INTERNATIONAL JOURNAL OF APPLIED BIOLOGY AND PHARMACEUTICAL TECHNOLOGY

Volume: 2: Issue-2: April-June -2011 IJABPT *ISSN 0976-4550*

HISTOPATHOLOGICAL LESIONS IN GILL OF AIR-BREATHING CAT FISH *MYSTUS CAVASIUS* EXPOSED TO OF ELECTROPLATING INDUSTRIAL EFFLUENT NICKEL

P. Palanisamy*, G. Sasikala, D.Mallikaraj, N. Bhuvaneshwari and G.M. Natarajan

*Post Graduate Department of Zoology, Government Arts College, Coimbatore, Tamil Nadu, India.

ABSTRACT: The gill which participate in many important functions in fish, such as respiration, osmoregulation and excretion. Electroplating industrial effluent nickel induced hyperplasia, multiple telangiectases (aneurysms), desquamation of the epithelial cells, complete fusion of secondary gill lamellae and congestion of blood sinuses were the significant histopathological lesions observed in the gill of *Mystus cavasius*.

Key words: Electroplating industry, nickel, *Mystus cavasius*, primary lamellae, secondary lamellae, gills, mucus secretion, hyperplasia, epithelial cells, interlamellar space,

INTRODUCTION

The teleost fish gills play an important role in maintaining of whole animal ionic homeostasis in both freshwater and sweater environment (Evans, 1993). The rapid increase in both human population and the number of industries has resulted in large amounts of industrial effluents and wastes being discharged into the water bodies. To date, relatively little known about the effects of various toxicants on the air-breathing, cat fish *Mystus cavasius*. Owing to their direct and continuous contact with the environment, fish gills organs for respiratory gas exchange, osmoregulation, excretion of nitrogenous waste products and acid-base regulation are directly affected by contaminants (Bhagwant and Hahee, 2002) Navaraj (2003) reported that electroplating industry effluent metals was highly affected the respiratory rate and oxygen consumption of fish, *Oreochromis mossambicus*. Metals may be taken up by aquatic organisms via the general body surface, across the gills and through the gut lining following the ingestion of food and metal containing particulate material (Brayan, 1971 Koli *et al*., 1978; Memmert, 1987 and Senders, 1997). Conklin *et al*. (1992) observed both a significant increase and rapid degeneration of chloride cells population in the gills of larvae brook trout exposed to soft and acid water. In addition filament length, lamellar length and the number of lamellae per filament were decreased.

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). 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 (NAS, 1975; WHO, 1991; Chau and Kulikousky, 1995).

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). Although aquatic organisms can accumulate in nickel from their surroundings there is little evidence of significant bio-magnification of nickel levels along food chains (NRCC, 1981; Sigel and Sigel, 1988 and WHO, 1991). Destruction of the gill lamellae by ionic nickel decrease the ventilation rate and may cause blood hypoxia and death (Ellgaard et al., 1998). The toxicity of nickel to aquatic life has been shown to vary significant with organism species, pH and water hardness (Birge and Black, 1990), 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). 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 over night in Bouin's and 10% natural buffered formalin. Gills were processed by double embedding technique. Sections were cut at 6μ thickness and stained with haematoxylin and eosin (dissolved in 70% alcohol) and mounted in canad balsam (Humason, 1972). The tissues samples were investigated by Olympus CX40 using a light microscope.

RESULTS AND DISCUSSION

Histopathology provides a rapid method to detect effects of irritants in various organs (Johnson et al., 1993). The exposure of fish to chemical contaminants likely to induce a number of lesions in different organs (Bucke et al., 1996). Gill (Poleksic et al., 1994) is a suitable organ for histological examination in order to determine the effect of pollution. Gill covers more than 60% surface area of the fish and its external location renders it the most vulnerable target organs for the pollutants (Roberts, 1989). Toxic substances can injure gills, thus reducing the oxygen consumption and disrupting the osmoregulator function of aquatic organisms (Saravana Bhavan and Geraldine, 2000). 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 (Oliveira et al., 1996 and Franchini et al., 1994). The normal gill of *Mystus cavasius* the control is essentially similar to that found in fish described in order teleost (Fig1-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.

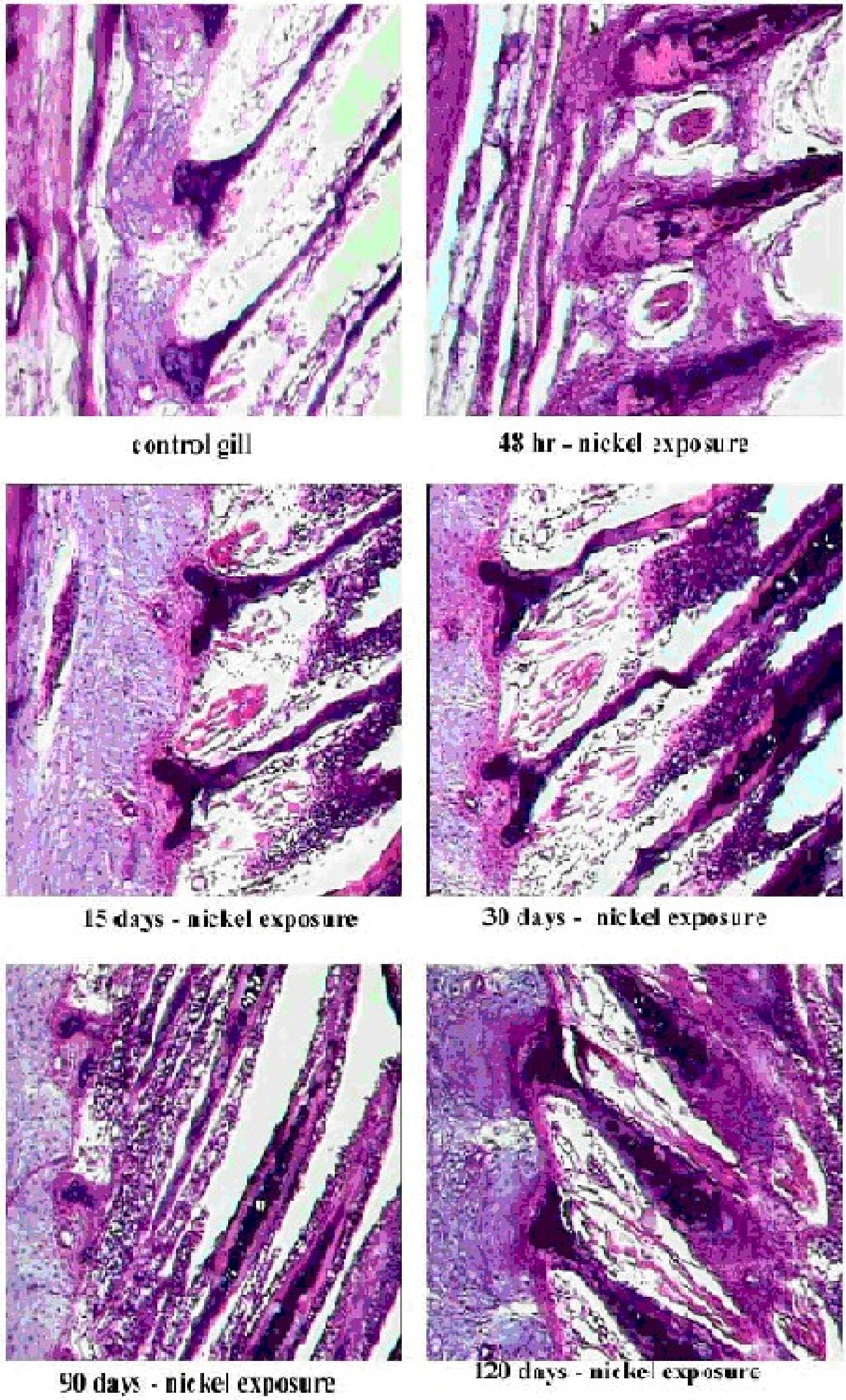

Figure-1: Control and tested Gills

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). 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).

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) 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) 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). Calcium levels in the plasma are very important for the control of membrane permeability.

Hunn (1984) suggested that 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 lead to increased permeability, and consequently an increase in efflux of ions. The present study also suggest that electroplating industrial effluent chromium might act synergistically to cause toxic effects on the air-breathing fish *Mystus cavasius*.

REFERENCES

A.K. Koli, W.T. Canty, K.I. Felix, R.J. Reed and R. Whitmore (1978). Trace metals in some fish species of South Carolina. Bull Env. Cont. Toxic., 20 (3). 328-331.

Bhagwant and KB. Hahee (2002). Pathologic Gill lesions in two Edible fish species, from the Bay of Poudred, Or, Mauritius. *Western Indian Ocean. J. Nar. Sci*.1. 35- 42.

Conklin *et al*., (1992). Effects of chronic exposure to soft, acid water on gill development and chloride cells numbers in embryo –larval brook trout. *Salvelinus fontinalis*. *Aquat. Toxicol.* 22. 39 -52.

D.E. Hinton and J.C. Lauren (1990). Integrative histopathology approaches to detecting effects of environmental stress on fishes. *Am. Fish. Soc. Symp*. 8. 51 – 66.

D.H. Evans (1993). Osmotic and ionic regulation In: *The physiology of fishes*, edited by D.H. Evans, Boca Raton, FL: CRC, pp 315 – 341.

E.S. Ellgaard, S.E. Ashley, A.E Langford. and D.C. Harlin, 1995. Kinetic analysis of swimming behaviour of the of the gold fish *Carassius auraties* exposed to Nickel; hypoactivity induced by sublethal concentrations. *Boll. Env. Con. toxicol.* 55. 926 – 936.

F, Frachini, Alessandrini and A.M. Fantin. (1994). Gill morphology and ATPse activity in the goldfish *Carassius carassius* var. auratus exposed to experimental lead intoxication. *Boll. Di Zool.,* 61. 29 – 37.

G.L. Humason (1972). *Animal tissue technique III* (Ed) W.H. Freeman and Co. San Francisco, U.S.A.

G.W. Bryan (1971). The effect of heavy metal (other than mercury) on marine and estuarine organisms. Proc. Roy. Soc. Lond. B. 177. 389 – 410.

H. Sigel and A. Sigel (1988). Metal ions in biological systems. Nickel and its role in biology. Marcel Dekker, New York. Vol. 23. pp 488.

J. Brusle, I. Gonzalez and G. Anadon (1996). The structure and function of fish liver. Fish morphology. In: Munshi JSD and Dutta HM(Eds). Newyork, Science Publishers Inc.

J. Muniz, and H. Leivestad (1980). Acidification effects on freshwater fish. Ecol. Impact Acid Preeip. Proc., Int. Conf. pp 84 -92.

J.B. Hunn (1985). Role calcium in gill function in freshwater fishes. *Com. Biochem. Physiol.* 82A : 543 – 547.

K. Nath, and N. Kumar (1989). Nickel induced histopathological alteration in the gill architecture of tropical freshwater perch, *Collisa fasciatus*. The science of the Total Environment. 80. 293 – 296.

K.R. Olsen and P.O. Fromm (1973). Uptake of chromium by fish gills. *Z. Zell. Sch.,* 143 – 434.

L.L. Johnson, C.M. Stehr, O.P. Olson, M.S. Myers, S.M. Pierce, C.A. Wigren, B.B. McCain and U. Varanasi (1993). Chemical contaminants and hepatic lesions in winter flounder (*Pleuronectes americanus*) from the Northeast Coast of the United States. *Environ Sci Technol*, 27. 2759 – 71.

M.A. Solangi and R.M. Overstreet (1982) Histopathological changes in two estuarine fishes, *Meriab berylllina* (Cope) and *Trinectes maculatus* exposed to crude oil and its water-soluble Gactions. *J. Fish Dis.* 5. 13-Z5.

M.E. Muller, D.A. Sanchez, H.L. Bergman, D.G. McDonald, R.G. Rhem, and C.M. Wood. (1991). Nature and time course of acclimation to alluminium in juvenile brook trout (*Salvelinus fontinalis*). II. Histology. *Can. J. Fish. Aquat. Sci.* 48. 2016 – 2027.

M.J. Sanders (1997). A field evaluation of the freshwater river crab Protamonautes warren: as a bio-accumulative indicator of metal pollution. Thesis. Rand Afrikaans University, South Africa.

National Academy of Science (NAS). 1975. *Medical and biological effects of environmental pollutants.* Nickel. National Research Council, National Academy of Science, Washington, D.C. pp 277.

National Research Council of Canada (NRCC). 1981. *Effects of nickel in the Canadian environment.* Publication No. NRRC, 18568, NRCC/ CNRC, Ottawa, Canada. pp 277.

P. Dhanapakiam, V.K. Ramasamy and R. Sampoorani (1998). A study on the histological changes in gills of *Channa punctatus* in Cauvery river water. *J. Envrion. Biol.,* 19. 265-269.

P. Doudorff and M. Katz 1953. A critical review of literature on the toxicity of industrial wastes and their components to fish. II. The metals as salts. II. *Sewage Ind Waste.,* 25. 802 – 839.

P. Saravana Bhavan and P. Geraldine (2000). Histopathology of the hepatopancreas and gill of the prawn *Macrobrachium malcolmsonii* exposed to endosulfan. *Aquat Toxicol*, 50. 331 – 339.

P.S. Navaraj (2003). Synergetic effect of metals of electroplating industry effluent on physiology of the Oreochromis mossambicus. *J. Phys. IV. France*. 107. p 925.

R.J. Robert (1989). Fish Pathology. 82A : 543 – 547.

Spry *et al*. (1981). The effects of environmental acid on freshwater fish with particular reference to the soft water lakes in Ontario and the modifying effects of heavy metals. A literature review. *Can. Tech. Rep. Fish. Aquat. Scie*. 999. 144.

U. Memmert (1987). Bioaccumulation of zinc in two freshwater or- _ ganisms *Daphnia magna*, Crustacea and *Brachydanio rerio*, . Pisces . Water Res. 21. 99 - 106.

V, Poleksic, V. Mitrovic-Tutundzic (1994). Fish gills as monitor of sublethal and chronic effects of pollution. Sublethal and chronic effects of pollutants on freshwater fish. In : R Muller and R. Lioyd (Eds).Oxford, UK, FAO Fishing News Book. 339 – 352.

W.J. Birge and J.A. Black (1980). Aquatic toxicology of nickel. *In: Nickel in the environment* (Nriagu, J.O (Ed.), John Wiley and Sons, New York, pp 349 – 366.

World Health Organization (WHO). 1991. *Nickel. Environmental Health Criteria,* 108, pp. 383.

Y.K. Chau, and Kulikovsky - Cordeiro, G.P. 1995. Occurrence of nickel in the Canadian environment. *Environmental Rev.,* 3. 95 – 120.