Available online at www.scholarsresearchlibrary.com

Scholars Research Library

European Journal of Zoological Research, 2013, 2 (1):6-9
(http://scholarsresearchlibrary.com/archive.html)

ISSN: 2278–7356

Ascorbate effect on the cadmium induced histopathological alterations in liver of the fresh water fish, *Channa orientalis* (schneider)

Borane Vijay Ramdas

Department of Zoology, Jijamata Arts, Science and Commerce. College, Nandurbar, (M.S.) India.

ABSTRACT

Fresh water fishes, Channa orientalis were exposed to chronic dose of $CdCl_2$ with and without ascorbic acid. Histopathological changes shows that the heavy metal enters the systems ultimately causing damage to liver rupture of cell membrane, accumulation of blood cell on exposure to heavy metal along with and without ascorbic acid. The groups exposed to heavy metals along with ascorbic acid showed recovery than those exposed to only heavy metal. Pre-exposed fishes to heavy metals showed fast recovery than those which were allowed to cure naturally.

Key words: Cadmium, Liver histology, Ascorbic acid (50mg/l.), *Channa orientalis* (Schneider).

INTRODUCTION

Toxicology is one of the most important multidisciplinary subjects dealing with the adverse effect of chemical and allied agents on living systems [1]. Heavy metals coming out from industrial effluents enter the fresh water habitats cause serious deterioration in water quality which may lead to mortality of aquatic organisms bring about histological changes in aquatic habitats. *Channa orientalis* is one of the local fresh water edible fish. Hence, it is prudent to study the chronic toxicity and the changes that occur in the histology. Most significant cases of long-term contamination are recorded when cadmium is deposited in bottom sedimentation [2], subsequently transferred into food chain in the aquatic environment. According to mode of action of the toxicant on different tissues certain architectural changes occur whose effect causes the death of the organisms [3].

Cadmium has no role in biological system that can produce marked alterations of the entire liver structure with degenerative and proliferative changes of non-target organisms [4]. Cadmium may induce oxidative damage in different tissues by enhancing peroxidation of membrane lipids in tissues and altering the antioxidant systems of the cells [5]. Pathological changes have been reported in fishes on exposure to different pesticides [6]. Administration of either Liv-52 alone or in combination with spirulina in cadmium-intoxicated rats caused more pronounced hepato-protective effects treated rats showed periportal inflammation, fatty change, congestion of sinusoids and microvesicular steastosis [7]. Histopathological lesions induced in the hepatopancreas of *Channa punctatus* and *Clarius batrachus* exposed to industrial pollutants [8]. Histopathological changes increase in the size of lumen and can be excretory or secretary activities of the cells [9]. The heavy metals produce cumulative deleterious effects on animals, however studies on the vitamin C against heavy metals toxicity in animal system is limited and inconclusive [10]. Ascorbic acid protects extra cellular protein function through gene expression highlighted [11]. Recovery by ascorbic acid in metabolic changes altered by heavy metal in bivalve [12]. Ascorbic acid being important constituent in cellular metabolism, the interactions of biomolecules gives proper idea of toxicant and its effect.

MATERIAL AND METHODS

Medium sized fresh water fishes, *Channa orientalis* were collected from Shiven river area Nandurbar Dist. Nandurbar. Physico-chemical parameters given [13]. The fishes were divided in to three groups A, B and C. Group

A as a control. Group B fishes were exposed to $LC_{50/10}$ dose of Cd^{++} (1.248ppm) as $CdCl_2$ for 45 days; while group C fishes were exposed to chronic concentration of heavy metal with ascorbic acid for 45 days. Fishes from B groups were divided into two D &E groups after 45 days. Group D were allowed to cure naturally while those of E groups were exposed to ascorbic acid (50 mg/l.). Histopathological changes were recorded from A, B and C group after 15 and 45 days of exposure and from D and E groups after 50^{th} and 55^{th} days of recovery. The tissue was placed in Bouin's fixative for 24hr. They were dehydrated in a graded series alcohols. Then embedded in paraffin, sectioned at 5µm thickness and stained with haematoxylin and eosin and mounted in Destrene Plasticizer Xylene [14]. Standard histopathological procedures [15], were followed for investigations. Histological effects are analyzed in comparison to control.

RESULT AND DISCUSSION

Photomicrographs of the slides showing the histopathological structure of the liver are given in Plates. The Physico-chemical properties are given in Table -1. Plate -I shows the normal structure of liver. From results it is clearly seen that liver tissue damage depend upon cadmium concentration and duration of exposure with and without ascorbic acid and during recovery are shows given in Plate -I, Plate II-A, Plate II -B.

Liver is the main organ responsible for detoxification of harmful substances. Toxicants reach to liver through circulation. Histological the liver is made of roughly hexagonal hepatic lobules (Plate I- b1 and b2) consist of cords of polygonal hepatic cells called hepatocytes concentrating towards the central vein. The photomicrographs (Plate II- A) histopathological changes in the liver after exposure to cadmium included, necrotic effect on hepatocytes, hyperplasia, vacuolization, rupture of the cell membranes resulting in to multinucleated regions and accumulation of RBC [16]. Liver revealed a foamy appearance, with large nuclei and prominent nucleoli, and vacuoles. The Plate II – B show the protection by the ascorbic acid and hence the impact of cadmium on the structure of liver during the exposure period is less and the structure comparatively appears normal. Fishes treated urea showed disintegration and separation of hepatic cells from blood vessels, vacuolization and necrosis in pancreatic cells [17]. Industrial effluents cause metabolic disorders, immunosuppression among fishes [18]. The presence of definite necrosis indicated capability of the toxin causing cell death [19].

The recovery in the structure of the liver after the discontinuation of cadmium. The rate of recovery as is evident from the histological structure of liver is faster in the ascorbic acid as compared to those recovered in the normal water. The alterations in the structure of the basement membranes can be recovered faster by ascorbic acid. During recovery, regeneration of hepatocytes with organized orientation of hepatic cords and bile passage [20], in *Puntitus ticto* after recovery of one month of atrazine exposure. Biological compounds with antioxidant properties contribute to the protection of cells and tissues against deleterious effects of reactive oxygen species and other free radicals. Vitamin E and C are potent free radical scavengers and prevent oxidative damage by utilizing free radicals [21]. Ascorbic acid plays important role in the collagen synthesis for the hydroxylation of the proline can correct the altered situation due to heavy metals. Collagen forms the dominant part of the basement membranes; its damage causes the improper supply to the overlying epithelia which are found to puff out from the basement membrane. Ascorbic acid acts as a detoxifier reduces the toxicity to offer protection to the cell from expansion or abnormalities in their structural features.

Table No-1: Physico-chemical parameters Of water used for experimentation

Temperature	25·1 ± 3·2⁰
PH	7 · 6 0 ± 0 · 3
Conductivity	140 ± 15.7 µ $mho^{-cm.}$
Free Co2	3·34 ±1· $3ml^{1.}$
Dissolved O2	6·3 ± 1·1ml^{1}.
Total Hardness	204 ± 12·0mg^{-1}.
Total Alkalinity	585·6 ± 32.8 mg^{-1}.
Magnesium	31·67 ± 2.9 mg^{-1}
Calcium	30·46 ± 3.06 mg^{-1}
Chloride	107·92 ± 16.34 mg^{-1}.

PLATE -I **PLATE II -A** **PLATE II -B**

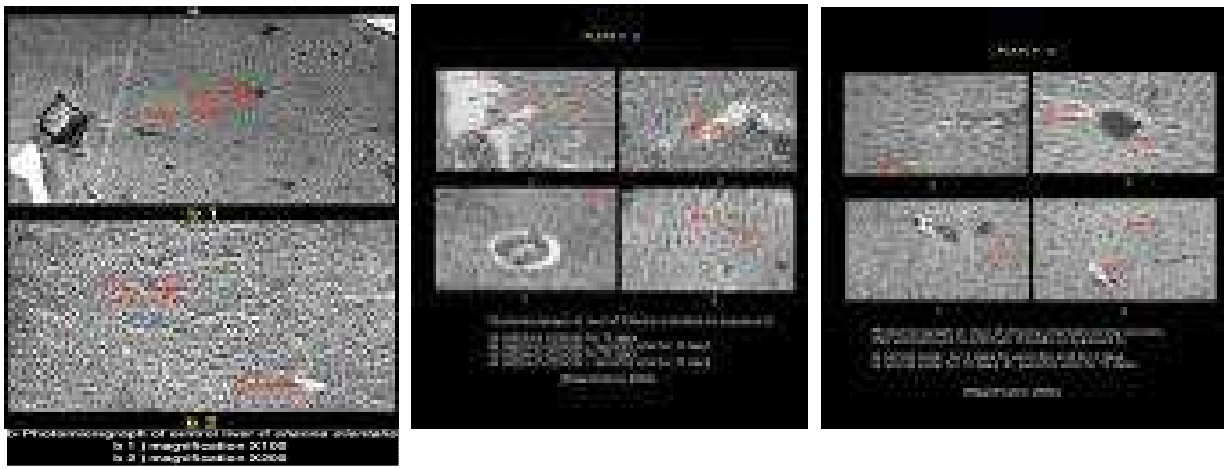

PLATE – I
HC=Hepatic Cord, CV =Central Vein

PLATE II - A=DT=Degenerated Tissue, HS=Hepatic Sinusoid,
NC= Necrosis, BS =Blood Sinus, H =Hemorrhage
BD = Balloon Degeneration, BVC= Blood Vessel Congestion
RHC= Ruptured Hepatic Cells

PLATE II - B= CV= Central Vein, DH = Degenerated Haepatocyte
NH= Normal Hepatocyte, BS = Blood Sinus,
DH = Degenerated Haepatocyte

CONCLUSION

The ascorbic acid act as an antioxidant and efficient inhibitor against cadmium chloride.

Ascorbic acid acts as a detoxifier reduces the toxicity of the heavy metals to offer protection to the cell from expansion or abnormalities in their structural features.

The photomicrographs (Plate II- A) histopathological changes in the liver after exposure to cadmium included, necrotic effect on hepatocytes, hyperplasia, vacuolization, rupture of the cell membranes resulting in to multinucleated regions and accumulation of RBC. Liver revealed a foamy appearance, with large nuclei and prominent nucleoli, and vacuoles. The Plate II – B show the protection by the ascorbic acid and hence the impact of cadmium on the structure of liver during the exposure period is less and the structure comparatively appears normal.

The rate of recovery as is evident from the histological structure of liver is faster in the ascorbic acid as compared to those recovered in the normal water. The alterations in the structure of the basement membranes can be recovered faster by ascorbic acid.

REFERENCES

[1] Lu; basic toxicology, fundamental target organs and risk assessment. Hemisphere publishing Corporation Washington**.1958**.
[2] D Leontovičová**;** Complex monitoring in selected profiles of state networks of water quality follow up in CHMU Periodicum fakulty ekologie an enviromentalistiky Technickej university Vozvolene. **2003,** *Vol. 10 Suppl.*
[3] DJH, Philips; PS Rainbow; Biomonitoring of trace aquatic contaminations, 2'nd Edn, Chapman and Hall, London**.1994**.
[4] JP Shukla; K Pandey; *Acta. Hydroch. Hydrob.* **1998,** *16: 537*-540.
[5]S Sarkar; P Yadav; R Trivedi ; AK Bansal ; D Bhatnagar;. *Trace Elem. Med. Biol.***1995,** *9:* 144.
[6] KS Tilak; K Yacob; *J. Eotoxicol. Environ. Monit.* **2002,** *12(1): 9*-15.
[7] K Jeyaprakash; P Chinnaswamy; *J. Ecotoxicol. Environ. Monit.* **2005,** *15(4) 333*-339.
[8] T Bhattacharya; AK Ray; S Bhattacharya**;** *Matsya*.**1984,** *11:1.*
[9] CS Panse; SK Deshpande; LR Tiwari; *Ecology and fisheries.***2010,***Vol.3 (1):19*-22.

[10] WHO; Laboratory manual for the examination of Human semen and Semen cervical mucus interaction, Geneva.**1980**.
[11] HR Griffiths; J Lunec; *Environ Toxicol Pharmacol*.**2001,** *10:173* – 182.
[12] AY Mahajan; SP Zambare**;** *Asian J. Microbiol. Biotech. Environ. Sci* .**2001,** *Vol. 3, No. (1- 2): 95*-100.
[13] APHA, AWWA AND WPCF; Standard methods for the examination of water and waste water APHA (17'th) INC, New York. **1985**.
[14] RAB Durvy; EA Willington**;** Caleton Histological Techniques 4'th edn. Oxford University Press. London. **1967**.
[15] RJ Robert; Fish pathology (2'nd edition), London, Billier, Tindall.**1989,** 11, pp 453.
[16] Mazher Sulthana; A Boja Rajan; *J. Aqua. Biol. Vol.* **2007,** *(22): 189*-192.
[17] Arshi, Rana; *Proc. Zool. Soc*. India. **2007,** *6 (**1**):121*-127.
[18] AK Pandey; GC Pandey; *Nat. J. life Sci.* **2004,** *1 (2):199*-203.
[19] Benjamin Nidhi, Ameeta Kushwah, RK Sharma; AK Katiyar**;** *Indian J. Expt. Biol.* **2006,** *Vol. 44 pp 228*-232.
[20] R Jain; KD Mishra; *Bull. Environ. Sci.* **2004,** *11*: 1-5.
[21] Y Suzuki; *J. Toxicol. Environ. Health.* **1980,** *6 (3):* 469.