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Effects of Mycotoxins and Mycotoxin Sequestering Agents on Mineral Nutrition

Key Findings

- Feeding mycotoxin contaminated feedstuffs will negatively impact animal performance, especially when animals are either under stress and/or immuno-compromised.
- Research has demonstrated the important role of trace minerals in optimizing animal performance.
- The potential exists for mycotoxin sequestering agents to negatively affect trace mineral nutrition.
- Based on limited data, some classes of mycotoxin sequestering agents may render some minerals and vitamins unavailable for absorption and metabolism.
- Providing a highly bioavailable source of trace minerals, such as Zinpro Performance Minerals®, is justified when dealing with mycotoxin contaminated feeds.

Introduction

Annual costs of mycotoxin mitigation and livestock losses have been estimated at \$466 million and \$6 million, respectively (CAST, 2003). Mycotoxins routinely occur in common feedstuffs such as corn, corn silage, small grains and small grain silage, especially when growing conditions are sub-optimal. It should be noted that mycotoxins may or may not be present in moldy feedstuffs. In addition, mycotoxins may be present in feeds that appear free of mold which makes mycotoxins a challenge for producers and nutritionists alike. The objectives of

this paper are to provide a brief review of the effects of mycotoxins on dairy cattle, present common mycotoxins and discuss the effects of mycotoxin sequestering agents on mineral nutrition.

General Effects of Mycotoxins

Large doses of mycotoxins may be a primary cause of production problems in a dairy herd. However, long term exposure to mycotoxins often contributes to chronic problems which result in higher incidence of disease, poor reproductive performance and/or lower milk yields. According to Whitlow and Hagler (2008), mycotoxins exert their effects through several means including 1) decreased feed intake, 2) reduced nutrient absorption and impaired metabolism, 3) altered function of the endocrine and exocrine systems, 4) suppressed immune function, 5) altered rumen microbial growth and 6) changes in white blood cell and neutrophil counts. The effects of mycotoxins are often compounded by the presence of other metabolic and environmental stressors which can lead to difficulties in diagnosis. This is especially apparent in immuno-suppressed animals such as transition dairy cows, receiving beef cattle and young animals with developing immune systems.

Degradation of mycotoxins by rumen microbes can provide some protection against acute toxicity from mycotoxins in feedstuffs (Jouany and Diaz, 2005). However, rumen degradation may lead to the production of toxic metabolites, resulting in cows exhibiting a wide range in severity of symptoms. In general, the



first signs of mycotoxicosis are observed in highly stressed cattle such as immuno-compromised transition dairy cows. In addition to the previously mentioned signs of mycotoxin exposure, cows may exhibit intermittent diarrhea (sometimes with bloody or dark manure), unthriftiness, rough hair coat and reduced reproductive performance (irregular estrus cycles, decreased conception rates, embryonic mortalities and pregnant cows displaying estrus). Additionally, the incidence of transition cow disorders is likely to increase and cows may not respond well to veterinary therapy (Whitlow and Hagler, 2008).

Common Mycotoxins

Following is an overview of six common mycotoxins, including a summary of common effects (Table 1, page 2). For information regarding the occurrence of common mycotoxins in shelled corn, refer to Table 2 (page 2).

Aflatoxins: Aflatoxins are extremely toxic, mutagenic and carcinogenic compounds produced primarily by the fungus *Aspergillus flavus* and *Aspergillus parasiticus*. Aflatoxins are most abundant in hot, humid geographic areas. Aflatoxin production can also surface in drought stressed or insect damaged corn and

small grains when breakage is increased. The FDA limits aflatoxin to <20 ppb in lactating dairy cattle feeds and to 0.5 ppb in milk. Generally speaking, milk aflatoxin concentrations equal 1.7% (range of 0.8 to 2.0%) of the aflatoxin concentration in the total ration dry matter (Whitlow and Hagler, 2008).

Deoxynivalenol (DON) or Vomitoxin: Deoxynivalenol is produced by *Fusarium* mold and can be commonly detected in feeds. The effect of DON on dairy cattle performance is not well established. Diaz *et al.* (2001) found feeding a mycotoxin sequestering agent significantly increased milk yield by 3 lb/day in dairy cattle fed diets containing 2.5 ppm of DON. Beef cattle and sheep appear to be more tolerant of DON than dairy cattle or swine and poultry. The FDA has established 5 ppm of vomitoxin in grain and grain byproducts as the advisory level for dairy cattle and recommends that commodities containing this level not exceed 40% of the ration dry matter.

T-2 Toxin: Although a potent *Fusarium* product, T-2 toxin is not commonly found in feedstuffs. T-2 toxin is associated with gastroenteritis, intestinal hemorrhages and death. Whitlow and Hagler (2008) reported T-2 levels in excess of 500 ppb to be intolerable and associated with reduced feed intake, bloody manure, enteritis, abomasal and ruminal ulcers and death. Due to immune incompetence, transition dairy cows seem to be the most sensitive group to this toxin. There are currently no FDA guidelines published for T-2 in feedstuffs.

Zearalenone: Zearalenone is produced by *F. graminearium* and several other species of *Fusarium*. Zearalenone resembles the structure of estrogen and can therefore elicit estrogenic responses in animals such as vaginal secretions, reproductive tract infections, poor reproductive performance, abortions and mammary gland enlargement of virgin heifers. Grazing dairy herds exposed to zearalenone had lower fertility with higher levels of blood and urinary zearalenone metabolites (Jouany and Diaz, 2005).

Table 1. Effects of Common Mycotoxins^a

| Mycotoxin | Effect |
|--|--|
| Aflatoxin | Acute: <ul style="list-style-type: none"> • inappetance, lethargy, ataxia, rough hair coat, pale skin, enlarged fatty livers Chronic: <ul style="list-style-type: none"> • decreased feed efficiency, decreased milk production, jaundice, decreased appetite, decreased resistance to disease, decreased vaccine-induced immunity |
| Deoxynivalenol (DON) or Vomitoxin | Altered rumen fermentation, reduced flow of utilizable protein to the duodenum |
| T-2 | Poor transition cow performance, low feed intake, low peak milk yields, sharp increase in incidence of disease, culling and death, absence of estrus cycles |
| Zearalenone | Increased vaginal secretions, reproductive tract infections, poor reproductive performance, abortions, mammary gland enlargement of virgin heifers |
| Fumonisin | Liver lesions, lymphocyte blastogenesis, elevated liver enzymes, decreased feed intake and milk yields |

^a Whitlow, L.W., and W.M. Hagler, Jr. 2008. Mold and mycotoxin issues in dairy cattle. Effects, prevention and treatment. Pp 80-89. Proc. Southeast Dairy Herd Mgmt. Conf., Macon, GA.
Ref. Slide # CG-320

Table 2. Occurrence of Common Mycotoxins in Shelled Corn^a

| Mycotoxin | Total Samples | Number Positive | % Positive | % Exceeding FDA Limit* or Advisory Level [†] |
|--|---------------|-----------------|------------|---|
| Aflatoxin | 516 | 52 | 10 | 0.4% > 20 ppb* |
| Deoxynivalenol (DON) or Vomitoxin | 1005 | 832 | 83 | 4.8% > 5 ppm ^{†b} |
| T-2 | 387 | 139 | 36 | NA |
| Zearalenone | 845 | 417 | 49 | NA |

^a Dairyland Laboratories, Inc., Arcadia, WI. Samples from Oct. 1, 2009 through January 18, 2010.
^b FDA advisory levels for deoxynivalenol (DON) or vomitoxin are 5 ppm in grain and grain byproducts destined for swine and other species (except cattle and chickens); not to exceed 20 percent of the diet for swine, and not to exceed 40 percent for other animal species; 10 ppm in grain and grain byproducts for ruminating beef and feedlot cattle older than 4 months and for chickens; not to exceed 50 percent of the diet.
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Whitlow and Hagler (2008) suggest that zearalenone levels as low as 300 ppb may be of concern, however, the FDA has no published guidelines for zearalenone levels.

Fumonisin: Fumonisin is a group of mycotoxins produced by the mold *Fusarium verticillioides*. Little information exists on the effects of fumonisin on ruminants. Research has found fumonisin unmetabolized in the feces of beef cattle fed corn contaminated with fumonisin.

However, the carryover of fumonisin into milk appears to be quite low. Whitlow and Hagler (2008) reported dairy cattle fed 100 ppm of fumonisin for 7 days prepartum through 70 days postpartum to produce 13 lb/day less milk due primarily to a decrease in feed intake. The FDA has established maximum levels of 10 ppm and 5 ppm for corn/corn byproducts and finished feeds, respectively. Feedstuffs contaminated with these levels should be limited to 50% of the diet, or less.

Table 3. Effects of Mycotoxin Sequestering Agents on Mineral Nutrition^a

| Sequesterant | Specie | Feeding Rate, % | Mineral Source | Mineral(s) Affected |
|-------------------------------|---------|-----------------|----------------|--|
| HSCAS^b | | | | |
| Chung <i>et al.</i> , 1990 | Poultry | 0.5 or 1.0 | Oxides | Decreased tibia Zn |
| Chestnut <i>et al.</i> , 1992 | Sheep | 2% | Oxides | Decreased Zn, Mn, Mg absorption |
| Silicates | | | | |
| Schell <i>et al.</i> , 1993 | Swine | 1% | Inorganics | Decreased Zn, Ca, Na absorption & retention |
| Ivan <i>et al.</i> , 1992 | Sheep | 0.5% | None | Decreased ruminal Zn, Cu and Mg solubility; Decreased Cu in liver and plasma |

^a Chestnut, A.B., P.D. Anderson, M.A. Cochran, H.A. Fribourg, and K.D. Gwinn. 1992. *J. Anim. Sci.* 70:2838-2846; Chung, T.K., J.W. Erdman, Jr., and D.H. Baker. 1990. *Poultry Sci.* 69:1364-1370; Schell, T.C., M.D. Lindemann, E.T. Kornegay, and D.J. Blodgett. 1993. *J. Anim. Sci.* 71:1209-1218

^b Hydrated sodium calcium aluminosilicate
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Ochratoxin A (OTA): Ochratoxin A is produced by a species of *Penicillium* and *Aspergillus*. Ochratoxin A is the primary causative agent of kidney disease in swine primarily through an inhibition in protein synthesis. Although OTA is rapidly degraded in the rumen, Whitlow and Hagler (2008) indicated chronic exposure and acute toxicities have been known to occur in cattle, including death and abortions from cattle consuming moldy hay containing OTA. Recently, Ranaldi *et al.* (2009) found that adequate zinc stores are required within intestinal cells to properly respond to an OTA challenge in humans. There are currently no FDA guidelines for OTA.

Effects of Mycotoxin Sequestering Agents on Mineral Nutrition

Research data on the impact of mycotoxin sequestering agents on trace minerals is limited and virtually no published data are available in dairy cattle at the time of this publication (Table 3). Therefore, a brief discussion on a limited number of classes of mycotoxin sequestering agents on mineral metabolism is provided.

Hydrated sodium calcium aluminosilicate (HSCAS): Hydrated sodium calcium aluminosilicate from natural zeolite functions to sequester positively charged or cationic compounds, mainly aflatoxins. Results from Chung *et al.* (1990) indicated young broiler chicks fed 0.5% or 1.0% dietary HSCAS in mycotoxin-free diets supplemented with inorganic (oxides) minerals does not impair manganese (Mn), vitamin A or riboflavin utilization but led to a decrease in tibia zinc (Zn). Chestnut *et al.* (1992) found HSCAS (fed at 2% DM basis) to impair Zn absorption in sheep while also reducing the absorption of magnesium (Mg) and Mn. In both of these studies supplemental zinc was provided as zinc oxide. Zinc plays a critical role in carbohydrate and energy metabolism, protein synthesis, epithelial tissue integrity, cell repair and division and vitamin A and E absorption and transport, which all affect immune function. Therefore, based on these studies, livestock fed diets containing HSCAS may benefit from supplementation with zinc as zinc amino acid complexes.

Silicate minerals: Silicate minerals are the largest class of mycotoxin sequestering agents and consists of phyllosilicate (mineral clays such as montmorillonite/

bentonite) and tectosilicate (zeolites) subclasses. The bentonites largely are used as a result of having a high degree of ion exchange capabilities and are primarily effective against aflatoxins (Diaz and Smith, 2005). Schell *et al.* (1993) used weanling and growing pigs to investigate the effect of sodium bentonite clay (1%) on mineral metabolism in diets with or without aflatoxin. Feeding aflatoxin contaminated feed increased phosphorus (P), sodium (Na) and Zn absorption and retention suggesting a possible increased metabolic demand for these minerals when aflatoxin is present. In addition, feeding sodium bentonite decreased Mg absorption regardless of the presence of aflatoxin. The addition of bentonite clay also decreased calcium (Ca) and Na absorption and retention in aflatoxin contaminated diets and decreased Na absorption when the feed was free of aflatoxin. Similar to the effects of HSCAS, Zn absorption and retention was decreased in diets supplemented with sodium bentonite. The effects of feeding sodium bentonite clay on iron (Fe) was confounded in this study by the increase in dietary Fe (446 ppm vs. 292 ppm) from the addition of the clay.

In addition to negatively affecting Zn, bentonite has also been shown to decrease copper (Cu) bioavailability in sheep (Ivan *et al.*, 1992) fed no supplemental trace minerals. Although the cation composition was not disclosed, bentonite was fed at 0.5% of the diet (as fed basis). In this study, bentonite decreased the ruminal solubility of Zn, Cu and Mg and led to significant decreases in Cu in both plasma (0.75 vs. 0.71 µg/ml) and liver (602 vs. 504 µg/g DM).

In conclusion, it appears utilizing silicate minerals as mycotoxin sequestering agents could lead to decreases in both Zn and Cu status. The interaction between complexed trace minerals and mycotoxin sequestering agents has not been researched. However, providing a portion of supplemental Zn and Cu as amino acid complexes may be warranted to improve the likelihood of maintaining optimal trace mineral status when diets contain silicate-based mycotoxin sequestering agents.

Others: Other mycotoxin sequestering agents include activated charcoal, cholestyramine, chlorophyllin and yeast cell wall-derived agents. Although these may be beneficial at reducing the impact of mycotoxins in humans, aquatic and other animal species, there is currently no data available on their interaction with mineral nutrition.

Conclusions

Timely harvest and proper storage of feedstuffs can significantly decrease the likelihood of mycotoxin contamination. However, poor growing and harvesting conditions often result from unpredicted changes in weather which may lead to the development of mycotoxins. Regardless of the type of mycotoxin(s) present, consider the following: 1) eliminate or reduce the feeding rate of the contaminated feed, 2) provide additional dietary protein, energy, fiber/buffer/additives which enhance rumen function and 3) incorporate a research proven mycotoxin sequestering agent to aid in decontaminating the feed (Table 4). One should note, based on limited data, some classes of mycotoxin sequestering agents may render some minerals and vitamins unavailable for absorption and metabolism. Therefore, providing a highly bioavailable source of trace minerals such as Zinpro Performance Minerals may be justified.

In summary, two important points are noteworthy regarding mycotoxins and the use of mycotoxin sequestering agents. First of all, feeding mycotoxin contaminated feedstuffs will negatively impact performance, especially animals under stress and/or immuno-compromised such as transition dairy cows. Research has demonstrated the important role of trace minerals in optimizing performance in these animals. Secondly, it is clear that there is potential for mycotoxin sequestering agents to negatively affect trace mineral nutrition. Therefore, including Zinpro Performance Minerals as a portion of your trace mineral program is justified when dealing with mycotoxin contaminated feeds.

Table 4. Potential solutions for dealing with mycotoxin-contaminated feeds

- **Eliminate or reduce the feeding rate of the contaminated feed**
- **Provide additional dietary protein, energy, fiber/buffer/additives which enhance rumen function**
- **Incorporate a research-proven mycotoxin sequestering agent to aid in decontaminating the feed**

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