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EFFECT OF AFLATOXIN CONTAMINATED FEEDS ON SOME FRESH WATER FISHES

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SUMMARY

Fingerlings of four species of fresh water fish namely Nile tilapia (*Oreochromis niloticus*), red tilapia (hybrid of *O. niloticus x O. mossambicus*), grey mullet (*Mugil cephalus*) and common carp (*Cyprinus carpio*) were graded into homogeneous size and kept in circular tanks. Healthy fishes were selected and used in four experiments to study: the sensitivity of tilapia and common carp to purified aflatoxin B1 (Experiments 1 and 2), and the effects of dietary aflatoxin on survival rate , growth performance, body composition, feed and nutrient utilization of fish (Experiments 3 and 4).

Pathogenicity of aflatoxin B1(AF B1) on Nile tilapia and common carp were judged by mean death time (MDT) and minimum lethal dose (MLD50) which were conducted by an intraperitoneal(IP) administration of doses of :0,100,200 and 400ng AF B1 and doses of 0,400,800 and 1200 ng AF B1/0.1 ml HPLC Methanol /fish for ten successive days in experiments 1 and 2 respectively.

The results of the 1*st* experiment showed that the low doses of AF B1 had no effects on the survival of fish. However, results in the 2*nd* experiment showed that 80% of common carp died on the 6 *th* day (150 hours), while only 40% of Nile tilapia were dead on the 8*th* day (190 hours). Thus the MLD50 in common carp was 80 ug/kg body weight and MDT was 150 hours. Gross lesions were enlarged pale liver and distended gall bladder.

The effects of different concentrations of dietary aflatoxin (0,476,952 and 1905 ug AF/kg diet) on survival rate, growth performance, feed and nutrient utilization of common carp and Nile tilapia in growing and feeding experiment were conducted for 14 weeks (Experiment 3). The survival rates were greatly decreased in aflatoxin treated groups of common carp as compared to the tilapia. Growth performance, feed and nutrient utilization were significantly ($P< 0.05$) decreased in aflatoxin treated groups. Body crude protein in tilapia was not affected with incerasing levels of dietary AF, however decreased in carp.

The effects of the high level of dietary aflatoxin (1905 ug/kg diet) on the survival rate, growth performance , feed and nutrient utilization were studied in growing and feeding experiment for 12 weeks by using Nile tilapia,red tilapia, common carp and grey mullet (Experiment 4). Survival rates were only 20% during the first 11 days of

the experiment for grey mullet and 82.5, 87.5 and 95% for common carp, red tilapia and Nile tilapia, respectively. However, there were no significant differences between AF- treated and untreated groups on growth performance between all tested fish species. Feed and nutrient utilization were significantly (P< 0.05) decreased with AF- treatment as compared with untreated groups (control).

The present results clearly showed that grey mullet is highly sensitive to aflatoxin followed by common carp, red tilapia and Nile tilapia, respectively. Aflatoxin treatment decreased the feed consumption, growth performance, feed and nutrient utilization.

Keywords : Fish, aflatoxin, contaminated feed, growth, nutrient utilization.

INTRODUCTION

Aflatoxins (AF) are a group of hepatotoxic compounds produced exclusively by *Aspergillus flavus* and *A. parasiticus* growing on feedstuffs. They were first discovered in 1960 as a result of turkey "X" disease which was responsible for the deaths of over 100000 turkey poults in England (Stevens *et al.*, 1960 and Wanrop, 1960). Aflatoxins (AF) are the most commonly occurring mycotoxins (AF B1,B2,Gl and G2) produced by fungi *Aspergillus*. Aflatoxin B1 (AF B1) is the most common and biologically active component (Busby and Wogan,1981), however AF G1 cannot be ignored in any assessment of the risk of natural contamination.

Abdel- Hamid (1985) investigated a total of 95 various Egyptian feedstuffs for presence of AF (B1,B2,G1 and G2) using TLC. Out of these samples 44.21 % were contaminated with aflatoxin (maize , rice crack , rice germ , rice germ cake, rice bran, wheat bran ,cottonseed cake, peanut and mixed feed for broilers, commercial feeds for layers, calf fattening and dairy animals). A total of 90.48% of the contaminated samples were contaminated with less than 100ppb total aflatoxin. Aflatoxin B1 was present alone so frequently (In 76.19% of the contaminated samples). The relationship between the concentrations of AF B2:G1:B1 were 1.0: 2.3: 22.4, respectively.

In 1960, the widespread occurrence of trout hepatoma was recognized in many hatcheries in the United States (Rucker *et al.*, 1961). Ashley and Halver (1963) found that the fish suffered from aflatoxicosis or hepatoma during early stage of tumor growth appeared normal. Moreover, enlargement of the liver and emaciation (poor growth) were noticed with progress of the disease. Solomon *et al.*, (1965) investigated the effects of some feeding stuffs on the liver of rainbow trout which received diet containing cottonseed meal or toxic groundnut meal with 100 ppm aflatoxin. Hyperplasia of the bile duct and cholangitis were observed in fishes fed with cottonseed meal and groundnut for three and half months,while these changes were absent in the control fishes. The lower dose of AF B1 (2 ug/kg feed) had no effect on body weight, condition, health or physiological state of common carp (*Cyprinus carpio*) and it left no aflatoxin residue in the muscles. In carp fed barley meal with addition of 200 ug AF B1/kg feed, reactive infiltrations in the area of intrahepatic bile ducts, dystrophy of liver tissue, serious dystrophic changes in nerve cells, and kidney damage with appearance of polymorphonuclear elements in tubules were found next to circulation disturbance in organs and tissue (Svokbodova *et al.*, 1982). Jantrarotai and Lovell (1990) found that means for growth rate, haematocrit, haemoglobin

concentration, and erythrocyte count of channel catfish (*Lctalurus punctatus*) fed 10,000 ug AF B1/kg of feed for 10 weeks were significantly lower than those of fish fed 2,154 ug/kg or lower concentration of AF B1. Gross appearance and behaviour of all fish were normal. Gastric glands in the stomach were necrotic and contained infiltration macrophages.

Because the effect of aflatoxin vary widely on different organisms; the effects may be quite different even with very closely related organisms. Although the effect of aflatoxin on some world fish has been investigated, yet its effects on Egyptian fish species is still lacking. Therefore, the aim of the present work is to study :- the susceptibility or sensitivity of Nile tilapia and common carp to purified aflatoxin B1, and the effect of dietary aflatoxin on survival rate, growth performance, body composition, feed and nutrient utilization of Nile tilapia, red tilapia,grey mullet and common carp.

MATERIALS AND METHODS

Four experiments were carried out at the Department of Animal and Fish Production, Faculty of Agriculture (Saba-Basha), Alexandria University.

1. Experimental design

Experiments 1 and 2 were designed to determine the mean death time (MDT) and the minimum lethal dose (MLD50) of purified aflatoxin B1 (AFB1) for Nile tilapia and common carp by intraperitoneal (IP) administration. Experiment 3 was designed to study the effect of different doses of dietary aflatoxin on surival rate, growth performance, body composition ,feed and nutirent utilization of Nile tilapia and common carp after 14 weeks of growing and feeding. Experiment 4 was designed to study the effect of high level of dietary aflatoxin on surival rate, growth performance, body composition, feed and nutrient utilization of Nile tilapia, red tilapia, grey mullet and common carp after 12 weeks of growing and feeding.

2. Experimental fish

Fingerlings of four fish species namely Nile tilapia (*Oreochromis niloticus*), red tilapia *(Oreochromis niloticus x Sarotherodon mossambicus),* grey mullet (*Mugil cephalus*) and common carp (*Cyprinus carpio*) were obtained from Bersik Fish Farm , El- Behera Governorate in May , 1991 . Fishes were graded into homogeneous sizes and were kept in $1m^3$ fiberglass circular tanks (each 1.5 m diamter). Healthy fish were selected for using in the injecting,growing and feeding experiments. At the start of each experiment, fish were weighed and randomly distributed into the experimental aquaria jars.

3. Aquaria

Experimental facilities were glass aquaria each measured 30 x 40 x 100 cm with capacity of 120 liters for each aquarium. However, 105 liters of water was only allowed .

4. Daily management

Fishes were fed twice a day for 6 days a week, for 10 days in experiments 1 and 2, and for 14 and 12 weeks in experiments 3 and 4, respectively. Daily feed allowance

was determined as a percentage of fish live body weight and readjusted biweekly. Feeding rates were 4% and 3% of live body weight of fishes in experiments 1 & 2 and 3 & 4 respectively.

Water temperature was kept at 28±1°C. Accumulated wastes were removed daily from the aquarium by siphoning. Equal amounts of water were replaced with well aerated tap water retained for 48 hours in a net covered storage tank. With wastes removal,about one half of water in each aquarium was changed daily. Continuous aeration was provided throughout air stones by using air pumps.

5. Pathogenicity of aflatoxin

The mean death time (MDT) was carried out as described by Anon (1963) and the minimum lethal dose (MLD50) was calculated after the formula of Reed and Muench (1938).

5.1. Preparation of aflatoxin

Pure crystalline aflatoxin B1 was diluted with High Performance Liquid Chromatography Methanol (HPLC Methanol).

In the 1*st* experiment the levels of aflatoxin B1 intraperitonealy injected were calculated to be 0, 100, 200, 400 ng AF B1/0.1 ml HPLC Methanol, while higher levels (0, 400, 800, 1200 ng AF B1/0.1 ml HPLC Methanol) were used in the 2nd experiment.

Dietary aflatoxin was produced by fermentation of rice grains with *Aspergillus parasiticus* (NRRL 2999) as described by Shotwell *et al.* (1966), modified by West *et al.* (1973). The rice was steamed, dried, ground to fine powder before fermentation. Dietary aflatoxin content of the experimental diets were spectrophotometrically determined by the method of Nabney and Nesbitt (1965) as modified by Wiseman *et al.* (1967).

5.2. Aflatoxin administration (Experiments 1 &2)

Fingerlings of two species of Nile tilapia and common carp (average weight 17.5 and 12.5 g/fish respectively) were used in experiments 1 &2 .Each treatement was conducted in duplicates . Sixten glass aquaria were used. Each aquarium was stocked with ten fishes from each species. Fishes in each aquarium were placed in a dip-net and intraperitoneally (IP) injected with 0.1 ml HPLC Methanol containing different levels of pure AF B1/ fish at 2 p.m. for ten successive days. However, control group was injected with 0.1 ml HPLC Methanol / fish per day only during the same injection period. Mortality during the experimental period (10-days) was recorded.

6. Feeding experiments (Experiments 3 and 4)

In experiment 3 sixteen glass aquaria were used. Each aquarium was stocked with 20 fish/ species (mono-culture) with an average initial weight of 4.3-4.4 g/fish. Fish were fed on the basal diets containing different levels of aflatoxion at 3% of its live body weight , twice daily at 10.00 and 16.00 hr for 14 weeks. At two weeks intervals, fish were weighed and examined for overt signs of aflatoxicosis .

Sixteen glass aquaria were used in experiment 4 too. Each aquarium was stocked with 20 fish of one tested fish species. Average initial weights were 7.1-7.3 g/fish. Fish were fed on the basal diet contaminated with the higher level of aflatoxion at 3%

of its live body weight, twice daily, at 10.00, 16.00 hr for 12 weeks. At 2 weeks intervals, fish were weighed and examined for overt signs of aflatoxicosis. At the end of the experiments 3 and 4 the number and body weight of fish in each aquarium were determined.

6.1. The basal diet

The basal diet was formulated as shown in table 1.

6.2. Preparation of dietary aflatoxin

Aflatoxin was produced through rice fermentation. Weight of rice powder, calculated to yield a final concentration of 476.19, 952.38 and 1904.76 ug AF/kg of feed, were incorporated manually into the diets 2,3 and 4, respectively instead of the same amount of yellow corn in the basal diet. The control diet was kept free from aflatoxin (Experiment 3). In experiment 4, the higher level of AF (1904.76 ug AF/kg feed) was compared with the control diet only.

Table 1. Feed ingredients and proximate chemicl analysis of the basal diet.

Item	%
Feed ingredient (%)	
Fish meal	30.0
Soybean meal	20.0
Yellow con	45.6
Bone meal	2.0
Maize oil	2.0
Vitamin mixture *	0.2
Mineral mixture*`	0.2
Chemical analysis (%):	
Dry matter (DM)	93.5
% on DM basis:	
Crude protein (CP)	31.57
Ether extract (EE)	7.62
Ash	9.40
Crude fiber (CF)	2.53
Nitrogen free extract (NFE)	48.88
Gross energy **(Kcal/g DM)	4.56

* Each kilogram of diet contained : 20000 IU vit. A, 2000 IU vit. D_3 and 400 IU vit. E. and 40 g manganese, 45 g zinc, 3 g copper, 0.3 g iodine, 0.1 g selenium and 30 g iron.

** Gross energy **(Kcal/g DM) was calculated according to NRC, 1983 using the calorific values: 5.65, 9.44 and 4.11 Kcal /g diet DM for protein, fat and carbohydrate, respectively.

7. Analytical methods

Chemical analysis of the experimental diets and fish body were done according to the method described in Association of Official Analytical Chemists (A.O.A.C. 1980).

8. Criteria Indices

The following indices were calculated :- average daily gain (ADG), specific growth rate (SGR %), feed/gain ratio (FCR), protein efficiency ratio(PER),protein productive

value (PPV %) and energy utilization (EU %) were calculated according to the following equations:
- average daily gain (ADG).
ADG = Wt-Wo/n.
Where
Wo: the initial fish weight at the start of the experiment.
Wt: the final fish weight at the end of the experiment
n : the duration period of the experiment in days.
- specific growth rate (SGR %).
SGR% = (Ln Wt-Ln Wo) 100/n.
Where:
Ln: the natural logarithm
Wo: the initial fish weight at the start of the experiment.
Wt: the final fish weight at the end of the experiment.
n : the duration period of the experiment in days.
- feed conversion ratio (FCR).
FCR = Dry matter intake (g)/weight gain (g).
- protein efficiency ratio (PER).
PER = Weight gain (g)/protein intake (g).
- protein* productive value (PPV %).
PPV % = (P-Po)100/Pi.
Where:
P : the protein content in whole fish body at the end of the experiment.
Po : the protein content in whole fish body at the start of the experiment.
Pi: the protein in feed intake.
* : protein was determined as nitrogen X 6.25
- energy utilization (EU %).
EU % = (E-Eo) 100/Ei.
Where:
E: the energy in whole fish body (kcal) at the end of the experiment.
Eo: the energy in whole fish body (kcal) at the start of the experiment.
Ei: the gross energy in feed intake (kcal).

9. Statistical analysis

experimental results were statistically analyzed according to Snedecor and Chochran (1971) and multiple range method of Duncan (1955) .

RESULTS AND DISCUSSION

Experiments 1 and 2 were conducted to study the susceptibility of Nile tilapia and common carp to purified AF B1 as judged by mean death time (MDT) and minimum lethal dose (MLD50) through the intraperitoneal(IP) administration. The results of the 1*st* experiment showed that the low doses of aflatoxin (0,100,200,400 ng AF B1/0.1 ml HPLC Methanol /fish /day for 10 successive days) had no effects on the survival rates of fishes . However the results of the 2*nd* experiment (Table 2) show the susceptibility of the tested fish species to a high dose of AF B1 (1200 ng AF B1/fish) .Values of MDT were 150 and 192 hrs in common carp and Nile tilapia, respectively. Moreover, MLD50 in common carp (80 ug/kg body weight) was higher than in Nile

tilapia. Mortality rates observed during the experimental period (10 days) were 80% and 40 % in common carp and Nile tilapia,respectively (Table 3)

Table 2. Mean death time (MDT)* of AF B1 in Nile tilapia (*O. niloticus*) and common carp (*Cyprinus carpio L.*) (Experiment 2)

Time hr.	Tilapia	Carp
24	0	0
48	0	0
72	0	0
96	0	0
120	0	1
144	0	2
168	1	0
192	0	1
216	1	0
MDT	192	150

* MDT of 5 injected fish

Table 3. Minimum leathal dose (MLD50) of AF b1 in Nile tilapia (T) (*O. niloticus)* and common carp (C) (*C. carpio L.*) (Experiment 2).

Item	AF B1 dose (ng /fish)							
	0		400		800		1200	
	T	C	T	C	T	C	T	C
Total No. of death*	0	0	0	1	0	1	2	4
Mortality %	0	0	0	20	0	20	40	80

* Five fish from each species were injected by each dose.

El-Banna (1995) reported that aflatoxin resulted in great economic loss to fish. The acute toxic effects include hemorrhaging and death, while the sub-acute or chronic forms could adversely affect growth, feed utilization efficiency and resistance to disease and also it might result in liver carcinoma. The present results indicated that MLD50 in common carp was 80 ug/kg , however, the MLD50 in rainbow trout was 0.5-1 mg/kg (Halver, 1966). Also,the obtained mortality rates were higher than that obtained by Bauer *et al.*, (1969).Variation in response to AF B1 has also been demonstrated for different species of salmonid (Bailey *et al.*, 1982). These data lead us to state that, common carp were more sensitive to aflatoxin than rainbow trout and Nile tilapia.

Table 3 shows that percent mortality in common carp was 2.5, 75, 55 and 60 versus 0, 10, 2 and 10 in Nile tilapia in dietary aflatoxin treated groups. The mortalities appeared on the 6 and 8*th* weeks for carp and tilapia respectively. Mortality in different species of fish receiving a dietary aflatoxin were also reported by other workers (Jackson *et al.*, 1968 and Sinnhuber *et al.*, 1974).

Feed intake (g DM/fish/98 days), growth performance, feed and nutrient utilization (Table 4) were significantly ($P<0.05$) higher in Nile tilapia than common carp and significantly ($P<0.05$) decreased with increasing the dietary aflatoxin levels. Meanwhile, protein and energy utilization were significantly decreased in aflatoxin-

treated groups. These decreases were reflected in slow growth rate and decrease of daily weight gains. These reductions in dietary aflatoxin-treated groups in the present study were attributable to the impairment of digestion and absorption due to different tested levels of aflatoxin (Osborne *et al.*, 1976 and Huff *et al.*, 1977). Furthermore, aflatoxin toxicity is expressed as the disruption of protein synthesis through conversion to 2,3 expoxide binding to DNA and inhibiting RNA synthesis (Yu,1981). However, no significant differences were observed in body dry matter due to different fish species or dietary aflatoxin treatment. Body crude protein was significantly ($P<0.05$) higher in tilapia than carp. Body crude protein was not affected with increasing dietary aflatoxin in tilapia and significantly ($P<0.05$) decreased with aflatoxin treatment in carp. The results showed also no significant differences in body ether extract in tilapia, however, a significant ($P<0.05$) increase in body ether extract was observed in the carp fed on dietary treated aflatoxin. The increased percentage of body ether extract in the carp agreed with the finding of Huff *et al.*(1986) and Merkley *et al.(*1987). They found that the accumulation of lipids in the liver increased significantly with increasing dietary aflatoxin concentration in chickens.

Mortality rate due to the high level of dietary aflatoxin (1904.76 ug/kg) was 80% in grey mullet during the first 11 days of the 4*th* experiment (Table 4). While the mortality of other species were 17.5 , 12.5 and 5.0 % in common carp, red tilapia and Nile tilapia respectively. These results indicate that mullet was highly sensitive to aflatoxin than common carp, red tilapia and Nile tilapia, respectively. Further investigation is needed to clarify this phenomenon.

Results of feed intake, growth performance, feed and nutrient utilization were significantly ($P<0.05$) decreased (Table 5) in the high dietary level of aflatoxin as compared with non aflatoxin treated group in all tested fish species. However, body ether extract was significantly ($P<0.05$) increased and body crude protein significantly ($P<0.05$) decreased in common carp only. All these findings supported the results of experiment 3. Meanwhile, no significant differences were reported in body dry matter due to fish species or aflatoxin treatment in experiment 4.

Symptoms and postmortem (PM) lesions of fish which received aflatoxin either by injetion or in feed were :

a) in common carp: appeared as orange or rusty spots on the dorsal surface and bilateral exophthalmia. Loss of appetite, sluggish movement and decrease in body gain were also observed. Postmortem (PM) lesions were paleness and enlargement of the liver with distension of gall bladder, and

b) in tilapia : sluggish movement, dark discolouration of caudal peduncle and tail fin and exophthalmia. Postmortem (PM) lesions were congestion in the kidneys, pale and enlargement in livers with distension of gall bladder.

Finally, it is concluded that :

(1)grey mullet(*Mugil cephalus*) was highly sensitive to the toxic effect of aflatoxin followed by common carp, (*Cyprinus carpio*) red tilapia (*Oreochromis niloticus* x *Sarotherodon mossabicus*) and Nile tilapia (*Oreochromis niloticus*) respectively.,

(2) dietary aflatoxin significantly ($P<0.05$) decreased feed intake, growth performance, feed and nutrient utilization, and

(3) body ether extract in common carp increased,while body crude protein decreased with increasing the dietary aflatoxin.

Table 4. Effects of different levels of dietary aflatoxin on mortality rate, growth performance, body composition, feed and nutrient utilization of Nile tilapia (T) and common carp (C) after 14 weeks of feeding (Experiment 3).

Item	Treatment *								LSD**
	1		2		3		4		
	T	C	T	C	T	C	T	C	
Mortality rate (%)	0.00	2.50	10.00	75.00	2.00	55.00	10.00	60.00	
Feed intake(g/fish)	20.80^{a}	14.10^{c}	19.80^{b}	12.60^{f}	19.50^{b}	12.60^{f}	17.40^{c}	12.30^{f}	0.427
Growth perfomance									
Initial weight (g/fish)	4.40	4.40	4.40	4.30	4.40	4.30	4.40	4.30	
Final weight (g./fish)	16.90	8.40	15.90	8.10	15.00	7.80	12. 00	5.90	2.069
Gain (g/fish)	12.50	4.00	11.50	3.80	10.60	3.50	7.60	1.60	0.387
Specific growth rate (%)	1.37^{a}	0.66^{c}	1.31^{a}	0.65^{c}	1.25^{ab}	0.60^{c}	1.02^{b}	0.32^{d}	0.210
Body composition (%)									
Dry matter (DM)	25.50	25.40	24.50	24.80	25.50	24.80	24.30	22.10	N.S.
% on DM basis									
Crude protein (CP)	57.90^{a}	54.10^{b}	57.90^{a}	50.70^{c}	57.80^{a}	50.40^{c}	57.80^{a}	51.00^{c}	1.987
Ether extract (EE)	21.60^{d}	34.60^{c}	21.80^{d}	38.70^{a}	22.20^{d}	39.10^{a}	22.50^{a}	37.10^{b}	1.541
Ash	20.00^{a}	11.30^{b}	20.30^{a}	10.60^{c}	20.00^{a}	10.50^{c}	19.70^{a}	12.00^{b}	0.851
Feed and nutrient utilization									
Feed conversion ratio(FCR)	1.66^{d}	3.53^{b}	1.72^{d}	3.32^{b}	1.84^{d}	3.60^{b}	2.29^{c}	7.69^{a}	0.335
Protein efficiency ratio(PER)	1.89^{a}	0.89^{c}	1.85^{a}	0.95^{c}	1.73^{ab}	0.88^{c}	1.38^{b}	0.41^{d}	0.315
Protein productrive value(PPV%)	28.50^{a}	15.80^{d}	26.50^{b}	14.80^{de}	26.10^{b}	14.10^{c}	19.70^{c}	5.90^{f}	1.095
Energy utilization (EU%)	18.50^{a}	15.20^{d}	17.00^{b}	16.50^{c}	17.00^{b}	14.50^{e}	13.10^{f}	8.40^{g}	0.391

* 1,2,3 and 4 diets contained 0, 100, 200 and 400 mg of dietary AF/kg diet, respectively.

** LSD: Least significant differences.

+Values in the same row with different superscripts differ significantly ($P<0.05$).

Table 5. Effect of the high level of dietary aflatoxin (1904,76 ug AF/kg diet) on mortality rate, growth performance, body composition, feed and nutrient utilization of grey mullet, Nile and red tilapia, and common carp after 12 weeks of feeding (Experiment 4).

Item	Treatment *								LSD**
	1				2				
	Grey mullet	Nile tilapia	Red tilapia	Common carp	Grey mullet	Nile tilapia	Red tilapia	Common carp	
Mortality rate (%)	0.0***	0.00	0.00	2.5	80***	5.00	12.50	17.50	
Feed intake (g DM/fish)	--	21.60^{c}	23.10^{a}	18.75^{e}	---	20.90^{d}	22.20^{b}	18.30^{f}	0.405
Growth performance									
Initial weight (g/fish)	---	$7.20_{}$	$7.30_{}$	$7.10_{}$	---	7.20_{b}	7.30_{ab}	$7.10_{}$	
Final weight (g/fish)	----	16.80^{ab}	18.40^{a}	11.60^{c}	---	15.60^{b}	16.70^{ab}	11.10^{c}	2.590
Gain (g/fish)	---	9.60^{b}	11.10^{a}	4.50^{d}	---	8.40^{c}	9.40^{b}	4.00^{d}	0.526
Speific growth rate (%)	---	1.01^{a}	1.10^{a}	0.58^{b}	---	0.92^{a}	0.99^{a}	0.53^{b}	0.20
Body cmposition (%)									
Dry matter (DM)	---	26.60	26.00	24.10	---	25.20	25.10	23.30	N.S.
% on DM basis									
Crude protein (CP)	---	60.95^{ab}	60.22^{b}	61.76^{a}	---	60.98^{ab}	60.07^{b}	57.15^{c}	1.451
Ether extract (EE)	---	19.35^{c}	18.78^{c}	27.04^{b}	---	20.42^{c}	19.93^{c}	31.35^{a}	1.765
Ash	---	19.70^{c}	21.00^{a}	11.20^{e}	---	18.60^{d}	20.00^{b}	11.50^{e}	0.921
Feed and nutrient utilization									
Feed conversition ratio (FCR)	---	2.25^{d}	2.08^{f}	4.16^{b}	---	2.49^{c}	2.36^{a}	4.60^{a}	0.405
Protein effeciency ratio (PER)	---	1.40^{b}	1.51^{a}	0.76^{e}	---	1.27^{d}	1.34^{c}	0.69^{f}	0.052
Protein productive value(PPV%)	---	23.55^{b}	24.73^{a}	13.59^{a}	---	19.70^{d}	20.83^{c}	9.64^{f}	0.449
Energy utilization (EU%)	----	12.57^{b}	14.18^{a}	9.82^{e}	---	10.41^{d}	12.18^{c}	8.85^{f}	0.315

* 1 and 2 diets containing 0 and 1904 76 ug /kg dietary aflatoxin, respectively.

** LSD: Least significant differences.

*** Results of grey mullet were not completed because 80% of aflatoxin-treated fish were dead at the 11th day of feeding.

+ Values in the same row with different superscripts differ significantly ($P<0.05$).

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أثر الاعلاف الملوثه بالافلاتوكسين على بعض اسماك المياه العذبة

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أصبعيات أربعة أنواع من اسماك المياه العذبة وهى البلطى النيلى (*Oreocohromis niloticus*) والبلطى الاحمر (هجين *O niloticus X O. mossabicus*) والبورى (*Mugil cephalus*) والمبروك العادى (*Cyprinus carpio*) تم تدريجهاحجميا ووضعت فى تنكات مستديرة وتم أختيار الافراد السليمة واستخدمت فى أربعة تجارب لدراسة مايلى :

١- حساسية البلطى النيلى والمبروك العادى للافلاتوكسين ب١ النقى (تجربة ١ ، ٢) و ٢- تأثير تلوث الاعلاف بالافلاتوكسين على الاعاشه والنمو والاستفادة من الغذاء والعناصر الغذائية(تجربه ٣ و ٤).

وقد قدر التأثير المرضى للافلاتوكسين ب١ النقى فى البلطى النيلى والمبروك العادى بمتوسط الوقت اللازم للموت بأقل جرعة قاتلة عن طريق الحقن فىالمنطقة البطنية بجرعات صفر، ١٠٠، ٢٠٠، ٤٠٠ نانوجرام افلاتوكسين ب١ وصفر ، ٤٠٠، ٨٠٠، ١٢٠٠ نانوجرام افلاتوكسين ب١ لكل ١ر مل HPLCميثانول لكل سمكة لمدة عشرة أيام متواصله فى التجربتين الاولىوالثانية على التوالى . واشارت نتائج التجربة الاولى الى عدم تأثر اعاشة الاسماك بالمستويات المنخفضة من الافلاتوكسين فى حين أوضحت نتائج التجربة الثانية ان٨٠ ٪ من اسماك المبروك العادى قد نفقت فىاليوم السادس (١٥٠ ساعة)بينما نفق ٤٠٪ فقط من البلطي في اليوم الثامن (١٩٠ساعة) وهكذا فأن اقل جرعة قاتلة للمبروك العادي كانت ٨٠ ميكروجرام افلاتوكسين ب١/ كجم من وزن الاسماك وكان اقل وقت لازم للنفوق ١٥٠ ساعة . والتشخيص المرضي تمثل في تضخم الكبد وظهور لون باهت وضمور في الحوصلة الصفراوية.

في التجربة الثالثة تم دراسة تأثير المستويات المختلفه من الافلاتوكسين فى العليقه (صفر، ٤٧٦ ، ٩٥٢، ١٩٠٥ ميكروجرام افلاتوكسين /كجم عليقة) على معدل الاعاشة والنمو والتركيب الكيميائي للجسم والاستفادة من الغذاء والعناصر الغذائية للمبروك العادي والبلطي ا لنيلي في تجربة نمو وتغذية لمدة ١٤ أسبوع . واوضحت النتائج ان معدل الاعاشة تناقص بشدة في المبروك العادي المعاملة بالافلاتوكسين مقارنة بالبلطي النيلى . وكانت معدلات النمو النسبى اقل جوهريا فى االاسماك المعاملة بالافلاتوكسين بالمقارنة بالكنترول. ولم تتأثر قيم المادة الجافة فى جسم الاسماك بالمعاملة بالافلاتوكسين. وكانت نسبة البروتين الخام فى جسم المبروك العادى تقل مع زيادة الافلاتوكسين فى العليقة في حين لم يتأثر البلطى بنفس الدرجة وكانت نسبة المستخلص الاثيرى في جسم المبروك العادى اعلى من البلطي فى حين كانت نسبة الرماد اعلى فى البلطى عن المبروك العادى وكانت معدلات الاستفادة من الغذاء والمواد الغذائية تقل مع زيادة نسبة الافلاتوكسين فى العليقة.فى التجربة الرابعة تم مقارنة تأثير المستوي العالى من الافلاتوكسين فى العليقة (١٩٠٥ ميكروجرام افلاتوكسين /كجم عليقة) بالعليقه الكنترول (الخاليه من الافلاتوكسين)على معدل الاعاشة والنمو والتركيب الكيماوى للجسم وكفاءة الاستفادة من الغذاء والعناصر الغذائية فى تجربة نمو وتغذيةأستمرت ١٢ اسبوع بأستخدام أسماك البلطى النيلى والبلطى الاحمر والمبروك العادى والبورى واظهرت النتائج ان نسبة النفوق كانت ٨٠٪ خلال الايام ال ١١ الاولى من التجربة بالنسبة للبورى فى حين أن نسبة الاعاش كانت ٨٢ ٪ ، ٨٧ ٪، ٩٥٪ للمبروك العادى

والبلطى الاحمر والبلطى النيلى على التوالى. ولم يكن هناك اى فروق جوهرية فى نمو الاسماك بين المجموعات المعاملة بالافلاتوكسين وغير المعاملة (كنترول) . ولم تتأثر نسبة المادة الجافة فى جسم الاسماك بالمعاملة بالافلاتوكسين فى حين انخفضت نسبة البروتين الكلى فى الجسم وخاصة فى اسماك المبروك العادى عن البلطى . واوضحت النتائج ان كفاءة الاستفادة من الغذاء العناصر الغذائية كانت تقل جوهريا عند المعاملة بالافلاتوكسين.

ويستخلص من نتائج الدراسة ان اسماك البورى كانت شديدة الحساسية للافلاتوكسين يليها اسماك المبروك العادى والبلطى الاحمر والبلطى النيلى على التوالى. المعاملة بالافلاتوكسين خفضت من معدلات النمو واستهلاك الغذاء وكفاءة الاستفادة من الغذاء والعناصر الغذائية.