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TOXIC STRESS OF CHROMIUM ON HISTOPATHOLOGY OF KIDNEY IN FRESH WATER TELEOST FISH CATLA CATLA

Zoology

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ABSTRACT

The fingerlings of fresh water teleost fish, Catla catla when exposed to different sublethal concentrations of chromium for a different periods1, 8, 16, 32 days of time brought changes in the structure and morphology of the osmatic organs such as kidney. In trivalent chromium exposed fish, a few pathological changes were seen during the initial days of exposure and then structural reorganization observed in later days of exposure, The degenerative changes observed in the kidney of the fish exposed to the sublethal concentration of hexavalent chromium were, Dilated Bowman's capsule with reorganization, indistinct capillary structure in the shrunken Glomerulus, tubular destruction, vacuolation in the cytoplasm of the tubular cells and a clear increase in periglomerular space and loss of cell architecture and degeneration of cell nuclei were observed.

KEYWORDS:

INTRODUCTION

The concentration of heavy metals is especially high in water bodies that deteriorate the life sustaining quality of water and damage both flora and fauna (Kasherwani, D., et al., 2009; Zyadah, M.A et al., 2000; Lliopoulou - Georguadaki, J.et al., 2001; Verma, et al., 2005; Sharma, et al., 2005). Heavy metals toxicity received considerable attention in aquatic organism especially in fish (Javed, M, 2002). The metals enter fish bodies through body surface, gill or the digestive tract (Vincent, S., 2002). Several workers reported on the effects of metals or chemicals or pesticides and pointed out the architectural damage to brain, gill, liver, kidney, heart, lung, muscle, testis, intestine in various animals (Jayantha Rao, 1982; Radhaiah, 1988; Vijay Joseph, 1989; Vani, 1991; Badri Sriman Narayanan et al., 1993; Manoranjitham et al., 1993; Pondy et al., 1997a, 1997b; Shukla et al., 2001, Glynn, 2003; Garg et al., 2004; Madhaveelatha, 2006; Sivaiah, 2006; Rajendra Prasad, 2007; Sukanya, 2007; Kishandar, 2007; Madhava Rao, 2007;Nagarjuna, 2007; Rajeswari, 2008).

Exposure of animals to contaminated water also causes severe pathological changes at the tissues level. Toxicological histopathology gives useful data concerning the changes induced by toxicants at cellular levels. All the tissues and organs in the body of an animal may be potential targets for the toxic effects of any chemical or heavy metal. The finer cytoarchitectural changes produced during chemical intoxication can be traced by microscopic examinations of the tissues; such studies may explain to certain extent the tissue specificity of the drug action. Their field of study is called histotechnology (Merck Source, 2002 and Stedman's medical dictionaries, 2005).

MATERIALS AND METHODS

Catla catla (Hamilton, 1822), the Indian major carp is an economically important edible fish, having a great commercial value, occurs abundantly in fresh water tanks and ponds, collected from the department of fisheries, Anantapur, Andra Pradesh, and were immediately transported in big fish containers in the laboratory Then they were released into large cement tanks contained of chlorinated tap water. The fish were fed with commercial fish pellets having around 40% protein content, and allowed to acclimatize for 15 days.

Then the fish were isolated into batches having weight of 10±2gms were maintained in static water without any flow. Water was renewed every day to provide fresh water rich in oxygen. The quality of dechlorinated tap water used for the experiment was analysed and various parameters such as dissolved oxygen - 6.8mg/l, alkalinity-130mg/l, hardness-125mg/l and pH-7.3 were measured and maintained. Water temperature was maintained between 22 ± 3oC as recommended by APHA during experiment. During experimentation water was aerated once a day to prevent hypoxic conditions. As the level of toxicity reported to vary with the interference of extrinsic and intrinsic factors like temperature, salinity, PH, hardness of water, exposure period, density of the animals, size, sex etc., (Sivaramakrishna et al., 1991), and precautions were taken throughout this investigation.

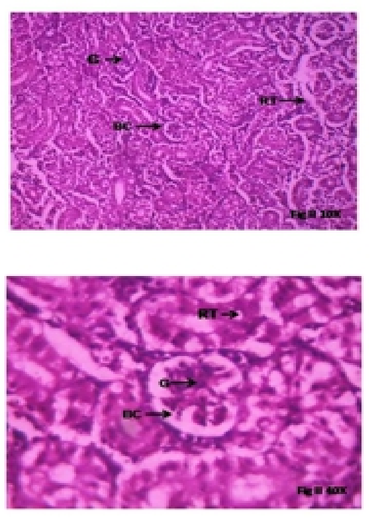
Lethal concentration (LC50) of chromium chloride (trivalent and hexavalent) to fish Catla catla was determined by "Probit method" of Finney (1971). Based on the fact that the effect of a metal on fish becomes consistent within 96 hour of exposure (Eisler, 1977), LC50S/96 hours of trivalent and hexavalent chromium are considered as lethal concentrations. So, about 1/10 th of the 96 h LC50 lethal concentration was taken as sublethal concentration i.e., 59.68mg/l, 100 mg/l(Cr as 35.40mg/lit) were the lethal concentrations, 5.96 mg / l of trivalent chromium and 10 mg /l(Cr as 3.54 mg/lit) of hexavalent chromium respectively was taken as the sublethal concentration for further studies.

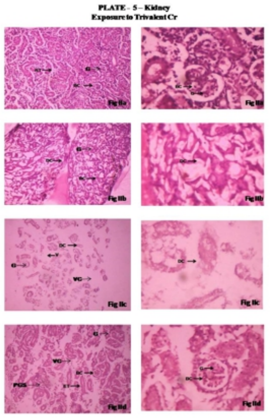
The effects of sublethal concentrations of trivalent and hexavalent chromium on the fish were studied at different periods of exposure in order to understand the influence of time over toxicity. Thus 1, 8, 16 and 32 days were chosen to observe the short term and long term effects of trivalent and hexavalent chromium on the fish Catla catla.After the completion of stipulated exposure period, the fish were sacrificed and isolated tissues such as kidneys under laboratory conditions for biochemical analysis and histopathological studies. These tissues were removed and washed with saline then fixed in buffer formalin (10%) processed for sectioning (5-6um)and staining with haematoxyline and eosin. The histological sections of the kidney were taken by adopting the procedure as described by Humason (1972). Photographs were taken.

PLATE - 4

Fig: II Control Kidney showing clear Bowman's capsule (BC); Glomerulus (G) and Renal tubules (RT), with lower magnification (10X); and Higher magnification (40X).

PLATE - 4
Control Kidney





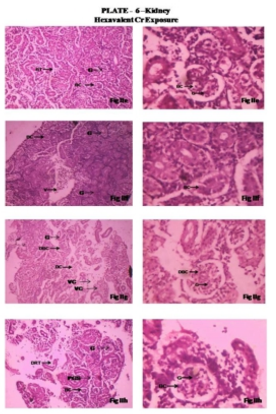


PLATE - 5

Fig: IIa Fish exposed to sublethal conc. of trivalent chromium at day1, kidney with clear Bowman's capsule (BC); Glomerulus (G) and Renal tubules (RT), with lower magnification (10X); and Higher magnification (40X).

Fig: IIb. Fish exposed to day 8, showing shrunken Glomerulus (G), dilation in Bowman's capsule (BC), destruction of tubules takes place, with lower magnification (10X); and Higher magnification (40X).

Fig: IIc. Fish exposed to day 16, dilated Bowman's capsule (BC), shrunken Glomerulus (G), destruction in tubules, vacuolation in cytoplasm (VC) and loss of architecture, with lower magnification (10X); and Higher magnification (40X).

Fig: IId. Fish exposed to day 32, dilated Bowman's capsule (BC), shrunken Glomerulus (G), destruction in tubules, vacuolation in cytoplasm (VC) periglomerular space (PGS), with lower magnification (10X); and Higher magnification (40X).

PLATE - 6 Fig: IIe. Fish exposed to sublethal conc. of hexavalent chromium at day 1, kidney with clear Bowman's capsule (BC); Glomerulus (G) and Renal tubules (RT), with lower magnification (10X); and Higher magnification (40X).

Fig: IIf. Fish exposed to day 8, shrunken Glomerulus (G) indistinct capillary structure, reorganization dilation in Bowman's capsule (BC), destruction in tubules, with lower magnification (10X); and Higher magnification (40X).

Fig: IIg. Fish exposed to day 16, dilated Bowman's capsule (BC), shrunken Glomerulus (G) indistinct capillary structure, destruction tubules, vacuolation in cytoplasm (VC), with lower magnification (10X); and Higher magnification (40X).

Fig: IIh. Fish exposed to day 32, dilated Bowman's capsule (BC), shrunken Glomerulus (G), tubular destruction, vacuolation in tubular cells, increase in periglomerular space (PGS), with lower magnification (10X); and Higher magnification (40X).

RESULTS AND DISCUSSION

The structure of normal control kidney of the fish consists of a clear Bowman's capsule; Glomerulus and Renal tubules were observed (Fig II). No significant changes were observed in the structure of the kidney of the fish exposed for a period of 1 day to trivalent chromium (Fig IIa). On exposure of the fish for a period of 8 days, certain changes were observed in the structure of kidney, as compared to control, shrunken Glomerulus with dilation in Bowman's capsule, destruction of tubules takes place (Fig IIb). On exposure of the fish for a period of 16 days, some degenerative changes were observed in the normal structure of the kidney, dilated Bowman's capsule, shrunken Glomerulus, to some extent destruction in the tubules vacuolation in cytoplasm loss of architecture were observed (Fig IIc). On exposure for a period of 32 days some more degenerative changes were observed dilated Bowman's capsule, shrunken glomerulus, destruction in the tubules, vacuolation in the cytoplasm of the tubular cells and to some extent periglomerular space were observed (Fig Iid).

No significant changes were observed in the structure of the kidney of the fish exposed for a period of 1 day to the sublethal concentration of hexavalent chromium (Fig IIe). On exposure for a period of 8 days, few changes were observed in the structure of kidney, shrunken Glomerulus with indistinct capillary structure, reorganization and also dilation in Bowman's capsule, destruction in tubules and tubular structure were observed (Fig IIf). On exposure for a period of 16 days, some degenerative changes were observed compare to control and day 1 and 8, reorganization and dilated Bowman's capsule, shrunken Glomerulus with indistinct capillary structure, destruction in the tubules to some extent vacuolation in cytoplasm of the tubular cells and loss of cell architecture were seen (Fig IIg).On exposure for a period of 32 days some more degenerative changes were observed with reorganized dilated Bowman's capsule, indistinct capillary structure shrunken Glomerulus, tubular destruction, vacuolation in the cytoplasm of the tubular cells and a clear increase in periglomerular space and loss of cell architecture and degeneration of cell nuclei were observed (Fig Iih).

The degenerative changes observed in the kidney of the fish exposed to the sublethal concentration of hexavalent chromium were supported by the metabolic disorders which occur in it. Dilated Bowman's capsule with reorganization, indistinct capillary structure in the shrunken Glomerulus, tubular destruction, vacuolation in the cytoplasm of the tubular cells and a clear increase in periglomerular space and loss of cell architecture and degeneration of cell nuclei were observed. Kidney is not only the excretory organ but also the osmoregulatory organ of the fish. The cellular damage which occurred in the kidney by the hexavalent chromium induction also impairs the osmoregulatory function which was evident from the impaired oxidative metabolism, which could be one of the main reasons for the death of the fish. Similar reason also suggested by Akhilender Naidu et al., (1983) for the death of Sarotherodon mossambicus exposed to the lethal concentration of mercury.

CONCLUSIONS

The changes which were taken place in the organs of the fish at the initial period of exposure to the sublethal concentration of hexavalent chromium, might be a part of defense mechanism, but on prolonged exposure the further accumulation of hexavalent chromium caused the condensation of the tissue nuclear material, there by the synthetic ability of the metallothionein were repressed and chromium ions destroyed the organ structures. The degree of destruction appeared to be linearly proportional to the period of exposure. The cellular damage which occurred in the kidney by the hexavalent chromium induction also impairs the osmoregulatory function which was evident from the impaired oxidative metabolism, which could be one of the main reasons for the death of the fish.

REFERENCES

1. Akhilender Naidu, K., Abhinendar Naidu, K. and Ramamurthi, R. 1984. Acute effects of mercury toxicity on some enzymes in liver of teleost Sarotherodon mossambicus. Ecotoxicol. Environ. Saf., 8(3):128-133.
2. Gardner, G.R. and Yevich, P.P. 1970. Histological and haematological responses of an esturine teleost to cadmium. J.Fish.Res.Bd.Can., 27: 2185-2196.
3. Gardner, G.R. and Yevich, P.P. 1970. Histological and haematological responses of an esturine teleost to cadmium. J.Fish.Res.Bd.Can., 27: 2185-2196.
4. Garg, U.K., A.K. Pal, G.J. Jha and S.B. Jadhao 2004. Pathophysiological effects of chronic toxicity with synthetic pyrethroid, organophosphate and chlorinated pesticides on bone health of broiler chicks. Toxicologic Pathology, 32:364-369.
5.. Javed, M., 2005. Heavy metal contamination of freshwater fish and bed sediments in the river Ravi stretch and related tributaries. Pakistan J. Biol. Sci., 8(10): 1337-1341.
6. Javed,M. 2002. J.Ani. Vet.Adv. 1: 16-19.
7. Javed,M. 2004. Ind. J.Biol. Sci.1: 59-62.
8. Jayantha Rao, K., Azhar Baig Md., Ramamurthy, R., Madhu, Ch. And Murthy, V.S.R. 1984. Chronic sublethal effects of phosphomidon on a fresh water teleost Tilapia mossambica. Bull. Environ. Sci., 1:3-5.
9. Jha, B.S. and Satheyendra Pandey, 1990. Toxicity of Lead nitrate to the fish, Channa punctatus (Bloch) Bio. J., 2 (1): 151-154.
10. Kasherwani, D., Lodhi, H. S., Tiwari, K.J., Shukla, S. and Sharma,U.D 2009. Asian J. Exp. Sci., 23: 149-156.
11. Lliopoulou-Georguadaki, J. and Kotsanis, N.: Bull. Environ.Condom. Toxicol.,(2001).66: 77-85.
12. Rajeswari, I.S.R. 2008. Toxicity of organophosphate Compound acephate on haematological, selected biochemical parameters and histological studies in wistar strain rats. Ph.D. Thesis, Sri Venkateswara University, Tirupati, India.
13. Sharma, R.K. andAgrawal,M. (2005) : J. Environ. Biol., 26: 301-313.
14. Verma, R.S., Khan,M.A., Tripathi, R., Shukla, S. and Sharma,U.D (2005) : Aquacult., 6: 57-62.
15. Vincent, S., Ambrose, T. and Selvanayagam, M 2002 : J. Environ.Biol., 23: 209-212. 16.Virchow, R. 1858. Die cellular pathologic in ihrur Bergundung auf physiologische and pathologische Gewelebre. Berlin: A. Hirschwold.
17. Zyadah, M.A. andAbdel-Bakey, T.C (2000) : Bull. Environ. Contam. Toxicol., 64: 740-747.