TOXICITY OF AFLATOXIN AND OCHRATOXIN AND THEIR OCCURRENCE IN FEED AND SOME ANIMAL PRODUCTS

*Elham, A. El-shewy; **Mona, A. Ashoub and ***Samia, M. El-Hoshy

*Department of animal medicine; **Department of Hygiene
Fac. Vet. Med. Zagazig University, Benha Branch
***Department of food hygiene Animal Health Research Institute

ABSTRACT  A total of one hundred and twenty samples of different types of animal and poultry feeds; sera and some products like milk and egg, and ninety four samples of liver and kidney from beef calves; buffalo calves; sheep and camel were collected from different farms in Kalubia and Alexandria governorates. These samples were examined for presence of aflatoxin B1 (AFB1) and Ochratoxin A (OTA). The results indicated the presence of various levels of Aflatoxin B1 and Ochratoxin A in the examined samples. The results encouraged us for further experiment investigating some toxic effect of detected mycotoxins in poultry, where fifty (one-day old) Lohman chickens were given AFB1 or OTA or combination of (AFB1 and OTA) daily for five weeks. The biological investigations revealed that the examined mycotoxins altered some serum biochemical parameters. Furthermore various levels of mycotoxin residues were detected in liver, kidney and muscles of treated birds. The public health hazards of Aflatoxin and Ochratoxin as well as suggestive control measures were discussed.

INTRODUCTION

Almost all plant products can serve as substrate for fungal growth and subsequent mycotoxin formation, thus providing the potential for direct contamination of human food and animal feed. When farm animal used for production ingested feed contaminated with mycotoxins, not only may a direct toxic effect on animals occur but there may be a carry-over of the toxins into milk and meat, thus creating a further avenue for human exposure to mycotoxins (WHO, 1979). Afzal et al.,(1979) reported that Aflatoxin containing feeds may lead to Aflatoxin contaminated milk and observed that aflatoxin contamination of most agricultural plant crops used in dairy cattle rations including grains, oil seeds, root crops and some forage crops, have been demonstrated in many countries.

Bryden et al.,(1980) detected AFB1 in Australian feed stuffs known to be either water damage or visibly mouldy. Highest contamination level was 0.7 mg/kg with average of 0.14 mg/kg, of feed. Sanli et al.,(1982) examined 96 samples of feed ingredients for laying hens and broilers and found that AFB1 could be detected in 62 samples, AFB2 in 29 samples and AFG1 in 18 samples. The mean total aflatoxin levels ranged from from 4.01 to 9.92 mg/kg feed.

Abd-El-Haleem, (1983) stated that out of 191
Toxicity of Aflatoxin and Ochratoxin and their Residues in some Animal Products

respectively.

Sampling:

Blood samples were collected from wing vein, allowed to stand for one hour at room temperature and then centrifuged at 3000 r.p.m. for 15 minutes for separation of serum. Serum samples stored at -20°C for evaluation of the effect of the examined mycotoxins on serum profile. Five chickens from each group were slaughtered after 5±6 weeks of treatment and specimens of liver, kidney and muscles were used for detection of mycotoxin residues.

Biochemical investigation:

The levels of some serum enzymes were estimated according to the following methods:

*serum alkaline phosphatase and (GOT&GPT)
were estimated according to the methods adopted by Bessey et al.,(1946) and Reitman & Frankel (1957) respectively *Serum urea; creatinine and uric acid were determined after Patton & Crouch, (1977); Henry, (1974) and Fossati et al., (1980).

Serum glucose; sodium and potassium were determined according to the methods described by Trinder, (1969); Guder et al., (1982) and Henry, (1974)

Tissue residues for mycotoxins were determined as previously mentioned in the first part of this study.

Statistical analysis were conducted after Snedecor, (1971).

RESULTS

The results are recorded in tables 1, 2, 3, 4, 5 and 6.

DISCUSSION

Mycotoxins are considered unavoidable contaminants in foods & feeds because agronomic technology has not yet advanced to the stage at which preharvest infection of susceptible crops by fungi can be eliminated (Wood, 1992).

Table (1) shows that animal feed represents a considerable source for Aflatoxin either to animals or to human through contaminated edible animal products. As indicated in this table, buffalo feed showed the highest level of contamination (12.22 ± 4.007 µg/kg) followed by yellow corn (6.25 ± 3.75) µg/kg, this give indication that yellow corn represent an important source for aflatoxins when used as ingredient in formulation of different kinds of feed. On contrast to yellow corn, broad beans showed the lowest level of contamination (3.83±0.16) µg/kg. However, the variation in the Aflatoxin content of different feed ingredients is dependent on so many factors like degree of damage in the grains or the condition of storage (Lillehøj et al., 1976). Aflatoxin content of examined layer, duck and broiler feed were more or less similar (5.83±1.19); (5.00±2.22) and (5.0±0.6) µg/kg respectively. Several studies recorded the presence of mycotoxins in feeds e.g. Sanli et al.,(1982) who detected levels of 4.018 - 9.2 mg/kg of Aflatoxin in laying hens and broilers feed; Hassan, (1990) recorded 50-250 ppb AFB1 in broiler and layer protein concentrate respectively. Also, Hegazy et al.,(1992) recorded that 10.17 to 54% of examined layer; broiler and fish meal samples respectively were positive for Aflatoxin contamination. Finally, we have to note that the concentration of AFB1in yellow corn was equal to the higher limit of Egyptian standard (10 mg AFB1/kg). (Egyptian organization for standardization & quality control, 1990). In the same aspect the commercial buffalo feed have twice as the egyptian standard 1990.

Table (2) shows that the highest concentrations of AFB1 was in serum of laying hens, however, the levels of Aflatoxin in serum of broiler was under the detection level. Aflatoxin B1 was detected in buffalo and duck serum (1.00±0.25; 1.765±1.23 µg/kg) respectively. Regarding the presence of Ochratoxin A in feed, serum, as tabulated in tables (1&2) the concentrations of OTA in serum of broiler chickens, laying hens, ducks, and buffaloes represented 1.117; 1.36; 1.020; 0.824 and 0.333 as their corresponding levels in feeds. Also from data of tables 1&2 we can observe that in laying hens, the concentration of AFB1 in serum was approximately 3.36 times as its concentrations in ingested feed. Serum from ducks ingested Aflatoxin contaminated feed contain 1.153 AFB1as ingested feed, while the concentration of AFB1 in serum of buffalo was 0.491 times ingested feed. Also, Truckssess et al., (1983a) observed the presence of (0.05-0.07) ng/g in serum of laying. Similarly Sova et al., (1989) found AFB1 in serum of laying hens in a concentration of 3.1 - 4.3 & 2.2 - 4.0 µg/kg after 4.8 hours of single oral dose of 10 mg AFB1. Moreover Marquardt et al.;(1988)
found that blood Ochratoxin concentration was a good indicator for the tissue concentration. The occurrence of Aflatoxin B1 and Ochratoxin A in foods (edible tissues; egg and milk) was tabulated in Table (3) where (20%; 14.26%; 33.33%; 23.33%; 23.08%; 50%; 30%; 20%; 10%; 30% and 40%) (13.3%; 21.14%; 25%; 23.08%; 30%; 30%; 20%; 20%; 30% and 30%) of liver and kidney of of beef calves and buffalo calves; sheep and camel: egg and milk were contaminated by (51.8; 17.18; 15.4; 135.2; 155.6; 168.9; 120; 95.5; 5.187 and 4.25 and (41.35; 31.68; 75.8; 111.1; 91.8; 95.1; 49.47; 73.61; 38.88 and 47.26 ug AFB1 and OTA/kg respectively.

The highest concentration of AFB1 was present in kidney of sheep (198.8 ug/kg) followed by liver of sheep (155.5 ug/kg), while the highest concentration of OTA was present in kidney of buffalo calves (111.1 ug/kg) followed by kidney of sheep (95.1 ug/kg). Yadgiri and Tulbule (1975) demonstrated that the hepatic metabolism of AFB1 by the livers of sheep & goats was faster than by the livers of cows and buffaloes, suggesting a possible correlation between metabolism and species susceptibility to Aflatoxin. From our results we can conclude that the kidney is sensitive indicator of Aflatoxin exposure. AFB1 was previously recorded in liver, kidney of cows by Mckinney et al., (1973) and Shreeve et al., (1979), similarly AFB1 was recorded in cow’s liver and kidney by Stubblefield et al., (1983) and Truckssess et al., (1983b) while Ertado et al., (1979) detected the presence of AFB1 in liver and kidney of pigs, Metwally et al., (1983) and Fink-Gremmels (1985) observed AFB1 in edible tissues. Also from Table (3) we can observe that in eggs AFB1 was equal to 0.886 times as in ingested feed, but in milk it was equal to 0.348 as ingested feed. Similar results obtained by many authors like Masri et al., (1967) who found that AFB1 itself was identified in about 0.3% of the ingested dose in cows, while in lactating ewes it was 0.1% of ingested dose (Nabney et al., 1967) Truckssess et al., (1983a) found that the concentration of AFB1 in egg was 0.24ug/g. Regarding the presence of OTA in liver and kidney of cows, Galtier and Alvinerie (1976) detected 5 ug OTA/kg in liver & kidney of cows, while several authors like Nortensen et al., (1983) and Kofer et al., (1991) detected OTA in liver and kidney of pigs. A similar results were recorded for OTA in eggs by Juszkiewicz et al., (1982) who found that the consumption of 10 mg OTA in feed resulted in 0.13 ug OTA/kg eggs. Also, Shehab (1995) detected AFB1 in balady eggs. From toxicological point of view, Schiller et al., (1983) set a guide line value of 10 and 20 p.p.b to be the permissible limit of Aflatoxin and Ochratoxin in food respectively and 0.1 p.p.b for Aflatoxin in fluid milk. Accordingly the level of Aflatoxin and Ochratoxin for liver and kidney samples of all studied animal species as well as milk samples were exceeded this limit. As the same time Ochratoxin level in examine egg samples was above the permissible limit while the level of Aflatoxin was within the acceptable limit.

From hygienic health point of view Aflatoxin may be regarded as a potent toxin, a carcinogen, teratogen and mutagen (Ueno and Ueno, 197; and Chu 1992). Also Ochratoxin may be considered as an environmental major factor in the occurrence of human renal disease nephropathy tumor and cancer (Bacha et al., 1996, Crepp, 1996 and Simon 1996).

Concerning the effect of AFB1 on some biochemical parameters, the level of serum ALP showed significant and highly significant increase in the fifth and sixth week, this was shown in Table (4). Our results are similar to some previous studies; as Lynch et al., (1973) and Van Dijk et al., (1984) in calves. In contrast Nowar et al., (1983) found that Aflatoxin decreased the level of ALP in rats. This table also showed the effect of AFB1 on serum level of glutamic oxaloacetic transaminase activities (GOT). AFB1 increased the level of GOT in the fifth week but decreased below the control level in the sixth week. The level returned to normal after cessation of dosing by the end of the fifth week indicating reversible effect of AFB1 on liver, our data agree with the results of Lynch et al., (1973) and Van Dijk et al., (1984) in calves, but disagree with the results of Nowar et al., (1983) who found that Aflatoxin decreased the level of GOT in rats. Serum glutamic pyrovic transaminase (GPT) levels were illustrated in Table (4) where there is a decrease in its level, this agree with the data recorded by Nowar et al., (1983) in rats. The level of uric acid increased in fifth week but significantly decreased below the control level in the sixth week, while urea level in serum decreases.
Table (1):- Occurrence of aflatoxin B₁ and ochratoxin A in animal feeds (in μg/kg):

<table>
<thead>
<tr>
<th>Feed Type</th>
<th>Aflatoxin B₁</th>
<th>Ochratoxin A</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Range</td>
<td>Mean ±S.E.</td>
</tr>
<tr>
<td>Broiler feed</td>
<td>6.66 - 3.75</td>
<td>5.063 ±0.60</td>
</tr>
<tr>
<td>Layer feed</td>
<td>8.75 - 2.25</td>
<td>5.85 ±1.195</td>
</tr>
<tr>
<td>Duck feed</td>
<td>9.50 - 0.504</td>
<td>5.002 ±0.222</td>
</tr>
<tr>
<td>Yellow corn</td>
<td>10 - 2.5</td>
<td>6.25 ± 3.75</td>
</tr>
<tr>
<td>Broad beans</td>
<td>4 - 3.33</td>
<td>3.83 ±0.168</td>
</tr>
<tr>
<td>Commercial</td>
<td>20 - 6.66</td>
<td>12.22 ±4.007</td>
</tr>
<tr>
<td>buffalo feed</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
at fifth and sixth week. The decrease in serum level of urea reported in this study was previously reported in pigs by Harvey et al., (1969). In contrast, serum level of creatinine was significantly increased, a similar result was obtained by Harvey et al., (1969) in pigs. A significant decrease in serum level of glucose and sodium was observed in our results, this also reported in pigs by Harvey et al., (1989).

Concerning the effect of OTA on some biochemical parameters, serum uric acid level significantly increased in fifth and sixth week when compared with the control indicating impaired renal excretory function. These changes suggest a possible alteration in nitrogen metabolism, the level didn't return to normal after cessation of dosing indicating irreversible effect of OTA on kidney. Our results were agree with the results of Kubena et al., (1988) in chickens and Abou Salem et al., (1994) in rats. The serum level of creatinine and potassium decreased in fifth week but increased in the sixth week, while serum urea level increased significantly in the fifth but decreased in the sixth week, this agree with the results of Kubena et al., (1988) in chickens and Abou Salem et al., (1994) in rats. The level of ALP, glucose and sodium in serum decreased significantly in fifth and sixth week of the experiment, this disagree with the results recorded by Kubena et al., (1983) and Bailey et al., (1989) in chickens. The level of GOT and GPT significantly increased in the fifth week then decreased in the sixth week, our results are similar to the results of Kubena et al., (1988) in chickens.

The coinoculated chickens with (AFB1 + OTA) together showed high significant increase in serum level of ALP, this effect mainly due to the effect of Aflatoxin because OTA alone didn't produce this increase, the present result agree with the result of Harvey et al., (1989) in pigs. Concerning the GOT, the combined toxicity significantly decreased the serum level of GOT. Also, AFB1 + OTA significantly decreased the serum level of GPT, this effect resemble the AFB1 effect. The effect of AFB1 + OTA on serum level of urea nitrogen was shown in Table 4 where the toxins produce a significant decrease in serum urea level. Similarly, Tapia & Seawright (1985) and Harvey et al., (1989) reported a reduction in urea nitrogen concentration with AFB1 + OTA combination. The mechanism of this interaction is poorly understood; however, the presence of Aflatoxin in renal proximal tubular cells may interfere with renal uptake or binding of OTA, therefore, reducing the ability of OTA to cause renal damage (Tapia & Seawright, 1985). Moreover, both mycotoxins produced highly significant higher levels of creatinin and significantly lower level of uric acid at the fifth week, then higher level in the sixth week, thus supporting the possibility of Aflatoxin interference with renal OTA metabolism. Also, both mycotoxins produced lower serum level of glucose and sodium, this observation was previously recorded in pigs by Harvey et al., (1989).

From Table 5 we observed that AFB1 appeared in chicken tissues after five weeks of treatment with AFB1, while AFM1 (produced by hydroxylation of the C-3 position of the bis-furan ring of AFB1) disappeared at the sixth week, also OTA was detected in chicken tissues after one week of cessation of treatment. The appearance of AFB1 in tissues of treated chickens after five weeks of treatment may be due to the normal enzymatic capacity of the liver to metabolize and excrete Aflatoxins is exceeded (Hendrickse, 1991). AFM1 & AFG1 disappeared from tissues of treated chickens with AFB1 in the sixth week. The concentrations of some mycotoxins residues at various interval of the experiment were also shown in Table 5, where we observed that the concentration of AFB1 increased from the fifth to sixth week of treatment which may be due to insufficient metabolism of Aflatoxins due to its hepatotoxic effect, in contrast to our results. Truckess et al., (1983) and Sova et al., (1989) mentioned that after 7 days of stopping treatment chickens with AFB1, the concentration of AFB1, in tissues decreased. On the other hand AFM1 & AFG1 disappeared in the sixth week, the concentration of OTA decreased from the fifth to sixth week.

The distribution of AFB1; AFM1; AFG1 and OTA in liver; kidney and breast muscle of chickens due to treatment with AFB1 and OTA was shown in Table 6, where treatment with AFB1 leads to appearance of AFB1 by 12%; 20% and 20%; AFM1 by 8%; 20% and 8% and AFG1 by 55%; 0.0% and 2% in liver; kidney and muscle of treated chickens respectively. While dosing AFB1 + OTA resulted in appearance of AFB1; AFM1; AFG1 and OTA in liver; kidney and muscle by
Toxicity of Aflatoxin and Ochratoxin and their Residues in some Animal Products

Table (2): Occurrence of aflatoxin B₁ and ochratoxin A in serum of chickens, ducks & animals (in ug/kg):

<table>
<thead>
<tr>
<th>Animal</th>
<th>Aflatoxin B₁</th>
<th>Mean ±S.E.</th>
<th>Ochratoxin A</th>
<th>Mean ±S.E.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Broiler</td>
<td>UDL</td>
<td>UDL</td>
<td>62.34 - 30</td>
<td>41.89 ± 10.27</td>
</tr>
<tr>
<td>Layer</td>
<td>*35.23 - 4.13</td>
<td>19.68 ±15.55</td>
<td>133.33 - 80</td>
<td>104.44 ±15.55</td>
</tr>
<tr>
<td>Duck</td>
<td>7.00 - 4.531</td>
<td>5.766 ± 1.23</td>
<td>85 - 25</td>
<td>58.33 ±17.64</td>
</tr>
<tr>
<td>Buffalo</td>
<td>6.25 - 5.75</td>
<td>6.00 ± 0.25</td>
<td>33.33 - 22.22</td>
<td>28.47 ±12.86</td>
</tr>
</tbody>
</table>

N.B.: UDL = under the detectable level * = one sample over the detectable level

Table (3): Occurrence of aflatoxin B₁ and ochratoxin A in edible tissues (in ug/kg):

<table>
<thead>
<tr>
<th>Item</th>
<th>Total samples</th>
<th>Positive samples</th>
<th>Contamination ppb</th>
<th>Total samples</th>
<th>Positive samples</th>
<th>Contamination ppb</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>Range</td>
<td>Mean</td>
<td>No</td>
<td>%</td>
</tr>
<tr>
<td>Beef calves</td>
<td>15</td>
<td>3</td>
<td>20%</td>
<td>25.9-70.1</td>
<td>51.8</td>
<td>2</td>
</tr>
<tr>
<td>a) Liver</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b) kidney</td>
<td>14</td>
<td>2</td>
<td>14.29%</td>
<td>16.16-18.2</td>
<td>17.18</td>
<td>3</td>
</tr>
<tr>
<td>Buffalo calves</td>
<td>12</td>
<td>4</td>
<td>33.3%</td>
<td>85.3-199.2</td>
<td>150.4</td>
<td>3</td>
</tr>
<tr>
<td>a) Liver</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b) kidney</td>
<td>13</td>
<td>3</td>
<td>23.08%</td>
<td>20.8-211</td>
<td>135.2</td>
<td>3</td>
</tr>
<tr>
<td>Sheep a) Liver</td>
<td>10</td>
<td>5</td>
<td>50%</td>
<td>85.8-233.3</td>
<td>155.5</td>
<td>3</td>
</tr>
<tr>
<td>b) kidney</td>
<td>10</td>
<td>3</td>
<td>30%</td>
<td>95.5-311</td>
<td>198.9</td>
<td>3</td>
</tr>
<tr>
<td>Camel a) Liver</td>
<td>10</td>
<td>2</td>
<td>20%</td>
<td>51.8-188.2</td>
<td>120</td>
<td>3</td>
</tr>
<tr>
<td>b) kidney</td>
<td>10</td>
<td>1</td>
<td>10%</td>
<td>95.5</td>
<td>-</td>
<td>2</td>
</tr>
<tr>
<td>Egg</td>
<td>10</td>
<td>3</td>
<td>30%</td>
<td>4.53-6.50</td>
<td>5.187</td>
<td>3</td>
</tr>
<tr>
<td>Milk</td>
<td>10</td>
<td>4</td>
<td>40%</td>
<td>4.5</td>
<td>4.25</td>
<td>3</td>
</tr>
</tbody>
</table>

Table (5): Percentage* and concentrations (in ug/kg) of different mycotoxin residues in tissue of chickens at various intervals of the experiment:

<table>
<thead>
<tr>
<th>Time in weeks</th>
<th>Fifth week</th>
<th>Sixth week</th>
</tr>
</thead>
<tbody>
<tr>
<td>%</td>
<td>Range</td>
<td>Average</td>
</tr>
<tr>
<td>AFB₁</td>
<td>33.33%</td>
<td>12.5-33.33</td>
</tr>
<tr>
<td>AFM₁</td>
<td>53.33%</td>
<td>4.5-12.5</td>
</tr>
<tr>
<td>AFG₁</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>OTA</td>
<td>13.33%</td>
<td>100-166.66</td>
</tr>
<tr>
<td>Total no of samples</td>
<td>15</td>
<td>12</td>
</tr>
<tr>
<td>% to whole +ve samples</td>
<td>27.27%</td>
<td>21.82%</td>
</tr>
</tbody>
</table>

* Percentage to whole no of +ve samples during the same week

Alex. J. Vet. Sci., Vol. 13, No. 3, October 1997 225
Table 6: Percentage and correlation (r/kg) of some mycosis in some chicken liver due to treatment (OTA and AFL)

<table>
<thead>
<tr>
<th>Treatment</th>
<th>OTA</th>
<th>AFL</th>
<th>r/kg</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>0</td>
<td>0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Treatment 1</td>
<td>0.2</td>
<td>0.1</td>
<td>0.3</td>
<td>0.4</td>
</tr>
<tr>
<td>Treatment 2</td>
<td>0.4</td>
<td>0.3</td>
<td>0.5</td>
<td>0.6</td>
</tr>
<tr>
<td>Treatment 3</td>
<td>0.6</td>
<td>0.5</td>
<td>0.7</td>
<td>0.8</td>
</tr>
<tr>
<td>Treatment 4</td>
<td>0.8</td>
<td>0.7</td>
<td>0.9</td>
<td>1.0</td>
</tr>
</tbody>
</table>

Note: OTA and AFL are the types of mycosis, and r/kg is the correlation coefficient.


Egyptian Organization for Standardization and Quality Control (1990):


Toxicity of Aflatoxin and Ochratoxin and their Residues in some Animal Products

pp.525.


الملخص العربي

"سمية الأفلاتوکسين وماكرونوكاس و التواليا في الأعلاف ومن بعض المنتجات الحيوانية.

د. الهام الشيوى
د. منى عشوب
كلية الطب البيطري
معهد بحوث صحة الحيوان

تم تجميع عدد من الاعلاف وإعداد عينات مختلفة من أعلا و معالج بعض الحيوانات و الطيع، و بعض المنتجات الحيوانية مثل الألبان و البيض و باقي عينة بحثية من (اعلاف
كل من دجاج التشمان و الدجاج البياض و البيض و ألعاب الجاموس و قوا الصويا و الجذرة الصفراء).

بالإضافة إلى عشيرة عينات مصل من كل من دجاج التشمان و الدجاج البياض و البيض و
الجاموس و عشيرة عينات من كل من البيض و لون الجاموس. أيضا تم تجميع أربع و تسعة
عينة (كبد و كلي) من عجل التشمان و الجاموس و الألعاب و الجذرة من محافظتين القيروانية
و السكندرية. تم استيفاء وجود الأفلاتوکسين ب 1 و الأوكراوكسین ب 1 و الأوكراوكسین ب فى كل العينات
السابقة . أظهرت النتائج تواجد مستويات من الأفلاتوکسين ب 1 و الأوكراوكسین ب فى
العينات المفحوصة. شجعتنا هذه النتائج على إجراء تجربة بيولوجية لمعرفة التأثير
السمى للمسوم الفطري الموجودة على الدجاج حيث تم إعطاء عدد خمسين كتكوت (سن
يوم) الأفلاتوکسين ب 1 أو الأوكراوكسین ب 1 أو مزيج منهما يوميا لمدة خمسة أسابيع.

أظهرت النتائج حدوث تغييرات بيوكيميائية مختلفة في مصل الدجاج بالإضافة إلى وجود
متغيرات من تلك السعوم في كبد و كلي و عضلات الطيع بالإضافة إلى هذه السعوم. أخيرا تم
مناقشة مخاطر الأفلاتوکسين ب 1 أو الأوكراوكسین ب على الصحة العامة وكذلك طرق
الوقاية من هذه السعوم .