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Histopathological alterations in the gill and kidney of an estuarine mullet, *Liza parsia* (Hamilton-Buchanan), caused by sublethal exposure to lead (Pb)

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ABSTRACT

In order to assess the effects of lead (Pb) on gill and kidney of an estuarine fish, *Liza parsia* the fish was exposed to sublethal concentration (0.5 ppm) of lead nitrate in sea water for 15 days. Gill responded initially with an enhanced secretion of mucus and oedematous separation of the epithelial lining cells from the basement membrane of the secondary gill lamellae. 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 during the period of observation. The entire interlamellar spaces became filled with the hyperplastic epithelium by day 15 of Pb exposure. Trunk kidney of the treated fish revealed initial hypertrophy and vacuolation of the epithelial cells lining, proximal convoluted tubules and shrinkage in the glomeruli resulting in dilation of Bowman's space on day 4. Necrotic changes like oedema, pycnosis, karyorrhexis, karyolysis, cytolysis and fibrosis were observed in the kidney of the fish exposed to Pb intoxication.

Introduction

Lead (Pb), a non-beneficial and non-essential heavy metal to animals is capable of causing hypertension, atherosclerosis, nephropathy, hepatopathy, neuropathy and neoplasia among mammals (WHO, 1977; DeMayo *et al.,* 1982). The presence of Pb in natural water is largely due to a variety of anthropogenic activities related to the increasing mining operations and industrial use (Ruvio, 1972). Occurrence of lead (Pb) in the marine/coastal waters and in fishes has been recorded from different parts of the world (Chow *et al.,* 1974; Stoeppler and Nurnberg, 1979; Buggiani and Vannucchi, 1980). Presence of Pb has also been recorded from water, sediments, zooplankton and fishes of Indian coasts and Andaman waters (Kureishy *et al.,* 1983). It is reported that there are certain "hot spots" of Pb pollution near Bombay (Patel *et al.,* 1985; Varshney and Abidi, 1992) and Madras (Nammalwar, 1987). We do have information about Pb toxicity in a

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the pillar **(pilaster) cell system** remained unaffected even by day 15 **of** Pb intoxication,

Nephrons of the **trunk** kidney of *Liza parsia* maintained in sea water (control) consisted of tufts of capillaries **forming glomerulus surrounded by Bowman's capsule,** a short neck, first and second **proximal** convoluted tubules **and** collecting duct. Tine interstitial **spaces of** trunk kidney **were** occupied by **the** actively dividing **haemopoetie** tissue (Fig. 9). Kidney of the **mullets** revealed swelling (hypertrophy) of **the** epithelial

with excessive **hypermia on** clay 12 of Pb

y of *Liza parsia* exhibiting
inkage in glomeruli and
clay 10 of Pb intoxication

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Kidney of fish accumulates Pb (Buggiani and Vannucchi, 1980) and is an important target organ for this heavy metal (Crandall and Goodnight, 1963; Sippel *et al.,* 1983). Crandall and Goodnight (1963) observed pathological symptoms like dilation of renal tubules and reduction of lymphoid tissue in the kidney of *Lebestes reticulatus* in response to prolonged (3 months) exposure to Pb (1.25 mg/1). Renal tissue of *Liza parsia* exhibited swelling (hypertrophy) of the cells lining the promixal convoluted tubules, glomerular shrinkage, oedema and degenerative changes like variolation, pycnosis, karyorrhexis, karyolysis and cytolysis of the cells (tubulonecrosis) till day 8 of Pb exposure. Occurrence of fibrosis under prolonged Pb intoxication (Figs. 13, 14) may probably represent an adaptive measure to protect the kidney against the pollutant (Roberts, 1989).

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