ENVIRONMENT AND HEALTH

Effects of Feeding Fusarium moniliforme Culture Material, Containing Known Levels of Fumonisin B1, in the Young Turkey Poults

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ABSTRACT The effects of feeding Fusarium moniliforme culture material, containing known concentrations of fumonisin B1 (FB1), were studied in turkey poulets. Day-old poulets were allotted randomly to dietary treatments containing 0, 0.41, 0.82, 1.23, 2.87, 4.10, 5.33, 6.56, and 7.79% fumonisin culture material (FCM). These levels of FCM supplied 0, 25, 50, 75, 100, 175, 250, 325, 400, and 475 mg FB1/kg of feed. Each dietary treatment was fed to six pen replicates of six poulets each for 21 d. Poulets fed FCM that supplied 325 to 475 mg FB1/kg diet had lower (P < 0.05) feed intakes and BW gains. Increased (P < 0.05) liver and pancreas weights were observed in poulets fed FCM that supplied > 175 mg FB1/kg. Poulets fed FCM that supplied 400 and 475 mg FB1/kg diet had increased (P < 0.05) red blood cell counts and increased (P < 0.05) serum concentrations of gamma glutamyl transferase and aspartate aminotransferase. Compared with controls, poulets fed FCM that supplied 25, and 75 to 475 mg FB1/kg had increased (P < 0.05) liver sphinganine:sphingosine ratios. Hepatocellular hyperplasia was mild at 75 and 100 mg FB1/kg diet, moderate to severe at 250 mg/kg FB1, and severe at 325 to 475 mg FB1/kg. Multifocal to generalized loss of cross striations and thinning of cardiomyocytes was observed in poulets fed FCM that supplied 475 mg FB1/kg diet. Results indicated that diets containing > 1.23% FCM that supplied ≥ 75 mg FB1/kg are toxic to young turkeys.

(Key words: fumonisin B1, turkey, Fusarium moniliforme, sphinganine, sphingosine)

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INTRODUCTION

Recently, a new group of water-soluble toxins produced by Fusarium moniliforme have been characterized. These toxins, which may be responsible for some of the toxicological effects of F. moniliforme, are collectively called fumonisins and include fumonisin A1, A2, B1, B2, B3, and B4 (Gelderblom et al., 1992). Fumonisin B1, (FB1) the major compound, has been shown to be the causative agent in equine leukoencephalomalacia (Marasas et al., 1988) and porcine pulmonary edema (Harrison et al., 1990). Fumonisin B1 is hepatotoxic in rats (Voss et al., 1990) and has been shown to cause morphological and functional changes in chicken macrophages in vitro, which suggests an immunosuppressive effect (Qureshi and Hagler, 1992). Initial studies with turkey poulets indicated that high dietary levels of FB1 (≥ 75 mg FB1/kg diet) supplied by F. moniliforme cultures caused poor performance, increased organ weights, diarrhea, biliary hyperplasia, hepatocellular hyperplasia, and rickets (Weibking et al., 1993a, 1995). However, there have been no reports to date on the effects of lower levels of FB1 in turkeys. Therefore, the objectives of the present study were to investigate further the toxic effects of FB1 present in F. moniliforme cultures, in young turkeys and to determine the minimum dietary concentration necessary to cause deleterious effects.

MATERIALS AND METHODS

Experimental Design and Birds

Three hundred and sixty day-old female Nicholas Large White poulets were allotted randomly to pens in a stainless steel chick battery and allowed to consume feed and water ad libitum. The experimental design consisted of 10 dietary treatments with six pen replicates of six birds allotted randomly to each dietary treatment. The day-old poulets were fed experimental diets from hatching to 21 d of age. Poulets were monitored daily for signs of morbidity and mortality. The animal care and use protocol was reviewed and approved by the University of Missouri-Columbia Animal Care and Use Committee.

Fumonisin Production and Analysis

Procedures for the production of fumonisin have been reported previously (Weibking et al., 1993b). The culture

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material was analyzed for FB1, FB2, and FB3 by HPLC by the procedure of Wilson et al. (1990).

**Diet Preparation**

Dietary treatments were prepared by substituting ground F. moniliforme M-1325 culture material for ground corn in a typical corn-soybean meal basal diet. Fumonisin culture material (FCM) contained 6,125 mg FB1/kg, 1,400 mg FB2/kg, and 535 mg FB3/kg by analysis and made up 0, 0.41, 0.82, 1.23, 1.64, 2.87, 4.10, 5.33, 6.56, and 7.79% of the respective diets and supplied 0, 25, 50, 75, 100, 175, 250, 325, 400, and 475 mg FB1/kg diet, respectively. Diets were formulated to be isocaloric and isonitrogenous and either met or exceeded the nutrient requirements of turkey poultss as recommended by NRC (1984). Diets and culture material were screened by the method of Rottinghaus et al. (1982) and found to be below detection limits for the following mycotoxins: aflatoxin (10 µg/kg), citrinin (100 µg/kg), sterigmatocystin (50 µg/kg), zearalenone (500 µg/kg), ochratoxin A (50 µg/kg), T-2 toxin (1 mg/kg), diacetoxyscirpenol (1 mg/kg), and vomitoxin (500 µg/kg). Culture material was also assayed and found to be below detection limits for fusarin C (500 µg/kg) and monoiliformin (500 µg/kg; assays performed by Glenn Bennett, USDA, Peoria, IL 61604).

**Sample Collection**

At the end of Weeks 1, 2, and 3 of the experiment, poultss were weighed individually and feed consumption was determined for each pen. On Day 21, blood samples were collected via cardiac puncture for serum biochemistry and hematologic determinations. Serum biochemical values were determined using an autoanalyzer.5 Hemoglobin was measured as cyanmethemoglobin.6 Red blood cell counts (RBC), mean cell volume (MCV), and hematocrits (HCT) were determined with a counter7 using instrument settings described by Steel et al. (1977). Mean cell hemoglobin (MCH) and mean cell hemoglobin concentration (MCHC) were calculated. Free sphinganine (SA) and free sphingosine (SO) were determined in livers by HPLC using the procedures described by Merrill et al. (1988). Following blood sampling, 12 poultss per treatment (2 poultss per replicate) were euthanatized with CO2, and selected organs were excised and weighed.

**Histopathology**

Post-mortem examinations were performed on six poultss (one poult per replicate) from each treatment group at 3 wk of age. Samples of proximal tibiotarsal bone, pectoral muscle, adductor muscle, ventriculus, proven-ticulus, lung, spleen, thymus, bursa of Fabricius, heart, duodenum, pancreas, jejunum, ileum, cecum, kidney, and liver were fixed in 10% neutral buffered formalin. Fixed tissues were trimmed, embedded in paraffin, sectioned at 4 µm, and stained with hematoxylin and eosin stain. Liver sections from all experimental groups were examined microscopically and liver histopathology was graded in a blind fashion.

**Statistical Analysis**

Data were analyzed by the General Linear Models procedure of SAS® (SAS Institute, 1985) as a completely randomized design. Mean differences were determined using Fisher's Least Significant Difference Test. Absolute organ weights were adjusted for final body weight by covariance analysis (Shirley, 1977). Statistical significance was accepted at P < 0.05.

**RESULTS**

The effects of dietary FCM on poult performance are presented in Table 1. Compared with controls, feed intakes and BW gains were reduced in poultss fed FCM that supplied 325 to 475 mg FB1/kg diet. Compared with controls, feed conversion was poorer in poultss fed 25 and 475 mg FB1/kg.

Compared with controls, increased liver weights were observed in poultss fed FCM that supplied 25, 50 and 175 to 475 mg FB1/kg, and increased proventriculus weights were observed in poultss fed FCM that supplied 400 and 475 mg FB1/kg (Table 2). Poultss fed FCM that supplied 175 to 475 mg FB1/kg had heavier pancreas weights than control poultss (Table 2). Kidney, bursa, gizzard, and spleen weights averaged 6.57, 1.04, 15.43, and 0.62 g, respectively, and were not influenced by dietary FB1 (data not shown).

Compared with controls, increased RBC were observed in poultss fed FCM that supplied 25, 50 and 175 to 475 mg FB1/kg, and increased proventriculus weights were observed in poultss fed FCM that supplied 400 and 475 mg FB1/kg (Table 2). Poultss fed FCM that supplied 175 to 475 mg FB1/kg had heavier pancreas weights than control poultss (Table 2). Kidney, bursa, gizzard, and spleen weights averaged 6.57, 1.04, 15.43, and 0.62 g, respectively, and were not influenced by dietary FB1 (data not shown).

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The effects of dietary FCM on serum chemistry are presented in Table 3. Poultss fed FCM that supplied 400 and 475 mg FB1/kg had higher gamma glutamyl transferase and aspartate aminotransferase (AST) levels than control poultss. Serum glucose, cholesterol, total protein, and albumin were not influenced by FCM and averaged 291 mg/dL, 97 mg/dL, 2.92 g/dL, and 1.39 g/dL, respectively (data not shown). Compared with controls, poultss fed FCM that supplied 25, 75 to 475 mg FB1/kg diet had increased liver SA:SO ratios (Figure 1).

With the exception of myocardial lesions in poultss fed 475 mg FB1/kg, treatment-related lesions were only observed in the liver. Hepatocellular hyperplasia (Figure
TABLE 1. Effects of Fusarium moniliforme culture material, containing fumonisins, on performance of turkey poults (1 to 21 d)

<table>
<thead>
<tr>
<th>Culture material (%)</th>
<th>Feed intake</th>
<th>Body weight gain (g)</th>
<th>Feed conversion (feed:gain)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 (0)</td>
<td>785b</td>
<td>532a</td>
<td>1.48bcd</td>
</tr>
<tr>
<td>0.41 (25)</td>
<td>842a</td>
<td>536a</td>
<td>1.57a</td>
</tr>
<tr>
<td>0.82 (50)</td>
<td>752b</td>
<td>516a</td>
<td>1.46d</td>
</tr>
<tr>
<td>1.23 (75)</td>
<td>790abcd</td>
<td>527a</td>
<td>1.50bcd</td>
</tr>
<tr>
<td>1.64 (100)</td>
<td>762bc</td>
<td>531a</td>
<td>1.44d</td>
</tr>
<tr>
<td>2.87 (175)</td>
<td>760bc</td>
<td>511ab</td>
<td>1.49bcd</td>
</tr>
<tr>
<td>4.10 (250)</td>
<td>721c</td>
<td>465b</td>
<td>1.54b</td>
</tr>
<tr>
<td>5.33 (325)</td>
<td>634d</td>
<td>416c</td>
<td>1.52bc</td>
</tr>
<tr>
<td>6.56 (400)</td>
<td>522e</td>
<td>332a</td>
<td>1.57a</td>
</tr>
<tr>
<td>7.79 (475)</td>
<td>19</td>
<td>15</td>
<td>0.02</td>
</tr>
<tr>
<td>SE</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Values within columns with no common superscript differ significantly (P < 0.05).

1Data are means of six replicate pens of six poults each.
2Dietary levels (milligrams per kilogram) of fumonisin B1 (FB1).
3Total fumonisin levels (FB1 + FB2 + FB3) were 0, 33, 66, 99, 132, 231, 330, 429, 528, and 627 mg/kg diet, respectively.

2) was mild at 75 and 100 mg FB1/kg, moderate to severe at 250 mg FB1/kg, and severe at 325 to 475 mg FB1/kg. Mild biliary hyperplasia was also observed in poults fed 475 mg FB1/kg. No lesions were seen in skeletal muscle, spleen, thymus, kidney, proventriculus, lung, bursa of Fabricius, duodenum, pancreas, jejunum, ileum, cecum, or proximal tibiatarsus of the control or treatment groups.

DISCUSSION

In contrast to previous studies (Weibking et al., 1993a, 1995), depression in poult performance did not occur until dietary levels of FB1 reached 325 mg FB1/kg feed. In two previous studies, depressions in intake and gain were observed in poults fed dietary levels as low as 75 mg FB1/kg for 3 wk (Weibking et al., 1993a, 1995). However, in another 3-wk study no depression in performance was observed in poults fed 75 mg FB1/kg feed (Weibking et al., 1994). In the studies in which performance was affected at 75 mg FB1/kg feed (Weibking et al., 1993a, 1995), a severe diarrhea was present after 8 d in poults fed FB1. Diarrhea probably accounted for the differences between those studies and the present study, in which no diarrhea was observed. In earlier studies, FB1 concentrations in FCM averaged 2,000 mg/kg or less, and in order to achieve the required dietary concentrations it was necessary to add significant quantities (> 10%) of FCM to the diets. Diarrhea appears to occur only when the level of FCM exceeds 10% of the diet. In the present study, the highest level of FCM incorporated in diets was 7.79%. Two possible mechanisms by which low concentration FB1 producing FCM cause diarrhea are proposed. An additional toxic metabolite may be present in low producing FB1 Fusarium moniliforme cultures. This hypothesis is supported by a recent report (Norred et al., 1991) indicating the presence of other cytotoxic water-soluble metabolites in F. moniliforme cultures. A second possibility is that an unknown toxic metabolite is present in all F. moniliforme cultures at a low concentration and a diarrhea-producing dose of this metabolite is not administered until greater than 10% culture material is included in the ration.

Increased liver weights were observed in poults fed 25 mg FB1/kg, which is the lowest level of FB1 that has
been shown to cause increased liver weights to date. In previous studies, increased liver weights have also been observed in poults fed 75 to 300 mg FB₁/kg (Weibking et al., 1993a, 1994, 1995), broilers fed 100 to 400 mg FB₁/kg (Brown et al., 1992; Ledoux et al., 1993a, 1994, 1995), and mice fed 175 to 475 mg FB₁/kg in this study has been observed previously in turkeys fed 100 and 200 mg FB₁/kg (Weibking et al., 1993a). In the very sensitive to dietary FB₁. The increase in AST activity observed in this study has not been observed previously in turkeys but has been observed in broilers fed 100 to 400 mg FB₁/kg (Ledoux et al., 1992).

The changes in hematology observed in this study were not observed in a previous study with poults fed 100 or 200 mg FB₁/kg (Weibking et al., 1993a). In the previous study, MCV and MCH were decreased in poults fed 200 mg FB₁/kg. These inconsistent results and those observed in studies with broilers (Weibking et al., 1993b) suggest that hematologic variables are not very sensitive to dietary FB₁.

The increase in AST activity observed in this study has also been observed previously in turkeys (Weibking et al., 1993a), broilers (Ledoux et al., 1992), rats (Voss et

### Table 2. Effects of *Fusarium moniliforme* culture material, containing fumonisins, on absolute organ weights of turkey poults

<table>
<thead>
<tr>
<th>Culture material (%)</th>
<th>Liver (g)</th>
<th>Proventriculus (g)</th>
<th>Pancreas (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 (0)³⁴</td>
<td>14.5⁹</td>
<td>3.16⁶</td>
<td>2.07⁴</td>
</tr>
<tr>
<td>0.41 (25)</td>
<td>16.6⁵bcd</td>
<td>3.15⁶</td>
<td>2.16⁵</td>
</tr>
<tr>
<td>0.52 (50)</td>
<td>16.6⁵bcd</td>
<td>3.16⁶</td>
<td>2.22⁶</td>
</tr>
<tr>
<td>1.23 (75)</td>
<td>15.3⁴de</td>
<td>3.33⁶</td>
<td>2.30⁶</td>
</tr>
<tr>
<td>1.64 (100)</td>
<td>15.1₁de</td>
<td>3.32⁵</td>
<td>2.24⁶</td>
</tr>
<tr>
<td>2.87 (175)</td>
<td>16.9⁴bcd</td>
<td>3.11⁴</td>
<td>2.35³</td>
</tr>
<tr>
<td>4.10 (250)</td>
<td>17.4⁴bc</td>
<td>3.34⁵</td>
<td>2.40⁶ bc</td>
</tr>
<tr>
<td>5.33 (325)</td>
<td>17.8⁴b</td>
<td>3.31⁶</td>
<td>2.40⁶</td>
</tr>
<tr>
<td>6.56 (400)</td>
<td>18.6⁴ab</td>
<td>3.45⁵</td>
<td>2.65³</td>
</tr>
<tr>
<td>7.79 (475)</td>
<td>20.9⁴a</td>
<td>3.63⁵</td>
<td>2.63⁶ab</td>
</tr>
<tr>
<td>SE</td>
<td>0.75</td>
<td>0.07</td>
<td>0.08</td>
</tr>
</tbody>
</table>

³⁴Values within columns with no common superscript differ significantly (P < 0.05).

³Absolute organ weights adjusted for final body weight by analysis of covariance.

⁴Dietary levels (milligrams per kilogram) of fumonisin B₁ (FB₁).

### Table 3. Effects of *Fusarium moniliforme* culture material, containing fumonisins, on hematology and serum chemistry of turkey poults

<table>
<thead>
<tr>
<th>Culture material (%)</th>
<th>RBC (×10⁹/mm³)</th>
<th>HCT (%)</th>
<th>HB (g/dL)</th>
<th>GGT (IU/L)</th>
<th>AST (IU/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 (0)³⁴</td>
<td>2.88⁶</td>
<td>37.2⁹</td>
<td>10.6⁹</td>
<td>11.6⁹</td>
<td>298ab</td>
</tr>
<tr>
<td>0.41 (25)</td>
<td>2.88⁶</td>
<td>36.7⁹</td>
<td>9.7⁹</td>
<td>11.8⁹</td>
<td>306bc</td>
</tr>
<tr>
<td>0.52 (50)</td>
<td>2.83⁵</td>
<td>36.8⁹</td>
<td>10.1⁹</td>
<td>12.5⁹</td>
<td>303bc</td>
</tr>
<tr>
<td>1.23 (75)</td>
<td>2.92⁴</td>
<td>37.9⁹</td>
<td>9.8⁹</td>
<td>12.1⁹</td>
<td>303bc</td>
</tr>
<tr>
<td>1.64 (100)</td>
<td>3.01⁴bc</td>
<td>39.0⁹</td>
<td>10.6⁹</td>
<td>11.4⁹</td>
<td>304bc</td>
</tr>
<tr>
<td>2.87 (175)</td>
<td>2.73⁴d</td>
<td>35.6⁹</td>
<td>9.6⁹</td>
<td>12.8⁴bc</td>
<td>319bc</td>
</tr>
<tr>
<td>4.10 (250)</td>
<td>2.85⁴d</td>
<td>37.3⁹</td>
<td>10.1⁹</td>
<td>12.2⁴bc</td>
<td>319bc</td>
</tr>
<tr>
<td>5.33 (325)</td>
<td>3.00⁴bc</td>
<td>39.4⁹</td>
<td>11.0⁹</td>
<td>12.8⁴bc</td>
<td>334bc</td>
</tr>
<tr>
<td>6.56 (400)</td>
<td>3.22⁴b</td>
<td>40.0⁹</td>
<td>11.8⁴bc</td>
<td>13.6⁴bc</td>
<td>374⁴bc</td>
</tr>
<tr>
<td>7.79 (475)</td>
<td>3.49⁴a</td>
<td>46.2⁴a</td>
<td>12.3⁴a</td>
<td>14.0⁴</td>
<td>390⁴</td>
</tr>
<tr>
<td>SE</td>
<td>0.08</td>
<td>1.2</td>
<td>0.4</td>
<td>0.4</td>
<td>21</td>
</tr>
</tbody>
</table>

³⁴Values within columns with no common superscript differ significantly (P < 0.05).

¹Data are means of six replicates of two poults each.

²RBC = red blood cells; HCT = hematocrit; HB = hemoglobin; GGT = γ γamma glutamyl transferase; AST = aspartate aminotransferase.

³Dietary levels (milligrams per kilogram) of fumonisin B₁ (FB₁).

⁴Total fumonisin levels (FB₁ + FB₂ + FB₃) were 0, 33, 66, 99, 132, 231, 330, 429, 528, and 627 mg/kg diet, respectively.
FIGURE 2. Photomicrographs (hematoxylin and eosin) of liver sections of a control poult and a poult fed 400 mg fumonisin B₁ (FB₁)/kg feed, supplied by Fusarium moniliforme culture material. Normal hepatocytes are evident in the control poult (A) and a severe generalized hepatocellular hyperplasia is evident in the poult fed FB₁ culture material (B). Bar equals 20 μm.

al., 1990), mice (Voss et al., 1992), pigs (Osweiler et al., 1992), and ponies (Wang et al., 1992) fed FB₁. However, in the previous study with turkeys, AST activity was elevated in turkeys fed levels as low as 100 mg FB₁/kg. Increases in serum AST values are indicative of altered hepatocyte membrane integrity with the leakage of this enzyme or hepatic necrosis (Duncan and Prasse, 1986).

Diets containing FCM that supplied 75 mg FB₁/kg or higher caused hepatic lesions in young turkeys. Biliary hyperplasia and hepatocellular hyperplasia have also been observed previously in FB₁-exposed turkeys (Weibking et al., 1993a, 1994, 1995), broilers (Brown et al., 1992; Weibking et al., 1993b), and rats (Voss et al., 1990). Hepatocellular hyperplasia is a likely mechanism for the increased liver weights observed in this study. The myocardial lesions observed in pouls fed 475 mg FB₁/kg in this study and in pouls fed 300 mg FB₁/kg in a previous study (Weibking et al., 1995) have not been reported previously in any other species.

Wang et al. (1991) found that FB₁ inhibition of de novo sphingolipid biosynthesis resulted in increased SA:SO ratios and proposed that this inhibition may be the initial molecular target of these toxins. The SA:SO ratios increase as a consequence of the accumulation of SA in tissues and serum resulting from FB₁ inhibition of N-acyltransferase, the enzyme required to convert SA to SO. Fumonisin B₁ inhibition of sphingolipid biosynthesis resulted in an increase in the SA:SO ratio in liver hepatocytes (Wang et al., 1991), cultured renal cells (Yoo et al., 1992), blood serum of ponies (Wang et al., 1992), blood serum, liver, lung, and kidneys of pigs (Riley et al., 1993), and blood serum, kidney, liver, and muscle of catfish (Goel et al., 1994). Similar increases in serum SA:SO ratios were observed in previous studies in turkeys fed the lowest level of FCM that supplied 75 mg FB₁/kg (Weibking et al., 1993a, 1994, 1995). In the present study, there was a three-, four-, and eightfold increase in SA:SO ratios, relative to controls, in livers of pouls fed 25, 75, and 475 mg FB₁/kg, respectively. Although not statistically significant, liver SA:SO ratios also increased twofold in pouls fed FCM that supplied 50 mg FB₁/kg diet. Wang et al. (1992) hypothesized that the depletion of complex sphingolipids (resulting from FB₁ inhibition of biosynthesis) and accumulation of SA may well be the cause of pathology observed in FB₁ toxicosis, because these sphingolipids are involved in regulation of cell surface receptors, ion pumps, and other systems vital for cell function and survival. However, the mechanism by which these alterations in sphingolipids cause tissue damage is still not known (Riley et al., 1993).

In pigs fed 5 to 175 mg FB₁/kg, significantly elevated SA:SO ratios were observed in liver, lung, and kidney at FB₁ levels as low as 23 mg/kg, but tissue damage occurred only in liver at 23 mg FB₁/kg. Damage to lung only occurred at 175 mg FB₁/kg and no damage was observed in kidney at any level of FB₁ (Riley et al., 1993). Riley et al. (1993) proposed the following hypotheses to explain these differences: It is possible that different tissues have different tolerances to elevated levels of sphingoid bases; elevations in free sphingoid bases may
be a benign early response; such elevations may not accurately assess the extent of complex sphingolipid depletion; sphingolipids may have a more critical role in maintaining cellular integrity or regulating cell function; tissue effects may be indirectly linked to sphingolipid alterations. Results of the present study indicated that although liver SA:SO ratios were increased in pouls fed 25 mg FB1/kg microscopic tissue changes only occurred in liver at 75 mg FB1/kg and in cardiac muscle at 475 mg FB1/kg diet.

In summary, results indicate decreased performance at 325 mg FB1/kg, increased liver weights at 25 mg FB1/kg, hepatic lesions at 75 mg FB1/kg, cardiac lesions at 475 mg FB1/kg, and a threefold increase in liver SA:SO ratios at 25 mg FB1/kg. These data and those of previous experiments suggest that levels of FB1 of 50 mg/kg or lower supplied by FCM does not cause significant deleterious effects in young turkey pouls. These results further suggest that fumonisin toxicity may not be of practical concern to the turkey industry since 50 mg FB1/kg is 10-fold higher than naturally occurring levels of FB1 (0.1 to 5 mg/kg) reported for U.S. corn crops (Rottinghaus et al., 1992; Holcomb et al., 1993; Murphy et al., 1993). This level of FB1 (50 mg/kg) is also twice as high as the average FB1 level reported in corn samples from feeds associated with PPE and ELEM (Ross et al., 1991). The possibility that there may be other toxic metabolites in these F. moniliforme cultures that may be responsible for causing diarrhea implies that F. moniliforme-contaminated commodities may be of concern to the turkey industry. Finally, although FCM used in this study more closely approximates natural contamination compared to the pure mycotoxin, it still does not represent natural contamination and the individual toxicity of some mycotoxins have been shown to be enhanced under natural conditions when they occur as co-contaminants (Huff et al., 1986). Culture material used in this study contained only fumonisins; however, F. moniliforme is known to produce several other toxins, including fusaric acid, fusarins, moniliformin, and giberellins (Nelson, 1992). Combinations of these and other Fusarium toxins may result in synergistic toxicity and may be responsible for previously reported disease syndromes in poultry.

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