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Histopathological study of nickel induced alterations in the fresh water bivalve, *Lammellidens marginalis*

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ABSTRACT: Chronic exposure for 30 days to nickel chloride (0.227ppm, equivalent to the $LC_{50/10}$ of 96 hours exposure) showed the results of histomorphological impact on gills and digestive glands of the bivalve, *Lammellidens marginnalis*. The study revealed that the gill lamellae and hepatic lobule were badly affected by salt of nickel chloride and the severity of the effect was time dependent.

Key words: *Lammellidens Marginalis*, Nickel, Histopatholgy, Fresh water bivalve

Introduction

Histological approach is the most valuable tool for assessing the action of toxicants at tissue level providing data concerning tissue damage and also it manifests structural and functional changes (Sprague, 1971) in tissues and organs. Histological studies have a way for understanding the pathological conditions of the animal by helping in diagnosing the abnormalities or damages of the tissues of animals exposed to toxic stress of heavy metals.

Heavy metal affects histological structure, physiology, biochemistry, cellular structures and molecular biology of normal tissues. Since heavy metals are known to interfere with functional groups of macromolecules, the presence of ionic heavy metal above a threshold level results in irrevocable alterations in the microenvironment of the cell, thus resulting in its morphological changes towards degeneration. Histopathological abnormalities caused due to toxicity of heavy metals in animals have been reported earlier (Shastry and Sunita, 1984; Shrivastava and Maurya, 1991). Many investigators have reported toxicant induced histopathological abnormalities and regenerative changes in certain tissues of various animals (Goel and Garg, 1980 and Bhattacharya and Ray, 1984).

Histological study provides data concerning tissue damage at structural and functional level (Sprague, 1971) in tissues and organs and may provide the site of the action of the toxicant. Histopathological changes in various tissues affected of various compounds of the heavy metals are mainly studied in invertebrates, amphibians and mammals.Nickel Toxicity to humans, includes carcinogenicity, reproductive and developmental toxicity, neurotoxicity, and acute toxicity. This depends on how much nickel a person has been exposed to, for how long, the nature of the nickel compound(s), and current state of health. An allergic skin reaction is the most common adverse health effect in people who are sensitive to nickel. People can become sensitive to nickel when jewellery or other products containing nickel are in direct contact with the skin. Once a person is sensitized to nickel, further contact with nickel will produce a reaction, most commonly a skin rash at the site of contact (dermatitis). Less frequently, some sensitized persons may have asthma attacks following exposure to nickel. Some people may react when they eat nickel in food, drink it in water, or breathe dust containing it.

Exposure to nickel salts can cause 'nickel itch', which causes burning and itching sensations in the hands, followed by abnormal redness of the skin and nodular eruption on the web of fingers, wrists and forearms. Nickel salts act as emetics when swallowed. Workers who accidentally drank water containing very high levels of nickel (100,000 times more than in normal drinking water) had stomach aches, and blood and kidney disorders. Nickel dust is irritating to the eyes, nose and throat. Lung effects, including chronic bronchitis, reduced lung function, and lung cancer, as well as nasal effects, including rhinitis, nasal sinusitis, nasal mucosal injury and sinus cancer, have been observed in workers who breathed high levels of nickel while working in nickel refineries or nickel processing plants. Nickel platers exposed to nickel sulfate and welders exposed to nickel oxides have been linked with asthma. Evidence for the carcinogenicity of nickel metal and other compounds is relatively weak or inconclusive, but insoluble dusts of nickel oxides, and soluble aerosols of nickel sulfate, nitrate, and chloride, have been implicated as potential carcinogens.

Nickel is released into the air by power plants and trash incinerators. Nickel can also end up in surface water when it is a part of wastewater streams.The larger part of all nickel compounds that are released to the environment gets adsorbed to sediment or soil particles and become immobile. In acidic ground however, nickel is bound to become more mobile in the groundwater.

There is not much information available on the effects of nickel upon organisms other than humans. The nontarget aquatic organisms are vulnerable to the nickel and cause the effects on it. This brings the biodiversity in danger and affects the whole biota of the aquatic body.

Materials and Methods

The selected model of animals, the freshwater bivalves, *Lamellidens marginalis* were collected from the Dhondwadi dam at Borana River Tq. Parli Vijanath, Dist. Beed (M.S.), India. After collection, the bivalves were acclimatized in the laboratory condition at room temperature for 2-3 days. The active acclimatized bivalves of approximately same size were selected for experiment.

Before starting the experiment, these bivalves were divided into two groups one group of bivalves was maintained as control while the second group was exposed to the chronic dose of nickel chloride (0.227 ppm, equivalent to the $LC_{50}/10$) for 30 days.

The experimental and control bivalves were dissected after15 and 30 days. The gill and digestive glands were fixed in Bouie's fluid, for 24hrs. Washed and dehydrated in alcohol grades, cleared in toluene and embedded in paraffin wax (58-60°C).

Prepared blocks of tissues were cut at the thickness of 6µ and stained with Mallory's triple stain. Stained slides with serial sections were examined under light microscope for histopathological impact. Gills and digestive glands of bivalves from both groups i.e. control & exposed were screened and photomicrographs are presented in the plates I and II.

Observations and Results

The animals exposed to Nickel chloride and non treated *L.marginalis* showed histomorphological differences in their tissue architecture as fallows.

Histology of gills

Histologically, each gill or ctenidium consists of two gill plates or demibranchs. Each gill plate is formed of two similar flaps or lamellae. They are joined to form water tubes opening into the mantle cavity. Lamellae are formed of numerous gill filaments which contain holes called ostia. The gill filaments are covered by different kinds of cilia and supported by two chitinous rods. The space between two lamellae of a gill plate contains blood vessels.

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Photomicroplate 1a shows histology of gills of bivalve in control group.

Gills of bivalves exposed to chronic dose of nickel chloride for 15 and 30 days showed histopathological changes in their architecture as shown in photomicroplates 1 b and 1 c respectively.

Chronic exposure to nickel Chloride induced significant pathological changes in histology of gills. The epithelial cells and connective tissue cells have lost their cellular structure. Epithelium became oedematic, necrotic and vacuolated. There was fusion of secondary lamellae and decrease in space between water tubes and interlamellar junction due to hypertrophy. Nickel exposure caused severe damage to cilia, chitinous rods and epithelia. The epithelial lining at the tip of gill filaments were disintegrated. There was damage to normal structure of gills which resulted in atrophy of secondary gill lamellae. Cytoplasm showed disintegration due to swelling in respiratory epithelium. Cilia became clumped resulting the filterfeeding mechanism and circulation of water.The blood vessels were ruptured causing hemorrhage. Some cells and nuclei were observed in abnormal shape.

Histology of digestive glands

Digestive glands also called hepatopancreas consists of numerous hepatic lobules. Photomicroplate 2 a shows histology of hepatopancreas of bivalves in control *L. marginalis*. Each lobule is lined by columnar epithelial cells and secretory cells resting on basement membrane. In the core of hepatic lobule there is a narrow lumen. Interlobular space is filled by thin layer of connective tissue. It is the storage house of metabolic reserve which is the source of energy during physiological stress. Its secretion also plays a vital role in digestion of food.

Hepatopancreas of bivalves exposed to chronic dose nickel for 15 and 30 days showed histopathological changes in their structure as observed in the photomicroplate 2 b and 2c respectively. Epithelial cells of the hepatic lobules were separated from the basement membrane and scattered in the lobules. There was increase in the size of lumen in the core of lobule. Necrosis and degeneration of the cells was observed. Connective tissue became very thin. The overall damage of the hepatic follicles was observed.

Discussion

Gulbhile (2006) studied mercuric chloride exposure on *Lamellidens Corrianus*, the lamellae of gill showed various changes such as repture of the ciliated epithelium, increase in the size of lamellae, increase in space between the inter lamellar junction and increase in space between the water tube and inner lamellar junctions. Normal structure of gills totally damaged or disturbed due to mercuric chloride showing fusion and atrophy of secondary gill lamellae, displacement and necrosis of outer layer of gill lamellar epithelium. After 24 to 96 hrs of exposure to mercuric chloride the change in cytoarcitecture of gill *Lamellidens Corrianus* were more severe as compared to those bivalve exposed to mercuric chloride.

In bivalves and gastropods, the digestive gland is the major site of heavy metal storage (Simkiss and Mason, 1983). The heavy metals are able to bioconcentrate in the animal tissues. Light microscopic structure of the digestive gland reveals the toxic effect on acute and chronic exposure to lethal and sub lethal concentrations of metal contaminants at the cellular level. Main histopathological features can be manifested as increase in tubular lumen diameter, irregular shape of digestive and basophilic cells and presence of vacuoles and detachment of tubular teguments.

A plan metric method was applied to measure the epithelial thickness of the tubular section of the marine gastropod *Littorina littorea* using a schematic diagram made by a drawing tube. Geometrical transformation of that shape into a hypothetical trapezium can assess the determination of the high index of the digestive tubule for both control and heavy metal exposed molluscs. Tubular atrophy can be easily distinguished from thinning of epithelial cells that resulted from environmental stress.

Mahajan (2005) found changes in the cytoarchitecture of hepatic lobules and their disruption on exposure to arsenic trioxide, lead nitrate and mercuric chloride in hepatopancreas of snail, *Bellamya bengalensis*. Suresh (2001) observed disorganized condition of hepatopancreas in *U. annulipes* in response to cadmium and mercury.

Bhavan and Geraldine (2000) observed extensive vacuolation in the cells of hepatopancreas in *U. triangularis* on exposure to urea and naphthalene. Mule (1990) studied the fluoride-induced changes in the hepatopancreas of fresh water mussel, *Indonaia caeruleus* and observed severe damage to the hepatopancreas and severity of effect was dose dependent. The tubules were disfigured and lumen size was enlarged, disorganization and extensive vacuolation in the cytoplasm of cells were observed.

Gulbhile (2006) observed that acute exposure to sodium arsenate and mercuric chloride induced severe changes in the digestive gland of freshwater bivalve, *Lamellidens corrianus*. The hepatic lobules of hepatopancreas exposed to sodium arsenate and mercuric chloride showed increased size of lumen in cells, space between epithelium and its basement membrane and the damaged epithelia.

Jawale (2006) studied hepatopancreas of bivalve, *Lamellidens corrianus* after chronic exposure to lead and zinc, the marked histopathological changes induced exhibited an initial reaction of epithelial damage, together with necrotic changes in basement membrane and intertubular connective tissue. She observed the epithelial necrosis, rupture of epithelial layer and sloughing of the epithelium after chronic exposure of lead and zinc.

In present investigation study the results of chronic exposure to nickel Chloride for 15 and 30 days duration induced significant pathological changes in histology of gills and Hepatopancres which alter the normal tissue architectures. These losses in normal tissue architecture lead to loss of normal physiological function of animals.

Fig 1a. Section of control gill, Fig 1b section of gill after exposure for 15 days to Nickel chloride (0.227ppm) and Fig. 1 c section of gill after exposure for 30 days to Nickel chloride (0.227ppm)

Fig-1 a

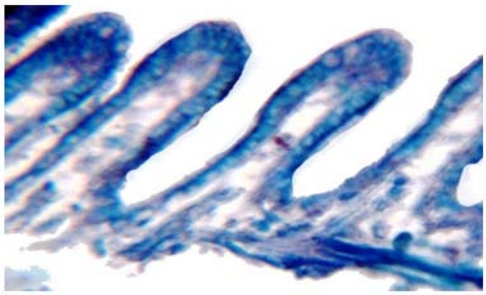


Fig. 1b

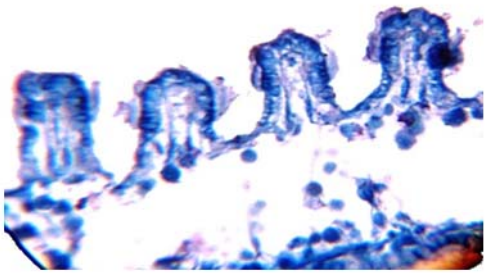


Fig.1 c

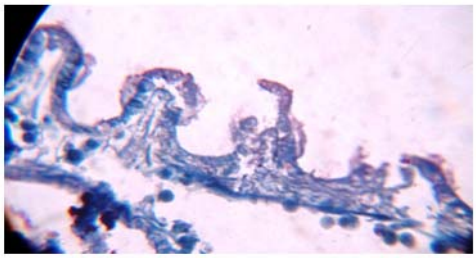


Fig 2 a. Section of control digestive glands, Fig 2b section of digestive glands after exposure for 15 days to Nickel chloride (0.227ppm) and Fig. 2 c section of digestive glands after exposure for 30 days to Nickel chloride (0.227ppm)

Fig. 2 a

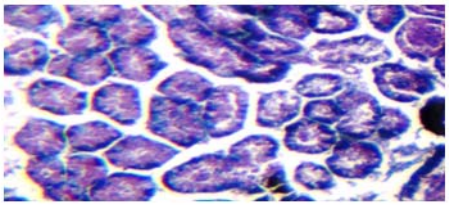


Fig. 2 b

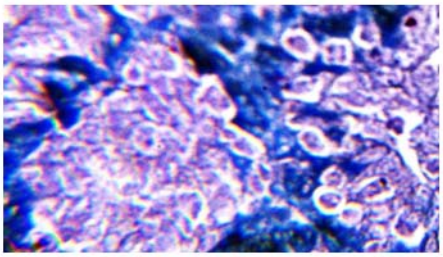
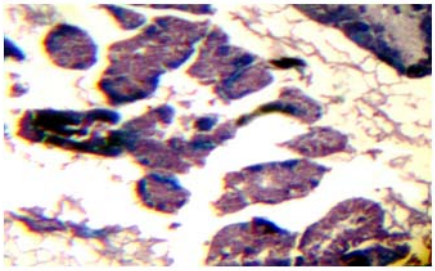


Fig. 2 c



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