Correlation between broiler aflatoxicosis and European production efficiency factor

A. M. Shareef*, O. S. Bayon** and T. S. Qubih**

*Department of Veterinary Public Health, **Department of Pathology, College of Veterinary Medicine, University of Mosul, Mosul, Iraq

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Abstract

Ten broiler flocks claimed to be suffering from aflatoxicosis in Ninavah governorate were subjected to study the correlation between natural contamination of feed samples with aflatoxin and European production efficiency factor (EPEF). Enzyme-linked immunosorbent assay (ELISA) method was adopted for estimation of AF level in broiler feeds. Growth performance parameters were recorded including final body weight, mortality, feed consumption and conversion ratio. Aflatoxins levels in feeds were ranged from 31 to 2381.8 ppb and EPEF was between 91.55-151.05. There was a highly negative correlation between AF concentration in broiler flock feeds and their EPEF (-0.828).

Keywords: Aflatoxin; Chicken; Feed.

Introduction

Aflatoxins are one of the most potent toxic substances that occur naturally. These are a group of closely related mycotoxins produced by fungi Aspergillus flavus and A. parasiticus (1). Aflatoxins are normally refers to the group of difuranocoumarins and classified in two broad groups according to their chemical structure; the difurocoumarocyclopentenone series (AFB1, AFB2, AFG1, AFM1, AFM2, AFM2A and aflatoxicol) and the difurocoumarolactone series (AFG1, AFG2, AFG2A, AFGM1, AFGM2, AFGM2A and AFB3) (2). Among 18 different types of aflatoxins identified, major members are aflatoxin B1, B2, G1 and G2 that contaminate agricultural products.
commodities and pose a potential risk to poultry (3). Aflatoxicosis, is one of the most commonly seen mycotoxins in commercial poultry worldwide, and here in Iraq (4). Aflatoxin contamination is more likely in the tropics or subtropics (5). In this survey, we tried to correlate natural aflatoxin broiler feed contamination with their production efficiency factor.

Materials and methods

Chicks and diet

Ten broiler flocks, reared in Al-Hamdanyea, at the east of Mosul province, were occupied with varying numbers of broilers. These flocks were subjected to weekly visits by veterinarians throughout 2006. It was revealed that repeated outbreaks of Aflatoxicosis, with different percentages of mortality had occurred. In most cases, broiler chickens show reduced weight gain, pale comb, and in sever cases their bellies were filled with fluids. On autopsy an edematous viscera, enlarged pale livers with lesions, enlarged kidney and spleen, and sometimes hydro pericardium was observed. The changeover from aflatoxin contaminated to AF free feed always improve the condition of affected birds, implicating that the levels of AF in the feed coupled with the bad management were the two main causes of frequent outbreaks of Aflatoxicosis.

Feed sampling

Broiler feed samples, were collected from 10 various broiler farms, at a rate of 500 gms/ton. They were analyzed for AF contamination in private veterinary laboratory in Mosul city.

Aflatoxin assay

Estimation of aflatoxin concentration in broiler feeds had been carried out using Neogen direct competitive ELISA method. It was briefly as follows: Five Gms sub sample from each collected 500 Gms, were grounded to pass 20 meshes. Extraction was performed with 25 ml 70% (v/v) methanol/water. Extracts were mixed with conjugate and then added to antibody –coated micro wells. After room temperature incubation, plates were washed and enzyme substrate was added and left to incubate for 30 minutes. Stop solution was added in the final step of analysis. Color intensity was measured optically with a micro plate reader ELx 800 at wavelength of 650 nm. Results were obtained by using computerized Neogen Veratox Software programmed version 2.0.16 (Neogen Corporation), USA.

EPEF calculation

For analysis of performance traits such as: live body weight, feed consumption, feed conversion ratio and mortality, EPEF (5) was used and estimated as follows:

\[
EPEF = \frac{C \times 1000}{A \times \frac{B}{\text{Age (days)}}}
\]

where:
- \(A\) = Total weight of live birds intended for slaughter
- \(B\) = Total weight of live birds intended for slaughter
- \(C\) = Total No. of reared birds
- \(B\) = Age (days)
- \(A\) = Total feed consumption (kg)

Results

Dead and eviscerated broiler birds from broiler flocks claimed from aflatoxicosis, showed pathological changes similar to changes in experimental aflatoxicosis. Liver, the principle organ, showed the characteristics lesions of yellow, ocher discoloration, with multifocal hemorrhages, and developed whitish foci due to the increase in liver lipid content (Figure1).

![Figure 1: Lethal aflatoxicosis in broiler birds caused liver congestion, discoloration, enlarged pancreas. Aflatoxin (1087 ppb) was detected in the feed given to these broilers](image)

The mean of aflatoxin (ppb) in the tested broiler feeds are presented in Table 1.
Table 1: Mean aflatoxin concentration in broiler feeds.

<table>
<thead>
<tr>
<th>Farm</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
<th>F</th>
<th>G</th>
<th>H</th>
<th>I</th>
<th>J</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean AF ppb</td>
<td>433</td>
<td>372</td>
<td>908</td>
<td>519</td>
<td>391</td>
<td>280</td>
<td>518</td>
<td>619</td>
<td>1087</td>
<td>425</td>
</tr>
</tbody>
</table>

From table 1, it is evident that mean AF concentration in these broiler feed were ranged from 280 ppb in farm F, to 1087 ppb in farm I. All feed samples collected from the diets of 10 broiler flocks showed high levels of AF contamination. These levels were higher than the permissible AF level of 20 ppb. Since, the lower level detected was that estimated in farm (F), and had AF level 10 times (280 ppb) more than the permissible limit. Two farms (B, E), (A, J), and (D, G), had levels between 300-400 ppb, 400-500 ppb and 500-600 ppb AF, respectively. Higher levels of AF, 600-700 ppb, 900-1000 ppb, and 1000-1100 ppb, were detected in the feed samples obtained from flocks (H, C and I), respectively.

The results of EPEF of the various broiler farms are presented in Table 2.

Table 2: European production efficiency factor of the various 10 broiler farms

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Farm</th>
<th>Total No. of birds housed</th>
<th>No. of dead birds</th>
<th>Mortality %</th>
<th>No. of slaughtered birds</th>
<th>Final mean BW of live birds(kg)</th>
<th>Total BW of slaughtered birds(kg)</th>
<th>Feed conversion ratio (kg/kg)</th>
<th>Total feed consumption (kg)</th>
<th>EPEF</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
<td>7845</td>
<td>1251</td>
<td>15.9</td>
<td>6594</td>
<td>1.75</td>
<td>11539</td>
<td>2.61</td>
<td>29160</td>
<td>118.79</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>10781</td>
<td>980</td>
<td>9.09</td>
<td>9801</td>
<td>1.85</td>
<td>18131.85</td>
<td>2.34</td>
<td>41200</td>
<td>151.05</td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>8810</td>
<td>2100</td>
<td>23.8</td>
<td>6710</td>
<td>1.65</td>
<td>11071</td>
<td>2.90</td>
<td>30840</td>
<td>92.07</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>8060</td>
<td>1560</td>
<td>19.3</td>
<td>6500</td>
<td>1.69</td>
<td>10985</td>
<td>2.70</td>
<td>29000</td>
<td>105.35</td>
</tr>
<tr>
<td></td>
<td>E</td>
<td>10648</td>
<td>1100</td>
<td>10.2</td>
<td>9548</td>
<td>1.80</td>
<td>17186</td>
<td>2.48</td>
<td>41393</td>
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<tr>
<td></td>
<td>F</td>
<td>12417</td>
<td>918</td>
<td>7.98</td>
<td>10581</td>
<td>1.90</td>
<td>20103</td>
<td>2.29</td>
<td>44724</td>
<td>148.52</td>
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<tr>
<td></td>
<td>G</td>
<td>11016</td>
<td>1800</td>
<td>16.3</td>
<td>9216</td>
<td>1.70</td>
<td>15667</td>
<td>2.70</td>
<td>40864</td>
<td>111.28</td>
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<tr>
<td></td>
<td>H</td>
<td>9391</td>
<td>1870</td>
<td>19.91</td>
<td>7521</td>
<td>1.65</td>
<td>12409</td>
<td>2.84</td>
<td>34000</td>
<td>98.43</td>
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<tr>
<td></td>
<td>I</td>
<td>7285</td>
<td>1785</td>
<td>24.5</td>
<td>5500</td>
<td>1.65</td>
<td>9075</td>
<td>2.89</td>
<td>25200</td>
<td>91.55</td>
</tr>
<tr>
<td></td>
<td>J</td>
<td>10114</td>
<td>1460</td>
<td>14.4</td>
<td>8654</td>
<td>1.75</td>
<td>15144</td>
<td>2.63</td>
<td>38616</td>
<td>119.84</td>
</tr>
</tbody>
</table>

From table 2, it is evident that there were different mortality percentages among different flocks, and were ranged from 7.98% in flock (F) to 24.5% in flock (I). It is also clear that there was a clear relation between feed AF contamination level and mortality percentages, i.e.; on an average there was 2% increase in mortality rate for each 100 ppb increase in feed AF contamination level.

Data of mean live body weight in different flocks showed proportional reduction of about 100 grams for each 100 ppb increase of feed AF contamination level. Feed
conversion ratio in different flocks was adversely affected by about 0.1 for each 100 ppb increase of feed AF contamination level.

EPEF in various broiler flocks, ranged from 92.07 in flock (C) to 151.05 in flock (B). This range is clearly associated with the growth parameters in various flocks. Thus, there were two types of correlations; Positive one between EPEF &three growth parameters namely, final body Weight (0.9787) (Figure 2), total body weight of slaughtered birds (0.8477) and feed consumption (0.7940) (Figure 3). The negative type of correlation was between EPEF and feed conversion (-0.9904) (Figure 4) and mortality percentages (-0.9799) (Figure5). From table1 and 2, there was also a negative correlation between aflatoxin and EPEF (-0.828) (Figure 6).

![Figure 2](image2.png)  
**Figure 2:** The negative correlation between aflatoxin concentration and live body weight.

![Figure 3](image3.png)  
**Figure 3:** The negative correlation between aflatoxin concentration and feed consumption.
Figure 4: The negative correlation between aflatoxin concentration and feed conversion.

Figure 5: The negative correlation between aflatoxin concentration and mortality.

Figure 6: The negative correlation between aflatoxin concentration and EPEF.
Discussion

Aflatoxin contamination of broiler feed commodities, and the natural aflatoxicosis in broilers have been reported world wide. In this study, broiler flocks showed both acute and chronic signs of aflatoxicosis, these signs were uneven growth, ataxia, recumbency, backward extended legs, paleness, poor appearance and varying percentages of mortality. Post-mortem findings of affected birds from these flocks show friable, enlarged, yellow colored, reticulated livers. Some birds exhibited enlarged pancreas and spleen. Others (6-8) also reported these symptoms and changes of field aflatoxicosis.

Jones et al. (9), defined poor broiler growers, those fed as low as 14 ppb aflatoxin, and they concluded that productivity losses in commercial broiler operations could occur when aflatoxin concentrations were below those shown by controlled research to be of concern in laboratory situation. Aflatoxin concentration associated with field problems has been reported to range from 30 ppb to 101,000 ppb (10), or feeding aflatoxin at a rate of 0.5, 1.25, and 2.5 ppm (11-14). In Mosul province (Iraq), field aflatoxicosis in broiler chickens had also been reported through feeding diets naturally contaminated with 38.4-511 ppb aflatoxin (4). Doer et al (15), found that feeding broilers for 7 wks feed naturally contaminated with aflatoxin ranged from 65-2700 ppb, showed significant reduction in broiler performance (reduction in growth rate and feed consumption and conversion), and increase in liver fat content. Others (16,17) also found that feeding broilers aflatoxin for 4 wks at a rate of 500 to 1000 ppb caused a reduction in body weight and feed conversion ratio.

In this study, mean aflatoxin concentrations of naturally contaminated feeds were ranged between 280-1087 ppb. Although this range occurs below the acute aflatoxin level of 2.5 mg/kg feed and more, and for poultry producers, sub clinical losses are of great economic importance than losses from acute effects. The clinical signs and lesions reported here gave an impression that these flocks were suffered from acute aflatoxicosis. This could be attributable to the interaction with other mycotoxins, (although not tested here), and interaction with the stresses of environment and production (18,19). It is also important to say that in field mycotoxicosis, naturally contaminated feeds are more toxic than feeds with the same level of a pure mycotoxin supplemented into the diet. Moreover, aflatoxin produced from culture is more toxic than pure aflatoxin added to the diets (20). Because broilers were fed a blend of compound feed and because mold produces an array of mycotoxins, many mycotoxins interactions are possible. The effect of aflatoxin on birds performance could be explained by the reduction in amount of nutrients available for use by birds, i.e., through reduction of vitamins, amino acids and energy, and could be due to GIT irritation and reduction in nutrients absorption (21). Therefore, the mechanism by which aflatoxin reduce growth rate is probably related to disturbances in protein, carbohydrate and lipid metabolism (22). Gross liver lesions were noticed in examined broiler birds by many dead and succumbed ones. These changes were also reported to be occurred when birds fed aflatoxin B1 at a rate of 500-1000 ppb (23). Here, mortalities in broiler flocks were recorded up to 25%. Rajion and Farrell (24), reported higher mortalities (58-73%) through feeding naturally aflatoxin contaminated moldy wheat. Mortalities due to aflatoxicosis could be moderated by many factors, such as sex, age, diet, and duration of exposure. However, aflatoxin can increase incidence of disease, through reduction in immunity, and may exhibit many clinical symptoms mixed with many disease profile (25-27). Broiler performance, expressed by EPEF formula, (which depends on number and weight of birds at the end of rearing period, their slaughter age, and the amount of feed they consumed), ultimately reflects the effect of any factor that could play a role in final production profile. The results represented in Figures 2-6, showed a clear negative relationship between aflatoxin concentration and overall production profile expressed in EPEF in broiler flocks. The negative correlations between aflatoxin concentration of broiler feeds and corresponding EPEF were (-0.9789) for live body weight; (-0.8477) for total body weight of slaughtered birds; (-0.7940) for feed consumption; (0.9904) for feed conversion and (0.9799) for mortality. There was a gradual decline in EPEF with each increase in aflatoxin concentration in broiler feeds. For each 100 ppb increase in aflatoxin in broiler feed, there was +2.25% increase in mortality per 10000 birds; -3 gms/bird in live body weight; -4 ton in feed consumption per 10000 birds; +0.27/bird for feed conversion ratio, based on the original number of birds housed centers of mycotoxin prevention on acquisition of mycotoxin free- diets and application of feed manufacturing and management practices that prevent mold growth and mycotoxin formation. This ideally requires access to sufficient laboratory capabilities to confirm the purchase of ingredients free of mycotoxins, proper storage of ingredients, and feed processing, shipping, and handling procedures to minimize formation of mycotoxins. Detoxification is a new approach to utilizing mycotoxin-contaminated feeds in preventing mycotoxicosis. Hydrated sodium-calcium aluminosilicate binds aflatoxin B1 in the digestive tract, possibly by sequestration, and reduce toxicity to chickens (18). Bentonite clay also ameliorate aflatoxicosis (28). New adsorbents like mycofix (29,30), are also effective in alleviating aflatoxicosis. Other important managemental efforts should be practiced for reduction of mold growth and aflatoxin formation. These include monitoring and controlling moisture content of feedstuffs below 15%; insect control, using mold inhibitors, pelleting feeds, adequate ventilation for controlling
humidity in confinement housing, continues analysis of feed for common mycotoxins, reduction of environmental and diet stresses and an addition of antioxidants like vitamin E and selenium.

Acknowledgement

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References


