

Histopathological Changes in Hepatopancreas of Freshwater Prawn, *Macrobrachium Kistnensis* Exposed to TBTCL

¹P.S. Kharat, ²T. S. Pathan and ³K.B. Shejule

¹Nutan Mahavidyalaya, Selu, Dist. Parbhani (M.S.) India

²Kalikadevi Arts, Commerce and Science College, Shirur Kasar Dist. Beed (M.S.) India

³Dr. Babasaheb Ambedkar Marathwada University, Aurangabad (M.S.) India

Abstract: We investigated the toxicity of TBTCL and its effects on Hepatopancreas of freshwater prawn, *Macrobrachium kistnensis* at different concentrations in present study. After exposure of different concentrations as 0.33 ppm, 0.26 ppm, 0.17 ppm and 0.09 ppm LC50 values for 24 h, 48 h, 72 h and 96 h respectively of tributyltin chloride, we found that the size of lumen was reduced after exposure of 0.26 ppm and Epithelial lining covering of the tubules were found ruptured and size of the lumen increased with finger like projection after 96 h exposure for 0.09 ppm. More number of tubules found to be ruptured

Key words: TBTCL • Histopathology • Hepatopancreas • *Macrobrachium kistnensis*

INTRODUCTION

A study of pathological change in the microanatomy of tissues is known as histopathology. Since the mid nineteenth century this branch of science has been successfully employed as a diagnostic tool in medical and veterinary science. Some environmental scientists are beginning to correlate the degree of cell damage to concentrations of the toxic substance and their synergistic or antagonistic interaction, [1, 2]. The histopathological studies not only give an early indication of pollutants hazards but also provide useful data on nature and degree of damage to cells and tissues. The histological techniques are promising area of research in aquatic toxicology as it gives the real picture of effects imposed and the involvement of chemical pollutants in the either disturbing or destroying the vital organs of living organisms. Histological analysis appears to be a very sensitive parameter and is crucial in determining cellular changes that may occur in target organs, such as the gills, liver and gonads, Dutta [3]. Histological responses may also serve as ecotoxicologically meaningful biomarkers since they form an important link between effects at the biochemical level and those measured in whole organisms, [4, 5]. Many workers have reported the degenerative changes in selected tissue of animals in response to

pollution by various toxicants, [6, 7]. Histological changes associated with pesticides in fish have been studied by many authors, [8, 9, 10].

The toxicity of organotin compound varies considerably according to the number and nature of organic groups with tin and tetra dialkyltin being the most toxic form. TBT compounds are already being used in variety of paint formulation either alone or as an active agent or in combination with metal ion compounds Evans, [11, 12]. TBT compounds are extremely effective and relatively economical as biocides, contributing to rapid uptake of organotin based paints by shipping industry and small boat owners in 1970's. These compound causes variety of effects in aquatic environment, [13]. They interfere and interact with various physiological activities of the organism like reproduction, which is an important biological phenomenon dominating all other physiological processes and are a need to the animal for continuity of their races, [14]. The organs like gills, ovaries and hepatopancreas are known to be the sensitive indicator of physiological disturbances. In crustacea the hepatopancreas was considered as purely digestive gland, beside this hepatopancreas act as a center of intermediary metabolism and an important storage depot like insect fat body and vertebrate liver and adipose tissue. The cellular diversity in hepatopancreatic tubules

of crustacea has been known for some years, [15]. Liver has the ability to degrade toxic compounds, but its regulating mechanisms can be overwhelmed by elevated concentrations of harmful substances and this could subsequently result in structural damage. Similar studies on various fish species, exposed to various toxicants, showed histopathological changes in the livers of those specimens, [16]. Nagbhushnam and Kulkarni [17] showed effects of cadmium chloride on freshwater prawn, *Macrobrachium kistnensis* showed many histopathological changes in hepatopancreas such as vacuolization of cell, winding of tubular lumen which ultimately resulted into syncytial mass containing large number of vacuolated cells and phagocytes. Bhodake [18] stated that organopesticides affect on the hepatopancreas of freshwater crab, *Barytelphusa cunicularis*. Doughtie and Rangrao [19] studied the histopathological changes in the hepatopancreas of grass shrimp exposed to chromium. Gangshettiwar [20] also found histopathological lesion in hepatopancreas of freshwater prawn, *Macrobrachium lamerrii* treated with phenol. TBT affects the *Oyster gigas* was first noticed by researchers [21]. Bruno and Ellis [22] observed histopathological changes in different tissues of Atlantic salmon, *Salmo salar L.*, attributed to use of tributyltin antifoulant. Bodar *et al.* [23] investigated cytological changes of digestive tract and storage cells in *Daphnia magna* exposed to cadmium and tributyltin. Dode [24] reported morbid changes in ovary and hepatopancreas in freshwater prawn, *Macrobrachium kistnensis* due to impact of organotin mellatoids copper.

Very few literatures were available on the impact of TBT compounds on histopathological aspects in crustacean. Hence the present investigation was under taken to find out histopathological changes in hepatopancreas in freshwater prawn, *Macrobrachium kistnensis* exposed to TBTCI.

MATERIALS AND METHODS

The fresh water prawns *Macrobrachium kistnensis* were collected from Kham river near Aurangabad, Maharashtra. The prawns were maintained in plastic trough containing aerated tap water. They were acclimatized for a week in laboratory condition. The water was changed every 24 h. Prawns were fed with green algae at alternative days. 1ppm stock solution of TBTCI was prepared in acetone Laughlin *et al.* [25]. Matured

healthy female prawns were selected for experiment. For each experiment 20 animals of approximately similar size (2.5 ± 1 cm in length) were exposed to 0.26 ppm and 0.09 ppm LC50 values of TBTCI at 24 h, 48 h, 72 h and 96 h respectively. Simultaneously group of 20 female prawns were also set up for the experimental control period in non-contaminated medium. Tissues such as gill, ovary and hepatopancreas were dissected out from control and experimental prawns and then fixed in Bouins fluid. Respective tissues were processed for microtechnique routine procedure.

RESULTS

Control Hepatopancreas: The tubules of hepatopancreas were enclosed by basal lamina and contained a central lumen. The hepatopancreas of *Macrobrachium kistnensis* consist of innumerable oval shaped tubules bound by loose connective tissue enclosing a large central activity of lumen. The tubules were composed of collemnar epithelial cells. Two types of cells were observed in each tubule beneath the epidermal layer, the first were columnar type of cell with nucleus towards base. i.e. absorptive cells. The second type of cells was larger in size having large globular mass of cells discharge their secretion into lumen of tubules i. e. secretory cell (Fig. 1).

Experimental Hepatopancreas: The hepatopancreas showed different changes in their architecture after exposure of different lethal concentrations of TBTCI for 0.33 ppm, 0.26 ppm, 0.17 ppm and 0.09 ppm LC50 values at 24 h, 48 h, 72 h and 96 h respectively. The 0.26 ppm of TBTCI treated prawns showed increased in absorptive cells and the lumen was filled with secretory fluid. After exposure of of TBTCI, the size of lumen was reduced (Fig. 2). Very few secretory globules were noticed in hepatopancreas prawns. Epithelial lining covering of the tubules were found ruptured and size of the lumen increased with finger like projection after 96 h exposure for 0.09 ppm more number of tubules found to be ruptured. The tubular absorptive cells were observed with pycnotic nuclei (Fig. 3).

DISCUSSION

Industrialization is linked to the economic growth and human progress. It is emphasized that increased industrialization lead to deterioration of environmental

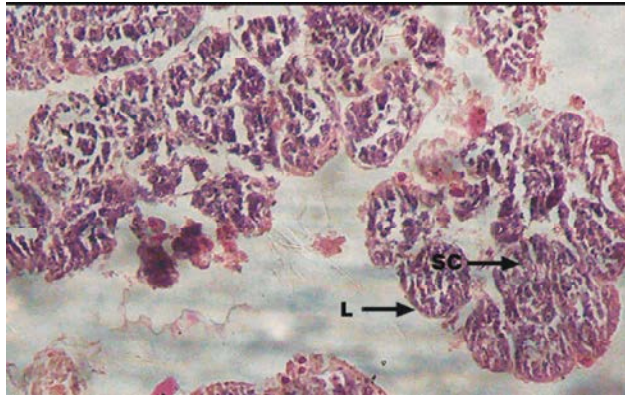


Fig. 1: (100x). Transverse section of the hepatopancreas of control prawn, *Macrobrachium kistnensis* showing Secretary cells (SC) and Lumen (L)

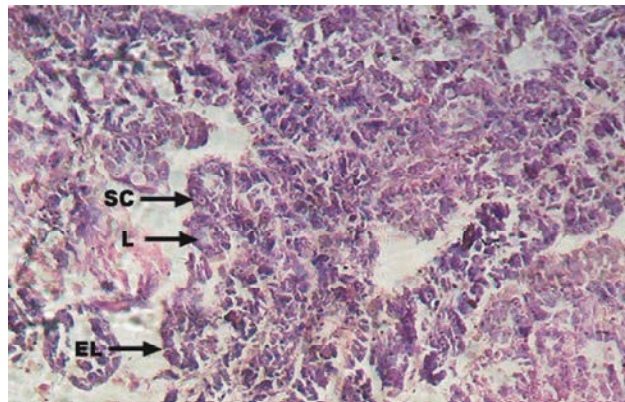


Fig. 2: (100x) After exposure lethal concentration at 0.26 ppm (LC of 48 hrs) of TBTCI Hepatopancreas showing Secretary cells (SC), Lumen (L) and Epithelial lining (EL)

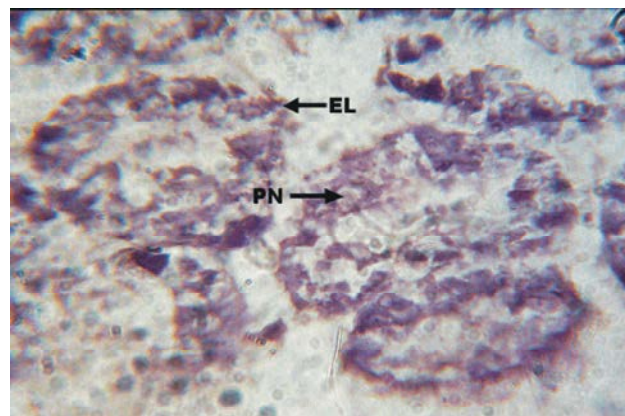


Fig. 3: (150x) After exposure lethal concentration at 0.09 ppm (LC of 48 hrs) of TBTCI Hepatopancreas showing Picnotic Nuclei (PN) and Epithelial lining (EL)

quality and subsequently to health hazardous of organisms. The rapid unplanned industrial progresses have added to the problem of pollution. Organotin is noted as one of the significant contributor to water pollution. The organotin pollution in the environment has risen to alarming proportion which has caused great

concern among the environmental toxicologist as the organotin residues that are released as non-biodegradable substances.

In the present investigation the freshwater prawn, *Macrobrachium kistnensis* was exposed to different concentration of TBTCI. In *Macrobrachium kistnensis*

hepatopancreas consist of innumerable oval shaped tubules bound by loose connective tissue enclosing a large central activity of lumen. The tubules were composed of columnar epithelial cells. Two types of cells were observed in each tubule beneath the epidermal layer. Columnar types of cell with nucleus towards base were called absorptive cells. Other type cells were larger in size having large globular mass of cells discharge their secretion into lumen of tubules called secretary cells (fig 3). Hepatopancreas of crustacea is an important digestive gland like liver in fishes and plays an important role in intermediate metabolism. In the present study prawns exposed to 72 h and 96 h at 0.17 ppm and 0.09 ppm exhibited various changes in the hepatopancreas. It was found that damage caused by TBTCI at these concentrations was more pronounced when compared with that of 24 h and 48 h exposure. The damage to the hepatopancreas showed, the tubules were almost in state of collapsing in most of the TBTCI treated prawns. The tubules were distinguished and lumen size was enlarged, disorganization and extensive vacuolization in the cytoplasm of cells were observed. Indira [26] reported vacuolization of cell, winding tubular lumen and phagocytic cell in freshwater prawn, *Caridina weberi* when exposed to TBTO and copper sulphate. The histological changes indicates that the animal were not in position to digest and store food properly because the damage of secretory and absorptive cells by TBTCI toxicity, can not produced secretory material which mainly contains digestive enzymes and hence no absorption of simple food by absorptive cell to store. The lack of nutrients also resulted in atrophy of hepatopancreas. Similar results were reported by many researchers [27, 28, 29].

CONCLUSION

The above histopathological changes showed that the severity of damage is dose and time dependent. Many others have reported pathological changes in different tissues of various organism induced by lethal effect of different pollutants. In conclusion we can say that the changes in the architecture of the hepatopancreas is due to the TBTCI toxicity and it should be control to save the aquatic biota.

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