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LEAD AND MERCURY ALTERS LIPID CONTENTS IN LIVER AND KIDNEY OF *HETEROPNEUSTES FOSSILIS*

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The toxic effect of lead nitrate ($Pb NO_3$) and mercuric nitrate ($Hg NO_3$) on the activity of a few lipids like phospholipids, Neutral Lipids in the liver and kidney of *Heteropneustes fossilis* have been analysed histochemically. Impact of these heavy metals on hepatic and nephric tissues interfere with protein and lipid metabolism. Our experimental evidences may be useful in revealing the mechanism of injury caused by these heavy metals.

INTRODUCTION

The heavy metals, greatly increasing in the biosphere to upset the natural balance of the ecosystem, are challenging the existance of life over this planet. Lead and mercury are recognised as toxic contaminents of our environment. Lead being a cumulative poison, even consumed as a small dosage, toxic effect produced in man and other organism (Harrison *et al.*,1971). According to Methis & Kevern (1975), this element is quite toxic to aquatic organism including fishes which are the most sensitive group. Mercury compounds show their significant effect on different tissues of the animals. They even disturb the composition and amounts of proteins, amino acids, carbohydrates, lipids and moisture contents (Doudroff & Katz, 1953; Merlini & Pozzi, 1977).

Lipids are the heat insulators and reserve suppliers of energy. They are present in all the vegetable and animal matter hence are widely spread in nature. Generally lipids in living cells, together with proteins, carbohydrates form an essential part of the colloidal complex of cytoplasm. Histological studies reveal that liver and kidney, being two vital organs of animal body undergo considerable change when the animals intake these heavy metals. It was felt that an animal model showing specific effects of these metal pollutants on the distribution of lipids mainly phospholipids and neutral iipids, could be useful in our understanding of physiological mechanism of damage. For specific localization of these toxic effects histochemical parameters were selected.

MATERIALS AND METHODS

Fishes were collected from local freshwater sources and maintained in the laboratory aquarium for experimentation. They were allowed to acclimatize to the laboratory conditions for six days. Specimens weighing 50-60 gms were selected and divided into three groups of 20 fishes each. The water was aerated with gas mixture of 15% O_2 and 5% CO_2 for two hours each day, the fishes were fed with commercial fish food, twice a day during the tenure of the experiment . The first group was maintained in a sub lethal medium containing 2.5 mg/l of lead nitrate for 15 days, while the second group was maintained in sub lethal medium containing 2.5 mg/l of mercuric nitrate for 15 days. The third group served as control. Two fishes in first and three fishes in second group died between 8-12 days while no mortality was recorded in third group.

After 15 days fishes were dissected from each of the group and liver and kidney were carefully removed and fixed in 10% neutral formaline (as a fixative). Paraffin sections were prepared and subjected to following histochemical tests.

(a) Neutral lipids - Propylene glycol sudan Black B method (Mc Manus, 1946).
(b) Phospho lipids - Baker's Acid - haematein test (Pearse, 1961).

RESULTS

Neutral lipids

Liver : Control fish liver showed a uniform reaction in sinusoids and parenchymal cells. Lead treated fishes showed accumulation of neutral lipids near the portal vein. The mercury fed fishes liver could exhibit a little stimulation in the centrolobular region. Details are exhibited in Table I.

Kidney : In control kidney, neutral lipids were localized mainly in the proximal convoluted tubules. After lead intoxication a stimulated reaction was observed in proximal and distal convoluted tubules and in medullary regions. The mercury fed fishes showed enormous increase in neutral lipids in proximal convoluted tubules. Details are exhibited in Table II.

Phospho Lipids

Liver : In control fishes, a unifrom distribution of phospholipids was observed throughout the liver lobules. In the liver of mercury fed fishes, the deposition of phospholipids was in the form of fatty cysts in the perilobular region of the lobule. The lead fed fishes liver also exhibited cysts but were comparatively smaller in size and were evenly distributed from centrolobular to perilobular regions. Details are exhibited in Table I.

Kidney : The control kidney showed a uniform distribution of phospholipids in the epithelial cells of the tubules. Lead fed fishes could show a stimulation of phospholipid activity in the medullary region. In mercury fed fishes, no stimulation was noticed in the kidney. Cyst formation after mercury fed, was also not observed in the kidneys. Detailed observations are exhibited in Table II.

Table I : Distribution of lipids in the liver of *H. fossilis* after exposure to lead nitrate and mercuric nitrate.

Object	Treatment	Centrolubular region	Peripor-region	Hepatic parenchyma	Smusoids	Bile Canalicules
Neutral Lipid	Control	±	±	++	++	-
	Lead	-	+++	±	±	-
	Mercury	+++	+	±	±	-
Phospho Lipid	Control	±	+	++	+	-
	Lead	+++	±	++	+	-
	Mercury	+	+++	++	±	

+++ : Intense; ++ : Moderate; + : Average; ± : Dull; -: No reaction.

Table II : Distribution of lipids in the kidney of *H. fossilis* after exposure to lead nitrate and mercuric nitrate.

Object	Treatment	Proximal con-voluted tubules	Disal con-voluted tubules	Glomeruli	Medullary region
Neutral Lipid	Control	±		-	+
	Lead	++	+	-	++
	Mercury	+++	+	-	+
Phospho Lipid	Control	±	±	-	+
	Lead	±	+	-	+++
	Mercury	+	+	-	+

+++ : Intense; ++ : Moderate; + : Average; ± : Dull; - : No reaction.

DISCUSSION

The lipids moistening the surface content of a surfactant which changes the surface tension properties of a liquid film in a manner on the extent of compression of the surface area. When a surfactant is concentrated in the liquid it dilutes other molecules of the solution, dilutes molecules which have high attractive force interactions and in this way lowers the surface tension at the interface. The surface tension reducing properties of these surfactant are dependent mainly upon phospholipids (Harlan *et al.*, 1966; Morgen *et al.*, 1956). Thus lipids and mainly the phospholipids passes greatest physiological importance in the liquid moistening process. Present histochemical studies show regional aggregation of neutral and phospholipids in the liver of *Heteropneustes fossilis* fed on lead and mercury. Kidney from both the groups showed a stimulated reaction. Accumulation of lipids in liver can be induced by a multiplicity of agents. Heard & Platt (1964), describe nutritional etiology of fatty liver. Recknagel & Ghoshal (1966), Rana (1974) studied accumulation of lipids after CCl_4 poisoning. Diluzio *&* Hartman (1967), studied in pathogenesis of ethenol induced fatty liver. However, no report is available after heavy metal poisoing. Like other hepatotoxins, lead and mercury also damage membranous structures promoting peroxidation of lipids. Increased rate of triglyceroid input or impairment of fatty acid oxidation thus caused would accentuate the accumulation of lipids inhibiting the biosynthesis of lipo-proteins. By present observations and discussion, it seems quite possible that lead and mercury increased glycerophosphate generation that diverts the available fatty acids away from the B - oxidation pathway of fatty acids catabolism. Thus it may be concluded that our laboratory experimental observations may be helpful to dispel doubts concerning the reliability of a heavy metal exposures to fishes. The data further will suggest the possibility of continuous exposures to lead and mercury may produce extensive change in the lipids contents of liver and kidney of *H. fossilis*.

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