EUROPEAN JOURNAL OF BIOMEDICAL AND PHARMACEUTICAL SCIENCES

http://www.ejbps.com

ISSN 2349-8870
Volume: 8
Issue: 10
435-440
Year: 2021

ADVERSE EFFECT OF CHROMIUM IN PISCES–TOXICITY EVALUTE IN *L.ROHITA*

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Article Received on 21/08/2021 **Article Revised on 11/09/2021** **Article Accepted on 01/10/2021**

ABSTRACT

Chromium toxicity is the affected by valance state chromium (III) is generally less toxic than the chromium (VI). There is equilibrium between the two forms under different conditions. Chromium resistance bacteria capable of reducing or transforming hexavalent to trivalent chromium were isolated from soil and water. Chromium apart from being an important metal present in substantial threat to aquatic life as well as human being. Because soluble chromium is extremely pernicious and shows mutagenic effects, due to its strong oxidizing nature. It destabilizes the ecosystem due to their toxic impact on biota and bioaccumulation in certain organisms. Occurrence of chromium varies in fishes, depending upon their ages, development as well as other physiological variables. It also produced cytotoxicity and detrimental impact on behaviour of fish such hypertrophy and paraplegia at gill epithelium, uneven swimming and suspended feeding. Various research studies indicated adverse effects of chromium in fish at haematological level, like anaemia, thrombocytopenia, decrease in haemoglobin and total erythrocyte count. At biochemical level, mostly decline in the contains of glycogen. Lipids and proteins were observed.

KEYWORDS: Chromium resistance bacteria, biological oxygen demand, chemical oxygen demand, extremely pernicious and mutagenic effects, toxic effect, haematology.

INTRODUCTION

Bioaccumulation of heavy metals contaminate land air and water. Industries such as cotton textiles, tannery, electroplating, iron and steel manufacturing industries, rubber paints are the major industries which polluted water. The major source of chromium is tannery and leather producing industries.[37] Chromium enters into water bodies and effect on biological and chemical oxygen demand.

Hexavalent chromium is a toxic industrial pollutant and classified carcinogen possessing mutagenic and teratogenic properties.[3] The field of ecotoxicology, fundamental research on the impact of toxic chemicals on terrestrial, freshwater, and marine ecosystems at the population or community level has undergone a particularly strong surge in aquatic system.[3] This scientific data discussed in this review provide a basis for understanding the potential impact and for advancing of our knowledge of the eco–toxicological effects and risk assessment of chromium.[3]

FATE OF CHROMIUM IN THE ENVIRONMENT

In biodegradation of chromium soils contain organic and other reducing agents which help to reduce hexavalent chromium to trivalent chromium. It is proven that Cr^{+6} is a strong oxidizing agent and can also be reduced by electron donors because low concentration oxygen is responsible for the reduction.[37]

$2CrO_4^{-2} + 2H_3O^- \leftrightarrow CrO_7^{-2} + 3H_2O$

$HCrO_4^- + HCrO_4^- \Leftrightarrow Cr_2O_7^{2-} + O$

$2Cr_2O_7^{2-} + 3C° + 16H^+ \longrightarrow 4Cr^{3+} + 3CO_2 + 8H_2O$

The pH of water has a profound influence on the reduction of chromium in the environment. Dimers predominant at pH=6.5 whereas monomers are more abundant at extremely low pH values (0.1).[3] A significant occurrence of dimmers is observed in Cr^{+6} solution is greater than 30mM, whereas monomers are dominant in concentration below 30mM.Microbial processes also transform Cr(IV) to the insoluble trivalent form which adsorbs on solid surfaces.[3] The most microbial reductions of Cr(IV) can be reduced to Cr(III) that precipitates as a fairly insoluble hydroxide.

CHROMIUM TOXICITY TO FISH

The aquatic toxicology of Cr depends on both biotic and abiotic factors. The biotic factors include the type of species, age and developmental stage. The temperature, concentration of Cr, oxidation state of Cr, pH, alkalinity, salinity and hardness of water constitute the abiotic factors. Moreover, lethal and sub–lethal concentrations of the metal and its speciation also determine the sensitivity of the individual organism.[3]

ACUTE TOXICITY

Acute toxicity occurs almost immediately in hours or days after an exposure. An acute exposure is usually a single dose or a series of doses received within a 24 hours period. Death is major concern in cases of acute exposure. Hydrogen cyanide, hydrogen sulphide, nitrogen dioxide etc. Acute toxicity is expressed in Lethal dose 50ppm and Lethal concentration 50ppm. This is the amount or concentration of the toxicant required to kill 50% of test animals. LD_{50} values are recorded in milligrams of toxicant per kilogram of body weight of the test animal–mg/kg or in parts per million–ppm. LC_{50} values are recorded in milligrams of toxicant per volume of air or water–ppm. Toxicants with high LD values are considered the least acutely toxic.

Table 1: Chromium content in fish with acute effects.[1]

Fish species	Chromium	Acute effects
Labeo rohita	39.40mg/L 96h–LC_{50}	Decreased in glycogen content, total lipid content and protein content of muscle, gill and liver.

EFFECT OF BEHAVIOUR OF FISHES

In the acute study with different concentration of potassium dichromate solution. It has been revealed that Rohu fingerlings lose its body balance after 24 hours when exposed to 28.99mg/l concentration. Activeness and swimming rate of the fingerlings have been found to vary after 24 hours when exposed to 56.59mg/l concentration. Fingerlings are found to be restless with highly decreased body balance and higher rate of mucus secretion at the aforesaid condition.[2]

Table 2: 96 h LC_{50} Values (mg/l) of Chromium toxicity for different experimental fishes.[1]

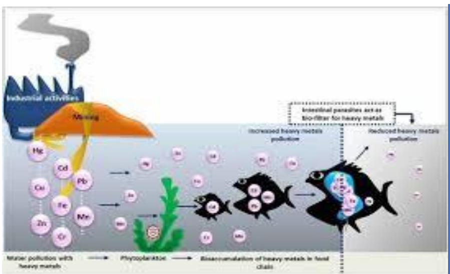
Sl.No.	Test Organisms	Time of Exposure	Test Conditions and Organism Size (if available)	96 h LC_{50} Values of Cr Concentration (mg/l)
1	*Labeo rohita*	96h	Renewal, (27.5±1)°C	39.40
		96h	Static; (22.7±1)°C	30.36
		72h	fingerlings; Temp(°C) 27.4–33.7	38.19
		48h	Temp (°C) 27.4–33.7; pH 7.4–7.8;	53.42
		24h	D.O. (4.6–6.2) mg/l; Total Alkalinity (140–190) mg/l; Total Hardness (120–230) mg/l	58.76

EFFECT OF MORTALITY OF FISHES

Acute toxicity test can provide environmental data very rapidly. According to the data we came to know that 96 hours LC_{50} value is 30.36mg/l and 33.39 mg/l in case of *Labeo rohita* (fingerlings). In case of *Labeo rohita* 50% lethal concentration value of chromium for the potassium dichromate salt is 39.40 mg/l.[2] In the experiment different ages of Rohu fishes are affected by rainbow trout for acute concentration of Cr (VI) who's sensitive 1.16–2.52times.[11] The exact causes of mortality due to heavy metal poisoning have been found to be multiple and depend on time concentration combinations. For the instance fluctuations in pH, temperature and other water quality parameters have some influence on the value of LC_{50}.[2]

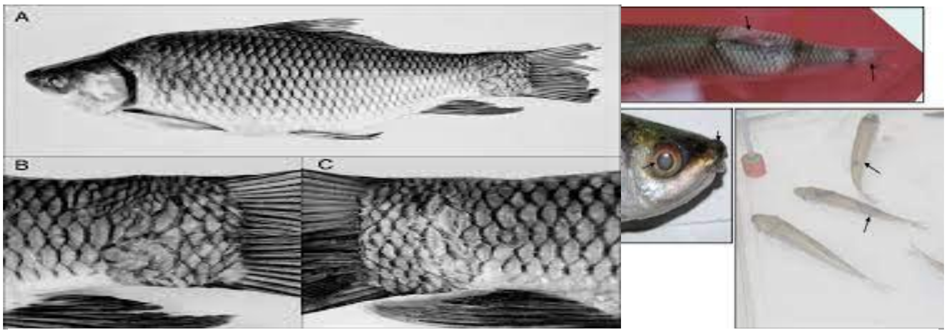
EFFECT ON CELLULAR SYSTEM

Due to acute toxicity fishes are also affected by cellular system. *In–vitro* cytological investigations have revealed that metal toxicity exerts influences on several cytological parameters including measurement of death of cell.[2] The inhibitory concentration (IC_{50}) values for the metals have indicated both Cr and Cd exert a more pronounced cytotoxic effect than the other metals (Zn & Cu).[2]



(1) Water is polluted by heavy metals.

(2) Toxic effect of Rohu fish.



(3) Fingerling following heavy metals. (4) scale deformities of Rohu fish.

Figure–1: Abnormalities in Rohu fish

EFFECT ON IMMUNE SYSTEM

The immune response and non–specific immunity in Rohu fish exposed to sub lethal concentrations of tannery effluent containing chromium calcium carbonate and sodium sulphate. Nonspecific immune mechanisms have been evaluated in terms of serum lysozyme activity, the production of intracellular reactive oxygen species (ROS) and reactive nitrogen intermediates (RNI) by peripheral blood leucocytes during laboratory studies. The chronic exposure of fish to 0.53% concentration of tannery effluent significantly suppressed the antibody response, nonspecific serum lysosome activity, and ROS and RNI production.[2] Similar responses have also been detected in fish exposed to a low concentration of tannery effluent, although to a lesser extent.

EFFECT ON BIOCHEMICAL TOXICITY

Chromium induces cumulative deleterious effects at both biochemical and enzymatic levels. *In-vitro* effect of Hg, Cd, Ni, Cr and Zn ions on the kinetic properties of NADPH cytochromeP_{450} reductase purified from leaping mullet. The results of indicate that Cr independents of its oxidation state, is a strong inhibitor of CYP_{450} reductase activity in fish.[11] This experiment was designed to investigate the hepatotoxicity of As and Cr (VI) to *L.rohita* at LC_{50} concentration for 24 hours and 96 hours exposure. Cr can alter the glucose transport rate in epithelial cells of the intestine. Glucose uptake by epithelial cells in the intestine of rainbow trout showed a diminished rate of glucose absorption. Some biochemical profiles were investigated in various organs like gill, liver, and muscle of *L.rohita* at lethal concentrations Cr (39.4mg/L).[3] The results of the study showed that glycogen, lipids, and protein levels were diminished significantly in all organs, which could be due to metallic stress or the prevalence of hypoxic or anoxic conditions. According to information that *L.rohita* exposed to Cr exhibits hypoxia with lower oxygen consumption.[11]

Table 3: Major histopathological alterations in different tissues of ROHU fresh water fishes.[1]

Tissues	Major Alterations
Liver	Hyperplasia, Necrosis of hepatic cells, Cellular disorganization in *Labeo rohita* and *Heteropneustes fossilis* after 60 days exposure to 96 h 1/10th $LC_{50.}$ Reduction of nucleus to cytoplasm ratio in liver cells in Rainbow Trout.
Kidney	Highly fenestrated Bowman's capsule, Constricted lumen of Renal tube, Glomerular disorganization in *Labeorohita* and *Heteropneustes fossilis* after 60 days exposure to 96 h 1/10th $LC_{50.}$
Intestine	Inner epithelial layers highly degraded in *Labeo rohita* and *Heteropneustes fossilis* after 60 days exposure to 96 h 1/10th $LC_{50.}$
Muscle	Loosening of muscle fibre with increased space between fibres in *Heteropneustes fossilis* after 60 days exposure to 96 h 1/10th $LC_{50.}$
Gill	High Lamellar degradation. Necrosis in epithelial cells. Thickening of blood vessels, Atrophied central axis after 60 days exposure to 96 h 1/10th LC_{50} in *Labeo rohita* and *Heteropneustes fossilis*. Hyperplasia of epithelial cells and epithelial lifting of secondary lamellae in Rainbow Trout.

Cr (VI) has been reported to inhibit the ion–dependent ATPases in gills, kidney, and intestine of costal teleost at different concentrations (5, 19, 15 mg/L) with a general dose and duration dependent trend.[3] The finding that Cr inhibits the activity of the ATPases in all three organs is crucial to understanding the toxic effects of the metal because such alterations have a great impact on osmoregulatory function and the transport system along the cell membrane. Similar results on the loss of osmoregulatory and respiratory abilities have been reported in rainbow trout.[3]

HEMATOLOGY AND IMMUNE SYSTEM STUDIES

Hematologic indices have different sensitivities to various environmental factors and chemicals. In fish, changes in these parameters and their peculiarities depend upon the concentration of heavy metals and duration of exposure. The alterations in the hematologic indices of fresh water fish exposed to Cr(VI) are well documented, and the metal is reported to induce a decrease in most blood parameters investigated. Studies on *L.rohita* exposed to Cr (VI) are well documented and the metal is reported to induce a decrease in the present of haemoglobin (Hb) and the total erythrocyte count at the end of both 24 hours and 96 hours.[3] After getting further studied that the anaemic state of the fish and could be due to iron to deficiency and its consequent decreased utilization for Hb synthesis. To expose of using potassium dichromate at different pH levels have shown blood coagulation an increasing in clotting time. The results are typical of thrombocytopenia, a condition caused by a deficiency of platelets in the blood. The Hb concentration significantly decreased at high pH and slightly increased at pH 5.0.[3] Fish exposed to all Cr concentrations had lower antibody titre values, reduced numbers of splenic and kidney plaque–forming cells, and high counts of splenic lymphocytes but reduced counts of kidney cells when compared with the control group. At all concentrations of Cr, a dose dependent Cr accumulation in kidney, liver, and spleen was found. A dose–dependent decrease in erythrocyte counts, Hb content, and packed cell volume indicated anaemia. The oxidative function of mitochondria was affected even at mM concentrations of Cr (VI). Fish exposed to Cr for 28 days also exhibited higher susceptibility to *Aeromonas hydrophila* infection than control fish.[3] Trout liver mitochondria were incubated in the presence of a micromolar concentration of potassium dichromate under several experimental conditions. Cr(VI) strongly inhibited both state 3 and state 4 respiration supplemented by NAD–linked substrates and slightly affected the respiration of FAD–linked substrates.[3] A decrease in the acceptor control index and alterations in respiration rate were also observed.

Table 4: Effect of 96h LC_{50} concentration of hexavalent chromium (39.40 mg/l) on haemoglobin per cent, total erythrocyte count and mean cell haemoglobin of control and exposed *Labeo rohita* for 24h and 96h (n = 15).[1]

Parameter studied	Period (hour)	Control X±SD	Experiment X±SD	Percent Change	Result
Haemoglobin (g%)	00	8.1±0.67	6.80±0.76	–16.05	P<0.05*
Total erythrocyte count (10^6/mm)	24	1.3±0.03	1.05±0.05	–17.43	P<0.05*
Mean cell haemoglobin (Pg)		63.6±0.5	64.6±0.72	–1.67	P>0.05#
Haemoglobin (g%)		8.1±0.67	4.40±0.55	–45.68	P<0.001**
Total erythrocyte count (10^6/mm)	96	1.3±0.03	0.76±0.19	–40.66	P<0.001**
Mean cell haemoglobin (Pg)		63.6±0.5	58.2±0.45	–8.46	P<0.05*

**Highly significant; *Significant; #Not significant;

BIOMARKERS OF CHROMIUM TOXICITY IN FISH

Biomarkers are the measurable indicators of some biological state or condition. The decrease in glycogen concentration of the tissues of *Labeo rohita* can be due to its enhanced utilization as an immediate source to meet energy demands under metallic stress. It could be due to the prevalence of hypoxic or anoxic conditions, which normally enhances glycogen utilization. These glycogen and its consequent depletion tissues may be attributed to hypoxia since it increases carbohydrate consumption.[3] Under hypoxic conditions, the fish derives its energy from anaerobic breakdown of glucose, which is available to the cells by the increased glycogenolysis.

Stress proteins like metallothionine take longer time to express in case of Cr exposure at sublethal concentrations. In *L.rohita*, a 96 hours LC_{50} exposure to a concentration of hexavalent chromium (39.4mg/l) significantly declines the tissue glycogen total protein, and total lipid content in liver, muscle, and gill tissues of the fish.[2] A number of biomarkers of chromium pollution is also found through some genotoxicity studies. MN–assay and BN–assay both are major tools of genotoxicity study. The report has revealed that significant ($p<0.05$) increase of the MN frequencies at different exposure concentrations compared to control group.The MN frequency increased significantly ($p<0.05$) with increasing concentration of potassium dichromate within treatment groups of experimental organisms *L.rohita.*[2] In *L.rohita*, the micronucleus percentage has been found to range from 0.16 to 0.32 in control group; whereas a maximum of 2.48% has been found in case of sub–lethal ($1/10^{th}$ 96 hours LC_{50}) concentration after 60 days.[2] Hexavalent chromium, a well-known carcinogen, employs genotoxic effects in addition to endocrine disruption in fresh water fishes. In plasma cortisol, TSH, free triiodothyronine (T_3),and free thyroxin (T_4) level can be used as a biomarker for endocrine function. The impact of chromium exposure has been evident with decreased plasma (T_4) levels in eels only when exposed to the metal. The genotoxicity has been recorded by the frequency of erythrocytic nuclear abnormalities (ENA) of the endocrine cells.[1,2] Histological alterations like cellular disorganization, hyperplasia, necrosis of hepatic cells, highly fenestrated Bowman's capsule, constricted lumen of renal tube, glomerular disorganization in kidney, highly degraded inner epithelial layer of intestine, high lamellar degradation, necrosis in gill epithelial cells, thickening of blood vessels and atrophied central axis of gill can be used as remarkable biomarker of chromium exposure of fresh water fishes.[2]

CONCLUSION

According to analytical techniques (such as cathodic stripping voltammetry, chromatography, ion exchange, capillary zone electrophoresis, selective co–precipitation and mass spectrometry) and geochemical modelling

(Florence and Batley 1980) are helped to find out the acute exposure to hexavalent chromium. It is proven that highly toxic to *Labeo rohita* and induced cumulative deleterious effects at various vital functional sites like metabolic rate, haematological indices and biochemical profiles. Though significant changes are observed both at the end of 24h and 96h exposure periods, these changes are more pronounced at the end of 96h suggestive of time–dependent toxicity. Cr has been designated as a priority pollutant due to its carcinogenicity in animals and mutagenicity in a number of bacterial species. River Ganga harbours several such industries in and around its banks that discharges waste into the river directly. This result ed in development of resistance to chromium in microorganisms for their proper survival.[37] The metal induced decrease in the total protein content could possibly affect the enzyme mediated bio defence mechanisms of the fish, which pose a serious threat to human beings by secondary poisoning through food chain. Further studies under sub–acute exposure is required to elucidate the subtle changes that occur in the above parameters under the impact of chromium.

ACKNOWLEDGEMENT

The author Tanima Debnath Sarkar is thankful to Zoology Department of Annamalai University, Chidambaram, Chennai for providing labs and necessary equipment to perform this project supervised by Dr.P.Senthil Elango, and Dr.M. Muthulingam. The author is blessed to complete the postgraduate course successfully to get M.Sc. degree.

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