COMPARATIVE ASPECTS OF *FUSARIUM* MYCOTOXICOSES IN SWINE, POULTRY AND HORSES

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ABSTRACT

A series of experiments have been conducted to determine the effect of feeding blends of grains naturally-contaminated with *Fusarium* mycotoxins on behavior and metabolism of starter pigs, mature horses, broiler chickens, laying hens and turkeys. Blends of naturally- contaminated corn and wheat were found to contain deoxynivalenol, 15-acetyldeoxynivalenol, zearalenone and fusaric acid. Feed intake was very significantly reduced when contaminated grains were fed to swine and horses. Much higher concentrations of mycotoxins were required, however, to reduce feed intake in poultry. It was determined that elevated brain concentrations of serotonin were seen in both swine and poultry but the anorectic effects of this neurochemical change were cancelled in poultry by a simultaneous elevation in brain catecholamine concentrations. Mycotoxin-induced metabolic changes were much more obvious in poultry than in other species. This was likely due to the lack of protective effect of anorexia. In broiler chickens there were elevations in red blood cell counts and concentrations of hemoglobin and uric acid. An even greater elevation in blood uric acid concentrations was seen in laying hens. Turkeys, which were more sensitive to the feeding of contaminated grains than broilers, had reduced plasma uric acid concentrations. Horses, like swine, appeared to be protected by the anorectic behavior induced by the feeding of contaminated grains with the only metabolic effect being elevated serum gamma-glutamyltransferase activities. Many of the behavioral and metabolic effects of the feeding of grains contaminated with *Fusarium* mycotoxins could be prevented by the use of a polymeric glucomannan mycotoxin adsorbent. It was concluded that species differences in the sensitivity to feed-borne *Fusarium* mycotoxins are mainly due to differing degrees of appetite suppression.

Introduction

Mycotoxins are fungal metabolites which can reduce performance and alter metabolism of livestock and poultry (Wannemacher *et al*., 1991). The pathological states arising from the consumption of feeds contaminated with mycotoxins are referred to as mycotoxicoses. Mycotoxins can be formed in the field preharvest and may continue to be formed under suboptimal storage conditions postharvest. High moisture content often predisposes feedstuffs to fungal growth and mycotoxin formation. Temperature is another key factor . Some fungi, such as *Aspergillus flavus*, are usually found in tropical and semi-tropical climates. This mold produces the carcinogenic hepatotoxin aflatoxin. *Fusarium* fungi, however, are more common in temperate climates and *Fusarium* mycotoxins are likely the most common mycotoxins on a global basis (Wood, 1992).

The global frequency of mycotoxin-contamination of feedstuffs and the severity of mycotoxicoses in livestock and poultry appear to be increasing in recent years. This may be due, in part, to increased monitoring of suspect materials and an increased awareness of the symptoms of mycotoxicoses by veterinarians and producers. Global climate change has also contributed to an increased frequency of mycotoxin contamination of feed grains. Drought, excessive rainfall and flooding can all promote mold growth. Increased international trading of feedstuffs has also contributed to the problem as this increases the chance that a given compound feed will contain components of widely varying geographical origins. Such blends of ingredients increases the chance of the feed containing mixtures of different mycotoxins. This can result in toxicological synergies that increase the severity of mycotoxicoses.

Symptoms typical of mycotoxicoses are often seen despite analysis of the feed that indicates only very low concentrations of mycotoxins (Trenholm *et al*., 1983). In this situation, it is not clear if a mycotoxin problem really exists, or if poor performance is due to management or nutritional factors. It is now known that unexpected toxicity may be due to toxicological interactions between different mycotoxins that exaggerate the toxicity. The likelihood of this occuring is greatest for the *Fusarium* mycotoxins. It has been shown that the feeding of naturally-contaminated feedstuffs produces greater toxicity than the feeding of an equivalent amount of purified mycotoxins (Tremholm *et al*., 1994). It has been shown that fusaric acid, the most common of the *Fusarium* mycotoxins (Bacon *et al*., 1996), can increase the toxicity of the trichothecene deoxynivalenol (vomitoxin, DON) (Smith *et al*., 1997). Fusaric acid, however, is seldom analyzed for in feeds due to its low toxicity when consumed in the absence of other toxins (Smith and MacDonald, 1991; Smith and Sousadias, 1993).

The approach most commonly used to overcome mycotoxicoses in livestock and poultry is the use of specialty feed additives referred to mycotoxin adsorbents (Romos *et al*., 1996). Hydrated sodium calcium aluminosilicate (HSCAS) has been shown to have potential to reduce aflatoxicosis but has been shown to not be effective against *Fusarium* toxicoses (Patterson and Young, 1993). Bentonite has been shown to be effective against T-2 toxin (Carson and Smith, 1983a) but only at levels that are not practical in animal feeds. Other types of clays also have some potential benefits against T-2 toxin but again only a very high levels of dietary inclusion (Smith, 1984). Alfalfa fibre can have protective effects against zearalenone (James and Smith, 1982; Stangroom and Smith, 1984) and T-2 toxin (Carson and Smith, 1983b) but alfalfa is also often a source of *Fusarium* contamination in diets. The problem of high levels of dietary inclusion has now been overcome with the development of polymeric glucomannan mycotoxin adsorbents (GMA) which are extracted from the cell wall of yeast.

Materials and methods

FEEDING TRIAL WITH HORSES

A study was conducted to determine the effect of feeding mature horses a blend of wheat and corn naturally-contaminated with *Fusarium* mycotoxins (Raymond *et al*., 2003) . Changes in intake of concentrate and blood metabolism and the efficacy of GMA to prevent these changes were determined. Nine non-exercising, mature mares were randomly assigned to one of three experimental concentrates for 21 days. The experiment was subsequently replicated in time. Concentrates included: (1) control (2) contaminated grains (3) contaminated grains + 0.2% GMA (Integral, Alltech Canada, Guelph, ON). Concentrates containing contaminated grains contained about 15 ppm deoxynivalenol, 0.75 ppm 15-acetyldeoxynivalenol, 12 ppm fusaric acid and 2.0 ppm zearalenone. The diets offered included 35% concentrates + 65% hay (up to 5 kg / head / day). The hay and straw bedding were not contaminated with *Fusarium* mycotoxins. Concentrate intake, blood biochemical profiles and serum immunoglobulin concentrations were determined weekly.

FEEDING TRIALS WITH SWINE

Neurochemical Study

Starter pigs (initial weight approximately 10 kg) were fed 5 diets (7 pens of 5 pigs per diet) for three weeks (Swamy *et al*., 2002b). The diets included: (1) control (2) blend of wheat and corn naturally-contaminated with *Fusarium* mycotoxins (3) contaminated grains + 0.2% GMA. The diets including contaminated grains contained about 5.5 ppm deoxynivalenol, 0.5 ppm 15-

acetyldeoxynivalenol, 0.3 ppm zearalenone and 28 ppm fusaric acid. At the end of the feeding period, animals were killed and brains were excised and dissected. Brain tissue concentrations of indoleamines and catecholamines and metabolites were analyzed. Blood samples were also taken after three weeks of feeding and hematological and serum chemistry measurements were taken.

Pair Feeding Study

Starter pigs (initial weight approximately 9 kg) were fed 5 diets (7 pens of 5 pigs per diet) for three weeks (Swamy *et al*., 2003a). The diets included : (1) control (2) low level of contaminated grains (2.2 ppm deoxynivalenol + 36.22 ppm fusaric acid) (3) high level of contaminated grains (2.9 ppm deoxynivalenol + 49.28 ppm fusaric acid) (4) high level of contaminated grains + 0.2% GMA (2.8 ppm deoxynivalenol + 20.93 ppm fusaric acid (5) pigs pairfed the control diet to the intake of pigs fed the high level of contaminated grains. Weight gains and feed intake were determined weekly. At the end of the experiment, all pigs were bled for determination of serum metabolites.

FEEDING TRIALS WITH POULTRY

Broiler Study

A total of 360 broiler chicks of a commercial strain were fed 4 diets for 56 days (Swamy *et al*., 2002a). The diets included: (1) control (2) low level of contaminated grains (4.7ppm deoxynivalenol + 20.6 ppm fusaric acid + 0.2 ppm zearalenone (3) high level of contaminated grains (8.2 ppm deoxynivalenol + 21.6 ppm fusaric acid + 0.56 ppm zearalenone (4) high level of contaminated grains + 0.2% GMA (9.7 ppm deoxynivalenol + 21.6 ppm fusaric acid + 0.8m ppm zearalenone). Weight gain and feed intake were determined weekly. Blood and biliary samples were collected after three and eight weeks and were analyzed for hematology and serum chemistry.

Layer Study

A total of 145 45 week-old laying hens were fed diets including: (1) control (2) contaminated grains (3) contaminated grains + 0.2% GMA for 12 weeks. The diets including contaminated grains were found to contain 12 ppm deoxynivalenol, 0.5 ppm 15-acetyldeoxynivalenol and 0.6 ppm zearalenone. Parameters measured after 4, 8 and 12 weeks included feed consumption, rate of lay, egg and eggshell quality and plasma chemistry.

Turkey Study

A total of 225 day-old male turkey poults were fed corn, wheat and soybean meal-based starter (0-3 weeks), grower (4-6 weeks), developer (7-9 weeks) and finisher (10-12 weeks) diets for a total of 12 weeks. Diets included: (1) controls (2) contaminated grains (3) contaminated grains + 0.2% GMA. Parameters measured included weight gain, feed consumption and serum chemistry.

Results and discussion

FEEDING TRIAL WITH HORSES

The feeding of contaminated grains to horses in the current study resulted in reduced concentrate intake compared to controls ($P<0.05$, Table 1). Supplementation with GMA to the blend of contaminated grains significantly improved concentrate intake compared to the feeding of contaminated grains. Consumption of forage remained unaffected regardless of diet fed. Gamma-glutamyltransferase levels were significantly higher in serum of horses consuming contaminated grain on days 7 and 14 but not on day 21, thereby implying that the horses might be adapting to the hepatotoxicity caused by the mixture of *Fusarium* mycotoxins. The feeding of GMA prevented this hepatotoxicity.

Table 1. Effect of feeding blends of grains naturally-contaminated with *Fusarium* mycotoxins on feed intake and hepatic metabolism of horses[1].

Diet	Concentrate Intake (kg / day)	GGT[2] Day 7 (U/ L serum)	GGT Day 14 (U/L serum)	GGT Day 21 (U/L serum)
Control	2.72[b]	11.44[b]	12.23[b]	11.49[b]
Contaminated	0.98[c]	22.98[c]	23.86[c]	15.27[b]
Contaminated + 0.2% GMA[3]	1.71[d]	8.57[b]	9.58[b]	8.24[b]

[1]From Raymond *et al*., 2003.
[2]Gamma-glutamyltransferase activity.
[3]Glucomannan mycotoxin adsorbent.
[b,c]Means in a column without a common superscript differ (P<0.05).

It has been reported that horses are quite resistant to the effects of deoxynivalenol-contaminated barley (Johnson *et al.*, 1997). The feeding of 1.27 kg of barley contaminated with 36-44 ppm deoxynivalenol per day for 40 days was determined to not affect feed intake or immunological status of horses. It is likely that the feeding of a blend of contaminated grains in the current study resulted in combinations of mycotoxins and toxicological synergies (Smith *et al*., 1997) that were not present in the study of Johnson *et al*. (1997). It was concluded that supplementation of GMA to diets naturally-contaminated with *Fusarium* mycotoxins was beneficial in alleviating reduced feed intake and metabolic changes in mature horses.

FEEDING TRIALS WITH SWINE

Neurochemical Study

It was observed that the feeding of grains naturally-contaminated with a combination of *Fusarium* mycotoxins for three weeks resulted in several neurochemical changes (Table 2). In the pons region there was a significant elevation in norepinephrine concentration and also an elevation in the 5-hydroxyindoleacetic acid : serotonin ratio. This ratio was also elevated in the hypothalamus where a decrease in dopamine concentration was seen. The feeding of GMA prevented most of these changes.

Table 2. Effect of feeding blends of grains naturally-contaminated with *Fusarium* mycotoxins on regional brain neurochemistry of starter pigs[1].

Diet	NE[2]	DA[3]	5-HIAA:5-HT (P)[4]	5-HIAA:5-HT (H)[5]
Control	3.71[a]	1.74[a]	0.57[a]	0.43[a]
Contaminated grains	3.09[b]	1.17[b]	0.71[b]	0.49[b]
Contaminated grains + 0.2% GMA[6]	3.45[a]	1.40[a]	0.67[a,b]	0.45[a]

[1]From Swamy *et al*., 2002a.
[2]Norepinephrine concentration (nmol / g pons tissue).
[3]Dopamine concentration (nmol / g hypothalamus tissue).
[4]Ratio of concentrations of 5-hydroxyindoleacetic acid to serotonin in pons tissue.
[5]Ratio of concentrations of 5-hydroxyindoleacetic acid to serotonin in hypothalamus tissue.
[6]Glucomannan mycotoxin adsorbent.
[a,b]Means within a column without a common superscript differ significantly (P<0.05).

The elevations in 5-hydroxyindoleacetic acid : serotonin ratios are similar to that seen when pigs were orally dosed with fusaric acid (Smith and MacDonald, 1991). Chronic administration of deoxynivalenol to pigs resulted in similar effects (Prelusky, 1993). Elevations in brain serotonin concentrations have been shown to result in reduced appetite, loss of muscle coordination and lethargy (Leathwood, 1987).

Pair Feeding Study

The effect of feeding blends on grains naturally-contaminated with *Fusarium* mycotoxins on weight gain, feed consumption and feed efficiency of starter pigs is given in Table 3. There was a significant linear decline in weight gain and feed consumption with the feeding of increasing levels of contaminated grains. Feed efficiency was largely unaffected by diet. The effect of diet on serum metabolites is given in Table 4. It was observed that pigs pairfed to the feed intake of those consuming the high level of contaminated grains had significantly higher concentrations of total serum proteins and globulins. This infers that the adverse effects of feeding grains naturally-contaminated with *Fusarium* mycotoxins are due not only to appetite suppression but are also due to metabolic changes. It is of note that the feeding of GMA prevented these changes. Serum concentrations of conjugated bilirubin are an index of hunger. This was reflected in the observation that serum conjugated bilirubin concentrations were significantly elevated in pairfed pigs compared to controls (P<0.0134). The toxic effects on feed intake resulting from the high mycotoxin concentrations used in the current study were too great to be overcome by the feeding of 0.2% GMA.

Table 3. Effect of feeding blends of grains naturally-contaminated with *Fusarium* mycotoxins on growth rate, feed consumption and feed efficiency of starter pigs 0-21d)[1].

Diet	Growth (kg/pig)	Feed Consumption (kg/pen)	Feed Efficiency (gain/feed)
Control	9.54	75.64	0.630
Low level of contaminated grains	7.88	61.32	0.637
High level of contaminated grains	6.89	50.44	0.680
High level of contaminated grains + 0.2% GMA[2]	6.38	52.84	0.609
Pairfed to high level of contaminated grains	6.58	48.39	0.691
Pooled standard deviation	1.79	6.49	0.039
Linear effect	<0.0001	<0.0001	NS[3]
Control vs pairfed	<0.0001	<0.0001	0.0173
Pairfed vs high level of contaminated grains	NS	NS	NS

[1]From Swamy *et al*., 2003.
[2]Glucomannan mycotoxin adsorbent.
[3]Not significant (P>0.05).

Table 4. Effect of feeding blends of grains naturally-contaminated with *Fusarium* mycotoxins on serum metabolites of starter pigs (0-21d)[1].

Diet	Total Proteins (g/L)	Globulins (g/L)	Albumin/ Globulin	Conjugated Bilirubin (umoles/L)
Control	46.33	18.00	1.608	0.166
Low level of contaminated grains	47.83	18.33	1.733	0.416
High level of contaminated grains	45.50	14.67	2.163	0.333
High level of contaminated grains + 0.2% GMA[2]	47.00	16.33	2.017	0.250
Pairfed to high level of contaminated grains	50.50	18.42	1.901	0.750
Pooled standard deviation	3.84	4.74	0.61	0.508
Linear effect	NS[3]	NS	0.01	NS
Control vs pairfed	<0.05	NS	NS	<0.01
Pairfed vs high level of contaminated grains	<0.02	<0.02	NS	NS

[1]From Swamy *et al*., 2003.
[2]Glucomannan mycotoxin adsorbent.
[3]Not significant (P>0.05).

FEEDING TRIALS WITH POULTRY

Broiler Study

It was observed that the feeding of increasing levels of contaminated grains to broilers resulted in a linear decrease in growth rates and feed intake (P < 0.05, Table 5). This was not seen, however, until birds were in the grower phase. Elevations in red blood cell counts and in serum hemoglobin and uric acid concentrations were noted at the end of the study (Table 6). Most of these effects were prevented by the feeding of GMA. Broilers were less sensitive than horses or swine with respect to reductions in feed intake caused by the feeding of contaminated grains. The absence of this protective behavior resulted in numerous adverse metabolic changes that progressed with the consumption of contaminated materials. The observation that the feeding of contaminated grains to broilers resulted in reduced growth only in the grower and finisher phases supports the concept that broilers do not exhibit feed refusal in a manner similar to swine fed *Fusarium* mycotoxin contaminated diets (Smith *et al*., 1997). The reason for this species difference has been shown to be differences in the effects on brain neurochemistry (Swamy *et al*., 2004b). The feeding of contaminated grains to pigs elevated brain serotonin concentrations. In broilers, such treatments elevated both serotonin and catecholamines thereby canceling the effect of serotonin on appetite suppression. It is likely that mycotoxin-induced growth suppression in the broilers was due to gradual alterations in metabolism that occurred with extended feeding of contaminated grains. The alterations in red blood cell counts and hemoglobin concentrations are similar to blood changes seen in ascites. In the current study, this may have been partially due to the hypotensive effect of fusaric acid. Reduced blood flow to the lungs may have constituted a stress which resulted in an adaptation and increased oxygen trapping capacity of the blood. The elevations in serum uric acid concentrations may be a reflectioin of the reduction in hepatic protein synthesis caused by the trichothecene mycotoxins deoxynivalenol and 15-acetyldeoxynivalenol. Elevated hepatic free amino acid concentrations would result in increased amino acid oxidation and increased nitrogen excretion in the form of uric acid.

Table 5. Effect of feeding blends of grains naturally-contaminated with *Fusarium* mycotoxins on weight gain and feed consumption of broiler chickens.[1]

Diet	Feed consumption (g/bird)[2]			Weight gain (g/bird)[3]		
	0 – 21 d	21 – 42 d	42 – 56 d	0 – 21 d	21 – 42 d	42 – 56 d
Control	908	2797	2544	435	1678	1303
Low Mycotoxins	841	2565	2437	376	1522	1274
High Mycotoxins	923	2392	2456	386	1479	1319
High Mycotoxins + 0.2% GMA[4]	968	2472	2532	392	1538	1348
SEM	37	60	27	6	16	13
Linear effect	NS[5]	0.05	NS	NS	0.04	NS

[1]From Swamy et al., 2004a.
[2]Values are least square means; n = 3.
[3]Values are least square means; n = 90.
[4]Glucomannan mycotoxin adsorbent.
[5]Not significant (P>0.05).

Table 6. Effect of feeding blends of grains naturally-contaminated with *Fusarium* mycotoxins on hematology, serum chemistry and breast meat coloration of broiler chickens.[1]

Diet	RBC[2]	Hb[3]	Uric Acid[4]	Redness[5]	Biliary IgA[6]
Control	2.66	95.0	259	0.45	7.54
Low Mycotoxins	2.84	101.1	286	0.67	7.28
High Mycotoxins	2.83	99.2	357	0.80	4.99
High Mycotoxins + 0.2% GMA[7]	2.54	91.2	281	0.21	6.54
SEM	0.04	1.37	10.9	0.07	0.29
Linear effect	0.01	0.01	0.009		0.01

[1]From Swamy et al., 2002a.
[2]Red blood corpuscle counts (10^{12} / L); n = 12.
[3] Hemoglobin concentration (ppm); n = 12.
[4] Uric acid concentration (umoles / L); n = 12.
[5] Unitless scale, 0 = green, 1 = red; n = 15.
[6] mm precipitate; n = 15.
[7]Glucomannan mycotoxin adsorbent.

Layer Study

The feeding of contaminated grains to laying hens decreased feed intake relative to controls in the first month but increased feed intake thereafter (Table 7). This resulted in a large deterioration of feed efficiency in the latter months of the experiment. Egg production also declined in months 1 and 2 (Table 8). Serum uric acid concentrations were highly elevated throughout the study when contaminated grains were fed (P < 0.001). This effect was prevented by the feeding of GMA. It can be concluded that laying hens are less sensitive to the anorectic effects of contaminated grains than broilers. This is perhaps why metabolic changes such as elevated serum uric acid concentrations in layers are exaggerated compared to those seen in broilers.

Table 7. Effect of feeding blends of grains naturally-contaminated with *Fusarium* mycotoxins on feed consumption and feed efficiency of laying hens.

	Feed consumption[1]			Feed efficiency[2]		
Diet	Wk 0 - 4	Wk 4 - 8	Wk 8 - 12	Wk 0 - 4	Wk 4 - 8	Wk 8 - 12
Control	119	120	117	1.88	1.92	1.90
Mycotoxins	106	127	132	1.94	2.29	2.23
Mycotoxins + 0.2% GMA[3]	114	124	121	1.90	2.10	1.94
Pooled SD	9	9	7	0.18	0.27	0.17
Control vs Mycotoxins	0.008	0.04	0.0001	NS[4]	0.001	0.001
Mycotoxins vs MTB-100	NS	NS	0.0006	NS	NS	0.003

[1]g / hen / day; n = 12.
[2]feed consumed / egg mass; n = 12.
[3]Glucomannan mycotoxin adsorbent.
[4]Not significant (P>0.05).

Table 8. Effect of feeding blends of grains naturally-contaminated with *Fusarium* mycotoxins on organ weights and plasma uric acid concentrations of laying hens.

	Organ weights (g)			Uric acid (umol / L)		
Diet	Liver	Spleen	Kidney	Wk 4	Wk 8	Wk 12
Control	44.5[1]	2.2	5.9	376	392	390
Mycotoxins	44.2	2.4	7.6	1009	1154	1030
Mycotoxins + 0.2% GMA[2]	46.2	2.2	6.6	500	539	487
Pooled SD	7.58	0.66	0.98	188	156	159
Control vs Mycotoxins	NS[3]	NS	0.002	0.001	0.001	0.001
Mycotoxins vs MTB-100	NS	NS	0.02	0.001	0.001	0.001

[1]n = 12.
[2]Glucomannan mycotoxin adsorbent.
[3]Not significant (P>0.05).

Turkey Study

The feeding of contaminated grains reduced growth rates in the starter, developer and finisher phases and overall (Table 9). The feeding of GMA prevented these effects. The most obvious effect of diet on plasma chemistry was on plasma uric acid concentrations (Table 10) . After 4 and 8 weeks of feeding contaminated grains, plasma uric acid concentrations were significantly reduced. This effect was not seen, however, with the feeding of GMA. At the end of the experiment, turkeys fed contaminated grains + GMA had significantly smaller spleens and kidneys and significantly larger bursas than birds fed unsupplemented contaminated grains.

Table 9. Effect of feeding blends of grains naturally-contaminated with *Fusarium* mycotoxins on growth of turkeys (g / bird / week).

Diet	Starter	Grower	Developer	Finisher	Overall
Control	217	378	748	723	517
Mycotoxins	196	492	657	637	496
Mycotoxins + 0.2% GMA[1]	213	548	756	852	592
Control vs Mycotoxins	0.01	0.01	0.01	0.05	0.05
Mycotoxins vs GMA	0.05	0.05	0.05	0.01	0.01

[1]Glucomannan mycotoxin adsorbent.

Table 10. Effect of feeding blends of grains naturally-contaminated with *Fusarium* mycotoxins on organ weights and plasma uric acid concentrations of turkeys.

	Organ weights (g)			Uric acid (umol / L)		
Diet	Bursa	Spleen	Kidney	Wk 4	Wk 8	Wk 12
Control	5.3[1]	5.6	16.1	437	339	276
Mycotoxins	5.5	6.1	17.7	299	210	305
Mycotoxins + 0.2% GMA[2]	6.5	5.1	15.8	321	197	235
SEM	0.30	0.33	0.71	27	15	43
Control vs Mycotoxins	NS[3]	NS	NS	0.001	0.001	NS
Mycotoxins vs GMA	0.03	0.05	0.05	NS	NS	NS

[1]n = 12.
[2]Glucomannan mycotoxin adsorbent.
[3]Not significant (P>0.05).

Broilers, layers and turkeys are all sensitive to *Fusarium* mycotoxicoses. The adverse effects of the diets fed in the current studies are greater than literature reports based on the DON content. This is likely because of the relatively short duration of previously reported trials. The blending of different naturally-contaminated grains, moreover, also results in a more complex mixture of mycotoxins thereby increasing the chances of toxicological synergy between mycotoxins.

Conclusions

It can be concluded that the adverse effects of *Fusarium* mycotoxins in a wide range of animal species can be overcome by the feeding of a suitable mycotoxin adsorbent such as GMA. This has important economic consequences when wide spread contamination of the feed supply forces the feeding of contaminated grains or when favorable pricing prompts the intentional feeding of contaminated materials.

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