



The Effects of Mycotoxins in Swine

A Review for Swine Producers

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INTRODUCTION

Mycotoxins are found in virtually all livestock feed. Mycotoxins are produced by several species of fungi. The fungi that produce mycotoxins of particular concern for the swine industry are *Aspergillus*, *Fusarium*, and *Penicillium*. While over 400 known mycotoxins and their different isolates have been identified, aflatoxin, fumonisin, ochratoxin, T-2 toxin, vomitoxin (deoxynivalenol), and zearalenone have captured the most attention. One or more of these mycotoxins contaminate an estimated one-quarter of world's food crops. Therefore, understanding how mycotoxins affect swine is important for producers to properly control them and prevent economic losses. Among these common mycotoxins, aflatoxin (AFL) is one of the most toxic to swine and is primarily produced by *Aspergillus* fungi species. A few hundred ppb (parts per billion; µg/kg) of aflatoxin-contaminated feed is especially dangerous to piglets.

The clinical symptoms of mycotoxin intoxication have been well established. However, while swine genetics and farm management have improved during the past few decades, it remains crucial to review how modern genetics respond to the dietary mycotoxin challenge. This paper reviews published data on the effects of mycotoxins in swine.

Abbreviated names for common mycotoxins used in the paper are:

AFL – aflatoxin FUM – fumonisin FUA – fusaric acid
 ZEA – zearalenone OTA - ochratoxin
 DON – deoxynivalenol (vomitoxin)

Five major classes of mycotoxins are described below in terms of their primary origins and biological effects on swine:

Mycotoxins	Primary Origins*	Effects on Swine
Aflatoxin	<i>A. flavus</i> <i>A. paracitrus</i> <i>A. nomius</i> <i>A. pseudotamarii</i>	<ul style="list-style-type: none"> • Reduces feed intake and weight gain • Reduces feed efficiency • Immunosuppressive • Increases mortality • Results in liver damage, such as fatty liver • Causes hemorrhaging of the kidney and intestine • Carcinogenic and teratogenic
Fumonisin	<i>F. moniliforme</i> <i>F. verticillioides</i>	<ul style="list-style-type: none"> • Reduces feed intake and weight gain • Affects sphingolipids metabolism • Results in liver damage • Results in pulmonary edema
Ochratoxin	<i>A. ochraceus</i> <i>P. verrucosum</i> <i>P. palitans</i>	<ul style="list-style-type: none"> • Reduces growth performance • Results in kidney damage and mild liver damage • Teratogenic and carcinogenic • Immunosuppressive
Trichothecenes Deoxynivalenol	<i>F. graminearum</i>	<ul style="list-style-type: none"> • Reduces feed intake and feed efficiency • Induces vomiting (DON) • Immunosuppressive (greater for T-2) • Myocardial and pancreatic lesions (T-2)
T-2 Toxin	<i>F. sporotrichioides</i>	
Zearalenone	<i>F. graminearum</i>	<ul style="list-style-type: none"> • Estrogenic effects: enlargement of vulva and reduced reproductive efficiency • Results in atrophy of ovaries, mammary gland, and testicles • Increases abortion

*A. – *Aspergillus*; F. – *Fusarium*; P. – *Penicillium*

AFLATOXIN

In early 1950, cattle and swine death losses were reported in the United States from the consumption of moldy corn (Sippel et al., 1953). Toxic substances from *Aspergillus* and *Penicillium* fungi were identified to have caused the problem (Burnside et al., 1957). British scientists first purified the substance, named aflatoxin, from peanut meal (Allcroft et al., 1961). Since then, many acute and chronic toxicity studies of AFL have been conducted and published. Aflatoxins occur in four major forms in grains: AFL B₁, B₂, G₁, and G₂. Aflatoxin B₁ is the most common and considered the most biologically active form.

Research completed by Schell et al. (1993) proved that young pigs are sensitive to dietary AFL. The group at Virginia Polytechnic Institute used a total of 198 weanling pigs in three trials. Pigs consumed AFL contaminated feed (500 or 800 ppb), which resulted in significant reductions of average daily gain (ADG; P<0.05) in all three trials. The results are shown in the Table 1. Reduced feed efficiency was observed in Trial I, in which 800 ppb AFL was fed, but average daily feed intake (ADI) was not affected. In Trial II (500 ppb AFL) and III (800 ppb AFL), the reduced ADG could have occurred mainly due to the reduced feed intake.

TABLE 1: Weanling pigs fed aflatoxin (AFL)-free or AFL-contaminated feed

AFL, ppb	Trial I – 4 wks (Initial wt. 10.7 kg)		Trial II – 5 wks (Initial wt. 9.6 kg)		Trial III – 4 wks (Initial wt. 10.0 kg)	
	0	800	0	500	0	800
ADG, g	640 ^a	480 ^b	660 ^a	460 ^b	630 ^a	520 ^b
ADFI, g	1320	1170	1410 ^a	970 ^b	1290 ^a	1020 ^b
G : F	0.48 ^a	0.41 ^b	0.47	0.47	0.49	0.51

^{ab} Results, within a trial, with different superscripts are significantly different

Coffey et al. (1989) conducted two trials to investigate the interaction of additional dietary nutrients and dietary AFL from contaminated corn. A total of 192 pigs were used in the first trial and 96 in the second. The control and contaminated corn contained 6 and 182 ppb AFL, respectively, and the dietary inclusion of corn varied between 62 and 73% for Study I and was a constant 73% in Study II. The diets were fed to pigs for 4 weeks in both studies. In the first study, a 2 x 2 x 2 factorial arrangement was used to examine dietary AFL, protein, and fat interactions. The results are shown in Table 2.

TABLE 2: Effect of aflatoxin (AFL), added fat, and dietary protein on performance of pigs.

	18% Dietary Protein			
	0% Fat		5% Fat	
	Clean*	AFL*	Clean	AFL
Average Daily Gain, g	380	290	360	310
Average Daily Feed Intake, g	680	560	640	580
Feed : Gain	1.81	1.92	1.76	1.85
	20% Dietary Protein			
	0% Fat		5% Fat	
	Clean	AFL	Clean	AFL
Average Daily Gain, g	370	370	360	350
Average Daily Feed Intake, g	670	640	620	610
Feed : Gain	1.84	1.75	1.70	1.74

* Clean – diet formulated with clean corn; AFL – diet formulated with corn containing 182 ppb aflatoxin B₁

Pigs fed a high protein (20%) diet showed no detrimental effects on performance by feeding diets containing contaminated corn. However, when 18% protein was fed ADG, ADI, and feed conversion was worse (P<0.05) in the pigs fed AFL contaminated corn. This suggests that pigs receiving higher dietary protein can tolerate greater AFL contamination.

In the second study by Coffey et al. (1989), a 2 x 2 x 2 factorial arrangement was also used to examine dietary aflatoxin, lysine, and methionine interactions when 18% dietary protein was fed. The results are shown in Table 3. A significant decrease in ADG was observed when AFL contaminated corn was added to the control diet (440 g vs 500 g; P<0.05). However, the ADG returned to the level of the control pigs when 0.25% synthetic lysine was added (510 g) to the AFL contaminated diet, resulting in a significant AFL by lysine interaction. When 0.15% synthetic methionine was added to the AFL contaminated diet ADG was only partially restored (P>0.05). The results suggest that a lysine-fortified diet can improve growth performance in pigs if feeding a marginal protein (18%) diet and AFL is present in the feed.

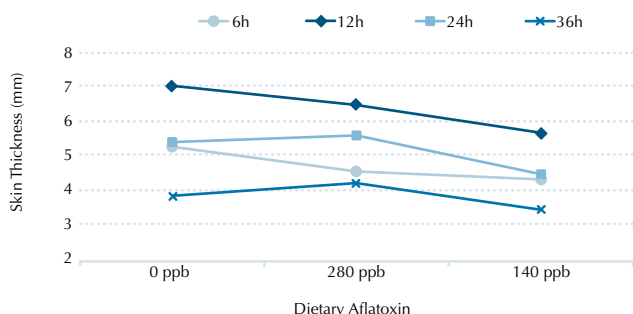
TABLE 3: Effects of lysine and methionine supplementation in aflatoxin (AFL)-contaminated feed in pigs.

	Control		+0.25% Lysine		+0.15% Methionine	
	Clean*	AFL*	Clean	AFL	Clean	AFL
Average Daily Gain, g	500	440	490	510	510	480
Average Daily Feed Intake, g	990	930	970	1,000	1,120	1,020
Feed : Gain	2.01	2.04	1.96	1.95	2.03	2.06

* Clean – diet formulated with clean corn; AFL – diet formulated with corn containing 182 ppb aflatoxin B1

Researchers at North Carolina State University (van Heugten et al., 1994) used a total of 288 pigs, weaned at day 21, in an immune response study by feeding various levels of AFL and methionine. The pigs were divided into 12 treatments in a 3 x 4 factorial arrangement with three levels of AFL (0, 140 and 280 ppb) and four levels of methionine (0, 0.15, 0.30 and 0.45%) added to the diets. Skin thickness was measured 6, 12, 24, and 36 hours after a subcutaneous injection of phytohemagglutinin to determine cellular immunity. The thinner the skin at the injection site suggests a depression in cellular immunity. The authors reported a significant linear AFL response on skin thickness at 12 hours ($P < 0.05$) and 24 hours ($P < 0.10$) post injection as seen by the decrease in skin thickness with increase in dietary AFL in Figure 1. Antibody response to sheep red blood cell and serum IgG, IgM concentrations were not affected by dietary treatments. Researchers concluded that low levels of AFL may depress certain cellular immunity and a methionine supplement did not improve immune functions in pigs fed aflatoxin.

FIGURE 1: Main effect of aflatoxin on skin thickness of pigs



Joint research between France and Romania on AFL was recently reported (Martin et al., 2002). The study used a total of 36 weanling pigs that were fed diets containing 0, 140, or 280 ppb AFL for 4 weeks. In the study, a high protein (21.32% CP for 21 days and 18.61% CP for last 8 days) diet was fed to the pigs. Growth performance, blood parameters, and cellular

immune response were measured and the ADG results are shown in the Table 4.

TABLE 4: Effect of low levels of aflatoxin (AFL) on weanling pigs weight gain.

Days Fed Diets	0 ppb AFL	140 ppb AFL	280 ppb AFL
	Average Daily Gain (g)		
0 to 15 d	252	210	191
0 to 22 d	349 ^a	349 ^a	218 ^b
0 to 30 d	489 ^a	453 ^a	326 ^b

^{ab} Results, within a row, with different superscripts are significantly different

Pigs fed the lowest level of AFL (140 ppb) showed no difference compared to pigs fed aflatoxin free diets in respect to weight gain. However, pigs fed 280 ppb showed significant reduction in weight gain ($P < 0.05$). There were no differences found in red blood cell numbers and their lymphocyte, monocyte, and neutrophil numbers or serum protein, albumin and globulin concentrations between treatments. Pigs fed 280 ppb dietary aflatoxin showed decreased pro-inflammatory cytokines expression (interleukin 1 and tumor necrosis factor mRNAs) and increased anti-inflammatory cytokines expression (interleukin 10 mRNA). This suggests a low dose of dietary AFL induced an alteration of immune response as well as depressed growth of pigs.

Not only do young pigs respond to AFL loads, older pigs also show detrimental effects of AFL-contaminated diets. Southern and Clawson (1979) fed thirty-two 53 kg growing-finishing pigs 20 to 1,480 ppb aflatoxin for 66 days. Average daily gain and ADI were decreased linearly ($P < 0.05$) as dietary AFL increased (Table 5). Feed conversion was not changed except for the pigs fed the highest level of AFL (1480 ppb). After the 66 day feeding trial, 4 of the 8 pigs fed 385, 750, or 1480 ppb AFL feed were fed the control feed for 7 days, while the other 4 remained on the toxin diets. The final weight (average of all pigs per treatment) after 66 days plus the 7 day withdrawal period was dramatically different between the control (106 kg) and the highest dietary aflatoxin (83 kg), a 23 kg difference. However, the 4 pigs per treatment consuming the control feed for the last 7 days (withdrawal, after feeding toxin) had increased ADI (+130 g/day), ADG (+290 g/day), and improved feed conversion (-3.25 g feed/g gain) compared to their counterparts consuming the AFL feed. The percent of liver weight relative to body weight and the incidence of liver lesions increased linearly as aflatoxin levels were increased (Table 6). Serum protein and its relative albumin and IgG (gamma globulins) concentrations were not affected by dietary aflatoxin in the study. However, the IgM fraction of the gamma

globulins was statistically higher ($P<0.05$) in the 750 and 1480 ppm AFL fed pigs; suggesting some immunological affects from AFL consumption.

TABLE 5: Effect of feeding different levels of aflatoxin in growing-finishing pigs.

AFL, ppb	20	385	750	1480
Average Daily Gain, g	770 ^a	670 ^b	570 ^b	410 ^c
Average Daily Feed Intake, g	2870 ^a	2530 ^b	2150 ^c	1610 ^d
Feed : Gain	3.74 ^a	3.78 ^a	3.71 ^a	3.97 ^b

TABLE 6: Effects of aflatoxin on pig liver size and lesions.

AFL, ppb	20	385	750	1480
Pig wt, kg	106	99	96	83
Liver wt, kg	1.39	1.53	1.52	1.49
% Liver to body wt	1.31 ^a	1.55 ^b	1.58 ^b	1.77 ^c
Visual lesion	1 out of 8	2 out of 8	4 out of 8	5 out of 7

In the CAST (2003) book several physical and chemical methods were reviewed for detoxification of mycotoxins in grain or feed, but variable results were discussed depending on mycotoxin. Heat treatment of moldy corn, soybean meal, peanut, etc. has shown to reduce the detrimental effects of AFL in animals. However, AFL is heat stable and will not be completely destroyed by thermal processing. Also, nutrients, such as lysine and methionine (Hale and Wilson, 1979), are damaged under high temperature treatment; and therefore poor digestibility and poor feed efficiency were obtained with heat treatment of moldy corn. In addition, there is a high energy cost associated with heat treatment of feed which may not be economically feasible.

The results from the study by Hale and Wilson (1979), in which pigs were fed heat-treated moldy corn, are shown in Table 7. Forty-eight pigs with an average starting weight of 18.3 kg were used in the 2 x 2 factorial study. Pigs were fed AFL contaminated or AFL free corn that was either unheated or heated to 160-180°C in an oven with a capacity of 1,500 kg per hour. The AFL contaminated corn averaged 383 ppb before heating and 60 ppb AFL after the heat treatment.

TABLE 7: Effects of heat treatment on aflatoxin (AFL) contaminated corn in growing pigs.

Corn Treatment	Heated Corn (160-180°C)		Unheated Corn	
	0 ppb	42 ppb	0 ppb	345 ppb
Total AFL ¹	0 ppb	42 ppb	0 ppb	345 ppb
Average Daily Gain, g	740	750	760	720
Feed : Gain, g:g	2.87	2.91	2.73	2.94
Nitrogen (N) retention, %	71.1	68.7	76.7	70.6
Digestibility, %				
Dry matter	83.9	82.6	84.4	83.4
Fat	42.0	37.7	41.6	37.2
Protein (based on N)	77.3	76.6	80.2	78.0

¹ Total analyzed aflatoxin in feed after unheated (383 ppb AFL) or heated corn (60 ppb AFL) was added to the diet

Pigs fed corn that was heated (for about an hour at > 160°C) showed less nitrogen retention, poor protein (nitrogen) digestibility, and increased ADI resulting in a numerically worse feed conversion than pigs fed unheated corn. The results were similar to pigs fed dietary AFL without heat treatment. Based on the data, we can conclude that heat is not a good option in treating AFL contaminated feeds.

The di-ketone structure of AFL is easily oxidized and generates peroxide and free radicals once it enters animal cells. Lindemann et al. (1993) fed pigs with a dietary supplement of selenium (Se) and folic acid (FA) to see if these nutrients would prevent oxidation damage in pigs. The results are shown in Table 8. Linear effects on performance were observed; as dietary AFL increased ($P<0.05$), ADG, ADI and feed efficiency decreased. The addition of 0.6 ppm Se did not improve ADG, feed intake or feed efficiency, but 2 ppm FA did improve ($P<0.05$) ADG by 40%. However, the addition of 0.5% of hydrated sodium calcium aluminosilicate (HSCA) did improve feed intake and weight gain (almost to the level of controls) but not feed efficiency.

TABLE 8: Effects of selenium (Se), folic acid (FA) and hydrate sodium calcium aluminosilicate (HSCA) on aflatoxin contaminated diets in pigs.

AFL, ppb	Basal		Basal +0.6 ppm Se	Basal +2 ppm FA	Basal +0.5% HSCA	
	0	420	840	840	840	
Average Daily Gain, g	520	460	280	310	370	480
Average Daily Feed Intake, g	1130	950	670	680	830	1170
Gain : Feed	0.58	0.52	0.37	0.44	0.48	0.47

OCHRATOXIN

Ochratoxin is commonly produced by *Penicillium* and *Aspergillus* fungi. The primary toxins are identified as ochratoxin A (OTA) and its less potent analog ochratoxin B. There is concern for public health since these toxins have been isolated in cereals, coffee beans, dried fruits and wine. Other than the aforementioned naturally contaminated foods, OTA from meat and organs of swine is also an important concern for human OTA consumption. The presence of OTA in human blood has been study in many different countries and regions. In one particular investigation in Canada, between 0.29 and 2.37 ng OTA/ml was found in human blood with a mean concentration of 0.88 ng OTA/ml (Scott et al., 1998). In a report by Stoev et al. (1998), 50 slaughtered pigs in Bulgaria (intended for human consumption) were inspected and found to have “enlarged mottled or pale kidneys”. Serum samples were taken from all of the pigs and were found to contain OTA.

Few recent studies have been conducted and published regarding ochratoxin in swine. During the late 1970s and early 1980s, high levels of ochratoxin were found in Danish barley. As a result, a series of experiments were completed by a Danish group (Madsen et al., 1982) using barley batches containing levels of OTA varying between 0 and 2,300 ppb. The study showed that under the specific pathogen free (SPF) conditions, with good nutrition and management, the pigs’ health in general was good even after feeding ochratoxin contaminated feeds. However, reductions in feed consumption, weight gain, and an increase of water consumption and subsequent urination were observed as dietary OTA levels increased. A dosage of 200 ppb OTA in feed had little effect on daily gain and feed efficiency in pigs; whereas a significant effect was found at dietary levels above 1,400 ppb. Conversely, after high OTA contaminated feed was replaced by normal feed, the pigs’ performance was restored to normal.

Marquardt and Frohlich (1992) at the University of Manitoba completed a thorough review on ochratoxin effects in poultry and other livestock. Several acute toxicity studies of ochratoxin had been conducted and discussed by the authors. Young pigs were shown to be more sensitive to the toxin than older pigs. The single dose of OTA that causes 50% mortality (LD50) for pigs was reported to range between 1 and 6 ppm (mg/kg body weight), with tissue damage observed mostly in the kidney. In comparison, the LD50 for AFL in pigs has been reported to be only 0.62 ppm (mg/kg body weight) and the liver is the primary organ affected (Leeson et al., 1995). Even though both OTA and AFL can be produced from the same genus of fungi (*Aspergillus*), but different species (*A. ochraceus* vs. *A. flavus*), it would appear that higher levels of dietary OTA are required to show detrimental effects in pigs.

Huff et al. (1988) reported results of an unpublished pig study in a paper about mycotoxin interactions in poultry and swine. In the study, pigs (unknown age or sex) were fed either 2.0 ppm AFL or 2.0 ppm OTA or a combination of the two toxins. Results show additive effects on body weight and body weight gain when both toxins were combined, but no synergistic effects (Table 9). However, unlike a difference in LD50 reported in the previous paragraph (less AFL required to cause death), in this study no differences were observed in body weight or weight gain between the OTA and AFL when fed at the same level. This would suggest that the performance effects are similar between toxins when fed above the acute toxic levels.

TABLE 9: Effects of ochratoxin, aflatoxin, and the combination on pig body weight

Aflatoxin, ppm	Ochratoxin, ppm	Body Weight, kg	Weight Gain, kg
0	0	33.7 ^a	18.2 ^a
2.0	0	29.7 ^a	13.5 ^b
0	2.0	29.9 ^a	13.8 ^b
2.0	2.0	24.6 ^b	8.8 ^c

FUSARIUM MYCOTOXINS

Fumonisin (FUM), fusaric acid (FUA), deoxynivalenol (DON; vomitoxin), and zearalenone (ZEA) are common toxins produced by *Fusarium* genus fungi. These toxins have received much attention recently due to companies promoting products that may neutralize the toxins biological effect, such as glucomannan and enzymes. The majority of the current papers published include the use of mycotoxin ameliorating compounds, but the emphasis of this paper is on the toxic effects of mycotoxins in swine.

Swamy et al. (2002) reported detrimental effects in pigs fed high levels of *Fusarium* mycotoxins. A total of 175 postweanling pigs, with an average starting weight of 10 kg, were used in the study. Pigs were fed control, toxin contaminated or toxin contaminated plus yeast cell wall (YCW) for 3 weeks. The toxin contaminated feed contained an average of 5.5 ppm DON, 400 ppb ZEA, and 26.8 ppm FUA. The control feed contained low levels of DON (0.8 ppm) and ZEA (< 100 ppb), but a high level of FUA (29.7 ppm). Pigs fed high levels of toxins showed reductions in ADG and ADI (P<0.05); however, toxins did not affect feed efficiency (Table 10). The addition of yeast cell wall further reduced (P<0.05) ADG (at 0.1 or 0.2% inclusion) and feed efficiency (0.1% inclusion only) and did not improve ADI. It is not understood why adding yeast cell wall had a negative

impact on the pigs performance. A possible explanation may be due to a shift of energy to the immune system activated by glucomannan from yeast cell wall.

TABLE 10: Effects of *Fusarium* mycotoxins in young pigs.

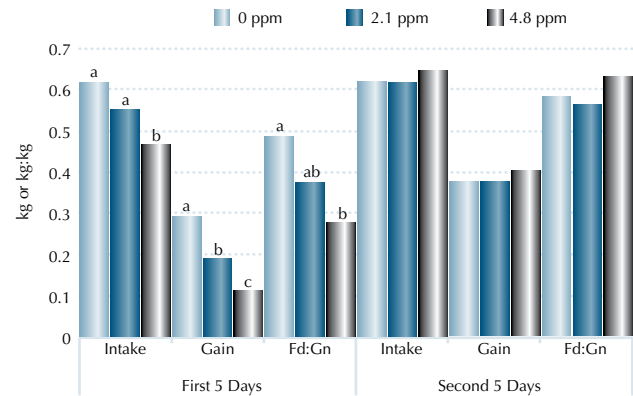
	Average Daily Gain, g	Average Daily Feed Intake, g	Gain : Feed
Toxins Free Diet	428	778	0.558
Toxins Diet*	280	524	0.533
Toxins Diet + 0.05% YCW	253	497	0.510
Toxins Diet + 0.1% YCW	216	486	0.444
Toxins Diet + 0.2% YCW	232	488	0.488

* Diets averaged 5.5 ppm DON, 400 ppb ZEA , and 26.8 ppm FUA.

In the study by Swamy et al. (2002), reduced relative liver and kidney weights, hematological parameters and serum calcium and phosphorus were also observed in the pigs consuming the toxin contaminated feed. Decreased serum calcium and phosphorus were also observed in a previous study in which pigs were fed naturally contaminated oats providing 3.5 ppm dietary DON (Bergsjö et al., 1993). However, in the same study, an increase in liver weights was documented when 3.5 ppm DON was fed. Swamy et al. (2002) reported an increase in serum IgA and IgM when pigs were fed the toxin contaminated feed, suggesting an immunostimulatory affect of the toxins. Drochner et al. (2004) also observed an increase in serum IgA when 0.6 and 1.2 ppm pure OTA was fed to female pigs for 8 weeks after weaning.

The University of Guelph (He et al., 1993) completed a study using a total of 30 pigs (3 boars and 3 gilts per treatment) weaned at day 28 and fed a starter diet for 2 weeks. The pigs were fed naturally occurring DON from moldy corn (480 ppm) or detoxified moldy corn (over 50% reduction) for 5 days followed by a non-contaminated control diet for another 5 days. The moldy corn was microbial detoxified by incubation with the contents of the large intestine of chickens. Results are shown in Figure 2. For the first 5 day period, pigs fed the diet (4.8 ppm DON) with moldy corn showed a significantly ($P < 0.05$) reduced feed consumption (-25%), weight gain (-57%), and feed efficiency (-45%) compared to the control pigs. When the moldy corn was detoxified and added to the diet (2.1 ppm DON) the pigs feed intake and feed efficiency was similar to the control pigs and weight gain was in between the controls and the pigs fed the moldy corn. After the contaminated feed was withdrawn at day 5 and replaced with a control diet for another 5 days, all pigs grew similar to pigs fed control diets (for both periods), suggesting the reduced weight gain was temporary and can be remunerated.

FIGURE 2: Performance of pigs fed moldy corn and detoxified moldy corn containing vomitoxin for 5 days (first 5 days) and then removing toxin feed for 5 days



abc Results are significantly different, within a performance parameter

In another study, naturally contaminated wheat providing low dietary levels of DON ranging from 0 to 840 ppb was fed to young male pigs for 28 days (Accensi et al., 2006). Results showed no impact ($P > 0.05$) on weanling pig feed intake, weight gain, hematological, biochemical and immune response.

Fumonisin are one of the most recently discovered economically important mycotoxins and is most toxic to horses, causing equine leukoencephalomalacia. Pigs follow horses in sensitivity to FUM, however horses are much more sensitive to acute levels. In pigs, FUM is most known for causing pulmonary edema.

Oswald et al. (2003) completed two studies supplying a 0.5 mg/kg body weight/day gavage of either purified or a crude extract of fumonisin (approximately 5 to 8 ppm dietary FUM) to young pigs for 6 days. On the last day of toxin treatment, half of the pigs in each treatment were orally inoculated with a pathogenic *Escherichia coli* and were then euthanized 24 hours later. The levels of FUM fed did not significantly ($P > 0.05$) affect performance, however there was a trend of decreased weight gain with FUM exposure. However, FUM did significantly increase *E. coli* colonization in the small and large intestine (Table 11).

TABLE 11: Effects of fumonisin (FUM) feeding on *Escherichia coli* colonization in sections of young pigs intestines

Intestinal Section Evaluated	FUM extract ¹		Pure FUM ¹	
	Control	FUM	Control	FUM
	24 hour <i>E. coli</i> colonization, log ₁₀ (CFU/g)			
Ileal	1.66	4.26	2.74	3.67
Cecum	2.99	5.85	3.72	5.07
Colon	3.32	6.03	3.73	5.62

¹0.5 mg FUM/kg body weight/day gavaged for 6 days followed by *E. coli* inoculation

Zearalenone is different from other *Fusarium* mycotoxins due

to its estrogenic effects. It has a strong impact on reproduction, including premature onset of puberty in gilts, difficulty of returning heat and rebred, abortion in sows, and decreased libido in boars.

Yang et al. (2008) conducted a trial on weaned female pigs to determine ZEA effects on performance and organ growth. Twenty pigs were used in the study and 0, 1, 2, or 3 ppm of purified ZEA was added to feed and fed for 3 weeks. The basal (control) feed was highly contaminated with up to 0.9 ppm ZEA, so the total ZEA concentration of each diet was higher (Table 12). Pigs that consumed between 11 to 51 mg of ZEA for the entire study (Table 12) showed no difference in weight gain, intake, or feed efficiency. However the uterus and ovary, kidney, and liver were significantly increased ($P < 0.05$) as ZEA consumption increased (Table 13).

TABLE 12: Effects of zearalenone feeding in young female pigs

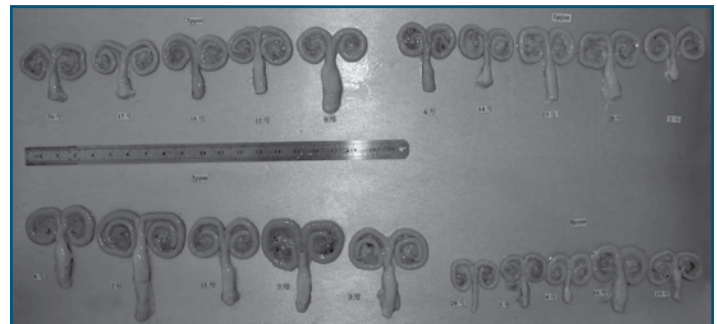
Analyzed dietary ZEA, ppm	Total ZEA Intake, mg	Average Daily Gain, g	Average Daily Feed Intake, g	Feed Conversion Rate
0.9	11.03	471	693	1.482
1.67	24.90	480	692	1.444
2.33	30.02	460	690	1.500
4.33	50.77	494	705	1.428

The heart, lung, spleen, stomach, and intestine sizes were not affected by feeding different levels of zearalenone. The dressing percent was numerically ($P > 0.05$; Table 13) decreased as dietary zearalenone increased. The slight decrease in dressing percent but increase in certain organs from ZEA exposure may provide some insight into the toxic anabolic effect due to the impact on organ muscle but not skeletal muscle. A visual comparison of uterus sizes is shown in the Figure 3.

TABLE 13: Effects of zearalenone (ZEA) on relative organ weight (percent of body weight) and dressing percentage.

Total ZEA Intake, mg	Uterus & Ovary Weight	Kidney Weight	Liver Weight	Dressing Percent
% of Body Weight				
11.03	0.0625 ^a	0.430 ^a	2.429 ^a	73.47
24.90	0.0986 ^{ab}	0.451 ^{ab}	2.535 ^{ab}	75.11
30.02	0.1214 ^b	0.502 ^b	2.759 ^b	72.46
50.77	0.2410 ^c	0.511 ^b	2.761 ^b	71.69

FIGURE 3: Uterus size of young female pigs fed different levels of zearalenone



Right Bottom – 0 ppm additional ZEA; Right Top – 1 ppm additional ZEA; Left Top – 2 ppm additional ZEA; Left Bottom – 3 ppm additional ZEA

In a reproductive study, thirty-six gilts at 91 days of gestation were fed *Fusarium* mycotoxins until farrowing, to understand the impact of DON and ZEA on reproduction (Diaz-Liano and Smith, 2006). The pregnant gilts were divided into three treatments and fed toxin free, toxin contaminated, or toxin contaminated plus 0.2% YCW for the last 3 weeks of gestation. The toxin diet contained 5.5 ppm DON and 0.3 ppm ZEA. Gilts fed toxin-contaminated diets had lower ($P < 0.05$) ADG and feed efficiency during the last 3 weeks of gestation (Table 14). There was no significant mycotoxin effect observed for feed intake, stillbirths, piglets born alive, total born per litter, number of mummies, and litter weight at birth. However, there was a trend to increase stillbirths and decrease percent born alive by feeding the toxin contaminated diet to the pregnant gilt.

TABLE 14: Effect of *Fusarium* mycotoxins in late gestation in gilts.

	Toxins Free Diet	Toxins Diet*
Feed Efficiency, kg:kg	0.5 ^a	0.2 ^b
Average Daily Gain, kg	1.1 ^a	0.6 ^b
Stillbirth, %	6.3	15.5
Born alive, %	90.5	80.7
Litter Weight, kg	11.6	12.5

* Diet contained DON 5.5 ppm and ZEA 0.3 ppm

MYCOTOXIN RESIDUES IN PORK

A USDA report circa 1970 indicated that no aflatoxin residues were found in pork and beef after feeding aflatoxin contaminated feeds; however, a significant increase of AFL M₁ (metabolite of AFB) in cow milk was observed (Table 15). Similar results were reported by Murthy et al. (1975) in a study where pigs were fed 870 µg AFL B₁/day from a complete

mixed diet (936 ppb dietary AFL B₁ supplied by peanut meal; Treatment 1). In the same study, 2 other sets of pigs were fed the protein portion (peanut and corn gluten meal) separately, containing either 3,986 ppb (Treatment 2) or 1,760 ppb (Treatment 3) AFL B₁, but the same quantity/day of the protein portion as Treatment 1. Treatments 3 and 4 were then allowed ad libitum access to the non-protein portion (AFL-free) after eating the entire AFL contaminated protein portion. The pigs consuming Treatments 2 or 3 had a calculated average daily intake of 1566 or 642 µg AFL B₁, respectively. Even though pigs on Treatment 3 consumed less AFL per day, small amounts of toxin residue were found in the kidney and liver, but not in Treatment 1. The authors suggest that because there was a lower intake of the non-protein portion by Treatment 3 pigs, there may be some protection against aflatoxicosis in the liver due to higher carbohydrate consumption in Treatment 1. A pig consuming 1566 µg AFL B₁/day had a much larger quantity of AFL B₁ in the liver, with some also found in the kidney, spleen, heart, and muscle. The AFL B₁ content of the organs in the aforementioned pig ranged from 0.1 to 6.1 ppb, with muscle at the lowest and liver the highest.

TABLE 15: Aflatoxin residues after feeding aflatoxin contaminated feeds

Species	AFL Dosage	Results
Swine	800 ppb growing-finishing	No AFL residue found in pork
Beef	1000 ppb growing-finishing	No AFL residue found in beef
Dairy	67~200 mg/week total	70~154 ppb AFL M ₁ in milk

A report distributed by FAO on various mycotoxin residues in animal protein is shown in Table 16. The sources of mycotoxin contamination were not discussed in the report. It is assumed the toxins mainly came from grains and finished feeds.

TABLE 16: Examples of food of animal origin which may be naturally contaminated with mycotoxins

Mycotoxins	Occurrence	Reported Highest Levels
Aflatoxin	Eggs	0.4 ppb
	Pig liver	0.5 ppb
Ochratoxin A	Pig liver	98 ppb
	Sausages	3.4 ppb
Zearalenone	Pig liver and muscle	10 ppb

CONCLUSIONS

In general, swine are very sensitive to AFL. Aflatoxin contamination increases mortality, reduces weight gain, feed intake, and ultimately profit of return. Tolerance of OTA in pigs appears to be higher than AFL because of the higher LD50

reported for OTA. Unlike poultry, moderate levels of *Fusarium* mycotoxins will cause subclinical effects, but not as severe as AFL and OTA on performance in pigs. The amount of DON required to cause the vomiting effect in swine is higher (20 ppm) than the level that causes complete feed refusal (12 ppm), so vomiting is less commonly found (CAST, 2003). Not all pigs will show signs of pulmonary edema from consuming FUM and the pigs that do not show signs can tolerate higher levels of FUM. After aflatoxin and ochratoxin, zearalenone may be the second most important toxin impacting swine production by impairing reproductive traits. Swine and their sensitivity to major mycotoxins are summarized in Table 17.

TABLE 17: Summary of mycotoxins sensitivities in swine

Mycotoxins	AFL	OTA	DON	FUM	FUA	ZEA
Sensitivity	++++	+++	++	++	+	++/-
Toxin Tolerance	10~100s ppb	1~5 ppm	5~10 ppm	5~10 ppm	100s ppm	Gender dependent

Mycotoxin binders are commonly used to reduce toxin adsorption in animals. A comprehensive review of various mycotoxin adsorbents has been written by Huwig et al. (2001). This review is a useful reference for nutritionists and producers who have hands-on knowledge of livestock feed production. It is well documented that toxin-contaminated feed reduces growth performance and alters the immune system in pigs. No matter how strong the nutrition and health program is, if mycotoxins can not be controlled, the greatest genetic potential will never be achieved. This certainly will reduce the amount of profit to be made.

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