A Review of Heavy Metals Toxicological Studies on Mangur (Clarias batrachus) Fish

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ABSTRACT

Mangur (Clarias batrachus) is an economically important carp. Heavy metal pollutants are accumulating in our aquatic systems due to natural as well as anthropogenic activities. Owing to the process of biomagnification, fish as well as human health are at risk for being at higher levels of food chain. In this review, we have summarized the results of recent heavy metal toxicological studies on mangur fish.

Keywords--- mangur fish, heavy metal toxicity, aquatic system pollution, Clarias batrachus

I. INTRODUCTION

Heavy Metal toxicity is one of the serious hazards to cause deleterious effects on animals, plants, humans. Of the various environmental contaminants, metals and metalloids are amongst the most commonly accumulated toxins in fish which can lead to health defects when consumed in amounts exceeding safe consumption levels. Sewage and industrial disposal has greatly increased the addition of heavy metals in the aquatic ecosystems.

Fish is an important part of human diet as well as a good indicator of environmental contamination by a number of substances, including heavy metals. However, fish has been considered at the top of the aquatic food chain, therefore, they normally can accumulate heavy metals from food, water and sediments. The accumulated toxic metals in fish can counteract their beneficial effects; several adverse effects of heavy metals to human health have been known for long time. This may include serious threats like renal failure, liver damage, cardiovascular diseases and even death [1][2][3][4].

The Magur fish, (Clarias batrachus L.) a type of fresh water scaleless teleost of comparatively medium size, are abundantly available in India and are good sources of protein. The varying sizes ranging from 10 or 15 g to about 200 to 300 gm are usually available in the local market in all the seasons. They can survive in air for a considerable length of time because of the presence of accessory respiratory organs suitable for air breathing. Polluted aquatic surroundings are affecting mangur fish adversely.

II. HEAVY METALS TOXICITY

Heavy metals may act as a source of contamination when significant changes of pH, redox potential, salinity, particulate matter or microbial activity occur in the environment. These changes can increase the mobility and transport of the metals in the aquatic media and make them bio-available to the biota. It is well known that metals can be bio-accumulated in fish tissues. The magnitude of bioaccumulation is a function of age, species, and trophic transfer. Within the same species, the concentrations of metals may vary with the age and body weight [5][6][7].

Pb, Ba, Cd, Hg, Cr, and As are classified as toxic heavy metals, and maximum residual levels have been prescribed for humans and have no established role in biological system, whereas metals such as Cu, Na, K, Ca, Mn, Se, Fe, and Zn are essential metals for fish metabolism but may also bioaccumulate and reach toxic levels that can potentially destroy the ecological environment. The bioaccumulation of heavy metals in living organisms and biomagnifications describe the processes and pathways of pollutants from one trophic level to another.

At low levels, some heavy metals such as copper, cobalt, zinc, iron and manganese are essential for the activity of many enzymes involved in biological processes but they could become toxic at high concentrations. For the normal metabolic activities, the essential metals like copper and zinc must be taken by the fish from water, food or sediment.

Metal contaminants are naturally present in the environment but can be increased through industrial activity and pollution. The concentrations and uptake of these metals in aquatic organisms are subject to
environmental and species-specific biological factors as well as the chemical and physical state of the metals. There are three possible ways of metal pollution through which metals enter the body of fish, the gills, digestive tract and body surface. High concentrations of heavy metals are found in fish liver, kidney, gills.

Multiple factors including season, physical and chemical properties of water can play a significant role in metal accumulation in different fish tissues. The gills are directly in contact with water. Therefore, the concentration of metals in gills reflects their concentration in water where the fish lives, whereas the concentration in liver represents storage of metals in water. High concentration of Pb, Cr and Ni in sediment and fish tissues has been reported specially in areas close to industries [8][9][10].

**ARSENIC**

Arsenic (As) is widely distributed in nature due to environmental sources and anthropogenic pollution which is largely due to smelting activities, glass manufacturing, manufacture and use of arsenic pesticides, herbicides, fungicides and wood preservatives. Arsenic has a complex chemistry and can be present in several organic (trivalent and pentavalent arsenic) and inorganic (elemental, trivalent and pentavalent arsenic) forms which vary in their degree of toxicity.

The impact of sublethal toxicity of sodium arsenite on hematological and certain biochemical parameters of the fresh water catfish *Clarias batrachus* has been analyzed following exposure of sublethal concentration (1 mg/L; 5% of LC50 value) of sodium arsenite for 10, 30, 45, and 60 days. Arsenic bioaccumulation in the blood tissue of the fish increased progressively with increased period of exposure. The values of total erythrocyte count (TECs), total leucocytes count (TLCs), hemoglobin concentration, and packed cell volume (PCV) 1.40 ± 0.03 ×10¹⁰/mm³, 174.83 ± 2.74 × 10⁹/mm³, 5.01 ± 0.26 g/100 ml, 25.00 ± 1.06 were observed respectively at the end of 60 days of exposure. The results of hematological indices were found to be 179.23 ± 8.81fl/cell for mean corpuscular volume (MCV), 35.92 ±1.89 pg/cell for mean corpuscular hemoglobin(MCH) and 20.17 ± 1.12 g/dl for mean corpuscular hemoglobin concentration (MCHC). The findings clearly indicated severe fish anemia due to the arsenic salt exposure. As per authors, the continued arsenic toxicity results in decreased serum protein concentration that might be a cause for the loss of weight as well as weakness in the fish [14].

**CADMIUM**

Cadmium (Cd) is a metal contaminant which is introduced into the environment through both natural processes (volcanic emissions and weathering of rocks) and anthropogenic activities such as the smelting of other metals, burning of fossil fuels, incineration of waste materials and the use of certain fertilisers. Cadmium is a nonessential heavy metal and considered as one of the most toxic water contaminants that could cause toxicity at each trophic level of the ecosystem. Fish exposed to high concentration of cadmium quickly develop lack of calcium and low blood hemoglobin.

Cadmium can readily cross various biological membranes, and once inside living cells, has a high affinity to bind to ligands and form Cd complexes which can be more stable. For example, in fish muscle most of the Cd present tends to bind to proteins. Cadmium absorbed into the fish body is therefore eliminated at a very slow rate, causing bioaccumulation in the body. Cadmium can enter fish by passive diffusion across the gills or by entering the marine food chain at the plankton and micro-organisms level and thereby entering fish through the diet.

Even at low concentrations, Cd and Cr are known to be highly toxic because Cd can replace Zn in some enzymes and may cause diseases. Cd has been shown to accumulate in bluegill Sun fish and it can restrict the oxygen metabolism in gills, liver and kidney in higher amount interfering in different biological functioning. Cd is released into the environment by industries like paints, alloys and found to be associated with bottom sediment and particles. Cd is very low in water as compared to sediment.

Presence of cadmium in water and its significant levels in fish tissues (muscle & gills) has been shown to cause disturbances in tuna. This trace metal affects the physiology of fish due to its chronic exposure to waterborne sub-lethal levels cadmium. Acute toxicity studies of cadmium on the edible carp, *Catla catla* revealed significant changes in the biochemical constituents of the fish like glucose, glycogen, total proteins, lipids and free amino acids.

In a study, the pattern of accumulation of cadmium and its affinity to selected tissues - gills, kidneys, liver, skin and muscle - of *Clarias batrachus* exposed to sublethal concentrations (7 ppm) of cadmium chloride was investigated. These tissues showed significant variations in the patterns of accumulation of cadmium. The rate of accumulation varies from tissue to tissue as well as at various exposure periods. The mean rate of accumulation after 60 days of exposure was in the order gills > kidneys > liver > skin > muscle. The difference in the rate of accumulation may be attributed to the proximity of the tissues to the toxicant medium, physiological state of the tissues and presence of ligands in the tissues having an affinity to cadmium.

In another study, the common histopathological changes that took place under heavy metal exposure were disintegration of mucosal epithelium, hyper trophy epithelial cells, increased number of goblet cells, sloughed off epithelial cells and mucous in the lumen, degeneration of the connective tissue of sub-mucosa and hyperemic blood vessels. Separation of mucosal epithelium,
especially in the villi and increased goblet cells were prominent in Cd-exposed fish [15][20][23].

**CHROMIUM**

Chromium does not normally accumulate in fish and hence, low concentration was reported even from the industrialized part of the world. A study has shown that a higher rate of uptake in young fish but the body burden of Cr declines with age due to the rapid elimination.

Cr exists in water in two forms; trivalent and hexavalent. It is the hexavalent state which is highly toxic. Due to its poor solubility it is not detected in water. Cr combines with water of greater hardness and can get accumulated in fish from water through the gills and transported via blood to various organs and tissues. The metal then reaches the gut where it is eliminated through feces. This could be the reason for low accumulation of Cr in the muscles of the fish species in general.

A study has been conducted to determine the median lethal concentration of chromium to the freshwater fish *Clarias batrachus* following standard procedures and was found that 40.56ppm, 38.15ppm, 36.65 ppm and 35.50ppm for 24h, 48h, 72h and 96hours LC50 doses, whereas LC0 as well as LC100 doses were 31 ppm and 40ppm respectively. The behavioural changes following metal stress included abnormal coughing and jerky movements, erratic swimming, hyperactivity changes in opercular movement, copious amount of mucus secretion all over the body and loss of equilibrium.

Further, sublethal toxic effect of chromium 14.2 ppm (40% of 96h LC50) on the testis of the fresh water fish *Clarias batrachus* were observed for a period of 45days. The histopathological changes observed as distortion of seminiferous tubules, disorganization of spermatogonia, spermatocytes and spermatids with cytoplasmic vacuolization and nuclear pycnosis. These alterations in the histology of testis in the fish *Clarias batrachus* exposed to sublethal chromium solution may be due to toxicity induced by the irritant present in the aquatic medium [16][27][38].

**COPPER**

Copper is essential trace metal in small concentration for several fish metabolic functions because of its specific incorporation into a variety of enzymes that play important roles in physiological processes, as well as, into some structural proteins. When exposed to toxic concentrations, organs of aquatic animals may accumulate copper, that can lead to redox reactions generating free radicals and, therefore, may cause biochemical and morphological alterations. Cu can induce respiratory distress in fish, and it is striking that the most hypoxia sensitive species is also the most Cu sensitive. Cu at sublethal concentration in fish decreases survival growth and reproductivity and mainly accumulates in gills and liver. Its bioaccumulation can decrease oxygen consumption.

The teleost liver maintains homeostasis of copper. In several fish species obtained from different parts of the world, the concentration of copper ranges from 0.5 to 28 mg/l. Copper is absorbed rapidly resulting in increased residue levels. At sublethal concentration copper causes retardation in growth, inhibition of respiratory enzymes in crayfish at 1.0 mg/l, changes in locomotors behavior of goldfish exposed to as low as 0.011 to 0.017 mg/l.

A study was conducted to determine the effects of sublethal concentrations of ZnSO4, CdCl2 and CuSO4 as a pollutant on some haematological parameters of *Clarias batrachus* were exposed to three sublethal levels. Sublethal concentrations used were 6.5 ppm for cadmium, 4.6 ppm for Zn and 0.7 ppm for Cu. The results obtained revealed that there was significant difference in hematocrit, Exposures of *Clarias batrachus* to sublethal concentrations of Zn and Cd resulted in significant decrease (p < 0.05) in erythrocyte count and haemoglobin content, while there was a slight increase in case of copper. The microscopical observations showed that exposure of *Clarias batrachus* to sublethal concentrations of heavy metals 6.5 ppm for cadmium, 4.6 ppm for Zn and 0.7 ppm for Cu resulted in some changes in the morphology of the R.B.Cs which tended to lose its ellipsoidal shape acquiring different shapes (poikilocytosis or anisocytosis).

The nucleic acid (DNA and RNA) content in different tissues such as brain, liver, kidney and ovary of copper sulphate exposed freshwater fish, *Clarias batrachus* had been studied. The changes in the nucleic acid content of the tissues have been observed. The DNA content in the ovary is higher in comparison to other tissues. The variation of DNA content in control and copper sulphate exposed fish is ovary > liver > brain > kidney. The RNA content also exhibited similar to that of DNA, having higher amount in the ovary and the variation is ovary > liver > brain > kidney. Although the degree of variation between the tissues remains same in both control and experimental groups, the nucleic acid content reduced under copper sulphate exposed freshwater fish, *Clarias batrachus* indicating copper sulphate as a pollutant effect the nucleic acid content in the tissue [17][34].

**LEAD**

Lead is one of the primary contaminants present in the environment and naturally occurs in rocks, soils and in the hydrosphere. However, Pb is also the most widely used metal and industrial Pb contributes a considerable quantity to that found in the natural environment. Large amounts of lead tetraethyl can be completely converted to aerosols through the combustion of gasoline, subsequently contributing to atmospheric Pb. The atmosphere, in turn, is
the main source of Pb deposition in the aquatic environment, therefore acting as a Pb pathway from the terrestrial to the aquatic environment. The toxicity of Pb is dependent on its chemical form where the organolead compounds are more toxic than the inorganic Pb form.

Fish have been reported to be very much sensitive to Pb; its uptake increases with increasing concentration in the environment. Fish living in contaminated sediment showed high Pb concentration. The higher Pb concentration has been reported in kidney, gills and liver of fish. Exposure to heavy metals resulted in slowed down development and growth rate, and reduced survival. Exposure to lead generally caused scoliosis in fish. Lead exposure caused a general increase in locomotor activity in mirror carp (Cyprinus carpio).

In zebrafish (Danio rerio) and fathead minnows (Pimephales promelas), lead exposure reduced feeding ability, as evidenced by feeding mistakes and increased prey-handling times. This reduction in feeding ability was attributed to psychomotor coordination, based on correlations between increased brain lead levels, increased serotonin and norepinephrine concentrations, and decreased feeding ability. Lead exposure induced similar increases in brain serotonin and decreases in GABA in walking catfish (Clarias batrachus). Although lead increased brain serotonin and norepinephrine levels, lead exposure did not increase dopamine levels in fathead minnows.

In a study, it had been shown that the RBC counts, haemoglobin percentage and serum protein levels were decreased significantly in comparison to control groups following lead exposure in Clarias batrachus [24][25][32].

**MERCURY**

Mercury (Hg) is a metal that is liquid at ambient temperature and pressure and can be present in several different chemical forms and compounds in the environment. Mercury is used for the production of paint, electrical equipment, batteries and fungicides as well as in medicine, dentistry, wood pulping and the military sector. In addition, mining contributes significantly to Hg water pollution whilst the burning of fossil fuels and the smelting of Pb, Cu and Zn ores are major sources of atmospheric Hg pollution.

A study was conducted to determine the combined effects of cadmium (Cd) and mercury (Hg) at sub-lethal concentrations for 32 days on histochemical localization of heavy metals and on serum biochemical parameters including serum glutamic-pyruvic transaminase (SGPT) enzyme activity; glucose, triglyceride, cholesterol and total protein concentrations in Clarias batrachus. Serum SGPT, glucose, triglyceride, cholesterol and total protein levels were significantly altered in fish exposed to Cd or Hg salt alone. However, combined exposure of Cd and Hg normalized all the above mentioned biochemical parameters. Histochemical analysis demonstrated enormous amount of metals in the liver and kidney tissues exposed to Hg and Cd alone. Mercury accumulation in C. batrachus was comparatively more than that of cadmium in both the tissues. While exposure Hg or Cd adversely altered the biochemical parameters in the test catfish, following the combined exposure of both the metals, the concentrations of metal accumulation were decreased in both the tissues of C. batrachus.

In another study, clumping of the epithelial cells at the base near the basement membrane was found out to be prominent in the case of the Cr- and Hg-exposure. Connective tissue degeneration in the lamina propria and submucosa of the intestine and dilated blood vessels with congestion of blood were also prominent in the Cr and Hg exposed fish.

In another study, adult cat fish treated separately with Cadmium Chloride (CdCl₂) and Mercury Chloride (HgCl₂) for 24, 48, 72, and 96hr at different dose levels showed significant behavioral changes in the Lethal concentration were determined by probit analysis method. The LC 50 values for CdCl₂, HgCl₂ were found as 8.21ppm, 1.85ppm; respectively. Among the toxicants selected, HgCl₂ is more toxic than CdCl₂ in cat fish [11][12][13][18][19].

**ZINC**

Zinc at certain concentration is desirable for the growth of freshwater animals but at high accumulation it becomes hazardous to the exposed organisms as the one’s consuming them directly or indirectly through food chain. Zinc is potential toxicant to fish which causes disturbances of acid-base and ion regulation, disruption of gill tissue and hypoxia. Zinc caused oxidative stress in estuarine teleostean, Fundulus heteroclitus.

A study was carried out on juvenile Clarias batrachus (L.) to investigate the effects of sub-lethal concentrations of copper and zinc (0.3, 0.4, 0.5 ppm) on the survival rate, oxygen consumption and histopathological changes in the gills of exposed fish. The results showed a decrease in survival rate with increasing concentration of each metal. Copper has the most toxic effect compared with zinc the survival rate has decreased from 50% to 10% for copper and from 70% to 20% for zinc with increasing concentration for 15 days. Oxygen consumption rate decreased with increasing concentration and there was a negative correlation between oxygen consumption and metal concentration. The exposure to each metal caused histopathological changes in the gill and resulted in separation of epithelial secondary gill lamellae, hyperplasia, fusion of secondary lamellae and necrosis [21][22][26].
III. CONCLUSIONS

The heavy metals are present at <0.1% level in earth’s crust. But anthropogenic, geochemical factors are releasing a large number of toxic heavy metals into the aquatic ecosystem.

In recent years the accumulation of heavy metals in aquatic systems has become a problem of great concern throughout the world. These metals may accumulate to a very high toxic levels and cause severe impact on the aquatic organisms without any visible sign. Increase in population, urbanization, industrialization and agricultural practices have further aggravated the situation [26][28][30][31].

Heavy metals thus discharged persist in the aquatic bodies and bioaccumulate along the food chain. Metal toxicity in fish is often characterised by gill damage and hypersecretion of mucus, ensuing mortalities are related to secondary physiological respiratory disturbances, i.e. ion regulatory and acid-base balance disturbances of which extent depends upon uptake and bio-accumulation of metals. Heavy metals at high concentrations can cause harmful effects on the biochemical system of fishes and this causes long term ecotoxicological effects on the organism that eat them [33][35][36][37].

Heavy metal concentrations are species, location and trophic level dependent which can result in considerable variation making comparison and meaningful interpretation difficult. Therefore, more research is required to cover each of these aspects. Research on: (1) trophic level disparities can aid the understanding as to how metals accumulate within the food chain, while (2) spatial scale studies (between and within species) may provide links between environmental pollution and the effects on fish contamination and consequently food safety and consumer health.

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