



Effect of Mercury in Ambient Water on Blood Glucose and some Biological Parameters of *Labeo Rohita* (Hamilton)

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ABSTRACT

To evaluate the relative effects of inorganic (HgCl_2) and organic (CH_3HgCl) mercury pollution on *Labeo rohita*, a series of experiments for a period of 75 days were carried out. Fishes were exposed to 0.005 and 0.01 ppm inorganic and organic mercury for a period of 60 days followed by 15 days of recovery by transferring the fishes to mercury free water. Effects on general behaviour, morphology, growth and mortality have been recorded. Biochemical changes on blood glucose level and glycogen level in liver and muscle were analysed. In general increased opercula movement, abnormal swimming, retarded growth, haemorrhage was far greater in fishes exposed to inorganic mercury except that excessive mucous secretion was greater in the later. Initial hyperglycaemia in the peripheral blood followed by subsequent hypoglycaemia was recorded.

Keywords- Mercury, *Labeo rohita*

INTRODUCTION

The increasing use of mercury based pesticides in agriculture; public health and intensive mining activity have increased the scope of disruption of ecological balances. These effluents discharged into fresh water resources affect aquatic fauna. Fish *Labeo rohita* constitute an important food. Many workers have studied that haematological parameters of fish are used as indicator of their physiological state and their study has become widespread in the control of pathogens and manipulation of stress in fish. Literature survey shows the influence of many factors like heavy metals (Ramalingam et al., 2000) pesticides (Mishra et al. 2002) and age, stress due to industrial waste, handling (Chatterjee, et al., 2004) etc. Yoshitomi *et al.* (1999) concluded that Cd contamination had morphological effect in the form of scale deformation in common carp, *Cyprinus carpio*. Sessa Srinivas and Rao (1999) study of effect of 96 h LC50 concentration of hexavalent chromium (39.40 mg/l) on the gills of *Labeo rohita* and revealed damage, fusion, bulging at distal parts and necrosis of secondary gill lamellae and atrophy of central axis. Rani (2000, 2015) observed congestion of primary rachis, bulging of primary and secondary gill lamellae, hypertrophy and hyperplasia of interlamellar cells, atrophy of several of secondary gill lamellae and necrosis in the gills of *Oreochromis mossambicus* with reference to Cd toxicity. Toxicity induced damage to gill surface is further corroborated from various other studied. Hypertrophy and thinning of epithelial cells in gill structure due to reaction to the heavy metals were adaptive responses to enhance oxygen diffusion during stress, whereas secretion

of mucous was to prevent the entry of toxicant through gill surface.

In the present work observations on general and feeding behaviour, growth, mortality, morphological changes biochemical studies on tissue glycogen (liver and muscle) and blood glucose have been attempted.

MATERIAL AND METHODS

Water quality parameters were measured using APHA (American Public Health Association). Standard methods for the examination of water and waste water (APHA, AWWA, WPCF, 1998).

Fry of *Labeo rohita* were brought from a nearby nursery pond and stocked in cement tank. They were acclimatised for 15 days in plastic pools (91cm diameter and 91 cm height), pelleted diet prepared by wheat bran and oil cake.

EXPERIMENTAL PROCEDURES

Experiment was set at fingerlings stage .On the basis of 96hrs Lc50 value for fingerlings of *Labeo rohita* (0.018 mg/l for mercury chloride) and 0.068mg/l for methyl mercury chloride four subs lethal concentrations were tested. Fingerlings of *Labeo rohita* were divided into five groups of 60 fishes in each (av. weight 25.55 ± 0.65 gm, average length 125 ± 0.38 mm).

- Group 1: Control (water free from mercury pollution)
- Group 2: 0.005 mg/l mercury chloride.
- Group 3: 0.01 mg/l mercury chloride.
- Group 4 : 0.005 mg/l methyl mercury chloride.

- Group 5 : 0.01 mg/l methyl mercury chloride.

Fishes were exposed in these groups for 60 days followed by 15 days recovery period (water free from pollution). Physico-chemical characteristics of the water during experimental period were recorded using standard methods (APHA, 1998).

Observations were taken at intervals of 7,15,30,60 & 75 days.

RESULTS

(1). The values of five important Physico-chemical characteristics of the water during experimental period were recorded and average value ranges are as follows:-

S.N.	Physico-chemical characteristics of the water	Average value ranges
01	Dissolved Oxygen	6.4-7.2 mg/l,
02	Ph	7.4 to 7.6
03	Temperature of water	18.5 degree C to 26 degree C.
04	Total alkalinity	139 to 147 mg/l.
05	Total hardness	216 to 225 mg/l.

(2). In behaviour, swimming movement increased and morphological changes were also observed as fishes were transferred to test concentrations. Mucous secretion increased after 7 days in G3 whereas in G4 and G5 secretion was relatively less.

The results of blood glucose level are presented in table giving data of key significance is given in Table1.

Table1: Blood glucose level in % increased (+) or decrease (-) as compared to control group G1

Groups	15 Days	45 days	60 Days	75Days (R)*
G2 (0.005mc)	+6.95	+3.07	-2.59	+6.40
G3 (0.01 mc)	+8.02	+2.56	-4.88	+5.12
G4 (0.005mmc)	+9.02	-2.30	-5.91	+3.00
G5 (0.01 mmc)	+11.80	-7.17	-10.0	+2.82

(R)*= Recovery period

From table it is evident that both organic and inorganic mercury pollution induced an initial hyperglycaemia. This continued for the first fortnight in all experimental groups but in the next 15 days a decreasing trend was observed in all except G2 (0.005mg/l mc) where the increase was up to a month. By 60th day blood glucose level was significantly ($P < 0.05$) below the control level in G3, G4 and G5. Though there was a decrease in G2 but it was not statistically significant.

Restoration to normal water conditions brought about a surprising rapid recovery bringing the value not only to the control level but the increase was more or less at a level seen after 15 days of treatment.

Changes in glycogen level both in liver and muscle are presented in Table 2 as below:

Table 2: Glycogen Content Depletion %

Group	Tissues	15 Days	60 Days	75 Days (R)*
G2 (0.005 mc)	Liver	4.12	21.72	19.53
	Muscle	12.14	25.00	21.66
G3 (0.01 mc)	Liver	11.19	27.57	23.08
	Muscle	16.79	36.67	26.67
G4 (0.005mmc)	Liver	20.04	28.90	27.73
	Muscle	20.71	46.67	33.33
G5 (0.01 mmc)	Liver	27.97	43.92	36.59
	Muscle	23.21	54.66	36.66

(R)*= Recovery

It is clear from the data that there is continuous and consistent depletion in glycogen content in liver and muscle during the exposure to the mercury at all levels, although greater depletion is observed with methyl mercury chloride as compared to mercury chloride.

Restoration of the fish to normal mercury free water after 60 days exposure did reverse contents of liver and muscle remained much below control level.

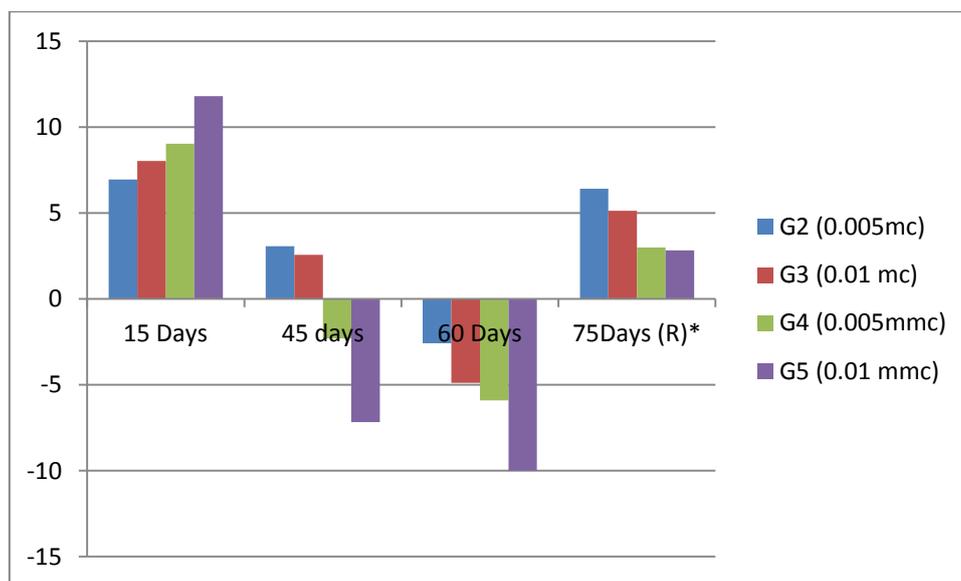


Figure – 1. Bar diagram showing Blood glucose level in % increased (+) or decrease (-) as compared to control group G1.

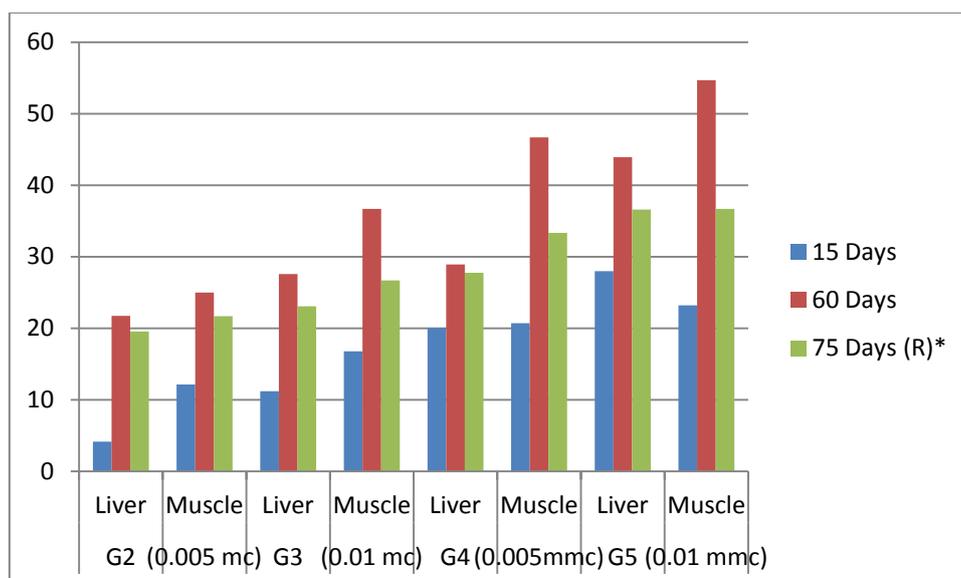


Figure - 2. Bar diagram showing Glycogen Content Depletion %

DISCUSSION

Earlier studies of different pollution in fishes recorded behaviour changes, biochemical changes and increased mucous secretion (Kamrav, 2002 and Khangaroot, 2002) on *Channa punctatus*. More or less similar to those recorded above were recorded in the present study on *Labeo rohita*. In the present work, exposure to mercury pollution followed by recovery treatment resulted in fruitful invention.

In present investigation, intensity of mucous secretion was found to be greater in fishes exposed to inorganic concentration of mercury. In present work, exposure to mercury pollution followed by recovery treatment is fruitful invention.

The initial hyperglycaemia and subsequent hypoglycaemia can be explained on the basis that the environmental stress induced by mercury pollution increase metabolic rate, thus resulting in rapid glycogenolysis such that reserve glycogen is metabolised resulting in hypoglycaemia in the blood.

This is further evidenced by consistent depletion in glycogen recorded. However with prolonged exposure to pollutant stress, accompanied by subnormal feeding and use of energy results in hypoglycaemia in peripheral blood. Hyperglycaemia and hypoglycaemia has been reported by several workers as common response to pollution of different kinds such as pesticides (Aditya, 2000, Paulose and Maheshwari, 2006), other heavy metals (Kaviraj & Korner, 1982; Jana & Bandyopadhyay, 1987).

Mercury intoxication also caused pronounced clinical symptoms in the grass carp. The nervous symptoms included nervousness, dullness, nudge, yawn and circling. Oliveira *et al.* (1996) reported that the nerves such as the optic, showed disorganized disposition of axons and mainly disruption and dissociation of myelin sheaths, leading to a decrease in mortality and increased in-coordination. Behavioral disturbances such as off feed and restlessness were also observed. Ribeiro *et al.* (1995) described that

olfactory organs were affected in mercury intoxication, which changed the normal behavior of the fish. These findings are in line with the present investigations.

However, most of the experiments were limited to few hours whereas present investigation is for longer period and has been tried to stimulate conditions which are likely to occur in nature in large bodies of water. Hence, both the experimental duration and subsequent recovery treatment acquire significant practical value.

REFERENCES

1. Aditya, A. K. and Chattopadhyay, S (2000). J. Environ. Biol., 21(1):55-57.
2. APHA (American Public Health Association). Standard methods for the examination of water and waste water. APHA, AWWA, WPCF, 16Ed, New York, 1998.
3. Chatterjee N, Pal AK, Manush SM, Das T, Mukherjee SC. 2004. Thermal tolerance and metabolic status of *Labeo rohita* and *Cyprinus carpio* early fingerlings acclimated to three different temperatures. J Therm. Biol. 29:265-270.
4. Jana, S. R and Bandyopadhyay. N (1987). Ecol., 5: (3), 488-493.
5. Kamrav, R.K. and Jain , S (2002) Nat. Semi. Environ. Poll. And Fish. Mana. 8th-10th Aug. 2002, Ujjain, M.P.
6. Kaviraj, A. and Konar , S.K. (1982) Geobios., 9 : (3) , 97-100.
7. Khangarot , B.S. and Prakash , V. (2002), 22nd Nat. Symp. On Biodi. And Reso. Manag., Lucknow.
8. Misra SM, Borana K, Pani S, Bajpai A, Bajpai AK. 2002. Assessment of heavy metal concentration in grass carp (*Ctenopharyngodon idella*). Polln. Res 21(1):69-71.
9. Oliveira, R. C. A. and R. F. Torres, 1995. Acute effects of evaluation of HgCl₂ on epidermis of *Trichomycterus brasiliensis*. Ecotoxicol. Environ. Saf., 32(3): 260-266.
10. Paulose P. V. and Kamlesh Maheshwari, 2006. Hepatocyte Damage in Indian Major Carp, *Labeo rohita* with Respect to Accumulation and Elimination of Mercury. *Asian J. Exp. Sci.*, Vol. 20, No. 2, 2006, 369-374
11. Ramalingam V, Vimaladevi V, Narmadaraji, Prabakaran, P. 2000. Effect of lead on haematological and biochemical changes in freshwater fish, *Cirrhinus mrigala*. Poll. Res 19(1):81-84.
12. Rani U. Cadmium induced bioaccumulation in tissue of freshwater teleost *Oreochromis mossambicus*. Ann. N.Y. Academy 2000; 1(919):318-320.
13. Rani, S., RK Gupta, Manju Rani. 2015. Heavy Metal Induced Toxicity in Fish with Special Reference to Zinc and Cadmium. International Journal of Fisheries and Aquatic Studies, 3(2): 118-123.
14. Ribeiro, C.A., L.M. Fernandes, C.S. Carvalho, R.I. Cardoso and N.M. Turcatti, 1995. Acute effects of mercuric chloride on the olfactory epithelium of *Trichomycterus brasiliensis*. Ecotoxicol. Environ. Saf., 31(2): 104-109.

15. Sessa SV, Rao BM. 1999. Chromium induced histological alteration in the gill of the fresh water teleost fish *Labeorohita* (Ham). Indian J Comp. Animal Physiol. 17(1):31-33.

induced scale deformation in carp *Cyprinus carpio*. Bull. Environ. Contam. Toxicol.60(4):639-644.

16. Yoshitomi T, Koyanra J, Lida A, Okamoto N, Ibeda Y. 1998. Cadmium