Association between aflatoxicosis and salmonella: A case study

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Summary: *We investigated a* Salmonella cholerasuis *outbreak in a 500-head finishing herd that occurred coincidentally with a change to a corn source contaminated with detrimental levels of aflatoxins. The diarrhea was resolved when pigs were switched to an aflatoxin-free corn source. In this article, we review the current nutritional alternatives being researched to prevent the negative effects of aflatoxins on pigs.*

Case Study

During the autumn of 1991, pigs in a 500-head, all-in-all-out finishing unit began to experience problems with diarrhea as they reached 125 lb (57 kg). High-health pigs were purchased as 40 lb (18 kg) feeder pigs from one source, and no vaccines were used other than for *Erysipelas rhusiopathiae.* Mecadox® (50 g per ton) was administered in feed until the pigs reached 75 lb (34 kg), after which they received BMD® (30 g per ton) as a growth promotant until finished. The pigs were housed 20–25 to a pen in a totally power-ventilated building with full slats. Each pig had a total finish-out space of 7 sq ft (0.6 m^2).

Diarrhea initially was observed in about 25% of the pigs. The disease syndrome quickly became a respiratory problem with accompanying death loss. Postmortems were performed on a sample of the dead pigs and laboratory tests confirmed a diagnosis of *Salmonella cholerasuis* infection. We administered traditional and extra-label injectable, water, and feed medications based on cultures and sensitivities. Despite these treatments, however, 15% of the pigs showing clinical signs died, and the other 10% were chronic poor-doing pigs. This mortality rate was very elevated for this herd; normally, the herd's death loss ranged from 1%–2%. The surviving pigs responded to treatment much more slowly than the majority of pigs we have treated for *Salmonella* outbreaks in other herds.

There had been no known stresses on the pigs and we question why these pigs began to experience diarrhea at 125 lb (57 kg), when most pigs traditionally break at 40–75 lb (18–34 kg) or shortly after they arrive on the farm. This herd never had a case of diagnosed salmonellosis nor had the farm of origin ever experienced a salmonellosis outbreak. We tested the feed (which contained neither meat nor bone meal) for calcium, phosphorus, protein, and *Salmonella* spp. The ingredient values were normal and no *Salmonella* spp. were isolated.

The only change that occurred was that a new bin of corn had been opened 2 weeks prior to the outbreak, and all pigs were being fed from this new bin. Because the feed was manufactured on the farm, the producer made it a practice to retain samples of every batch of feed. Feed consumption records indicated that both feed consumption and average daily gain (ADG) had decreased (by 15% and 25%, respectively). With some batches of feed the pigs initially refused the feed but would eventually eat it.

We collected three 0.5-lb (227 g) samples of the current ration, one from the top of the bin and two from the auger, as well as the ration that was being fed prior to the outbreak, and submitted these in plastic whirlpacks to North Dakota State Diagnostic Laboratory, Fargo, North Dakota for analysis. The feed was analyzed for vomitoxin, aflatoxin, and T-2 mycotoxins. All samples were negative for T-2 toxin. While the old feed sample was negative for both vomitoxin and aflatoxin, a current feed sample from the newly-opened bin contained vomitoxin concentrations of 0.2 ppm and aflatoxin concentrations of 300 ppb (300 parts per 1×10^9). (These results represent only these feed samples, and toxin concentrations probably varied depending upon location in the bin.) We concluded that the aflatoxin-contaminated feed in this herd compromised the pigs' immune and metabolic systems and contributed to this outbreak of *S. cholerasuis.*

Because we believed that the aflatoxin in the feed was associated with the *S. cholerasuis* infection, we switched to a new corn source that was negative for both vomitoxin and aflatoxin. The pigs that had been nonresponsive to treatment began to show marked improvement (i.e., increased activity levels, increased feed intake, improved ADG, and improved body condition) within 1–2 weeks. No recurrence of the diarrhea or respiratory disease was noted, and cull rates returned to the pre-outbreak levels normal for this herd. This may have been due to the natural progression of the disease, but the rapid turnaround also may have been the result of the removal of the toxins and the pigs' ability to resume growth on aflatoxin-free corn.[1]

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Perspectives from the field are reviewed for their logic, relevance, and presentation.

Literature Review

Aflatoxin and vomitoxin are two common mycotoxins that cause clinical signs in swine. Mycotoxins are byproducts of the metabolic processes of molds that colonize plant parts, most commonly corn and wheat. To grow and produce their respective toxin, each mold requires that a substrate be present in the grain and that conducive environmental conditions prevail. Aflatoxin, the most common toxin found in corn, is produced by the fungus *Aspergillus flavus*, a common soil fungus. The fungus can only colonize kernels of corn and small grains that have been damaged. It grows best and produces the most toxin at 18% moisture and at temperatures of approximately 79°F (26°C).[2]

Table 1

Effects of graded levels of aflatoxin on finishing swine

	Dietary aflatoxin levels ppb			
Performance value	20	385	750	1480
Average daily gain (kg)	0.77	0.67	0.57	0.41
Average daily feed intake (kg)	2.87	2.53	2.15	1.61
Feed : gain ratio	3.74	3.78	3.71	3.97

Aflatoxins primarily target the liver,[1,2] and when present at concentrations of 1000 ppb can cause signs consistent with liver damage. Hepatitis, necrosis of liver cells, prolonged clotting time, and death by severe hemorrhage can be observed. Subacute aflatoxin poisoning is manifested by hepatic lipidosis, portal fibrosis, and proliferation of bile duct epithelium, depressed protein formation, and hepatic tumors.[2] Signs of chronic aflatoxin poisoning (observable with concentrations of 300–410 ppb) include poor growth rates, poor feed conversion, decreased feed intake, increased mortality, impaired liver function and necrosis, and altered immune responses.[5]

Investigators who conducted a vaccine response trial to measure the response of pigs fed aflatoxin to *Erysipelas rhusiopathiae* (as measured by ELISA) found that although all pigs mounted an immune response (with no significant difference among controls and principals) and that there was no difference in the proliferative responses of lymphocytes to mitogens, pigs fed aflatoxin had significantly lower complement fixation titers ($P < .05$) than pigs fed an aflatoxin-free diet. Complement is necessary to enhance immune responses. There was also an increase in serum IgG and IgM in the aflatoxin-positive groups as opposed to the negative controls. Livers of aflatoxin-positive pigs were enlarged and toxic histological damage was apparent on the parenchyma of the liver. The reduction in complement could be explained by the extensive liver damage, and the increases of IgG and IgM may be the result of the impaired catabolic activity of the liver.

Yang[3] suggested that suppression of peripheral blood, splenic, and thymic lymphocytes would occur only in cases of acute aflatoxicosis, and that death occurs before a decrease in weight loss would occur. These investigations showed that aflatoxins impair elements of the host's defense mechanism, such as complement production, and that the resulting suppression of the immune system may explain why pigs fed aflatoxin have enhanced susceptiblity to *Serpulina hyodysenteriae* and *S. cholerasuis*.[4]

Aflatoxins can cause other problems that can contribute to a concurrent disease problem. Southern and Clawson[1] found that ADG and average daily feed consumption were linearly reduced ($P < .05$) as the concentration of dietary aflatoxin was increased at or above 385 ppb. Feed efficiency was not significantly influenced by aflatoxin concentrations of 385 ppb and 750 ppb but was depressed ($P < .05$) when 1480 ppb was fed (Table 1). When half of these pigs at the end of a 66-day feeding trial were fed aflatoxin-free diets for 7 days, they consumed more feed (2.18 versus 2.05 kg per day), gained at a faster rate, (0.59 versus 0.30 kg per day) ($P < .01$), and were more efficient (3.66 versus 6.91 kg feed per kg gain) than those pigs that remained on their respective aflatoxin-containing diets. The pigs' total serum proteins, albumin, and IgG were unaffected ($P > .10$) by aflatoxin consumption. The IgM fraction was increased ($P < .05$) in pigs being fed 750 and 1480 ppb of aflatoxin. As the concentration of toxin was increased, there was an increase in liver weight as a percentage of the final pig weight, indicating that the liver had been damaged ($P < .05$). These results agree with work performed by Monegue (cited in Southern and Clawson).[1]

In another investigation,[4] pigs fed aflatoxin gained significantly less weight than did controls ($P < .01$). In this experiment, there were two aflatoxin-positive groups:

- one group fed a diet containing aflatoxin concentrations of 300 ppb; and
- one group fed a diet containing aflatoxin concentrations of 500 ppb.

Although there were initially no differences in feed consumption, as the experiment progressed the feed consumption of the aflatoxin-positive groups was lower than the controls. Beyond the fifth week the decreases in feed consumption were dose dependent. The group fed 500 ppb had the poorest feed conversion, whereas the feed efficiency of the group fed 300 ppb was intermediate when compared to the controls.

The negative effects of aflatoxin-contaminated feed on growth parameters can be partially counteracted by feeding increased concentrations of protein, dietary fat, lysine, or methionine.[1] Feed intake, average daily gain, and feed : gain ratios remain at acceptable levels when aflatoxin-contaminated feed is supplemented with crude protein (Table 2). A lack of dietary protein may interfere with the ability of the liver to synthesize enzymes important for metabolizing and

Table 2

Performance comparison of diet modifications in two trials

Diet	ADG kg	FI kg/d	F : G
Trial 1			
CP 18%	0.37	0.66	1.78
CP 18% + 182 ppb AFB	0.30	0.57	1.88
Trial 2			
CP 18% + 186 ppb AFB	0.44	0.93	2.00
CP 18% + 186 ppb AFB + 0.25% lysine	0.51	1.00	1.95

detoxifying the aflatoxin molecules, exposing it longer to the toxic effects of aflatoxin. Since aflatoxins are known to interfere with the digestion of dietary fats and fat-soluble vitamins, adding more fat to an aflatoxin-contaminated ration may obviate the need for supplementing essential fatty acids. This is particularly important, since aflatoxin B_1 alters the integrity of cell membranes and its effect on lipid metabolism is the primary cause of lesions during aflatoxicosis.[5]

An additional control method is the use of aluminosilicates and other clay binders (at a 1% concentration). These have been reported to bind aflatoxins, and offset the negative effects of aflatoxin contamination in feed. Schell, et al., reported that adding clays or other binders to aflatoxin-contaminated swine diets could offset 85% of its negative effect on growth parameters, and that these clay compounds do not adversely affect normal pigs. [6,7]

Implications

- Decreased feed consumption, poor feed conversion, and impaired growth rate were associated with aflatoxin consumption.
- As with any chronic nonresponsive disease, it is important to include mycotoxins in the differential diagnosis and laboratory workup.
- Using production records can facilitate a tentative diagnosis of aflatoxicosis, because ADG and feed intake are more sensitive parameters than feed efficiency in cases of aflatoxicosis.
- Feed should be stored in a cool, dry place and handled carefully to avoid providing an environment conducive to the molds that secrete mycotoxins.
- If aflatoxin contamination has already occurred, adding protein, lysine, or fat to the rations can partially or completely offset its negative effects on growth parameters. Adding clay or aluminosilicates to the diet can help to bind the toxins. Blending the contaminated corn with aflatoxin-free corn can also help offset depressed growth (Table 3). These measures, however, must be cost-effective.
- Aflatoxins can damage the liver, cause death loss, and can alter the immune responses of swine. Pigs whose immune systems have been compromised by the ingestion of aflatoxins are more susceptible to infection from such pathogens as *S. hyodysenteriae* and *S. cholerasuis,* even in herds with no previous history of such problems.

Table 3

Recommended maximum feeding levels for aflatoxin feed

Pig	aflatoxin level (ppb)
Breeding herd	100
Young pigs	20
Growing pigs	20
Finishing	200

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