Effects of feeding a blend of grains naturally contaminated with *Fusarium* mycotoxins on feed intake, serum chemistry, and hematology of horses, and the efficacy of a polymeric glucomannan mycotoxin adsorbent

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ABSTRACT: The feeding of *Fusarium* mycotoxin-contaminated grains adversely affects the performance of swine and poultry. Very little information is available, however, on adverse effects associated with feeding these mycotoxin-contaminated grains on the performance of horses. An experiment was conducted to investigate the effects of feeding a blend of grains naturally contaminated with *Fusarium* mycotoxins on feed intake, serum immunoglobulin (Ig) concentrations, serum chemistry, and hematology of horses. A polymeric glucomannan mycotoxin adsorbent (GM polymer) was also tested for efficacy in preventing *Fusarium* mycotoxicoses. Nine mature, nonexercising, light, mixed-breed mares were assigned randomly to one of three dietary treatments for 21 d. The horses were randomly reassigned and the experiment was subsequently replicated in time following a 14-d washout interval. Feed consumed each day was a combination of up to 2.8 kg of concentrates and 5 kg of mixed timothy/alfalfa hay. The concentrates fed included the following: 1) control, 2) blend of contaminated grains (36% contaminated wheat and 53% contaminated corn), and 3) blend of contaminated grains + 0.2% GM polymer. Diets containing contaminated grains averaged 15.0 ppm of deoxynivalenol, 0.8 ppm of 15-acetyldeoxynivalenol, 9.7 ppm of fusaric acid, and 2.0 ppm of zearalenone. Feed intake by all horses fed contaminated grains was reduced ($P < 0.001$) compared with controls throughout the experiment. Supplementation of 0.2% GM polymer to the contaminated diet increased ($P = 0.004$) feed intake of horses compared with those fed the unsupplemented contaminated diet. Serum activities of $\gamma$-glutamyltransferase were higher ($P = 0.047$ and 0.027) in horses fed the diet containing contaminated grain compared with those fed the control diet on d 7 and 14, but not on d 21 ($P = 0.273$). Supplementation of GM polymer to the contaminated diet decreased ($P < 0.05$) serum $\gamma$-glutamyltransferase activities of horses compared with those fed unsupplemented contaminated diet on d 7 and 14. Other hematology and serum chemistry measurements including serum IgM, IgG, and IgA, were not affected by diet. It was concluded that the feeding of grains naturally contaminated with *Fusarium* mycotoxins caused a decrease in feed intake and altered serum gamma glutamyltransferase activities. The supplementation of GM polymer prevented these mycotoxin-induced adverse effects.

Key Words: Fusaric Acid, *Fusarium*, Horses, Mycotoxins, Vomitoxin

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Introduction

*Fusarium* fungi are commonly found in temperate climates and *Fusarium* mycotoxins are likely the most economically significant grain mycotoxins on a global basis (Wood, 1992). *Fusarium* mycotoxins, however, have also been found to contaminate pastures and forages. Among the trichothecene mycotoxins, deoxynivalenol (DON, vomitoxin) is a major contributor to reduced feed intake in animals. Trichothecene mycotoxins have also been shown to be potent immunosuppressive agents (Bondy and Pestka, 2000).

Pigs are the most sensitive species to feeding DON, and it has been shown that feeding DON-contaminated grains reduces feed intake and weight gain of pigs (Trenholm et al., 1994). A synergistic interaction between DON and fusaric acid (FA) on growth rate of pigs fed blends of naturally contaminated grains has been demonstrated (Smith et al., 1997). Very little information is available, however, on the toxicity associated
with the feeding of *Fusarium* mycotoxin-contaminated grains on feed intake and metabolism of horses. Johnson et al. (1997) found no effect on feed intake, serum chemistry, or hematology when horses were fed barley naturally contaminated with 36 to 44 ppm of DON for 40 d.

Polymeric mycotoxin adsorbents have been reported to prevent the deleterious effects of mycotoxins by reducing intestinal absorption of mycotoxins and preventing subsequent transport to target tissues (Ramos et al., 1996). A polymeric glucomannan mycotoxin adsorbent (GM polymer) has shown beneficial effects in preventing *Fusarium* mycotoxicoses in poultry and swine (Raju and Devegowda, 2000; Swamy et al., 2002a,b).

The objectives of the current experiment were to investigate the effects of feeding a blend of grains naturally contaminated with *Fusarium* mycotoxins on feed intake, serum immunoglobulin (Ig) concentrations, serum chemistry, and hematology of horses. A polymeric mycotoxin adsorbent (GM polymer) was also tested for efficacy in preventing *Fusarium* mycotoxicoses.

### Materials and Methods

#### Experimental Animals and Diets

Mature, nonexercising, light, mixed-breed mares (n = 9) were assigned randomly to one of three dietary treatments for 21 d. The horses were randomly reassigned and the experiment was subsequently replicated in time following a 14-d washout interval. Feed consumed each day was a combination of up to 2.8 kg of concentrates and 5 kg of mixed timothy/alfalfa hay (Table 1) formulated to meet the nutritional requirements of a mature, nonworking horse (NRC, 1989). The concentrates fed included the following: 1) control, 2) blend of contaminated grains (36% contaminated wheat and 53% contaminated corn), and 3) blend of contaminated grains + 0.2% GM polymer (MTB-100, Alltech Inc., Nicholasville, KY). Water was provided ad libitum.

The horses were housed at the Equine Research Centre (Guelph, ON, Canada) in individual 3.6 m² boxstalls with 8 h of group turnout on paddocks with minimal pasture a day. Horses were examined daily for any adverse clinical signs.

#### Animal Care

This experiment was reviewed and approved by the University of Guelph Animal Care Committee. Animals were managed and cared for according to the guidelines of the Canadian Council on Animal Care.

#### Analysis of Dietary Mycotoxins

Dietary and bedding materials were analyzed for DON, 3-acetyldeoxynivalenol, 15-acetyldeoxynivalenol, nivalenol, T-2 toxin, iso T-2 toxin, acetyl-T-2 toxin, HT-2 toxin, T-2 triol, T-2 tetraol, fusarenon-X, diacetoxyscirpenol, scirpentriol, 15-acetoxyscirpenol, neosolaniol, zearalenone, zearalenol, and fumonisin using a combination of gas chromatography and mass spectrometry (North Dakota State University, Fargo, ND). The detection limit for these mycotoxins was 0.2 μg/g. Briefly, the mycotoxins were extracted from 25 g of ground sample with 100 mL of acetonitrile:water (84:16) for 1 h on a horizontal shaker. A 6-mL aliquot of the supernatant was gravity filtered through 1.5 g of C₁₈:alumina (1:1) and a 2-mL aliquot of the eluent was evaporated at 65°C for 30 min. The residue was derivatized (N-trimethylsilylimidazole +

### Table 1. Composition of dietary concentrates and hay (as-fed basis)

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Controla</th>
<th>Mycotoxinb</th>
<th>0.2% GMC</th>
<th>Hay</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nutrient composition</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CP, %</td>
<td>13.21</td>
<td>14.44</td>
<td>14.30</td>
<td>15.82</td>
</tr>
<tr>
<td>DE, Mcal/kgf</td>
<td>2.30</td>
<td>2.30</td>
<td>2.30</td>
<td>1.73</td>
</tr>
<tr>
<td>Ca, %f</td>
<td>0.29</td>
<td>0.29</td>
<td>0.29</td>
<td>0.38</td>
</tr>
<tr>
<td>P, %f</td>
<td>0.24</td>
<td>0.24</td>
<td>0.24</td>
<td>0.18</td>
</tr>
</tbody>
</table>

aDiet with control corn and wheat.

b,cMycotoxin-contaminated diets supplemented with 0 and 0.2% glucomannan polymer, respectively.

dProvided the following per kilogram of diet: calcium, 3.3%; phosphorus, 2.0%; sodium, 4.0%; magnesium, 0.5%; potassium, 1.3%; iodine, 10 mg; copper, 520 mg; sulphur, 0.2%; iron, 1,866 mg; zinc, 1,570 mg; cobalt, 6 mg; fluorine, 100 mg; vitamin A (retinyl palmitate), 114,400 IU; vitamin K₃, 47 mg; thiamin, 87 mg; riboflavin, 84 mg; pantothenic acid, 258 mg; niacin, 410 mg; pyridoxine, 67 mg; folic acid, 19 mg; biotin, 3,545 μg; vitamin B₁₂, 348 μg.

eAnalyzed (AOAC, 1980).

fCalculated.
Table 2. Mycotoxin content of experimental diets, hay and bedding material

<table>
<thead>
<tr>
<th>Dietary group</th>
<th>DON</th>
<th>FA</th>
<th>ZEA</th>
<th>15-ADON</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>0.7</td>
<td>5.4</td>
<td>&lt;0.1</td>
<td>&lt;0.2</td>
</tr>
<tr>
<td>Mycotoxin</td>
<td>14.1</td>
<td>6.4</td>
<td>2.0</td>
<td>0.7</td>
</tr>
<tr>
<td>0.2% GM</td>
<td>15.9</td>
<td>12.9</td>
<td>2.0</td>
<td>0.8</td>
</tr>
<tr>
<td>Hay</td>
<td>&lt;0.1</td>
<td>12.3</td>
<td>&lt;0.1</td>
<td>&lt;0.2</td>
</tr>
<tr>
<td>Straw bedding</td>
<td>1.2</td>
<td>3.9</td>
<td>&lt;0.1</td>
<td>&lt;0.2</td>
</tr>
</tbody>
</table>

*aOther mycotoxins (i.e., T-2 toxin, iso T-2 toxin, acetyl T-2 toxin, HT-2 toxin, T-3 triol, T-2 tetraol, fusarenon-X, 3-acetyl deoxynivalenol, nivalenol, diacetoxyscirpenol, scirpentriol, 15-acetyl scirpenol, neosolaniol, zearalenol, fumonisin B1, and aflatoxins [G1, G2, B1 and B2]) were below detection limits (2 ppm for fumonisin B1, 0.01 ppm for aflatoxins, and 0.2 ppm for the other mycotoxins).

*bDeoxynivalenol.

*cFusaric acid.

*dZearalenone.

*2T-2 toxin (Deoxynivalenol).

*315-acetyldeoxynivalenol.

*4Diet with control corn and wheat.

*5Mycotoxin-contaminated diets supplemented with 0 and 0.2% glucomannan polymer, respectively.

trimethylchlorosilane + n,o-bis(trimethylsilyl)trifluoroacetamide + pyridine) to form trimethylsilyl (TMS) ester derivatives of tricothenes and estrogens. The TMS-mycotoxin derivatives were separated on a gas chromatograph (Shimadzu, QP 5050), using a Restek (Restek Corp., Bellefonte, PA) 30-m RTX 35 × 0.25 mm × 0.25 μm phase capillary column, and then assayed by select ion monitoring and electron ionization in a mass spectrometer, using three or four ion fragments for identification and quantification of each mycotoxin. The capillary column was held at 90°C for 1 min after on-column injection, heated to 210°C at 40°C/min, and then heated to 310°C at 5°C/min and held for 1 min (25 min total) before recycling. Helium linear velocity at 110°C was 40 cm/s.

Dietary and bedding material aflatoxin contents (G1, G2, B1, and B2) were analyzed by HPLC (Animal Health Laboratory, Laboratory Services Division, University of Guelph, Guelph, ON, Canada). The detection limit for these mycotoxins was 0.002 μg/g (Tarter et al., 1984). Fusaric acid content was determined by the HPLC method of Matsui and Watanabe (1988) as modified by Smith and Sousadias (1993) and confirmed by Porter et al. (1995; 0.77 μg/g detection limit).

Experimental Parameters Studied

Feed Intake and Weight Gain. At each feeding (0800 and 1600), the quantity of concentrate consumed by each horse was recorded. The horses were weighed weekly during the trial.

Hematology and Serum Biochemical Analysis. Blood samples were drawn by jugular venipuncture (7, 14, and 21 d). Blood samples were subjected to hematology and serum chemistry determinations (Laboratory Services Division, University of Guelph, Guelph, ON). Red blood cell count, mean corpuscular volume, and hemato-crit were determined and mean corpuscular hemoglobin concentrations were calculated. Hemoglobin was measured as cyanmethemoglobin after lysing the red blood cells using an Advia 120 Hematology System (Bayer Inc., Healthcare Division, Toronto, ON, Canada). Complete blood cell counts (differential leukocyte count) were performed manually to test for changes in absolute numbers of leukocytes, lymphocytes, segmented neutrophils, band neutrophils, monocytes, eosinophils, and basophils.

Serum concentrations of total protein, albumin, globulin, glucose, β-hydroxybutyrate, haptoglobin, urea, cholesterol, creatinine, bilirubin, calcium, phosphorus, magnesium, sodium, potassium, chloride, and activities of alkaline phosphatase, glutamate dehydrogenase, aspartate aminotransferase, γ-glutamyltransferase (GGT), and creatine kinase were determined using a Hitachi 911 autoanalyzer (Roche Diagnostics, Hoffman-La Roche Ltd., Montreal, QC, Canada).

Serum Immunoglobulin Concentrations. Concentrations of IgA, IgM, and IgG were determined in serum samples obtained on day 21 using the radial immunodiffusion technique of Mancini et al., 1965 (Animal Health Lab Services). Briefly, serum was placed into plates containing buffered agarose with monospecific antisera. The plates were incubated at room temperature for 18 to 24 h. The diameter of the subsequent diffusion ring was used to determine the concentration by comparing with a standard curve.

Statistical Analysis

Data were subjected to Levene’s homogeneity of variances test before the analysis for treatment differences. Data were analyzed by ANOVA using the GLM procedure of SAS as a completely randomized design within the period (SAS Inst., Inc., Cary, NC). Effects of period and period × diet interaction were tested in the model, and the same were removed from the model when found nonsignificant. The effect of feeding Fusarium mycotoxin-contaminated diets was determined by employing a simple contrast between the horses fed the control diet and those fed the mycotoxin-contaminated diet. The ability of the GM polymer to prevent Fusarium mycotoxin-induced effects was tested by simple contrasts between the horses fed the mycotoxin-contaminated diet with and without 0.2% GM polymer and between horses fed the control diet and the GM polymer-supplemented diet (Kuehl, 2000). Statements of statistical significance were based on P < 0.05.

Results

Dietary Mycotoxin Concentrations

The analyzed concentrations of mycotoxin in the diets are given in Table 2. Deoxynivalenol and FA were found in the control diet, whereas only DON was detected in the straw bedding. Zearalenone and 15-acetyl DON
Table 3. Concentrate intake of horses fed blends of grains naturally contaminated with \textit{Fusarium} mycotoxins$^a$

<table>
<thead>
<tr>
<th>Dietary group</th>
<th>0 to 7 d</th>
<th>0 to 14 d</th>
<th>0 to 21 d</th>
<th>7 to 14 d</th>
<th>14 to 21 d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control$^b$</td>
<td>2.80</td>
<td>2.80</td>
<td>2.80</td>
<td>2.80</td>
<td>2.80</td>
</tr>
<tr>
<td>Mycotoxin$^c$</td>
<td>1.03</td>
<td>1.02</td>
<td>1.00</td>
<td>1.01</td>
<td>0.96</td>
</tr>
<tr>
<td>0.2% GM$^d$</td>
<td>1.81</td>
<td>1.71</td>
<td>1.64</td>
<td>1.60</td>
<td>1.52</td>
</tr>
<tr>
<td>SEM</td>
<td>0.09</td>
<td>0.08</td>
<td>0.07</td>
<td>0.08</td>
<td>0.06</td>
</tr>
<tr>
<td>Control vs. mycotoxin$^{ef}$</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mycotoxin vs. 0.2% GM$^g$</td>
<td>0.004</td>
<td>0.011</td>
<td>0.009</td>
<td>0.032</td>
<td>0.011</td>
</tr>
<tr>
<td>Control vs. 0.2% GM$^h$</td>
<td>0.001</td>
<td>0.001</td>
<td>&lt;0.001</td>
<td>0.001</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

$^a$All dietary treatments included 5 kg/d of mixed timothy/alfalfa hay.
$^b$Values are least squares means; n = 6 horses.
$^c$Mycotoxin-contaminated concentrate containing 36% contaminated wheat and 53% contaminated corn.
$^d$Mycotoxin-contaminated concentrate supplemented with 0.2% glucomannan polymer.
$^e,f,g$Simple contrasts comparing control diet with mycotoxin diet, mycotoxin diet with 0.2% GM, and control diet with 0.2% GM, respectively.

were detected in the mycotoxin-contaminated diets with and without GM polymer in addition to DON and FA.

Feed Intake and Changes in Body Weight

Feeding of the contaminated diet to horses resulted in reduced feed intake compared with those fed the control diet throughout the experiment (Table 3; $P < 0.001$). Supplementation of 0.2% GM polymer to the contaminated diet improved ($P = 0.004$) feed intake of horses compared with those fed the unsupplemented contaminated diet but not when compared with the control diet (Table 3). Consumption of forage remained unaffected regardless of diet fed (data not shown). Body weights of horses were unaffected by diet (Table 4).

Hematology and Serum Chemistry

Serum activities of GGT were higher ($P = 0.047$ and 0.027) in horses fed the contaminated diet compared with those fed the control diet on d 7 and 14, but not on d 21 ($P = 0.273$; Table 5). Supplementation of the contaminated diet with 0.2% GM polymer significantly decreased ($P = 0.008$ and 0.005) the increase in serum activities of GGT on d 7 and 14, but the decrease was not significantly different from the control diet. Other hematology and serum chemistry parameters were not significantly affected by the dietary treatments (Tables 6, 7, and 8).

Serum Immunoglobulin Concentrations

Serum IgG, IgA, and IgM concentrations were not affected by diets (Table 6).

Discussion

Dietary Mycotoxin Concentrations

The variation in the level of DON (14.1 and 15.9 ppm) in the two contaminated diets may have been due to

Table 5. Effect of feeding blends of grains naturally contaminated with \textit{Fusarium} mycotoxins on serum γ-glutamyltransferase activity levels (U/L) in horses

<table>
<thead>
<tr>
<th>Diet</th>
<th>GGT$^a$</th>
<th>7 d</th>
<th>14 d</th>
<th>21 d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control$^b$</td>
<td>11.44$^b$</td>
<td>12.23</td>
<td>11.49</td>
<td></td>
</tr>
<tr>
<td>Mycotoxin$^c$</td>
<td>22.98</td>
<td>23.86</td>
<td>15.27</td>
<td></td>
</tr>
<tr>
<td>0.2% GM$^d$</td>
<td>8.57</td>
<td>9.58</td>
<td>8.24</td>
<td></td>
</tr>
<tr>
<td>SEM</td>
<td>1.88</td>
<td>1.69</td>
<td>1.35</td>
<td></td>
</tr>
<tr>
<td>Control vs. Mycotoxin$^e$</td>
<td>0.047</td>
<td>0.027</td>
<td>NS$^i$</td>
<td></td>
</tr>
<tr>
<td>Mycotoxin vs. 0.2% GM$^f$</td>
<td>0.008</td>
<td>0.004</td>
<td>NS$^i$</td>
<td></td>
</tr>
<tr>
<td>Control vs. 0.2% GM$^g$</td>
<td>NS$^i$</td>
<td>NS$^i$</td>
<td>NS$^i$</td>
<td></td>
</tr>
</tbody>
</table>

$^a$Serum glutamyltransferase activity (U/L).
$^b$Diet with control corn and wheat.
$^c$Mycotoxin-contaminated concentrate containing 36% contaminated wheat and 53% contaminated corn.
$^d$Mycotoxin-contaminated concentrate supplemented with 0.2% glucomannan polymer.
$^e,f,g$Simple contrasts comparing control diet with mycotoxin diet, mycotoxin diet with 0.2% GM, and control diet with 0.2% GM, respectively.
$^h$Values are least squares means; n = 6 horses.
$^i$Not significant.
Effect of feeding blends of grains naturally contaminated with *Fusarium* mycotoxins on selected serum protein fractions in horses

<table>
<thead>
<tr>
<th>Diet</th>
<th>TPa</th>
<th>Albb</th>
<th>Globc</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>7 d</td>
<td>14 d</td>
<td>21 d</td>
</tr>
<tr>
<td></td>
<td>7 d</td>
<td>14 d</td>
<td>21 d</td>
</tr>
<tr>
<td></td>
<td>7 d</td>
<td>14 d</td>
<td>21 d</td>
</tr>
<tr>
<td>Controld</td>
<td>68.05h</td>
<td>70.57</td>
<td>68.38</td>
</tr>
<tr>
<td>MYCotoxin*</td>
<td>70.63</td>
<td>72.63</td>
<td>71.12</td>
</tr>
<tr>
<td>0.2% GMf</td>
<td>68.98</td>
<td>70.95</td>
<td>70.31</td>
</tr>
<tr>
<td>SEMg</td>
<td>1.01</td>
<td>0.98</td>
<td>0.99</td>
</tr>
<tr>
<td>Control</td>
<td>68.05h</td>
<td>70.57</td>
<td>68.38</td>
</tr>
<tr>
<td>MYCotoxin*</td>
<td>70.63</td>
<td>72.63</td>
<td>71.12</td>
</tr>
<tr>
<td>0.2% GMf</td>
<td>68.98</td>
<td>70.95</td>
<td>70.31</td>
</tr>
<tr>
<td>SEMg</td>
<td>1.01</td>
<td>0.98</td>
<td>0.99</td>
</tr>
</tbody>
</table>

*a,b,c,dValues are least squares means; n = 6 horses.

Effect of feeding blends of grains naturally contaminated with *Fusarium* mycotoxins on selected serum protein fractions in horses

<table>
<thead>
<tr>
<th>Diet</th>
<th>IgAa</th>
<th>IgMb</th>
<th>IgGc</th>
<th>Total Igd</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>7 d</td>
<td>14 d</td>
<td>21 d</td>
<td></td>
</tr>
<tr>
<td></td>
<td>7 d</td>
<td>14 d</td>
<td>21 d</td>
<td></td>
</tr>
<tr>
<td></td>
<td>7 d</td>
<td>14 d</td>
<td>21 d</td>
<td></td>
</tr>
<tr>
<td>Controlf</td>
<td>2.98</td>
<td>0.65</td>
<td>20.19</td>
<td>23.82</td>
</tr>
<tr>
<td>Mycotoxin*</td>
<td>3.14</td>
<td>0.64</td>
<td>25.40</td>
<td>29.18</td>
</tr>
<tr>
<td>0.2% GMf</td>
<td>3.41</td>
<td>0.74</td>
<td>28.39</td>
<td>32.54</td>
</tr>
<tr>
<td>SEMh</td>
<td>0.66</td>
<td>0.06</td>
<td>2.06</td>
<td>2.43</td>
</tr>
</tbody>
</table>

*a,b,c,dValues are least squares means; n = 6 horses.

The variation in the FA concentrations in the contaminated diets and presence in the control diet may be caused in part by contaminated soybean meal (Smith and Sousadias, 1993; Smith et al., 1997). Matsui and Watanabe (1988) reported that soybean plants could be contaminated with FA. Bacon et al. (1996) stressed that given the numerous common *Fusarium* species that produce FA, the natural occurrence of this compound as a contaminant in foods and feeds should be considered commonplace.

The presence of FA in the test diets, whether contributed from contaminated grains or contaminated soybean meal, needs to be addressed in the context of possible synergistic interactions between FA and DON. Smith et al. (1997) reported a synergistic interaction between DON and FA on weight gains of pigs. Fusaric acid has a very low acute toxicity compared with trichothecene mycotoxins (Hiyakawa et al., 1969). Fusaric acid is a contaminant in foods and feeds should be considered commonplace.

Reports of feeding zearalenone-contaminated diets to pigs indicated that 1 ppm is the minimal concentration required to produce hyperestrogenism (James and Smith, 1982). The minimal concentration required to produce symptoms in the horse is unknown. A field outbreak of zearalenone mycotoxicosis in horses was associated with corn screenings containing approximately 2.6 ppm of zearalenone (Gimeno et al., 1983). The content of deoxynivalenol or FA was not determined. The dietary zearalenone content of about 0.7 ppm in the current experiment should not have caused metabolic effects in the horses tested. *Fusarium graminearum* fungi can produce deoxynivalenol and zearalenone simultaneously in infected corn and wheat (Cote et al., 1985). Toxicological synergism between DON and zearalenone has not been observed in swine (Cote et al., 1985) or mice (Forssel et al., 1986).

Feed Intake and Changes in Body Weight

It was reported that feeding barley naturally contaminated with DON (36 to 44 ppm) to horses did not cause...
Table 8. Effect of feeding blends of grains naturally contaminated with *Fusarium* mycotoxins on d 21 complete and differential white blood cell counts in horses

<table>
<thead>
<tr>
<th>Diet</th>
<th>WBC&lt;sup&gt;a&lt;/sup&gt;</th>
<th>SNC&lt;sup&gt;b&lt;/sup&gt;</th>
<th>LC&lt;sup&gt;c&lt;/sup&gt;</th>
<th>MC&lt;sup&gt;d&lt;/sup&gt;</th>
<th>EC&lt;sup&gt;e&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control&lt;sup&gt;f&lt;/sup&gt;</td>
<td>7.05&lt;sup&gt;j&lt;/sup&gt;</td>
<td>4.07</td>
<td>2.46</td>
<td>0.27</td>
<td>0.13</td>
</tr>
<tr>
<td>Mycotoxin&lt;sup&gt;g&lt;/sup&gt;</td>
<td>7.20</td>
<td>3.46</td>
<td>3.26</td>
<td>0.26</td>
<td>0.17</td>
</tr>
<tr>
<td>0.2% GM&lt;sup&gt;h&lt;/sup&gt;</td>
<td>6.83</td>
<td>3.32</td>
<td>3.01</td>
<td>0.25</td>
<td>0.19</td>
</tr>
<tr>
<td>SEM&lt;sup&gt;i&lt;/sup&gt;</td>
<td>0.20</td>
<td>0.20</td>
<td>0.21</td>
<td>0.02</td>
<td>0.02</td>
</tr>
</tbody>
</table>

<sup>a</sup>White blood cell count, 10<sup>9</sup>/L.<br>
<sup>b</sup>Segmented neutrophil count, 10<sup>9</sup>/L.<br>
<sup>c</sup>Lymphocyte count, 10<sup>9</sup>/L.<br>
<sup>d</sup>Monocyte count, 10<sup>9</sup>/L.<br>
<sup>e</sup>Eosinophil count, 10<sup>9</sup>/L.<br>
<sup>f</sup>Concentrate with control corn and wheat.<br>
<sup>g</sup>Mycotoxin-contaminated concentrate containing 36% contaminated wheat and 53% contaminated corn.<br>
<sup>h</sup>Mycotoxin-contaminated concentrate supplemented with 0.2% glucomannan polymer.<br>
<sup>i</sup>No significant effect of diet on differential leukocyte count was observed (P > 0.05).<br>
<sup>j</sup>Values are least squares means; n = 6 horses.

feed refusal (Johnson et al., 1997). These results are in contrast with those of the current experiment, in which a reduction in feed intake was observed in horses fed contaminated grain containing 14 ppm of DON. Johnson et al. (1997) fed only one source of contaminated grain as compared to the current study in which a blend of naturally contaminated grains was used. A blend would more likely contain combinations of mycotoxins that could act synergistically to decrease feed intake.

It was reported that the horses were fed 2.7 kg of contaminated barley per day, an amount of grain similar to that consumed in the current study, but it is unclear if the contaminated barley was mixed with other uncontaminated feed ingredients. The researchers did not report levels of FA or other possible *Fusarium* mycotoxins, and did not provide information on the use of a control diet or nutritional information on the diet used.

Results of the current experiment suggest a relatively high degree of reduced feed intake when horses are fed concentrates containing a blend of grains naturally contaminated with *Fusarium* mycotoxins. Trichothecene mycotoxins inhibit cellular protein synthesis to varying degrees. This property is likely the cause of many of the pathologies associated with trichothecene toxicosis. T-2 toxicosis results in hyperaminoacidemia (Wannemacher and Dinterman, 1983) and this is likely due to inhibition of hepatic protein synthesis (Meloche and Smith, 1995). Subsequent elevations in blood tryptophan can result in increased concentrations of tryptophan in the brain. Tryptophan is the precursor of the neurotransmitter serotonin and the serotonergic neurons are thought to be important mediators of behaviors such as appetite, muscle coordination and sleep. Serotonin synthesis in the brain is poorly regulated and can be promoted by increased intracellular concentrations of tryptophan (Leathwood, 1987).

Body weights of horses in the current experiment were unaffected by diet. The horses used were mature and non-exercising. It is likely that each supplementation period of 21 d was not long enough for weight loss on the maintenance diet fed.

Hematology and Serum Chemistry

Little work has been reported regarding the effect of feeding *Fusarium* mycotoxin-contaminated grains to horses on hematologic or serum biochemical parameters. Serum activities of the hepatic membrane-associated enzyme, GGT, were higher in horses fed the contaminated diet when compared with the control diet on d 7 and 14, but not on d 21. These findings are in contrast to those of Johnson et al. (1997). Such differences may be due to the previously described differences in experimental protocols. An increase in the activity of this enzyme indicates either hepatocellular damage or enzyme induction. The lack of differences found in serum activities of gamma-glutamyltransferase on d 21 implies that the horses may have adapted to the hepatotoxicity caused by the combination of *Fusarium* mycotoxins.

Serum Immunoglobulin Concentrations

Limited information has been published on the possible immunomodulatory effects of feeding *Fusarium* mycotoxin-contaminated grains to domestic animals. Johnson et al. (1997) fed 36 to 44 ppm of DON to horses and found no significant effects of diet on serum IgA and IgG concentrations. Serum IgM concentrations were not measured. These results are in agreement with the current experiment. Increased serum IgA and IgM concentrations were found in pigs fed the same blend of contaminated grains as the current study (Swamy et al., 2002b). Ingested DON affects intestinal Ig synthesis. Specifically, DON stimulates intestinal IgA production in mice, leading to an elevated concentration of circulating serum IgA (Dong et al., 1991 and Pestka et al., 1989).

Effect of GM Polymer Supplementation

The use of adsorbents, such as activated charcoal, silicates, bentonites, clays and zeolites, in preventing mycotoxicosis has been extensively studied in livestock
exposed to dietary mycotoxins (Ramos et al., 1996). These compounds have sometimes proven impractical due to high dietary inclusion rates. Glucomannan polymer derived from *Saccharomyces cerevisiae* is an organic adsorbent. Glucomannan polymer improved weight gain and feed intake and reduced organ weights in broiler chickens fed aflatoxins (Swamy and Devegowda, 1998) and aflatoxins and T-2 toxin (Raju and Devegowda, 2000). Previous studies in our laboratory indicated that the supplementation of *Fusarium* mycotoxin-contaminated diets with GM polymer prevented some of the mycotoxin-induced alterations in hematology, serum chemistry, biliary IgA concentrations, and brain neurotransmitter concentrations (Swamy et al., 2002a,b). The reduction in consumption of naturally contaminated grains was partially prevented by the feeding of GM polymer in the current study. The increase in serum GGT activities when contaminated grain was fed was prevented with the inclusion of GM polymer, suggesting that toxin absorption was reduced below the threshold of biological activity. Target organs associated with the mixture of mycotoxins present in the experimental diets can be liver, brain, and reproductive organs.

**Implications**

Horses chronically fed *Fusarium* mycotoxin-contaminated grains exhibited a reduction in feed consumption. This effect can have wide implications for the horse industry. Inclusion of contaminated grains in rations for horses should therefore be minimized. Further research is required to determine the effect of *Fusarium*-induced feed reduction and metabolic changes on athletic performance of horses. The capability of glucomannan polymer to decrease the *Fusarium*-induced reduction in feed intake and prevent metabolic changes shows promise for the horse industry.

**Literature Cited**


