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Biochemical alteration in blood metabolite levels of *Heteropneustes fossilis* due to Cadmium Chloride toxicity

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ABSTRACT

The present study reveals alteration in blood metabolite due to Cadmium chloride toxicity on *Heteropneustes fossilis*. For 15 days (subchronic exposure) Hyperglycaemia was observed due to hypermetabolic state of fish and hypoglycaemia condition in chronic exposure of 30 days was seen due to adaptive tolerance and exhaustion of energy. The protein depletion in Blood plasma was observed for both the exposure periods suggesting neogluconeogenesis and biochemical transformation. The level of cholesterol in serum reflecting decreasing trends indicating impaired Liver function and steroidogenesis.

Keywords: *Heteropneustes fossilis, Cadmium chloride, Blood Metabolite.*

INTRODUCTION

Intense activity in agriculture fields and industrial sectors has inevitably enhanced the level of heavy metals in waste water reservoir (Kumari, *et al*., 2006; Indira and Shivaji, 2006 and Sharma and Choudhary, 2007). Heavy metals are serious pollutants of the aquatic environment due to their persistence and biomagnifications properties (Young, 2005. Senthil kumar *et al.*, 2007; Sobha, *et al.*, 2007; Hano, *et al*., 2016).

The inherent toxicity of a heavy metal disturbs the dynamic life processes in biological system by combining with macromolecule and metabolites. Cadmium is considered as non-essential element has no positive role in biological function of organisms in general & human in particular. Cadmium is highly toxic which can impart a cumulative poisonous effect on mammals causes hyper pressure, renal impairment, gastrointestinal dysfunctions, anemia, emphysema, prostate cancer & other (Jha and Jha, 2011 and Bedii, *et al.*, 2005).

Some of the works have been reported on fish due Cadmium toxicity (Mark, *et al.*, 1974., Shaby and Gupta, 1994., Muley, *et al.*, 2006., Rakesh, *et al.*, 2007.,Rekha Rani, *et al.*, 2008., Jha and Jha, 2011., M. Saeed Heydamejad, *et al.*, 2013) but still more work is needed in this direction.

In the light of the above facts fish *Heteropneustes fossilis* was selected for biochemical study to see the alteration in blood metabolites due to $CdCl_2$ exposure for subchronic (15 days) & chronic (30 days) period.

MATERIALS AND METHODS

To determine LC_{50} of cadmium chloride exposure to *Heteropneustes fossilis*, a static bio-assay test was done following the method of APHA and sublethal concentration (4 mg/L) was calculated by employing the formula of Hart, *et al*. (1945).

Sublethal concentration of $CdCl_2$ was given to fish *Heteropneustes fossilis* [20 – 22 cm (L), 35 – 41 g (W)] for the period of 15 and 30 days side by side control groups was also

run in equal volume of water. The exposure was renewed at every 24 hrs and at the termination of exposure period *i.e.* 15 days & 30 days the *Heteropneustes fossilis* was subjected to anaesthetized with 1:4000 MS 222 (Tricane methane sulphonate) and processed for quantitative analysis of blood glucose by the method of Dabowski, 1962, plasma protein by Varley, *et al*. (1980) and serum cholesterol by Kabara, *et al*. (1966).

RESULTS

The fish *Heteropneustes fossilis* under 15 and 30 days exposure of $CdCl_2$ shows decline trends in the level of protein and cholesterol for both exposure periods but different results was obtained in blood glucose *i.e.* hyperglycemia in 15 days exposure and hypoglycemia in 30 days exposure period.

The blood glucose elevation to 68.09 ± 0.08 mg/ 100 ml of blood against control value of 61.25±0.28 in 15 days exposure period where as 30 days showed decline by 36.14% (control 63.63 ±0.14 and treated 40.78 ±0.13). The plasma protein level showed decrease for both the exposure periods. The level shows decline for both 15 days and 30 days exposure 7.19 ± 0.13 and 4.17 ±0.10 from 9.41 ±0.17 and 8.60 ±0.05 respectively. These two values are significant at P<0.001 (Table 1).

The serum cholesterol showed decrease for both the exposure periods (Table 1) and the decrease by 13.80% (142.14± 0.09 from 164.90 ± 0.17) in 15 days and 15.28 % decrease (136.96±0.21 from 161.68±0.20). Both are significant at P<0.001

Table 1

Profiles of Blood glucose, plasma protein & serum cholesterol in normal and $CdCl_2$ treated fish, *Heteropneustes fossilis* (Values are ± SE of five fishes in each group)

Sl. No.	Parameters	Days	Control	Treated	Student 't' Test P value	% increase (+) or Decrease(-)
1	Blood Glucose (mg/100 ml)	15	61.25 ± 0.28	68.09 ± 0.08	P<0.05	+11.14
		30	63.63 ± 0.14	40.78 ± 0.13	P<0.001	-36.14
2	Plasma Protein (mg/100 ml)	15	9.41 ± 0.17	7.19 ± 0.13	P<0.001	-23.59
		30	8.60 ± 0.05	4.17 ± 0.10	P<0.001	-51.51
3	Serum cholesterol (mg/100 ml)	15	164.90 ± 0.17	142.14 ± 0.09	P<0.001	-13.80
		30	161.68 ± 0.20	136.96 ± 0.21	P<0.001	-15.28

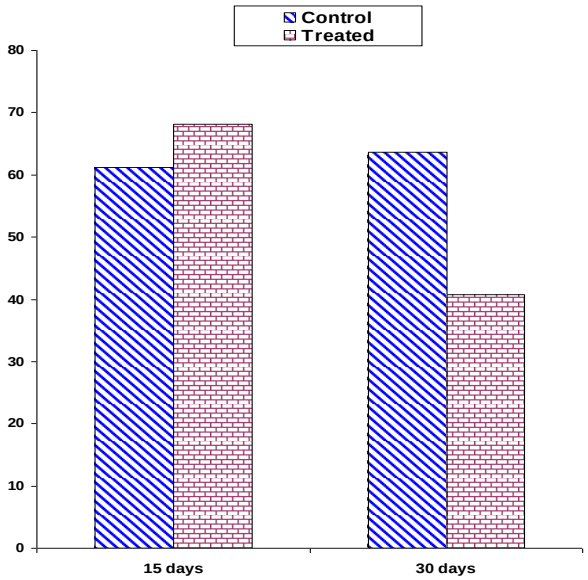

Plate 1. Profiles of blood glucose in normal and $CdCl_2$ treated *Heteropneustes fossilis*

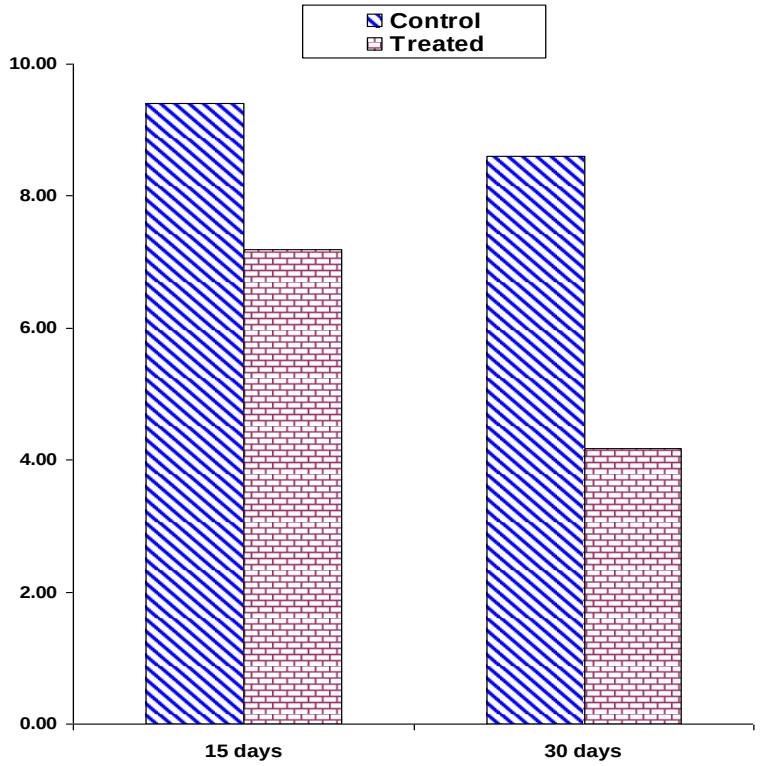

Plate 2. Profiles of plasma protein in normal and $CdCl_2$ treated *Heteropneustes fossilis*

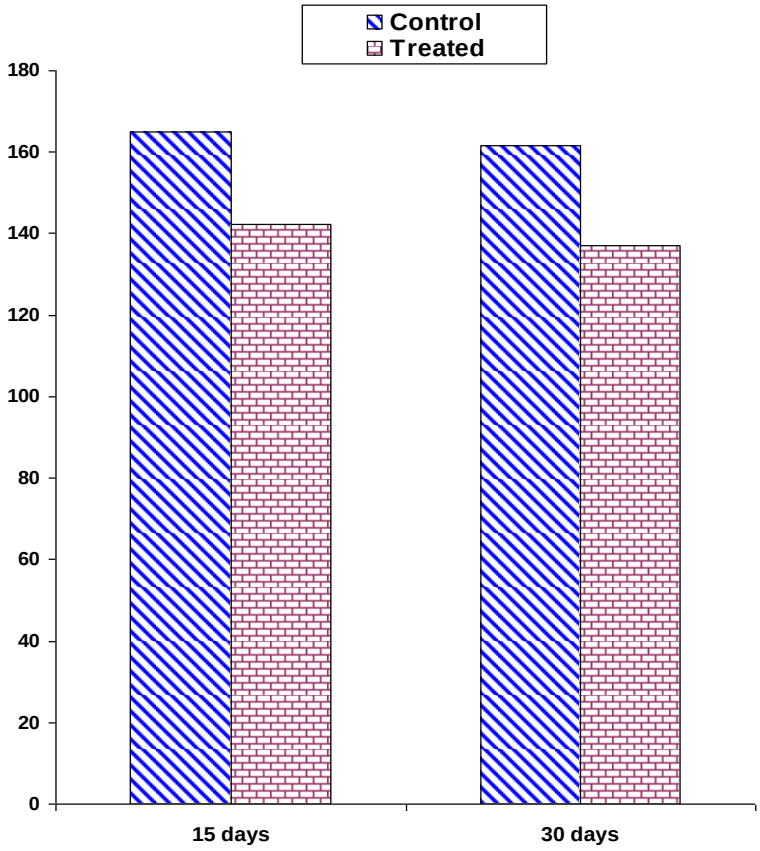

Plate 3. Profiles of serum cholesterol in normal and $CdCl_2$ treated *Heteropneustes fossilis*

DISCUSSION

Blood Glucose:

The significant rise in blood glucose for 15 days exposure period is suggestive to hyper metabolic states of fish under metal intoxication, thus glucose intake gets elevated to fulfill the excessive demand of energy in toxic stress. The result in present investigation is similar to the work of Jha (1992), Roy (2003), Seema, *et al*. (2005), Rekha Rani, *et al.* (2008). This elevation may also be attributed to release of adrenal medullory amines under stressful condition.

For 30 days exposure period, decline in glucose level was observed. The hypoglycemia condition seems to be adaptive tolerance of fish and exhaustion of energy under $CdCl_2$ exposure.

This condition has also been presented by Rajan (1990) and Jha (1992). The condition can be supported by the contention that cadmium gets accumulated and causes pancreatic necrotic lesion.

Plasma Protein:

The present study has given level of plasma protein similar to the investigation made by Jana and Bandyopadhyay (1987), Jha (1991), Siddique and Chang (2014). The decline in plasma protein level can be explained that there is a progressive degradation of protein & biochemical transformation of the protein nitrogen in other nitrogenous product as suggested by Mandal (2009). This can also be explained in context of metal toxicity of neoglucogenelis to maintain carbohydrates metabolism in stress of metal (Harper, 1985).

Serum Cholesterol:

The present finding reflects decrease in serum cholesterol due to cadmium toxicity for both the exposure periods. The result exhibited shows similarity with the findings of Jha (1992), Mandal and Jha (2009) and Heydamejad, *et al*. (2013).

Review of the literature suggested that metals have been reported to induce both decrease & increase in cholesterol level as decreases suggested by Jha (1992) and Heydamejad, *et al.* (2013) and increase by Sastry and Sharma (1980), Jha and Jha (2011). Present findings reveal that cholesterol is raw material of steroidogenesis, so its depletion reflects impaired liver function and disturbed steroidogenesis.

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