

## RESEARCH ARTICLE

EFFECT OF HEAVY METALS ON FRESH WATER TELEOST FISH *O. MOSSAMBICUS*

Mukesh Kumar Napit

Department of Zoology, Swami Vivkanand Govt. College Berasia, Bhopal

## ARTICLE INFO

## Article History:

Received 05<sup>th</sup> September, 2015Received in revised form 08<sup>th</sup> October, 2015Accepted 10<sup>th</sup> November, 2015Published online 28<sup>st</sup> December, 2015

## Keywords:

Heavy Metals, Histopathology, Liver and Kidney Changes, *O. mossambicus*.

## ABSTRACT

Increasing number and amount of industrial, agricultural and commercial chemicals discharged in to the aquatic environment have lead to various deleterious effects on the aquatic organisms. The liver and kidney plays central role in the process of digestion and excretion metabolism. This organ has a number of functions in the body, including detoxification, plasma protein synthesis, glycogen storage and production of metabolites necessary for digestion. In the present study fish was exposed to 0.25 ppm. of heavy metals (LC50/6 values of 72 hours) up to 18 days. The present study of Cadmium trioxide treated fish, *O. mossambicus* liver, kidney shows disarrayed cords and showing pronounced vacuolization and necrosis. Karyolysis is also observed. Scattered erythrocytes, pronounced damage has been observed. Severe damage in comparison in 18 days exposure indicate that damage in the liver, kidney are indirectly related to dose and duration of treatment.

© Copy Right, Research Alert, 2015, Academic Journals. All rights reserved.

## INTRODUCTION

Fish are especially susceptible to environmental variations and respond more sensitively to pollutants than numerous mammals. Liver, kidney, lungs are a very interesting model for the study of interactions between environmental factors and hepatic structures and functions. As the liver, kidney lungs are involved in detoxification of pollutants, it merits analysis. Cellular coagulation, necrosis, increased cytoplasmic eosinophilia, and subcapsular necrosis foci observed in teleost. Casillas, (1983) Changes found in the diameter, cellular coagulation, and necrosis in the hepatocytes of *H. fossilis* exposed to malathion Datta, (1993), Investigated the chronic toxicity of ammonia in the gills, liver and kidney of juvenile *M. aurata*. Wajsbrot, (1993) Acute and chronic toxicity of ammonia to *Rainbow trout*. Information is available on the pathological changes produced by ammonia in the liver from the study Mukherji, (1975) The fresh water teleost fish, *O. mossambicus*, is an edible fish, and is economically important as the protein food sources of the rural areas of Central Madhya Pradesh. Hence the present investigation was undertaken to study the histopathological changes occurring in the liver gills and kidney after exposure to heavy metals

## MATERIALS AND METHODS

Live specimens of fish *O. mossambicus* were collected from running and standing water bodies of Dist. Bhopal, stored in 50 litre glass aquarium containing dechlorinated water with aeration system, during experiment the fishes fed with leafy green cabbage. The physiological properties of water as follows. PH 7.4 Air temp. 35.0°C, water temp 27.5, Dissolved oxygen 4.56 Bicarbonates 10, Total alkalinity 10.50 and

Acidity 4.36 (expect PH and temp.). All parameters are expressed in mg/l. Animals were acclimatized to laboratory condition for 6 days. During the experiment healthy fishes of uniform size and weight were selected for the treatment. Fishes were divided in to two groups; one group was maintained as control while the remaining one group was separately exposed to the chronic treatment. Each groups contain 6 fishes, were exposed to heavy metals to different concentration separately for studying LC50, in aquaria containing 50 liter of water. During this period the medium was changed 24 hourly. The LC50 values for different intervals 12, 24, 36, 48, 72 and 96h were calculated by Probit analysis method Finney, (1971)

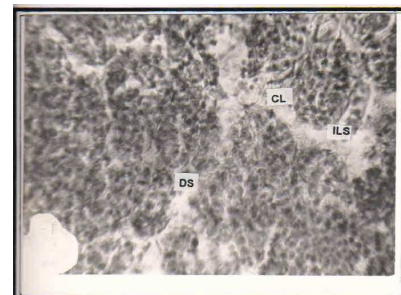
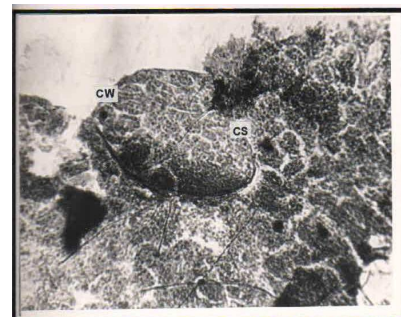


Figure 1

In the chronic treatments the experimental groups of fishes were exposed to 0.5211 ppm. of heavy metals (LC50/6 values of 96 hours) up to 18 days. At the end of 6, 12 and 18 days of treatment, the control and experimental fishes were scarified by decapitation method, liver, gill and kidney were dissected out from the fish and preserved overnight in Bouins and 8% neutral buffered formalin, following paraffin embedding, section were cut out at 3- 6 $\mu$  and were stained with haematoxylin and counter stained with eosin.

## OBSEVATIONS AND RESULTS

The cellular organization was damaged to a greater extent. The hepatic and renal cells lost their normal polygonal shape with excentric nuclei. The cell membrane rupture and fusion between two or more cells took place, exhibiting binucleate, or multinucleate cells at several areas. Some cells became necrotic and complete extrusion of nuclei occurred, after 12 days exposure. The decrease in diameter was due to shrinkage of the cell. The nuclei became pyknotic. There was some degeneration of the cell membrane and vacuolization in the cytoplasm observed after 18 days exposure

## DISCUSSION

Because of the deleterious impact of pollutants in aquatic ecosystem. The histo- cytological responses of fishes to various classes of xenobiotic compounds need to be determined and characterized Hinton, (1978) Histopathological biomarkers of xenobiotic exposure and histopathology of fish liver have been increasingly recognized as valuable tools for the detecting chronic effects of contaminants exposure and uptake on aquatic organisms. Myers, (1998) and Pietrapiana (2002) Hepatic lesions, including neoplasm, focal lesions and degenerative necrosis lesions are commonly detected in fish from contaminated environment Mayes, (1986) Such lesions are similar to those experimentally induced by toxicants or a carcinogenic exposure in fish under chronic exposure to contaminated sediments and diets. Therefore, they have been related to environmental contaminant exposure in several field studies

Moore, (1994) Histopathological lesions caused by pesticides and industrial pollutants have been amply reported in short term as well as long term exposures. Chemically induced liver injury resulting from chronic exposure can produce marked alterations of the entire liver structure with degenerative and proliferative changes. Kendall (1977) Reported histopathological lesions induced in the hepatopancreas of *Channa punctatus* and *Clarias bartachus* exposed to

industrial pollutants mercuric chloride, cadmium chloride and a factory effluent Sastry (1978)

In the present investigation, histopathologically heavy metals treated fish; *O.mossambicus* liver gills and kidney shows disarrayed cords and hyper-trophied hepatocytes showing pronounced vacuolization and necrosis. Pyknotic nuclei and karyolysis are also observed. Hyperemic blood capillaries observed with scattered erythrocytes, pronounced damage has been observed. The major histopathological lesions in the liver and gills of fish, *O.mossambicus* included parenchymal disorganisation, cytoplasmic vacuolation, and nuclear pycnosis. The number of apparently intact hepatocytes was greatly reduced but in such cells nuclear enlargement was seen. Severe damage in comparison in 18 days exposure indicates that damage in the liver gills and kidney are indirectly related to dose and duration of treatment.

## References

1. Bhattacharya, T., Ray, A. K. and Bhattacharya, S., 1984, *Matsya*, 11, 1.
2. Casillas, E. M. Myers, and a Warren, 1983, *Aquatic Toxicol*, 3:61-78.
3. Datta, H., Adhikari, M. S., Singh, N. K., Roy, P. K. and Munshi, J. S. D., 1993 b, *Environ. Contam. Toxicol.*, 51:895-900.
4. Finney, D. J., 1971, *Probit analysis* 3rd ed, 444pp Cambridge University Press, London.
5. Hinton, D. E., J. E. Klaunig and M. M. Lipsky, 1978, *Mar. Fish Rev.*, 40, 47-50.
6. Kendall, M. W., 1977, *Bull. Environ Contam. Toxicol.*, 18, 143-151.
7. Mayes, M. A., Lexender, H. C., Hapkins, D. L. and Latvatis, P. B., 1986, *Environ, Toxicol*, 5, 437 - 442.
8. Miller, D. C., Poucher, S., Cardin, J. A. and Hanson, D., 1990, *Arch. Environ. contam, Toxicol*, 19, 40-48.
9. Moore, M. J. and M. S. Myers, 1994, CRC press, Boca Raton, FL., pp. 327-386
10. Mukherji, S. and Bhattacharya S., 1975, *Indian J. Exp. Biol.*, 13, 571-573.
11. Myers, M. S., L. L. Johnson, O. P. Olson, C. M. Stehr, B. H. Horness, T. K. Collier and B.B. McCain *Toxi.*, 1998, *Pollut. Bull.*, 37, 92-113.
12. Pietrapiana, D., M. Modena, P. Guidetti, C. Falugi and M. Vacchi, 2002, *Mar. Pollut. Bull.*, 44, 238-243.
13. Sastry, K. V. and Gupta, P. K., 1978, *Environ. Res.* 16, 270-278.
14. Wajsbrot, N. A., Gasith, A., Diamant and Popper, D. M. 1993, *J. Fish Biol.*, 42: 321 - 328.

\*\*\*\*\*