



Effect of Mercury Chloride on Histological Impairments in the Gill of Freshwater Fish *Hypphthalmichthys Molitrix* (Valenciennes)

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ABSTRACT:

Introduction: The widespread use of heavy metals in recent years, due to accelerated industrialization and modernization, has led to the overexploitation of natural resources and severe environmental pollution. Various pollutants such as organic and inorganic compounds, nanoparticles, organometallic compounds, and radioactive isotopes have contaminated ecosystems at an alarming rate. Among these, heavy metals have emerged as major environmental contaminants because of their high density, atomic weight, and toxic nature. Heavy metal pollution poses a significant threat to aquatic life and human health worldwide.

Objectives: The primary objective of this study was to examine the effects of sublethal concentrations of mercury chloride on the histology of gills in the freshwater fish *Hypphthalmichthys molitrix*.

Methods: Specimens of *Hypphthalmichthys molitrix* were exposed to sublethal concentrations of mercury chloride for durations of 10, 20, and 30 days. After exposure, gill tissues were collected and processed for histopathological examination to identify structural and cellular alterations.

Results: The exposed fish exhibited marked histopathological alterations in the gills, including fusion of secondary gill lamellae, necrosis, rupture of gill epithelium, secretion of mucus, and shortening of secondary lamellae. The severity of these pathological changes increased progressively with the duration of exposure (10, 20, and 30 days).

Conclusion: The findings suggest that exposure to sublethal concentrations of mercury chloride induces significant histopathological damage in the gills of *Hypphthalmichthys molitrix*. These alterations indicate impaired respiratory function and overall physiological stress, emphasizing the toxic impact of mercury contamination on freshwater fish

1. Introduction

Water pollution is mostly caused by increased urbanization, industry, and rapid population growth beside water bodies like lakes, reservoirs, and rivers, this, in turn, leads to the degradation of the ecosystem [1]. Contamination with heavy metals is a significant issue in expanding cities of emerging countries, mostly as a result of unchecked pollution levels brought by root causes which including industrial expansion and significant increases in traffic powered by petroleum fuels [2]. Both naturally occurring and human-caused environmental releases of heavy metals occur. Heavy

metals originate from multiple sources, including soil erosion, mining activities, industrial discharges, urban runoff, wastewater discharge, crop pesticides, and the natural weathering of the Earth's crust [3].

Heavy metals naturally exist in various concentrations in earth's crust, soil, air, water and all biological matter and they have been spread widely as a result of anthropogenic activities such as cement production, iron steel industry, steam power plants, glass production, garbage and waste mud incineration facilities, mining activities, smelters and foundries, piping, combustion and traffic. They can also diffuse around by natural



events such as wind, soil erosion and volcanic activity [4]. Pollution and corresponding risks that come into existence by this rapid increase in agricultural activities, population growth, urbanization and industrialization are critical issues about environment. There is no doubt that the most dangerous chemical pollution in water is heavy metal contamination. Heavy metals constitute a significant ecological and health concern due to their toxicity and ability to accumulate in living beings. Heavy metals have a strong influence on the stability of ecosystems but also have adverse effects on humans. Even though some of the heavy metals such as zinc, iron, cobalt and copper are essential for enzymatic activity and other biological processes at low levels they become toxic when they exceed certain limit. On the other hand other metals such as lead, cadmium and mercury have no essential role in living organisms and are toxic even at too low concentrations [5]. Heavy metals include metals and metalloids with high atomic weight and density that exist naturally in the environment at low levels [6]. They are persistent, stable, and nonbiodegradable [7]. They are transported through the earth's spheres: atmosphere (air), geosphere (soil), hydrosphere (water), and biosphere (biotic) and recycled by biogeochemical cycles [8,9]. Heavy metals are classified into essential and non-essential heavy metals. Essential heavy metals are needed in small quantities by the human body to ensure their normal functioning. High quantities can become toxic and alter the normal biochemical processes of the human body's functions. Non-essential heavy metals like mercury (Hg) is not needed by the human body and can adversely affect human health [10]. In the aquatic environment, mercury is transformed into a more toxic form, methyl mercury (MeHg) (CH_3HgX , where X is Cl^- , OH^- , or organic thiols and other reduced sulfur ligands (R-S^-)), by microorganisms present in the sediment. Aquatic organisms readily absorb MeHg, which is then bioaccumulated and biomagnified within the aquatic food chain [11].

Various factors may affect the mercury and methyl mercury concentrations in fish. Some of those factors are related to the fish like trophic level, species, size, age, gender, habitat, and niche. Abiotic factors can also influence mercury levels like water column, temperature, salinity, and deposition from anthropogenic emissions from industrial and petroleum

activities. The contamination of fish with Hg and MeHg poses a significant challenge to public health. Fish is considered one of the most important sources of healthy proteins and is a staple food item for many communities worldwide. In addition, fish is rich in omega 3, docosahexaenoic acid, linolenic acids, unsaturated fatty acids, micro- and macronutrients, and different vitamins. Besides, fish consumption was proven to be highly effective in reducing the risk for non-communicable diseases (NCDs), particularly cardiovascular diseases and rheumatoid arthritis, and is essential for normal neuronal development in children [12].

A major concern associated with fish consumption is human exposure to the toxic Hg and MeHg [13]. A wide range of factors have affected the Hg levels in the aquatic environment around the Arabian Gulf region. The Arabian Gulf has a physiologically arid environment, hot climate, high water salinity, and poor water circulation resulting in long water residence time. It is a semi-closed Gulf that receives a limited amount of freshwater input from rivers in the northern part and low precipitation levels. The region also relies on water desalination plants, wastewater treatment plants, and power plants established in the coastal areas. This increase in mercury levels can pose a risk to the Gulf population since fish is a major food source of the region due to its abundance and easy availability that remains unaffected by the arid climate, unlike other meat sources. To plan appropriate responses at both the upstream and downstream levels, it is necessary to investigate all possible causes and effects relationships of mercury. Thus, understanding the source-receptor relationships associated with sources of mercury emissions, transformations, and cycling in each of the environmental compartments is needed [14]. Numerous studies on Hg in the environment have focused on the Hg biogeochemical cycling in aquatic systems, the presence and bioaccumulation of Hg in seafood, and the risk of Hg exposure from fish consumption [12].

2. Minamata disease, a devastating neurological disorder caused by mercury poisoning, remains a haunting reminder of the consequences of environmental negligence. Tragically, this resulted in widespread neurological and physical impairments, affecting 1000s of individuals, including unborn babies. The symptoms of



Minamata disease can vary in severity but often include sensory disturbances (such as numbness and tingling), muscle weakness, vision and hearing impairments, difficulty walking, and cognitive dysfunction. In severe cases, the disease can lead to paralysis, coma, and even death. Minamata disease is primarily caused by the ingestion of methyl mercury, a highly toxic form of mercury [15]. The main sources of methyl mercury exposure in the context of Minamata disease are industrial waste and contaminated seafood. Mercury, a heavy metal, is released into the environment through industrial processes, such as chemical production, mining, and waste incineration. These pollutants find their way into water bodies, where they are converted by bacteria into methyl mercury, a bioavailable form that can easily accumulate in aquatic organisms. In the case of Minamata, a local chemical factory released methyl mercury-contaminated wastewater into the Minamata Bay over several decades. The heavy metal mercury converted in to methyl mercury by the action of microbes which is present in the aquatic medium. The methyl mercury accumulated in the phytoplankton, zooplankton, small fish followed by larger fish through food chain. This bioaccumulation continued until the mercury concentration reached dangerous levels, and the local population, largely dependent on seafood, was exposed to the toxin. One of the most heart-wrenching aspects of the Minamata tragedy is the fact that it affected not only those who directly consumed contaminated seafood but also unborn children whose mothers were exposed during pregnancy. The disease caused severe developmental issues, known as congenital Minamata disease, leaving affected children with life-long disabilities. The treatment of Minamata disease focuses on alleviating symptoms and preventing further exposure to methyl mercury. However, it's important to note that there is no cure for the disease, and the damage caused by methyl mercury poisoning is often irreversible. Management of the disease primarily revolves around supportive care and therapies aimed at improving the quality of life for affected individuals. Histological analysis appears to be a

very sensitive parameter and is crucial in determining cellular changes generated by pollutants exposure that may occur in target organs. *Hypophthalmichthys molitrix* is one of the exotic fish. It is an edible freshwater fish of great importance. Hence, it is prudent to study the effect of sublethal concentration of heavy metal, mercury chloride on histopathological alterations in gill.

2. Material and Methods

Procurement and rearing of experimental fishes

Hypophthalmichthys molitrix exotic carp commonly known as silver carp is widely distributed in the freshwater. *Hypophthalmichthys molitrix* was collected from the fish farm located in Puthur, 10 km away from the University Campus. The collected fishes without least disturbance were transported in polythene bags filled half with water. About 100 fishes were put in each bag and water was well aerated, using pressurized air from a cylinder. This mode of transit proved successful, since there was no mortality in all consignments throughout the study.

The fishes brought to the laboratory were acclimatized in cement tank for a fortnight before they were used for the experiment. The fish tanks were kept free from microbial infection by washing with potassium permanganate solution. the fish were disinfected with 0.1% potassium permanganate solution and were maintained for three weeks in well aerated tap water. Test fishes were critically screened for the signs of disease, stress, physical damage and mortality. The injured, severely diseased, abnormal and dead fishes were discarded. Feeding was discontinued two days prior to the commencement of the experiments to reduce the additive effects of animal excreta in the test trough. The fishes were exposed to sublethal concentration of mercury chloride and control for the period of 10, 20 and 30 days. A control group was maintained with identical environment. The toxicant water and normal water was renewed every day. The fish were sacrificed from both experimental and control groups on 10, 20 and 30 days of exposure periods.

Experimental Design

Hypophthalmichthys molitrix weighing 14-16 g were divided into 4 groups and stocked at random into 4 different group were assigned.



The following experimental groups were conducted in the freshwater fish *Hypophthalmichthys molitrix* for the period of 30 days.

Group I: The first group was maintained in free from mercury chloride and served as the control

Group II : Fish exposed to 1/15th of the LC₅₀ of low sublethal concentration of mercury chloride for the period of 10, 20 and 30 days.

Group III : Fish exposed to 1/10th of the LC₅₀ of medium sublethal concentration of mercury chloride for the period of 10, 20 and 30 days.

Group IV : Fish exposed to 1/5th of the LC₅₀ of high sublethal concentration of mercury chloride for the period of 10, 20 and 30 days.

Fishes were exposed to sublethal concentrations of mercury chloride separately in plastic troughs and control fishes were also maintained separately. They were fed on ad libitum diet of rice bran and oil cake. The medium was renewed daily with sublethal concentration of the mercury chloride. After the exposure period, *Hypophthalmichthys molitrix* were sacrificed and the gills were removed for histopathological examination.

Histological studies

To examine the extent of cellular damage caused by mercury chloride the gills of the control and treated fish tissues were fixed in Bouin's fluid. After 24 hours, the standard histological technique was followed by the method of [16]. The tissues were dehydrated in ascending grades of alcohol. After dehydration in graded series of alcohol, they were cleared in acetone and xylol. Then, they are treated with cold and hot infiltration and embedded in paraffin wax (58°C - 60°C). Serial sections were cut at 6 to 8 µm thickness, deparaffinized in xylol, passed through descending grades of alcoholic series, hydrated in water, stained in Heidenhain's iron haematoxylin and counterstained with aqueous eosin. Stained sections were mounted in DPX for microscopic observations.

3. Results

Gill histology

The control gills of *Hypophthalmichthys molitrix* comprised of laterally compressed leaf like gill

filaments (primary gill lamellae) arranged alternately on either side of the interbranchial septum. Each primary filament bore a row of secondary gill lamellae on both sides perpendicular to its long axis. Primary gill lamellae comprised of a central core of cartilaginous rod, lining epithelial cells and blood vessels whereas

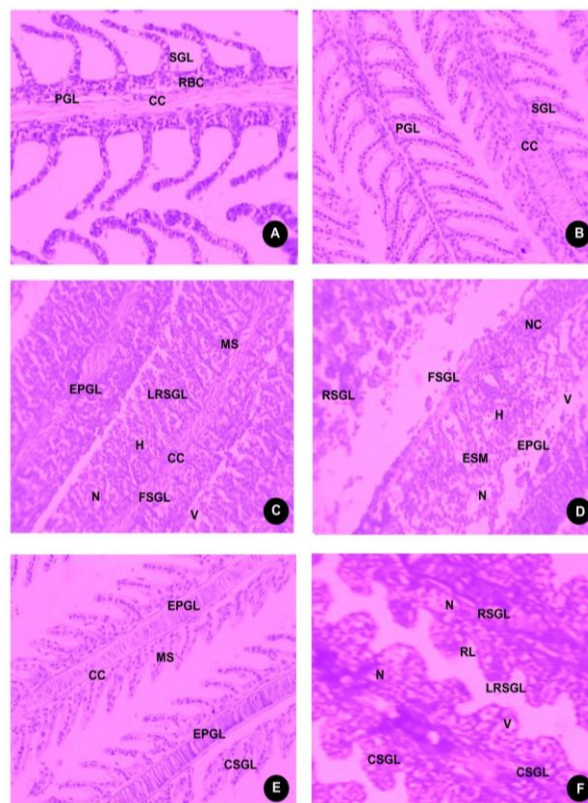
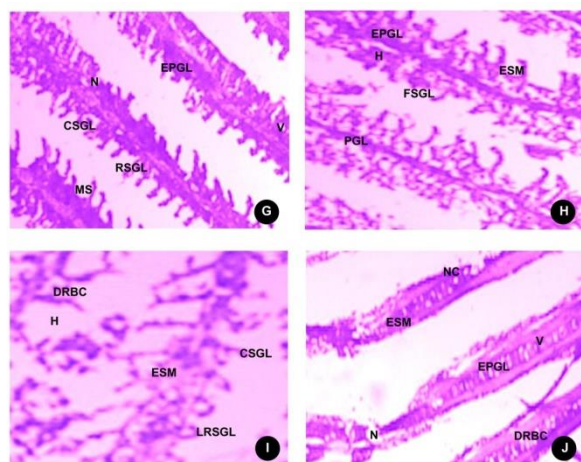


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secondary lamellae consisted of a layer of flattened epithelial cells attached to the basement membrane, contractile pillar cell system and blood spaces (Fig 1).

Gill histopathology

The fish, *Hypophthalmichthys molitrix* exposed to low, medium and high sublethal concentrations of heavy metal mercury chloride for 10, 20 and 30 days showed marked histopathological changes in their gill. Excessive secretion of mucous in the intercellular spaces, fusion of secondary gill lamellae, reduction in length, swelling of epithelial cells of secondary lamellae, swelling of primary lamellae, damaged RBC cells, cytoplasmic vacuolization and necrosis, hypertrophy were some of the observable changes. The severe pathological changes of gill lamellae were noticed on 30th day of exposure (Plate 1 B-J).



- A – Gill section taken from control fish showing normal histoarchitectural pattern (XCa 200)
 B – Gill section taken from low sublethal concentration of mercury chloride treated fish for the period of 10 days (XCa 200)
 C – Gill section taken from medium sublethal concentration of mercury chloride treated fish for the period of 10 days (XCa 200)
 D – Gill section taken from high sublethal concentration of mercury chloride treated fish for the period of 10 days (XCa 200)
 E – Gill section taken from low sublethal concentration of mercury chloride treated fish for the period of 20 days (XCa 200)
 F – Gill section taken from medium sublethal concentration of mercury chloride treated fish for the period of 20 days (XCa 200)
 G – Gill section taken from high sublethal concentration of mercury chloride treated fish for the period of 20 days (XCa 200)
 H – Gill section taken from low sublethal concentration of mercury chloride treated fish for the period of 30 days (XCa 200)
 I – Gill section taken from medium sublethal concentration of mercury chloride treated fish for the period of 30 days (XCa 200)
 J – Gill section taken from high sublethal concentration of mercury chloride treated fish for the period of 30 days (XCa 200)
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|---|---|
| CC – Central core | RL – Reduction in length |
| FSGL – Fusion of secondary gill lamellae | LRSGL – Length reduction of secondary gill lamellae |
| MS – Mucous secretion | SGL – Secondary gill lamellae |
| EPGL – Enlargement of primary gill lamellae | H – Hypertrophy |
| V – Vacuolization | NC – Necrosis |
| CSGL – Curling of secondary gill lamellae | RSGL – Rupturing of secondary gill lamellae |
| DRBC – Damaged red blood corpuscle | ESM – Excessive secretion of mucus |
| RBC – Red blood corpuscle | |

Fig 1. Histology and histopathology of gill

4. Discussion

Gill

Histological alterations act as a biomarker of contaminants and provide direct and indirect effects on animal tissues. It also indicates the whole population's health in the ecosystem [17]. Histopathological studies are commonly used to evaluate the toxic effects of pollutants and provide a useful means to evaluate water quality [18,19]. Cadmium is biologically nonessential, but is an important metal for industrial applications (Wu *et al.*, 2009) and can damage gills. Chloride cells are the primary targets of water borne cadmium, because this metal decreases the activity of gill Ca^{++} ATPase, which leads to hypocalcemia in the fishes and causes problems in calcium homeostasis [20]. In addition cadmium has negative effects on respiratory functions and osmoregulation [19,21].

Gills perform several vital functions, such as excretion of metabolic waste, ion regulation and gas exchange [17]. Fish gills are also vulnerable to pollutants in

water because of their large surface area and external location. Gills apart from being the primary respiratory organ in fishes, are also responsible for ion regulation. For this reason, fish gills are considered to be the most appropriate indicators of water pollution levels. In fish the gill is the most important organ for respiration and osmoregulation and it is the first organ to which, pollutant comes into contact. Hence, it is more vulnerable to damage than any other tissue [21]. Gills represent a thin and extensive surface in intimate contact with water. They carry out three main functions, gas exchange, ion regulation and excretion of metabolic waste products. Due to the constant contact with the external environment, gills are the first target of waterborne pollutants [22]. Heavy metals enter the fish mainly through the gills, thus being the first target of the metal. The effect of cadmium on fish gill (Na^+ , K^+)-ATPase, as well as its sensitivity to the metal, differs from species to species and probably from organ to organ [23]. Fish gills are very sensitive to changes in the composition of the environment and are an important indicator of waterborne toxicants. Consequently, injury to gill epithelium is a common response observed in fish exposed to a variety of contaminants. The severity of damage to the gills depends on the concentration of the toxicant and the period of exposure [24-28].

Gill covers more than sixty percent surface of the fish and its external location renders the most vulnerable target organ for the pollutants [29]. Mercury, widely used by humans, contaminates aquatic environments, entering fish through the food web via bio-accumulation and bio-magnification. Anthropogenic activities, especially coal fired thermal power plants are major sources, releasing mercury into the air, which eventually contaminates water bodies and sediments. This leads to methyl mercury accumulation in fish, posing risks to their health and ultimately to humans who consume contaminated fish, leading to neurological damage and other health issues. The higher organisms in the food chain the greater the degree of accumulation. Fishes are therefore particularly sensitive to environmental contamination of the water and pollutants may cause significant impairments of certain physiological and biochemical processes which can result in serious tissue damage [30,31].



The present investigation fish, *Hypophthalmichthys molitrix* when exposed to high sublethal concentration of mercury chloride for 10, 20 and 30 days showed marked histopathological changes such as excessive secretion of mucous in the intercellular spaces, fusion of secondary gill lamellae, reduction in length, swelling of epithelial cells of secondary lamellae, cytoplasmic vacuolization and necrosis were some of the observable changes in the gill. The severe pathological changes of gill lamellae were noticed on 30 days exposure. Similar histopathological changes have been observed in lead exposed estuarine mullet, *Liza parsia* [32] and in DDT exposed estuarine mullet, *Liza parsia* [33]. Gills showed necrosis and atrophy of the gill lamellae. Severe edema, hyperplasia, fusion and focal desquamation of the epithelial lining of the secondary lamellae also were detected. The gill arch showed numerous mononuclear leucocytic infiltrations, edema, congestion and the apex of gill filaments showed congestion, hyper activation of the mucous and chloride cells [34]. Observed that Ariel detergent exposure induced separation of epithelial layer around respiratory lamellae and atrophy of respiratory lamellae [35]. The plant toxin exposed Indian hill stream fish showed collapsed tips of the swollen gill filaments, mucous exudation and reduction in the lamellar spaces [36]. These responses can impair gill functions and could eventually lead to the death of the fish [37].

In the present study on *Hypophthalmichthys molitrix* the nature of histopathological changes observed in the gill exposed to sublethal concentration of mercury chloride, lend additional support to those recorded for *Channa punctatus* exposed to sugar mill effluent [38], *Heteropneustes fossilis* exposed to hydrocarbon and blue gill exposed to alkyl benzene sulfonate. The disorganization of secondary gill filaments, development of vacuoles, necrosis of nucleus and increased number of mucous cells during monocrotophos, fenvalerate and monocrotophos-fenvalerate mixture treatment in *Labeo rohita* [21]. Histological examination of the *Catla catla*, gills of treated fish showed epithelial hyperplasia, epithelial necrosis, lamellar atrophy, and curling of secondary lamellae after heavy metal exposure [39]. In the present investigation the histopathological changes in gill is evident in the sublethal concentration of mercury chloride exposed *Hypophthalmichthys molitrix* [40].

Severe gill lesions at longer period exposure such as excessive secretion of mucous in the intercellular spaces, swelling of epithelial cells, rupturing of secondary gill lamellae, vacuolization and necrosis were some of the observable changes in the gill were very much evident [41].

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