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Impact of Water Pollution on Histopathological and Electrophoretic Characters of *Oreochromis niloticus* Fish

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Abstract: The present study suggested in order to explore the capability of two environmental pollutants namely copper sulfate ($CuSO_4$) and lead acetate $(CHCOO)_3$ Pb to induce histopathological changes and changes in the electrophoretic pattern of serum proteins in aquatic organisms. To achieve such a purpose, *Oreochromis nilotica* were chosen as a test material for the study. After determination of LC_{50} of both pollutants, fish were treated with 1/5, 1/10 and 1/20 of LC_{50} of either $CuSO_4$ or $(CHCOO)_3$ Pb for a periods of 2, 4 and 6 weeks. The histopathological studies revealed that both chemicals under study are capable of inducing changes in different fish organs (gills, liver, kidneys and spleen) after 2 and 6 weeks of treatment and at three different concentrations. These histopathological changes were positively correlated in its effects with the increase of pollutants concentration and time of exposure. Concerning the electrophoretic patterns. The control samples exhibited 12 bands with different densities and intensities. The total number of bands were 11, 16 and 5-13 bands after exposure to 1st, 2nd and 3rd concentrations of copper sulfate, respectively. The total number of bands were ranged from 8-17, from 9-13 and from 9-12 bands after exposure to 1st, 2nd and 3rd concentrations of lead acetate respectively.

Key words: Heavy metals, *Oreochromis niloticus*, histopathology, serum protein electrophoresis

INTRODUCTION

With the growth of civilization, an increasing number of chemicals are being introduced to our environment. These chemicals are hazardous to living organisms, to humans and to our ecosystems. The aquatic environment is particularly sensitive to the toxic effects of contaminants since a considerable amount of the chemicals used in industry, urbanization and in agriculture enter marine and other aquatic environments.

Heavy metal contamination has been reported in aquatic organisms (Adham *et al*., 2002; Olojo *et al*., 2005). These pollutants build up in the food chain and are responsible for adverse effects and death in the aquatic organisms (Farkas *et al*., 2002).

The fish, as a bioindicator species, plays an increasingly important role in the monitoring of water pollution because it responds with great sensitivity to changes in the aquatic environment. The sudden death of fish indicates heavy pollution; the effects of exposure to sub lethal levels of pollutants can be measured in terms of biochemical, physiological or histological responses of the fish organism (Mondon *et al*., 2001).

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Nile tilapia, *Oreochromis niloticus*, is one of the most common freshwater fishes used in toxicological studies (Figueiredo-Fernandes *et al*., 2006a, b; Garcia-Santos *et al*., 2006), because it present a number of characteristics that may make it an appropriate model that can be used as indicator species in biomonitoring programs (Gadagbui *et al*., 1996).

Gills are the first target of waterborne pollutants due to the constant contact with the external environment, as well as the main place for copper uptake (Campbell *et al*., 1979). It is well known that changes in fish gill are among the most commonly recognized responses to environmental pollutants (Mallatt, 1985; Au, 2004).

Metals can either increase or decrease hepatic enzyme activities and can lead to histopathological hepatic changes, depending on the metal type and concentration, fish species, length of exposure and other factors (Paris-Palacios *et al*., 2000).

Smith (1990) reported that gel electrophoresis was a powerful technique for fish-stock identification. The protein electrophoresis was successfully employed to study the variations among marine fish populations.

Subsequently this study was planned to investigate the capability of copper sulfate and lead acetate to induce histopathological and electrophoretic changes in aquatic organisms.

In order to achieve such a purpose *Oreochromis nilotica* fish were chosen and employed.

Moreover the study was carried out in the following steps:

Step 1 : Determination of the acute lethal concentration LC_{50}/72 h of copper sulfate and lead acetate.

Step 2 : Investigation of the effect of the two chemicals on histopathology and protein electrophoresis patterns at different exposure intervals, at various concentrations levels.

MATERIALS AND METHODS

A total number of 300 apparently healthy fish namely *Oreochromis niloticus* were obtained from Al-Abbassa fish farm (Abo Hammad, Al-Sharkia province, Egypt). The sampled fish were approximately 20-40 g in weight. Fish transported to the aquaria located in the laboratory in glass jars full of water, each aquarium of 80×40×50 cm with capacity of 160 L. The aquaria were supplied with the equipments for water quality control (pumps, filters and thermostats) and filled with dechlorinated tap water treated to be clear and safe for the fish and partially changed every other day. Fish were acclimatized to laboratory condition for at least two weeks before experiments during which they fed twice daily on the fine fish commercial diet.

Chemicals

Copper Sulfate

Copper was provided in the form of copper sulfate salt [$CuSO_4.5H_2O$; M.W. 249.68].

Lead Acetate

Lead was provided in the form of lead acetate salt [$(CH_3COO)_2Pb.\ 3H_2O$; M.W.379.34].

Determination of the Acute Lethal Concentration Dose (Lc_{50}/72 h) For Copper and Lead

The LC_{50}/72 h was calculated according to the standard procedure of Weil (1952) by the following Eq.

$$\text{Log } LC_{50} = \text{Log } D_a + d\,(f+1) \text{ For } K = 3$$

Where:

LD_a = The logarithm of the lowest dosage level
d = The logarithm of the constant ratio between dosage levels
f = A constant value obtained from the tables according to the mortality data and number of the animals
K = No. of fish in each replicate

Histopathological Examination

The collected specimens of the various organs (gills, liver, kidneys and spleen)from fish treated with copper sulfate and lead acetate were fixed in 10% buffered formalin solution then processed for histopathological studies in ascending grades of ethyl alcohol, cleared in xylol, then embedded in paraffin wax. Paraffin sections of about 5-7 microns thickness were prepared and then stained with hematoxylin and eosin (Bancrift *et al*., 1996).

Electrophoretic Studies of Serum Protein

The fish of controlled and treated groups were bled by cardiac puncture; the freshly drawn blood was allowed to stand for 1 h at room temperature and was then left to contract at 4°C for 6 h. Serum was separated by centrifugation at 3000 rpm for 3 min then transferred to eppindrof tubes, kept at -20°C till electrophoresis.

Sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) was performed according to the method of Laemmli (1979) after being modified by Studier (1973).

RESULTS AND DISCUSSION

Determination of the acute lethal concentration dose LC_{50}/72 h of copper and lead on *Oreochromis nilotica*

According to the equation of Weil (1952) the LC_{50}/72 h of copper sulfate and lead acetate for *Oreochromis nilotica* were 40.6 and 422.5 mg L^{-1}, respectively.

Histopathological Findings

Copper

Gills

At 2 weeks post-intoxication, the examination of gills exhibited congestion of gill arch and lamellar blood vessels, together with edema, hemorrhage and leukocytic infiltration. Lamellar epithelial hyperplasia was observed (Fig. 1). At 6 weeks gills showed epithelial hyperplasia of the secondary lamellae with desquamation of the gill epithelial from the underlying basement membrane (Fig. 2). These gill histological alterations has been observed by several authors in fish submitted to copper (Karan *et al*., 1998; Chen and Lin, 2001; De Boeck *et al*., 2001).

António Figueiredo-Fernandes *et al*. (2007) found that the main histopathological changes observed in gills exposed to 2.5 mg L^{-1} of waterborne copper for a period of 21 days were edema, lifting of lamellar epithelia and an intense vasodilatation of the lamellar vascular axis. Although less frequent, lamellar fusion caused by the filamentar epithelium proliferation and some lamellar aneurisms were also found.

Liver

At 2 weeks liver revealed congestion and hemorrhage in the hepatic sinusoids with dilation of hepatic vessels. The hepatic cells suffered vacular degeneration and fatty changes (Fig. 3). At 6 weeks the hepatic cells exhibited vacular degeneration and other hepatic cells were necrotic showed pyknotic and karyolitic nuclei (Fig. 4). Most of pancreatic acini were atrophid and showed degenerative changes in the pancreatic acinar cells (Fig. 5). António Figueiredo-Fernandes *et al*. (2007) exposed Nile tilapia,

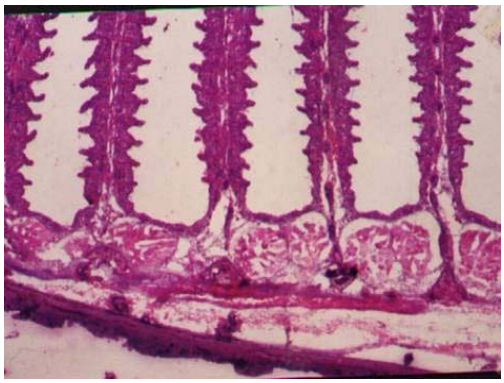

Fig. 1: Gills, 2 weeks of copper toxicosis showing lamellar epithelium hyperplasia with oedema and congestion with gill arch and lamellar blood vessels (H and E, X100)

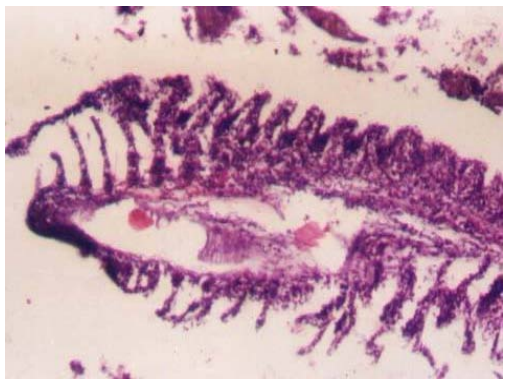

Fig. 2: Gills, 6 weeks of copper toxicosis showing hyperplasia of secondary lamellae with desquamation of gill epithelium (H and E, X400)

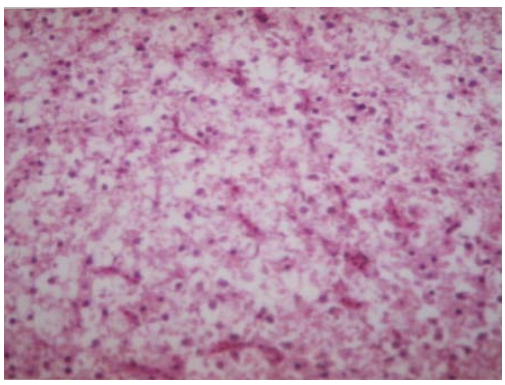

Fig. 3: Liver, 2 weeks of copper showing vacular degeneration of the hepatic cells beside congestion and hemorrhage of the hepatic sinusoids (H and E, X 400)

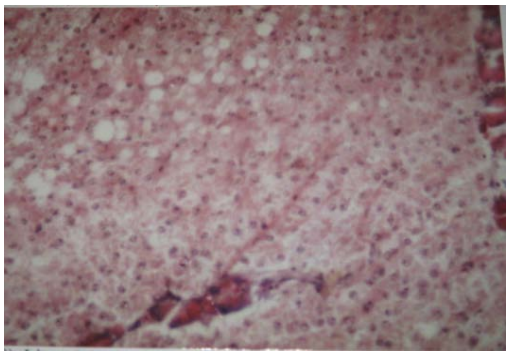

Fig. 4: Liver, 6 weeks of copper toxicosis showing vacular degeneration and necrosis of hepatic cells (H and E, X 400)

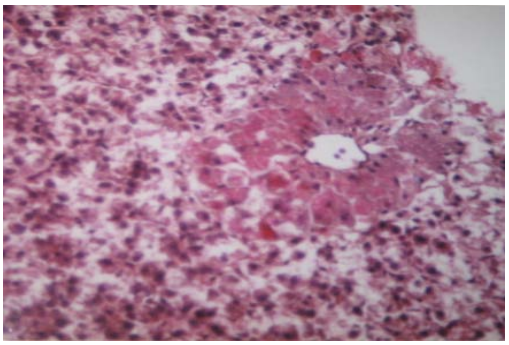

Fig. 5: Liver, 6 weeks of copper toxicosis showing atrophy of pancreatic acini and degenerative changes in pancreatic acinar cells (H and E, X 400)

Oreochromis niloticus, of both sexes to 0.5, 1.0 and 2.5 mg L^{-1} of waterborne copper for a period of 21 days. The liver of control group exhibited a quite normal architecture, while the fish exposed to copper showed vacuolation and necrosis. These hepatic alterations were more evident in fish exposed to 1.0 and 2.5 mg L^{-1} copper concentrations. The number of hepatocytes nucleus per mm^2 of hepatic tissue decreased with the increase of copper concentration. In contrast, the hepatic somatic index was high in fish exposed to 2.5 mg L^{-1} of copper. Alterations in the size of nucleus have been previously regarded by Paris-Palacios *et al*. (2000) in *Brachydanio rerio* exposed to sub lethal concentrations of copper sulphate. Braunbeck *et al*. (1990) referred that alterations in size and shape of nucleus have often been regarding as signs of increased metabolic activity but may be of pathological origin.

Kidneys

At 2 and 6 weeks the kidneys showed sever degenerative changes of the renal tubular cells, coagulative necrosis and hyalin cast. Lymphatic infiltration around the degenerative renal tubules and decrease number of melanomacrophage center were noticed (Fig. 6). Wangsongsak *et al*. (2007) found prominent tubular and glomerular damage in the kidney of common silver barb, *Puntius gonionotus*, exposed to the nominal concentration of 0.06 mg L^{-1} Cd for 60 days. Also Koponen *et al*. (2001) stated that histopathological study of bream (*Abramis brama*) and asp (*Aspius aspius*) living in a PCB-polluted freshwater lake revealed abnormal cellular changes in the renal corpuscle of both species, dilation of glomerular capillaries (DGC), mesangial edema (ME), an adhesion between visceral and parietal layers of Bowman's capsule (ABC) and filling of Bowman's space (FBS).

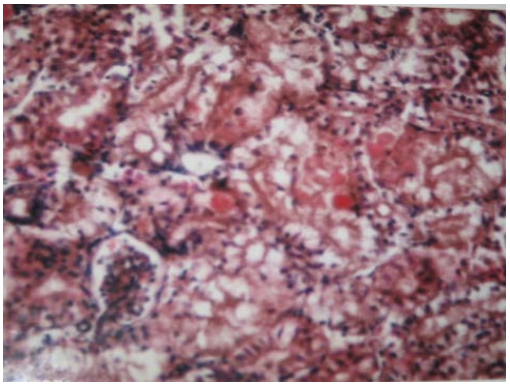

Fig. 6: Kidney, 6 weeks of copper toxicosis showing tubulonephrosis and coagulative necrosis (H and E, X 400)

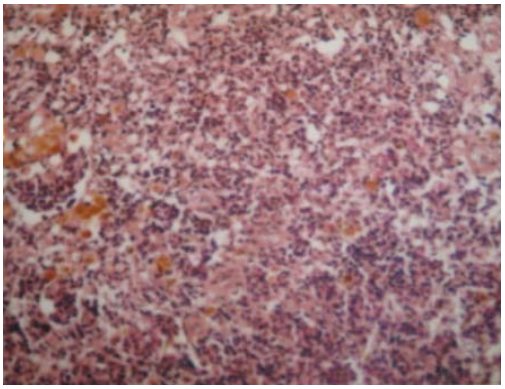

Fig. 7: Spleen, 6 weeks of copper toxicosis showing depletion of lymphoid follicles and coagulative necrosis infiltration (H and E, X 400)

Spleen

At 2 weeks spleen showed congestion of the splenic vessels, depletion of lymphoid follicles and hyper activation of melanomacrophages centers.

At 6 weeks spleen exhibited depletion of the lymphoid follicles and activation of melanomacrophage centers. Focal coagulative necrosis could be detected throughout the splenic parenchyma (Fig. 7). Bano and Hasan (1990) found that the pathomorphological alterations in relation to mercury toxicity in spleen were associated with the disorganization of the splenic cords resulting in the displacement of lymphatic tissue cells within the substance of splenic pulp. Marked depletion of the red pulp was noticeable.

Lead

Gills

At 2 weeks post intoxication gills revealed congestion, edema and extravasted erythrocytes with lymphocytic infilteration in gill arch (Fig. 8). Hyper activation of the epithelial cells of lamellae and congestion of the central lamellar vein were seen. At 6 weeks gills showed severe degenerative change, vacuolation and necrosis of lamellar epithelial cells (Fig. 9). Congestion of central lamellar vein and hyperplasia of lamellar epithelial were evident. Some studies revealed that interstitial edema is one of

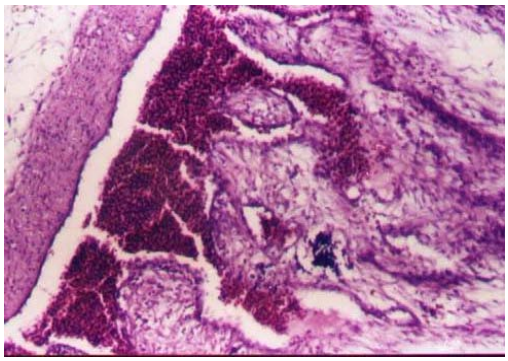

Fig. 8: Gills, 2 weeks of lead toxicosis showing oedema and extravasated erythrocytes with lymphatic infiltration in gill arch (H and E, X100)

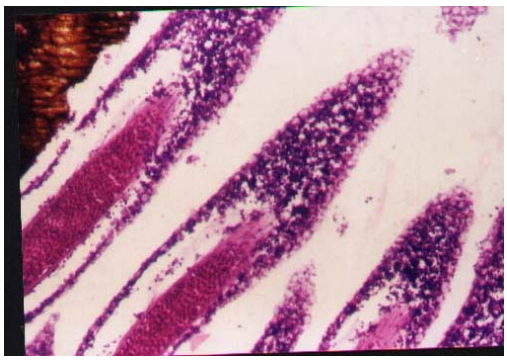

Fig. 9: Gills, 6 weeks of lead toxicosis showing vacuolation and necrosis of lamellar epithelial cells beside congestion of central lamellar vein (H and E, X100)

the more frequent lesions observed in gill epithelium of fish exposed to heavy metals (Mallatt, 1985). Edema with lifting of lamellar epithelium could be serve as a mechanism of defense, because separation epithelial of the lamellae increases the distance across which waterborne pollutants must diffuse to reach the bloodstream (Arellano *et al*., 1999). Also Onwumere and Oladimeji (1990) observed the accumulation of heavy metals and histopathology in *Oreochromis niloticus* exposed to treated petroleum refinery effluent from the Nigerian National Petroleum Corporation, Kaduna. The accumulation was, in decreasing order, Pb, Fe, Zn, Cu, Mn, Cr, Ni and Cd, examination of the organs for histopathology revealed damages to the gills. Gills with edematous fused lamellae congested with blood were observed. Koca *et al*. (2005) studied water quality and the distribution of some heavy metals in gills of Lepomis gibbosus from the Cine Stream and observed a significant decrease in mean length of primary and secondary lamellae. Moreover, cellular proliferation developed with secondary lamellae fusion, ballooning degenerations or club deformation of secondary lamellae, as well as distribution of necrotic, hyperplastic and clavate secondary lamellae.

Liver

At 2 weeks: liver showed vacuolar degeneration of the hepatocytes in addition to congestion of the central vein and hepatic sinusoid with focal hemorrhage (Fig. 10). At 6 weeks: liver exhibited severe diffuse vacuolar degeneration of hepatocytes and necrosis of some hepatocytes (Fig. 11). There was

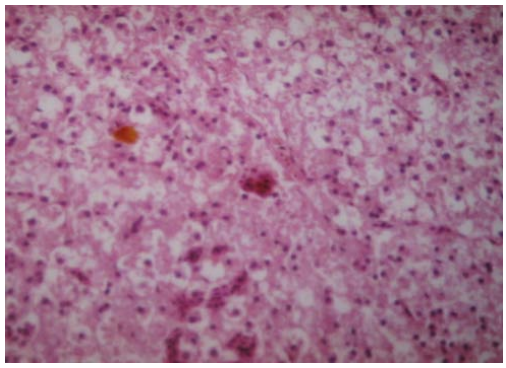

Fig. 10: Liver, 2 weeks of lead toxicosis showing vacular degeneration of hepatocytes and focal hemorrhage (H and E, X 400)

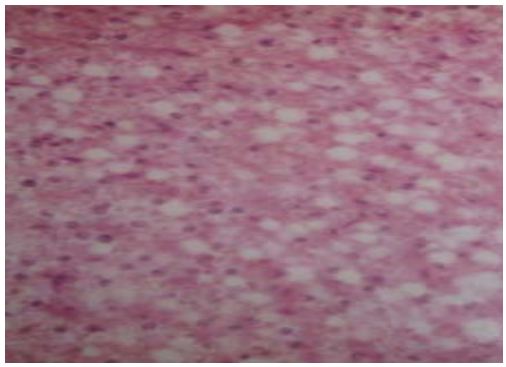

Fig. 11: Liver, 6 weeks of lead toxicosis showing vacular degeneration and necrosis of hepatocytes (H and E, X 400)

vacuolation of the pancreatic acinar cells. Several studies had shown a variety of changes in the liver of *O. niloticus*, resulting from exposure to different toxic chemicals (Visoottiviseth *et al*., 1999; Figueiredo-Fernandes *et al*., 2006a, b). Koca *et al*. (2008) investigated the histopathological effects of water pollution on two fish species caught from the Buyuk Menderes River and from its tributary. They found that Maximal metal accumulation was observed in the liver, the changes included swollen and ruptured parenchymal cells, loss of cord structure, vacuoles filled with cellular debris, focal necrosis and a significant increase in Kupffer cells.

Kidney

At 2 weeks kidneys showed tubulonephrosis and focal hyaline cast. At 6 weeks kidneys revealed vacuolar degeneration and extensive necrosis of epithelial renal tubules in addition to hyaline cast (Fig. 12). Bravo *et al*. (2005) stated that Kidney tubule alterations after exposure of (*Caquetaia kraussii* and *Colossomna macropomum*) to an herbicide, triazine, included loss of plasmalemma and cell interdigitations, misshaped mitochondria, decrease in rough endoplasmic reticulum and free polysomes and the presence of autophagic vacuoles and primary lysosomes. These alterations at the cellular level may explain fish behaviour in terms of kidney tubule pathology and relative amounts and conditions of organelles within affected cells.

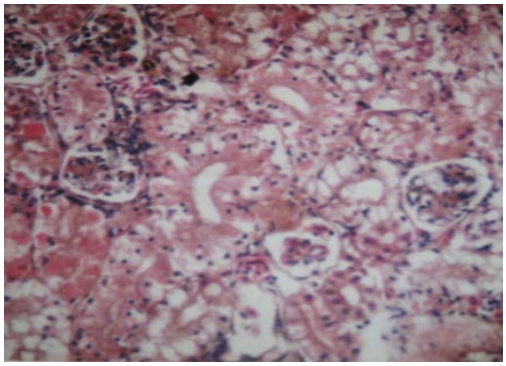

Fig. 12: Kidney, 6 weeks of lead toxicosis showing necrosis of renal tubules (H and E, X 400)

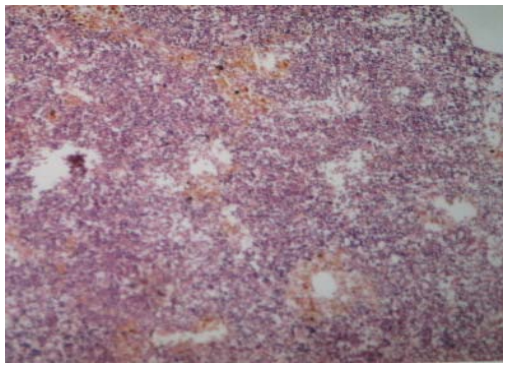

Fig. 13: Spleen, 6 weeks of lead toxicosis showing focal parenchymal edema, depletion of lymphoid follicle and excessive hemosiderosis (H and E, X 100)

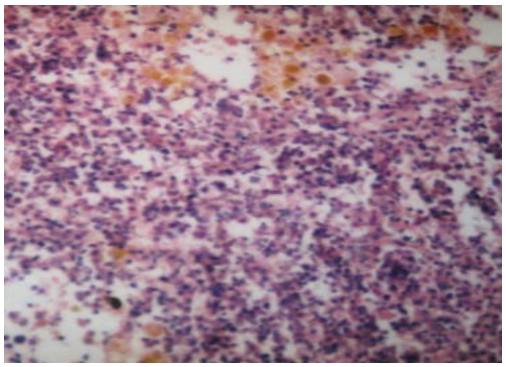

Fig. 14: Higher magnification of photo (13) showing depletion of lymphoid follicles (H and E, X 400)

Spleen

The most common pathological changes at 2 weeks and 6 weeks intoxication were focal parenchymal edema, depletion of lymphoid follicles in addition to excessive hemosiderosis (Fig. 13, 14). El-Sayed and Saad (2007) found hyperactivation of melanomacrophage centres of spleen in The monosex Nile tilapia, *Oreochromis niloticus* L., that exposed to subacute concentration

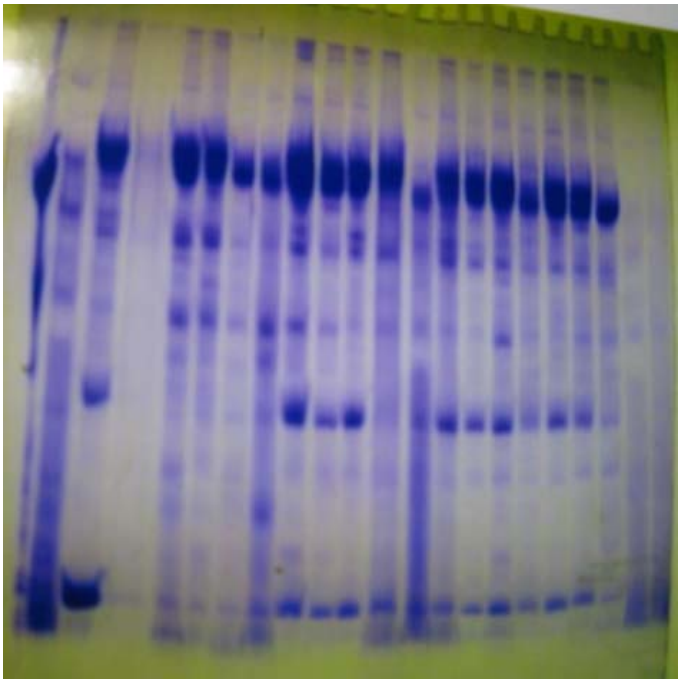

Fig. 15: Variation in electrophoretic patterns of serum proteins for *Oreochromis niloticus* treated with different concentrations of copper sulfate and lead acetate

(1.46 g L^{-1}) of a pyrethroid insecticide, deltamethrin for 28 consecutive days, similar alterations in the mosquitofish, *Gambusia affinis*, exposed to two sublethal concentrations of deltamethrin (0.25-0.50 μg L^{-1}) for periods of 10, 20 and 30 days, have been observed by Cengiz and Unlu (2006).

Electrophoretic Studies

According to molecular weights, a total of 26 bands were detected. The control samples exhibited 12 bands with different densities and intensities (Fig. 15).

Toxicants can enter the bloodstream through the gills or the gastrointestinal tract (Doving, 1991).

Thus, evaluation of fish blood provides valuable information concerning the physiological response of fish to changes in the external environment, including the presence of toxicants (Van Vuren *et al*., 1994).

Variation in serum proteins electrophoresis of *O. niloticus* after exposure to copper sulfate.

The total number of bands was 11, 16 and 5-13 bands after exposure to 1st, 2nd and 3rd concentrations of copper sulfate respectively.

Dutta *et al*. (1992), Richmonds and Dutta (1992) and Datta-Munshi *et al*. (1999) reported decreases in the high-mobility proteins when fish were exposed to OP pesticides for 24 h. The above authors suggested that the high-mobility proteins include albumin.

Fish exposed to the three concentrations shared two common bands (band 13 at M.W. 77.858 and band 25 at M.W. 17.393). Dutta *et al*. (1992) found a new protein fraction when Indian catfish were exposed to malathion for 48 h. Formation of the new protein may be attributed to the cellular damages caused by this pesticide. Tissue damage would result in leakage from the plasma membrane of cellular proteins, for instance, intracellular enzymes, into the blood on the other hand fish exposed to 1st and 2nd concentrations shared one specific band at M.W. 30.165. One band

(band 16 at M.W. 61.401) was missing after exposure to the three concentrations of copper sulfate for different exposure times. In addition one specific band at M.W. 56.477 was appeared as a result of these treatments. Rizkala *et al*. (1997) studied the genotoxic effects of different dose (1/10, 1/5 and LC_{50}) of organophorus compound (Hinosan) and carbamate (sevin) for 30 days on the electropherogams of sarcoplasmic proteins of common carp (*Cyprinus carpio*) with regard to the number, mobility and density of fractions. They found that:

- Ten percent untreated normal gel electrophoresis of protein extracted from untreated normal specimens yielded 14 bands. These bands were to be: the fastest mobile band, 4 distal bands, 5 mid bands, 3 proximal bands and the last mobile band fractions 1 and 4a showed the highest and lowest density percentage of all bands, respectively.
- Hinosan exposed specimens yielded 13-15 bands. The percentage of appearance (polymorphism) ranged from 33-100%. Anew band 8b appeared with 33.3% polymorphism at 1/10 LC_{50} concentration in addition band 4b only appeared in ½ LC_{50} dose with 50% polymorphism on the other hand, at 1/5 and ½ LC_{50} band 10 disappeared completely.
- The difference in number of protein bands was marked in sevin treated specimens. 13-16 fractions were scannered with the three dose studied. At ½ LC_{50} dose, fractions 4b and 8b appeared newly. On the other hand, fractions 6 and 10 completely disappeared at both 1/10 and 1/5 LC_{50} dose percentage of polymorphism ranged from 33.3-100%.
- Comparison of the relative mobilites of bands between control and exposed hinosan and sevin specimens showed some amount of variations especially in the proximal fractions.
- Studying the effects of different concentrations of Hinosan and sevin on changes in the relative proportions of protein fraction detected that band 9 was the most strengthen fractions appeared in the investigation. However, total distal fraction recorded a significant variation when fish were exposed to both pesticides.

Table 1 represented the similarity matrix between different combinations of treatments with copper sulfate. The highest value of similarity matrix was that obtained in case of control/C_3d (-0.083). The lowest was that between control/C_3-a (0.553).

Variation in serum protein electrophoresis of *O. nilotica* after exposure to lead acetate.

The total number of bands were ranged from 8-17, from 9-13 and from 9-12 bands after exposure to 1st, 2nd and 3rd concentrations of lead acetate.

Pottinger and Carrick (1999) reported that the aquaculture environment exposes the fish to a repeated acute stress which has deleterious effects on growth, reproduction and the immune response.

Fish exposed to the three concentrations for different exposure times shared one specific band at M.W. 17.393. Two common bands at MW_S 61.401 and 26.13 were missed after exposure to the three concentrations for different exposure times. Chiesa Maria *et al*. (2006) evaluated the effects of weekly administration of sub lethal Pb (as acetate, 50 mg kg^{-1}) during 6 weeks on the profile of the serum

Table 1: The similarity matrix between fish populations treated with copper sulfate

	Control	C_3-a	C_3-b	C_3-c	C_3-d	C_2-a	C_2-b
C_3-a	0.553						
C_3-b	0.331	0.52					
C_3-c	0.309	0.333	0.488				
C_3-d	-0.083	0.219	0.136	0.463			
C_2-a	0.256	0.184	0.386	0.474	0.256		
C_2-b	0.256	0.356	0.185	0.474	0.573	0.35	
C_1-a	0.30	0.272	0.57	0.234	0.30	0.677	0.357

Where, C_1: First concentration of copper sulfate (1/5 LC_{50}). C_2: Second concentration of copper sulfate (1/10 LC_{50}). C_3: Third concentration of copper sulfate (1/20 LC_{50}). a: Two weeks exposure time. b: Four weeks exposure time. c: Six weeks exposure time. d: Eight weeks exposure time

Table 2: The similarity matrix between fish populations treated with lead acetate

	Cont	L_3-b	L_3-c	L_3-d	L_2-a	L_2-c	L_2-d	L_1-a	L_1-b	L_1-c
L_3-b	0.462									
L_3-c	0.378	0.089								
L_3-d	0.226	0.299	0.537							
L_2-a	0.00	-0.08	0.632	0.463						
L_2-c	0.137	0.15	0.588	0.299	0.404					
L_2-d	0.30	0.031	0.603	0.456	0.389	0.686				
L_1-a	0.025	0.19	0.243	0.511	0.404	0.359	0.459			
L_1-b	0.30	0.359	0.288	0.456	0.389	0.195	0.212	0.296		
L_1-c	0.051	0.04	0.158	0.219	0.167	0.566	0.61	0.31	0.104	
L_1-d	-0.08	-0.03	0.378	0.536	0.463	0.624	0.613	0.674	0.144	0.386

Where: L_1: First concentration of lead acetate (1/5 LC_{50}). L_2: Second concentration of lead acetate (1/10 LC_{50}). L_3: Third concentration of lead acetate (1/20 LC_{50}). a: Two weeks exposure time. b: Four weeks exposure time. c: Six weeks exposure time. d: Eight weeks exposure time

proteins of the adult South American toad, Bufo arenarum (Anura: Bufonidae). The electrophoretic patterns of serum proteins pointed out the presence of four fractions: the metal provoked a significant decrease in both total proteins and albumin fraction; among the globulin fractions, the G3 resulted augmented. These findings may be related to the impact of lead on the toads' hepatic cells and immune system.

After exposure to the 1st concentration of lead acetate besides missing of bands 16 and 22, band 12 present only after 2 weeks exposure time, band 8 present only after 6 weeks exposure time while band 18 observed only after 4 weeks exposure time. While after exposure to the 2nd concentration bands 18, 23 and 26 absent after the three exposure times. Furthermore after exposure to the 3rd concentration beside absence of band 23 band 18 observed only after 2 weeks exposure time. Shenker *et al.* (1993) found a suppressive effect on immune system of fish as result of Zn, Cu and Hg exposure even at very low concentrations. Ramalingam (1982) who was working on *T. mosambicus* and showed a decrease in liver and muscle proteins by electrophoresis after malathion treatment. Also, Bano and Hasan (1990) and Murray *et al.* (1993) reported that the decrease in serum albumin could be attributed to its disturbed synthesis due to functional effects on liver. Heavy metals lead to liver damage in fish. And Lachapelle *et al.* (1993) observed a decline in albumin secreted by hepatocytes by approximately 38% as a result of mercury toxicity.

Table 2 represented the similarity matrix between different combinations of treatments with lead acetate. The highest value of similarity matrix was that obtained in case of co control/L_1d (-0.083).The lowest was that between control/L_3-b (0.462).

His research concluded that the water pollution especially with heavy metals have histopathological and electrophoretic effects on fishes exposed to it which may lead to health risks among humans through chronic consumption of such fishes. This work also recommends the use of *Oreochromis niloticus* in order to assess the extent of pollution in aquatic environment.

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