

Toxicology Study on Lead Nitrate Induced Fresh Water Fish *Cirrhinus mrigala* (Hamilton)

S.Celine Hilda Mary^{1*}, S.Silvan², EK.Elumalai³

¹Assistant Professor , P.G. and Research Department of Biochemistry
St.Joseph's College of arts and science (autonomous),
Cuddalore-1.Tamilnadu, India.
celinehildamary@yahoo.com

²Assistant Professor, Department of Biochemistry,
Achariya Arts and Science College,
Villianur, Puducherry-605110. , India.
bio.silvan@gmail.com

³Assistant Professor, Department of Applied Microbiology,
Achariya Arts and Science College,
Villianur,Puducherry-605110. India.
elumalai.mi@gmail.com

Abstract

Heavy metals occur naturally in the marine environment. These heavy metals enter the aquatic systems by direct discharges via industrial and urban effluents. The heavy metals leads to affect the aquatic environments, So Toxic effect study was conducted to investigate the effect of lead nitrate induced histological alterations in the gills of freshwater fish, *Cirrhinus mrigala* which were kept in aqueous solution of lead nitrate of lethal concentrations of 24 days. The histopathological effect of lead nitrate in the gills. liver and muscles of fish is characterized by complete fusion of gill lamellae, edema in the filamentary epithelium etc. Therefore, the present investigation illustrates that the presence of lead nitrate in water are toxic to fishes.

Keywords: *Cirrhinus mrigala*, Toxic, Lead nitrate ,Fish

1. Introduction

Millions of fish are killed every year by a wide variety of different pollutants from many sources like municipal, agricultural and industrial. From industrial and agricultural operations, these compound find their way in to the natural water resources and affect the aquatic organism [1]. Some toxicants contained in the industrial effluents have been reported to be toxic, depending on the dose and exposure duration, and they can impart serious damage to aquatic life [2]. The pollutants build up in the food chain are responsible for the adverse effects and finally death of aquatic organisms.

Heavy metals are serious pollutants in the water environment and are accumulated by aquatic organisms [3,4]. Certain metals (such as lead) and its compounds accumulate in the wild fish, particularly in gills and liver tissues [5]. Because lead occurs in nature and it can arise from a variety of sources, it is important to know more about the toxicity of this metal and chronic effect of its compounds

in water. Pollution by lead metal has been recognized for a long time; it does not affect merely aquatic life, as contamination to have occurred everywhere on Earth by industrial and many natural activities.

Fish are relatively sensitive to changes in the surrounding environment including the increase in pollution as showed by previous studies. Early toxic effects of pollution may, however, only be evident on cellular or tissue level before significant changes can be identified in fish behavior or external appearance. The present investigation was therefore conducted to determine the toxicity, lead to fresh water fish species and responses of different age groups to metal's toxicity.

2. Materials and Methods

2.1. Sample

The live specimens of adult fresh water fish *cirrhinus mrigala* (hamilton),(both sexes, body weight 30-100g) were collected from the pond water located at the Nellikuppam

area in Cuddalore district. They were acclimatized to the laboratory condition for few hours in plastic fish tanks. Physiochemical characteristics of the tap water used in experiment were temperature 26°C and pH-8. The toxicant was dissolved in water and mixed well in fish tank. The tanks were provided with continuous aeration and were maintained under normal condition. During acclimatization, the fish were fed with commercial pelleted food or fish feed, 2-3 times per day. The water was renewed after four days and the concentration of toxic substance was changed for every 96 hrs.

2.2. Biochemical analysis

In the present investigation 96h LC50 was taken to study the effect of lead nitrate on biochemical constituents. The vital tissues like muscle, liver, gill, of the fish were taken for the estimation of total protein, cholesterol and for the estimation of the activity of the four enzymes viz ALP, AST, ACP, SGOT, SGPT and for the estimation of the antioxidant activity viz., catalase, & ascorbic acid.

2.3. Experimental design

In short term exposure the fish were subjected to 0.052mg/l (0.001 of 96hrs LC50). In the second time exposure, the fish were subjected to 0.52mg/l (0.01 of 96hrs LC50) for the duration of 8 days at two time interval of 4days. The third time 1.04g/l (0.1 of 96hrs LC50) for the duration of 12 days at three time interval of 4 days. Concurrently a control group was also used for comparison by using the saline water. Six fishes from each group was sacrificed at the end of 24th day.

3.Result

The results showed lead nitrate administration resulted in a significant increase in the activities of GPT, GOT and ACP. ALP showed a significant decrease in the level than in control group (Table.1).

The activities of enzymic antioxidants (SOD and CAT) were significantly decreased in the tissues (gill, muscle and liver) of lead nitrate in toxic fish(Table.2)

The level of cholesterol increased in toxic fish. The liver has an higher concentration than in muscle and gill. The decrease in the level of protein, when compare to the control group. In protein the organ level will be increased.

Table-1: Variations of enzyme activity in Gill, Muscle and Liver exposed to different concentration of lead nitrate.

Parameter	Gill		Muscle		Liver	
	Normal	Toxic	Normal	Toxic	Normal	Toxic
GOT	23.6±2.41	25.8±2.03	21.8±2.15	23.5±2.20	24.1±2.51	25.0±2.53
GPT	96.2±4.16	98.0±4.3	95.8±4.08	96.3±4.10	97.1±4.631	98.0±4.71
ALP	8.0±4.42	7.4±4.309	8.9±4.63	8.1±4.37	7.8±3.87	7.0±3.53
ACP	25.6±3.6	30.3±3.758	21.9±2.94	23.6±3.11	27.1±3.44	27.9±3.53

Table-2: Variations of antioxidant activity in Gill, Muscle, and Liver exposed to different concentration of lead nitrate

Parameter	Gill		Muscle		Liver	
	Normal	Muscle	Normal	Toxic	Normal	Muscle
CAT	136.6 ± 12.60	125.8 ± 11.05	125.8 ± 13.8	122.1 ± 13.10	114.8 ± 11.8	109.0 ± 11.3
ASCO	23.4 ± 4.07	21.1 ± 3.83	21.8 ± 2.90	20.07 ± 2.76	20.6 ± 3.0	20.0 ± 2.9
RBIC						
ACID						

Table-3: Variations of biochemical parameters in gill, muscle and liver exposed to different concentration of lead nitrate

	Gill		Muscle		Liver	
	Normal	Toxic	Normal	Toxic	Normal	Toxic
Protein	8.4±3.69	6.9±3.431	7.2 ± 3.544	7.0 ± 3.4	9.8±4.017	8.6 ± 3.9
Cholesterol	114.6 ± 3.4	120.9 ± 3.5	130.87 ± 4.56	134.0 ± 4.9	133.41± 4.86	136.0 ± 5.2

3.1.Histopathological studies

Histopathological studies of Gill, Muscle and Liver of fishes in different concentration of Lead Nitrate intoxication are shown.

In Gill the secondary lamellae are intact and the mild hypertrophy in seen. In toxic fish after the exposure of lead nitrate the fragmentation of primary lamellae and the erisions in secondary lamellae are seen(Figure 1,2).

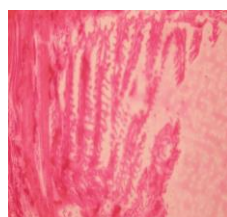


Fig.1: Normal Gill

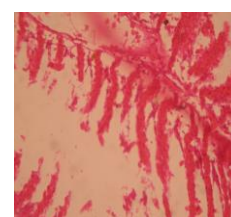


Fig.2: Toxic Gill

In Muscle mild intra muscular edema was observed . In toxic fish the elongation and separation of muscle fibers observed and the fibers are seem to be less compact after the exposure of different concentration of lead nitrate on fish(Figure 3,4).

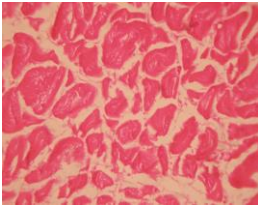


Fig.3: Normal Muscle

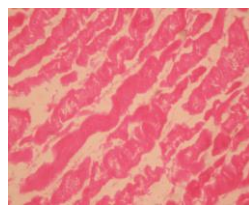


Fig.4: Toxic Muscle

In liver the normal liver architecture were changed after the sublethal concentration. It shows complete disintegration. Marked necrosis with hyper vacuolization is observed (Figure 5,6).

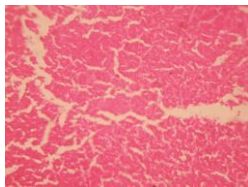


Fig.5: Normal liver

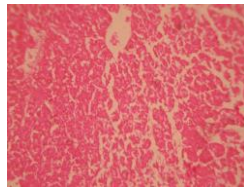


Fig.6: Toxic liver

4. Discussion

The level of GOT, GPT, ALP and ACP in gill, muscle, and liver was found that the enzyme activities were increased after the exposure to lead (II) nitrate toxicity. GPT, GOT and ACP shows different levels after the exposure of lead nitrate, the gill shows increased concentration than in liver and muscle. In ALP the increased concentration in muscle than in liver and gill.

GPT levels increase in toxic hepatitis and viral hepatitis has reported changes in biochemical constituents. GOT and GPT values increase in experimental fish and rise in the activities of transaminases is due to toxication which suggests enhanced protein catabolism and probable hepatocellular damage in the organism.[6]

The phosphatase (ACP and ALP) are important biomarkers because they are involved in adaptive cellular response to the potential cytotoxicity and genotoxicity of pollution. The increase in some of the concentration may be as results of liver damage or arrested bone growth[7]. Table.3 Shows the level of SOD, CAT and ascorbic acid in gill, muscles, and liver. It was found that the antioxidant activities were decreased after the exposure to lead toxicity. The decreased activities of SOD, CATALASE AND ASCORBIC ACID

may indicate disturbance in the cell organells. Such damage to cell organells has been reported in various studies[8]

Histopathological alterations can be used as indicators of the effects of various pollutants on the organism including fish, and reflection of the overall health of the entire pollution. According to studies shown that the exposure of fish to pollutants, that is agricultural and industrial chemicals, were resulted in several pathological changes in different tissues of fish[9].

In the present study showed that the exposure of *Cirrhinus mrigala* to lead nitrate caused pathology in fish organs such as gill, muscle and liver, so based on the test report, we conclude that heavy metals(Lead Nitrate) increase the toxicity to aquatic environment and leads affect fish. In future heavy metal resistant fish varieties might be develop to overcome this problems.

References

1. Tilak KS, Veerajah K, Thathaji PB. and Butchiram MS.Toxicity studies of butachlor to the freshwater fish, *Channa punctatus*(Bloch). *J. Environ. Biol.* 2007 28: 485-487.
2. Vinodhini, R. and Narayanan 2009. The impact of toxic heavy metals on the hematological parameters in common carp, *Cyprinus carpio* L.Iran. *J. Environ. Hlth. Sci. Eng.* 6: 23-28.
3. Lioyd M. Pollution and fresh water fish. Fishing new books.1992.pp. 77-85.
4. Papatransion E King PE. Ultra structure studies on the relation to cadmium accumulation. *Aquat. Toxicol.*, 1993; 3(40):273-284.
5. Crespo S, Nannotte G, Coline DA, Leray, C. Nonnote L and Aubree A. Morphological and functional alterations induced in trout intestine by dietary cadmium and lead. *J. Fish. Biol.*, 1986;28(1):69-80.
6. Praful, Godkar B, Darshan, Godkar. P. Textbook of medical laboratory echnology, Bhalani Publishing House, Mumbai, India, 2003.
7. Leohner TW, Reash RJ. Williams M. Assessment of tolerant sunfish populations (*Lepomis spp*) inhabiting selenium- laden coal ash effluent. *Tissue biochemistry evaluation. Ecotoicol. Envir. Saf.* 2001;50.217-224.
8. Bhattacharya A, Chatterjee A, Ghosal S, Bhattacharya SK. Antioxidant activity of active tannoid principles of *Emblca officinalis* (amla). *Ind. J. Exp. Biol;* 1999; 37: 676-680.
9. Mohamed FAS. Histopathological studies on *Tilapia zillii* and *Solea vulgaris* from Lake Qarun, Egypt. *World J. Fish Mar. Sci.* 2009.1: 29-39.

Author profile:

S.Celine Hilda Mary.

Assistant Professor ,

P.G.and Research Department of Biochemistry, St.Joseph's college
of arts and science (autonomous), Cuddalore-1.Tamilnadu,

India.celinehildamary@yahoo.com