**International Journal of Environmental Sciences**
ISSN: 2229-7359
Vol. 11 No. 22s, 2025
https://www.theaspd.com/ijes.php

# Observations Of Histopathological Responses In Freshwater Murrels Channa Punctatus And Catfish Heteropneustes Fossilis Exposed To Copper And Zinc

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**ABSTRACT**
*Water pollution plays a primary role in the destruction of aquatic ecosystems. Anthropogenic activities involve the increased release of various toxic chemicals which ultimately reach the aquatic environments and become responsible for their degradation. Toxicity in fish is the culmination of a series of events involving various physical, chemical, and biological processes. The present investigation was planned to have a deeper insight into various toxicological changes with the impact of heavy metals copper and zinc on different freshwater fishes Channa punctatus and Heteropneustes fossilis. The main objectives were to investigate its toxicity on histological alteration of the gill of these experimental fishes. The percentage of mortality was significantly increased with the increase in concentrations of the copper and zinc toxicant at 96 hr duration of the experiment (F=848.13: C. punctatus; F=1122.78: H. fossilis; p<0.05: for copper, and . F=1393.42: C. punctatus; F=1649.67: H. fossilis; p<0.05: for zinc). On the basis of mortality of C. punctatus and H. fossilis under the exposure of copper and zinc heavy metals, $LC_{10}$, $LC_{25}$ and $LC_{50}$ values were calculated by probit analysis. The histopathological observations resulted that the severity and frequency of organ lesions were found to be more pronounced in fish treated with higher concentrations and even low concentrations at long-term exposure of heavy metals. The heavy metals induced marked histopathological changes in the gills the changes induced the bulging of the tip of the primary gill filament with distortion of the shape of the secondary filament gill lamellae. There was the tendency of fusion of disorganised secondary gill filament, epithelial proliferation, necrosis and haemorrhage in the central venous sinus of primary gill lamellae, congestion of blood vessels, hypertrophy of epithelial cells, clumping of chloride cells, pillar cells and blood channels, lamellar aneurysm and oedema in blood vessels and secondary lamellae.*

***Keywords:*** *Cooper, Zinc, histopathological, gill.*

## 1. INTRODUCTION

The freshwater ecosystem is under increasing threat due to the rapidly expanding human population and subsequent modernization process resulting in huge exploitation of nature. Rivers are very vulnerable, since waste in effluents from industries, domestic and farm open directly into them. Among the heavy metals, Cu and Zn are highly toxic to aquatic animals and have gained wide interest in the scientific community in recent years due to their potential human health hazards (Jezierska and Witeska, 2001). They are the most abundant necessary trace element and ubiquitous aquatic pollutants (Lobo et al., 2016).

Aquatic bodies may extensively be contaminated with heavy metals released from domestic, industrial, and other man-made activities. Heavy metal exposure proves to be useful for animal and human health at a limited concentration. Some of the heavy metals are essential to living organisms and they are commonly found in natural waters but high concentrations and accumulation of them may become toxic. Several pollutants including toxic heavy metals like cadmium (Cd), copper (Cu), lead (Pb), mercury (Hg) and zinc (Zn) are found to be universally present in rivers, lakes, reservoirs and are destructive to aquatic life and can exhibit toxic effects and death in the aquatic systems at elevated concentrations (Chavan and Jawale, 2013). However, at high concentrations, it accumulates in different organs, damage tissues and interferes with normal growth (Eriksen et al., 2001; Wepener

et al., 2001; Paquin et al., 2002; Lodhi et al., 2006) and have a lethal effect on the ecological balance of the recipient environment and the diversity of aquatic organisms (Vosyliene and Jankaite, 2006; Vinodhini and Narayanan, 2008).

As effluent accumulates in different tissues of the body, it is necessary to study in detail the histopathological changes of fish in order to know the extent of the damage. Hinton et al., 1992; Schwaiger et al., 1997; Teh et al., 1997). Gill is the first target of waterborne pollutants due to the constant contact with the external environment (Perry and Laurent, 1993) and is both morphologically and physiologically complex, performing multiple functions, such as gas exchange, ion and water exchange, acid-base balance, nitrogenous waste excretion, toxicant uptake, detoxification and several other metabolic transformations (Evans et al., 1988; Tang and Lee, 2011). The gills are in intimate contact with the liquid. As a result, the concentration of metals in a fish's gills reflects the concentration of those metals in its water of origin. Pollutants can directly cause degeneration or necrosis of gill tissues (Camargo and Martinez, 2007; Ayandiran et al., 2009). Histopathological and physiological changes in gills under acute and chronic exposure to heavy metals have been studied in many fish species (Rajbanshi and Gupta, 1988; Sola et al., 1995). Studies have shown that the intensity of lesions depends on the type of pollutants, concentrations and time of exposure which can further impair gaseous exchange (Mazon et al., 2002), thus compromising the survival of exposed individuals. In fishes, it is observed that the external organs are affected due to toxic chemicals, causing loss of equilibrium, increase in opercular movements, to and fro irregular vertical movements, finally leading to death. This may be attributed to the significant damage to the internal organs. The present study was planned to have a deeper insight into histopathological changes of the gill tissue with the impact of heavy metals copper and zinc on freshwater fishes Channa punctatus and Heteropneustes fossilis.

## 2 Materials And Methods

### 2.1 Experimental animal collection and maintenance

The fish were handled in accordance with local/national guidelines for experimentation on animals and all care was taken to prevent cruelty of any kind. Channa punctatus and Heteropneustes fossilis specimens employed in the current study, were collected from three different sampling places of lake Bhaghar in Tehsil Ramnagar, Barabanki (U.P.). Fish were acclimatized to laboratory conditions for 15 days. During this period, fish were maintained in 200 L capacity glass aquaria with proper care and an oxygenation system. Fish were treated with 0.05% $KMnO_4$ solution to remove dermal infection if any. For the entire duration of the experiment, fish were maintained under a natural light / dark cycle and fed regularly with commercial fish food pellets and goat liver. Diseased and dead individuals were removed immediately if any.

### 2.2 Estimation of median lethal concentration

A static toxicity bioassay was used to determine the median lethal concentration: $LC_{50}$ of different heavy metals (Zn and Cu). Ten healthy fish were exposed to different concentrations of Cu in these fishes were as follows: 0.5 mg/l - 8 mg/l in Heteropneustes fossilis and 0.1 mg/l - 1.4 mg/l in Channa punctatus. The different concentrations of Zn were as follows: 15 mg/l - 50 mg/l in Heteropneustes fossilis and 10 mg/l - 45 mg/l in Channa punctatus. The effects of each concentration were tested in triplicate with ten animals to verify reproducibility. Fish were not given any food during the experiment. Mortality was observed every day in each concentration.

### 2.3 Histological analysis

After obtaining 96 hr $LC_{50}$ of Cu and Zn, fish were exposed to 96 hr $LC_{50}$ for 30 days. Fish were sacrificed immediately at the end of the exposure period. Gill tissue was fixed in bouin's fluid for 24 h, dehydrated through a graded ethanol series and embedded in paraffin. Tissue sections (5 mm thick) were stained with haematoxylin–eosin. The thin sections of the tissues were stained by haematoxylin and eosin for observation by the Olympus bright field microscope.

## 2.4 DATA ANALYSIS

The fish mortality was presented in percentage. It was defined as the number of dead fish divided by total number of exposed fish in the aquarium multiplied by hundred. It was recorded every 24 hr up to 96 hr study period. The data were analysed statistically by one-way analysis of variance (ANOVA). The $LC_{50}$ with 95% confidence limit was estimated by probit analysis (Finney, 1971) with the statistic software (IBM SPSS version 20). The histological sections of Gill tissues were examined at 10X magnification under Olympus bright field microscope.

## 3 RESULTS

### 3.1 Lethal toxicity test

The result showed that both heavy metals copper and zinc were highly toxic to fish. The percentage of mortality was significantly increased with the increase in heavy metal concentrations at 96 hr duration of the experiment ($F=848.13$: C. punctatus and $F=1122.78$: H. fossilis $p<0.05$: copper exposure and $F=1393.42$: C. punctatus and $F=1649.67$: H. fossilis $p<0.05$: zinc exposure). Based on the mortality of C. punctatus and H. fossilis under the exposure of copper and zinc heavy metals, $LC_{10}$, $LC_{25}$ and $LC_{50}$ values were calculated by probit analysis (Table 1 and Table 2). The observation of heavy metals toxicity indicated that copper was more toxic as compared to zinc in different fishes. Among the fishes, Channa punctatus was significantly highly sensitive towards copper toxicity ($R^2=0.98$) as compared to H. fossilis ($R^2=0.976$) and both fishes showed similar sensitivity towards zinc toxicity (C. punctatus: $R^2=0.932$ and H. fossilis: $R^2=0.932$).

### 3.2 Histology of gills in Channa punctatus

The histological study of the gills shows a typical structural organisation of respiratory lamellae in unexposed fish. The gill tissue was composed of numerous gill filaments which have two rows of primary gill lamellae and secondary lamellae that run perpendicular to each filament. The lamellae were lined by a squamous epithelium composed of pavement and non-differentiated epithelial cells. Each lamella was made up of two sheets of epithelium delimited by many pillar cells. Between the lamellae, the filament was lined by a thick stratified epithelium constituted by several cellular types, such as chloride cells, mucous or goblet cells and pavement cells. Mucus cells and pavement cells were also present in the epithelium of the filament and at the base of lamellae. The gill filaments were covered with squamous pavement cells showing characteristic concentric patterns of micro ridges (Figure 1).

#### 3.2.1 Histopathology of gills under copper and zinc exposure

The heavy metals induced marked pathological changes in the gills the changes induced the bulging of the tip of the primary gill filament with distortion of the shape of the secondary filament gill lamellae. There was the tendency of fusion of disorganised secondary gill filament, epithelial proliferation, congestion of blood vessels, hypertrophy of epithelial cells, lamellar aneurysm, clumping of chloride cells, pillar cells and blood channels. and oedema in blood vessels and secondary lamellae. Fish exposed to copper showed necrosis and haemorrhage in the central venous sinus of primary gill lamellae. There was a number of activated goblet cells observed all over the region of the gill filament with detachment & hypertrophy on the central core of primary lamellae (Figure 1).

Zinc toxicity induced the detachment of the secondary epithelial cells from pillar cells, due to severe degeneration and necrosis, congestion of the central venous sinus of primary lamellae. Zinc toxicity induced sloughing of secondary lamellae and proliferation of epithelial cells such as chloride cells, pillar cells pavement cells, severe hyperplasia and necrosis of the cells of the central core and central venous sinus of primary gill filament and complete detachment & degeneration of secondary epithelial cells.

### 3.3 Histology of gills in Heteropneustes fossilis

The histological study of the gills showed a typical structural organisation of the respiratory lamellae in the untreated fish. There were four-gill arches and each arch was composed of numerous gill filaments with two rows of semicircular secondary lamellae that were aligned along both sides of the primary gill lamellae. The primary gill lamellae consisted of a centrally placed rod-like central axis with chloride cells and with blood vessels on either

side. The lamellae were lined by squamous epithelium and many capillaries split by pillar cells run parallel along the surface. Between the lamellae, the filament was lined by a thick stratified epithelium constituted by several cellular types, such as chloride, mucous and pavement cells. The secondary gill lamellae or respiratory lamellae was intact and covered by a thin layer of epithelial cells. The stratified epithelium was with a marked mucous cell layer and chloride cell. The secondary gill filament was consisting of pillar cells completely fused with the epithelial lining of the secondary gill lamella, and gill filament (Figure 2).

**3.3.1 Histopathology of gills under copper and zinc exposure**

Gills are found to be the primary site of copper accumulation in gills in H. fossilis. The fish gill is very sensitive to physical and chemical alteration of the aquatic medium. Thus, any histopathological changes in the gills might impair the respiratory function of the gills by reducing respiratory surface area resulting in hypoxia, and respiratory failure problems and this may lead to the death of fish. The sub-lethal concentration of copper for 15 days and 30 days exposure showed to damage gill- lamellae and gill-epithelium. The epithelial covering showed vacuolation, necrosis and haemorrhagic manifestation. The epithelial detachment, hyperplasia fusion of gill filaments and inflammation in gill lamellae were other indications of gill damage. The exposed fishes showed excessive secretion of mucous in the inter-lamellar spaces, degenerative changes in epithelial cells of secondary gill filaments, necrotic changes in inter-lamellar epithelial cells and twisting and clubbing of secondary gill lamella tips. As the concentration of heavy metal and duration of exposure increased, the fusion of secondary gill lamellae, reduction in length, swelling and bulging of epithelial cells of secondary lamellae and cytoplasmic vacuolization, necrosis in the tip of secondary gill lamellae and degeneration in gill rays were observed (Figure 2). The exposed group of fish treated with zinc showed noticeable pathological changes in fish gill architecture. The changes included the curling of secondary lamellae and desquamated epithelium. Other changes were observed such as rupture and breakdown of pillar cells, hyperplasia of epithelial cells and lifting of secondary gill lamellar epithelium. The gills of experimental fish showed extensive oedema of the epithelial cells and blood congestion (aneurism) in many areas of secondary lamellae with complete damage and breakdown of the pillar cell system at 30 days. Shortened and clubbing of ends of the secondary gill lamellae, the fusion of adjacent secondary gill lamellae and necrosis in the primary lamellae were well marked. Besides these changes, pyknotic nuclei, lamellar clubbing, rupture of secondary lamellar tips, oedema and rupture of epithelial cells were also observed at 15 days of exposure (Figure 2)

**Table 1:** $LC_{10}$, $LC_{25}$ and $LC_{50}$ values with its 95% confidence limit (upper and lower limit) at 96 hr exposure period in different freshwater fishes: Channa punctatus and Heteropneustes fossilis exposed to copper heavy metal.

| **Lethal Concentrations** | **Copper Toxicity** | **95 % confidence limits** | |
|---|---|---|---|
| | | **Upper Limit** | **Lower Limit** |
| **Channa punctatus** | | | |
| $LC_{10}$ | 0.19 | 0.24 | 0.12 |
| $LC_{25}$ | 0.42 | 0.46 | 0.37 |
| $LC_{50}$ | 0.68 | 0.72 | 0.64 |
| **Heteropneustes fossilis** | | | |
| $LC_{10}$ | 0.88 | 1.72 | 0.52 |
| $LC_{25}$ | 2.24 | 2.47 | 1.9 |
| $LC_{50}$ | 3.74 | 3.94 | 3.54 |

**Table 2:** $LC_{10}$, $LC_{25}$ and $LC_{50}$ values with its 95% confidence limit (upper and lower limit) at 96 hr exposure period in different freshwater fishes: Channa punctatus and Heteropneustes fossilis exposed to Zinc heavy metal.

| **Lethal Concentrations** | **Zinc Toxicity** | **95 % confidence limits** | |
|---|---|---|---|
| | | **Upper Limit** | **Lower Limit** |
| **Chnna punctatus** | | | |

**International Journal of Environmental Sciences**
ISSN: 2229-7359
Vol. 11 No. 22s, 2025
https://www.theaspd.com/ijes.php

| | | | |
|---|---|---|---|
| $LC_{10}$ | 16.65 | 19.68 | 12.19 |
| $LC_{25}$ | 22.84 | 25.09 | 19.85 |
| $LC_{50}$ | 29.72 | 31.80 | 27.66 |
| **Heteropneustes fossilis** | | | |
| $LC_{10}$ | 21.57 | 24.66 | 16.95 |
| $LC_{25}$ | 27.79 | 30.08 | 24.71 |
| $LC_{50}$ | 34.70 | 36.79 | 32.62 |

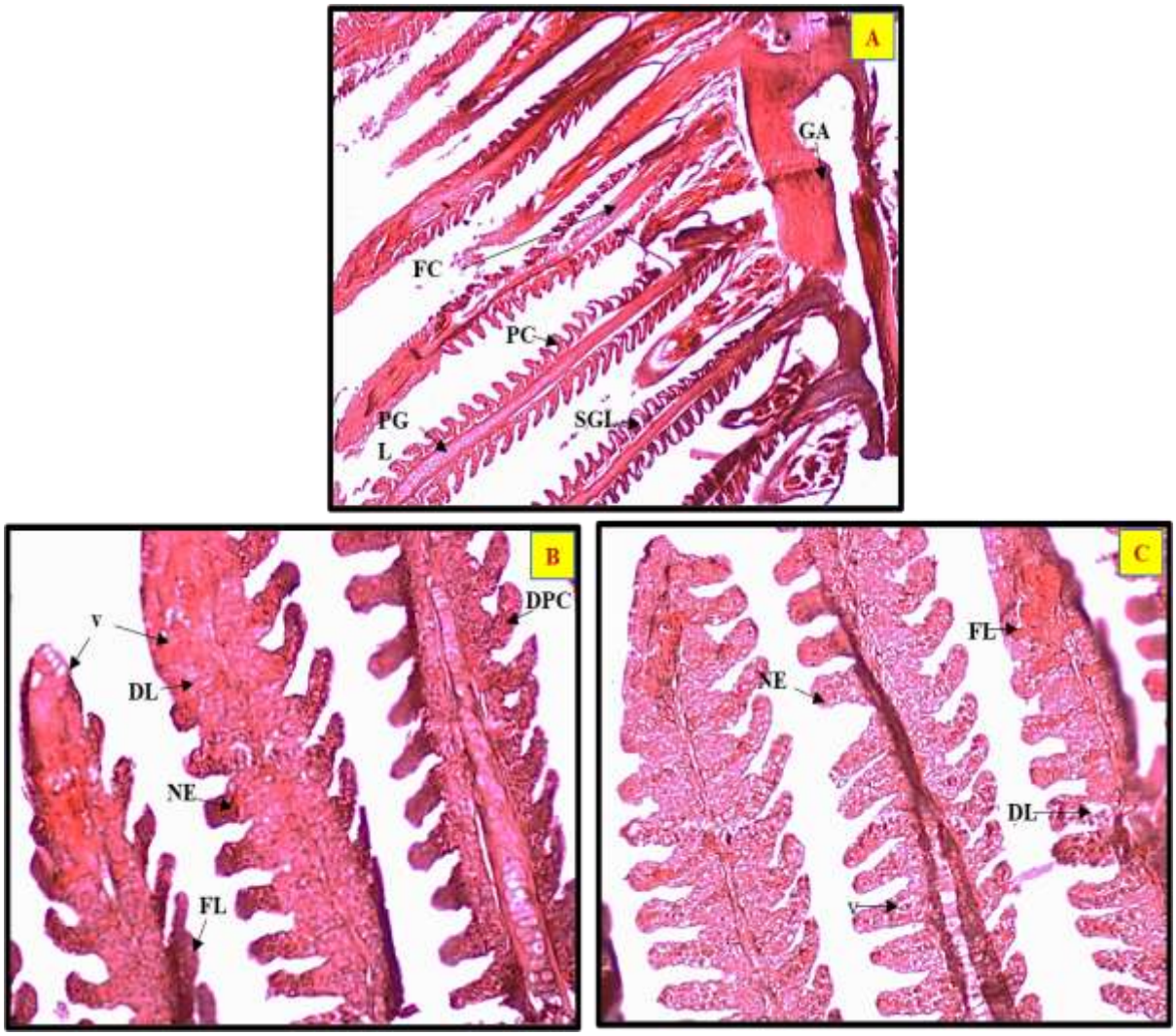


Figure 1: Photomicrograph of histological sections of gill of freshwater fish Channa punctatus was showed using haematoxylin & eosin stain, represented section of control fish (A) and exposed fish to 96 hr $LC_{50}$ copper (B) and zinc (C) for 30 days. The control section represents hepatocytes with their uniform nuclei, gill arch (GA), filament cartilage (FC), normal pilar cell (PC), primary gill lamella (PGL), secondary gill lamella (SGL) while treated fish to heavy metals showed cytoplasmic vacuolation (V), fused lamella (FL), necrosis (NE), damaged lamella (DL), degeneration of Piller cell (DPC). Images were acquired at 20x magnification.

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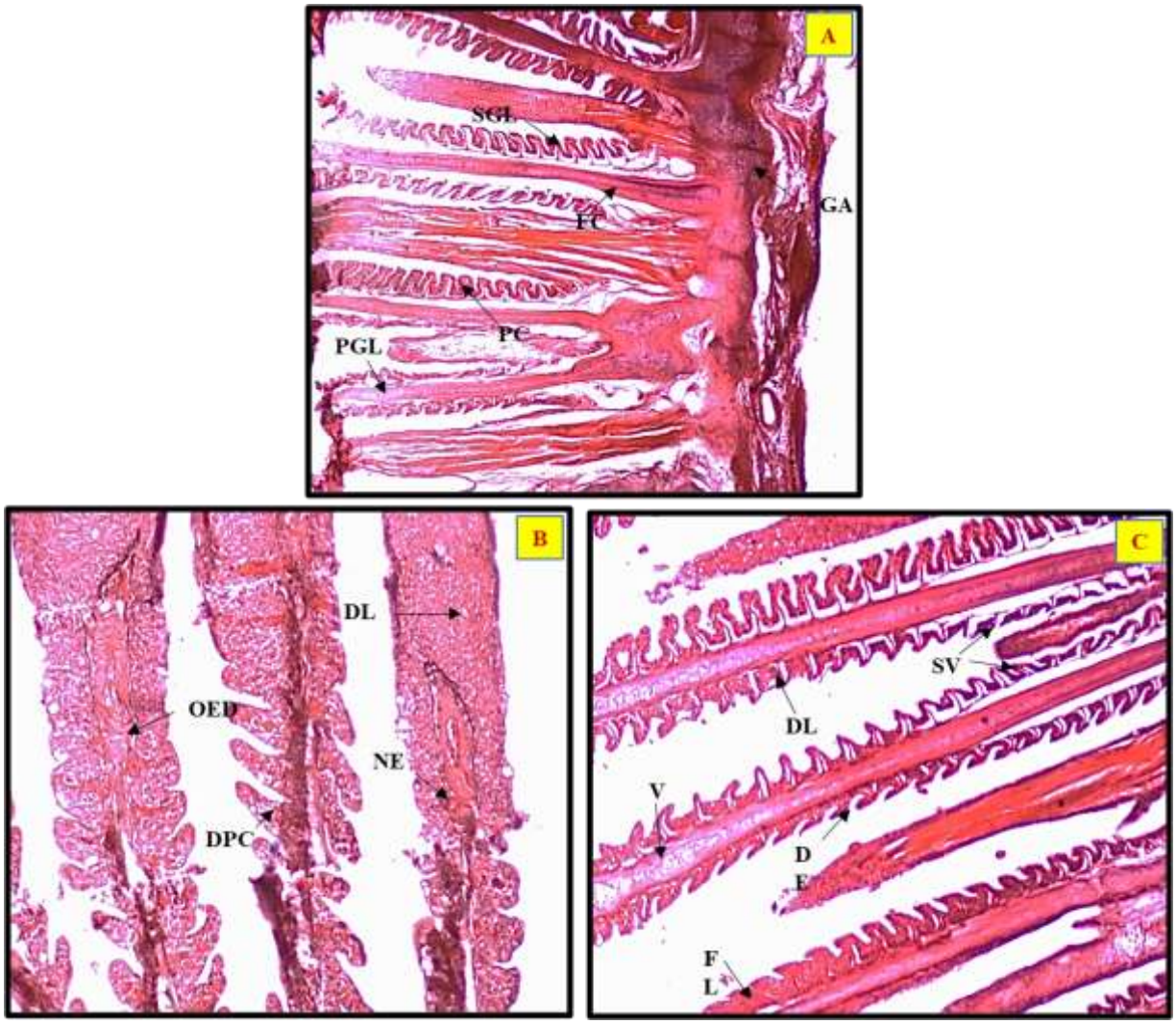


Figure 2: Photomicrograph of histological sections of gill of freshwater catfish Heteropneustes fossilis was showed using haematoxylin & eosin stain, represented section of control fish (A) and exposed fish to 96 hr $LC_{50}$ copper (B) and zinc (C) for 30 days. The control section represents hepatocytes with their uniform nuclei, gill arch (GA), filament cartilage (FC), normal pilar cell (PC), primary gill lamella (PGL), secondary gill lamella (SGL) while treated fish to heavy metals showed cytoplasmic vacuolation (V), fused lamella (FL), necrosis (NE), damaged lamella (DL), short villi (SV), degeneration of epithelium (DE), degeneration of piller cell (DPC) and oedema (OED). Images were acquired at 20x magnification.

## DISCUSSION

The present study showed that copper and zinc caused many histopathological defects in the gill tissue of C. punctatus and H. fossilis. The extensive architectural loss was observed in the gills of copper and zinc treated group. In the present study, after 30 days of exposure to high concentrations of heavy metals, epithelial necrosis, hypertrophy of the epithelial cells, rupture of gill epithelium, haemorrhage at primary lamellae and sloughing of respiratory epithelium were noted. The lifting of the epithelium, oedema, epithelial necrosis, fusion of adjacent secondary lamellae and haemorrhage at primary lamellae were observed in the gills of the fish examined after 28 days of exposure to zinc.

Another important histopathological change observed in the copper and zinc treated group was hyperplasia. Morphologically, hyperplasia refers to augmentation in the number of normal cells that constitute a given tissue. Gill alterations such as hyperplasia of the epithelial cells can be considered adaptive, since they increase the distance between the external environment and blood, serving as a barricade to the entrance of contaminants. Gill hyperplasia might serve as a protective mechanism leading to a decrease in the respiratory surface and an

increase in the toxicant-blood diffusion distance. Extensive epithelial desquamation was also observed in the treated group. It is well known that changes in fish gill are amongst the most commonly recognised responses to environmental pollutants (Mallatt, 1985; Laurent and Perry, 1991; Au, 2004). After acute exposure to hexavalent chromium, Channa punctatus exhibited marked degenerative changes in the histology of gills, kidney and liver tissues (Mishra and Mohanty, 2008). The gills of copper treated sea bass exhibited lamellar telangiectasis (localised dilation of blood vessel). This appearance of the secondary lamellae results from the collapse of the pillar cell system and breakdown of vascular integrity with a release of large quantities of blood that push the lamellar epithelium outwards (Alazemi et al., 1996). Complete lamellar fusion may have reduced the total surface area for gas exchange. Otherwise, they increase the distance of the water–blood barrier, which together with epithelial lifting and the increase in mucus secretion may drastically reduce the oxygen uptake. Epithelial necrosis and rupture of gill epithelium are direct deleterious effects of the irritants. The histopathological changes of gills an result in hypoxia and respiratory failure problems with ionic and acid-base balance (Alazemi et al., 1996). The histopathological changes observed in the gills of C. punctatus and H. fossilis in the present study are in good agreement with the results of (Jauch, 1979; Oliveira Ribeiro et al., 2000; Cerqueira & Fernandes, 2002; Parashar and Banerjee, 2002; Rao et al., 2003; Martinez et al, 2004; Olojo et al, 2005; Athikesavan et al.,2006; Giari, et al., 2007; Jiraungkoorskul et al., 2007; Gurcu et al., 2010).

## CONCLUSION

In conclusion, this study revealed that copper and zinc are more toxic to freshwater fishes that as evidenced by severe histopathological changes in vital organs like gills. Therefore, the use of metals on/near fish farms or in areas close to aquatic environments should be discouraged.

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