Comparative ameliorative effect of Hydrated sodium calcium aluminosilicate and *Saccharomyces cerevisiae* (Brewer's yeast) against toxic impact of aflatoxin B₁ in *Oreochromis niloticus* (Nile tilapia)

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Abstract

Different ways have been used in an attempt to decrease the risk of aflatoxicosis in fish. This study was undertaken to compare the possible alleviative effects of hydrated sodium calcium aluminosilicate (HSCAS) and *Saccharomyces cerevisiae* against the toxic impact of aflatoxin B₁ (AFB₁) on *Oreochromis niloticus* (*O. niloticus*). Therefore, 180 normal cultured monosex *O. niloticus* were randomly allocated into 6 equal groups. Group 1, was received the basal ration only. Group 2, was fed a basal ration supplemented with 0.5% HASCAS. Group 3, was fed a basal diet enriched with 0.25% *Saccharomyces cerevisiae*. Group 4, was received a diet intoxicated with 2.5 ppm aflatoxin B₁. Group 5, was fed a diet intoxicated with 2.5 ppm AFB₁ with 0.5% HASCAS. Group 6, was fed a diet intoxicated with 2.5 ppm AFB₁ with 0.25% *S. cerevisiae*. AFB₁ intoxication induced mortality 16.67 %, leucopenia, lymphopenia, neutrophilia with a significant decrease in phagocytic % and index. Furthermore, significant increases in serum creatinine, ALT and ALP as well as a significant decrease in total protein, albumin and globulin were recorded. Moreover, accumulation of aflatoxin residues in *O. niloticus* flesh (5 ppb) and liver (15 ppb). While, supplementation of AFB₁ intoxicated diet either with *S. cerevisiae* or HSCAS ameliorated the drastic effects of aflatoxin on *O. niloticus* and *S. cerevisiae* appear to be more effective in the protection of fish from aflatoxicosis than HSCAS.

Keywords: Aflatoxin B₁, Residues, Hematology, Phagocytosis, HSCAS, *Saccharomyces cerevisiae*

Introduction

Mycotoxins are unavoidable contaminants in foods and feed stuffs and are a major problem throughout the world [1]. Aflatoxins (AFs) are a group of structurally related mycotoxins produced as food-borne metabolites by toxigenic strains of *Aspergillus parasiticus*, *Aspergillus flavus* and to lesser extent *Aspergillus nomius* [2]. Aflatoxins have a serious impact on the cultured fish, inducing disease with elevated death-rate and a steady decrease fish quality, in this manner revealing a critical issue in aquacultures [3]. Aflatoxin B₁ is the most common and toxic aflatoxins for human, land animals and aquatic organisms, due to its strong carcinogenic, immunosuppressive and mutagenic effects [4].

In spite of good screening programs, election of high quality feed ingredients and raw materials and good storage it is extremely hard to ensure the nonappearance of mycotoxins in aquaculture feeds. Subsequently, it is insistent to find appropriate ways to face the problem via an effective handling of the hazards caused by mycotoxins contamination [5]. Hydrated sodium calcium aluminosilicate (HSCAS) clay considered an easy, inexpensive and effectual way of aflatoxicosis as it firmly and specifically captures aflatoxins in GIT, lowering their bioavailability and accompanied problems [6,7].

Brewer's yeast (*Saccharomyces cerevisiae*) was found to have an important antagonistic role toward aflatoxicosis and has immunostimulant in chickens and quails [8,9]. The yeast preparations appear to be effective on a broad range of mycotoxins [10], due to the ability of glucomannans from *S. cerevisiae* to adsorb mycotoxins [11]. *S. cerevisiae* able to degrade aflatoxin and other mycotoxins as T-2 toxin and zearalenone [12,10]. Therefore, the present experiment was carried out to compare the preventive impacts of HSCAS and

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Saccharomyces cerevisiae against the drastic effects of AFB1 on Oreochromis niloticus.

Material and Methods

Aflatoxin B₁ (C₁₇H₁₂O₆), generated by toxigenic Aspergillus flavus utilizing polished raw rice as a growth substrate [13] with minor modifications [14].

HSCAS: Trade name, Condition feed, is commercial product made in India and imported by Pharma chemical international company and is composed of Hydrated sodium calcium aluminosilicate 100%.

Saccharomyces cerevisiae: Trade name, Diamond v original xp, it contains dried yeast (Saccharomyces cerevisiae) fermented product 100%, made in U.S.A.

Experimental Fish

One hundred and eighty apparently normal cultured monosex Oreochromis niloticus, with average body weight (35±5 g), were obtained from Abbassa Fish hatchery, Sharkia Governorate, Egypt. Fish were transported in polyethylene bags filled with one third dechlorinated water enriched by air (2/3) to the laboratory of Pharmacology Department, Faculty of Veterinary Medicine, Zagazig University. Fish were acclimated for 2 weeks and kept in glass aquaria filled with chlorine free tap water under laboratory conditions (natural photoperiod 12 h and temp (25.5±2 °C). Persistent aeration was kept in each aquarium by an electric air pump and temp was maintained by heaters. The water parameters (dissolved oxygen, pH and electric conductivity of the tap water) used in this study were measured using Hack Method (Sigma Laboratory) following WHO, (2001).

Diets used for experimental fish

The experimental fish were fed on basal diet (contained 30.38% crude protein and 3000 kcal/kg metabolizable energy which composed of fish meal, poultry by product, soybean, vegetable oils, wheat flour, yellow corn, minerals and vitamins mixture. The basal ration was formulated from commercial constituents and was compressed (1mm size pellets) at Fish Research Unit, Faculty of Veterinary Medicine, Zagazig University. Fish were fed twice daily (8 am and 2 pm) at the rate of 3% of their biomass.

Experimental protocol

After adaptation period, the healthy fish were haphazardly assigned into six equal groups (3 replicates/group), each replicate contains 10 fish kept in well prepared and persistently aerated aquarium (80x40x30cm) containing dechlorinated tap water, experiment had lasted for 42 days.

Group 1 received a basal ration only. Group 2 (HSCAS group): Fish were received a basal diet supplemented with 0.5% HSCAS [15]. Group 3 (S. cerevisiae group): Fish were received a basal ration enriched with 0.25% Saccharomyces cerevisiae [15]. Group 4 (AFB₁ group): Fish were received a diet intoxicated with 2.5 ppm aflatoxin B₁ [16]. Group 5 (AFB₁+HSCAS group): Fish were fed a diet intoxicated with 2.5 ppm AFB₁ and supplemented with 0.5% HSCAS. Group 6 (AFB₁+S. cerevisiae group): Fish were fed a diet intoxicated with 2.5 ppm AFB₁ and supplemented with 0.25% S. cerevisiae.

Hematological, immunological and biochemical analysis

Blood samples were collected on 1st, 2nd, 4th and 6th weeks of the experiment from the caudal blood vessels. Three blood samples were collected from each group. The 1st sample was gathered in clean sterilized tubes containing heparin as anticoagulant for estimation of phagocytic activity according to method previously illustrated [17,18]. The 2nd blood sample was collected in clean sterilized tubes containing EDTA for hematological examination. The 3rd blood sample was collected in plain centrifuge tubes without anticoagulant then centrifuged at 3000 rpm for 15 minutes for serum separation. Hepato-renal injury biomarkers were assessed in the separated serum. Serum alanine aminotransferase (ALT) was measured [19] and alkaline phosphatase (ALP) [20]. Total protein, albumin and creatinine were detected [21-23] respectively.
Measurement of AFB<sub>1</sub> residues

Muscle and liver samples from five fish of each group on 1<sup>st</sup>, 2<sup>nd</sup>, 4<sup>th</sup> and 6<sup>th</sup> weeks of the experiment, were pooled and thoroughly homogenized in a mortem. AFB<sub>1</sub> was extracted, filtrated and quantitatively measured by HPLC [24].

Statistical analysis

All data were analyzed using the SPSS program using one-way ANOVA. Duncan's Multiple Range Test (DMRT) was used to determine differences among means at significance level of 0.05.

Results

Effects on survival rate

Fish fed AFB<sub>1</sub> intoxicated diet showed 83.33% survival rate. Adding of HSCAS or S. cerevisiae to AFB<sub>1</sub> intoxicated diet increased the survival rate to 96.67%.

Effects on some hematological parameters

Inclusion of AFB<sub>1</sub> to the *O. niloticus* ration evoked a significant reduction in the total RBCs, Hb concentration and PCV% in descending manner at different experimental periods comparing with those fed a basal diet only (Fig 1 A, B & C). Group 5 and 6 revealed a significant elevation in RBCs count, Hb and PCV% compared to fish received AFB<sub>1</sub> intoxicated ration only (Group 4). Addition of *S. cerevisiae* to AFB<sub>1</sub> intoxicated diet (Group 6) induced a significantly higher RBCs count, Hb content and PCV% when compared with fish group treated with aflatoxin B<sub>1</sub> and HSCAS.

![Figure 1: Effect of aflatoxin B<sub>1</sub> and different antimycotoxins on serum level of RBCs (A), PCV% (B) and Hb concentration (C) of *O. niloticus* during different experimental periods](image-url)
Fish fed diet enriched with *S. cerevisiae* (Group 3) showed a marked elevation in total leukocytic and lymphocytic counts comparing with the control group, but fish in HSCAS group revealed non-significant change in comparison with the control group (Table 1). The total leukocytes count significantly decreased (P<0.05) in descending manner at different experimental periods in AFB₁ group. Leucopenia, lymphopenia and neutrophilia are the main picture of the leukogram of AFB₁ group. Fish of group 5 and 6 showed a marked increase in WBCs counts compared to fish in group 4 throughout the experimental periods. Addition of *S. cerevisiae* to AFB₁ intoxicated diet induced a significant increase in WBCs count and lymphocytic counts when compared with AFB₁+HSCAS group.

### Table 1: Effect of aflatoxin B₁ and different antimycotoxins on total WBCs, lymphocytes and neutrophils of *O. niloticus* at different experimental periods

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Period</th>
<th>G1</th>
<th>G2</th>
<th>G3</th>
<th>G4</th>
<th>G5</th>
<th>G6</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>WBCs (10⁶/mm³)</td>
<td>1st week</td>
<td>25.70 ± 0.05 b</td>
<td>25.69 ± 0.02 b</td>
<td>26.51 ± 0.05 a</td>
<td>22.46 ± 0.05 b</td>
<td>23.71 ± 0.04 c</td>
<td>25.02 ± 0.06 c</td>
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<tr>
<td></td>
<td></td>
<td>2nd week</td>
<td>25.60 ± 0.04 b</td>
<td>25.62 ± 0.03 a</td>
<td>26.60 ± 0.03 a</td>
<td>20.75 ± 0.07 c</td>
<td>23.02 ± 0.05 d</td>
<td>24.15 ± 0.03 c</td>
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<tr>
<td></td>
<td></td>
<td>4th week</td>
<td>25.58 ± 0.03 b</td>
<td>25.56 ± 0.01 a</td>
<td>26.70 ± 0.01 a</td>
<td>18.74 ± 0.03 c</td>
<td>21.76 ± 0.04 c</td>
<td>23.19 ± 0.03 c</td>
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<tr>
<td></td>
<td></td>
<td>6th week</td>
<td>25.67 ± 0.02 b</td>
<td>25.70 ± 0.02 c</td>
<td>27.25 ± 0.32 a</td>
<td>16.64 ± 0.24 a</td>
<td>20.48 ± 0.37 c</td>
<td>22.99 ± 0.37 c</td>
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<tr>
<td></td>
<td></td>
<td>1st week</td>
<td>46.00 ± 0.32 c</td>
<td>45.40 ± 0.32 c</td>
<td>44.00 ± 0.32 c</td>
<td>49.60 ± 0.24 b</td>
<td>48.40 ± 0.24 b</td>
<td>47.20 ± 0.37 c</td>
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<tr>
<td></td>
<td></td>
<td>2nd week</td>
<td>45.80 ± 0.37 d</td>
<td>46.00 ± 0.37 d</td>
<td>44.60 ± 0.24 d</td>
<td>55.00 ± 0.32 d</td>
<td>49.40 ± 0.24 c</td>
<td>48.00 ± 0.37 c</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4th week</td>
<td>46.20 ± 0.24 e</td>
<td>46.60 ± 0.24 e</td>
<td>44.60 ± 0.24 e</td>
<td>55.60 ± 0.32 e</td>
<td>47.40 ± 0.24 b</td>
<td>46.40 ± 0.24 e</td>
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<tr>
<td></td>
<td></td>
<td>6th week</td>
<td>45.80 ± 0.37 e</td>
<td>46.40 ± 0.37 e</td>
<td>44.20 ± 0.24 e</td>
<td>56.60 ± 0.32 e</td>
<td>46.40 ± 0.24 b</td>
<td>45.40 ± 0.37 e</td>
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<tr>
<td></td>
<td></td>
<td>1st week</td>
<td>38.00 ± 0.32 b</td>
<td>37.60 ± 0.32 b</td>
<td>39.80 ± 0.20 a</td>
<td>36.00 ± 0.24 d</td>
<td>37.60 ± 0.24 b</td>
<td>39.00 ± 0.24 d</td>
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<tr>
<td></td>
<td></td>
<td>2nd week</td>
<td>38.20 ± 0.37 b</td>
<td>38.80 ± 0.37 b</td>
<td>40.80 ± 0.37 b</td>
<td>39.60 ± 0.24 d</td>
<td>35.40 ± 0.32 e</td>
<td>38.40 ± 0.32 e</td>
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<tr>
<td></td>
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<td>4th week</td>
<td>38.40 ± 0.24 c</td>
<td>39.60 ± 0.24 c</td>
<td>40.60 ± 0.24 d</td>
<td>28.60 ± 0.24 e</td>
<td>36.80 ± 0.20 d</td>
<td>39.20 ± 0.20 b</td>
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<tr>
<td></td>
<td></td>
<td>6th week</td>
<td>38.40 ± 0.24 c</td>
<td>38.60 ± 0.24 c</td>
<td>41.00 ± 0.37 a</td>
<td>27.60 ± 0.37 a</td>
<td>38.8 ± 0.20 b</td>
<td>39.60 ± 0.24 b</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± standard error, n=5. Means within the same row carrying different superscripts are significant at (P<0.05).

**Effects on immune status**

The addition of biological antmycotoxin *S. cerevisiae* to the basal diet (Group 3) significantly increase both phagocytic % and index when compared with control group, while adding of HSCAS to the basal diet (Group 2) evoked non-significant changes in phagocytic % or index comparing with control (Fig 2 A & B). There was a significant decrease (P<0.05) in phagocytic % and index in descending manner at different experimental periods as indicator for nonspecific immunity in AFB₁ group. The addition either of HSCAS or *S. cerevisiae* to aflatoxicated diet significantly increased (P<0.05) both phagocytic % and index compared to AFB₁ group all over the experimental periods. AFB₁, *S. cerevisiae* group showed a significant improvement in the phagocytosis when compared to HSCAS+AFB₁ group.

**Effects on some biochemical parameters**

Fish fed diet intoxicated with 2.5 ppm AFB₁ displayed a marked elevation in ALT, ALP and creatinine (Figs 3 A, B & C). Those fish also displayed a marked reduction in...
serum total protein, albumin and globulin (Table 2). Addition of HSCAS or S. cerevisiae could ameliorate the alterations of these parameters compared with AFB₁ group. Group 6 showed significant decrease in ALT, ALP and creatinine as well as significant increase in serum total protein, albumin and globulin compared to group 5 throughout the experimental period.

**Effects on aflatoxin residues**

Exposure of fish to AFB₁ intoxicated diet for 42 day resulted in accumulation of aflatoxin residues in *O. niloticus* flesh and liver in ascending manner at different experimental periods till reach high levels (5 ppb) in muscle and (15 ppb) in liver at the end of experiment. Addition of chemical antimycotoxin HSCAS or biological one S. cerevisiae to AFB₁ treated diet significantly reduced (P<0.05) aflatoxin residues in both liver and muscle of *O. niloticus* all over the experimental periods. Fish in group 6 showed a significant reduction in aflatoxin residues followed fish in group 5 (data not shown).

**Discussion**

The mortality rate was increased in fish fed aflatoxin B₁ intoxicated diet (16.7%) in comparison with control group (0%). Fish death may be due to the organs dysfunction, anemia and impaired immunity caused by aflatoxicosis as recorded in our study. In similar way, Santacroce *et al.*, [3] stated that the mortality rate increased in fish fed diets contaminated with aflatoxin. Addition either of HSCAS or *S. cerevisiae* to aflatoxin toxicated diet, improved the survival rate (96%) when compared with aflatoxicated diet only. This could be attributed to the ability of both antimycotoxins to bind aflatoxin in the gastrointestinal tract decreasing its uptake and bioavailability [25]. Our results were reinforced by Pooramini *et al.*, [26].

**Figure 2:** Effect of aflatoxin B₁ and different antimycotoxins on serum level of phagocytic % (A), and phagocytic index (B) of *O. niloticus* at different experimental periods.
AFB$_1$ had adverse impacts on fish hematological parameters, as it significantly decreased RBCs, PCV%, Hb concentration, total WBCs and lymphocytes. Meanwhile, neutrophils significantly increased. Lowering of RBCs, PCV %, Hb concentration indicated anemia, possibly due to the hemopoietic organs damage mainly anterior kidney [27] or an increase of RBCs destruction in hematopoietic tissues [28]. While, the reduction in leukocytic count may be due to the release of epinephrine during stress, which is capable of causing the spleen contraction and a decrease of leucocytes count, which accordingly results in the weakening of the immune system [29], renders the fish vulnerable to infection. Besides, the release of neutrophils into the blood occurs as a non-specific response to a variety of stress stimuli in mammals and fishes [30]. Our findings were supported by those reported for fish aflatoxicosis in _O. niloticus_ [16,15].

Marked elevation in WBCs, RBCs, PCV % and Hb in groups 5 and 6 compared with fish in group 4. This can be explained by the ability of _S. cerevisiae_ to degrade mycotoxins and prevent their toxic effects [31] and the ability of HSCAS to bind AF strongly and prevent its absorption across the gastrointestinal tract [32]. Our results were reinforced by Osman et al., [33].

In the present study, AFB$_1$ significantly reduced both phagocytic % and index, which proved the immunosuppressive effect of aflatoxin. Our results were supported by Sahoo and Mukherjee, [34] who reported that AFB$_1$ cause suppression of neutrophil function, macrophage phagocytic activity, humeral immune response and globulin levels in rohu (_Labeo rohita_). Furthermore, Rodríguez-Cervantes et al., [35] stated that aflatoxins induced chronic alterations in the immune system of aquatic organisms.

![Figure 3: Effect of aflatoxin B$_1$ and different antimycotoxins on serum level of ALT (A), ALP (B) and creatinine (C) of _O. niloticus_ during experimental periods.](image-url)
Adding either HSCAS or *S. cerevisiae* to aflatoxicated diet significantly improved phagocytic % and index comparing to AFB1 intoxicated group. These results may be attributed to the ability of HSCAS to bind AFB1 in the gastrointestinal tract, reducing bioavailability to the blood stream [36]. Furthermore *S. cerevisiae* contains various immunostimulating compounds [37] beside its ability to capture the mycotoxin molecule changing it into nontoxic substance [10]. Our results are nearly agreed with Wang et al., [38] who reported that β-glucan enhance phagocytic index in *O. niloticus* fed on aflatoxin treated diet.

Concerning liver and kidney function, fish fed AFB1 treated diet showed a significant increase in ALT, ALP and creatinine as well as significant decrease in total protein, albumin and globulin indicating the stressful effects of AFB1 on the hepatic and renal tissues and impairment of their function. The reduction in total protein and albumin could be attributed partly to the damaging effects of AFB1 on hepatic cells [34], which was detected in this study, as evidenced by the increase in serum ALT and ALP activities, whereas the reduced globulin levels in AFB1-treated fish may be due the result of lymphocytolysis [39]. Our results were strengthened by those reported for aflatoxicosis in *O. niloticus* [40].

Supplementation of AFB1 toxicated diet either with HSCAS or *S. cerevisiae* could alleviate the alterations of biochemical parameters compared to AFB1 group. This could explain by the ability of *S. cerevisiae* and HSCAS to bind with mycotoxins and limit their bioavailability in the digestive tract and protect animals against its adverse effects. Our results were reinforced by those previously recorded [41,15,42].

Table 2: Effect of aflatoxin B1 and different antimycotoxins on serum total protein, albumin and globulin of *O. niloticus* during experimental periods

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameter</th>
<th>Period</th>
<th>G1</th>
<th>G2</th>
<th>G3</th>
<th>G4</th>
<th>G5</th>
<th>G6</th>
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<tbody>
<tr>
<td></td>
<td>Total protein (g/dl)</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>1st week</td>
<td>7.03 ± 0.09 a</td>
<td>7.02 ± 0.07 a</td>
<td>7.07 ± 0.07 a</td>
<td>4.94 ± 0.07 a</td>
<td>5.99 ± 0.03 a</td>
<td>6.21 ± 0.06 a</td>
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<tr>
<td>2nd week</td>
<td>7.19 ± 0.12 a</td>
<td>7.07 ± 0.11 a</td>
<td>7.13 ± 0.10 a</td>
<td>4.19 ± 0.04 a</td>
<td>5.36 ± 0.05 a</td>
<td>5.92 ± 0.03 b</td>
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<tr>
<td>4th week</td>
<td>7.03 ± 0.06 a</td>
<td>7.04 ± 0.06 a</td>
<td>7.08 ± 0.05 a</td>
<td>3.45 ± 0.07 a</td>
<td>5.15 ± 0.05 a</td>
<td>5.52 ± 0.04 a</td>
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<tr>
<td>6th week</td>
<td>6.94 ± 0.07 a</td>
<td>6.94 ± 0.06 a</td>
<td>7.01 ± 0.05 a</td>
<td>2.87 ± 0.09 a</td>
<td>5.01 ± 0.03 a</td>
<td>5.25 ± 0.02 a</td>
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<tr>
<td>1st week</td>
<td>4.20 ± 0.02 a</td>
<td>4.20 ± 0.02 a</td>
<td>4.22 ± 0.05 a</td>
<td>3.78 ± 0.02 c</td>
<td>3.99 ± 0.02 a</td>
<td>4.05 ± 0.02 a</td>
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<tr>
<td>2nd week</td>
<td>4.21 ± 0.02 c</td>
<td>4.21 ± 0.02 b</td>
<td>4.21 ± 0.05 c</td>
<td>3.20 ± 0.02 b</td>
<td>3.76 ± 0.02 b</td>
<td>3.93 ± 0.02 b</td>
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<tr>
<td>4th week</td>
<td>4.16 ± 0.01 c</td>
<td>4.18 ± 0.03 c</td>
<td>4.19 ± 0.10 b</td>
<td>2.99 ± 0.02 b</td>
<td>3.38 ± 0.10 a</td>
<td>3.47 ± 0.01 a</td>
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<tr>
<td>6th week</td>
<td>4.16 ± 0.01 a</td>
<td>4.17 ± 0.01 a</td>
<td>4.19 ± 0.02 b</td>
<td>2.42 ± 0.02 a</td>
<td>3.29 ± 0.02 a</td>
<td>3.36 ± 0.01 b</td>
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<td>1st week</td>
<td>2.83 ± 0.01 a</td>
<td>2.82 ± 0.02 a</td>
<td>2.85 ± 0.02 a</td>
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<td>1.92 ± 0.02 a</td>
<td>2.13 ± 0.01 a</td>
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<tr>
<td>2nd week</td>
<td>2.98 ± 0.10 a</td>
<td>2.86 ± 0.08 b</td>
<td>2.92 ± 0.08 a</td>
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<td>1.62 ± 0.02 b</td>
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<td>4th week</td>
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<td>1.77 ± 0.03 a</td>
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<tr>
<td>6th week</td>
<td>2.78 ± 0.06 b</td>
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<td>2.82 ± 0.07 a</td>
<td>0.44 ± 0.02 a</td>
<td>1.73 ± 0.02 a</td>
<td>1.89 ± 0.02 a</td>
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</table>

Values are expressed as mean ± standard error, n=5. Means within the same raw carrying different superscripts are significant at (Ps0.05).
Maintainable mycotoxin residues in fish are a food safety problem [43]. In the current work, exposure to 2.5 mg AFB₁/kg diet for 42 day resulted in accumulation of aflatoxin residues in Nile tilapia flesh and liver in ascending increase manner at different experimental periods till reach high levels (5 ppb) in muscle and (15 ppb) in liver at the end of experiment. Most of AFB₁ residues were recorded mainly in the liver. These findings indicate that liver has an essential role in AFB₁ metabolism, toxic metabolites activation or detoxification [44,45]. Our results strengthened by Deng et al., [46] who detected AFB₁ residues in Nile tilapia liver when received smaller doses of toxin (less than 2 mg/kg). Moreover, Rajeev Raghavan et al., [47] concluded that residual AFB₁ was detected at high levels (5 ppb) in fish musculature after prolonged feeding of sea bass with low levels of AFB₁.

Supplementation of AFB₁ toxicated diet either with HSCAS or S. cerevisiae reduced residues of aflatoxin significantly in liver and muscle of O. niloticus indicating the protection of fish liver and musculature against AFB₁ residues by HSCAS and S. cerevisiae through their ability to bind aflatoxin and formation of adduct which is not affected by the gastrointestinal tract enzymes consequently reduce the toxin bioavailability.

**Conclusion**

From the previously mentioned outcomes, it could be concluded that the supplementation of AFB₁ intoxicated diet either with 0.25% S. cerevisiae or 0.5% HSCAS succeed in mitigation of the drastic effects of aflatoxin on survival rate, hematology, phagocytosis and biochemical parameters as well as its residues in O. niloticus. Furthermore, S. cerevisiae appear to be more effective in protection of fish from aflatoxicosis than HSCAS.

**Conflict of interest**

The authors declare that they have no conflict of interest.

**Acknowledgments**

Authors would like to acknowledge Prof. Dr. Mahmoud Araf Mohamed, Chief Researcher of Biochemistry, Toxicology and Feed Deficiency Department, Animal Health Research Institute, El-Dokki, for his help in AFB₁ preparation.

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المتلازمة العربية

التأثُر التحليمي المقارن لكالسوم ألمونوسيلك الصوديوم وسكرومٍسز سرٍفٍسٍاي (خمرة بروٌر) ضذ التأثٍر السام للأفلاتوكسٍه B1 فً أسماك البلاط النٍلً عثذ انعهٛى فؤاد عثذ انعهٛى 1، عشج جلال 2، شٓٛزج يحًٕد 2، عشج جلال 1، شٓٛشج يحًٕد 2، ككح انطة انثٛطزٖ، جايعح انشلاسٚك، انشلاسٚك 44511، انشزلٛح، يصز 2يعٓذ تحٕز صحّ انحٕٛاٌ – يعًم فزعٗ انشلاسٚك – يصز ذعرثز انثزٔج انسًكّٛ يٍ أْى يصادر انثزٔذٍٛ انحٕٛاَٗ. ٔٔجٕد انسًٕو انفطزّٚ فٗ الأعلاف يٍ أْى انًشكلاخ انرٗ ذٕاجّ الاسرشراع انسًكٗ. ذى اجزاء ْذِ انذراسّ لاخرثار ذأثٛز سى الأفلاذٕكسٍ ٔيضاداخ انسًٕو انفطزّٚ عهٗ صحح ٔيُاعح ٔتماٚا سًٕو الأفلاذٕكسٍ فٗ أسًان انثهطٗ انُٛهٗ. فٗ ْذِ انذراسّ ذى اسرخذاو 181 سًكّ تهطٗ ٚرزأح أٔسآَى تٍٛ 35-41 جزاو ذى ذمسًٛٓى انٗ سرح ي جًٕعاخ (كم يجًٕعّ ذشًم 31: سًكّ) كم يجًٕعّ يمسًّ عهٗ ثلاثح أحٕاض، ٔانًجًٕعاخ كاٜذٗ انًجًٕعّ الأٔنٗ: ْٗ يجًٕعّ ضاتطّ ذرغذٖ عهٗ عهٛمّ بساسّٛ تذٌٔ أٖ اضافاخ. انًجًٕعّ انثانثّ: ْٗ يجًٕعّ ذرغذٖ عهٗ عهٛمّ بساسّٛ يع اضافّ 1,5 % سٛهٛكاخ انًجًٕعّ انزاتعّ: ْٗ يجًٕعّ ذرغذٖ عهٗ عهٛمّ بساسّٛ ذحرٕٖ عهٗ 2,5 يجى أفلاذٕكسٍ ب 1. عهٗ كجى عهٛمح يع اضافّ 1,25 % خًٛزِ انًجًٕعّ انخايسّ: ْٗ يجًٕعّ ذرغذٖ عهٗ عهٛمّ ذحرٕٖ عهٗ 2,5 يجى أفلاذٕكسٍ ب 1/كجى عهٛمح يع اضافّ 1,25% خًٛزِ. ٔأٔضحد انُرائج أٌ اضافح الأفلاذٕكسٍ انٙ عهٛمح الأسًان أدخ انٙ يعذلاخَفٕق انٙ 16%. ٔيع اضافح انسٛهٛكاخ ٔانخًٛزِ انٙ انعهٛمّ أدخ انٗ ذعذٚم ْذا الاَخفاض. الأسًان انرٙ ذى ذغذٚرٓا عهٙ عهٛمّ يهٕثّ تالأفلاذٕكسٍ أظٓزخ اَخفاض يعُٕ٘ فٙ َسثح انثزٔذٍٛ ٔالأنثٕٛيٍٛ ٔانجهٕتٕٛنٍٛ ٔذحسُد ْذِ انُرائج تاضافح كلا يٍ انسٛهٛكاخ ٔانخًٛزِ. الأسًان انرٙ ذى ذغذٚرٓا عهٙ عهٛمّ يهٕثّ تالأفلاذٕكسٍ أدخ انٙ ذعذٚم ْذا الاَخفاض يع اضافح يضاداخ انسًٕو أد٘ انٙ اَخفاض يعُٕٖ فٙ َسثح تماٚا سًٕو الأفلاذٕكسٍ فٙ انعضلاخ ٔانكثذ ْذِ انشٚادِ ذصاعذّٚ عهٙ يذار انرجزتّ ٔاضافح يضاداخ انسًٕو أد٘ انٙ اَخفاض يعُٕٖ فٙ َسثح تماٚا انسًٕو فٙ كلا يٍ انعضلاخ ٔانكثذ. ٔانجذٚز تانذكز اٌ اعطاء يضاد انسًٕو انكًٛٛائٙ (انسٛهٛكاخ).