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Changes induced by Cadmium chloride on tissue metabolites of an air breathing fish - *Heteropneustes fossilis*

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ABSTRACT

The present study reveals changes in tissue metabolites due to Cadmium chloride toxicity on Heteropneustes fossilis for the period of 15 days (subchronic exposure) and 30 days (chronic exposure). Depletion in muscles and liver protein was observed due to blocking of mRNA synthesis and impending energy demand due to toxicity stress. The level of lipid in muscle and liver has been observed in depleted states reflecting lipid hydrolysis under high energy requirement during stress. It is believed that metal cadmium chloride impairs pancreas function there by decrease in lipid level has been observed under 15 days and 30 days exposure periods.

INTRODUCTION

The history of the water pollution is distinctly related to the human civilization. The pollutants mainly enter various water reservoir in varied form viz pesticides, acids, alkalides and heavy metals. In recent years heavy metal pollution has become a subject of considerable interest in the field of aquatic toxicology. Of all heavy metals, cadmium is one which does not have any biological significance and is known to be extremely toxic due to persistence nature (Thiruvallvan *et al.*, 1997; Gernhofer *et al*., 2001, Rauf *et al*., 2009).

The toxic impact of cadmium has been worked out by some workers (Vander Oost *et al*., 2003; Vutukuru *et al*., 2005 and Nawaz *et al*., 2010).

In the light of the above facts it was thought worthwhile to evaluate the subchronic and chronic effect of $CdCl_2$ on tissue metabolites level of the fresh water teleost *Heteropneustes fossilis* (Bloch) as biochemical changes give first hand information about the stress much before the gross sign of histopathological damage.

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 tissue protein by method of Folin-Lowry method (Lowry *et al*., 1951) and tissue lipid by employing method adopted by Folch *et al*., (1957).

RESULTS

The fish *Heteropneustes fossilis* under 15 and 30 days exposure of $CdCl_2$ shows decline trends in the level of protein in liver and muscles and lipid profile also showed decrease trends in both 15 and 30 days exposure periods.

The hepatic protein depletes to 66.21±0.04 from normal level of 74.24±0.02 in 15 days exposure period where as 30 days showed decline by 15.16% (control 73.24 ±0.02 to 62.13±0.03 in treated). The muscles protein level showed decrease for both the exposure periods. The level shows decline for both 15 days and 30 days exposure 76.37 ± 0.03 & 68.42 ±0.02 from 80.24 ±0.08 & 80.20 ±0.05 respectively. These two values are significant at $P<0.001$ (Table 1)

The liver lipid showed depletion for both the exposure periods (Table 2) & the decrease by 8.82% (40.27± 0.04 from 44.17 ± 0.03) in 15 days and 14.59 % decrease (36.17±0.04 from 40.35±0.13) in 30 days. Both are significant at $P<0.001$.

The muscle lipid showed declined for both the exposure periods (Table 2) and the decrease by 39.85% (3.26± 0.04 from 5.42 ± 0.04) in 15 days and 58.27 % decrease (2.22±0.07 from 5.32±0.02) in 30 days. Both are significant at $P<0.001$.

Table 1: Profiles of Liver Protein and Muscle Protein, in normal and $CdCl_2$ treated fish, *Heteropneustes fossilis*
(Values are ± SE of five fish in each group)

Sl. No.	Parameters	Days	Control	Treated	Student 't' Test P value	% increase (+) or Decrease(-)
1	Liver Protein (mg/100 gm)	15	74.24±0.02	66.21±0.04	P<0.01	-10.81
		30	73.24±0.02	62.13±0.03	P<0.001	-15.16
2	Muscle Protein (mg/100gm)	15	80.24±0.08	76.37±0.03	P<0.001	-4.82
		30	80.20±0.05	68.42±0.02	P<0.001	-14.68

Table 2: Profiles of and Liver and Muscle Lipid, in normal and $CdCl_2$ treated fish, *Heteropneustes fossilis*
(Values are ± SE of five fish in each group)

Sl. No.	Parameters	Days	Control	Treated	Student 't' Test P value	% increase (+) or Decrease(-)
1	Liver Lipid (mg/100 gm)	15	44.17 ± 0.03	40.27± 0.04	P<0.01	- 8.82
		30	40.35±0.13	36.17±0.04	P<0.001	- 14.59
2	Muscle Lipid (mg/100gm)	15	5.42 ± 0.04	3.26± 0.04	P<0.001	- 39.85
		30	5.32±0.02	2.22±0.07	P<0.001	- 58.27

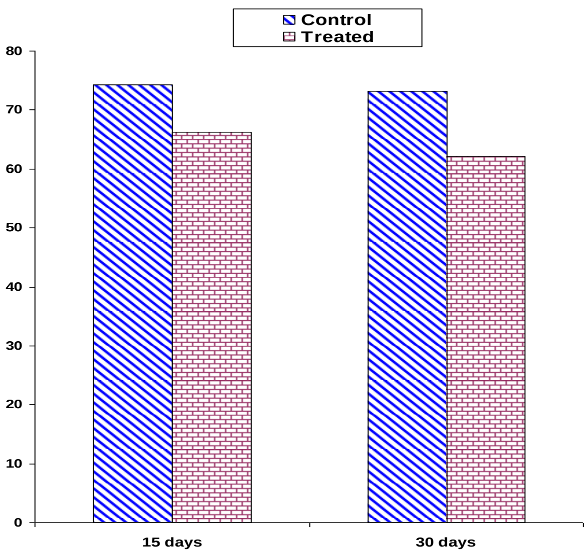

Plate 1: Profiles of Liver Protein in Normal and $CdCl_2$ Treated *Heteropneustes fossilis*

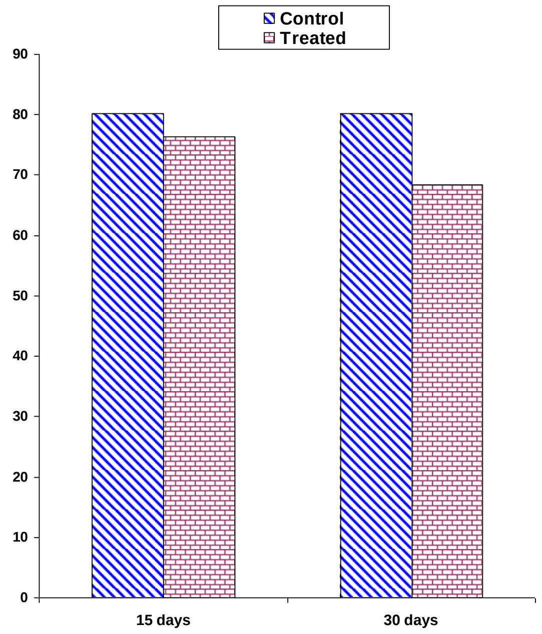

Plate 2: Profiles of Muscle Protein in Normal and $CdCl_2$ Treated *Heteropneustes fossilis*

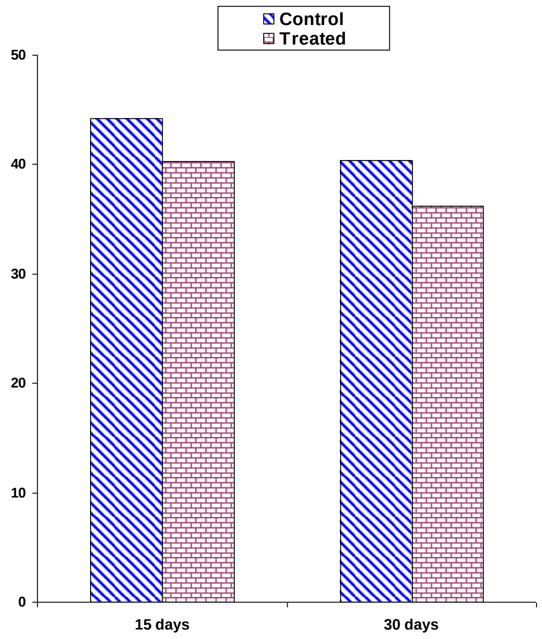

Plate 3: Profiles of Liver Lipid in Normal and $CdCl_2$ Treated *Heteropneustes fossilis*

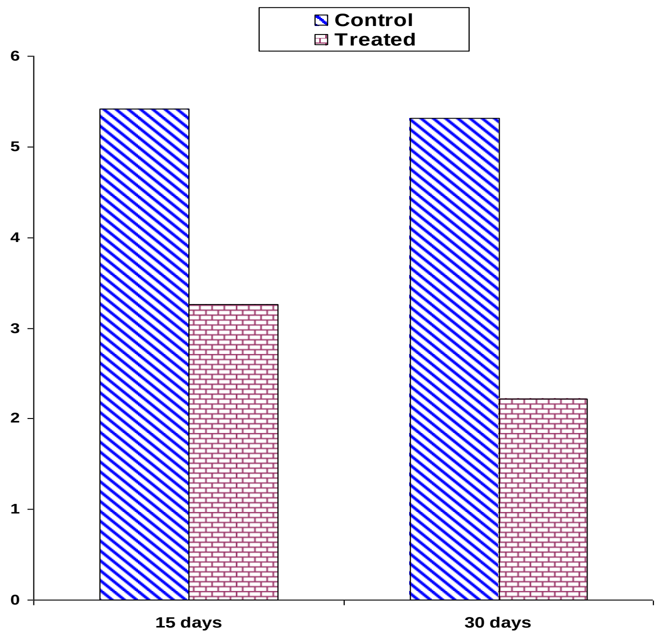

Plate 4: Profiles of Muscle Lipid in Normal and $CdCl_2$ Treated *Heteropneustes fossilis*

DISCUSSION

PROTEIN

Depletion in tissue protein reveals the requirements of high energy to maintain homeostasis under metal stress. It seems possible that under metal toxicity impending energy demand for tissue repair and detoxification procedure causes decrease in protein contents (De Smet and Blust G.A., 2000 and Senthil *et al*., 2007). Metals have also been reported to block mRNA synthesis and there by stopping transcription (Jana, 1981; Jha, 1992; Mandal ., 2009 and Bais and Lokhande, 2012).

The possibility of protein depletion may be associated to gluconeogenchis by utilizing protein in stress (Akhter Ali Siddque & Suponglemla Chang, 2014).

LIPID

Lipids serve as high source of energy and are utilized prior to protein under stress of metal (Roy, 2003 and Mandal S, 2009). Lipid being major calorie contributor and also helps in structural existence of cell membrane. The metal cadmium is supposed to cause impairment in lipid metabolism and adversely affecting its level (Mulay *et al*., 2006, Rekha Rani *et al*., 2008).

The depletion muscle lipid is an outcome of lipid hydrolysis under high energy demand during stress. The metal ability to disturb pancreas function might have contributed in decline of lipid content (Harper *et al*., 1985; Jha and Jha, 2011 and Hano *et al*., 2016).

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