimatization, the fishes were reared in tank until there was less than 10 percent mortality in 4 days perior to the beginning of the test as suggested by Anderson(1977). The water in the experimental trough was changed daily and also aeration was stopped to avoid the possible oxidation of the toxicants.

Experimental design

The toxicant exposure was done by 24 hour or renewal bioassay system. For analysis sublethal toxicity, 2 groups of 10 fish each were exposed separately and arsenic trioxide (2.73ppm : 10 % 96 hours LC_{50}). Solution prepared in well water. The experimental medium was prepared by dissolving cadmium chloride at 6 ppm having dissolved oxygen 5.8 ppm, PH7.4, water hardness 30.3mg/l (APHA *et al*., 1992) and water temperature 28± 2 C. Each group was exposed to 50 l of the experimental medium. Parallel groups of 10 fish each were kept in separate aquaric containing 50 l of well water as control. Feeding was allowed in the experimental as well as control groups every day for a period of 3 hours. Before the reneval of the medium through out the tenature of the experimental.

Estimation of LC_{50} value

Perior to the commencement of the experiment, 96 hr medium lethal concentration as (96 hr LC_{50}) of mercuric chloride for *Oreochoromis mossambicus* was estimated (Hamilton *et al* 1977). And 24 hrs renewal bioassay system and was found

BIOCHEMICAL STUDIES

After experiment, the fish each from the respective experimental as well as control groups were sacrificed. The gills, liver and kidney were isolated from the fish and used for various study.

Estimation of glycogen and glucose

Kemp and Kits van Heijningen (1954) was employed for the quantitative estimation of glycogen and glucose. The tissues were isolated and homogenized in 5.0 ml of 80% methanol and centrifuged at 3,000 rpm for 15 minutes. The supernatant containing free glucose was decanted into a calibrated test tube. The residue was set apart for the quantitative estimation of glycogen.

Estimation of Glycogen

The residue left after methanol extraction was homogenised in 5.0 ml of deprotenizing solution (5-0 ml of TCA and 100 mg of $AgS0_4$ in 100 ml of distilled water) and heated at 100° C over a water bath for 15 minutes. The mixture was cooled and made up to 5.0 ml with deproteinizing solution once again and later centrifuged at 2,000 rpm for 10 minutes. The clear supernatant was collected for the estimation of glycogen.

Estimation of glucose

To the decanted solution approximately 10.0 mg of activated animal charcoal powder was added. The methanol was allowed to evaporate by warming the solution over a water bath for 30 minutes. Deproteinizing solution (100 gm. of TCA in 100ml of distilled water) was added to the residual aqueous solution to bring the total volume to 5.0 ml. The suspension was centrifuged at 2,000 rpm for 15 minutes and the clear supernatant was used for the estimation of free glucose.

Quantitative estimation of glycogen and glucose

1.0 ml of the respective sample was taken in a separate test tube and 3.0 ml of concentrated sulfuric acid was added to it. The mixture was heated in a boiling water bath for 6.0 minutes and subsequently cooled in running tap water. The intensity of the colour developed was measured in a UV. Spectrophotometer against the reagent blanks (3.0 ml of concentration sulfuric acid) at 520 nm. The quantitative of glucose and glycogen present in the respective sample

s were read form the standard graph drawn previously form known quantities of the sample. The glucose and glycogen values are expressed as mg/ g wet weight of tissues.

Statistical analysis

Statistical significance was evaluated by using ANOVA followed by Duncan Multible Range Test (DMRT) Duncan (1957).

3.RESULTS

Level of glycogen in gill tissue

The level of glycogen was 5.11 ±1.11 mg/g well wt of tissue I in the control gill tissue. At sub lethal concentration of arsenic, the gill tissue showed the decreased trend of glycogen (2.81±1.81mg/g wet wt. of tissue). The percent change over the control was –45.00(Table1).

Level of glycogen in liver tissue.

In the normal liver tissue, the level of glycogen content was 9.63±1.35. When the fish exposed to arsenic, the level of glycogen content was decreased upto 4.31±1.62 g/g wet wt. of tissues. The percent change over control was – 55.08(Table1).

Level of glycogen in kidney tissue

The level of glycogen present in the kidney tissue of normal fish was 6.72±1.81 mg/g wet wt of tissue. The level of glycogen was decreased upto 3.26±0.81 mg/g wet wt. of tissue when the fish exposed with sub lethal concentration of arsenic. The percent change over control was – 52.08(Table1).

Table 1Level of glycogen in the selected tissue of fresh water fish *Labeo rohita* exposed with sub-lethal concentration of arsenic

Tissues	Control	21 days	% COC
Gill	5.11±1.62	2.81±1.81	-45.00
Liver	9.63±1.67	4.31±1.62	-55.24
Kidney	6.72±1.81	3.22±1.71	-52.08

Mean ± S.D. of six individual observations;* Significance ($p<0.05$) Group I compared with group II Values are expressed as (mg/g wet wt. of tissue)

Level of glucose in gill tissue

The level of glucose was 3.40 ±1.41 mg/g well wt of tissue in the control gill tissue. At sub lethal concentration of arsenic, the gill tissue showed the increased trend of glucose (4.88±1.12 mg/g wet wt. of tissue). The percent change over the control was 43.52 (Table 2).

Level of glucose in liver tissue.

In the normal liver tissue, the level of glucose content was 8.30±1.26. When the fish exposed to arsenic, the level of glucose content was increased upto 12.16±1.18 g/g wet wt. of tissues. The percent change over control was 46.50(Table 2).

Level of glucose in kidney tissue

The level of glucose present in the kidney tissue of normal fish was 4.71±1.62 mg/g wet wt of tissue. The level of glucose was increased upto 6.62±1.87 mg/g wet wt. of tissue when the fish exposed with sub lethal concentration of arsenic. The percent change over control was 40.55(Table 2).

Table 2 **Level of glucose in the selected tissue of fresh water fish *Labeo rohita* exposed with sub-lethal concentration of arsenic**

Tissues	Control	21 days	% COC
Gill	3.40±1.41	4.88±1.01	43.52
Liver	8.30±1.57	12.16±1.17	46.50
Kidney	4.71±1.62	6.62±1.87	40.55

Mean ± S.D. of six individual observations;* Significance ($p<0.05$) Group I compared with group II Values are expressed as (mg/g wet wt. of tissue)

4. DISCUSSION

The heavy metals are known to elicit changes in the biochemical constituents of fish there by altering the metabolic pathway [Sarkar and Medda, 1993]. Toxic exposure of organixms interferes with organ integrity at the biochemical level and unlimitedly gives rise to affect at the individual levels [Smolders *et al*., 2002]. The heavy metal and pesticides are found to influence the biochemical composition of fishes [Shakoori *et al*., 1997; Kaviraj *et al*., 2000].

Carbohydrates are an important sources of energy required to various metabolic activities of the living organisms, the energy being derived as a result of oxidation. They are mainly in the form polysaccharides and disaccharides, which are hydrolyzed into monsaccharides by enzymes of digestive tract. At the time of physiological demand for energy, glucose is oxidized to co_2, water and energy. In the absentee of physiological demand for energy, excess glucose may be converted to glycogen and is stored in the Linver, and muscle and other tissues [Saskin, 1941].

Glycogen is an important role in the carbohydrate metabolism. It is one of the sensitive biochemical indicators which reflects changes in the normal activity of various functional system [Metalev *et al*.,1983]. Glucose is an important biochemical substances which fives the immediate energy to organisms. It act as a sensitive biochemical indicator of the environmental stress [Meteleve *et al*., 1983].

The present study showed the level of glycogen decreased and glucose increased in the brain, gill liver and kidney tissue of *Labeo rohita* exposed to arsenic trioxide. This results indicates and extensive utilization of energy stores. Perhaps this stopped up titillation is to meet the extra demands of energy necessitated by the quick and brisk movement which shows in the behavioral pattern of the fish during stress. The reduction in stored glycogen content in the respective tissues

of fish treated with arsenic metals. The liberated glucose mobilized form livers glycogen are transported to other organs through blood to meet the energy requirements necessitated by the allele rated movements of the fish under the toxicity or arsenic.

Colley *et al*., (2001) reported that the glycogen content reduced in the liver tissue of *Oncorhynchus mykiss* exposed to dietary effluent. Radhakrishnaiah *et al*., (1992) have reported that glycogen content decreased in muscle are liver tissue of Labeo rohita exposed to copper. Alkakhera *et al*, (2005) have reported the depletion of glycogen reserved of liver in at razine treated animal.

Patil and Dhande, (2000) reported that a fall in glycogen in the tishes exposed to heavy metal. Black *et al*., (1960) reported that reeducation in glycogen level is thought to be the result of detoxification of act vive moieties of muscular exercise on hiver glycogen energy reserves in fish. Dezwann and Zendee, (1989) have observed the a reducation in tissue glycogen content due to decrease in synthesis or break down as consequence of toxic stress. Samuel and Satry, (1989) reported the level of glycogen decreased in *Channa punctatus* exposed to moncrotophos. Bakthavathsalam and Srinivasa Reddy (1985) have reported the similar fluctuation in *Anabas testudineus* exposed to disyston.
Ghosh (1986) has reported that the loss of glycogen is liver and musgle of tish might be attributed to the toxicogenic effects of the fish, *Channa punctatus* Karuppasamy (1999) observed the glycogen level of liver, muscle, and gill shows a decrease in *Channa punctatus* exposed to phenyl mercuric acetate. Sheela and Muniandy (1992) have suggested that the decrease in muscle glycogen in channan punctatus might be due to increased glycogholysis. Mary Chandravthy and Reddy, (1996) have observed a drop in glycogen content in kidney and intestine of *Anabas scandens* exposed to lead nitrate. Verma and Tonk (1983) have stated the decreased glycogen content in the liver and muscle of fish exposed to mercury.

In the present study, the level of glucose increased in the drain, gill, liver and kidney tissue to *Labeo robita* exposed arsenic. This result indicates that the glycogenolysis take place in the liver, where by the reserved glycogen is being slowly converted into glucose. Koundinya and Ramamurthi (1978) have reported the increase in glucose level in *Saratherodon mossambicus* exposed to pesticide. Hinston *et al*., (1973) have reported that maximum glycogen depletion corresponds to dramatic increase in glucose level in the fish *Channa punctatus* exposed to pollutants. They suggest that it might be due to some of the hepatic glycogen gaffing converted to glucose via the intermediate glucose-6 phosphate getting and entering the circulation.

The present study suggests that glycogen is being a ready source of energy, reduction in glycogen is probably due to more rapid breakdown, when releases glucose into circulatory system to meet the increased energy requirement in a stressful condition. Bakthavathsclam and Srinivasa Reddy (1985) noticed similar increase of glucese in *Anabas testudineus* exposed to disyston. Chowdhury (2004) reported the similar result in *Orxorhychus mykiss* exposed to cadmium. Radhakrishnaiah *et al*., (1992) reported that the level of glucose increased in the blood of *Labeo rohita* exposed to copper.

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