International Journal of Enhanced Research in Science, Technology & Engineering
ISSN: 2319-7463, Vol. 13 Issue 9, September-2024, Impact Factor: 8.375

Selenium Induced Histopathological Changes in Gills of Fresh Water Fish *Lepidocephalichthys Thermalis.*

Tejali S. Ayare[1], Trupti V. Mhaskar[2]

[1,2]Dept. of Zoology, B.N.N. College Bhiwandi, Dist.-Thane, Maharashtra.

ABSTRACT

Selenium occurs in soluble forms like selenate. In humans it is a trace element nutrient which functions as co-factor for reduction of antioxidant enzymes. Besides, it also plays a role in the functioning of thyroid. However, it is toxic in large doses. Information on the acute biological effects of the element is limited to birds and mammals. In the present study an attempt has been made to study the effects of selenium on fresh water fish *L. thermalis*. The results of acute toxicity studies are discussed. The findings are supported by histological changes in gills.

Keywords: Selenium, Acute toxicity, Gills.

INTRODUCTION

Selenium is an essential micronutrient for living organisms. It is a component of the unusual amino acids selenocysteine and selenomethionine. In humans, selenium is a trace element nutrient which functions as cofactor for reduction of antioxidant enzymes such as glutathione peroxidases and thioredoxin reductase. It also plays a role in the functioning of the thyroid gland by participating as a cofactor for thyroid hormone deiodinases. Dietary selenium comes from nuts, cereals, meat, fish, and eggs.

Although selenium is an essential trace element it is toxic if taken in excess. Information on the acute biological effects of this elements is generally limited to birds and mammals, although its effects on fish have been reported by a few scientists (Eillis et. al., 1937; Weir and Hine, 1970; Huckabee and Griffith, 1974; Niimi and Laham, 1975; Takahiko Sato et. al., 1980; Hilton et. al., 1980; Aziz and Chambon, 1981; Ward et. al., 1981 Sorensen et. al., 1982 a, 1982 b, 1983 a, 1983b, 1984; Sorensen and Bauer, 1984; Ates, B. et. al.,2008; Ohlendorf, H.M. et. al., 2011; Yeganeh, S. et. al., 2016; Naderi, M. et. al., 2018b; Ziaei-Nejad, S. et. al., 2021; Pan, B. et. al.,2022).

The overall format of the present study consist of toxicity tests exposing groups of test organisms to selenium in discrete solutions. The test was conducted at lethal or acute concentrations. The response chosen for the acute toxicity experiments was percent mortality. In acute toxicity tests, methodology given by Sprague (1973) and static bioassay method standardized by APHA (1975) was followed.

An attempt has been made in the present study to evaluate the intensity of the damage caused to the different organs of fish subjected to acute concentration of selenium. The organ taken for the histopathological study was gills which were in intimate contact with polluted water.

MATERIALS AND METHODS

The test fish were regularly brought from the local supplier and acclimatized for about a two weeks period before being used for tests. During acclimatization they were fed on dried Tubifex worms once a day at a fixed time (9 am). They were maintained in sufficiently large aquaria of 90 x 45 x 45 cms size so as to prevent overcrowding. Aged tap water was used to maintain the fish. It was aerated continuously and water was changed after every two days. The apparently healthy and diseased free fish from the stock were selected for the toxicological studies.

Preliminary small scale pilot tests were performed to determine the range of concentrations for the pollutant. This helped to determine the concentrations to be selected for the final test. The range of concentrations were selected in such a way that

they resulted in 0-100% mortality of the fish tested from 24 to 96 hrs. of exposure. Experiments were performed in triplicate. Control test without pollutant was simultaneously performed exactly under analogous conditions.

At the end of 96 hrs, the fish from control and different experimental groups were scarified. The gills and liver were immediately fixed in aqueous Bouin's fluid. The fixed tissues were reactively processed and paraffin of 5 to 8 microns were cut and stained with hematoxylin and alcoholic eosin.

Observation:

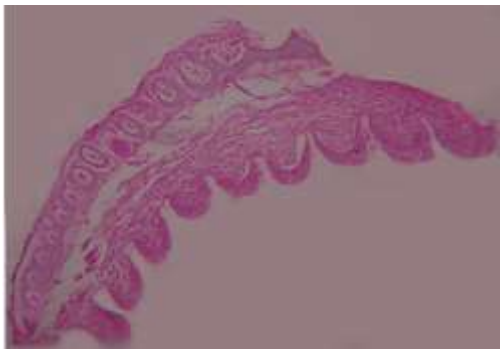
Table 1
%mortality of *L. thermalis* when exposed to various concentrations of Selenium.

Concentration (ppm)	Exposure Period			
	24hrs.	48hrs.	72hrs.	96hrs.
10 ppm	00%	00%	00%	10%
15 ppm	10%	10%	40%	60%
20 ppm	20%	20%	60%	80%
25 ppm	30%	30%	80%	90%
30 ppm	50%	60%	100%	--
40 ppm	80%	100%	--	--
Control	00%	00%	00%	00%

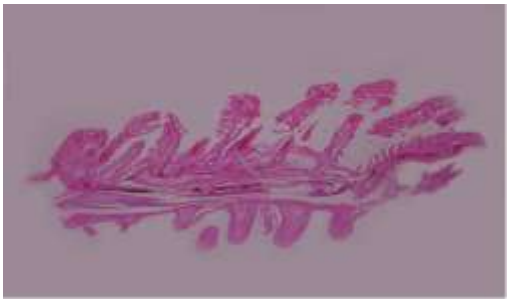
Table 2
LC_{16}, LC_{50} and LC_{84} values for *L. thermalis* exposed to Selenium.

Lethal concentrations of Selenium	Selenium			
	Exposure period			
	24 hrs.	48 hrs.	72 hrs.	96 hrs.
LC_{16}	19.25 ppm	17.25 ppm	10.75 ppm	6.00 ppm
LC_{50}	31.75 ppm	26.25 ppm	17.75 ppm	13.75 ppm
LC_{84}	44.50 ppm	35.50 ppm	24.75 ppm	22.25 ppm

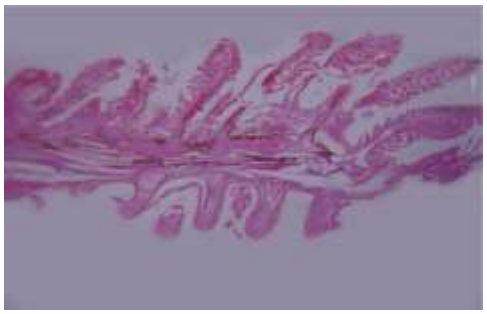
The fish were exposed to the different concentration and mortality was observed after 24, 48, 72, and 96 hours. The tests were started in the morning. The fish were considered dead when there were no respiratory and other movements and showed no response to prodding with a glass rod. Dead fish were removed and examined for morphological changes. No mortality was recorded in control tank during experimental period.



(A)

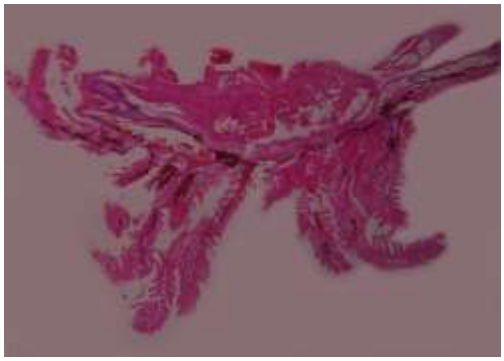


(B)



(C)

(D)



(E)

(A)- Microphotograph showing the structure of normal gill of *L.thermalis.*
(B)- Microphotograph showing the structure of gill of *L.thermalis* exposed to 10 ppm Se.
(C)- Microphotograph showing the structure of gill of *L.thermalis* exposed to 15 ppm Se.
(D)- Microphotograph showing the structure of gill of *L.thermalis* exposed to 20 ppm Se.
(E)- Microphotograph showing the structure of gill of *L.thermalis* exposed to 25 ppm Se

When compared with the gills of control fish, with the gills of fishes subjected to selenium treatment showed pronounced histopathological changes. In experimental fishes, marked degenerative changes were noticed in the epithelial cells, pillar cells, blood cells, cartilage cells and blood capillaries. In different regions of gill filaments debris of damaged cartilage cells and blood cells were observed. Occlusion of interlamellar spaces was comparatively lesser with less accumulation of mucous. The epithelial lining of the secondary lamellae showed stripping away from the pillar cells.

RESULTS AND DISCUSSION

The results of toxicity for selenium is represented in Table 2.2 to 2.4. Table 2.3 indicates the LC_{50} values for selenium which are 31.75, 26.25, 17.5 and 13.75 ppm for 24 hrs, 48 hrs, 72 hrs and 96 hrs respectively.

Observations on the fish ***L. thermalis,*** when exposed to toxicity tests of selenium revealed increased in toxicity with increase in concentration and exposure period. Observations on survival of ***L. thermalis*** is influenced by duration of exposure period and concentration of the toxicant. Increase in mortality rate was observed with increase in concentration of the toxicant and exposure period.

In the present investigation, histopathological changes observed in the gills exposed to selenium can be attributed to irritation followed by injuries to the cells since gills are in direct contact with the toxic medium, they are the organs which are first affected by the lethal and sub-lethal toxic concentrations of many substances (Ellis, 1937; Lloyd, 1960; Crandall and Goodnight, 1963; Baker, 1969; Skidmore and Tovell, 1972; Bilinski and Jones, 1973; Hinton et. al., 1973; Gupta and Rajbanshi, 1979; Srivastava and Srivastava, 1979).

Lethal concentrations of selenium produced severe histopathological changes in the gills of ***L. thermalis***. The gill filaments were seen to be covered by thick mucus film, the fusion of epithelial layers of adjacent gill filaments, hemorrhage & degeneration or complete disruption of cellular and tissue components of gills were some of the changes observed following lethal treatment of selenium. This damage implicated on the gills result in a fall in the capacity of the gills for gaseous exchange.

CONCLUSION

In Sum, it can be concluded that, when the ***L.thermalis*** was exposed to lethal concentrations of Selenium, it affects general physiological status of the organisms. This is reflected by alteration in oxygen consumption. Further it can be concluded that the metal under investigation also damage vital organs of the body like gills which obstacle in the process of respiration. Leading to the death of the fish which is an ultimate effect of any toxicant.

Metals are quite resistance to biodegradation and persist in the environment for long time. It is therefore, necessary to develop proper sites for land disposal of metal waste after suitable treatment. A scientifically designed treatment, storage and disposal Facility (TSDF) would require heavy investment. Regulatory agencies should encourage industry to develop such infrastructure on co-operative basis for proper management of their wastes.

In order to bridge the gap between availability and demand of certain metals like zinc, lead in the cadmium, mercury, selenium, chromium, etc. in the country. There is a need to encourage their reuse, recovery and recycling. Laws should be made to allow imports of wastes containing over 60 percent of residue of above mentioned heavy metals by such importers who have environmentally sound technology to recycle metals and disposal facility for leftover residue. Imports of rest of metal wastes should be strictly prohibited.

Industry based on metals including metal processing and recycling itself is largely concentrated in small scale and medium scale sector. Due to lack of modernization in this sector serious threat is caused to environment and sustainable development. Technocrats and decision makers play important role to cope up this problem. There is an urgent need for standard setting process. Social cultural, economic and nutritional status of our population should be taken into consideration while setting standards.

REFERENCES

1. APHA; AWWA and WPCF. 1975. Standard methods for the examination of water and waste water 14th ed. Washington D.C.
2. APHA 2017. Standard Methods for the Examination of Water and Wastewater (23rd ed.). Washington DC American Public Health Association.
3. Ates, B., Orun, I., Talas, Z.S., Durmaz, G., Yilmaz, I., 2008. Effects of sodium selenite on some biochemical and hematological parameters of rainbow trout (*Oncorhynchus mykiss* Walbaum, 1792) exposed to Pb 2+and Cu 2+. Fish Physiol. Biochem. 34, 53–59. https://doi.org/10.1007/s10695-007-9146-5.
4. Aziz G, and Paul Chambon. 1981. Fluormetry of selenium in urine Clin. Chem. 26 (9), 1272 – 1274
5. Baker, J.T.P. 1969. Histological and electron microscopical observations on copper poisoning in the winter flounder *(Pseudopleuronectus americanus)*. J. Fish. Res. Bd. Can. 26: 2785-2793.
6. Bilinski, E. and Jonas. R.E.E. 1973. Effects of cadmium and copper on the oxidation of lactate by rainbow trout *(salmo gairdneri)* gills. J. Fish. Res. Board. Can. 30: 1553-1558.
7. Crandall C.A. and C.J. Goodnight. 1963. The effects of sublethal concentrations of several toxicants to the common guppy *Lebistes reticulates*. Trans Am. Microsc Soc 82, 59-73.
8. Ellis M.M., Motley, H.L., Ellis, M.D. and Jones R.O. 1937. Selenium poisoning in fishes Proc. Soc. Exp. Biol, Med, 36: 519-22
9. Gupta A.K. and V.K Rajbhanhsi. 1979. Pathological changes resulting from bioassay of copper to *Heteropneustes fossils* (Bloch) Proc. Symp. Environ. Biol 167-172. Academy of Enviromental Biology, India.
10. Hinton D.E., M.W. Kendall and B.B. silver. 1973. Use of histologic and histochemical assessments in the prognosis of the effects of aquatic pollutants. Biological methods for assessment of water quality. ASTM STP 528, American Society for Testing and Materials. 88:194-208
11. Hilton J.W., Hodson P.V. and Singer S.J. 1980. The requirement and toxicity of selenium in rainbow trout *(Salmo gairdneri)* J; Nutr, 110, 2527-3
12. Huckabee , J.W. and N.A.Griffith. 1974. Toxicity of mercury and selenium to the eggs of *(Cyprinus Carpio)* Trans, Am. Fish. Soc 102: 822-825
13. Lloyd R. 1960. The toxicity of zinc sulphate to rainbow trout Ann. Appl. Biol. 48, (1), 84-94
14. Naderi, M., Salahinejad, A., Ferrari, M.C.O., Niyogi, S., Chivers, D.P., 2018b. Dopaminergic dysregulation and impaired associative learning behavior in zebrafish during chronic dietary exposure to selenium. Environ. Pollut. 237, 174–185. https:// doi.org/10.1016/j.envpol.2018.02.033.
15. Niimi A.J. and Q.N. Laham. 1975. Selenium toxicity on the early life stages of zebrafish *(Brachydanio rerio)* J. Fish. Res. Board Can. 32: 803-806
16. Ohlendorf, H.M., Covington, S.M., Byron, E.R., Arenal, C.A., 2011. Conducting sitespecific assessments of selenium bioaccumulation in aquatic systems. Integr. Environ. Assess. Manag. 7, 314–324. https://doi.org/10.1002/ieam.157.
17. Pan, B., Wang, Y., Li, D., Wang, T., Du, L., 2022. Tissue-specific distribution and bioaccumulation pattern of trace metals in fish species from the heavily sedimentladen Yellow River, China. J. Hazard Mater. 425, 128050 https://doi.org/10.1016/j. jhazmat.2021.12805
18. Skidmore J.F. and PWA Tovell. 1972. Toxic effects of Zn sulphate on the gills of rainbow trout water Research 6, 217-230
19. Sorensen E.M.B. and T.L. Bauer. 1984. A correlation between selenium aculumulation in sunfish and changes in condition factors and organ weight Environ pollut Ser A 34, 357-66

20. Sorensen E.M.B. Harlon C.W. and Bill J. S. 1982 b. Renal changes in selenium exposed fish., Am. J. Foresn Med, Pathol 3 : 123 – 129.
21. Sorensen E.M.B., Cumble P.M., Baure T.L., Bell J.S. and Harlan C.W. 1984. Hispathological, hematological condition factor and organ weight changes associated with selenium accumulation in fish from Belews lake, North Carolina Arch, Environ Contamin Toxicol 13, 153-62
22. Sorensen E.M.B.., Bell J.S. and Harlan C.W. 1983 b. Hispathological changes in selenium exposed fish. Am. J. Forens Med. Pathol 4. 111-23.
23. Sorensen, E.M.B., Bauer, T.L.Bell. J.S. and Harlan, C.W. 1982 a. selenuim accumulation and cytotoxicity in teleost following chronic environmental exposure. Bull Environ Cantamin and Toxicol 20, 688 – 696.
24. Sprague J.B. 1973. The ABC's of pollutant bioassay using fish in biological methods for assessment of water quality (ed) J. Cairnor and K.L. Dickson Am. Soc. For testing and materials 6-50
25. Srivastava and Srivastava. 1979. Toxicity of urea to *channa punctatus* (Bloch) tolerance and histopathological changes in certain tissues. Proc. Symp Environ Blot 183 – 189.
26. Takahiko Sato, Youki, OSE and Tsutomu Sakai. 1980. Toxicological effect of selenium on fish, Environ pollut (Ser A) 21: 217-224.
27. Thomas, J.K., Wiseman, S., Giesy, J.P., Janz, D.M., 2013. Effects of chronic dietary selenomethionine exposure on repeat swimming performance, aerobic metabolism and methionine catabolism in adult zebrafish (Danio rerio). Aquat. Toxicol. 130, 112–122. https://doi.org/10.1016/j.aquatox.2013.01.009.
28. Ward G.S., T.A. Hollister, P.T. Heitmuller and P.R Parrish. 1981. Acute and chronic toxicity of selenium to estuarine organisms, Northeast Gulf Sci 4(2) 73-78
29. Weir P.A. and C.H. Hine. 1970. Effects of various metals on behaviour of conditioned Goldfish, Arch. Environ Health 20: 45-51.
30. Yeganeh, S., Adel, M., Ahmadvand, S., Ahmadvand, S., Velisek, J., 2016. Toxicity of organic selenium (Selemax) and its effects on haematological and biochemical parameters and histopathological changes of common carp (Cyprinus carpio L., 1758). Toxin. Rev. 35, 207–213. https://doi.org/10.1080/15569543.2016.1213749
31. Ziaei-Nejad, S., Hosseini, S.M., Mortezaei, S.R.S., 2021. Effects of selenium nanoparticles supplemented feed on biochemical indices, growth and survival of Yellow-Tail Seabream (Acanthopagrus latus). J. Agr. Sci. Tech. 23, 1001–1011. http://jast.moda res.ac.ir/article-23-44533-en.html.