*Available online at*www.scholarsresearchlibrary.com

Scholars Research Library

Archives of Applied Science Research, 2016, 8 (7):1-3
(http://scholarsresearchlibrary.com/archive.html)

ISSN 0975-508X
CODEN (USA) AASRC9

Effect of Chromium trioxide on Liver biochemistry of freshwater fish, *Channa punctatus (*Bloch)

Mamta Pal and S. P. Trivedi

Environmental toxicology Laboratory, Department of Zoology, University of Lucknow (U.P.), India

---

ABSTRACT

The present study was aimed to investigate the impact of Chromium trioxide on biochemical parameters of freshwater fish, Channa punctatus exposed to sub lethal concentration for 7, 15, 30 and 60 days. Aspartate transaminase (AST), Alanine transaminase (ALT), Orthophosphoric Monoester Phospho-hydrolase (OMP) and Orthophosphoric acid Monoester Phospho-hydrolase (OAMP) activities have been analysed for different exposure. Results illustrates that AST, ALT, OMP and OAMP were found to be increases in comparison to control. However, the data's were collected are statistically significance at p<0.05, P<0.01 and P< 0.001 level.

Key words: Chromium trioxide, Biochemical parameters and *Channa punctatus*

INTRODUCTION

Heavy metals pollution in water bodies are mainly caused by industrial, consumer waste or from acidic rain, it breaking down the soils and releasing heavy metals into rivers, lakes, streams, and ground water [1]..Heavy metals are essential to living organisms in varying amounts, however chromium plays an important role in growth and carbohydrates and; lipids metabolism [2]. At higher concentrations (200-300 mg/L), chromium is a highly toxic, mutagenic [3] teratogenic [4], carcinogenic [5]. It is highly mobile and enters into the food chain [6]. Chromium exists primarily in Cr (III) and Cr (VI) oxidation states but Cr (VI) is found to be more toxic in the environment due to its higher solubility and mobility [7-8].

Since chromium contributes its important role in industrial pollution causes abnormalities in the fish body, besides of this, biochemical profiles in fish and other aquatic animals due to heavy metal pollution acts as a bio-indicators for monitoring of aquatic ecosystem [9-10]. In view of this the present work was undertaken to know the impact of Chromium trioxide on biochemical parameters of freshwater fish, *Channa punctatus* exposed to sub lethal concentration for 7, 15, 30 and 60 days.

MATERIALS AND METHODS

The experimental fish, *Channa punctatus* of approximately equal in size (11±2 cm) and weight (15±2 g) were collected with the help of fisherman from local source of Lucknow. The experimental fish was properly washed with tap water and treated with 0.02% $KMNO_4$ and 0.004% formalin solution to remove any contaminants. The fish were acclimatized to laboratory conditions for 30 days before starting experiment and were fed TOKYO (made in Japan) on 7 pm everyday. The fish were divided into 5 equal groups consisting of 10 each and each group was transferred separately to glass aquaria of 100 liter volume. Group I fish were maintained as control without any treatment, the group II, III, IV and V fish were exposed to sub lethal concentration of Chromium trioxide for 7,15, 30 and 60 days. Now, 10 % homogenates of liver were prepared in ice cold 0.25 M sucrose and then centrifuge at 2500 rpm for 15 minutes, supernatant was used for biochemical analysis. Biochemical analysis *viz*; Aspartate transaminase (AST), Alanine transaminase (ALT), Orthophosphoric Monoester Phospho-hydrolase (OMP) and Orthophosphoric acid

Monoester Phospho-hydrolase (OAMP) activities have been performed for different exposure. These biochemical analyses were done as per standard methods of Reitman and Frankel [11] and Bodansky [12]. The data shown are the average of three replicates ± SD and statistical significance was tested at p<0.05, P<0.01 and P< 0.001 level.

RESULTS AND DISCUSSION

In present investigation, Biochemical analysis *viz*; Aspartate transaminase (AST), Alanine transaminase (ALT), Orthophosphoric Monoester Phospho-hydrolase (OMP) and Orthophosphoric acid Monoester Phospho-hydrolase (OAMP) activities were done and summarized in Table-1. Aspartate transaminase (AST) activities are found to be increases as compared to control (0.38±0.010). After 7, 15, 30 and 60 days exposure of sub lethal concentration of Chromium trioxide, 18.42 %, 36.84%, 57.89% and 86.84% rise in AST activities was found respectively. Whereas, in case of Alanine transaminase (ALT) activities inconsequential changes seen up to 15 days, with increases of 7.09% for 7 days and 16.92% for 15 days but after 30 and 60 days of exposure the increase in ALT activities was observed as 26.15% and 32.30% respectively as compared to control (0.65±0.0153). Orthophosphoric Monoester Phospho-hydrolase (OMP) also showed an increasing trend as compared to control (0.11±0.020) and was recorded as 90.90%, 154.54%, 227.26% and 300% respectively after 7, 15, 30 and 60 days exposure of sub lethal concentration of Chromium trioxide. However, Orthophosphoric acid Monoester Phospho-hydrolase (OAMP) activities was found to be increased as compared to control (0.15±0.020), by 46.66%,113.33%, 153.33% and 293.33% respectively after 7, 15, 30 and 60 days exposure.

Table-1: Showing Enzyme activities in liver of *Channa punctatus* exposed to CrO_3

Parameters	Control	7 Days	15 Days	30 Days	60 Days
AST	0.38±0.010	0.45±0.15^{n}	0.52±0.010***	0.60±0.0153***	0.71±0.010***
ALT	0.65±0.0153	0.70±0.020 ^{n}	0.76±0.010***	0.82±0.010***	0.86±0.0153***
OMP	0.11±0.020	0.21±0.015 ^{n}	0.28±0.015***	0.36±0.010***	0.44±0.020***
OAMP	0.15±0.020	0.22±0.015 ^{n}	0.32±0.010***	0.38±0.041^{n}	0.44±0.015***

*± SD; *P<0.05, **P<0.01, ***P< 0.001, n = Non signi ficant*

Results illustrates that Aspartate transaminase (AST), Alanine transaminase (ALT), Orthophosphoric Monoester Phospho-hydrolase (OMP) and Orthophosphoric acid Monoester Phospho-hydrolase (OAMP) activities elevated on exposure of sub lethal concentration of Chromium trioxide for 7, 15, 30 and 60 days.

Whereas, there is an increase in the activities of GOT and GPT has been reported in *Cyprinus carpio* exposed to cadmium [13]. It was reported that blood enzymes were highly raised in the fish treated with heavy metals *viz*; cadmium, zinc and copper [14-16]. Accoding to Shakoori *et al* [17] blood enzymatic activity is increased due to leakage of these enzymes from hepatic cells causes raising levels in blood or increased synthesis or enzyme induction of these enzymes. Elevated serum aminotransferases causes myocardial and hepatic toxicity leading to extensive liberation of the enzymes into the blood [18-19]. Monitoring of liver enzymes leakage into the blood has useful tool to study liver toxicology [20]. Due to increase in GOT and GPT transaminases might be attributed to tissue damage especially liver [21]. These enzymes activity were found to increase on exposure to heavy metals in different fish species [22]. It was reported that chronic exposure of fish to metals like Zn, Cu and Cd raised the levels of plasma GOT and GPT [23]. Whereas, Shaheen and Akhtar [24] observed the stress reactions in fish causes reduction in their immune potential on prolonged exposure to various sublethal concentrations of Cr(VI) and also found that changes in the haematological and biochemical parameters causes a deleterious effect on fish immunity. It was reported that alteration in the enzymatic activities are due to Various types of liver damage or biliary tube obstruction [25-26].

Results of the present investigation are more or less similar to the previous authors and also in agreement with their findings.

REFERENCES

[1] EE. Obasohan, JAO.Oronsaye and OL. Eguavoen. *Afr. Sci*., **2008**, 9(1), 13-23.
[2] A.Taylor. *Ann. Clin. Biochem*. **1999**, 33, 486-510.
[3] L. Arreola-Mendoza, JL. Reyes, E. Melendez, D. Martín, MC. Namorado, E. Sanchez and LM. Del Razo. *Toxicology*, **2006**, 218, 237-264.
[4] MI. Yousef, FM. El-Demerdash, KI. Kamil, and FA. Elaswad. *Reproduc. Toxicol*., **2006**, 21, 322-328.
[5] M. Prabakaran, C. Binuramesh, D. Steinhagen and M.Dinakaran. *Ecooxcol. Environ. Saf.*, **2007**, 68(3), 372-378.
[6] M. Goldoni, A. Caglieri, D. Poli, MV. Vettori, M. Corradi, P. Apostoli, and A. Mutti. *Anal. Chim. Acta***, 2006**, 562, 229- 235.
[7] M. Reynolds, E, Peterson, G. Quievryn, A, Zhitkovich. *Journal of Biological Chemistry*, **2004**, 279(29), 30419-24.

[8] S.Vutukuru. *International Journal of Environmental Research and Public Health*, **2005**, 2(3), 456-62.
[9] V, Luskova. *Acta Scientiarum Naturalium Acad Scientiarum Bohemicae Brno NS*. **1997**, 31(5), 31-70.
[10] H. Abbas, H. Mahmoud, J. Miller. Online Journal of Veterinary Research. 2007, 11:54-74.
[11] S.Reitman and S.Frankel. *J. Clin. Pathol*, **1957**, 28, 56- 59.
[12] A.Bodansky. In Hawk's physiological chemistry (*Ed. B.L. Oser*) *Mc Grant-Hill*, New York. **1965**, 1118.
[13] H. De Smet and R. Blust. *Toxicol. Lett.* **1990**, 54, 199-205.
[14] AM. Hilmy and NEI. Domaity. *Comp.Biochem.Physiol*., **1987**, 87, 297-301.
[15] V. Karan and S. Vitorovic *et al*. *Ecotoxicol. Environ. Saf.*, **1998**, 40, 49-55.
[16] HS. Singh and TV. Reddy. Ecotoxic.Environ.Saf. **1990**, 20, 30-35.
[17] AR. Shakoori J. Alam *et al. Proc. Pak.Congr. Zool*.**, 1990**, 10, 61-81.
[18] AG. Heath. *CRC press Inc*. Boca. Raton Florida. U.S.A. **1987**.
[19] S.Abo-Hegab, MGE. Kamel and WD. Labib. *Proc. Zool. Soc. A.R.E*. **1993**, 24, 183-197.
[20] AGM. Osman, M. Koutb and AEH. Sayed. *Journal of Photochemistry and Photobiology B: Biology*, **2010**, 99, 1-8.
[21] VR. Chavan, and DV. Muley. *Int.J.Curr.Microbiol.App.Sci.*, **1914**, 3(5), 277-288.
[22] IAA. Mekkawy, UM. Mahmoud, FT. Wassif and M. Naguib. *Fish Physiology and Biochemistry*, **2011**, 37(1), 71-84.
[23] RV. Zikie, AS. Stajn, SZ. Pavlovie, BI. Ognjanovic and ZS. Saicic. *Physiological Research*, **2001**, 50(1), 105-11.
[24] T.Shaheen and T. Akhtar. *Turk J Zool*., **2012**; 36(5), 682-690.
[25] O. Fırat and F. Kargın. *Arch. Environ. Contam. Toxicol*., **2009**, 58(1), 151-7.
[26] M., Oner, G. Atli, and M. Canli. *Bullet. Environ. Contam. Toxicol*. **2009**, 82(3), 317-21.