# Detection of the effect of aflatoxin $B_1$ contaminated ration on Nile perch *Lates niloticus*

**Arafa, M.M.*, Ahmed Taha Ali** and Samia Fathi Mohammed***
* Chemistry Dept. and ** Fish Disease Dept.
Animal Health Res. Inst., Dokki, Giza, Egypt.

**Abstract**
The chemical analysis of ration, introduced to *Lates niloticus* fish reared in an experimental aquarium in a fish productive area in Ismailia Governorate was done. The analysis revealed the presence of high aflatoxin $B_1$ levels. The chemical analysis of the tissues of the fish showed elevated aflatoxin $B_1$ levels in the liver, kidney and musculature.
The fish showed sings of lethargy, sluggish movement, loss of appetite, excessive mucous over the skin, protruded eye and yellowish coloration of the body. The postmortem examination exhibited that liver was friable and dark in colour and viscera was congested. The gall bladder was enlarged and filled with bile. Biochemical analysis of the sera revealed significant increase in the alkaline phosphatase (ALP), aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activities, creatinine, urea, uric acid, cholesterol concentrations and albumin/ globulin (A/G) ratio. On the other hand, serum total protein (TP), albumin, globulin, sodium, potassium, calcium and inorganic phosphorus concentrations were significantly decreased. From the present investigation it is advisable to feed the fish with healthy aflatoxin free rations.

***Key words:***
*Fish, aflatoxin, liver, kidney, musculature,* ALP, AST, TP, *albumin, globulin, creatinine, urea, uric, cholesterol, sodium, potassium, calcium, phosphorus.*

## Introduction

Aflatoxins are secondary metabolites produced in specific ecological conditions by some strains of molds belonging to the groups of *Aspergillus flavus*, *A. parasiticus* and *A. nomius* **(Miliţă *et al.,* 2010).** Aflatoxin $B_1$ is known to be the most significant form that causes serious risk to animals and human health. Aflatoxins $B_1$ and $B_2$ adversely affect the growth, feed utilization and results in deleterious impact on the production of fish **(Adeniji *et al,* 2014).** The carcinogenic effect of aflatoxin $B_1$ has been found in fish **(Murjani, 2003).** Domesticated rainbow trout (*Oncorhynchus mykiss*) that were fed a pellet feed prepared with cottonseed meal contaminated with aflatoxins, developed liver tumors **(Ashley, 1970).**

The potential for development of aflatoxicosis increases in warm-water fishes, such as tilapia and channel catfish because plant ingredients, formulated in their diets have a higher potential than animal ingredients for contamination with aflatoxins. In tropical and subtropical conditions, this potential is further increased due to storage under humid and hot conditions. International trade in affected commodities and exposure to aflatoxins are worldwide concerns and the economic impact due to animal losses can be enormous **(Russo and Yanong, 2010).**
The fish, when exposed to the aflatoxin-contaminated feed suffer from a series of adverse complications ranging from developmental abnormalities to reproductive failure. Importantly the ingested aflatoxins are not totally

eliminated from their system, instead they accumulate and cause greater threat not only to themselves but also to fish eaters. The subtle effects often go unnoticed and in the long run results in increased incidence of liver cancer **(Madhusudhanan *et al.,* 2006).** Aflatoxins lower production efficiency of cultured fish by reducing growth rates, impairing immunity, and, in some cases, causing mortality. Storing feed properly (in a cool, dry area on pallets and at least one foot away from any walls) can prevent unnecessary economic losses **(Russo and Yanong, 2010).** Common effects of aflatoxicosis in finfish include poor growth, pale gills, reduced RBCs, anemia, impaired blood clotting, damage to liver, decreased immune responsiveness and increased mortality **(Goldblatt, 1976).**

The aim of the present work is to throw light on the harmful effects of aflatoxin $B_1$ on the health, liver and kidney functions and some biochemical changes in the blood serum of *Lates niloticus* fish.

## Material and methods

**1- Fish.**

Ten *Lates niloticus* weighing 3-5 kg were collected from an experimental traditional aquarium, exhibited health problems and high mortality rate in a fish productive area in Ismailia Governorate. Another 10 apparently healthy fish were collected and kept as control. Fish were transported alive to the Fish Disease Dept. in Animal Health Res. Inst.

**2- Water analysis.**

Water samples were collected from the affected and control fish aquaria and kept in closed glass bottles according to **(Tucker, 1992).** Water was subjected to chemical analysis for pH, dissolved oxygen ($DO_2$), hardness, phosphorus, nitrate, nitrite and ammonia according to **(Tucker, 1992)** and **(Clescerl *et al.* 1999)**, lead, cadmium and iron according to **(Jackson, 1973).**

**3- Fish ration analysis.**

The rations of the affected fish and control fish were chemically analyzed for the detection of aflatoxins, oxidative rancidity and acid number according to **(AOAC, 1980).**

**4- Clinical examination.**

The fish were clinically examined using the methods described by **(Lucky, 1977)**.

**5- Postmortem examination.**

Postmortem examination of gills, abdominal cavity and internal organs was carried out according to **(Amalacher, 1970).**

**6- Blood samples.**

Blood samples were collected from the caudal vein of each fish in either group according to **(Stoscopf, 1992)**. Each blood sample was kept in sloped glass tube at 4°C over night. Serum was then separated by centrifugation of blood at 3000 rpm for 20 minutes for biochemical analysis.

**7- Biochemical analysis.**

Serum alkaline phosphatase (ALP) activity was determined according to **(Marsh *et al.,* 1959)** by *Spectrum Diagnostics, Egypt* kits. Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activities in the blood serum were determined according to **(Reitman and Frankel, 1957)** using test kits of *Coral Co., India*. Serum total protein (TP) was measured by the method described by **(Cannon *et al.,* 1974)**; *Spectrum Diagnostics, Egypt* kits. Serum albumin was measured after **(Doumas *et al.,* 1971)** by *Vitro Scient, Egypt* kits. Blood serum globulin level and albumin/globulin ratio (A/G ratio) were calculated mathematically according to **(Coles, 1986)**. Serum creatinine concentration was determined using the method adopted by **(Bowers and Wong, 1980)**; *Spectrum Diagnostics, Egypt* kits. Serum urea level was determined according to **(Fawcett and Scott, 1960)** by *Bio-Diagnostic Kits, Egypt.* Determining serum uric acid concentration was performed by the assay of **(Fossati *et al.*, 1980)** *Spectrum Diagnostics, Egypt* kits. Cholesterol level was determined according to **(Young, 1990)** by *Vitro Scient, Egypt* kits. Sodium concentration in serum was measured according to **(Henry *et al.,* 1974)**; *Vitro Scient, Egypt* kits. Serum potassium concentration was determined as described by **(Hillman *et al.,* 1967)**; *Spectrum Diagnostics, Egypt* kits. Serum calcium concentration was

determined according to **(Barnett, 1965)** by *Spectrum Diagnostics, Egypt* kits. Serum inorganic phosphorus concentration was determined according to **(Daly and Ertingshausen, 1972)** by *Spectrum Diagnostics, Egypt* kits.

**Statistical analysis.**

Data were presented as mean ± standard error (SE) and the significance of differences was evaluated using analysis of variance (ANOVA) (**SPSS 14, 2006)**.

## Results

**Water chemical analysis.**

Table (1) shows the chemical criteria of the aquaria water samples of affected fish and control fish.

**Aflatoxin $B_1$ in ration.**

Chemical analysis showed that aflatoxin $B_1$ in the ration of affected *Lates niloticus* was significantly elevated comparing with control (table, 2).

**Aflatoxin $B_1$ in fish tissues.**

Table (3) represents that aflatoxin $B_1$ residues appeared in the liver, kidney and musculature of affected culture fish. No detectable levels of aflatoxin $B_1$ in the tissues of control fish.

**Clinical signs.**

Clinical signs in *Lates niloticus* fed ration containing aflatoxin $B_1$ included lethargy, sluggish movement, loss of appetite, increased mucous over the skin, exophthalmia and yellowish coloration of the body. High mortality rates were observed.

**Postmortem findings.**

The liver was friable and dark in colour and viscera was congested in *L. niloticus* fed ration containing aflatoxin $B_1$. The gall bladder was enlarged and filled with bile.

**Biochemical changes.**

As shown in table (4), ALP, AST and ALT activities, creatinine, urea uric acid, cholesterol concentrations and A/G ratio in the serum of *Lates niloticus* fed ration contained aflatoxin $B_1$ were significantly higher than those of control fish. On the other hand the table showed that serum TP, albumin, globulin, sodium, potassium, calcium and inorganic phosphorus concentrations in the *L. niloticus* fed ration containing aflatixin $B_1$ were significantly decreased comparing with control fish.

**Table (1).** Chemical parameters of control and affected fish aquarium water (n=10).

| Parameter | Control | Affected aquarium | Standard value* |
|---|---|---|---|
| pH | 7.5 | 8.5 | 6.5-9.0 |
| $DO_2$ (mg/L) | 5.5 | 5.1 | ≥ 5 |
| Hardness (mg/L as $CaCO_3$) | 90.0 | 125.0 | 10-400 |
| Phosphorus (mg/L) | 1.3 | 1.5 | ≤ 3.0 |
| Nitrate (mg/L) | 2.5 | 3.0 | ≤ 3.0 |
| Nitrite (mg/L) | 0.1 | 0.1 | ≤ 0.2 |
| Ammonia (un-ionized; mg/L) | 0.01 | 0.01 | ≤ 0.0125 |
| Lead (mg/L) | 0.01 | 0.02 | ≤ 0.03 |
| Cadmium (mg/L) | 0.0005 | 0.0005 | ≤ 0.003 |
| Iron (mg/L) | 0.01 | 0.01 | ≤ 0. 15 |

* Standard water parameters related to **(Swann, 1997).**

**Table (2).** Chemical analysis of ration offered to affected *Lates niloticus*.

| Parameter \ Group | Control | Affected aquarium |
|---|---|---|
| Aflatoxin $B_1$ (ppb) | 20.50 ± 1.20 | 168.65 ± 9.33** |
| Rancidity | 18.42 ± 1.63 | 22.00 ± 1.54 |
| Peroxide value | 25.74 ± 1.85 | 30.50 ± 2.26 |

Each value represents Mean ± SE; n=10.
** Significant difference against control at $p \leq 0.01$.

**Table (3).** Aflatoxin $B_1$ level in *Lates niloticus* tissues (ppb).

| Organ \ Group | Control | Affected fish |
|---|---|---|
| Liver | n.d. | 5.68 ± 0.24 |
| Kidney | n.d. | 5.20 ± 0.31 |
| Musculature | n.d. | 4.26 ± 0.21 |

Each value represents mean ± SE; n=10.
n.d. non detectable.

**Table (4).** Blood serum biochemical parameters in *Lates niloticus* fed aflatoxin $B_1$ contaminated ration.

| Parameter \ Group | Control | Affected fish |
|---|---|---|
| ALP (U/L) | 80.30 ± 3.59 | 103.64 ± 4.22** |
| AST (U/L) | 38.35 ± 1.65 | 64.4 ± 2.15** |
| ALT (U/L) | 20.58 ± 1.75 | 55.46 ± 2.19** |
| TP (g/dl) | 5.61 ± 0.37 | 4.11 ± 0.31** |
| Albumin (g/dl) | 3.25 ± 0.19 | 2.53 ± 0.10** |
| Globulin (g/dl) | 2.46 ± 0.11 | 1.66 ± 0.07** |
| A/G ratio | 1.30 ± 0.06 | 1.53 ± 0.07* |
| Creatinine (mg/dl) | 1.20 ± 0.08 | 1.62 ± 0.09** |
| Urea (mg/dl) | 8.74 ± 0.34 | 15.62 ± 0.68** |
| Uric acid (mg/dl) | 4.85 ± 0.23 | 5.83 ± 0.21** |
| Cholesterol (mg/dl) | 185.62 ± 5.11 | 224.50 ± 2.11** |
| Sodium (mM/L) | 125.50 ± 5.23 | 108.10 ± 4.42* |
| Potassium (mM/L) | 5.43 ± 0.25 | 4.56 ± 0.18* |
| Calcium (mg/dl) | 11.27 ± 0.65 | 7.86 ± 0.26** |
| Inorganic phosphorus (mg/dl) | 4.86 ± 0.22 | 4.02 ± 0.20* |

Each value represents mean ± SE; n=10.
* Significant difference against control at $p \leq 0.05$.
** Significant difference against control at $p \leq 0.01$.

**Discussion**

The chemical properties of water samples in the affected *Lates niloticus* culture were within the safe limits for fish according to **(Swann, 1997).**

The aflatoxin $B_1$ concentration in the rations of affected fish was significantly high comparing with those of control. In addition, aflatoxin $B_1$ residues were present in the affected *L. niloticus* liver, kidney and musculature.

*Lates niloticus* fed on ration containing aflatoxin $B_1$ exhibited signs of lethargy, sluggish movement, loss of appetite, increased mucous over the skin and protrusive eye while, fish body had yellowish colour. High mortality rates were observed in the affected fish.

The postmortem lesions included deformed liver and viscera. The gall bladder was enlarged and filled with bile. These clinical and anatomical findings nearly confirm those of **(El-Barbary and Mehrim, 2009 and Mehrim *et al.,* 2006)** aflatoxicated *Oreochromis niloticus* fish.

Respecting the blood serum biochemical findings, the enzymes, ALP, AST and ALT activities were significantly elevated in the serum of *Lates niloticus* fed on ration containing aflatoxin $B_1$ comparing with control. Nearly similar results where stated by **(El-Sayed and Khalil, 2009)** who recorded increase in serum transaminases and alkaline phosphatase activities in sea bass exposed to prolonged oral administration of aflatoxins and **(Selim *et al.,* 2014)** who observed increases in serum AST and ALT in *Oreochromis niloticus* fed aflatoxin $B_1$ containing ration. However our result nearly disagreed with that of **(Huang *et al.,* 2014)** who stated that the exposure of gibel carp fish to aflatoxin $B_1$ in diet at levels up to approximately 2000 μg $AFB_1$ $kg^{-1}$ had no significant effect on serum AST and ALT enzymes activities. The increase in the liver enzymes activities may be due to hepatic cell injury or increased synthesis of the enzymes by the liver as clarified by **(Yang and Chen, 2003)**.

Serum TP, albumin and globulin concentrations were significantly lowered while A/G ratio was increased in *Lates niloticus* fed on aflatoxin $B_1$ containing rations comparing with control. Nearly similar results were recorded by **(Sahoo and Mukherjee, 2001)** who recorded reductions of total protein, globulin levels and enhanced albumin-globulin ratio in Indian major carp (*Laboe rohita*) fish exposed to aflatoxin $B_1$ and **(Selim *et al.,* 2014)** who recorded a decrease in plasma proteins and globulin in *Oreochromis niloticus* fed aflatoxin $B_1$ containing ration. Blood proteins are used for energy production during toxicity and the protein catabolism is induced by stress **(Pfeifer and Weber, 1979)**. Moreover, aflatoxin hepatotoxicity leads to alterations in protein synthesis and cellular integrity of the liver **(Jindal *et al.,* 1994)**. Serum globulin reduction may be related to hemopoietic toxicity (anterior kidney and spleen) and lymphocytolysis **(Sahoo *et al.,* 2001)**. Aflatoxin $B_1$ also proved to be immunosuppressive in fish **(Sahoo and Mukherjee, 2001).** So, the increase of A/G ratio in *L. niloticus* fed on aflatoxin $B_1$ containing ration in the present study may be due the immunosuppressive effect of the aflatoxin.

Serum creatinine, urea and uric acid concentrations were significantly increased in *Lates niloticus* fed on aflatoxin $B_1$ containing ration comparing with control. Our findings are likely in agreement with those of **(Zaki *et al.,* 2010)** who found that aflatoxicosis increased urea and creatinine in *Tilapia Zilli* fish and **(Selim *et al.,* 2014)** who stated that aflatoxin $B_1$ increased serum creatinine level in *Oreochromis niloticus*. The increase of serum creatinine concentration fish fed on aflatoxin $B_1$ containing ration may be attributed to renal damage **(El-Boshy *et al.,* 2008).** The elevation of urea in the present study may be due to gill dysfunction **(Lockhart and Metner, 1984)**. The increase of uric acid levels in aflatoxin $B_1$ affected fish could be resulted from liver damage induced by aflatoxin **(Carlson *et al.,* 2001 and Ottinger and Kaattari, 1998)**.

Serum cholesterol level was significantly elevated in aflatoxin affected *L. niloticus*. Nearly

similar result was concluded by **(Zaki *et al.*, 2010)** in *Tilapia zilli* fish supplied with aflatoxin contaminated ration. In the current study, the hypercholesterolaemia in aflatoxin $B_1$ affected fish may be due to liver injury.

Significant decrease in the blood serum minerals, sodium, potassium, calcium and inorganic phosphorus levels occurred in the aflatoxin $B_1$ affected *Lates niloticus* comparing with control. The gills are the major sites of osmotic and ionic regulation in fish, any changes in gill morphology may result in perturbed osmotic and ionic status **(Al-Attar, 2007).** In the present study, hyponatraemia in the aflatoxin $B_1$ affected fish might reflect a decrease in the sodium influx rate through the gills as showed by **(Martinez *et al.*, 2004)**. The hypokalaemia in the affected fish might result from impaired active reabsorption of potassium in renal tubules **(Gill *et al.*, l989).** The hypocalcaemia observed in the aflatoxin $B_1$ fed *L. niloticus* may be due to diffusional losses caused by increased permeability of gill epithelium to water and ions **(Giles, 1984),** defective intestinal calcium absorption or impaired calcium reabsorption in the renal tubules **(Gill *et al.*, 1988).** The decreased serum inorganic phosphorus may be attributed to disturbance in metabolism resulted from gastrointestinal malfunction **(Glavind and Tryding, 1960).**

Conclusion: In the present study, aflatoxin $B_1$ residues appeared in the tissues of *Lates niloticus* fed on rations polluted with that toxin. The aflatoxin $B_1$ had bad impacts on the organs function and blood serum parameters. So, it is advisable to control the feeding of fish and supplying the fish with healthy aflatoxin free rations.

## References

**Adeniji, C.A.; Okiki, P.A.; Rasheed, A.M. and Bolaji, R. (2014).** Mouldy groundnut cake and hydrated sodium calcium aluminosilicate in practical diet for African catfish *Clarias gariepinus* (Burchell, 1822). Fish. Aquac. J., 4: 1

**Al-Attar, A.M. (2007).** The influences of nickel exposure on selected physiological parameters and gill structure in the teleost fish, *Oreochromis niloticus.* J. Biol. Sci., 7 (1): 77-85.

**Amalacher, E. (1970).** Textbook of fish disease. Tropical Fish Hobbyist, pp. 143-146.

**AOAC-Association of Official Analytical Chemists (1980).** Methods of analysis Washington, D.C. USA.

**Ashley, L.M. (1970).** Pathology of fish fed aflatoxins and other antimetabolites In: A symposium on diseases of fishes and shellfishes. American Fisheries Society Special Publication 5, 366-379.

**Barnett, R.N. (1965).** A scheme for the comparison of quantitative methods. Am. J. Clin. Pathol., 43: 562.

**Bowers, L.D. and Wong, E.T. (1980).** Kinetic serum creatinine assays. II. A critical evaluation and review. Clin. Chem., 26: 555-561.

**Cannon, E.C.; Olitzky, I. and Inkpen, J.A. (1974).** Proteins. In: Clinical chemistry principles and technics, 2nd ed. R.J. Henery, D.C. Cannon, J.W. Winkelman, editors, Harper & Row, New York, pp 407-421.

**Carlson, D.B.; Williams, D.E.; Spitsbergen, J.M.; Ross, P.F.; Bacon, C.W.; Meredith, F.I. and Riley, R.T. (2001).** Fumonisin $B_1$ promotes aflatoxin $B_1$ and N-methyl-N'-nitro-nitrosoguanidine-initiated liver tumors in rainbow trout. Toxicol Appl Pharmacol. 1, 172 (1): 29-36.

**Clescerl, L.; Greenberg, A. and Eaton, A. eds. (1999).** Standard methods for examination of water and wastewaters, 20th edn. Association, pp.1325.

**Coles, E.H. (1986).** Veterinary Clinical Pathology, 4th Edition W.B. Sanders Company, Pliladelphia, 22-23.

**Daly, J.A. and Ertingshausen, G. (1972).** Direct method for determining inorganic phosphate in serum with the "CentrifiChem". Clin Chem. 18 (3): 263-265.

**Doumas, B.T.; Watson, W.A. and Biggs H.C. (1971).** Albumin standards and Management of serum albumin with bromocresol green. Chin. Chim. Acta., 31:87-96.

**El-Barbary, M.I. and Mehrim, A.I. (2009).** Protective effect of antioxidant medicinal

herbs, rosemary and parsley, on subacute aflatoxicosis in *Oreochromis niloticus*. J. Fish. Aquat. Sci., 4 (4): 178-190.

**El-Boshy, M.E.; El-Ashram, A.M.M. and Abd El-Ghany, N.A. (2008).** Effect of dietary beta-1, 3 glucan on immunomodulation on diseased *Oreochromis niloticus* experimentally infected with aflatoxin $B_1$. 8th International Symposium on Tilapia in Aquaculture.

**El-Sayed, Y.S. and Khalil, R.H. (2009).** Toxicity, biochemical effects and residue of aflatoxin b1 in marine water-reared sea bass (*Dicentrarchus labrax l.*). Food and Chemical Toxicology, 47 (7): 1606-1609.

**Fawcett, J.K. and Scott, J.E. (1960).** A rapid and precise method for the determination of urea. J. Clin. Path., 13 (2): 156-159.

**Fossati, P.; Prencipe, L. and Berti, G. (1980).** Use of 3,5-dichloro-2-hydroxybenzene-sulfonic acid/4-aminophenazone chromogenic system in direct enzymatic assay of uric acid in serum and urine. Clin. Chem., 62: 227-231.

**Giles, M. (1984).** Electrolyte and water balance in plasma and urine of rainbow trout Salmo gairdneri during chronic exposure to cadmium. Can. J. Fish Aquat. Sci., 41: 1678-1685.

**Gill, T.S., Jagdish, C.P. And Jaishree, P. (1988).** Gill, liver and kidney lesions associated with experimental exposure to carbaryl and dimethoate in fish, Puntius conchonius (HAM.). Bull. Environ. Contam. Toxicol., 41: 71-78.

**Gill, T.S.; Pant, J.C. and Tiwari, H. (1989).** Cadmium nephropathy in freshwater fish Puntius conchonius (HAM.). Ecotox. Environ. Safe,. 18: 165-172.

**Glavind, J. And Tryding, N. (1960).** Acta physiologicl. Scand. 49 (97) cited by Artman, B.R.(1969).

**Goldblatt, L.A. (1976).** Significance of aflatoxin in foods. Proc. 80th Annual Conference of the Association of Food and Drug Officials, Atlanta, Georgia, June 22, pp: 191 – 201.

**Henry, R.F., Cannon, D.C. and Winkelman, J.W. (1974).** Clinical Chemistry Principles and Techniques. 2nd Ed. Harper and Roe, Hagerstown, M.D.

**Hillman, G.; Beyer, G. and Klin, Z. (1967).** Determination of potassium concentration. Chem. Clinic. Biochem., 5: 93.

**Huang, Y.; Han, D.; Xiao, X.; Yang, Y.; Jin, J.; Chen, Y and Xie, S. (2014).** Effect of dietary aflatoxin $B_1$ on growth, fecundity and tissue accumulation in gibel carp during the stage of gonad development. Aquaculture, 429: 236-242.

**Jackson, M.L. (1973).** Textbook of Soil Chemical Analysis, Prentice Hall of Indian Private Ltd.

**Jindal, N.; Manipal, S.K. and Mahajan, N.K. (1994).** Toxicity of aflatoxin B1 in roiler chicks and its reduction by activated charcoal. Res. Vet. Sci. 56:37–40.

**Lockhart, W.L. and D.A. Metner (1984).** Fish chemistry as a pathology tool, pp. 73-86. In: Contaminant Effects on Fisheries. Vol. 16 (Cairns, V.W., P.V. Hodson and J.O. Nriagu eds.). New York: Wiley Interscience.

**Lucky, Z. (1977).** Textbook for the diagnosis of fish diseases, Amerind Publishing Co. PVT. Ltd., New Delhi.

**Madhusudhanan, N.; Kavithalakshmi, S.N.; Shanmugasundaram, E.R.B. and Shanmugasundaram, K.R. (2006).** Aflatoxin $B_1$-induced DNA damage in *Labeo rohita*: protective effect of an antioxidant supplement, *Amrita Bindu*. Basic & Clinical Pharmacology & Toxicology, 98: 473-479.

**Marsh, W.H.; Finger hut, B. and Kirsch, E. (1959).** Adaptation of an Alkaline phosphatase method for automatic colorimetric analysis. Clin. Chem., 5: 119.

**Martinez, C.B.R.; Nagae, M.Y.; Zaia, C.T.B.V. and Zaia, D.A.M. (2004).** Acute morphological and physiological effects of lead in the neotropical fish Prochilodus lineatus. Braz. J. Biol., 64: 797-807.

**Mehrim, A.I., Abdelhamid, A.M., Abou-Shousha, A.,Salem, M.F.I. and El-Sharawy, M.A.M.M. (2006).** Nutritious attempts to detoxify aflatoxic diets of tilapia fish: 2- Clinical, biochemical and histological

parameters. J. Arab. Aquacult. Soc., 1: 69-90.

**Miliţă, N.M.; Mihăescu, G.; Chifiriuc, C. (2010).** Aflatoxins-Health risk factors. Bacteriol. Virusol. Parazitol. Epidemiol. 55: 19-24.

**Murjani, G. (2003).** Chronic aflatoxicosis in fish and its relevance to human health. Central Institute Of Fresh Water Aquaculture. India.

**Ottinger, C.A. and Kaattari, S.L. (1998).** Sensitivity of rainbow trout leucocytes to aflatoxin $B_1$. Fish Shell Fish Immunol, 8:515.

**Pfeifer, K.F. and Weber, L.J. (1979).** The effect of carbon tetrachloride on the total rotein concentration of rainbow trout, Salmo gairdneri. Comp. Biochem. Physiol. 64(C):37 –42.

**Reitman, S. and Frankel, S. (1957).** A colorimetric method for the determination of serum glutamic oxalacetic and glutamic pyruvic transaminases. Amer. J. Clin. Path., 28: 56.

**Russo, J.R. and Yanong, R.P.E. (2010).** Molds in fish feeds and aflatoxicosis. Fact Sheet FA-95, Institute of Food and Agricultural Sciences, University of Florida.

**Sahoo, P.K. and Mukherjee, S.C. (2001).** Immunosuppressive effects of aflatoxin B1 in Indian major carp (*Laboe rohita*). Microbiol. Infectious Dis. 24: 143-149.

**Sahoo, P.K.; Mukherjee, S.C.; Jain, A.K. and Mukherjee, A. (2001).** Histopathological and electron microscopic studies of gills and opisthonephros of Rohu, Labeo rohita to acute and subchronic aflatoxin B1 toxicity. Asian Fish Sci 16:257–268.

**Selim, K.M.; El-hofy, H. and Khalil, R.H. (2014).** The efficacy of three mycotoxin adsorbents to alleviate aflatoxin $B_1$-induced toxicity in *Oreochromis niloticus*. Aquacult. Int., 22: 523-540.

**SPSS 14 (2006).** Statistical package for social science, SPSS for windows release 14.0.0, Standard version, SPSS Inc.

**Stoskopf, M.K. (1992).** Clinical Pathology. In: "Fish Medicine", M.K. Stoskopf (ed.), 1st edn., W.B. Saunders Company, Philadelphia.

**Swann, L. (1997).** A Fish Farmer's Guide to Understanding Water Quality. Aquaculture extension, Illinois-Indiana Sea Grant Program. AS-503, Purdue University, IL-IN, pp. 1-8.

**Tucker, C.S. (1992).** Water analysis. In: Fish Medicine, M.K. Stoskopf (ed.) 1st edn., W.B. Saunders Company, Philadelphia, pp. 166-197.

**Yang, J. and Chen, H. (2003).** Serum metabolic enzyme activities and hepatocyte ultra structure of common carp after gallium exposure. Zoological Studies, 42 (3): 455-461.

**Young, D.S. (1990).** Effects of drugs on clinical laboratory tests. Third edition, 3: 6-12.

**Zaki, M.S.; Fawzi, O.M.; Omar, S.; Khafagy, M.; Fawzy, M. and Awad, I. (2010).** Diminution of aflatoxicosis in *Tilapia zilli* fish by dietary supplementation with fix in toxin and *Nigella sativa* oil. Nature and Science, 8 (2): 43-49.

# قياس تأثير الأفلاتوكسين بي1 في أسماك قشر البياض النيلي

**محمود محمد عرفة* - أحمد طه علي** - سامية فتحي محمد***

* قسم الكيمياء ، ** قسم أمراض الأسماك

معهد بحوث صحة الحيوان بالدقي - جيزة – مصر

**الملخص العربى**

تم فحص العلف المقدم إلى أسماك قشر بياض مرباة في حوض تجريبي في منطقة إنتاج سمكي بمحافظة الإسماعيلية. وقد أظهر التحليل وجود مستويات عالية من الأفلاتوكسين بي1. كما أظهر الفحص الكيميائي لأنسجة الأسماك وجود نسبة عالية من الأفلاتوكسين بي1 في الكبد والكلى والعضلات.

وقد ظهر على الأسماك أعراض الخمول وبطء الحركة وفقدان الشهية وزيادة المخاط على الجلد وجحوظ العين واصفرار لون الجسم. وأظهرت الصفة التشريحية أن الكبد سهل التفتيت وداكن اللون والأحشاء محتقنة وانتفاخ الحوصلة المرارية واكتنازها بالعصارة الصفراوية. وقد لوحظ من التحليل الكيميائي الحيوي لأمصال دم السمك وجود زيادة معنوية في أنشطة إنزيمات الفوسفاتاز القاعدي والأسبارات أمينوترانسفيراز والألانين أمينوترانسفيراز وزيادة معنوية في تركيزات الكرياتينين واليوريا وحمض اليوريك والكوليستيرول ونسبة الألبيومين/ جلوبيولين. ومن ناحية أخرى حدث نقص معنوي في تركيزات البروتين الكلي والألبيومين والجلوبيولين والصوديوم والبوتاسيوم والكالسيوم والفوسفور غير العضوي في مصل دم هذه الأسماك. لذلك ينصح بالعناية بغذاء الأسماك وتغذيتها على عليقة خالية من الأفلاتوكسين.