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# REDUCING THE TOXICITY OF AFLATOXIN $B_1$ BY DIFFERENT ADSORBENTS IN FISH

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## ABSTRACT

Reduction of aflatoxicosis in Nile tilapia (*Oreochromis niloticus*) fish was exmined by adding eight commercial adsorbents from Egyptian market to aflatoxin $B_1$ contaminated diets in a feeding trial for 8 weeks. Twenty hundred and ten growing Nile tilapia (*Oreochromis niloticus)* fish were assigned to ten experimental diets. There were 3 replicate glass aquariums of 7 fish / replicate. The 1st diet served as a control (commercial diet) (C), the 2nd one was contaminated with 9 mg aflatoxin $B_1$ / Kg diet (A) and the other experimental diets contained the same level of aflatoxin $B_1$ plus 0.5% of adsorbents from I to VIII . Adsorbent I was modified yeast cell wall, II was bentonite, III was tri - star , IV was mycobond, V was egy - tox, VI was moldstop super, VII was fungstat-k and VIII was moldstop mycobind plus.

Aflatoxin $B_1$ caused significantly ($P \leq .05$) loss in live body weight which was 6.09; 11.25; 17.34 and 22.87% of the treated fish at 2 , 4 , 6 and 8 weeks, respectively. Mortality rate increased significantly ($p \leq 0.05$) (47.62 % versus 4.76% for the control) by aflatoxin. Also, aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activites increased significantly by aflatoxin but the total protein and albumin decreased.

Adding the adsorbents caused significantly ($P \leq .05$) reduce the toxic effect of aflatoxin on loss of body weight (the improvement ranged from 14.53 to 95.57%) according to the kind of adsorbent and experimental duration. Also significantly ($P \leq .05$) decrease in the mortality rate and improved the blood parameters ($p \leq 0.05$) were caused by adsorbents.

These results suggested that adding adsorbents specially adsorbent IV (Mycobond) and VI (mold stop super) to fish diet contaminated with aflatoxin $B_1$ had benifical effects in fish feeding.

## INTRODUCTION

Aflatoxins are mycotoxins produced as secondary metabolites by *Aspergillus flavus* and *Aspergillus parasitcus* (Cheeke and Shull,1985).Todays it is estimated that mor than 25% of the world cereals are contaminated with know mycotoxins (Devegowda *et al.*, 1998). In Egypt, the aflatoxins and other mycotoxins are frequently detected in feedstuffs (Abdelhamid, 1990 &1993a, Abdelhamid *et al.*,1996 and Aziz *et al.*, 1997). The problems with mycotoxins do not end in feed refusal or redution of animal performance but many of these mycotoxins transfere into the meat or milk (Devegowda *et al.*, 1998).

The common effect of aflatoxicosis includes poor growth, anemia, impaired blood clotting, sensitivity to bruising, damag of liver and other organs, decreased immune response, increased mortality (Lovell,1991 and Abdelhamid *et al.*,1997 and 2002 a&b). Also, mycotoxins had carcinogenicity,

hepatitis, nephritis, dermatitis and genacologic forms (Abdelhamid and Dorra,1993 and Abdelhamid *et al* .,2002 a,b &c and 2003).

Many different methods (physical, chemical and biological techniques) were carried out for detoxification of mycotoxins (Abdelhamid, 1993 b and Abdelhamid *et al.*, 2002 & 2003 a, b, d). The most applied method for protecting animals against mycotoxicosis is the utilization of adsorbents mixed with the feed which are supposed to bind the mycotoxins efficiently in the gastrointestinal tract (Nowar *et al.*, 1996; Huwig *et al.*, 2001; Abd El-Baki *et al.*, 2002 and Abdelhamid *et al.*, 2002 c & 2003 and Shehata, (2002). Modified yeast cell wall mannanoligosaccharide (MOS) is based on an esterified glucomannan derived from the cell wall of a selected strain of *Saccharomyces cervisiae.* It causes stimulation of specific immune system, increased antibody titer values against infection and adsorption of mycotoxins (Devegowda *et al* ., 1998 and Shehata ,2002).

The present study was carried out to evaluate the efficiency of 8 commerical adsorbents to aflatoxin $B_1$ contaminated diet in reducing the aflatoxicosis in fish.

## MATERIALS AND METHODS

The exprimental work of this study was carried out in-door wet Lab. In the Aquaculture Research Lab., Abbassa, Abo-Hamad, Egypt. *Asperigllus flavus* MD 341, was obtained from the Central Lab. of Residues in Aagric. Products, Agric. Pesticides Research Centre, Dokki, Egypt, for production of the aflatoxin $B_1$ . *A. flavus*_ was grown on yeast extract sucrose (YES) containing 2% yeast extract and 20% sucrose. The substrate was dispensed in conical flask. The flasks were then autoclaved for 15 minutes at 121 $C^0$, then cooled and inoculated with spore suspension and incubated for 9 days at 25 – 29 C0. Aflatoxin was extracted from liquid media according to Davis et al (1966). Aflatoxin concentration was determined using the methods Shih and Marth (1969) and A.O. A. C. ( 1984). The media was found to contain aflatoxin B1 alone. Twenty hundred and ten Nile tilapia (*Oreochromis niloticus*) were randomly assigned to each of ten dietary treatments (Table 1) (21 fish in each). For each of ten treatments there were 3 replicate glass aquarium of 7 fish per aquarium for a total of 21 fish/ treatment. Eight commercial adsorbents in market in Egypt were tested. Adsorbents at a rate of 0.5% were added to ground commercial diet and pelleted agin. Commercial diet Product of Factory of General Organization for Fish Development was used in the exp. it consisted of fish meal, soybean meal, meat meal, yellow corn, bone meal, mixture of vitamins and minerals. The chamical composition was adopted according to A.O.A.C. (1980). Filterate of A. flavus sprayed on pelleted diets to obtain 9 mg/kg feed. The dimensions of each glass aquarium were 150 X 50 X 50 cm. This glass aquariums were supplied with dechlorinated tap water and continous aeration was adapted by using an air pump and airstones.Water temperature was 22°C ± 2°C. Sediment was filtered by siphon method daily and water was completely changed every 3 days.

Table (1): Experimental treatments

| No. | Treatments |
|---|---|
| 1- | Control (commercial diet) (C ) |
| 2- | Control contaminated with aflatoxin $B_1$ (9 mg/kg) ( A ) |
| 3- | A + 0.5% adsorbent I (Modified yeast cell wall) |
| 4- | A + 0.5% adsorbent II (Benontite) |
| 5- | A + 0.5% adsorbent III [Tri star (organic acid and silicate salts)]. Each Kg contain 300g formic acid, 150g probionic acid, 300g glutofid, 150 g precipitate of silica, 100 g calcium carbonate. German Co. for Vet. Medicine and Feed Additives. |
| 6- | A + 0.5% adsorbent IV [Mycobond (natural mineral compound with a high adsorption and binding capacity)]. Product of Optivite International Ltd, Main Street, Laneham, Retford, Notts, United Kingdom . |
| 7- | A + 0.5% adsorbent V [Egy-Tox (adsorption for toxin and fungcidal)]. It contain gentiana CA, MG,K,$Al_2SLo_3$ . Product of Egyption - Holand Co. |
| 8- | A+ 0.5% adsorbent VI [Moldstop super (used for control the molds and adsorption of its mycotoxin)]. Each Kg contain 200g calcium probionate, 100g Kaolin, 100g aluminum silicate, 10g copper sulphite. Product of Smart Vet. |
| 9- | A + 0.5% adsorbent VII [Fungstat- k (contains mixtures of organic acids and silicate salts)]. Product of Pharma Swede – Egypt. |
| 10- | A + 0.5% adsorbent VIII [Moldstop mycobind plus (composed of 50% : propionic acid, ammonium propionate, natural extracts, emulsifiers, antioxidant (BHA) and 50 % : unique combination of specially selected carriers with mycobinding activity- HSCAS, completed by amorphus silicum dioxide. Product of IMPEX TRACO ( Beligum ), Sole agent : NILE VET . |

The fish were fed 2 times a day (900 and 1600 h.) at a rate of 2% of the total body weight ( as recomonded by Parrel et al., (1986 ). The fish were weighted every two weeks for 8 weeks. At the end of experiment 6 fish from each treatment (2 fish/ replicate) were scarificed for collection of the blood. Blood was take from the caudal vein using sterilized syringe for seperating serum. Serum was analysed for total protien, albumin, aspartate aminotransferase (AST) and alanine aminotransferase (ALT) using commercial kits purchased from Diamond Diagnostics Company, Egypt.

Data of the experiment were statistically analyzed according to Snedecor and Cochran, (1982). Significant differences between treatment means were tested by Duncan's Multiple Range Test (Duncan, 1955).

## RESULTS AND DISCUSSION

### 1- Chemical composition:

The chemical composition for commerical diet as dry matter basis was 80.00, 30.00, 8.50, 3.93, 37.57, 20.00 for OM, CP, CF, EE, NFE and Ash, respectively.

### 2- Growth performance:

Data presented in Table (2) showed that aflatoxin $B_1$ caused a significantly loss in live body weight (40.09 g at 8 weeks versus 48.52 g at start of exp.).

**Table (2) : The effect of aflatoxin $B_1$ ( 9 mg/kg diet ) on fish performance and its modification by adsorbents**

| Parameters | Weeks | Treatments | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Control | Aflatoxin | Afl+ I | Afl+ II | Afl+ III | Afl+ IV | Afl+ V | Afl+ VI | Afl+ VII | Afl+ VIII |
| Live body weight (g) | Initial | 47 73±1 85 | 48.52±2.33 | 47.64±1.67 | 48.86±3.07 | 47.86±1 70 | 48.91±1.47 | 47.26±1.84 | 47.53±1.76 | 47 72±1.99 | 48 23±2.26 |
| | 2 weeks | 48.63±1.94 a | 45.67±2.19 c | 46.24±1.63 b | 46.61±2.95 b | 46.66±1 61 b | 48.76±0 41 a | 46.69±1.66 b | 47.28±1.76 b | 47 27±1 82 b | 46.48±2.58 b |
| | 4 weeks | 49.43±1.98 a | 43.87±2.16 e | 44.84±1.65 d | 45.61±3.19cd | 45.76±1.70 cd | 48.28±1.47 b | 46.26±1.84 c | 47.53±1.76 b | 46.87±1 99 c | 45.23±2.26 d |
| | 6 weeks | 50.53+1.89 a | 41.77±2.00 f | 43.04±1.49 e | 44.71±3.12de | 44.71±1.62 d | 47.75±1.18 b | 45.54±1.56 cd | 48.03±1.76 b | 46.27±1 90 c | 43 55+2.81 e |
| | 8 weeks | 51.98±1 89 a | 40.09±2.02 e | 42.15±1.53 d | 44.01±3.11 c | 43.73±1 56 cd | 47.42±1.23 b | 44.70±1.70 c | 48.55±1.87 b | 45.38±2.05 c | 42.38±2.93 d |
| Change in body weight (%) | 2 weeks | 0 | -6.09 | -4.91 | -4.15 | -4.05 | 0.27 | -3.99 | -2.78 | -2.80 | -4.42 |
| | 4 weeks | 0 | -11.25 | -9.29 | -7.73 | -7.42 | -2.33 | -6.41 | -4.00 | -5.18 | -8.50 |
| | 6 weeks | 0 | -17.34 | -14.82 | -11.52 | -11.52 | -5.50 | -9.88 | -4.95 | -8.43 | -13.81 |
| | 8 weeks | 0 | -22.87 | -18.91 | -15.33 | -15.87 | -8.77 | -14.01 | -6.60 | -12.70 | -18.47 |
| Improvment of body weight by adsorbents (%) | 2 weeks | - | - | 19.38 | 31.86 | 33.50 | 104.43 | 34.48 | 54.35 | 54 02 | 27.42 |
| | 4 weeks | - | - | 17.42 | 31.29 | 34.04 | 79.29 | 43.02 | 64.44 | 53.96 | 24.44 |
| | 6 weeks | - | - | 14.53 | 33.56 | 33 56 | 68.28 | 43.02 | 71.45 | 51.38 | 20.36 |
| | 8 weeks | - | - | 17.32 | 32.97 | 30.61 | 61.65 | 38.74 | 71. 14 | 44.47 | 19.24 |
| | Average | - | - | 17.16 | 32.42 | 32.93 | 78.41 | 39.82 | 65.35 | 50.96 | 22.87 |
| Body weight gain (g / 2 weeks) | 2 weeks | 0.90±0 09a | -2.85±0.15e | -1.40±0.03c | -2.25±0.31d | -1.20±0 09c | -0.15±0.09b | -0.57±0.26b | -0.25±0.05b | -0.45±0.17b | -1.75±0.33cd |
| | 4 weeks | 0.80±0.05a | -1.80±0.09f | -1.40±0.13ef | -1.00±0.26e | -0.90±0.09de | -0.48±0.18cd | -0.43±0.18c | 0.25±0.09b | -0.40±0.05cd | -1.25±0.25e |
| | 6 weeks | 1.10±0.13a | -2.10±0.17e | -1.80±0.26e | -0.90±0.09cd | -1.05±0.17d | -0.53±0.16c | -0.72±0.09cd | 0.50±0.09b | -0.60±0.09cd | -1.68±0.16e |
| | 8 weeks | 1.45±0 05a | -1.68±0.08f | -0.89±0.05de | -0.70±0.08d | -0.98±0 16de | -0.33±0.09c | -0.84±0.16de | 0.52±0 09b | -0.89±0.19de | -1.17±0.12e |
| | Average | 1.06±0.02a | -2.11±0.08g | -1.37±0.08f | -1.21±0.11ef | -1.03±0 08e | -0.23±0.06c | -0.64±0 06d | 0.26±0.02d | -0.59±0.04d | -1.46±0.18f |
| Relative growth rate (%) (RGR) | 2 weeks | 1.89±0.11a | -5.87±0.09e | -2.94±0.07c | -4.60±0.55d | -2.51±0.09c | -0.31±0.17b | -1.21±0.53b | -0.53±0.11b | -0.94±0.30b | -3.63±0.88cd |
| | 4 weeks | 1.65±0.06a | -3.94±0.27e | -3.03±0.30de | -2.15±0.74cd | -1.93±0.25cd | -0.98±0.42bc | -0.92±0.38b | 0.53±0.18a | -0.85±0.10bc | -2.69±0.60d |
| | 6 weeks | 2.23±0 35a | -4.79±0.18e | -4.01±0.51e | -1.97±0.10cd | -2.29±0.34d | -1.10±0.38c | -1.56±0.19cd | 1.05±0.21b | -1.28±0.21c | -3.71±0.59e |
| | 8 weeks | 2.87±0.14a | -4.02±0.32f | -2.07±0.18de | -1.57±0.20cd | -2.19±0.33de | -0.69±0.2c | -1.84±0.40de | 1.08±0.15b | -1.92±0.44d | -2.69±0.48e |
| | Average | 2.16±0.08a | -4.66±0.05f | -3.01±0.16de | -2.57±0.31de | -2.23±0.16d | -0.77±0.19c | -1.38±0.10c | 0.53±0.05b | -1.25±0.11c | -3.20±0.60e |
| Mortality rate (%) ( MR) | | 4.76±4.77d | 47.62±4.77a | 9.53±4.77cd | 9.53±4.77cd | 28.57±8.26b | 9.53±4.77cd | 19.05±4.77bc | 9.53 ± 4.77cd | 19.05±4 77bc | 19.05±4.77bc |

**Means in the same row bearing different letters differ significantly ( $p \leq 0.05$ ).**
**RGR = Final live body weight – Initial live body weight / Initial live body weight x 100**
**MR = No.of fish at start of exp. - No.of fish at end of exp. / No.of fish at start of exp. X100**

The bad effects of aflatoxin $B_1$ on growth performance (live body weight, body weight gain and relative growth rate) agreed with the findings of Jantrarotai and Lovell (1990) who reported that channel catfish fed 10 mg aflatoxin $B_1$/Kg feed for 10 weeks had shown a significant decrease in growth rate. Also, EL-Said, (1997) rerorted that 3 mg aflatoxin / Kg diet of *Oreochromis_aureaus* for 90 days caused a clear growth depression, were the loss in body weight gain was 4.33%. However, the effect of mycotoxin on fish depends on potency of mycotoxin, dose, species and strain of the fish, state of health, stage of life, temperature of the water and presence or absence of substances that can modify the toxicity (El-Said, 1997). The decrease of growth rate by aflatoxin may be due to disturbances of one or more basic metabolic processes (carbohydrate, lipid or protein metabolise) in the liver and loss of appetite (Cheeke and Shull, 1985). Also, it might be due to detoxification process in the body utilizing glutathione enzymes. Glutathione is partly composed of methionine and cystein, hence this detoxification process depletes the metabolic availabilty of methionine leading to poor growth and feed efficiency (Devegowda *et al.*, 1998).

Addition of the adsorbents reduced ($P \leq 0.05$) the toxic effect of aflatoxin $B_1$. Since, the average body weight gain ( g / 2 weeks ) ranged from + 0.26 to – 1.46 versus – 2.11 without adsordents. The average improvement in body weight gain for the total period as % from aflatoxin $B_1$ alone was 78.71; 65.35; 50.96; 39.82; 32.93; 32.42; 22.87 and 17.16 for adsorbents IV ; VI ; VII ; V ; III ; II ; VIII and I, respectively. Generally, the diminished effect of aflatoxin on body weight ranged from 14.53 to 95.57% according to the kind of adsorbent and experimental duration. However, the best results were obtained by adding adsorbent IV. Diminished effect of the adsorbents on body weight gain agreed with the findings of Araba and Wyatt, (1991) who reported that 0.5 and 1% HSCAS diminished growth inhibitory effect on broiler chickens by 38 and 84%. Also, Kubena *et al.*, (1988) reported high diminshing effect (55 to 100%). Bentonite (0.5 and 1%) reduce the inhibitory effect of aflatoxin on growth rate of broiler chickens by 46 and 84% (Araba and Wyatt , 1991) and 87 and 89% for pigs (Lindemann *et al.*, 1993). MOS redued the liver cholesterol and liver fat levels which increased by aflatoxin (Park *et al.*, 1996). These results indicate that MOS decrease the aflatoxin effect. Reducation of aflatoxin effect by MOS may be due to its effect on stimulating the specific immune system (Savage *et al.*, 1996).

However, the differences between adsorbents in their ability to reduce mycotoxin toxicity depend on type and concentration of mycotoxin, the adsorbents, grinding diameter (Ramos and Hernandez, 1996 and Lemke *et al.*, 1998). The most important feature of the adsorption is the physical structure of the adsorbent, i.e the total charge and charge distribution, the size of the pores and the accessible surface area. On the other hand , the properties of the adsorbent molecules, the mycotoxins, like polarity, solubility, size, shape and in case of ionized compounds charge distribution and dissocation constants play a significant role too ( Huwig *et al.*, 2001 ) .

3- Mortality rate:

The mortality rate (Table 2) was significantly increased ($p \leq 0.05$) in fish fed aflatoxin $B_1$ contaminated diet (47.62% in comparison

with 4.76% for control). These results agreed with reported by El-Said, (1997) who reported that 3 mg aflatoxin/kg feed caused 16.76% mortality in *Oreochromis niloticus* after 90 days. The incidence of death may be due to the disturbance of organs function, since, the aflatoxicosis caused liver neoplasm, necrosis of hepatocytes and degenerative changes in pancreatic and kidney tissues of rainbow trout (Halver, 1967). Also, Lovell, (1991) reported that aflatoxin caused damage of liver and other organs, thereby caused poor growth, anemia, impaired blood clotting, sensitivity to burising, decreased immune responsiveness and increased mortality. Also, liver tumor, necrosis and basophilia of hepatocytes, largement of blood sinusoids in the kidney, accumulation of iron pigments in the intestinal mucosa and epithelium and necrosis of gastric glands can be caused. Post mortem examination for fish fed aflatoxin $B_1$ contaminated diet showed, pale liver with congested patches and pin point hemorrhages or yellowish in color. Distended gall bladder was noticed with pale kidney. These findings agreed also with the post morten lesions described by El-Said (1997).

Addition of adsorbents reduced ($p \leq 0.05$) the mortality rate. The reduction in mortality rate by adsorbent I, II & IV, was > VI, VII & VIII > III. Generally, all adsorbents reduced the mortality rate. Since, it ranged from 9.53 to 28.57% versus 47.62 % for aflatoxin alone. Although, the adsorbent I reduce the mortality rate the improvement in body gain was in low magnitude, these results may be due to its ability on stimulation of the immunity system (Savage *et al.*, 1996 and Shehata, 2002). These results for mortality agreed with the findings of Kubena *et al.*, (1991) who found that 0.5% HSCAS caused 68% decrease in the mortality rate of growing male turkey poults by aflatoxin. Also, Abd El-wahhab, (1996) reported that no mortality occurred in pregnant rats dosed orally with aflatoxin $B_1$ (2 mg/kg body weight) during gestation days 6-13 when combined with 0.5% HSCAS in comparison with 9% for aflatoxin alone. The decrease mortality rate by adsorbents may be due to there ability for absorption of mycotoxins in the gastrointestinal tract and thereby decreasing toxic effects on animals (Galvano, *et al.*, 2001).

4- Blood parameters :

Data of blood parameters determination are shown in Table (3). Total protein and albumin concentrations were significantly decreased in fish fed aflatoxin contaminated diet. These results agree with the results obtained by Mamdouh (1996) who found decrease in serum total protein of *Oreochromis niloticus* fed on ration containing 1, 2 and 3 ppm aflatoxin $B_1$ for 21,42 and 63 days. Also, El-Said (1997) reported that 1.5 and 3 mg aflatoxin/kg diet for 90 days decreased serum total protein for *Oreochromis aureaus*. The decrease in total protein and albumin may be attributed to: aflatoxin interaction with protein synthesis and cellular integrity in liver (Patterson, 1976), plasma proteins are used for energy production during pollutant toxicity or in increasing of protein catabolism induced by stress in order to supplementary energy (Mazeaud *et al.*, 1977 and Pfeifer and Weber, 1979), and binding of aflatoxin with DNA which lead to inhibition of DNA synthesis and RNA formation which is responsible for protein synthesis (Mamdouh, 1996).

**Table (3) : The effect of aflatoxin $B_1$ ( 9 mg/kg diet ) on serum constituents of fish and its modification by adsorbents**

| Parameters | Treatments | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Control | Aflatoxin | Afl+ I | Afl+ II | Afl+ III | Afl+ IV | Afl+ V | Afl+ VI | Afl+ VII | Afl+ VIII |
| Total protein (g/dl) | 4.29±0.04a | 3.07±0.22b | 3.11±0.29b | 3.77±0.24ab | 3.55±0.22b | 3.69±0.12ab | 3.62±0.06b | 3.33±0.01b | 3.11±0 28b | 3.21±0.22b |
| Index | 100 | 71.56 | 72.49 | 87.88 | 82.75 | 86.01 | 84.38 | 77.62 | 72.49 | 74.83 |
| Albumin (g/dl) | 2.97±0.09a | 2.40±0.25c | 2.47±0.03bc | 2.80±0.15ab | 2.47±0.17bc | 2.40±0.06c | 2.40±0.12c | 2.43±0.03c | 2.40±0 21c | 2.53±0.19bc |
| Index | 100 | 80.81 | 83.16 | 94.28 | 83.16 | 80.81 | 80.81 | 81.82 | 80.81 | 85.19 |
| AST (u/l) | 29.50±1.26cd | 38.67±1.20a | 34.67±1.45ab | 33.67±0.67bc | 28.33±1.86d | 33.33±0.88bc | 32.00±1.72bcd | 28.33±2.67d | 33.00±1.16bcd | 35.67±0.67ab |
| Index | 100 | 131.08 | 117.53 | 114.14 | 96.03 | 112.98 | 108.47 | 96.03 | 111.86 | 120.92 |
| ALT ( u/l) | 7.00±0.29b | 8.83±0.88a | 8.60±0.38a | 6.50±0.29b | 6.17±0.33b | 6.33±0.33b | 6.83±0.17b | 6.67±0.17b | 6.75±0 14b | 6.40±0.21b |
| Index | 100 | 126.14 | 122.86 | 92.86 | 88.14 | 90.43 | 97.57 | 95.29 | 96.43 | 91.43 |

**Means in the same row bearing different letters differ significantly ( $p \leq 0.05$ )**

Addition of adsorbents increased or improved ($p \leq 0.05$) the total protein and albumin. The total protein as % from the control ranged from 87.88 to 72.49% versus 71.56% for aflatoxin $B_1$ without adsorbent. Also, the albumin with adsorbents ranged from 94.28 to 80.81% versus 80.81 % for aflatoxin alone.

Aspertate aminotransferase (AST) and alanine aminotransferase (ALT) enzymes increased significantly ($p \leq 0.05$) in fish fed aflatoxin $B_1$ contaminated diet. These results agreed with the findings of Carpenter *et al.*, (1995) on rainbow trout; Mamdouh (1996) on *Oreochromis niloticus* and El-Said (1997) on *Oreochromis aureaus*. The increase in AST and ALT levels indicated to damage of the liver and probably kidney. Evidence for acute aflatoxin $B_1$ nephrotoxicity was provided by distended gall bladders indicating disrupte osmoregulation (i,e. water retention) as reported by Carpenter *et al.*, 1995).

It could be concluded from the results of this work that adding 0.5% adsorbents specially adsorbents IV (Mycobond) VI (mold stop super) to a diet contaminatad with 9 mg aflatoxin B1/kg may provide a safe and practial method for reduction of aflatoxicosis in fish.

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## تقليل سمية الأفلاتوكسين $B_1$ بالمواد المدمصة المختلفة فى السمك

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تم دراسة تقليل تسمم أسماك البلطي النيلي بالأفلاتوكسين $B_1$ بواسطة إضافة العديد من المواد المدمصة المنتشرة في مصر (٨ مواد ) إلى العليقة الملوثة في تجربة تغذية لمدة ٨ أسابيع . أستخدمت ٢١٠ سمكة بلطي نيلي متوسط وزنها عند البداية ٤٧جم، تم تقسيمها على عشرة معاملات ( ٢١ سمكة في كل معاملة ) واحتوت كل معاملة على ٣ مكررات، في كل مكرره ٧ سمكات . المعاملة الأولى غذيت على عليقة تجارية ( كونترول ) ، المعاملة الثانية غذيت على عليقة ملوثة بالأفلاتوكسين $B_1$ ( ٩ملجم / كجم عليقة )، والمعاملات الثمانية المتبقية احتوت نفس المستوى من الأفلاتوكسين $B_1$ + ٠,٥ % من المواد المدمصة المختلفة التي تم دراستها. المادة المدمصة الأولى كانت جدر خلايا الخميرة المحسنه ، المادة الثانية هي البنتونيت ، المادة المدمصة الثالثة تراي ستار ، المادة الرابعة ميكوبوند ، المادة الخامسة ايجي توكس ، المادة السادسة مولد ستوب سوبر ، المادة السابعة فنجستات - ك ، المادة الثامنة مولد ستوب ميكوبايند بلس.

الأفلاتوكسين $B_1$ عند اعطاؤه في عليقة السمك أحدث انخفاض معنوي في وزن الجسم . الإنخفاض في وزن الجسم الحي كنسبة منوية كان ٦,٠٩ ، ١١,٢٥، ١٧,٣٤ ، و ٢٢,٨٧ % عند الأسبوع٢، ٤ ، ٦ ، ٨ على التوالى. زيادة معنوية في نسبة النفوق حدثت نتيجة تناول الأفلاتوكسين $B_1$ حيث كانت النسبة ٤٧,٦٢% مقارنة بـ ٤,٧٦% للكنترول . كما حدثت تغيرات معنوية في مكونات الدم حيث زاد نشاط انزيمات الاسبرتيت أمينوترانزفيريز AST وكذلك الألانين أمينوترانزفيريز ALT ، بينما انخفض تركيز البروتين الكلي والألبيومين .

إضافة المواد المدمصة قللت التأثيرات السامة للافلاتوكسين معنويا حيث أحدثت : تقليل تأثير الأفلاتوكسين $B_1$ على وزن الجسم بمقدار ١٤,٥٣ إلى ١٠٤,٤٣% على حسب نوع المادة المدمصة وطول فترة التجربة . كما أحدثت انخفاض معنوى في نسبة النفوق عند إضافة المواد المدمصة وكذلك تحسن معنوى في مكونات الدم .

هذه الدراسة تقترح إضافة المواد المدمصة خاصة المادة الرابعة (ميكوبوند) والمادة السادسة (موند ستوب سوبر) لعلائق الأسماك الملوثة بالأفلاتوكسين$B_1$ لتقليل السمية .