

Nickel Induced Histopathological Alterations in the Kidney of Indian Common Carp *Labeo Rohita* (Ham.)

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Abstract- Nickel used in industries when released into the water bodies affects the biota. Histopathological alterations were seen in kidney due to chronic exposure of the fish, *Labeo rohita* to the nickel chloride 4mg/l concentration which is $1/10^{\text{th}}$ of its 96 h LC₅₀ value, for 30 days. The primary site of tissue damage was noticed in the form of edema and loss of interstitial tissue in case of the experimental fish. After ten days of exposure to nickel chloride, the cells lining the uriniferous tubules appeared swollen and vacuolated. Their nuclei were also hypertrophied. Hydropic degeneration was seen at some places. Fishes exposed to sublethal concentration of nickel chloride for thirty days exhibited haemorrhage in the glomeruli. Nephrosis and advanced vacuolation was observed. Tubular and interstitial tissue was seen disintegrating. Disruption of the tubular cells resulted as the kidney had been induced to function actively under pollution stress to eliminate the toxicants. The accumulation of heavy metal salt and its degraded products might have a direct effect on enzymes of nuclear matrix, damaging the renal nuclei.

Keywords: Nickel, *Labeo rohita*, Kidney, Nephrosis

Introduction:

The deleterious effects of effluents on the water quality in rivers and lakes as well as on the biotopes, particularly on the fish population, have been known for many years. Due to the rapid industrial development during last three decades in India, the disposal of industrial effluents has become a serious problem. A huge amount of wastewater generated from factories is discharged on land or into the running water. Wastewater is characterized by low pH, high BOD and COD values and contains a high percentage of organic and inorganic materials (Chauhan, 1991). Increase in organic load, depletion in oxygen content and destruction of aquatic life including in water are some of the major problems created due to disposal of effluents in fresh water. Subsequently, these toxic elements may cause physical problems to human beings, animals by entering the food chain.

A special characteristic of heavy metal chemicals is their strong attraction for biological tissues and, in general slow elimination of these chemicals from biological systems. Once in a system, they remain for relatively long periods. Heavy metals are environmentally stable, and as long as the chemicals are present they may induce their toxic effect. It is necessary for toxicologists to understand the mechanisms of the toxic effect to recognize the clinical sign (Vinodhini and Narayanan 2009).

In context to the heavy metal toxicity, the use of fish as an indicator organism for the heavy metal pollution of its

environment and its possible unfitness for human consumption from a toxicological point of view and also, the use of fish to study the physiological behavior of heavy metal are considered in investigations.

Nickel is widely used in electronics, coins steel alloys, batteries, food processing, paper and pulp industries, fertilizers, petroleum refining vehicles, aircraft plating, finishing, steam generation power plants and stainless steel. Nickel is also used as catalyst in the hydrogenation of fats and oils. Nickel finds numerous applications in many industries because of its corrosion resistance high strength and durability, pleasing appearance, good thermal and electrical conductivity and alloying ability. The production of alloys accounts for approximately 75% of total nickel consumption.

However, long term effects of sublethal concentrations of heavy metal like nickel on different aspects of physiology of fishes is not well understood. Kidneys besides liver are involved in the detoxification and removal of toxic substance circulating in the blood stream. Histopathology is used to study the impact of toxic materials as it provides the real picture of the toxic effects of xenobiotics in vital functions of a living organism (Ramalingam, 1985). Hence, it was thought to investigate the impact of long-term exposure of the fish, *Labeo rohita* to compound like nickel chloride on its kidney.

Material and Methods

Disease-free fish, *Labeo rohita* were bathed in 1% KMnO₄ solution and acclimated in big glass aquarium of 400 to 450 liter capacity for a period of 15 days. Chlorine free aged tap water was used in the aquaria. The water had pH 8.2 ± 0.2 ; hardness 280 mg/l; D.O. 6.2 mg/l; total alkalinity 310 mg/l and temperature $25 \pm 2^{\circ}\text{C}$. The fish were fed with rice bran daily at 10.30 am. The water in the aquaria was changed daily after the consumption of food supplied. The healthy fish of both the sexes and uniform size and weight ($125 \pm 2\text{g}$) were selected from the lot for experimental purpose. Initially 96h LC₅₀ dose was determined for nickel heavy metal compound by the method as described in standard methods by the APHA (1998).

Forty healthy fish from the stock were selected and were divided into two groups.

Group-I: Consisted of 10 fish in aged tap water which served as control. **Group-II:** Consisted of 10 fish kept in toxicant water containing 4mg/l nickel chloride for 30 days. The fish

were exposed to 4mg/l of nickel chloride which is 1×10^{th} of the 96h LC₅₀ concentration.

To avoid the effects of starvation, the fish were fed on the rice bran at the average feeding rate of 25mg food \ gm fish \ day. The toxicant solution and the aged tap water (control) were renewed every day in the morning after removing the unused food, to maintain uniform test concentration throughout the experimental period.

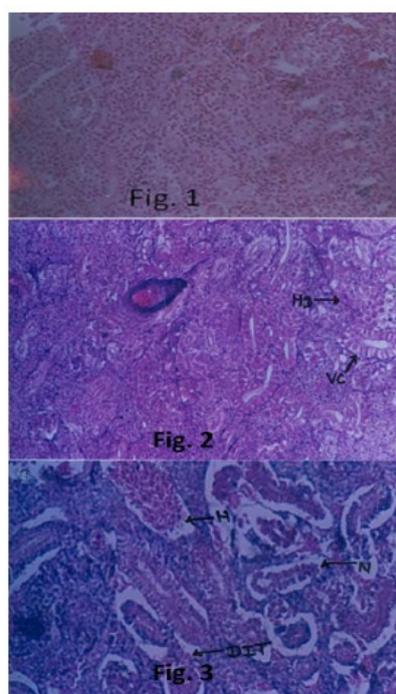
The controls as well as the experimental fish were sacrificed on the day 10 and 30. The kidney tissue was cut into small pieces of desirable size and fixed immediately into aqueous Bouin's fluid. The tissue was further processed by standard methods as described by Weissman (1972). The sections were cut and stained with haematoxylin-eosin, processed further, cleaned and then observed under microscope.

Observations

In fishes, the kidney is functionally as well as structurally differentiated into head kidney and trunk kidney. Histologically the trunk kidney is rich in nephrons and it takes the function of excretion. The intertubular space is full of lymphoidal tissue. The tubules are lined with tall columnar cells with distinct nuclei. The haemopoietic tissue in intertubular spaces has parenchymatous cells with distinct nuclei (Fig. 1).

After ten days of exposure to nickel chloride, proximal tubules showed vacuolated cells and vacuoles were noticed all around the section. The cells lining the uriniferous tubules appeared swollen and vacuolated. Their nuclei were also hypertrophied. Hydropic degeneration was seen at some places (Fig. 2). Fishes exposed to sublethal concentration of nickel chloride for thirty days exhibited haemorrhage in the glomeruli. Nephrosis and advanced vacuolation was observed (Fig. 3). Tubular and interstitial tissue was seen disintegrating.

Discussion



Fig, 1	T.S. of kidney, of the fish, <i>Labeo rohita</i> illustrating normal structure showing sections of uriniferous tubules with enraptured epithelial lining with distinct nuclei. Iron-haematoxylin – Eosin preparation. X400
Fig, 2	T.S. of kidney, illustrating the histomorphological changes after exposure of the fish, <i>Labeo rohita</i> to nickel chloride for 10 days as evidenced by Hydropic degeneration (HD) and vacuolated epithelial cells of uriniferous tubule (VC) Iron-haematoxylin – Eosin preparation. X400
Fig, 3	T.S. of kidney, illustrating the histomorphological changes after exposure of the fish, <i>Labeo rohita</i> to nickel chloride for 30 days as evidenced by haemorrhage if the glomeruli (H), nephrosis (N) and disintegrating interstitial tissue (DIT) Iron-haematoxylin – Eosin preparation. X400

The kidney plays a principal role in the accumulation, detoxification, and excretion of Ni and is considered to be a target organ for Ni toxicity (WHO, 1991; Eisler, 1998). Kidney is highly susceptible to toxic injury because of its high blood supply. The nickel induced histopathological changes observed in the kidneys of the fish, *Labeo rohita* are similar to pathological changes observed in other fishes due to heavy metal toxicity. Gardner and Yevich (1970), Wobesor (1975), Singhal *et al.*, (1996) reported necrosis and tissue damages in renal tubules of fish with heavy metal intoxication. Athikesavan *et al.* (2006) reported time dependent histopathological abnormalities in kidney of *Hypophthalmichthys molitrix* after nickel exposure.

The anatomical interdependence of structures in kidney implies that damage to one almost always secondarily affects the other. Edema is the first sign of any renal manifestation. Saxena & Saxena (2008) and Parvathi *et al.* (2011) have also reported the similar findings. Ruqaya *et al.* (2013) reported prominent tubular degeneration with severe interstitial mononuclear inflammatory cell infiltration along with mild interstitial and glomerular congestion during the winter seasons to severe tubular atrophy with endocrine type in the kidneys of *Schizothorax niger* when exposed to the heavy metal contamination. The lesions in nephrons and haemopoietic tissues suggest that both osmotic and ionic regulation were impaired upon exposure to sublethal concentrations of heavy metal salts. These findings are well in agreement with the earlier researchers (Kumar and Pant, 1981; Jarup *et al.*, 1993; Ray and Banerjee, 1998; Kumata and Kumar, 1998; Rana and Raizada, 2000; Abou Hadeed *et al.* 2008).

At end of 30 days treatment, ruptured cells, syncytical condition and pyknotic nuclei with aggregation of nuclei were seen due to the damage of plasma membrane. The glomerulus structure was disrupted, and the convoluted and uriniferous tubules are enlarged. The evidence of nephrotoxicity indicated by changes such as pyknotic nuclei, microvacuole degeneration, and shrinkage of glomeruli, swelling, and necrosis of the tubular epithelium in the second proximal tubule were reported by Athikesavan *et al.* (2006) these changes closely agree with the present findings.

In fishes, kidney is the main organ of excretion, therefore, the toxicant has to pass through it in its degraded forms, which may be more toxic, and as the histological units of kidney are more delicate, the lesions are seen in these parts of the kidney. The damage in the kidney due to heavy metals is mostly due to hormonal imbalance in toxic stress (Gliess, 1983). The accumulation of heavy metal salts and their degraded products might have a direct effect on enzymes of nuclear matrix, damaging the renal nuclei.

Conclusions

All these histological lesions indicated that the metal species enter in the cells by readily crossing the cell membranes where they might interfere with the enzyme systems resulting into morphological damage jeopardizing the vital functions like respiration and excretion. Disruption of the tubular cells resulted as the kidney had been induced to function actively under pollution stress to eliminate the

toxicants. The results of these studies may provide guidance to selection of tissues to be considered in the field biomonitoring efforts designed to detect the bioavailability of nickel and early-warning indicators of nickel toxicity in fish.

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