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Protection by zinc against mercury toxicity in the intestine of a Catfish-*Heteropneustes fossilis*-A Biochemical study

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Abstract

Present study deals with the investigation of toxic effects of Mercury and zinc and the role of zinc in the simultaneous treatment of mercury and zinc, in the intestine of a catfish *H. fossilis*. Biochemical studies had shown decrease in glucose and protein level and increase in alkaline phosphatase due to Hg (0.0l mg/l) treatment for 30 days. When treated with Hg and Zn simultaneously, values for all these parameters were comparable to that of control group, suggesting protective role of Zn against Hg toxicity.

Keyword:- *Mercuric chloride, Zinc sulphate, Intestine, Toxi*

Introduction

Environmental pollution due to heavy metals as a result of rapid industrialization has been reported in different parts of globe including India (Ansari *et al*., 1991; Long *et al.*, 1991; Adrienne and Resmissan, 1998; Govil *et al.,* 1999). The toxicity of mercury was known as early as 16th century and it has been fovnd highly toxic to both humans and animals (Clarkson, 1997). Mercury is widely used in electrical apparatus, chlorine industry, caustic soda and caustic potash industry, chloro-alkali industry, in ayurvedic medicines and also in dentistry (Margarat *et al.*, 2001

Accumulation of mercury in different tissues in various fishes has been reported (Dhanekar *et al.*, 1987; Mason *et al.*, 2000; Lima *et al.*, 2005). Hg is corrosive to the intestinal tract and can damage liver, kidney, if taken in sufficient amount (Gold water, 1971; Hommond, 1971).

Protection against heavy metal toxicity by herbal compound (Geed, 1992; Kothari *et al.*, 1999), essential metal (Bhoraskar and Kothari, 1993; Chen *et al.*, 2001) antioxidants (Potdar, 2007) has been reported. Zn is an essential metal and its pretreatment is known to provide protection against Cadmium (Peter, 1984). With this view in mind, present study was undertaken to assess protective role of zinc against mercury toxicity in the intestine of *Heteropneustes fossilis*.

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Materials and Method

Living and healthy specimens of *H.fossilis* were purchased from local market of Indore. Fish were acclimatized to laboratory conditions for 7 days. Analytical grade mercury chloride (BDH) and zinc sulphate (BDH) were used. 96 hrs LC_{50} for mercury chloride and zinc was found to be 0.5 mg/l and 600 mg/l respectively. Fishes were divided into four groups. Group 1st served as control group. Details of experimental groups are given in Table-1.

Table-1: Experimental groups of Fish

Group No.	Treatment
I	Control (without Poison)
II	Exposed to 0.01 mg/l $HgCl_2$
III	Exposed to 10 mg/l $ZnSO_4$
IV	Exposed to 0.01 mg/l $HgCl_2$ + 10 mg/l $ZnSO_4$

The duration of experiment was 30 days. The water of all aquariam was changed every 4th day and heavy metal salts were introduced into 2nd 3rd & 4th groups immediately after the water was renewed. Chopped prawns were given daily at a fixed time. No artificial aeration was done during experiment. Fishes from each group tissue was used for assaying level of total

protein (Lowery *et al.*, 1951), total glucose (Trinder, 1969) and alkaline phosphatase (ALP) activity (Wooton, 1964). Student "t" test was used to determine statistical significance of protein, glucose and ALP.

Results and Discussion

During this study reduced protein level was recorded due to mercury as compared to the control group in the intestine of *H. fossilis*. Zn alone enhanced the protein level, while Hg and Zn in combination maintained protein level near normal level (Table-2 Fig. 1). Depletion in protein content under the mercury stress has been reported earlier (Ramalingam and Ramalingam, 1982; Sharma, 1997) finding of this study are in accordance with the earlier reports.

Table-2: Protein concentration (mg/ml) in intestine of *H. fossilis*

Groups	Organ
	Intestine
I	7.13±0.41
II	3.98±0.20[a]
III	8.91±0.43[c]
IV	8.01±0.81[x]

Note: Data are means ±SEM. (n=7); x, p <0.001 as compared to the respective values of Hg group; a, p <0.001 and c, p <0.05 as compared to the respective control values; NS= Non significant I Vs III

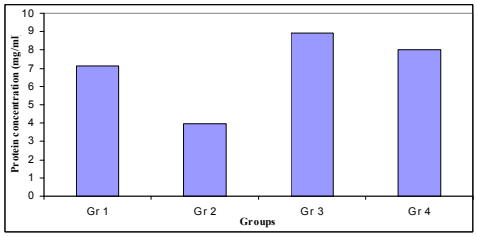

Fig. 1: Protein concentration (mg/ml) in intestine of *H. fossilis*

Reduction in protein level may be attributed to the impairment of food intake (Neff, 1985) and interference in protein synthesis due to mercury poisoning like other heavy metals (Suverson, 1977) and utilization of endogenous protein for maintenance of energy supply (Ramalingam and Ramalingam, 1982). Similar to protein, glucose level also significantly fell down due to Hg intoxication but increased in the presence of low concentration of Zn in group-III. In group-IV (Hg + Zn treated) glucose content in tissue, reached almost near to the normal level (Table-3, Fig. 2), suggesting protective role of Zn against Hg intoxication.

Table-3: Glucose concentration (mg/ml) in intestine of *H. fossilis*

Groups	Organ
	Intestine
I	15.20±0.60
II	10.20±0.41[a]
III	18.31±1.02[c]
IV	13.98±0.88[y]

Note: Data are means ±SEM. (n=7); y, p<0.01 as compared to the respective values of Hg groups; a, p<0.001; b and c, p<0.05 as compared to the respective control value

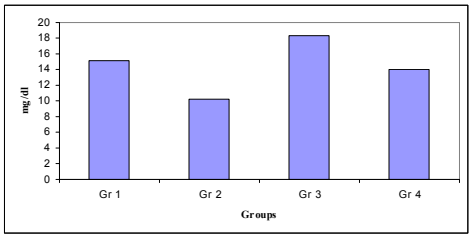

Fig. 2: Glucose concentration (mg/ml) in intestine of *H. fossilis*

Depletion in glucose content in intestine may be attributed to depletion of normal food intake due to Hg poisoning (Geed, 1992) and disturbed carbohydrate metabolism. Depletion in the level of glucose due to Zn (Kothari and Soni 2004) has been reported in the past. Protective effect of Zn against Hg toxicity also been reported by Fukino *et al.*, 1986 in rats.

During this study Hg enhanced the ALP activity, while exposure to Zn inhibited enzyme activity. However catfish exposed to Hg and Zn simultaneously was able to maintain ALP activity near normal (Table-4, Fig-2).

Duration dependent effect of Hg poisoning on ALP activity has been reported in intestine of *H. fossilis*. Both rise in ALP activity due to Hg in fish (Potdar,

Table-4: Alkaline phosphatase (KA unit) activity in intestine of *H. fossilis*

Groups	Organ
	Intestine
I	1.33±0.05
II	2.01±0.12[a]
III	0.99±0.04[a]
IV	1.30±0.08[x]

Data are means SEM. (n=7).

x, p<0.001 and y, p<0.01 as compared to the respective values of Hg group.

a, p<0.001; b, p<0.01 and c, p<0.05 as compared to the respective control values

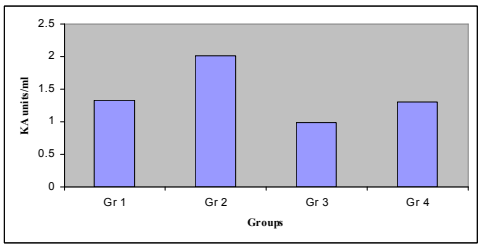

Fig. 3: Alkaline phosphatase (KA unit) activity in intestine of *H. fossilis*

2007) and fall in ALP activity due to Zn poisoning (Kothari and Soni, 2004) are known to occur. Findings of this study are in accordance with the earlier reports. It is known that alkaline phosphatase in intestinal brush border plays a critical role in the absorption of various macromolecules from the lumen to the tissue interior (Sinha, 1979; Chakrabarty and Sinha, 1982). Both the loss (Rodin and Crowson, 1962) and increase (Jeelani and Shaffi, 1986) in the ALP activity have associated with tissue necrosis and structural damage. The result of this study clearly revealed that alteration in the value of protein, glucose & ALP activity caused due to mercury intoxication were maintained near normal in the presence of zinc sulphate. This suggests that zinc provided protection against mercury caused disturbances in biochemical parameters.

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