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Histopathological studies of *Anabas testudineus* Bloch 1792 on exposure to aquatic toxicants of Buckingham canal, Chennai, Tamil Nadu, India

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Abstract
Heavy metal contamination have devastating effects on the ecological balance of the recipient environment and a diversity of aquatic organisms. Among animal species, fishes are the inhabitants that cannot escape from the detrimental effect of these pollutants. Fish are widely used to evaluate the health of aquatic ecosystems because pollutants build up in the food chain and are responsible for adverse effects and death in this ecosystem. Pollutants induce mortality, genotoxicity and histopathology thus impairing respiration, metabolism and enzyme activities in affected fishes. Therefore, in the present work the histopathological alterations caused to *Anabas testudineus* on exposure to aquatic toxicants present in the Buckingham canal, Chennai, Tamil Nadu, India were studied.

Keywords: *Anabas testudineus*, histopathology, gill, muscle, liver, intestine, kidney

1. Introduction
Buckingham canal water contains different kinds of dissolved and particulate matters including gases and solids. The chemical nature of water affects phytoplankton, zooplankton and other aquatic animals, fishes being the major among them. Fish forms a rich source of food and an important element in economy of many nations but the present scenario is that the fish diversity and its abundance in such aquatic systems are declining due to environmental pollution [1]. Heavy metals are recognized as one of the most hazardous environmental pollutants and are toxic to many living organisms. Environmental exposure to heavy metals has been reported to cause disease in human and other mammals [2]. In recent years, there has been a growing interest about the effects of heavy metals on the health of fish. This aspect is of great importance with development of fisheries, which are often located in rivers, ponds or estuaries subjected to industrialization or mining practices [3]. The natural aquatic systems may extensively be contaminated with heavy metals released from domestic, industrial and other man-made activities [4,5]. Heavy metal contamination may have devastating effects on the ecological balance of the recipient environment and a diversity of aquatic organisms [6-8]. Among animal species, fishes are the inhabitants that cannot escape from the detrimental effect of these pollutants [9]. Fish are widely used to evaluate the health of aquatic ecosystems because pollutants build up in the food chain and are responsible for adverse effects and death in the aquatic systems [10, 11]. Studies carried out on various fishes have shown that heavy metals may alter the physiological activities and biochemical parameters both in the tissues and in blood [12-14]. Environmental pollutants indicate that the sub lethal doses of most of the pesticides cause varying extent of histopathological alterations to different organs in fishes; the amount of damages are usually dependent on dose, duration of exposure and type of pesticide [15,16]. It is reported that most of the pollutants induce mortality, genotoxicity and histopathology thus impairing respiration, metabolism and enzyme activities in affected fishes [17]. Therefore, in the present study the histopathological alterations caused to *Anabas testudineus* on exposure to aquatic toxicants present in the Buckingham canal, Chennai, Tamil Nadu, India were studied.

2. Materials and methods
2.1 Study area
Chennai (Madras) the capital of Tamil Nadu is situated on the eastern coast of India, on 13 0 4' north latitude and 8 0 15' east longitude. There are three water ways that flows through the city, *viz*., Cooum river, Adayar river and Buckingham canal. The Buckingham canal is a man-made water canal linking the abovementioned two rivers. The portion north of the Cooum is known as the north Buckingham canal, and the portion south of the Cooum as the south Buckingham canal.

The canal extends from Nellore in Andhra Pradesh to Marakkanam near Puducherry. The length of this canal in Andhra Pradesh is 257km, and 163km is in Tamil Nadu. Approximately, 31km is within the city limits of Chennai (Figure 1). Within the city of Chennai the canal is badly polluted from sewage and industrial effluents, and the silting up of the canal has left the water stagnant, creating an attractive habitat for mosquitoes. The water quality is considered to be highly toxic and completely non-potable. The three waterways are severely polluted in Chennai city, particularly the Buckingham canal by sewage, industrial wastes, storm water drainage and garbage, as a result of haphazard urbanization.

2.2 Test organism

Taxonomic position:

Kingdom	: Animalia
Phylum	: Chordata
Sub Phylum	: Vertebrata
Class	: Pisces
Order	: Anabantifromes
Family	: Anabantidae
Genus	: *Anabas*
Species	: *testudineus*

Anabas testudineus commonly called climbing perch is an extremely hardy, small, brown or dark greenish-brown fish (Figure 2A), native to Southeast Asia. It is highly adapted to life in a seasonal tropical environment. It can tolerate very turbid and brackish water conditions; possesses an accessory air-breathing organ that enables it to survive out of water for several days; and uses its highly mobile sub operculum and strong fin spines to pull itself over land to move between bodies of water. The fish has the ability to aestivate during the dry season. Under extreme circumstances it is even able to aestivate for several weeks by burying itself into moist ground [18]. It inhabits the majority of drainage systems across its native range and has been recorded in many different habitat-types including swamps, marshes, lakes, canals, pools, small pits, rice paddies, puddles, tributaries and main river channels. Though primarily a lowland freshwater species, it also occurs in brackish coastal environments in some areas.

2.3 Histopathology

The histopathological observations were made from gill, muscle, liver, intestine and kidney of the control and experimental groups exposed to aquatic pollutants. Both control and experimental fish were sacrificed and their tissues (gill, muscle, liver, intestine and kidney) were fixed in 10% neutral buffered formalin. The fixed tissues were dehydrated in an increasing gradient of alcohol (70, 80, 90 and 100%) for thirty minutes each and were eventually dried in acetone and cleared in xylene for thirty minutes [19]. The tissues were then infiltrated by embedding in molten wax and sectioned at 8µ. The paraffin sections were then mounted on a slide, stained with haematoxylin and counterstained with eosin. To study the pathogenicity of aquatic pollutants, the section of tissues were observed under a microscope and were photographed at low and high power of magnification using Nikon micro photographic equipment.

3. Results

3.1 Gill

The gill arches of *Anabas testudineus* from the control group showed normal arrangement pattern of primary and secondary lamellae. Projecting on the lateral sides of primary lamellae are the secondary lamellae. The surface of the secondary lamellae was covered with a delicate layer of a simple squamous epithelium, the active exchange pillar cells. In the core of the primary lamellae was a rigid mass of cartilaginous tissues around which were traces of vascular channels (Figure 3A & B). The chloride cells were more frequent at the base of the secondary lamellae. Varied morphological changes occurred in the gill tissue of the affected *Anabas testudineus* and the gill exhibited marked alterations in their epithelia. The epithelium was no longer continuous, particularly the more delicate respiratory lamellae. Thus, there was fusion at adjacent secondary lamellae as a result of hyperplasia. Oedema at the secondary lamellae and swelling of the epithelia cells were observed. The pillar cells were altered and blood spaces expanded (Figure 3C & D). The elongated secondary lamellae with club-like structures were the other pathological changes observed in the histological sections of gills of the *Anabas testudineus* affected with aquatic pollutants of Buckingham canal.

3.2 Muscle

A transverse section of *Anabas testudineus* skeletal muscle showed bundles of muscle fibers. It consists of long multinucleated muscle fibres with peripheral oval nuclei, innervated by somatic nerves. Striated appearance of muscle was due to alternative arrangements of light and dark bands. The entire muscle was surrounded by dense connective sheath (epimysium) while the bundle of muscle fibre by the perimysium and individual muscle fibre surrounded by endomycium was present in the control fish (Figure 3E & F). Degeneration of muscle fibers and infiltration with mononuclear cells were observed in pollutant affected fish (Figure 3G & H). Some muscle fibers appeared abnormal with less of sarcoplasm and pyknotic nuclei and also showed variation in size and shape. Oedema between muscle bundles and splitting of muscle fibers was also observed in *Anabas testudineus* when compared to the normal architecture of muscle tissue of the control fish.

3.3 Liver

A section of the liver seen under the microscope revealed the general histology of the organ in the control group. It exhibited the typical parenchymatous appearance. Each tube was surrounded by a very thin connective tissue capsule which extended as a trabecular into the body of the lobes and dividing them into irregularly shaped lobules. The liver hepatocytes were polygonal shaped with central spherical nucleus. The cells were arranged as irregular cord-like structure in the section separated by sinusoids (Figure 3I & J). Histomorphology of liver specimens exposed to aquatic toxicants of the Buckingham canal showed diffuse changes in the hepatic parenchyma of *Anabas testudineus*. There were large vacuoles within the cytoplasm resulting from cell membrane degeneration. Many of the nuclei became pyknotic with gradual process of cytolysis. The cytoplasm was highly

vacuolated while the nuclei continued to be pyknotic. Focal necrosis was noticed in many regions with increase in cytolysis. Lesions were predominantly observed in liver of affected fish, odematous condition, vacuolization, marked degeneration and constriction of the sinusoids appeared in the parenchymatous tissue of the liver affected *Anabas testudineus*. Enlargement of hepatocytes in proportion to the increase in size of the nuclei, and increase in density of connective tissue with imminent congestion at the sinusoidal spaces were significant. There were large vacuoles within the cytoplasm resulting from cell membrane degeneration. Haemorrhages leading to extensive necrosis of hepatic cells were also noticed (Figure 3K & L).

3.4 Intestine

Histopathological examinations of the intestine of *Anabas testudineus*, revealed degenerative changes in the serosa, mucosa, longitudinal and circular muscle layers and submucosa. The columnar epithelial cells situated at the base and tips of the villi were degenerated and formed a syncytial mass. Mucous secreting goblet cells revealed an increase in their size, about to burst. The lumen of the intestine was filled with mucus plug (Figure 3M & N). The villi also displayed degeneration and necrosis at their tips when compared to control fish (Figure 3O & P).

3.5 Kidney

Lesions were observed in the histological studies of affected kidney of *Anabas testudineus*. Hypertrophy of renal cells, changes in the nuclear structure, formation of vacuoles, necrosis and degeneration of renal components were noticed. Hyperplasia was seen in the tubular epithelium (Figure 3Q & R). Parenchymatous cells revealed marked oedema and congestion of sinusoids. Renal tubules presented hypertrophy and reduced inter-tubular spaces. Tubular epithelium showed necrotic changes characterized by karyorrhexis and karyolysis at the nuclei of the affected cells. Lumen of the tubules was invariably dilated. Interstitium was markedly infiltrated with mononuclear cell when compared to the control fish (Figure 3S & T).

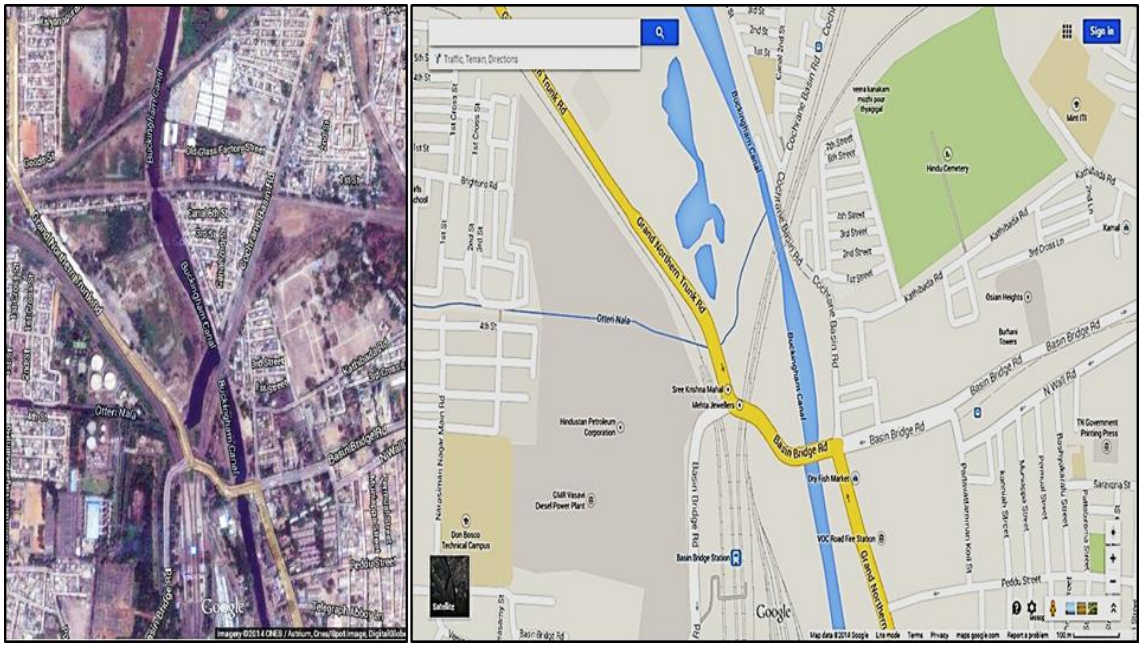

Fig 1: Study area – Buckingham canal

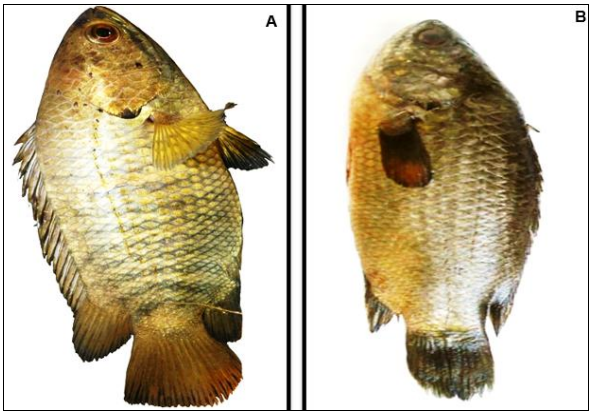

Fig 2: *Anabas testudineus*. A: control; and B: experimental

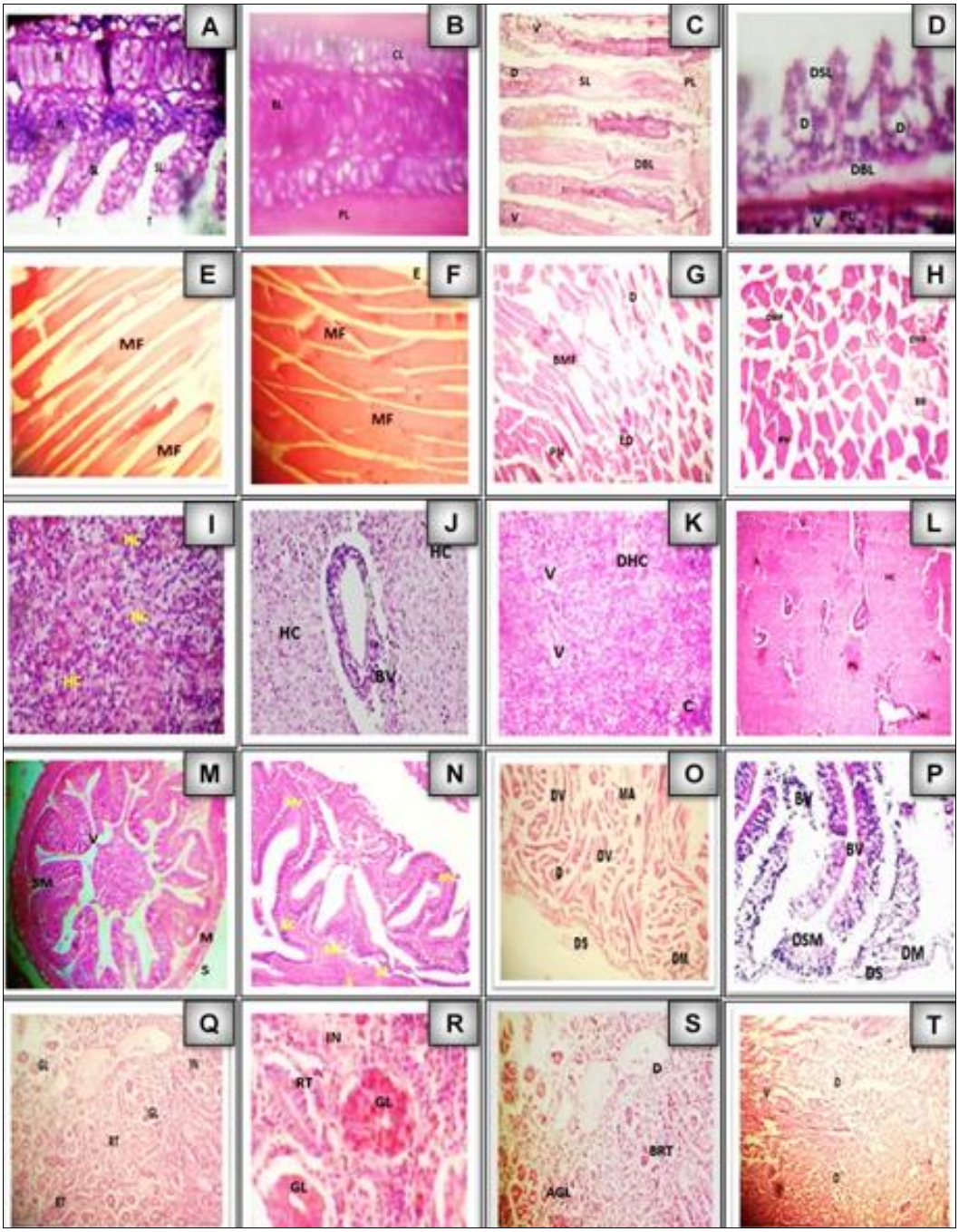

Fig 3: *Anabas testudineus*. A & B: Gill-control, C & D: Gill-treated. PL- Primary Lamellae, SL- Secondary Lamellae, BL-Blood Vessel, T- Tip of Secondary Lamellae, D-Degeneration, DBL-Degeneration of Blood Vessel, V-Vacuolation, DSL-Degeneration of Secondary Lamellae. E & F: Muscle-control, G & H: Muscle-treated. MF-Muscle Fibre, D-Degeneration, ED-Edema, PN-Pyknotic Nuclei, DMF-Degeneration Muscle Fibre. I & J: Liver-control, K & L: Liver-treated. HC-Hepatocytes, DHC-Degeneration of Hepatocytes, BV-Blood Vessel, V-Vacuolation, N-Necrosis, PN-Pyknotic Nuclei, DBL-Degeneration of Blood Vessel. M & N: Intestine-control, O & P: Intestine-treated. M-Mucosa, SM-Sub-Mucosa, S-Serosa, V-Villi, GC-Goblet Cell, NV-Necrosis of Villi, DV- Degeneration of Villi, MA-Mucus Accumulation. Q & R: Kidney-control, S & T: Kidney-treated: GL-Glomerulus, RT-Renal Tubule, IN-Interstitium, AGL-Affected Glomerulus, D-Degeneration, DRT-Degeneration of Renal Tubule, V-Vacuolation. (All magnifications at 10 X)

4. Discussion

Histology is a useful technique for investigating the toxic effect of various pollutants. Such a study also offers opportunity to locate the effect of pollutants in various organs and systems of animals. Histopathology provides a rapid method to detect effects of irritants in various organs. The exposure of fish to chemical contaminants is likely to induce a number of lesions in different organs [20]. Gill, intestine [21], kidney [22] and liver [23] are suitable organs for histological examination in order to determine the effect of pollution. This type of study in fish has been to a great extent but was handicapped due to lack of adequate histological literature concerning various fish organs [24]. Considerable interest has been shown in recent years in histopathological studies while

conducting sub-lethal tests in fish. Tissue changes in test organisms exposed to sub-lethal concentration of toxicant are a functional response of organisms which provides information on the nature of toxicant as well as the changes resulting in various biochemical, physiological, and histological alterations in vital tissues [25-27]. There is considerable information indicating that pesticides and heavy metals are responsible for many adverse effects in fishes and other animals from the histopathological and histochemical points of view [27-29].

In fish, gill is the first organ to which any pollutant comes into contact. Fish gill is very sensitive to changes in the composition of the environment and is an important indicator of water-borne toxicants. Consequently, injury to gill epithelium is a common response observed in fish exposed to a variety of contaminants. The severity of damage to the gills depends on the concentration of the toxicant and the period of exposure. *Anabas testudineus* affected with aquatic pollutants of Buckingham canal water sample showed an extensive damage to their gill architecture in the present study and this is in agreement with the earlier observations [27, 30, 31]. The gill is an important site for the entry of heavy metals that provokes lesions and gill damage [32]. The mechanism of toxicity of nickel is likely similar to that of other transitional metals *viz*., copper, cadmium, cobalt and lead and could be related to its activity in enhancing lipid peroxidation, a widely accepted general mechanism for cellular injury and death [33]. Many aquatic pollutants were assayed for their deleterious effects on the cellular ultra-structure of the gill epithelium [34, 35]. Wenkuan *et al*. [36] observed massive damage to gill architecture. Hughes *et al*. [37] found that among cadmium, chromium and nickel, only nickel had a concentration-dependent deleterious effect on the diffusing capacity of the gill and its ability to function in gas exchange. High contents of suspended solids present in the electroplating effluent might have caused the mortality, by blocking the gills of the experimental fish as reported by Kondal *et al*. [38]. The present study also revealed similar effect on the gills of *Anabas testudineus.*

Variations in the epithelial surface of gills showed important physiological adaptations relating to the area available for increased gaseous exchange [39]. Their vulnerability is thus considered because of their external location and for the fact that they are in intimate contact with water and are thus liable to damage by irritant materials. The lamellar reduction of the treated fish must have been caused by respiratory stress as the sum of all the physiological responses by which an animal tries to maintain or re-establish a normal metabolism in the face of a physical or chemical force. Haemorrhage and sloughing of the bronchial arteries at the opercular end of the primary lamellae can disrupt the circulation of the deoxygenated blood *via* the bronchial arteries into the secondary lamellae in a direction opposite to that of water flow. As a result, oxygen uptake is hampered which can cause asphyxiation, tissue necrosis and finally death. A number of pathological changes have been reported in fishes exposed to different heavy metals [40-45]. The changes include the bulging of tips of primary gill filaments and loss of secondary filament's original shape. The pillar cell nucleus showed necrosis and developed vacuoles in the secondary gill epithelium, with eventual fusion of secondary lamellae near the tips of gill filaments [46]. The biological function of the inflammatory response was to destroy or "wall off'' irritating substances so that damaged tissue may be healed. Eller [47] described induced histopathological changes in cut throat-trout and reviewed the gill lesions in freshwater teleosts. Sultana and Sharief [29] reported that these pathological changes in the gills might have resulted in such a shift from aerobic to anaerobic pathway in tissues of fish under exposure to toxicity. Tilak *et al.* [48] also reported on these lines in fishes exposed to different pesticides.

In the present study, it was observed that toxic exposure caused lamellar telangiectasis (clubbed appearance) along with oedema and mucoid metaplasia in *Anabas testudineus*. The clubbed appearance of lamellae is due to lamellar hyperplasia in which cells are derived from primary lamellae and migrate to the distal end. This result in accumulation of cells at the leading edge of secondary lamella, which is colloquially called 'clubbing' of lamellae [49]. Mucoid metaplasia was also very distinctly observed as the entire inter lamellar space seems to be filled up by such cells. Similar observations have also been reported on exposure to fenvalerate and EC 20% [44]. Cellular hyperplasia is an important feature in gills affected by pollution [23, 29]. The changes in gills were adaptations by the fish to cope with challenge of the toxicant. For example, atrophy or dystrophy, curving, clubbing and fusion of the secondary lamellae were attempts by the fish to reduce available surface area to the water sample. But this may result in the reduction of available surface for respiration and ionic exchange, consequently resulting in an internal hypoxic and toxic environment. Oedema recorded in the gills was due to failure of the sodium pump occasioned with the toxicant leading to accumulation of Na^+ and the diffusion of K^+ outside [50]. Vascular changes in the gills of exposed fish could be an attempt by the fish to supply more blood to the gills to increase oxygen uptake and supply to the internal organs. Further, the histological changes in the skeletal muscle of *Anabas testudineus* exhibited in the present study were degeneration and necrosis of muscle fibers together with infiltration of the mononuclear cells, thus, indicating reduction in the nutritive and economic value of the fish. Similar observations were made by Sultana [51] and Gayathri and Sultana [27] in their studies on *Oreochromis mossambicus.*

Liver being the main metabolic factory of the body serves several basic functions like metabolism, storage and secretion of bile. Liver accumulates more toxicants than other organs of body. Liver is the organ, which metabolizes the toxicants and excretes it out. Since metabolism of proteins, fats and carbohydrates and detoxification of endogenous waste products and drugs take place in liver, it is more liable to injury from toxicants. In liver sections of normal fish *Anabas testudineus*, the hepatocytes form a cord-like pattern, large in size, polygonal in shape with homogenous eosinophilic cytoplasm and centrally located nuclei. A large number of blood sinusoids separate the hepatic cords one from another. Exposure to toxicants induced obvious histopathological changes in the liver. The hepatocytes have lost their normal architecture and a large number of these cells appeared with pyknotic nuclei. The intra hepatic blood vessels were dilated

and congested with blood and inflammatory leucocytic infiltrations. Numerous hepatocytes showed marked cytoplasmic vacuolization [27]. In aquatic organisms, liver is greatly affected by pesticidal contaminants [47]. Binding of toxic substance with plasma proteins affects the excretion. Intracellular binding proteins are important in accumulating and storing toxicants within the liver. Metallothionein, a binding protein in liver and kidney binds metals. Considering the importance of liver, several studies were undertaken to explore the histopathological changes in liver and reported cellular level complexities and reasons for mortality [52-54]. Most of the histopathological lesions observed in the liver of *Anabas testudineus* in the present study were similar to those reported in earlier studies on other fishes with different toxicants. One of the important functions of liver is to eliminate toxicants through metabolism [47]. Hence, the liver becomes hyper-active to eliminate the intoxicants. Due to the hyper activity and accumulation of compounds, the cells may become larger in size and to meet the requirement, cells proliferate much faster, which may be the reasons for hyperplasia and hypertrophy. Similarly, the liver tissue will try to avoid such intoxicant being absorbed for which the epithelial tissues will lift up to avoid the toxicants. Richmonds and Dutta [55] reported that the nucleus become larger in malathion treated Blue-Gill, *Lepomis macrochirus*. Abraham and Tresa [56] and Sultana and Bojarajan [21] also reported such conditions in fishes and aquatic crustaceans. Similar changes were observed in the liver of *Anabas testudineus* due to impact of water pollutants of Buckingham canal in the present study. The liver cells were degenerated with necrosis which appeared as focal areas with lymphocytic infiltration. A large number of cells suffered from fatty degeneration. Similar change was observed in the liver of *Anabas testudineus* in the present study.

Histological changes in the liver of *Anabas testudineus* in the present study are in conformity with *Oreochromis mossambicus* exposed to the organophosphate monocrotophos [21, 57]. Elezaby *et al.* [58] studied the effect of malathion on *Oreochromis niloticus* and their results showed that this insecticide induced many histopathological changes in the liver and gills of the fishes. These changes were haemorrhage, necrosis and destruction of lamellae of the gills, and necrosis and lipidosis in the liver. Kurian *et al*. [59] and Altinok and Capkin [23] observed extensive degeneration of cytoplasm with pyknosis of nuclei and loss of glycogen in liver tissue of *Heteropneustes fossilis* and *Anabas testideneus* while subjecting them to acute thiodan and biopesticide (Neemax) toxicity. The liver of fish does not show the diversity of pathology as seen in higher animals, probably as a result of lack of Kuppler cells in the liver sinusoid [60]. However, its susceptibility to a number of toxic substances and the consequential metabolic disturbances cannot be over emphasized [61]. The high proportion of fibrotic tissue within the lobules and per biliary connective tissue of the treated specimens indicated hepatic cirrhosis. It is thus believed that the most dramatic cirrhosis found in fish as per biliary cirrhosis of the hepato-renal syndrome was associated with dietary toxicity [59]. The most frequent of the degeneration was hepatocytes enlargement with large vacuoles and sinusoid conjection, pyknosis and karyolysis observed in cases of severe intoxication with pollutants [62]. The shrinkage of the hepatic cells can result in cirrhosis and the contractions of the blood vessels thereby greatly impede the portal flow through the liver. The normal functions of the liver that involves conversion of glucose to glycogen for storage, regulation of lipids and deamination of aminoacids are impaired. The blockage of the sinusoids makes the blood flow from the hepatic artery and veins into the central vein rather difficult. The sinusoids widen to make up the right volume of blood in the central vein. The function of the canaliculi that forms the bile duct is hampered and as such, bile secreted from gall bladder cannot adequately get into the duct [61].

The intestine in the control fish had a normal lining of simple mucoid columnar epithelium, which is visible as long folds with sub-mucosa overlying the folds having eosinophilic granule cells, and is limited by dense mucosa and fibroblastic layer. *Anabas testudineus* affected with aquatic pollutants, exhibited necrosis of epithelial cells and reduction in the length of the intestinal folds. In addition, there was flattening of intestinal folds, rupture of muscular layer, reduction of villi and necrosis were prominent. Degeneration of peritoneal lining, vacuolization, histolysis of columnar cells and reduction of villi were the other marked changes seen in the present investigation. Histopathology of intestine in caged and feral freshwater fish revealed lifting of columnar epithelium of villi and hyperplasia as responses constituting the defense mechanisms of fish when exposed to the toxicants [63]. Reports on degenerative changes and rupture in tip of villi, loss of structural integrity of mucosal folds and degeneration and necrosis of sub-mucosa in the intestine of *Channa punctatus* after the exposure to carbofuran and aquatic pollutants of Arakkonam lake are found in literature [64, 65]. Sultana and Bojarajan [21] reported on vacuolization in submucosa and circular muscles and dilation of columnar and goblet cells of mucosal folds. Destruction of columnar epithelium, submucosa fused with muscles and serosa was found in disrupted condition after ten days of exposure to malathion [66]. These histopathological changes were observed in the intestine of *Cyprinus carpio* subjected to chlorinated pesticide and also in the present study.

Studies on Arakkonam lake pollutants affecting intestine of *Channa punctatus* and *Oreochromis mossambicus* also revealed degenerative changes in the serosa, longitudinal and circular muscle layers and the mucosa. Ruptured villi into pieces and their accumulation in the cavity of intestine along with goblet cells and mucus was also observed in *Mystus vittatus* treated with chromium [30], in *Oreochromis mossambicus* treated with heavy metals [21] and also due to aquatic toxicants [17,27,65]. A major effect on *Anabas testudineus* due to pollutants exposure was the enormous reduction in the size of the liver. The hepato-somatic index also showed a significant decrease. It has been pointed out that in fish and in higher vertebrates, the liver next to kidney is the major storage organ for metals and they induce various changes in the liver for the altered activity of certain liver enzymes to severe liver cirrhosis [67, 68].

Kidney acts as a detoxifying organ by collecting and removing toxic materials along with nitrogenous waste products. Gupta and Dalela [69] and Gayathri and Sultana [27] reported histological changes in kidney of *Notopterus notopterus* and

Oreochromis mossambicus exhibiting degeneration and dissolution of epithelial cells of renal tubules, vacuolization, loss of nuclei, shrunken and ruptured glomerulus at some places, tubular necrosis, especially in the convoluted portion, and inflammation of glomerulus leading to impaired kidney functions like polyuria, polydipsia, and increased build-up of non-protein nitrogen. The renal corpuscles were highly damaged exhibiting hypertrophy and necrosis following subtle exposure to phenolic compounds. Similar observations were made by Tilak *et al.* [48] under chlorpyrifos toxicity in *Cirrhinus mrigala*. The kidney plays a principal role in the accumulation, detoxification, and excretion of waste products. In the control fish, the trunk kidney involved in excretory function was formed by a large number of nephrons each having a renal corpuscle. The corpuscle is the proximal part of the tubule, which consists of two parts, a glomerulus and a capsule. In the present study, the severely shrunk lumen of tubules were suggestive of hindered tubular reabsorption in treated *Anabas testudineus*. Athikesavan *et al*. [70] reported that the histopathological abnormalities in kidney of *Heteropneustes molitrix* were time dependent and exhibited tubules with hyperplasia and hypertrophic nuclei, haemolysis of erythrocytes, cytoplasmolysis, karyolysis and vacuolization, ruptured cells, syncytical condition and pyknotic nuclei with aggregation of nuclei due to the damage of plasma membrane at the end of thirty days on exposure to nickel. The glomerulus structure was disruptured, and the convoluted and uriniferous tubules were enlarged. Similar changes were observed in the kidney of *Anabas testudineus* in the present study.

The exposure of aquatic toxicants to fish caused clear pathological symptoms in the fish as evidenced by the structural deformities and abnormal behavior. Agitated behavior exhibited by the test fish by sudden darting, erratic swimming, feeble or rapid opercular movement, gasping indicated immediate response to the toxicant-induced stress, and an attempt by the fish to overcome the stress and adapt to the new environment as an innate phenomenon. The severity of the symptoms was in accordance with the nature of the toxicant, dose and duration of exposure. [1, 48, 71]. It was very clear that the fish was most affected when the toxicants were acting in a synergistic manner compared to independent exposure. Hence, impairment of organs by the overall effect of the pathological changes in the exposed fish will have grave consequence with respect to the normal fish [72].

5. Conclusion

Pathological effects shown by toxicants are not uniform in the entire population but vary with the concentrations of toxicants, age and health of the test organism which contribute to the severity of the symptoms. The results indicate that the toxic accumulation gradually increases during the exposure period. The results of these studies may provide guidance to selection of tissues to be considered in the field of bio monitoring efforts, designed to detect the bioavailability of aquatic pollutants/ heavy metals and early-warning indicators of aquatic toxicity in fish. The results of the present study indicated that the aquatic pollutants of Buckingham canal also cause a reaching consequence in *Anabas testudineus* in the aquatic system. The results further suggest that even smaller concentrations of any toxicant in the environment can induce major histological changes and more care and vigil is needed before dumping municipal and industrial wastes/pesticides/biopesticides or organic/inorganic manure into agricultural fields or environment.

6. References

1. Ganesan S, Sultana M. Impact of aquatic pollutants on the protein profile of the fish, *Tilapia mossambicus* from Chromepet lake, Chennai, India. Journal of Aquatic Biology. 2010; 24(1):151-156.
2. Friberg LT, Piscator M, Nordberg G, Kjellstrom T. Handbook on the toxicology of metal. Vol. II. Amsterdam, 1986.
3. Allen PC. Effects of acute exposure to cadmium (II) chloride and lead (II) chloride on the haematological profile of *Oreochromis aureus* (Steindachner). Comparative Biochemistry and Physiology. 1993; 105:213-217.
4. Conacher HB, Page BD, Ryan JJ. Industrial chemical contamination of foods (review). Food Additives and Contaminants. 1993; 10(1):129-143.
5. Velez D, Montoro R. Arsenic speciation in manufactured seafood products: A review. Journal of Food Protection. 1998; 61(9): 1240-1245.
6. Ashraj W. Accumulation of heavy metals in kidney and heart tissues of *Epinephelus microdon* fish from the Arabian Gulf. Environmental Monitoring and Assessment. 2005; 101(1-3):311-316.
7. Vosyliene MZ, Jankaite A. Effect of heavy metal model mixture on rainbow trout biological parameters. Ekologika. 2006; 4:12-17.
8. Farombi EO, Adelows OA, Ajmoko YR. Biomarkers of oxidative stress and heavy metal level as indicators of environment pollution in African cat fish (*Clarias gariepinus*) from Nigeria Ogun river. International Journal of Environmental Research and Public Health. 2007; 4(2):158-165.
9. Clarkson TW. Human toxicology of mercury. Journal of Trace Elements in Experimental Medicine. 1998; 11(2-3):303-317.
10. Yousuf MHA, El-Shahawi. Trace metals in *Lethrinus lentjan* fish from Arabian Gulf: metal accumulation in kidney and heart tissues. Bulletin of Environmental Contamination and Toxicology. 1999; 62(3):293-300.
11. Farkas A, Salanki J, Specziar A. Relation between growth and the heavy metal concentration in organs of bream *Abramis brama* (L). populating lake Balaton. Archives of Environmental Contamination and Toxicology. 2002; 42(2):236-243.
12. Tort L, Torres P. The effects of sub-lethal concentration of cadmium on hematological parameters in the dog fish, *Scyliorhinus caniccula*. Journal of Fish Biology. 1988; 32(2):277-282.
13. Siraj BP, Rani UA. Cadmuium induced antioxidant defense mechanism in freshwater teleost *Oreochromis mossambicus* (Tilapia). Ecotoxicology and Environmental Safety. 2003; 56(2):218-221.
14. Sultana M, Samuel VKHC, Bhat SA. Impact of aquatic toxicants on the blood chemistry of the fish, *Anabas*

testudineus from Buckingham canal, at Uppalam area, Chennai, TN. India. International Journal of Environmental Sciences. 2012; 3(3):353-359.
15. Camargo MM, Martinez CB. Histopathology of gills, kidney and liver of a neotropical fish caged in an urban stream. Neotropical Ichthyology. 2007; 5:327-333.
16. Agamy E. Histopathological liver alterations in juvenile rabbit fish (*Siganus canaliculatus*) exposed to light Arabian crude oil, dispersed oil and dispersant. Ecotoxicology and Environmental Safety. 2012; 15:171-179.
17. Suresh M, Kalander A, Sultana M. Molecular identification of glucose transporter 4 and MyoD of *Therapon jarbua* skeletal muscle from polluted Ennore creek. International Journal of Innovation. 2013; 4(2):8-12.
18. Biswas B, Shah MS. Taxonomic comparison of local and Thai Koi (*Anabas testudineus*, Bloch) from Khulna, Bangladesh. SAARC Journal of Agriculture. 2009; 7(1):19-28.
19. Pearse AGE. Histochemistry theoretical and applied. Vol. II (New York: Churchill Livingston Inc). 1985, 441-1055.
20. Bucke D, Vethaak D, Lang T, Mellergaard S. Common diseases and parasites of fish in the north Atlantic: Training guide for identification. International Council for the exploration of the sea techniques in Marine Environmental Sciences, Copenhagen, 1996.
21. Sultana M, Bojarajan A. Histopathological lesions induced by copper sulphate, lead nitrate and zinc sulphate in the liver, intestine and kidney of *Tilapia mossambica*. Journal of Aquatic Biology. 2007; 22(1):189-192.
22. Bucher F, Hofer R. The effects of treated domestic sewage on three organs (gill, kidney, liver) of brown trout (*Salmo trutta*). Water Research. 1993; 27:255-261.
23. Altinok I, Capkin E. Histopathology of lethal toxin-induced effects and histopathological rainbow trout exposed to sub-lethal concentrations study of common carp (*Cyprinus carpio*). Toxicologic Pathology. 2007; 35:405-410.
24. Hinton DE, Lauren DJ, Gian CS. Cellular composition and ultrastructure of hepatic neoplasm's induced by diethyl nitrosamine in *Oryzias latipes*. Marine Environmental Research. 1997; 24:307-310.
25. Anbu RB, Ramaswamy M. Adaptive changes in respiratory movements of an air breathing fish, *Channa striatus* (Bleeker) exposed to carbamate pesticide, sevin. Journal of Ecobiology. 1991; 3:11-16.
26. Geraldine P, Bhavan PS, Kaliamurthy J, Zayapragassarazan Z. Intoxication of dichlorvos in the freshwater prawn *Macrobrachium malcolmsonii*. Journal of Environmental Biology. 1999; 20:141-148.
27. Gayathri V, Sultana M. Histological alterations in the gills of *Tilapia mossambicus* exposed to kerosene. International Journal of Biosciences and Technology. 2012; 5(9):45-48.
28. Verma SR, Sharma P, Tyagi A, Rani S, Dalela RC. 1983. Pollution and saprobic status of eastern Kalinadi. Limnologica (Berlin). 1983; 15(1):69-133.
29. Sultana M, Sharief DS. Effect of heavy metals on the histopathology of gills and brain of *Tilapia mossambica*. Journal of Aquatic Biology. 2004; 19(1):165-168.
30. Srivatsava VMS, Mauriya RS. Effect of chromium stress on gill and intestine of *Mystus vittatus*. Journal of Ecobiology. 1991; 3(1):69-71.
31. Sujatha LB. Studies on the physiology, haematology and histology in the Indian major carp, *Catla catla* (Hamilton) as influenced by individual and synergistic toxic effects of a pesticide and two metallic compounds. Ph.D. thesis, University of Madras, Chennai, Tamil Nadu, India. 2006.
32. Bols NC, Brubacher JL, Ganassin RC, Lee LEJ. Ecotoxicology and innate immunity in fish. Developmental and Comparative Immunology. 2001; 25(8):853-873.
33. Carter DE. Oxdiation reduction reactions of metal ions. Environmental Health Perspectives. 1995; 103:17-19.
34. Bunton TE, Poynton SL. *Kudoa* sp. (*Myxosporea multivalvulida*) infection in juvenile white perch, *Morone americana* (Gmelin): Histopathology and spore morphology. Journal of Fish Diseases. 1991; 14:589-598.
35. Sathe PS, Basu A, Mourya DT, Marthe BA, Gogate SS, Banerjee K. A cell line from the gill tissues of Indian cyprinoid *Labeo rohita*. In Vitro Cellular and Developmental Biology. 1997; 33(6):425-427.
36. Wenkuan L, Xiang Y, Xiurong W, Li WK, Yu X, Wen XR. Studies on the gill histopathology of silver carp, *Hypophthalmichthys molitrix* infected by *Dactylogyrus vaginulatus* and its treatment. Journal of Fishery Sciences of China. 1994; 18(4):305-311.
37. Hughes GM, Perry SF, Brown VM. A morphometric study of effects of nickel, chromium and cadmium on the secondary lamellae of rainbow trout gills. Water Research. 1979; 13:665-679.
38. Kondal JK, Gupta S, Saxena PK. Acute toxicity of vegetable oil factory effluent to some freshwater teleosts in relation to size. Toxicology Letters. 1984; 21:155-162.
39. Kendall MW, Dale JE. Scanning and transmission electron microscope observations of rainbow trout gill. Journal of the Biological Board of Canada. 1979; 36:1072-1079.
40. Susan AT. Toxicity and effect of fenvalerate to the 3 major Indian carps, *Labeo rohita*, *Catla catla*, *Cirrhinus mrigala* (Ham.) by gas liquid chromatography. Pollution Research. 1994; 18(1):57-59.
41. Vijayalakshmi S, Tilak KS. Effect of pesticides on the gill morphology of *Labeo rohita*. Journal of Ecotoxicology and Environmental Monitoring. 1996; 6(1):59-64.
42. Veeraiah K. Cypermethrin toxicity and its impact on histochemical and histological changes in the Indian major carp, *Labeo rohita* (Ham.). Ph.D. thesis, Nagarjuna University, Guntur, India, 2001.
43. Tilak KS, Veeraiah K, Reddy JV. A study on nitrite-nitrogen effect on haemoglobin content of the fish *Puntius sophore* and *Channa punctatus*. Pollution Research. 2001a; 20(2):179-181.
44. Tilak KS, Veeraiah K, Yacob K. Studies on histopathological changes observed in the gill, liver, and kidney of *Ctenopharyngodon idella* exposed to technical fenvalerate and EC 20%. Pollution Research. 2001b; 20(3):387-393.
45. Tilak KS, Yacobu K. Toxicity and effect of fenvalerate

on fish *Ctenopharyngodon idellua*. Journal of Ecotoxicology and Environmental Monitoring. 2002; 12:9-15.
46. Lohar PS. Comparative toxicity of four heavy metals in freshwater fishes. Journal of Aquatic Biology. 2000; 15(1/2):95-98.
47. Eller LL. Gill lesions in fresh water teleosts. In: Rivebelin. Migaki WEG. (Ed). The pathology of fishes, University of Wisconsin Press's. 1971, 305-330.
48. Tilak KS, Veeraiah K, Rao K. Histopathological changes observed in the gill, liver, brain and kidney of the Indian major carp *Cirrhinus mrigala* exposed to chlorpyrifos. Pollution Research. 2005; 24(1):101-111.
49. Roberts RJ, Rodger HD. Chapter 3: The pathophysiology and systematic pathology of teleosts. In: Roberts RJ (Ed). Fish pathology. Saunders Publishing, London. 2001, 55-133.
50. Mitchell, Cortran. Cell injury adaptation and death. In: Kumar V, Cortran R, Robins SL (Eds). Robbins basic pathology. Saunders, New Delhi, India. 2000, 3-32.
51. Sultana M. Heavy metal contamination in the Double lake (Erettai eri), Kolathur, Chennai, Tamilnadu. Journal of Aquatic Biology. 2002; 20(1):53-57.
52. Jana S, Bandopadhyaya S. Effect of heavy metal on some biochemical parameters in the freshwater fish *Channa punctatus* (Bloch). Environment and Ecology. 1987; 5:488-490.
53. Hemmaid KZ, Kaldas S. Histopathological and histochemical alterations occur in liver of fishes (*Oreochromis niloticus*) exposed to sub-lethal concentrations of ammonia. Journal of Union Arabian Biology. 1994; 01A:83-102.
54. Vurk S, Sharma RC. Biochemical changes induced by nickel and chromium in the liver of *Cyprinus carpio*. Pollution Research. 1999; 18(3):217-222.
55. Richmonds C, Dutta HM. Histopathological changes induced by malathion in the gills of bluegill *Lepomis macrochirus.* Bulletin of Environmental Contamination and Toxicology. 1989; 43:123-130.
56. Abraham K.M, Tresa R. Study on the gill of field crab, *Paratelphusa hydrodromus* (Herbst.) exposed to nickel. Journal of Environmental Biology. 2002; 23(2):151-155.
57. Desai AK, Joshi VM, Ambadka PM. Histological observations on the liver of *Tilapia mossambica* after exposure to monocrotophos an organophosphorous insecticide. Toxicology Letters. 1984; 21:325-331.
58. Elezaby MM, El-Serafy S, Heckmann R, Sharf-Eldeen KH, Seddek MM. Effect of some toxicants on the fresh water fish *Oreochromis niloticus*. Journal of the Egyptian-German Society of Zoology. 2001; 36:407-434.
59. Kurian J, Esakku S, Palanivelu K, Selvam A. Studies on landfill mining at solid waste dumpsites in India. Proceedings Sardinia '03, Ninth International Landfill Symposium, Cagliari, Italy. 2003, 248-255.
60. Ellis JE, Wiens JA, Rodell CF, Anway JC. A conceptual model of diet selection as an ecosystem process. Journal of Theoretical Biology. 1976; 60:93-108.
61. Olojo EAA, Olurin KB, Mbaka G, Oluwemimo AD. Histopathology of gill and liver tissue of the African catfish *Clarias gariepinus* exposed to lead. African Journal of Biotechnology. 2005; 4(1):117-122.
62. Jiraungkoorskul W, Upatham ES, Kruatrachue M, Pokethitiyook P. Biochemical and histopathological effects of glyphosate herbicide on Nile tilapia (*Oreochromis niloticus*). Environmental Toxicology. 2003; 19:260-267.
63. Tuvikene A, Huuskonen S, Koponen K, Ritola O, Mauer Ü, Lindström-Seppä P. Oil shale processing as a source of aquatic pollution: monitoring of the biologic effects in caged and feral freshwater fish. Environmental Health Perspectives. 1999; 107:745-752.
64. Muley DV, Kamble GB, Gaikwad PT. Endosulfan toxicity in the freshwater fish *Tilapia mossambica.* Proceedings of Academy of Environmental Biology. 1996; 5(1):49-55.
65. Dheenadayalamurthy. Characterization and impact of aquatic pollutants on the biochemical, histopathological and molecular changes in various tissues of a fish, *Channa punctatus* and *Oreochromis mossambicus*. Ph.D. thesis, University of Madras, Chennai, Tamil Nadu, India. 2012.
66. Bakthavathsalam R, Ramalingam R, Ramaswamy A. Histopathology of liver, kidney and intestine of the fish *Anabas testudineus* exposed to furadan. Environment and Ecology. 1984; 2(4):243-247.
67. Tafenelli R. Toxicity and pathogenic effects of intraperitonial infections of cadmium chloride to gold fish; Ph.D. thesis, Oklahoma State University, US. 1972.
68. Nordberg GF. Health hazards of environmental cadmium pollution. Ambio. 1974; 3(2):55-66.
69. Gupta AK, Dalela RC. Kidney damage in *Notopterus notopterus* (Pallus) following exposure to phenol compounds. Journal of Environmental Biology. 1987; 8:167-172.
70. Athikesavan S, Vincent S, Ambrose T, Velmurugan B. Nickel induced histopathological changes in the different tissues of freshwater fish, *Hypophthalmichthys molitrix* (Valenciennes). Journal of Environmental Biology. 2006; 27(2): 391-39.
71. Galat DL, Post G, Kerfe TJ, Boucks GR. Histopathological changes in the gill, kidney and liver of Lohonta cut throat trout, *Salmo clarki*, Henshawi, living in lakes of different salinity – alkalinity. Journal of Fish Biology. 1985; (27):533-552.
72. Fatma A, Mohamed S. Bioaccumulation of selected metals and histopathological alterations in tissues of *Oreochromis niloticus* and *Lates niloticus* from lake Nasser, Egypt. Global Veterinaria, 2008; 2(4):205-218.