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Study of histopathological lesions in gill of air breathing cat fish

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Abstract
The gills play an important role in maintaining of whole animal ionic homeostasis in both freshwater and sweater environment. The present study suggest that electroplating industrial effluent chromium might act synergistically to cause toxic effects on the air-breathing fish Mystus Cavasius

Keywords: Histopathological Lesions and Breathing Cat Fish

Introduction
Histopathological changes in animals tissues are powerful indicators of prior exposure to environment stressors and are net result of adverse biochemical and pysilogical changes in an organisms. For filed assessment histopathology is often the easiest method assessing both short and long term toxic effects (Hinton and Lauren, 1990) [1]. Human activities that contribute to nickel loading in aquatic and terrestrial ecosystem include mining, smelting, refining, alloy processing, scrap metal reprocessing, electroplating, fossil fuel combustion and waste incineration (Chau and Kulikousky, 1995) [2]. Nickel accumulates in fish tissues and cause alterations in gill structure, including hypertrophy of respiratory and mucus cells, separation of the epithelial layer from the pillar cells system, cauterization and sloughing and necrosis of the epithelium (Nath and Kumar, 1989) [3]. Although aquatic organisms can accumulate in nickel from their surroundings there is little evidence of significant bio-magnification of nickel levels along food chains. Destruction of the gill lamellae by ionic nickel decrease the ventilation rate and may cause blood hypoxia and death.
The toxicity of nickel to aquatic life has been shown to vary significant with organism species, pH and water hardness (Birge and Black, 1990) [4], but elevated concentrations can cause sublethal effects. The present study was undertaken to elucidate the sublethal concentrations of industrial effluent nickel toxicity by observing the histological changes of gills of the Mystus cavasius.

Materials and Methods
Healthy live fishes Mystus cavasius (average length 15 – 25 g, 10-20 cm) were procured locally and acclimatized at 28°C and 12:12 L:D cycle for minimum 2 weeks prior testing. The fish were acclimated to the laboratory conditions with softened tap water under following conditions: Ca, 0.725mm, Mg, 0.135mm, pH 7.1±0.43, DO, 7.4 + 0.2 mg/l. The test medium was maintained daily (Sprague, 1971) to maintain the constant toxic concentration. The Percentage of survival of M. cavasius at various concentration of waste water was determined by adopting procedure laid down by Doudoroff and Katz (1953) [5].
Fishes in group A were maintained as controls whereas those in group B were exposed to electroplating industrial effluent nickel. 25 fishes were exposed to sublethal concentration (0.5%) of nickel in exposed to the period of 24h, 48h, 15d, 30d, 90d and 120 d. At different exposure period gills were dissected out by killing and preserved overnight in Bouin's and 10% natural buffered formalin.
Gills were processed by double embedding technique. Sections were cut at 6m thickness and stained with haematoxylin and eosin (dissolved in 70% alcohol) and mounted in canad balsam (Humason, 1972) [6]. The tissues samples were investigated by Olympus CX40 using a light microscope.

Results and Discussion
Toxic substances can injure gills, thus reducing the oxygen consumption and disrupting the

osmoregulator function of aquatic organisms (Saravana Bhavan and Geraldine, 2000) [7]. 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 toxicants and the period of exposure (Franchini *et al.,* 1994) [8]. The normal gill of Mystus cavasius the control is essentially similar to that found in fish described in order teleost as shown in Fig. 1 control).

In the fish exposed to sublethal concentration of electroplating industry effluent nickel (0.5%) after 24hr, the gill showed swelling of the epithelial cells.

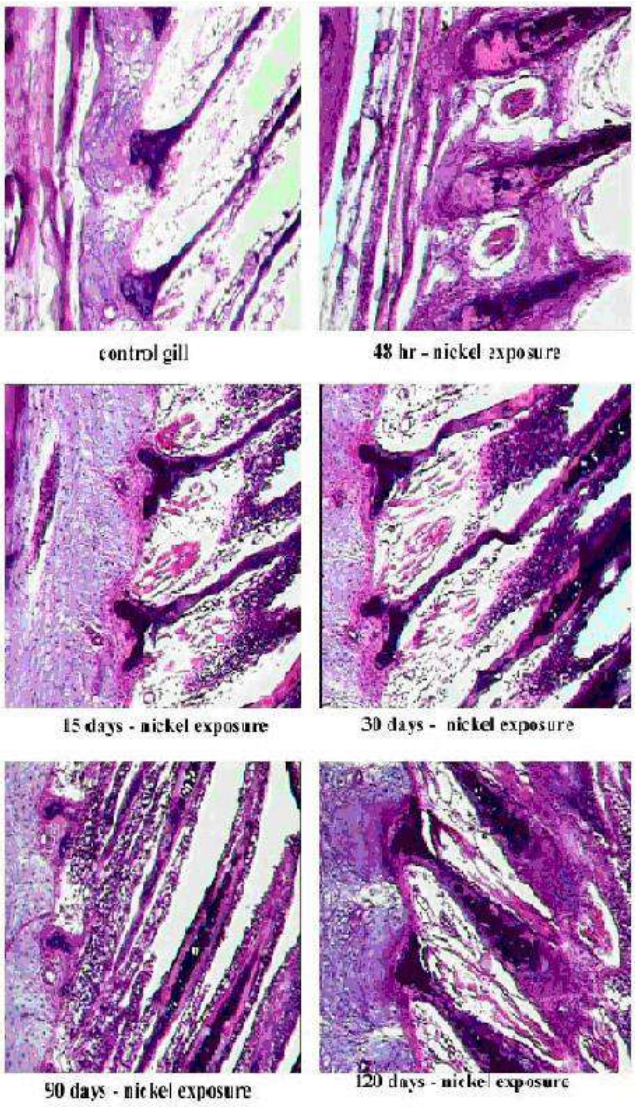

Fig 1: Control and tested Gills

Gills of the fishes are highly susceptible to water soluble toxicants when immersed in it. In the sublethal concentration 24hr exposure, mucus secretion at the gill surface forming thick coat cover it is, reported to be a protective device checking further penetration of the toxicants in the fish tissue via the gill (Solangi and Oversheet, 1982) [9]. The elicited excessive secretion of mucus in the interlamellar spaces at 15 days exposure.

The increased mucus secretion is also helpful in attenuating the osmotic influence of environmental stress in teleosts gills. The presence of mucus in the bellowing dilatation observed in the gill filaments may be considered as ion traps to concrete trace elements from water and favour cell adhesion between the neighbouring secondary lamellae serving to protect epithelia against both mechanical abrasion and infection as suggested by Olson and Fromm (1973) [10].

Dilation of blood vessels and partial (oedematous) separation of epithelial lining cells from the basement membrane of secondary gill lamellae was observed at 30 d exposure. Further, a few epithelial cells of secondary gill lamellae exhibited necrotic changes and cellular hyperplasia also noted at 90 d exposure. The entire interlamellar space were filled with the hyperplastic epithelium at 120 days exposure of the nickel. The present study confirmed the work of Dhanapakiam *et al.* (1998) [11] who have also reported similar degenerative changes in the respiratory epithelium of the fishes exposed to aquatic pollutants and metals. These pathological changes in respiratory gill might have been resulted into a shift from aerobic to anaerobic pathway in tissues under stress of the pollutants. Muller *et al.* (1991) [12] concluded that as a result of lamellar fusion, the lamellar surface area may be reduced by as 75%. This would impair ion uptake and oxygen delivery to the tissues. Enlargement of non-tissue space could result inadequate gas exchange, and consequently in a reduced diffusion capacity. These structural lesions of the gill could contribute to an increase in permeability. Increased gill permeability to cations could be the most important factor in the ion loss. It has been shown that under the normal conditions the permeability of the epithelial ells is determined by the characteristics of the cellular membrane and of the tight junctions that interconnect the epithelial cells (Muniz, J.P. and Leivestad, I.I. 1980) [13]. Calcium levels in the plasma are very important for the control of membrane permeability.

Conclusion

The permeability of the cellular membrane to water and ions is determined by the amount of calcium bound to the negatively charged groups of the membranes. The loss of bound calcium from the membrane and the tight junction of fish gill caused by the trace metals could to increased permeability consequently an increase in efflux of ions.

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