Mold and Mycotoxin Issues in Dairy Cattle: Effects, Prevention and Treatment

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Take Home Messages

- Molds occur commonly in feedstuffs and can infect cattle causing a mycosis.
- Molds also produce poisons called mycotoxins that can affect cattle consuming contaminated feeds.
- The mycotoxins of greatest concern to dairy cattle include: ergots produced in small grains, fescue and other grass; aflatoxin, which is generally produced by *Aspergillus* mold; deoxynivalenol, zearalenone, T-2 toxin, and fumonisin, which are produced by *Fusarium* molds; and ochratoxin, PR toxin, mycophenolic acid and roquefortine C produced by *Penicillium* molds.
- Hundreds of mycotoxins are known, causing an array of symptoms including reduced feed consumption, reduced nutrient utilization, altered rumen fermentation, suppressed immunity, low production, poor reproduction, irritated tissues and death.
- Diagnosis is difficult because many mycotoxins exist, sampling is inaccurate, analyses are limited, and symptoms are varied and nonspecific.
- Diet management may reduce the impact of mycotoxins. Diets should be formulated and fed to reduce nutritional stress (such as transitional diets), and to supply sufficient protein, energy, fiber, antioxidant nutrients, and buffers.
- Experimentally, mycotoxin binders have been effective at partially reducing the effects of some mycotoxins, but no products are currently approved by the Food and Drug Administration.

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■ Mycotoxins

Toxigenic molds (fungi) produce mycotoxins that cause an undesirable effect (mycotoxicosis) when animals are exposed. There are hundreds of chemically diverse mycotoxins known, but few have been extensively researched and even fewer have good methods of analysis available. The primary classes of mycotoxins are aflatoxins, zearalenone, trichothecenes, fumonisins, ochratoxin A and the ergot alkaloids.

■ Molds Can Cause Disease

A mold (fungal) infection resulting in disease is referred to as a mycosis. Fungal pathogens have been associated with bovine diseases, including mycotic hemorrhagic bowel syndrome (HBS) in dairy cattle. Reducing animal exposure to moldy feeds and mycotoxins can help control mycoses such as HBS. A commercial feed additive with anti-fungal and adsorbent properties appears to reduce HBS.

■ Mold Growth, Mycotoxin Formation

Many species of fungi produce mycotoxins in feedstuffs. Mold growth and mycotoxin production are related to plant stress caused by weather extremes, to insect damage, to inadequate storage practices and to faulty feeding conditions. Mold contamination in the field is related to environmental conditions such as heat, water and insect damage, which cause plant stress and result in large variations in mycotoxin occurrence. In storage, similar environmental stresses are the major factors influencing mycotoxin contamination of feedstuffs. Molds can grow on feeds containing more than 12-15% moisture. In wet feeds such as silage, higher moisture levels allow mold growth if oxygen is available. Because most molds are aerobic, high moisture storage (silage and high moisture grain) that excludes air can help prevent mold growth.

Aspergillus species normally grow at lower water activities and at higher temperatures and thus aflatoxin in corn is more likely in heat and drought stress associated with warmer climates.

The *Fusarium* species are important plant pathogens that can proliferate pre-harvest, but continue to grow post-harvest. In corn, *Fusarium* molds are associated with ear rot and stalk rot, and in small grains, they are associated with diseases such as head blight (scab). In wheat, *Fusarium* is associated with excessive moisture at flowering and early grain-fill stages. In corn, *Fusarium graminearum* is referred to as a red ear rot and is more likely in a

cool, wet growing season and with insect damage. *Fusarium* ear rots that produce fumonisins are referred to as pink ear rots and vary in their environmental requirements. They are more likely with dry conditions occurring in mid-season followed by wet weather.

The individual *Penicillium* species have variable requirements for temperature and moisture, but are more likely to grow under post-harvest environment, in cooler climates, in wet conditions and at a lower pH. *Penicillium* molds are a major contaminant of silage, probably because they are acid tolerant.

■ Mycotoxin Occurrence

Worldwide, it is estimated that 25% of crops are affected by mycotoxins annually, which would extrapolate to billions of dollars. Annual economic cost of mycotoxins to the U.S. agricultural economy is estimated to average $1.4 billion. Economic losses are due to effects on livestock productivity, losses in crops and the costs and effects of regulatory programs directed toward mycotoxins.

Mycotoxins routinely occur in feeds including corn silage and corn grain at unsuitable concentrations as shown in table 1.

Table 1. Occurrence of five mycotoxins in corn silage, corn grain and in all feed samples submitted for analysis by producers in North Carolina over a nine year period.

Mycotoxin	Feedstuff	Number of samples	Positive above limits, %	Mean	Standard deviation
Aflatoxin, >10 ppb	Corn Silage	461	8	28	19
	Corn Grain	231	9	170	606
	All Feeds	1617	7	91	320
Deoxynivalenol, > 50 ppb	Corn Silage	778	66	1991	2878
	Corn Grain	362	70	1504	2550
	All Feeds	2472	58	1739	10880
Zearalenone, > 70 ppb	Corn Silage	487	30	525	799
	Corn Grain	219	11	206	175
	All Feeds	1769	18	445	669
T-2 toxin, > 50 ppb	Corn Silage	717	7	569	830
	Corn Grain	353	6	569	690
	All Feeds	2243	7	482	898
Fumonisin, > 1 ppm	Corn Silage	63	37	--	--
	Corn Grain	37	60	--	--
	All Feeds	283	28	--	--

■ Mycotoxin Effects

Mycotoxins, in large doses, can be the primary agent causing acute health or production problems in a dairy herd. But more likely, mycotoxins are a factor contributing to chronic problems including a higher incidence of disease, poor reproductive performance, or suboptimal milk production. Ruminal degradation of mycotoxins helps to protect the cow against acute mycotoxin toxicity, but also masks effects in dairy cows, which may contribute to chronic problems associated with long term consumption of low levels of mycotoxins. As production stresses have increased in dairy cows, management details have become more closely scrutinized and the effects of mycotoxins have been more widely recognized.

Mycotoxins exert their effects through several means:

- reduced intake or feed refusal;
- reduced nutrient absorption and impaired metabolism;
- altered endocrine and exocrine systems;
- suppressed immune function;
- altered rumen microbial growth.
- cellular death

Because of limited diagnostic tools, the determination of a mycotoxin problem becomes a process of elimination and association. Certain basic observations can be helpful.

- Mycotoxins should be considered as a possible primary factor resulting in production losses and increased incidence of disease.
- Documented symptoms in ruminants or other species can be used as a general guide to symptoms observed in the field.
- Systemic effects as well as specific damage to target tissues can be used as a guide to possible causes.
- Post mortem examinations may indicate no more than gut irritation, edema, or generalized tissue inflammation.
- Because of the immune suppressing effects of mycotoxins, increased incidence of disease or atypical diseases may be observed.
- Responses to added dietary adsorbents or dilution of the contaminated feed may help in diagnosis.
- Feed analyses should be performed, but lack of accurate sampling is a major problem

Symptoms can be nonspecific and wide-ranging and may result from a progression of effects or from opportunistic diseases. Symptoms vary depending on the mycotoxins involved and their interactions with other stress factors. In many cases, mycotoxins simply increase the severity of existing problems, allowing more disease. Stressed cows, such as those in early lactation, are most affected, perhaps because their immune systems are already suppressed. Symptoms can include: reduced production; reduced feed consumption; intermittent diarrhea (sometimes with bloody or dark manure); reduced feed intake; unthriftiness; rough hair coat; lameness; and reduced reproductive performance including irregular estrous cycles, embryonic mortalities, pregnant cows showing estrus, and decreased conception rates. Rates of disease, culling and death generally increase over

time.

■ Safe levels of mycotoxins

Some of the same factors that make diagnosis difficult also contribute to the difficulty of establishing levels of safety. These include lack of research, sensitivity differences across animal species, imprecision in sampling and analysis, the large number of potentially different mycotoxins, and interactions with stress factors or other mycotoxins. In the U.S., the **FDA** (Food and Drug Administration-Center for Veterinary Medicine) has established action, guidance and advisory levels for some mycotoxins. Feeds with mycotoxin(s) that exceed the action, advisory or guidance levels may be considered by FDA as adulterated. Grains with mycotoxin(s) exceeding the highest action, advisory or guidance levels may be considered as unfit for use in animal feed (Henry, 2006).

Standards for acceptable concentrations of mycotoxins should be conservatively low due to non-uniform distribution, uncertainties in sampling and analysis, the potential for multiple sources of mycotoxins in the diet, and interacting factors affecting toxicity.

■ Toxicity of Individual Mycotoxins

Aflatoxin

Aflatoxins are extremely toxic, mutagenic, and carcinogenic compounds produced by *Aspergillus flavus* and *A. parasiticus*. Aflatoxin B1 is excreted in milk in the form of aflatoxin M1. The FDA limits aflatoxin to no more than 20 ppb in lactating dairy feeds and to 0.5 ppb in milk. A thumb rule is that milk aflatoxin concentrations equal about 1.7% (range from 0.8 to 2.0%) of the aflatoxin concentration in the total ration dry matter. Cows consuming diets containing 30 ppb aflatoxin can produce milk containing aflatoxin residues above the FDA action level of 0.5 ppb. Aflatoxin appears in the milk rapidly and clears within three to four days.

Symptoms of acute aflatoxicosis in mammals include: inappetance, lethargy, ataxia, rough hair coat, and pale, enlarged fatty livers. Symptoms of chronic aflatoxin exposure include reduced feed efficiency and milk production, jaundice, and decreased appetite. Aflatoxin lowers resistance to diseases and interferes with vaccine-induced immunity. FDA action levels for aflatoxin are presented in table 2 (Henry, 2006).

Table 2. Action levels for total aflatoxins in livestock feed, (Henry, 2006)

Class of Animal	Feed	Aflatoxin Level
Finishing beef cattle	Corn and peanut products	300 ppb
Beef cattle, swine or poultry	Cottonseed meal	300 ppb
Finishing swine over 100 lb.	Corn and peanut products	200 ppb
Breeding cattle, breeding swine and mature poultry	Corn and peanut products	100 ppb
Immature animals	Animal feeds and ingredients, excluding cottonseed meal	20 ppb
Dairy animals, animals not listed above, or unknown use	Animal feeds and ingredients	20 ppb

Deoxynivalenol (DON) or Vomitoxin

Deoxynivalenol is a *Fusarium* produced mycotoxin commonly detected in feed. It is sometimes called vomitoxin because it was associated with vomiting in swine. Surveys have shown DON to be associated with swine disorders including feed refusals, diarrhea, emesis, reproductive failure, and deaths. The impact of DON on dairy cattle is not established, but clinical data show an association between DON and poor performance in dairy herds. DON has been associated with altered rumen fermentation and reduced flow of utilizable protein to the duodenum. Beef cattle and sheep seem to be more tolerant of DON, having consumed levels of 21 ppm of dietary DON without obvious effects. Perhaps DON serves as a marker, indicating that feed was exposed to a situation conducive for mold growth and possible formation of several mycotoxins. Much of the consumed DON is metabolized in the rumen, but a derivative (DOM1) can be found at low concentrations in milk. FDA has set advisory levels for DON as shown in table 3 (Henry, 2006).

Table 3. Advisory levels for deoxynivalenol (vomitoxin) in livestock feed (Henry, 2006)

Class of Animal	Feed Ingredients & Portion of Diet	DON Levels in Grains & Grain By-products and (Finished Feed)	
Ruminating beef and feedlot cattle older than 4 months	Grain and grain by-products not to exceed 50% of the diet	10 ppm	(5 ppm)
Chickens	Grain and grain by-products not to exceed 50% of the diet	10 ppm	(5 ppm)
Swine	Grain and grain by-products not to exceed 20% of the diet	5 ppm	(1 ppm)
All other animals	Grain and grain by-products not to exceed 40% of the diet	5 ppm	(2 ppm)

Toxin (T-2)

T-2 toxin is a very potent *Fusarium* produced mycotoxin that occurs in a low proportion of feed samples (<10%). T-2 has been associated with gastroenteritis, intestinal hemorrhages and death. Dietary T-2 of >500 ppb are considered intolerable and associated with poor feed consumption, bloody feces, enteritis, abomasal and ruminal ulcers, and death. T-2 seems to be most severe on transitioning cows resulting in poor adjustment in early lactation, low feed intake and low peak milk production. Incidence of disease, culling and death are sharply increased. Absence of estrous cycles have been noted in dairy cows exposed to T-2, but probably results from the gross toxicity of T-2 and not to estrogenic effects. The FDA has no guidelines for T-2 in feed.

Zearalenone (ZEA)

Zearalenone is a *Fusarium* produced mycotoxin associated with ear and stalk rots in corn and with scab in wheat. Zearalenone has a chemical structure similar to estrogen and can produce an estrogenic response in animals. Much of the ZEA consumed is metabolized in the rumen, but the derivatives retain estrogenic activity. Case reports have related ZEA to estrogenic responses in ruminants including increased vaginal secretions, reproductive tract infections, poor reproductive performance, abortions, and mammary

gland enlargement of virgin heifers. Controlled studies with ZEA at high levels have failed to reproduce the degree of toxicity that has been associated with ZEA contaminated feeds in field observations. This may be due to inability to adequately analyze for conjugated forms of ZEA occurring naturally. Field observations suggest that ZEA may be of concern at levels as low as 300 ppb in the diet. There appears to be a minimum toxic dosage of ZEA that reduces fertility with higher doses resulting in minimal additional effect. Zearalenone has been measured experimentally in blood and urine. The FDA has established no guidelines for ZEA in feed (Henry, 2006).

Fumonisins (FB)

Fumonisins produced by *F. verticillioides* were first isolated in 1988. They cause leukoencephalomalacia in horses, pulmonary edema in swine, and hepatotoxicity in rats. Fumonisin B1 is the most toxic and is carcinogenic in rats and mice and is thought to be a promoter of esophageal cancer in humans. Fumonisins are structurally similar to sphingosine, a component of sphingolipids, which are in high concentrations in certain nerve tissues such as myelin. Fumonisin toxicity results from blockage of sphingolipid biosynthesis and thus degeneration of tissues rich in sphingolipids.

While FB is much less potent in ruminants than in swine or horses, it has been shown toxic to sheep, goats, beef cattle, and dairy cattle. Extent of rumen degradation has not been established. In cattle, FB cause liver lesions, lymphocyte blastogenesis and elevated enzymes indicative of liver damage. Dairy cattle (Holsteins and Jerseys) fed diets containing 100 ppm FB for approximately 7 days prior to freshening and for 70 days thereafter demonstrated lower milk production (6 kg/cow/day), explained primarily by reduced feed consumption. Fumonisin carryover from feed to milk is thought to be negligible. Guidance levels for FB are in table 4 (Henry, 2006).

Table 4. Guidance levels for total fumonisins in animal feeds (Henry, 2006)

Class of Animal	Feed Ingredients & Portion of Diet	Levels in Corn & Corn By-products	Levels in Finished Feeds
Equids and Rabbits	Corn and corn by-products not to exceed 20% of the diet **	5 ppm	1 ppm
Swine and Catfish	Corn and corn by-products not to exceed 50% of the diet**	20 ppm	10 ppm
Breeding Ruminants, Breeding Poultry and Breeding Mink*	Corn and corn by-products not to exceed 50% of the diet**	30 ppm	15 ppm
Ruminants ≥3 Months Old being Raised for Slaughter and Mink being Raised for Pelt Production	Corn and corn by-products not to exceed 50% of the diet**	60 ppm	30 ppm
Poultry being Raised for Slaughter	Corn and corn by-products not to exceed 50% of the diet**	100 ppm	50 ppm
All Other Species or Classes of Livestock and Pet Animals	Corn and corn by-products not to exceed 50% of the diet**	10 ppm	5 ppm

***** Includes lactating dairy cattle and hens laying eggs for human consumption.
** Dry weight basis.

Ergot alkaloids, including fescue toxicity

One of the earliest recognized mycotoxicoses is ergotism caused by a group of ergot alkaloids. They are produced by several species of *Claviceps*, which infect the plant and produce toxins in fungal bodies called sclerotia or ergots, which are small black colored bodies similar in size to the grain. Symptoms are directly related to dietary concentrations and include reduced weight

gains, lameness, lower milk production, agalactia and immune suppression. Sclerotia concentrations above 0.3% are related to reproductive disorders.

Fescue grass infected with *Neotyphodium* or *Epichloe* can contain toxic ergot alkaloids. Affected cattle show symptoms including lower weight gains, rough hair coat, increased body temperature, agalactia, reduced conception, and gangrenous necrosis of the extremities.

Ochratoxin A (OTA)

Ochratoxin A is produced by species of *Penicillium* and *Aspergillus* and is a causative agent of kidney disease in pigs. The primary toxic effect is inhibition of protein synthesis. In cattle, OTA is rapidly degraded in the rumen and thus thought to be of little consequence unless consumed by pre-ruminant calves, however chronic exposure and acute toxicities are thought to occur in cattle. Moldy hay containing OTA has been implicated in cattle deaths and abortions. The FDA has no guidelines for ochratoxin in feed (Henry, 2006).

PR Toxin, Roquefortine C and Mycophenolic Acid

PR toxin, roquefortine C and mycophenolic acid are a few of the mycotoxins produced by *Penicillium* molds. *Penicillium* grows at a low pH and in cool damp conditions and has been found to be a major contaminant of silage. PR toxin, produced by *P. roquefortii*, has been suggested as a major causative agent associated with moldy corn silage problems and has been shown to be the best marker for mycotoxin associated health problems in dairy herds. In Europe, grass and corn silage have shown a high occurrence of *P. roquefortii* (40% of samples) and associated with cattle disorders. In mice, rats and cats, PR toxin causes increased capillary permeability resulting in direct damage to the lungs, heart, liver and kidneys. PR toxin, roquefortine C, and mycophenolic acid have also been associated with dairy herd health problems including abortions, retained placenta and death. Currently no commercial lab analyzes feeds for PR toxin, but the service is expected in 2008.

Patulin

Patulin is produced by *Penicillium*, *Aspergillus* and *Byssochlamys*. Patulin has been found in grains, especially wet grains, and silage. Patulin is antibiotic against gram-positive bacteria. Patulin at high doses reduces VFA production, fiber digestion and bacterial yield in rumen cultures. The potential for patulin toxicity of livestock is thought to be low, but case studies of cattle toxicities are reported.

Forage Mycotoxins

The array of mycotoxins found in forages can be different than those found in grains, and these different mycotoxins may be of major importance in mycotoxicoses of ruminants. Many different fungi have been isolated from forages and the vast majority of forages contain mold. Some of the dominant genera in forages are *Penicillium, Mucoraceae, Monascus, Aspergillus, and Fusarium*.

Mycotoxin Testing

Analytical techniques for mycotoxins are continually improving. Several commercial laboratories are available and provide screens for an array of mycotoxins. Cost of analyses has been a constraint but can be insignificant compared with the economic consequences of production and health losses related to mycotoxin contamination.

The accurate determination of mycotoxin concentrations in feedstuffs depends on a number of factors. First, a representative sample must be drawn from the lot, but because mycotoxins are not evenly distributed in feeds, as much as 90% of the error for a single analysis is due to sampling. Once collected, samples should be handled to prevent further mold growth. Wet samples may be frozen or dried before shipment, and transit time should be minimized.

Mold spore counts provide a gross indication of the potential for toxicity but presence of mold does not correlate well with the presence of individual mycotoxins. Mold identification can be useful to suggest which mycotoxins are potentially present.

Prevention and Treatment

Adapted crop varieties with resistance to fungal disease or to insect damage (Bt hybrids) have fewer field produced mycotoxins. Positive field factors are irrigation, timely harvest, avoidance of harvesting lodged or field damaged materials and avoiding kernel damage.

Harvested grains should be dried to below 15% moisture and preferably to <13% to help compensate for non-uniform moisture concentrations throughout the grain mass. Because high temperatures increase the amount of free moisture (water activity), grain should be drier when stored at high temperatures. Storage must be sufficient to eliminate moisture migration, moisture condensation and leaks. Aeration helps reduce moisture migration

and non-uniform moisture concentrations. Commodity sheds should protect feedstuffs from rain and have a vapor barrier in the floor to reduce moisture. Bins, silos and other storage facilities should be cleaned to eliminate sources of inoculation. Check stored feed at intervals to determine if heating and molding are occurring. Organic acids can be used as preservatives for feeds too high in moisture for proper storage.

Mold will grow in moist hay, but it is sometimes difficult to achieve adequate dry down which is related to moisture at harvest, air movement, humidity, air temperature, bale density and the storage facility. Rate of dry down can be improved by ventilation, creation of air spaces between bales, reduced size of stacks, alternated direction of stacking and avoidance of other wet products in the same area.

Production of mycotoxins in silage can be reduced by following accepted silage making practices aimed at preventing deterioration primarily by quickly reducing pH and eliminating the oxygen. Accepted silage making practices emphasize:

- harvesting at the proper moisture content;
- chopping uniformly at the proper length;
- filling the silo rapidly;
- packing the silage sufficiently to exclude air;
- using an effective fermentation aide; and
- covering completely and well.

Infiltration of air after ensiling can allow growth of acid tolerant microorganisms, an increase in the pH and then mold growth. *Penicillium* molds are acid tolerant and can grow if any air is present. Microbial or other additives that enhance fermentation and rapidly reduce pH can reduce mold growth and mycotoxin formation. Chemical treatments that inhibit microbial growth have also been used effectively. Ammonia, organic acids, sulfates, urea and nitrates are shown to be at least partially effective at inhibiting mold growth. Organic acids have been used to treat the entire silage mass, or to selectively treat the outer layers of the silo. Organic acids are sometimes used during feedout to treat the silo feeding face in an effort to reduce deterioration of the feeding face. Treatment of the TMR with organic acids can reduce heating in the feed bunk. Silo size should be matched to herd size to insure daily removal of silage at a rate faster than deterioration. In warm weather, it is best to remove a foot of silage daily from the feeding face. The feeding face of silos should be cleanly cut and disturbed as little as possible to prevent aeration into the silage mass. Silage (or other wet feeds) should be fed immediately after removal from storage. Spoilage should not be fed and feed bunks should be cleaned regularly.

As with silage, high moisture grains or wet byproduct feeds must be stored at proper moisture content to exclude air and stored in a well maintained and managed structure. Wet feeds must be handled in quantities that allow them to be fed out rapidly. Organic acids can help prevent mold and can extend storage life.

Nutritional factors may be helpful in reducing effects of mycotoxins. Increasing nutrients such as protein, energy and antioxidants may be advisable. Animals exposed to aflatoxin show marginal responses to increased protein. In some situations, poultry respond to water soluble vitamins or to specific minerals, but data is lacking for cattle. Acidic diets seem to exacerbate effects of mycotoxins, and therefore adequate dietary fiber and buffers are recommended. Because a robust rumen fermentation can help destroy mycotoxins, cows may benefit from feed additives that enhance rumen function. Because mycotoxins reduce feed consumption, feeding management to encourage intake can be helpful. Dry cows, springing heifers and calves should receive the cleanest feed possible. Transition rations can reduce stress in fresh cows and reduce effects of mycotoxins. Strategic use of mold inhibitors can be beneficial.

■ Mycotoxin Adsorbents (Binders)

The addition of mycotoxin binders to contaminated diets may be the most promising dietary approach to reduce effects of mycotoxins. A binder decontaminates mycotoxins in the feed by binding them strongly enough to prevent toxic interactions with the consuming animal and to prevent mycotoxin absorption across the digestive tract. Therefore, this approach is often considered as preventative rather than a therapy.

Potential absorbent materials include activated carbon, aluminosilicates (clay, bentonite, montmorillonite, zeolite, phyllosilicates, etc.), complex indigestible carbohydrates (cellulose, polysaccharides in the cell walls of yeast and bacteria such as glucomannans, peptidoglycans, and others), and synthetic polymers such as cholestryamine and polyvinylpyrrolidone.

Overall, the benefits are variable by type and amount of binder, specific mycotoxins and their amounts, animal species, and interactions of other dietary ingredients. No adsorbent product is approved by the FDA for the prevention or treatment of mycotoxicoses. Several of these adsorbent materials are recognized as safe feed additives (GRAS) and are used in diets for other purposes such as flow agents or pellet binders.

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