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EFFECT OF FEED CONTAMINATION ON THE PRODUCTIVE PERFORMANCE AND BODY COMPOSITION OF Nile Tilapia (*Oreochromis niloticus*) fingerlings

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ABSTRACT

The present experiment was conducted to study the effect of mercury and lead contaminated diets on the productive performance and histological changes of Nile tilapia *(Oreochromis niloticus).*

A number of 315 Nile tilapia fingerlings were equally distributed among 21 glass aquarium (15 fish each) of 60 liter each (60 x 40 x 25 cm).

Seven experimental diets were tested. T1 was considered as the control diet (uncontaminated diet). T2, T3 and T4 diets were contaminated with mercury of 5, 10 and 15 mg/Kg diet, respectively. While T5, T6 and T7 diets were contaminated with lead of 5, 10 and 15 mg/Kg diet, respectively. By the end of the experiment (10 weeks) growth performance and feed utilization parameters of the experimental fish were calculated. Also all fish were killed for histological studies of gill and liver at the end of the trial.

Results indicated that as the level of mercury or lead increased in the diets, average daily gain (ADG), specific growth rate (SGR) and protein efficiency ratio (PER) decreased. Feed conversion ratio (FCR) increased. Negative values of the former parameters were obtained for the 10 and l5 mg/kg diets for both mercury and lead contaminated diets. Also, mercury and lead concentrations in muscles and liver have increased as a result of increasing their concentrations in diets. Abnormal histological changes of gill and liver were observed as the levels of mercury and lead increased in the diets.

Keywords: Toxicity, pollutants, mercury, lead, Nile tilapia (Oreochromis niloticus)

INTRODUCTION

Fish constitute to be an important source of protein for many people throughout the world. Metals are natural constituents of the fresh water environment. Some metals are essential for life, some are merely beneficial while many are highly toxic (Kalay *et al.,* 1999).

Over the last few decades, there has been growing interest in determining heavy metal levels in the marine environment and their effects on fish performance and the body composition (Tariq *et al., 1993).*

There is an increasing awareness of the potential hazards that exist due to the contamination of freshwater impoundments by toxic metals associated with the mining industry (Kelly, 1988; Queksy *et al., 1998;* Feather and Koen *1975).*

Toxicological and environmental studies have prompted interest in the determination of toxic elements in food. The ingestion of food is an obvious exposure mean to metals, not only because of contaminated

foodstuffs but also because of contamination during processing (Steve HaiL, 1995 ;*).*

Mercury and lead are the most important pollutants because their effects on marine organisms and for their potentially hazardous to human. Mercury and lead were selected because they are known to be accumulated in the environment and in fish, and they cause serious adverse health effects if consumed. Mercury enters the environment through the leaching of soil due to acid rain, coal burning, or industrial, household, and mining wastes which cause damage to nervous system, kidneys and vision. While lead enters the environment through paint, mining wastes, water from lead pipes and automobile exhaust which cause damage to kidneys, nervous system, learning ability, ability to synthesize protein, and nerves and red blood cells (Du Preez *et al.,* 2003). In some instances, fish catches were banned for human consumption because their total mercury content exceeded the maximum limits recommended by the Food and Agriculture Organization (FAO) and the World Health Organization (WHO). Also, the likelihood of mercury toxicity from fish consumption has been identified in other parts of the world (Rose *et al .,*1999; Inkship *and* Piotrowsk ,1985; Piotrowsk and Inkship, *1981).*

Therefore, the objective of the present work is to study the effect of mercury and lead contaminated feed on productive performance and histological changes of Nile tilapia *(Oreochromis niloticus)* fingerlings.

MATERIALS AND METHODS

This experiment was carried out at the department of animal production, Faculty of Agriculture, Ain Shams University, Egypt for a period of 10 weeks, using Nile tilapia fingerlings *(Oreochromis niloticus)* of 10g average body weight. The experiment was conducted in an open system consisting of 21 glass aquarium, 3 per each treatment of 60 liter each. Each aquaria was stocked with15 fish.

Experimental design:

Seven experimental diets were tested. T1 was considered the control diet (uncontaminated diet). T2, T3 and T4 diets were contaminated with mercury of 5, 10 and 15 mg/Kg diet, respectively. While T5, T6 and T7 diets were contaminated with lead of 5, 10 and 15 mg/Kg diet, respectively. Formulation and chemical composition of the experimental diets are shown in Table (1).

Fish were fed daily at 3% of live body weight for 10 weeks. At the 7^{th} day of each week, fish in each aquarium were weighed and the quantity of the feed offered was readjusted for next week. By the end of the experimental period, growth performance of studied fish and feed utilization parameters were calculated, and also all fish were killed for histological studies of gill and liver.

Chemical composition of the experimental diets and fish muscles, and liver were determined according to AOAC 1990**.** According to the data of body weight, the following parameters were calculated:

Average daily gain (ADG): ADG= (w_2-wt_1)/t

Specific growth rate (SGR): SGR= (Ln wt_2- Ln wt_1) x100/t , where

wt_1 = initial weight(g), wt_2 = final weight(g), Ln = Natural logarithm,

t = Experimental period (days).

Feed conversion ratio (FCR): Total feed intake / final body weight - initial body weight

Protein efficiency ratio (PER): Final body weight - initial body weight / protein intake

Histological examinations:

According to Mello and Vidal (1980), fresh samples were taken immediately after anatomical procedures from Gill and liver. The samples were fixed in 10% formalin dehydrated in ascending grades of ethyl alcohol, cleared in xylene, saturated by soft paraffin wax (m.p.50). Then embedded in hard paraffin wax (m.p.55) and sectioned at 4-5 thickness using a rotary microtome, then stained with hematoxylin and eosin.

Statistical analysis:

The obtained data were statistically analyzed according to Steel and Torrie (1980) using SAS program (1998). Differences between means were tested for significance according to Duncan's multiple range test (Duncan, 1955).

Table(1):Experimental diets formulation and their chemical composition (g\100g diet).

Ingredients	T1 (Control)	T2	T3	T4	T5	T6	T7
Fish meal (72%cp)	10.7	10.7	10.7	10.7	10.7	10.7	10.7
Meat meal (50%cp)	11	11	11	11	11	11	11
Soybean(48%cp)	9	9	9	9	9	9	9
Gluten(60%cp)	8	8	8	8	8	8	8
Corn	31	31	31	31	31	31	31
Rice bran	26	26	26	26	26	26	26
Oil fish	1	1	1	1	1	1	1
Sun flower Oil	1	1	1	1	1	1	1
Salt	2	2	2	2	2	2	2
Vitamin Mineral mix	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Dry matter	89.90	90.40	90.40	90.40	89.90	90.20	90.00
Crude protein	30.00	29.89	29.89	29.87	29.85	29.88	29.89
Ether extract	8.14	7.50	7.75	7.00	8.00	7.32	7.75
Crud fiber	3.73	3.87	3.78	4.59	3.83	3.52	4.30
Ash %	12.24	12.68	12.54	12.82	12.14	12.54	12.89
Nitrogen free extract (NFE)[1]	35.79	36.46	26.44	36.12	26.08	26.94	35.17
Digestible energy Kcal/100g diet2	294.84	289.87	292.08	284.59	293.41	298.16	289.54

[1]NFE = 100- (Moisture + crude protein + Ether extract + Ash + Fiber).

[2] Digestible energy based on 5.0 Kcal/g protein, 9.0 Kcal/g lipids, and 2.0 Kcal/g carbohydrates (Wee and Shu, 1989)

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RESULTS AND DISCUSSION

Effects of mercury contamination on growth rate and feed utilization parameters

The results presented in Table (2) clearly showed that as the level of dietary mercury increased the growth of fish significantly (p<o.o.1) decreased. Negative growth rate were observed at 10 and 15 mg mercury /kg diet. The present results were in good agreement with those obtained by *Sivakami et al.* (1995) who reported that exposure of fish to mercury led to significant decrease in both feed intake and growth rate. Decreased growth rate may be due to the greater expenditure of energy towards overcoming the stress caused by the heavy metals toxicity. They stated that high concentrations of inorganic mercury at a level of 10 g/kg diet resulted in severe pathological changes in the intestine. *Berntssen et al.* (2004) reported that inorganic mercury accumulation in kidney tissue had significantly increased in fish fed 10 mg Hg\ kg for a period of 4 months. *Friedmann et al.* (1996), observed an increase in production of stress hormones as well as a reduction in growth of juvenile walleye *(Stizostedion vitreum)* fed mercury contaminated diets of 0.1 mg/kg diet. Andrew *et al.,* (1996) stated that dietary mercury significantly impaired both growth and gonadal development in males which was reflected as reduced fish length, weight and gonad somatic index after fish fed on diets contaminated with 1.0 μg Hg/g diet. Friedmann *et al.* (1996) showed that even low levels of dietary mercury inhibited growth of juvenile walleye *(stizostedion vitreum).* There were negative relationships between fish sizes and metal levels in most cases.

Table (2) indicated that the control treatment had the best feed conversion ratio, while as long as mercury level increased in the diet, the feed conversion ratio increased. Their results might be due to the retarded growth rate of fish fed on diets contaminated with mercury. Ayyat and El-Marakby (2005) stated that feed conversion ratio' values of fish fed on diets contaminated with mercury at 50 or 100 mg/kg were lower by 9.8 and 16.4%, respectively, compared to those obtained from fish fed on uncontaminated diets. Table (2) showed gradual decrease in protein efficiency ratio values as the level of mercury in the diet increased .The highest value (p<0.01) was recorded in T1 (2.4573) whereas The lowest value (-2.2350) was recorded for T4. These results are positively correlated with those of growth rate.

Inhibition of protein synthesis could be due to the fact that inorganic mercury is known to reduce the active absorption of amino acids and sugars (Miller., 1981; Farmanfarmaian *et al*., 1985; Chauncey *et al*., 1988). Gill *et al*. (1990) also reported that the high concentrations of mercury inhibit intestinal enzymes.

Effects of lead contamination on growth rate and feed utilization parameters

The results presented in Table (3) showed that as the level of dietary lead increased, fish growth decreased. The highest ADG was observed for T1, whereas the lowest one was recorded for T7. Negative ADG values were recorded at 10 and 15 mg lead /kg diet.

Table (2): Effect of dietary mercury levels on growth performance of tilapia and feed utilization parameters

Treatment	ADG	SGR	PER	FCR
T1	$0.6449^{a} \pm 0.0038$	$2.5337^{a} \pm 0.1093$	$2.4573^{a} \pm 0.1255$	$1.2193^{b} \pm 0.25425$
T2	$0.1676^{b} \pm 0.0038$	$1.0690^{c} \pm 0.1093$	$1.1314^{b} \pm 0.1255$	$2.6730^{a} \pm 0.25425$
T3	$-0.0683^{c} \pm 0.0038$	$-1.0380^{c} \pm 0.1093$	$-0.9693^{c} \pm 0.1255$	$-1.0170^{d} \pm 0.25425$
T4	$-0.1156^{d} \pm 0.0038$	$-2.7230^{d} \pm 0.1093$	$-2.2350^{d} \pm 0.1255$	$-3.1200^{c} \pm 0.25425$

Values within the same column with different superscripts are significantly different (p<0.01)

Table (3): Effect of dietary lead levels on growth performance of tilapia and feed utilization parameters

Treatment	ADG	SGR	PER	FCR
T1	$0.6449^{a} \pm 0.0007$	$2.5336^{a} \pm 0.0005$	$2.4573^{a} \pm 0.0007$	$1.2193^{b} \pm 0.0005$
T5	$0.0623^{b} \pm 0.0007$	$0.5560^{b} \pm 0.0005$	$0.5326^{b} \pm 0.0007$	$5.6556^{a} \pm 0.0005$
T6	$-0.0550^{c} \pm 0.0007$	$-0.7780^{c} \pm 0.0005$	$-0.7050^{c} \pm 0.000$	$-4.2846^{d} \pm 0.0005$
T7	$-0.0886^{d} \pm 0.0007$	$-1.6606^{d} \pm 0.0005$	$-1.3960^{d} \pm 0.0007$	$-2.1570^{c} \pm 0.0005$

Values within the same column with different superscripts are significantly different (p<0.01)

Eisler, (1988); *Burger* and *Gochfeld*, (2000) stated that Lead toxicity disturb metabolism and consequently fish growth decrease. *Amusan et, al. (2002)* found that *Limicolaria jlammea* which were treated with high lead concentrations showed a high mortality rate. The present data clearly showed gradual decrease in protein efficiency ratio as the level of lead in the diet increased.

The highest value (p<0.01) was recorded for the control treatment (2.4 5 73) whereas the lowest value (-I. 396) was recorded for treatment (7). These results were positively correlated with those of growth rate. The present data clearly indicated that the control treatment had the best significant (p<0.01) feed conversion. Increasing lead level in the diet led to deterioration of feed conversion ratio. These results are mainly due to the negative average daily gain of fish feed on diet containing lead.

Effect of dietary mercury levels on its concentrations in tilapia muscles and liver

The results presented in Table (4) clearly showed a significant (p<0.01) gradual increase in muscle content of mercury as the level of mercury increased in the fish diet. The same trend was recorded for liver mercury. Results of Table (4) and fig (10) clearly indicated a significant

($p<0.01$) positive relationship between the concentration of liver mercury and the level of diet mercury.

Effect of dietary lead levels on its concentrations in tilapia muscles and liver

The results presented in Table (5) clearly also showed a significant ($p<0.01$) gradual increase in muscle lead as the level of lead increased in the fish diet. There was a significant ($p<0.01$) positive relationship between the concentration of liver lead and the level of diet lead. Widianarko *et al.,* (2000).investigated the relationship between metal (Pb, Zn, cu) concentration and fish *(poecilia reticulate)* size and found that there was significant decline in lead concentration with the increase in size. There were negative relationships between fish sizes and metal levels in most cases (Canli *et al., 1998).*

Histological study:

Effect of dietary mercury and lead levels on histopathological changes of tilapia gill

The histopathological changes of gill as affected by mercury and lead are shown in Fig (1) and (2), respectively. The histological alterations in the gills of fish Nile tilapia affected by mercury and lead include dilation of the marginal channel, hyperplasia of the epithelial cells and lifting of the lamellar epithelium. Coutinho and Gokhale (2000) found epithelial lifting in the gills of carps *(Cyprinus carpio)* and tilapias *(Oreochrom is mossambicus)* exposed to the effluents of a wastewater treatment plant. Engelhardt *et al.* (1981) observed epithelial lifting and lamellar fusion in rainbow trouts *(Oncorhynchus mykiss)* exposed to petroleum residues. Similar alterations in the gills have also been reported in the fish exposed to metals alterations are non-specific and may be induced by different types of contaminant.

Table (4): Effect of dietary mercury levels on its concentrations in tilapia muscles and liver

Treatment	Mercury in muscle	Mercury in liver
Treatment 1	$0.0003^{C} \pm 0.2897$	$0.000^{d} \pm 0.4791$
Treatment 2	$1.3010^{b} \pm 0.2897$	$1.3010^{c} \pm 0.4791$
Treatment 3	$7.3290^{a} \pm 0.2897$	$7.3290^{b} \pm 0.4791$
Treatment 4	$7.7317^{a} \pm 0.2897$	$9.3167^{a} \pm 0.4791$

Values within the same column with different superscripts are significantly different ($p<0.01$)

Table (5): Effect of dietary lead levels on its concentrations in tilapia muscles and liver

Treatment	Mercury in muscle	Mercury in liver
Treatment 1	$0.000^{d} \pm 0.0055$	$0.000^{d} \pm 0.2099$
Treatment 5	$0.1355^{c} \pm 0.0055$	$1.2905^{c} \pm 0.2099$
Treatment 6	$0.3483^{b} \pm 0.0055$	$5.1155^{b} \pm 0.2099$
Treatment 7	$0.6340^{a} \pm 0.0055$	$6.1340^{a} \pm 0.2099$

Values within the same column with different superscripts are significantly different ($p<0.01$)

The gills perform important functions in fish, such as respiration, osmoregulation and excretion. They remain in close contact with the external environment, and particularly sensitive to changes in the quality of the water (Poleksic and MitrovicTutundzic, 1994; Mazon *et al.,* 2002*;* Fernandes and Mazon, 2003).

High levels of mercury and lead caused alterations in gill of fish like epithelial lifting, hyperplasia and hypertrophy of the epithelial cells, besides partial fusion of some secondary lamellae. These are examples of defense mechanisms, and cause an increase of the distance between the external environment and the blood, thus serve as a barrier to the entrance of contaminants (Mallatt, 1985; Hinton and Lauren, 1990; Poleksic and Mitrovic-Tutundzic, 1994; Fernandes and Mazon*.,* 2003).

As a consequence of the increased distance between water and blood due to epithelial lifting, the oxygen uptake is impaired. However, fish have the capacity to increase their ventilation rate, to compensate low oxygen uptake (Fernandes and Mazon, 2003). Most part of the gill lesions caused by sublethal exposures affects lamellar epithelium (Hinton and Lauren, 1990). Some alterations in blood vessels may also occur, when fish suffer from severe type of stress. In this case, damaged pillar cells can result in an increased blood flow inside the lamellae, causing dilation of the marginal channel, blood congestion or even an aneurysm (Takashima and Hibya, 1995; Rosety-Rodrguez *et al.,* 2002).

The formation of an eurysm is related to the rupture of the pillar cells (Heath, 1987; Martinez *et al.,* 2004) due to a bigger flow of blood or even because of the direct effects of contaminants on these cells. This is a severe type of lesion, recovery from which is possible, but more difficult than the epithelial changes (Poleksic and Mitrovic- Tutundzic, 1994).

Winkaler *et al* (2001) found anomalies such as hyperplasia, hypertrophy, dilation of the marginal channel and aneurysms in another Neotropical fish, Astyanax altiparanae, collected in Cambe stream, which corroborates the hypothesis that the water of this stream is really contaminated and that exposure to this water causes structural damage to the fish gill.

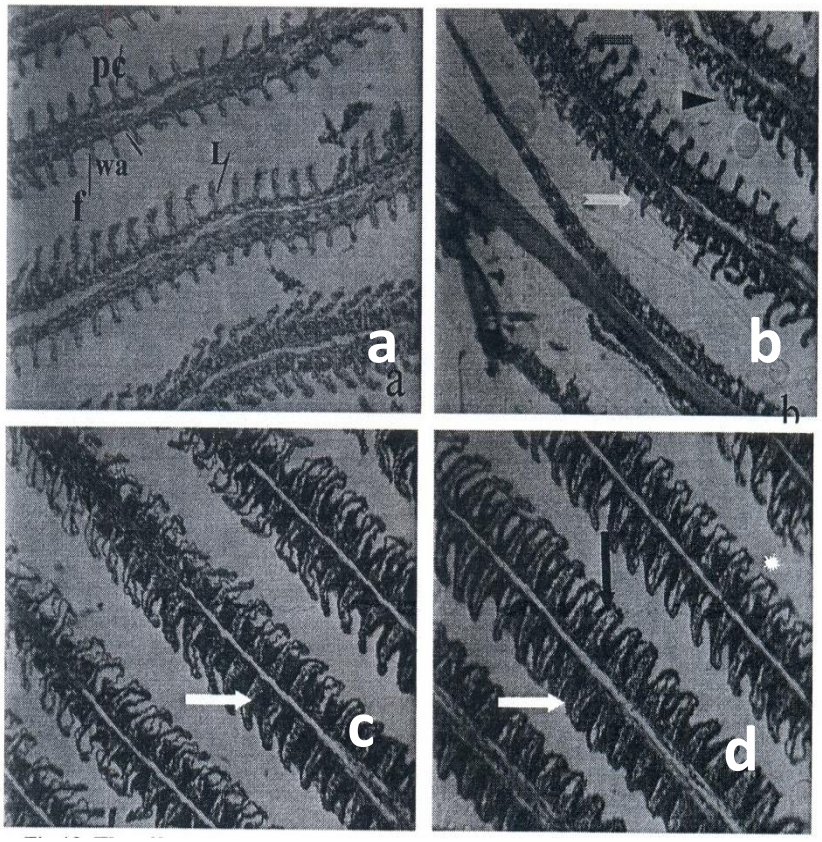

Fig (1) The effect of mercury on the gill of tilapia

Photomicrographs of the gill of *Oreochromis niloticus,.* a) Normal aspect of the gill, showing the filament (f), the lamellae (L), the water channel (wa), a pillar cell (pc); b)(O.5mg\kgHg)lamellae with the marginal channel dilated (black arrow), hyperplasia of the epithelial cells (gray arrow) and epithelial lifting (arrowheads);c) (lOmg\kgHg) fusion of lamellae (white arrow); d)(l5mg\kgHg) lamellar partial fusion of some lamellae (white arrow) and hypertrophy of the lamellar epithelium (black arrow) and epithelium rupture with hemorrhage (*)

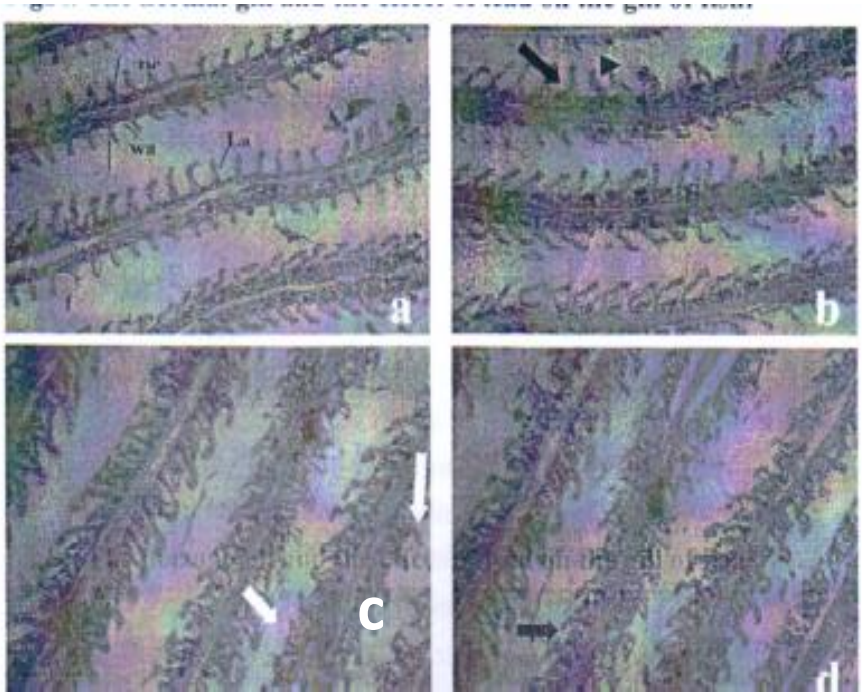

Fig (2) The effect of lead on the gill of tilapia

Photomicrographs of the gill of *Oreochromis niloticus,.* a) Normal aspect of the gill, showing the filament (f), the lamellae (L), the water channel (wa), a pillar cell (pc); b)(O.5mg\kgHg)lamellae with the marginal channel dilated (black arrow), hyperplasia of the epithelial cells (gray arrow) and epithelial lifting (arrowheads);c) (lOmg\kg pb) fusion of lamellae (white arrow); d)(l5mg\kg pb) lamellar partial fusion of some lamellae (white arrow) and hypertrophy of the lamellar epithelium (black arrow) and epithelium rupture with hemorrhage (*) .

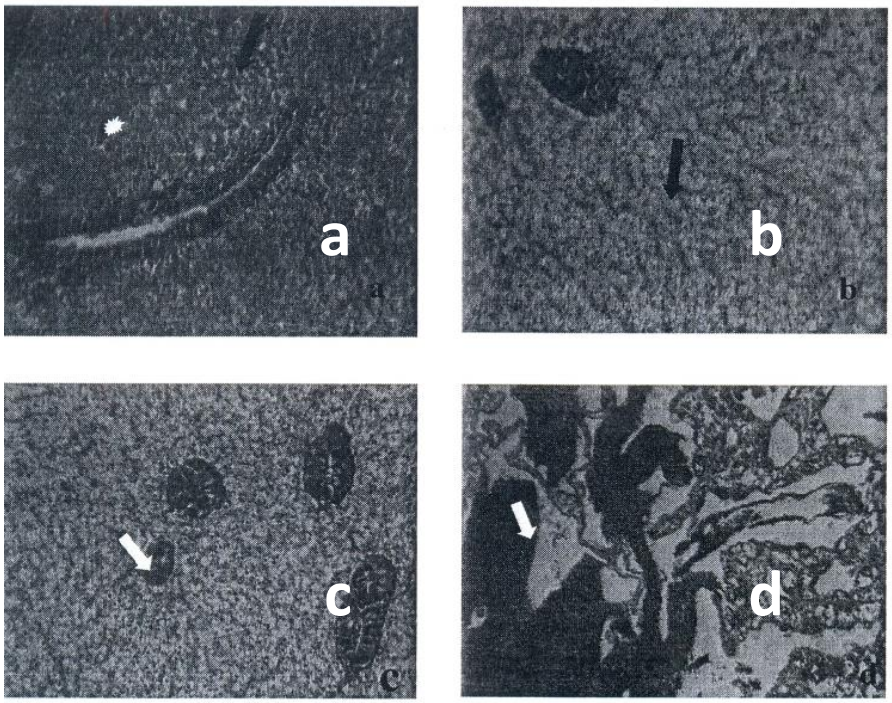

Fig (3) The effect of mercury on the liver of tilapia

Photomicrographs of the liver of. *Oreochromis niloticus* a) normal hepatic tissue, showing hepatocytes with granular cytoplasm (*); b) (5mgpb\kg) hepatocytes with irregular shaped nucleus (black arrows); c) (IOmgpb\kg) melanomacrophages aggregate, close to a vessel (white arrow) and cytoplasm vacuolation d) (15mgpb\kg)irregular-shaped nuclei, cytoplasm and nuclear degeneration

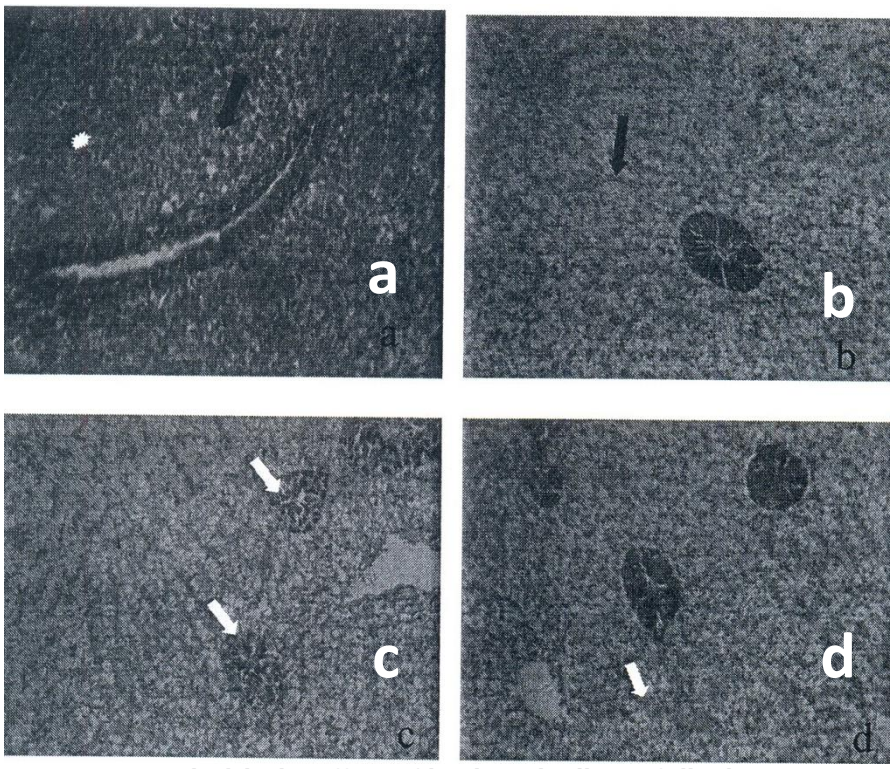

Fig (4) The effect of lead on the liver of tilapia

Photomicrographs of the liver of. *Oreochromis niloticus* a) normal hepatic tissue, showing hepatocytes with granular cytoplasm (*); b) hepatocytes with irregular shaped nucleus (black arrows),; c) melanomacrophages aggregate, close to a vessel (white arrow) and cytoplasm vacuolation d) irregular-shaped nuclei.

Effect of dietary mercury and lead levels on histopathological changes of tilapia liver

The histopathological changes of liver compared with normal liver and as affected by mercury and lead are shown in figs. (3) and (4) respectively.

The main alterations found in the liver were irregular-shaped nuclei, nuclear hypertrophy, nuclear vacuolation and the presence of eosinophilic granules in the cytoplasm, bile stagnation was identified as brownish-yellow granules in the cytoplasm. Cytoplasm and nuclear degeneration was also very common; melanomacrophages were identified as rounded aggregates of cells containing dark-yellowish granules of various sizes, normally close to the vessels.

These maybe due to the fact that liver is the organ associated mostly with the detoxification and biotransformation process is the liver, (Van der Oost *et*

al., 2003) it is also one of the organs most affected by contaminants in the water Rodrigues and Fanta, (1998).

Abnormalities such as irregular shaped hepatocytes, cytoplasm vacuolation and nucleus in a lateral position, close to the cell membrane, were also described in the siluriform *Carydaras paleatus* contaminated by organophosphate pesticides (Fanta *et al.,* 2003). Vacuoles in the cytoplasm of the hepatocytes can contain lipids and glycogen, which are related to the normal metabolic function of the liver. Depletion of the glycogen in the hepatocytes is usually found in stressed animals (Hinton and Lauren, 1990; Wilhelm Filho *et al.,* 2001), because the glycogen acts as a reserve of glucose to supply the higher energetic demand occurring in such situations (Panepucci *et al.,* 2001).

Pacheco and Santos; (2002) found a vacuolization increase in the hepatocytes as a signal of degenerative process that suggests metabolic damage, possibly related to exposure to contaminated water. The histopathological changes in the liver cause metabolic problems as well. Evidence for this is the bile stagnation in liver of most fish studied. This lesion characterized by the remains of the bile in the form of brownish-yellow granules in the cytoplasm of the hepatocytes. They stated that the bile is not being released from the liver. This accumulation of bile indicates possible damage to the hepatic metabolism (Fanta *et al*., 2003). The presence of bile stagnation and melanomacrophages in great quantity in the livers is strong evidence that these organs suffered structural and metabolic damage due to exposure to the Cambe stream water, reinforcing the idea that this environment is really impaired (Marina *et al., 2007).*

An increase in the density of the melanomacrophage aggregates, as observed in the liver of P. lineatus in this study, is generally related to important hepatic lesions (Panepucci *et al.,* 2001), such as degenerative and necrotic processes. This was related, in Pleuronectes american us, to contamination with P AHs (polycyclic aromatic hydrocarbons) and pesticides in urban areas on the USA coast (Chang *et al.,* 1998).The function of the melanomacrophages in the liver of fish remains uncertain, but some studies have suggested that it is related to destruction, detoxification or recycling of endogenous and exogenous compounds (Haaparanta *et al., 1996).*

CONCLUSIONS

The present study demonstrated the negative effects of mercury and lead contaminated diets on performance and histological changes of Nile tilapia. The high level of mercury and lead caused severe growth reduction, high mortality and abnormal histological changes in the gill and liver, which are reflections of the impairment of the fish metabolic functions. It is therefore recommended that contaminated water and feed should be avoided in order to achieve high performance, no negative histological changes in the fish and ensure safe food for human. Further studies are needed to determine the minimum toxins levels that may affect the fish and the consumer.

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أثر التلوث الغذائي على الأداء الإنتاجي وتركيب الجسم في إصبعيات اسماك البلطي النيلي

دينـــا عبـــاس عبـــدالباري١ ، محمـــد فتحـــي عثمـــان٢ ، حمـــدي محمـــد خطـــاب٢ و أشرف هاشم جمعه١

١- المركز الإقليمي للأغذية والأعلاف ـ مركز البحوث الزراعية ـ الجيزة ـ مصر

٢- قسم الإنتاج الحيواني ـ كلية الزراعة ـ جامعة عين شمس ـ شبرا الخيمة ـ القاهرة ـ مصر

الهدف من هذه الدراسة تقدير تراكم العناصر الثقيلة في أسماك البلطي النيلي التي تعرضت للتلوث بعنصري الزئبق والرصاص في العليقة وأثرها على المظاهر الإنتاجية والتغيرات الهستولوجية.

تم تقسيم الأسماك لسبعة معاملات لكل معاملة ثلاث أحواض زجاجية تحتوي كلا منها على خمسة عشر سمكة بمتوسط وزن ١٠ جرام وتمت تغذية الأسماك على عليقة تحتوي على ٣٠% بروتين بواقع ٣% من وزن الجسم . تم إختبار مستويات مختلفة لكل من الزئبق والرصاص في العليقة (٥ ، ١٠ ، ١٥ ملي جرام زئبق / كجم عليقة) و (٥ ، ١٠ ، ١٥ ملي جرام رصاص / كجم عليقة) للعلائق من ٢ إلى ٧ على التوالي، ومقارنتها بعليقة خالية من عنصري الزئبق والرصاص.

تمت دراسة التأثيرات المختلفة لكل من الزئبق والرصاص على متوسط الوزن ومعدل النمو النوعي ومعدل الإستفادة من البروتين ومعدل التحويل الغذائي لوحظ أنه بزيادة مستويات كلا من الزئبق والرصاص يقل معدل الزيادة اليومية ومعدل النمو النوعي ومعدل الاستفادة من البروتين مقارنة بالعليقة الخالية من عنصري الزئبق والرصاص بينما يزداد معدل التحويل الغذائى.

أخذت عينات من الكبد والخياشيم للدراسة الهيستولوجية التي أوضحت أنه بزيادة نسب كلا من الزئبق والرصاص في العلائق تحدث تغيرات غير طبيعية لكل منهما وتزداد تلك التغيرات بزيادة تركيز هذين العنصرين.