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HISTOLOGICAL VARIATIONS IN FRESHWATER TELEOST, *TILAPIA MOSSAMBICA* (PETERS) ON EXPOSURE TO ARSENIC

A. Shobha Rani and T. Naga Raju
Department of Zoology, Osmania University, Hyderabad-500 007, Andhra Pradesh, India

ABSTRACT

The sublethal toxicity of sodium arsenite on histopathology of tissues such as liver, kidney and gill was investigated in freshwater teleost fish, *Tilapia mossambica*, exposed to 1/3rd of sublethal concentration (10ppm) of the chemical for a period of 4 and 14 days. Though, acute exposure has not resulted in death, it led to many morphological and histological variations such as histolysis, lesions, necrosis and vacuolation etc. There are many degenerative changes in the tissues studied suggestive of impairment of normal function when compared to controls.

INTRODUCTION

Arsenic is mainly released into the environment through industrial processes during the preparation of base metals and thermal power generation besides arsenic based compounds, which are extensively used such as pesticides, insecticides, herbicides and fungicides (IARC 1988, Webb 1966). Arsenic is one of the major contaminants of marine and freshwaters and it is accumulated by organisms inhabiting these waters. Histological assessment proved the toxic nature of chemicals with tissue alterations on subsequent exposure. Several studies involving arsenic have demonstrated various tissue alterations with attendant physiological impairment (Oladimeji et al. 1994, Sakurai et al. 1998, Sorensen et al. 1979).

The arsenic level in food with the exception of fish is usually below 1 mg/kg (Leonard & Lauwery 1980). The source of arsenite toxicity to human beings originates through the food chains, especially by consumption of fish living in arsenic contaminated waters. In this investigation an attempt has been made to study the histopathology of select tissues such as liver, kidney and gill of *Tilapia mossambica*, exposed to arsenic.

MATERIALS AND METHODS

The fish were obtained from the Department of Fisheries, Hyderabad, Andhra Pradesh. The fish having 16 ± 2 cm in length and 50 ± 5 g in weight were selected for the experiment. After the process of acclimatization and washing with 0.1% $KMnO_4$ solution, fish were exposed to 1/3rd of sublethal concentration (10ppm) of sodium arsenite for a period of 4 and 14 days. A parallel set without the chemical was maintained as control. Both, experimental and control fish, were sacrificed and tissues such as liver, kidney and gills were used for histopathological preparations. The slides were prepared according to standard histological procedure. The slides were studied under microscope to observe histopathological details of liver, kidney and gill.

RESULTS

In the present study several histological changes were observed as a result of arsenic toxicity. The damage in major organs such as liver, kidney and gill has been described using microphotographs

(Figs. 1-9). The changes observed in this investigation were time dependent after exposure to sodium arsenite. The details are as follows:

Liver: The liver of control fish is characterized by continuous mass of hepatic cells. These cells are polygonal, large, each with a centrally placed rounded nucleus and homogenous cytoplasm (Fig. 1). In the present investigation on exposure to arsenic for 4 and 14 days, several changes were observed in the liver of *Tilapia mossambica*. On exposure to arsenic for 4 days (Fig. 2), moderate changes occurred in the shape of the hepatocytes and this may be probably due to of histolysis and lesions. The cell wall of certain hepatocytes became irregular and hepatocytes became vacuolar. On exposure for 14 days (Fig. 3), several areas of vacuolation of cytoplasm of hepatocytes were visible in the majority of the hepatic cells. Cloudy appearance of hepatic cells was observed. Position of nucleus slightly changed in few hepatocytes. Necrotic bodies were found in various locations in hepatocytes and between hepatocytes.

Kidney: Normal kidney showed coiled uriniferous tubules, glomeruli with rounded and prominent nuclei in the centre and Bowman's capsule (Fig. 4). Epithelial cells are clearly visible. Edema is the first sign of any renal manifestation as seen in the interstitium in the present case. In the exposure to arsenic for 4 days (Fig. 5), shrinkage of glomeruli and tubular degeneration took place. On exposure to 14 days, there was destruction and compression in the uriniferous tubules (Fig. 6). The nuclei of tubule cells were slightly displaced towards the periphery. Cloudy appearance and hydrophic degeneration of interstitial tissues were observed.

Gill: Normal gill (Fig. 7) showed primary gill lamellae with gill arch and gill rakers. Secondary gill lamellae are arranged in series. The results of the present investigation revealed striking changes in the gill structure as a consequence of arsenic toxicity. On exposure to arsenic for 4 days there was a swelling in the epithelial cells of secondary gill lamellae (Fig. 8). In certain areas, primary gill lamellae were completely disorganised. On exposure for 14 days, shrinkage in secondary gill lamellae and cellular damage of interbranchial space were observed (Fig. 9). Disintegration of gill epithelium, lymphocyte infiltration and loss of few lamellae were observed. The tip of the secondary gill lamellae with blood vessels became rounded swollen mass with more red blood cells inside (hematomass).

DISCUSSION

The histological changes that have occurred in the tissues studied in this investigation reflect the toxicity of arsenite on fish, which indicates a clear-cut derangement of normal function of these tissues, and this is in agreement with the reports of many workers in different animals (Hughes & Thompson 1996, Sorensen et al. 1979).

Liver is the vital organ for detoxification and metabolizes various compounds, which are eliminated through various routes. However, liver is susceptible to a number of chemical substances and undergoes cellular damage. Liver binds arsenic avidly and is generally considered as one of the major target organs in case of arsenic poisoning (Gilderhus 1966, Sarin et al. 1999). Histological changes range from fatty infiltration, central necrosis, cirrhosis and vacuolation of the cytoplasm of hepatic cells (Gilderhus 1966). Many nuclei were displaced (Sorensen et al. 1980). Small spaces in between the hepatic cords, change in the position of the nucleus, damage of connective tissue of liver after exposure to endosulfan and carbaryl on fish was reported (Sudha & Neeraja 1998). In the present study, the liver showed a distinct and disorganised appearance due to the presence of necrotic bodies, lesions, vacuoles in the hepatocytes.

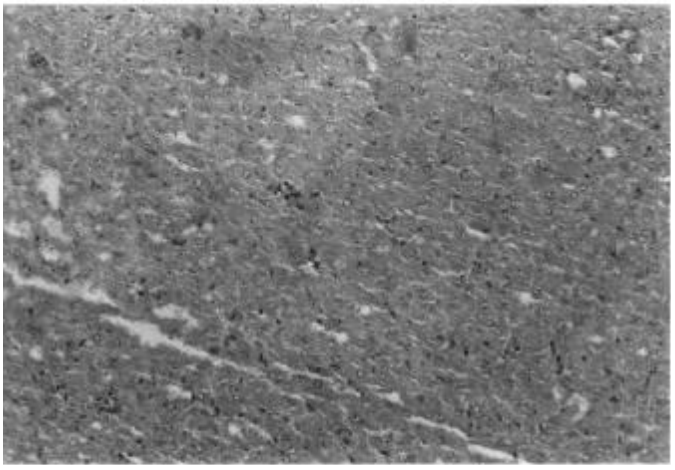

Fig. 1: Liver (Control): Radially arranged hepatocytes, clearly visible nuclear membrane and nuclei of hepatocytes.

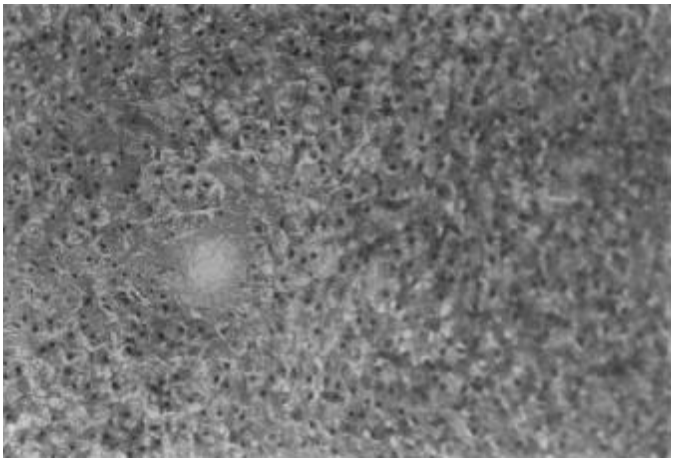

Fig. 2: Liver (Sublethal arsenic exposure for 4 days): Irregular cell walls of certain hepatocytes and vacuolar hepatocytes.

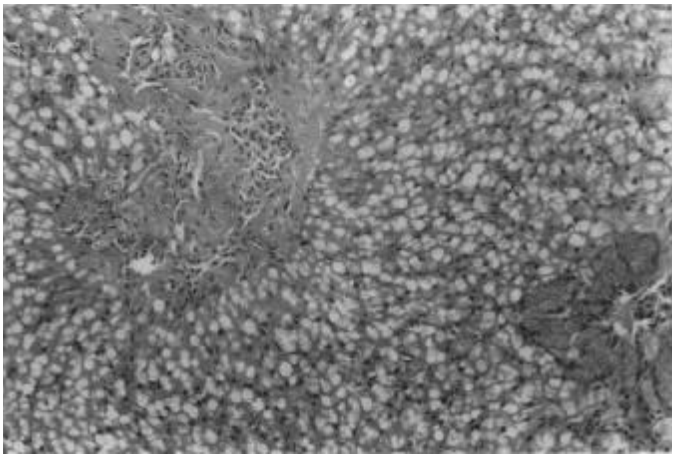

Fig. 3: Liver (Sublethal arsenic exposure for 14 days): Several areas of necrosis and vacuolation of the cytoplasm of hepatic cells.

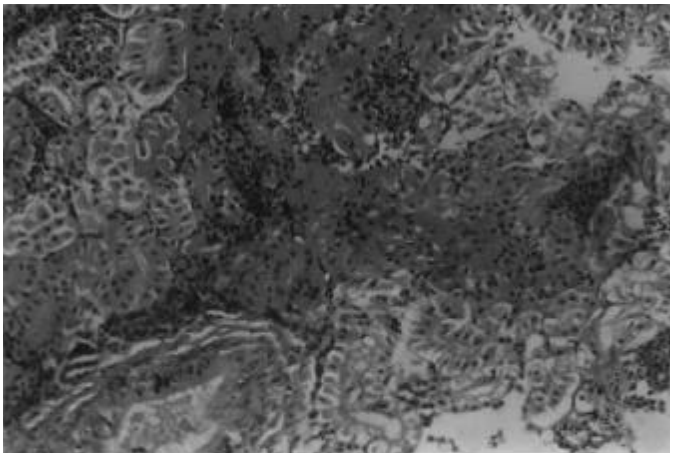

Fig. 4: Kidney (Control): Clearly visible uriniferous tubules and Bowman's capsules.

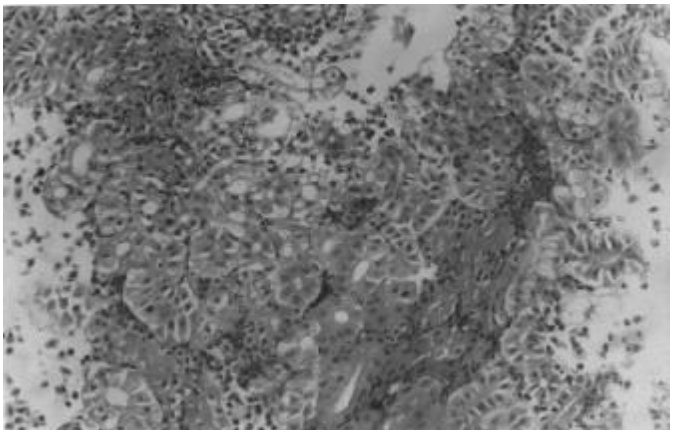

Fig. 5: Kidney (Sublethal arsenic exposure for 4 days): Shrinkage of glomeruli and tubular degeneration.

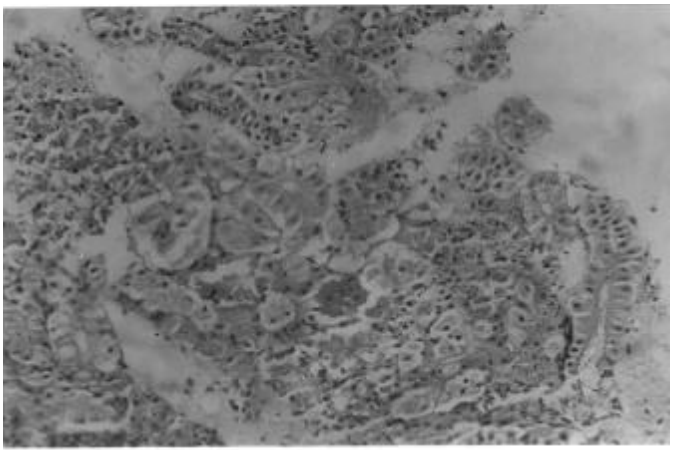

Fig. 6: Kidney (Sublethal arsenic exposure for 14 days): Compression of uriniferous tubules, cloudy appearance and swelling of interstitial tissues.

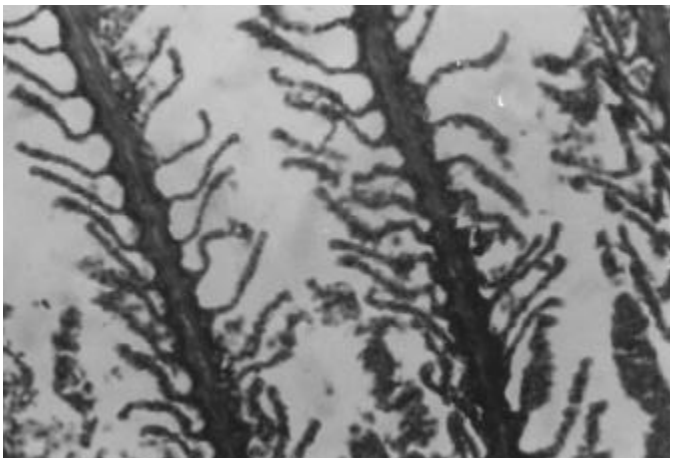

Fig. 7: Gill (Control): Secondary gill lamellae are arranged in series.

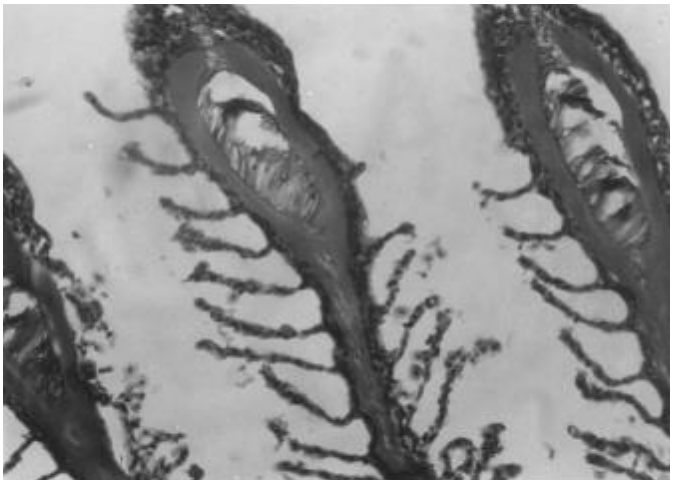

Fig. 8: Gill (Sublethal arsenic exposure for 4 days): Swelling in the epithelial cells of secondary gill lamellae clearly visible.

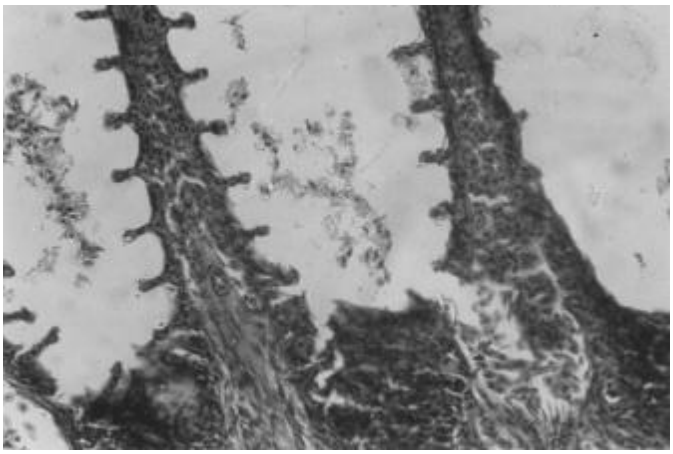

Fig. 9: Gill (Sublethal arsenic exposure for 14 days): Shrinkage and damage of secondary gill lamellae.

The kidney also accumulates arsenic and is considered to play a major role in the metabolism and excretion of arsenic (Sorensen et al. 1979). On exposure to arsenic, swollen mitochondria and increased numbers of autophagic lysosome-like bodies have been reported in rats (Brown et al. 1976) and in green sunfish (Sorensen et al. 1979). Edema and loss of interstitial tissue of kidney of *Labeo rohita* induced by tannery and textile dyeing effluents were reported (Rana & Raizada 2000). Necrosis and vacuolation of glomerular cells and necrosis of blood vessels were reported in *Tilapia mossambica* on exposure to sodium fluoride (Muley et al. 1996).

The sensitive nature of gill tissue and its respiratory surface coming into direct contact with toxicants present in the aquatic environment, renders it as the first organ to be affected by the toxicants among the other organs. Swelling of gill filaments and lamellae, reduction in interlamellar spaces and cyst formation were reported in fish exposed to cadmium (Kapila & Ragothamam 1999). It was reported that thinning of interlamellar epithelium, destruction in the arrangement of pillar cells and red blood cells, formation of hematomass within the secondary lamellae of gill tissue of *Labeo rohita* exposed to mercury (Jagadeesan 1999).

The present observations are in accordance with findings of various other authors cited above. The results of the present investigation revealed histopathological changes as a consequence of arsenic exposure. Fish exposed to sublethal concentration of arsenic for a period of 4 days and 14 days showed degenerative changes in select tissues such as liver, kidney and gill that are suggestive of dysfunction of these important tissues which may lead to an altered physiology of the whole animal.

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