

Bioaccumulation and histopathological alterations of the heavy metals in *Oreochromis niloticus* fish

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Abstract: Copper, lead, cadmium and mercury concentrations were recorded in water and tissues of *Oreochromis niloticus* from Egyptian fish farms in 2007-2009. Histopathological alterations in fish tissues were also studied. Bioconcentration factors of copper, lead, mercury and cadmium in liver and muscle tissue were (3.93 & 3.87), (8.10 & 7.60), (0.79 & 50.0) & (38.25 & 30.25), respectively. Mercury was the most bioaccumulated and biomagnified metal in the muscles, while Cu was the least. The concentration of cadmium, lead and copper were highest in liver and lowest in kidney tissue, while mercury (Hg) concentrations were highest in muscles, lowest in kidney tissue. Several histopathological changes were noted in muscles, liver, gills, kidney and intestine tissue attributable to heavy metals exposure. [Nature and Science. 2010;8(4):147-156]. (ISSN: 1545-0740).

Key words: Bioconcentration, copper, lead, cadmium, mercury, Tilapia, Pollution, histopathology.

1. Introduction

Metal contamination of aquatic ecosystems has long been recognized as a serious pollution problem. When fish are exposed to elevated levels of metals in a polluted aquatic ecosystem, they tend to take these metals up from their direct environment (Seymore 1994). Heavy metal contamination may have devastating effects on the ecological balance of the recipient environment and a diversity of aquatic organisms (Farombi *et al.* 2007).

Transport of metals in fish occurs through the blood where the ions are usually bound to proteins. The metals are brought into contact with the organs and tissues of the fish and consequently accumulated to a different extent in different organs and tissues of the fish. Most heavy metals released into the environment find their way into the aquatic environment as a result of direct input, atmospheric deposition and erosion due to rainwater, therefore aquatic animals may be exposed to elevated levels of heavy metals due to their wide use for anthropogenic purposes (Kalay and Canli, 2000). Heavy metals are non-biodegradable and once they enter the environment, bioconcentration occurs in the fish tissue in the case of aquatic environment, by means of metabolic and biosorption processes (Wicklund-Glynn 1991).

Heavy metals such as cadmium, lead, copper and more specifically mercury are potentially harmful to most organisms even in very low concentrations and have been reported as hazardous environmental

Pollutants able to accumulate along the aquatic food chain with severe risk for animal and human health.

Toxic heavy metal contamination mostly occurred in aquaculture farms and frequently occurs in groundwater,

rivers, estuaries, wetland and coastal areas. Of particular concern are the highly toxic non-nutrient elements such as mercury (Hg), lead (Pb), and cadmium (Cd).

The presence of pollutants have been associated with decreased fertility and other reproductive abnormalities in birds, fish, shellfish and mammals and also altered immune function. Heavy metals like mercury and cadmium are known to accumulate in marine organisms and cause rapid genetic changes (Nimmo *et al.* 1978, Nevo *et al.* 1986).

The toxicity of these elements is due to their ability to cause, oxidative damage to living tissues. Damage includes enhanced lipid peroxidation, DNA damage, enzyme inactivation and the oxidation of protein sulfhydryl groups (Taiz and Zeiger 1998). Toxic heavy metal can cause dermatological diseases, skin cancer and internal cancers (liver, kidney, lung and bladder), cardiovascular disease, diabetes, and anaemia, as well as reproductive, developmental, immunological and neurological affects in the human body. Metal contamination sources are typically derived from natural sources: mining, industrial waste discharges, sewage effluent, harbor activities and agrochemicals etc.

It is also possible that environmental toxicants may increase the susceptibility of aquatic animals to various diseases by interfering with the normal functioning of their immune, reproductive and developmental processes (Couch and John, 1978).

Prolonged exposure to water pollutants even in very low concentrations have been reported to induce morphological, histological and biochemical alterations in the tissues which may critically influence fish quality.

According to EPA guidelines, "the BCF

(Bioconcentration Factors) is defined as the ratio of chemical concentration in the organism to that in the surrounding water. Bioconcentration occurs through uptake and retention of a substance from water only, through gill membranes or other external body surfaces. In the context of setting exposure criteria it is generally understood that the terms "BCF" and "steady-state BCF" are synonymous. A steady-state condition occurs when the organism is exposed for a sufficient length of time that the ratio does not change.

The present study was carried out to investigate the bioaccumulation of heavy metals (lead, copper, cadmium and mercury) in the tissues of *Oreochromis niloticus* and to determine the histopathological changes caused by the residues of these metals in their organs.

2. Material and Methods

SAMPLING

The water samples were obtained from different farms derived their water supply from some River Nile ramifications. Forty eight water samples and one hundred adult freshwater tilapia (*Oreochromis niloticus*) ranged between 100- 150 g in weight were collected from 12 Tilapia farms located in 6 Governorates (Kafer Al-Sheikh, Ismailia, Kaliobea, Damietta, Al-Fayum and Behera) during 2007-2009. At laboratory, the fish samples were washed with deionized water and wrapped separately in acid washed polyethylene bag and stored frozen at -20°C until analysis was carried out.

PROCEDURES:

PREPARATION AND ANALYSIS OF WATER SAMPLES:

The analysis of water samples was carried out according to A.P.H.A. (1992). The water samples were preserved by the addition of one ml of concentrated nitric acid per liter until the time of analysis. The water samples were filtered through 0.45µl membrane filter. The required volume (100 ml) of the filtrate was collected to measure lead, cadmium, mercury and copper levels in water samples by using Air/Acetylene Flame Atomic Absorption Spectrophotometer (UNICAM 696 AA Spectrometer). Flameless Atomic Absorption Spectrophotometer equipped with (MHS) mercury hydride system "Cold Vapour Technique" was used for determination of mercury levels in examined water samples.

PREPARATION AND ANALYSIS OF FISH SAMPLES:

Procedure (A): Each sample was represented by one gram of tissues dissected from the gills, liver, kidney and muscles, then placed in a clean screw-capped tube

and digested according to the method described by Finerty *et al.* (1990). The obtained solutions were then analyzed by using Air/ Acetylene Flame Atomic Absorption Spectrophotometer (UNICAM 696 AA Spectrometer) for determination of copper (Cu), lead (Pb), cadmium (Cd) and mercury (Hg) levels in examined samples.

Procedure (B): The measurement of the mercury concentration in examined fish samples was carried out at minimal temperature for all fish samples where 0.5 gram macerated fish tissues was digested according to the technique described by Diaz-Ravina *et al.* (1994). About 5 ml stannous chloride solution were added to the obtained solutions to reduce mercury to elemental form and then analyzed by using Flameless Atomic Absorption Spectrophotometer equipped with "MHS" mercury hydride system "Cold Vapour Technique".

HISTOPATHOLOGICAL EXAMINATION

Tissue specimens from fresh Nile Tilapia were taken (gills, muscles, livers, intestine and kidney) and fixed in 15 % buffered neutral formalin. They were processed to obtain five micron thick paraffin sections then stained with Hematoxylin and Eosin (Bancroft *et al.*, 1996) and examined under light microscope.

STATISTICAL ANALYSIS

Data were analyzed using Analysis of Variance (ANOVA) and means were separated by Duncan at a probability level of < 0.05 (SAS Institute 2000).

3. Result

Results are shown in Table 1 (Heavy metal concentrations in water of Nile Tilapia farms) and Table 2 (Concentration of heavy metals in fresh Nile tilapia tissues). Figure A-1-12 (The histopathological alterations in Tilapia tissues) , Figure (B)-2(Mean concentrations of Cu, Pb, Cd and Hg in water of fish farms in different Governorates and the permissible limits according to WHO,1984) and Figure (C)-3 (Mean residual accumulations of Cu, Pb, Cd and Hg in tissues of *Oreochromis niloticus* and the permissible limits according to WHO,1984).

Table 1, showed that the mean concentration of copper in water of Tilapia farms was 0.65 ± 0.01 ppm, while Table 2 showed the mean concentrations of copper in gills, liver, kidney and muscles of Tilapia (were 4.8 ± 0.05 , 2.56 ± 0.21 , 1.52 ± 0.06 and 2.54 ± 0.05 ppm, respectively). The BCF of copper in liver and muscles was 3.93 and 3.87, respectively. The mean concentration of lead in water of Tilapia cultures was 0.20 ± 0.07 ppm, while the mean concentrations of lead in gills liver, kidney and muscles, were 0.483 ± 0.05 , 1.523 ± 0.02 , 0.155 ± 0.02 and 1.521 ± 0.02 ppm,

respectively. The BCF of lead in liver and muscles was 8.10 and 7.60 ppm, respectively. The mean concentration of cadmium in water of Tilapia farms was 0.04 ± 0.009 ppm, while the mean concentrations of cadmium in gills liver, kidney and muscles were 0.891 ± 0.05 , 1.523 ± 0.02 , 0.212 ± 0.02 and 1.21 ± 0.05 ppm, respectively. The BCF of cadmium in liver and muscles was 38.25 and 30.25 ppm, respectively.

The mean concentration of mercury in water of Tilapia farms was 0.07 ± 0.009 ppm, while the mean concentrations of mercury in gills liver, kidney and muscles were 0.04 ± 0.002 , 0.055 ± 0.003 , 0.020 ± 0.005 and 3.50 ± 0.22 ppm, respectively. The BCF of mercury in liver and muscles was 0.79 and 50 ppm, respectively. The histopathology of different Tilapia tissues revealed that there are several histopathological changes in different Tilapia organs (muscles, liver, gills, kidney and intestine) as shown in Figure (A1).

Gills showed mild congestion and edema of the primary lamellae (Figure A1-8). Severe edema, hyperplasia, fusion and focal desquamation of the epithelial lining of the secondary lamellae as seen in Figure (A1)-9. The gill arch, especially at the bases of the gill filaments, showed numerous mononuclear leukocytic infiltration, edema and congestion. The apex of gill filaments showed congestion, hyper activation of the mucous and chloride cells with epithelial vacuolation of the secondary lamellae.

Liver showed degeneration of the hepatocytes and intravascular haemolysis in blood vessels as shown in Figure (A1 -2), congestion of central vein, hemorrhages (Figure A1-3), nuclear pyknosis in the majority of hepatic cells (FigureA1-4) and the metal-binding proteins were accumulated in the nuclei of hepatocytes 42% of the examined adult freshwater tilapia (*Oreochromis niloticus*) were showed histopathological alterations.

Kidney The kidney is composed normally of numerous renal corpuscles with well developed glomeruli and a system of tubules. The proximal segment is covered by tall columnar epithelial cells with basal nuclei and brush border located along the cell apices. The distal segment was lined with large, relatively clear columnar epithelial cells with central nuclei and the brush border was reduced or not present. The glomerulus is larger in diameter than the distal segment, containing columnar epithelial cells with basal nuclei and no brush border (Figure A1-10). In our study, the kidney showed hydropic swelling of tubules,

sometimes with pyknotic nuclei and many necrotic areas as well as swollen proximal epithelial cells with necrotic nuclei as noticed in Figure A1- 11.

Muscular tissues Several histopathological alterations were seen in the muscles of Tilapia which included degeneration in muscle bundles with aggregations of inflammatory cells between them and focal areas of necrosis. Also, atrophy and edema of muscle bundles as well as splitting of muscle fibers were seen as in Figure.A1-6.

The pathological findings in the intestine included atrophy in the muscularis, degenerative and necrotic changes in the intestinal mucosa and submucosa with necrotized cells aggregated in the intestinal lumen, edema and atrophy in the submucosa as shown in Figure A1-12.

Table 1. Heavy metal concentrations in water of Nile Tilapia farms.

Metal	In water samples (mg/L)			Occurrence %
	Min.	Max.	Mean \pm SE	
Copper	0.044	0.887	0.65 ± 0.01	35%
Lead	0.04	0.29	0.20 ± 0.07	82%
Cadmium	0.001	0.082	0.04 ± 0.009	72%
Mercury	0.01	0.11	0.07 ± 0.009	12%

Table 2. Concentration of heavy metals (ppm) in fresh Nile Tilapia tissues.

Metal Tissue	Copper	Lead	Cadmium	Mercury
Gills				
Mean	4.8±0.05	0.483±0.05	0.891±0.05	0.04±0.002
Min	1.32	0.02	0.11	0.002
Max	6.22	1.21	1.82	0.24
Liver				
Mean	2.56 ± 0.21	1.523 ±0.02	1.523 ± 0.02	0.055 ± 0.003
Min	1.22	0.01	0.20	0.001
Max	3.55	3.20	2.43	0.72
Kidney				
Mean	1.52±0.06	0.155±0.02	0.212±0.02	0.02±0.05
Min	0.21	0.11	0.09	0.002
Max	2.42	2.02	0.89	0.12
Muscles				
Mean	2.54±0.05	1.52±0.02	1.21±0.05	3.50±0.33
Min	0.21	0.892	0.55	1.32
Max	2.8	1.00	1.780	5.240

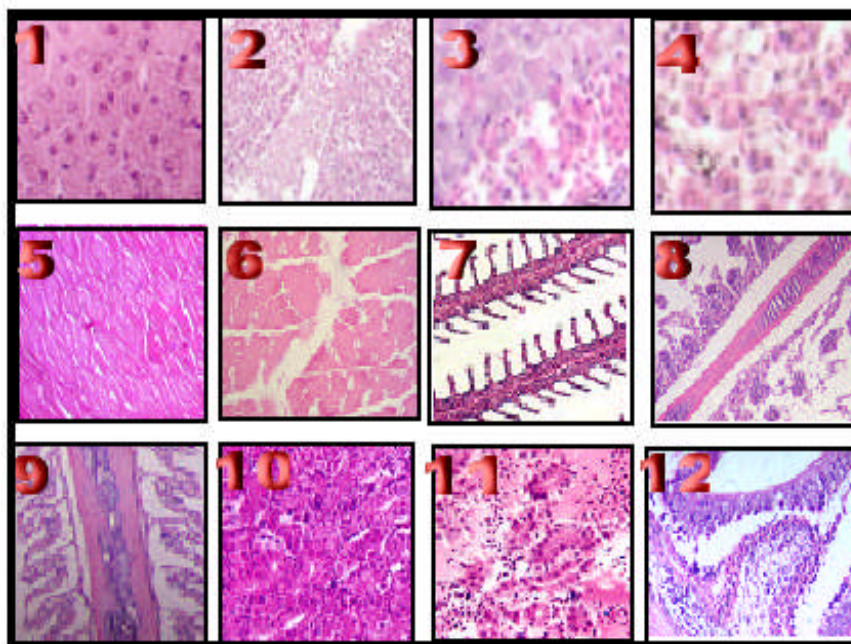


Figure A-1 (1-12). **1:** Liver of *Tilapia nilotica* fish showing the normal structure (X400). **2:** Liver of *Tilapia nilotica* fish showed degeneration of the hepatocytes and intravascular haemolysis in blood vessels. **3:** Liver of *Tilapia nilotica* fish showing haemorrhage (X400). **4:** The liver showed congestion and central vein, nuclear pyknosis in the majority of hepatic cells. (X400). **5:** Muscle bundles of *Tilapia nilotica* fish showing the normal structure (X400). **6:** Degeneration in muscle bundles with focal area of necrosis (X400). **7:** Gills: Gills of *Tilapia nilotica* fish showing the normal structure (X100). **8:** Degenerative and necrotic changes in the epithelium of gill filaments and secondary lamellae (X400). **9:** Edema in secondary lamellae and gill filaments (X400). **10:** Kidney showing the normal structure (X400). **11:** Severe degenerative and necrotic changes in the renal tubules with focal areas of necrosis (X400) and aggregations of inflammatory cells. **12:** Degeneration, haemorrhage in the submucosa and aggregations of inflammatory cells in the mucosa and submucosa (X400).

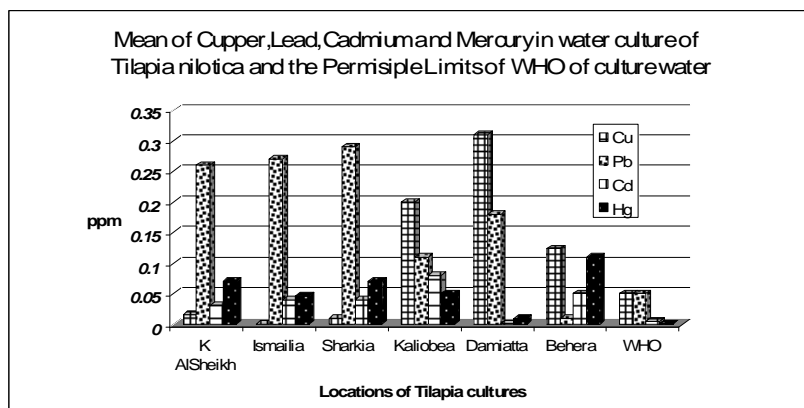


Figure (B)-2: Mean concentrations of Cu, Pb, Cd and Hg in water of fish farms in different Governorates and the permissible limits according to WHO (1984).

Table 3. Permissible limits of various heavy metals

Metal	Permissible	Country and reference
Copper	1.00 ppm	WHO (1984)
	20.0 ppm	South Africa (Foodstuffs, cosmetics and disinfectants Act. No. 54 of 1972)
	20.0 µg/g	Spain: Boletin Oficial del Estado (1991)
Lead	0.05 ppm	WHO (1984)
	0.1 mg/kg	Egypt "E.O.S.Q.C. (1993)
	0.5 ppm	FAO/WHO (1992)
	5.0 µg/g	Spain: Boletin Oficial del Estado (1991)
Cadmium	0.005 ppm	WHO (1984)
	0.05 ppm	FAO/WHO (1992)
	0.1 mg/kg	Egypt "E.O.S.Q.C. (1993)
	1.0 µg/g	Spain: Boletin Oficial del Estado (1991)
Mercury	0.001 ppm	WHO (1984)
	0.5 mg/kg	Egypt "E.O.S.Q.C. (1993)
	0.5 ppm	FAO/WHO (1992)
	1.0 µg/g	Spain: Boletin Oficial del Estado (1991), Schuhmacher and Domingo (1996)

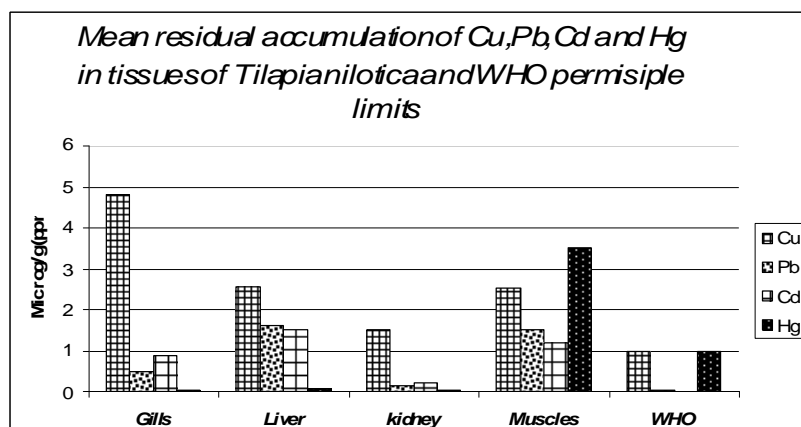


Figure (C)-3: Mean residual accumulations of Cu, Pb, Cd and Hg in tissues of *Oreochromis niloticus* and the permissible limits according to WHO (1984).

4. Discussion

Mean copper concentration in water of Tilapia farms was 0.65 ± 0.01 ppm and the maximum permissible limits recommended by WHO (1984) is 0.05 ppm, while in flesh was 2.54 ± 0.05 ppm. The recorded results of copper concentrations in fish were lower than the permissible limits intended by Foodstuffs, Cosmetics and Disinfectants (1972) [20.0 ppm] and Boletin Oficial del Estado (1991) in Spain [20.0 $\mu\text{g g}^{-1}$] and Schumacher and Domingo (1996). The BCF were; 3.93 and 3.87ppm in liver and muscles, respectively.

It is shown from Table.1 that the lead concentration in Tilapia tissues was exceed the permissible limit recommended by E.Q.S.Q.C. (1993). This result was nearly higher than those reported by Seddek *et al.* (1996) and Marouf and Dawoud (2006), they recorded levels ranged from 0.42 to 0.74 ppm. This result was much higher than those recorded by Suppin *et al.*, (2005) and Celik and Oehlenschlager (2007), they recorded levels varied from 0.04 ppm to 76.1 ppb.

High levels of lead may be attributed to presence of industrial and agricultural discharges, motor boat traffics and also from mine and smelting operations.

Lead is non-essential element and higher concentrations can occur in aquatic organisms close to anthropogenic sources. It is toxic even at low concentrations and has no known function in biochemical processes (Burden *et al.*, 1998). It is known to inhibit active transport mechanisms, involving ATP, to depress cellular oxidation reduction reactions and to inhibit protein synthesis (Waldorn and Stofen 1974). Lead was found to inhibit the impulse conductivity by inhibiting the activities of monoamine oxidase and acetylcholine esterase to cause pathological changes in tissue and organs (Rubio *et al.*, 1991) and to impair the embryonic and larval development of fish species (Dave and Xiu, 1991).

Mean cadmium concentration in water of Tilapia farms was 0.04 ± 0.009 ppm and the maximum permissible limits recommended by WHO (1984) is 0.005 ppm, while in flesh was 1.21 ± 0.05 ppm. The recorded results of cadmium concentrations in fish were higher than the permissible limits intended by Boletin Oficial del Estado (1991) in Spain [1.0 $\mu\text{g g}^{-1}$], FAO/WHO (1992) [0.05 ppm] and Egyptian Organization for Standardization and Quality Control "E.O.S.Q.C" [0.1 mg kg^{-1}]. The BCF were; 38.25 and 30.25ppm in liver and muscles, respectively. This result agree with that obtained by Daoud (1999) who reported that the cadmium concentrations in water and fish were higher than the maximum permissible limits recommended by WHO (1984). The presence of cadmium in fish in Egypt was detected by Seddek (1996) with mean levels

of 0.62 ppm in *Oreochromis* fish and 0.39 ppm in *Bagrus Byad* fish. Our result was nearly parallel to those reported by Celik and Oehlenschlager (2007) who recorded Cd concentration with levels varied from 0.1 to 0.8 ppm. Cadmium is highly toxic non-essential heavy metal and it does not have a role in biological processes in living organisms. Thus even in low concentration, cadmium could be harmful to living organisms (Burden *et al.*, 1998). The value of cadmium accumulation in liver of Tilapia was (1.523 ± 0.02) $\mu\text{g g}^{-1}$ dry weight. High accumulation of cadmium in liver may be due to its strong binding with cystine residues of metallothionein.

The high levels of Cd may be attributed to industrial and mining operations as well as the phosphate fertilizer which is considered the main source of Cd in the environment (Dimari *et al.* 2008).

Mean mercury concentration in water of Tilapia cultures was 0.07 ± 0.009 ppm and the maximum permissible limits recommended by WHO (1984) is 0.001 ppm, while in flesh was 3.50 ± 0.22 ppm. The recorded results of mercury concentrations in Tilapia tissues were higher than the permissible limits intended by Boletin Oficial del Estado (1991) in Spain [1.0 $\mu\text{g g}^{-1}$], FAO/WHO (1992) [0.5 p.p.m] and Egyptian Organization for Standardization and Quality Control (E.O.S.Q.C) (1993) [0.5 mg kg^{-1}]. The BCF were; 0.79 and 50.0 in liver and muscles respectively. These findings coincide with those reported by Daoud *et al.* (1999) and Tantawy (1997).

Conama (2005) recommend a maximum concentration of 0.0002 mg Hg l^{-1} in water supplies used for rearing fish species destined for human consumption in Brazil. This value is very similar to those recommended by Malaysia National Water Quality Standards (Doe-Um, 1986). Meanwhile, the most notorious mercury compounds in the environment are monomethyl and dimethyl salt of mercury which are soluble. They are produced from inorganic mercury in sediment by anaerobic bacteria through the action of methyl-cobalamine and intermediate in the synthesis of methane and get into natural water (Manahan, 1989). The average (88.9%) of total mercury in fish musculature was in the form of methyl mercury (Bishop and Neary, 1974) which is lipid soluble and easily absorbed and distributed through biological system.

This element is one of the most toxic metals, which are introduced into the natural environment by human interferences (Buhl, 1997). Some papers have reported situations where high mercury levels were detected in water, mainly nearby gold extraction locations (Maurice-Bourgoin *et al.* 2000; Dolbec *et al.* 2001) and industrial zones (Kime 1998, Sunderland and Chmura 2000). According to Allen (1994), the exposure of *Oreochromis aureus* to 0.5 mg Hg l^{-1} caused

a raise in the number of leukocyte and erythrocyte within 24 hours. Gill and Pant (1985) also reported hematological anomalies in *Barbus conchoniis* exposed to 0.18 mg Hg l⁻¹ in acute test.

It can be noticed that the highest bioaccumulation were observed in the organs mainly implicated in metals metabolism. The concentration of cadmium (Cd), lead and copper in tissues was high in the following order; liver > muscles > gills > kidney, while mercury (Hg) concentrations were high in the muscles > liver > gills > kidney. Oladimeji and Offem (1989) noticed that the gills of *Oreochromis niloticus* consistently accumulated higher amount of lead as lead nitrate.

BCF obtained for Pb, Cu, Cd and Hg in the muscles of Tilapia were all greater than 1.00ppm which indicated that the metals were highly bioaccumulated and biomagnified (according to Falusi and Olanipekun 2007). Mercury was the most bioaccumulated and biomagnified of all metals studied in the muscles of the *O. niloticus*, while Cu was the least one.

From the results of this study, the concentrations of different metals investigated in the tissues of Tilapia (gills, liver, kidney and muscle) except copper exceed the acceptable levels proposed for human consumption (USEPA 1995).

The histopathological alterations attributed to the prolonged exposure to heavy metals resulted in respiratory, osmoregulatory and circulatory impairment. These findings were demonstrated by Fernandes *et al.*, (2008). Moreover, Alvarado *et al.* (2006) reported that, the dramatic increase of chloride cells in the gills that produces epithelial thickening of the filament epithelium enhances migration of chloride cells up to the edge of the secondary lamellae and provokes the hypertrophy and fusion of secondary lamellae. These could be considered as unspecific biomarker responses of heavy metals exposure and disturbed health of fish.

Gills showed edema of the primary lamellae; severe edema, hyperplasia, fusion and focal desquamation of the epithelial lining of the secondary lamellae were observed. According to Mallatt (1985), the edema of the gill epithelium is one of the main structural changes caused by the exposure to heavy metals. Our results confirm this lesion of heavy metals exposure. These alterations have been reported for other species exposed to heavy metals particularly Cd (Gardner and Yevich 1970; Karlsson-Norrgrén *et al.* 1985; Pratap and Wendelaar Bonga 1993; Thophon *et al.* 2003) and sometimes referred as a first sign of pathology (Thophon *et al.* 2003). Cellular proliferation in the gill epithelium is also observed in fish exposed to different pollutants as described by Gardner and Yevich 1970 and Thophon *et al.* 2003. Lifting, swelling, and hyperplasia of the gill epithelium could serve as a defense function, as these alterations

increase the distance across which waterborne irritants must diffuse to reach the bloodstream. Lamellar fusion could be protective once it reduces the amount of vulnerable gill surface area (Mallatt 1985). However, branchial responses that serve to slow entry of toxicants have the undesirable side effect of impairing gas exchange. This was described by Benson *et al.*, (1987) who observed a fall in respiratory function of *Notemigonus crysoleucas* exposed to Cd.

The liver showed degeneration of the hepatocytes, congestion of central vein and nuclear pyknosis in the majority of hepatic cells. These findings were apparent as the liver considered the organ of detoxification, excretion and binding proteins such as metallothionein (MTs). The metal-binding proteins were present in the nuclei of hepatocytes suggested that the increase in the cell damages (De Smet and Blust 2001). Similar results were observed by Van Dyk (2003) and Mela *et al.* (2007). Liver of fish is sensitive to environmental contaminants because many contaminants tend to accumulate in the liver and exposing it to a much higher levels than in the environment, or in other organs (Heath 1995).

Pandey *et al.*, (1994) described the alterations in liver and intestine of *Liza parsia* exposed to Hg Cl₂ (0.2 mg Hg l⁻¹) for 15 days. Similarly, Oliveira Ribeiro *et al.* (2002) reported serious injuries in gills and olfactory epithelium of *Salvelinus alpinus* exposed to 0.15 mg Hg l⁻¹.

Similar alterations in muscles and kidney of Tilapia were observed in several species of fish exposed to heavy metals and these alterations were described by Oliveira Ribeiro *et al.* (2002), Jiraungkoorskul *et al.* (2003), Thophon *et al.* (2003) and Gupta and Srivastava (2006).

The result indicates that the heavy metal contamination definitely affects the aquatic life of the fresh water fish. Hence, a scientific method of detoxification is essential to improve the health of these economic fish in any stressed environmental conditions. However, the high concentrations of the analyzed metals in the whole body tissues investigated could be due to the storage role played by these tissues.

Fish contaminated by heavy metals suffers pathological alterations, with consequent inhibition of metabolic processes, hematological changes, and decline in fertility and survival.

It can be conclusively deduced from this study that fish has the tendency to bioaccumulate heavy metals in a polluted environment. Since virtually all metals investigated were found in higher concentration, so government should intact laws that will ensure that industries make use of standard waste treatment plants for the treatment of their wastes before they are being discharged into water bodies.

Acknowledgements

This research was sponsored by Department of Animal Hygiene and Environmental Sanitation (Faculty of Veterinary Medicine, Cairo University). We thank Dr. Kawkb A. Ahmed, Dept. of Pathology, Faculty of Veterinary Medicine, Cairo University for technique assistance.

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2/9/2010