Cytoarchitectural changes of chromium toxicity in the gill of fresh water edible fingerlings of fish, *Cyprinus carpio*

G.Tulasi* and K. Jayantha Rao

Division of Toxicology, Department of Zoology, Sri Venkateswara University, Tirupati – 517502. AP., India

*E-mail: g.tulasi1979@rediffmail.com

Fishes come into contact with multiple metal contaminants in the aquatic environment and biomagnifies the pollutants. Pollutants build up in the food chains are responsible for adverse effects in aquatic organisms. Gills are generally considered as good indicators of water quality and critical organs of respiratory, osmoregulatory and excretory functions. Heavy metals accumulated in the tissues of fish generally lead to environmental oxidative stress in fish. Effects of water pollution are not only devastating to people but also to animals. Polluted water is unsuitable for drinking, recreation, agriculture and industry. It diminishes the aesthetic quality of lakes and rivers. More seriously contaminated water destroys aquatic life and reduces its reproductive ability, which is eventually hazardous to human health.

Fingerlings of fish, *Cyprinus carpio* with average length of 7cm and weight 6±1gm were acclimatized for 10 days with water with a exposure of 12hr light and 12 hr darkness. After acclimatization, fishes were divided into four groups each having ten fishes. First group considered as control. Remaining three groups exposed to chromium at 7ppm for duration of 7 days, 15 days and 30 days. Water was changed on alternate days and fishes were fed with commercial fish fed.

The gills exhibited a pathological change after chromium exposure at 7ppm.

The investigation of chromium toxicity effects at 7days, 15days and 30days of experimental fish showed degenerative changes in epithelial cells of respiratory lamellae and twisting of tips in secondary gill filaments. In few cases of fish exposed to chromium, the gills showed moderate necrotic changes in inter lamellar epithelial cells. Infiltration of cells in the primary axis and moderate changes were observed.

The gills of *Labeo rohitha* exposed to tannery effluent revealed fusion and clumping of primary lamellae and epithelium (Dhanapakiam *et al.*, 2004). Gills used as models for studies of environment impact such as xenobiotic agents (Fanta *et al*., 2003). Degenerative changes in the lamellae and edema were observed in the gills of fish exposed to heavy metals (Osman *et al*., 2009), fusion of secondary lamellae giving a club - shaped appearance of filaments (Khan *et al*., 2004), contraction and sloughing of respiratory epithelium in mercury exposed group of fish, *Channa puncta* (Gupta Neeraj and Dua Anish 2002), sub lethal ammonia concentration of Nile tilapia, *Oreochromis niloticus* on gill tissues displayed hyperemia, fusion in secondary lamellae (Benli Ac *et al*., 2008).

Toxicants intake or an adaptive response to pollutants may be due to increased capillary permeability in gills (Olurin *et al*., 2006). Nucleus with degenerative changes in parenchyma cells with necrosis in *C. carpio* due to heavy
metals was observed by Vinodhini and Narayana, 2009. Atrophy and fusion of gill filaments, disorganization and rupture in the secondary lamellae in gills of *Clarias gariepinus* exposed to lead toxicity (Adeyemo, 2008); epithelial hyperplasia and fusion of adjacent lamellae of *Catla catla* exposed to lead (Palaniappan *et al*., 2008). Fusion of secondary lamellae was also observed in the gills of fish from stream polluted by industrial, domestic and agricultural waste (Camargo and Martinez, 2007) and arsenics (Hwang and Tsai, 1993).

Various histological and physiochemical changes in a fresh water fish, *Tilapia mossambicus* under sublethal concentrations of phosphomidon and heptachlor was observed by Jayantha Rao, *et al*., 1984. Similarly degenerative and necrotic changes in white bass, *Lates calcarifer* exposed to cadmium was reported by Thophon *et al*., 2003.

Environmental conservationist point of view, chemicals should be degraded at faster rate, otherwise problems like bio accumulation and bio magnification will arise, which makes the non target life less fit for better survival.

**REFERENCES**


Benli Ac, Koksal G & Ozkul A (2008), Chemosphere, 72 (9) : 1355 – 1358.


[MS received 29 November 2011; MS accepted 12 February 2012]
**Fig. A - control gill (100X)**
SGF - secondary gill filament
RGF - respiratory gill filament
PA - primary axis

**Fig. B - 7 days gill (100X)**
RL - respiratory lamellae
PA - Primary axis

**Fig. C - 15 days gill (100X)**
HGSGF - hemorrhage in secondary gill filaments
NSGF - necrosis in secondary gill filaments

**Fig. D - 30 days gill (100X)**
NILS - necrosis in interlamellar space
ASGF - atrophy of secondary gill filaments
HGPA - hemorrhage in primary axis
NRL - necrosis in respiratory lamellae

**Fig. E - 30 days gill (400X)**
CSGF - clubbing of secondary gill filaments,
SNILS - seviour necrosis in inter lamellar space
SELSGF - separation of epithelial layer in SGF
FRL - fragmentation of respiratory lamellae

**Fig. F - 30 days gill (400X)**
NRL - necrosis in RL
ATRL - atrophy of RL
IFCPA - infiltration of cells in PA

*Disclaimer:* Statements, information, scientific names, spellings, inferences, products, style, etc. mentioned in *Current Biotica* are attributed to the authors and do in no way imply endorsement/concurrence by *Current Biotica*. Queries related to articles should be directed to authors and not to editorial board.