Mycotoxins: A Review of Dairy Concerns

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Introduction

Molds are filamentous (fuzzy or dusty looking) fungi that occur in many feedstuffs including roughages and concentrates. Molds can infect dairy cattle, especially during stressful periods when they are immune suppressed, causing a disease referred to as mycosis. Molds also produce poisons called mycotoxins that affect animals when they consume mycotoxin contaminated feeds. This disorder is called mycotoxicosis. Mycotoxins are produced by a wide range of different molds and are classified as secondary metabolites, meaning that their function is not essential to the mold's existence. The U.N.'s Food and Agriculture Organization (**FAO**) has estimated that worldwide, about 25% of crops are affected annually with mycotoxins (Jelinek, 1987). Such surveys reveal sufficiently high occurrences and concentrations of mycotoxins to suggest that mycotoxins are a constant concern. Tables 1 and 2 provide mycotoxin occurrence and concentration of farmer submitted feedstuffs in North Carolina over several years.

Mycotoxins can be formed on crops in the field, during harvest, or during storage, processing, or feeding. Molds are present throughout the environment. The spores are high in the soil and in plant debris and lie ready to infect the growing plant in the field. Field diseases are characterized by yield loss, quality loss, and mycotoxin contamination. Mold growth and the production of mycotoxins are usually associated with extremes in weather conditions leading to plant stress or hydration of feedstuffs, insect damage, poor storage practices, low feedstuff quality, and inadequate feeding conditions.

It is generally accepted that the *Aspergillus, Fusarium* and *Penicillium* molds are among the most important in producing mycotoxins detrimental to cattle. The mycotoxins of greatest concern include: aflatoxin, which is generally produced by *Aspergillus* mold; deoxynivalenol, zearalenone, T-2 toxin, and fumonisin, which are produced by *Fusarium* molds; and ochratoxin and PR toxin produced by *Penicillium* molds. Several other mycotoxins such as the ergots are known to affect cattle and may be prevalent at times in certain feedstuffs.

There are hundreds of different mycotoxins, which are diverse in their chemistry and effects on animals. It is likely that contaminated feeds will contain more than one mycotoxin. This paper is directed toward those mycotoxins thought to occur most frequently at concentrations toxic to dairy cattle. A more extensive review is available in the popular press (Whitlow and Hagler, 2004).

Major toxigenic fungi and mycotoxins thought to be the most prevalent and potentially toxic to dairy cattle.

Fungal genera	Mycotoxins
Aspergillus	Aflatoxin, Ochratoxin, Sterigmatocystin, Fumitremorgens, Fumigaclavines, Fumitoxins, Cyclopiazonoic Acid, Gliotoxin
Fusarium	Deoxynivalenol, Zearalenone, T-2 Toxin, Fumonisin, Moniliformin, Nivalenol, Diacetoxyscirpenol, Butenolide, Neosolaniol, Fusaric Acid, Fusarochromanone, Wortmannin, Fusarin C, Fusaproliferin
Penicillium	Ochratoxin, PR Toxin, Patulin, Penicillic Acid, Citrinin, Penetrem, Cyclopiazonic Acid, Roquefortine, Isofumigaclavines A and B, Mycophenolic Acid
Claviceps	Ergot alkaloids in seed/grain of small grains, sorghum, grasses
Epichloe and Neotyphodium	Ergot alkaloids in fescue grass.

Stachybotrys   Stachybotryotoxins, Trichothecenes

Table 1. Percentage of feeds positive for mycotoxins in all feeds submitted by North Carolina dairy producers over a 13-year period (Whitlow et al., 1998).

	Aflatoxin n[1]=3266	Deoxynivalenol n=5053	Zearalenone n=4563	T-2 Toxin n=5136	Fumonisin n=822
Low, range	5-19 ppb	<500 ppb	100-299 ppb	50-99ppb	<5 ppm
%[2]	6.4	18.2	7.1	1.5	32.6
High, range	≥ 20 ppb	≥500 ppb	≥300 ppb	≥100 ppb	≥5 ppm
%	4.0	28.2	8.3	6.6	9.4
Total positive, %	10.4	46.2	15.4	8.1	42.0

[1]n = number of samples
[2]% = percentage of samples positive within given concentrations

Table 2. 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 (Whitlow et al., 1998).

	Corn Silage			Corn Grain			All Feeds		
	n[1]	% Pos[2]	mean ± s.d.[3]	n	% Pos	mean ± s.d.	n	% Pos	mean ± s.d.
Aflatoxin	461	8	28 ± 19	231	9	170 ± 606	1617	7	91 ± 320
Deoxynivalenol	778	66	1991 ± 2878	362	70	1504 ± 2550	2472	58	1739 ± 1880
Zearalenone	487	30	525 ± 799	219	11	206 ± 175	1769	18	445 ± 669
T-2 toxin	717	7	569 ± 830	353	6	569 ± 690	2243	7	482 ± 898
Fumonisin	63	3		37	60		283	28	

[1]n = number of samples
[2]% = percentage of samples positive above given concentrations
[3]mean ±s.d. = mean of the positive samples plus and minus the standard deviation

Aflatoxin production by *Aspergillus flavus* in corn is favored by heat and drought stress associated with warmer climates. *Fusarium* molds commonly affect corn, causing ear and stalk rots, and small grains, causing head blight (scab). In wheat, excess moisture at flowering and afterward is associated with increased incidence of mycotoxin formation. In corn, *Fusarium* diseases are more commonly associated with warm conditions at silking and with insect damage and wet conditions late in the growing season or late harvest. *Penicillium* molds grow in wet and cool conditions and require little oxygen.

Mycotoxins can increase the incidence of disease and reduce production efficiency in cattle (Coulombe, 1993; Joffe, 1986; Pier, 1992). Mycotoxins 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. They exert their effects through four primary mechanisms:

1) intake reduction or feed refusal,
2) reduced nutrient absorption and impaired metabolism;
3) alterations in the endocrine and exocrine systems; and
4) suppression of the immune system.

Recognition of the impact of mycotoxins on animal

production has been limited by the difficulty of diagnosis. Symptoms are often nonspecific and the result of a progression of effects, making a diagnosis difficult or impossible because of the complex clinical results with a wide diversity of symptoms. The difficulty of diagnosis is increased due to limited research, occurrence of multiple mycotoxins, non-uniform distribution, interactions with other factors, and problems of sampling and analysis.

Because of the difficulty of diagnosis, the determination of a mycotoxin problem becomes a process of elimination and association. Certain basics can be helpful:

1) Mycotoxins should be considered as a possible primary factor resulting in production losses and increased incidence of disease.
2) Documented symptoms in ruminants or other species can be used as a general guide to symptoms observed in the field.
3) Systemic effects as well as specific damage to target tissues can be used as a guide to possible causes.
4) Post mortem examinations may indicate no more than gut irritation, edema, or generalized tissue inflammation.
5) Because of the immune suppressing effects of mycotoxins, atypical diseases or increased incidence of disease may be observed.
6) Responses to added dietary sorbents or dilution of the contaminated feed may help in diagnosis.
7) Feed analyses should be performed, but accurate sampling is a problem (Schiefer, 1990).

Symptoms of a mycotoxicosis in a dairy herd vary depending on the mycotoxins involved and their interactions with other stress factors. The more stressed cows, such as fresh cows, are most affected; perhaps because their immune systems are already suppressed. Symptoms of mycotoxins may be nonspecific and wide ranging. Symptoms may be few or many. Symptoms may include: reduced production; reduced feed consumption; intermittent diarrhea (sometimes with bloody or dark manure); reduced feed intake; unthriftiness; rough hair coat; and reduced reproductive performance including irregular estrous cycles, embryonic mortalities, pregnant cows showing estrus, and decreased conception rates. There generally is an increase in incidence of disease; such as displaced abomasum, ketosis, retained placenta, metritis, mastitis, and fatty livers. Cows do not respond well to veterinary therapy.

Molds Can Cause Disease

A mold (fungal) infection resulting in disease is referred to as a mycosis. Fungal pathogens include: *Aspergillus fumigatus, Candida albicans, Candida vaginitis,* and certain species of *Fusarium.*

Aspergillus fumigatus has been proposed as the pathogenic agent associated with mycotic hemorrhagic bowel syndrome (**HBS**) in dairy cattle (Puntenney et al., 2003). *A. fumigatus* is thought to be a fairly common mold in both hay (Shadmi et al., 1974) and silage (Cole et al., 1977). While healthy cows with an active immune system are more resistant to mycotic infections, dairy cows in early lactation are immune suppressed (Kehrli et al., 1989a,b) and HBS is more likely in fresh cows (Puntenney et al., 2003). It is theorized that in mycosis, mycotoxins produced by the invading fungi can suppress immunity; therefore increasing the infectivity of the fungus. *A. fumigatus* produces several mycotoxins, including gliotoxin; which is an immune suppressant. Gliotoxin has been present in animals infected with *A. fumigatus* (Bauer et al., 1989). Reeves et al. (2004) using an insect model demonstrated the significance of gliotoxin in increasing the virulence of *A. fumigatus*. Niyo et al. (1988a, b) have demonstrated that in rabbits, T-2 toxin decreased phagocytosis of *A. fumigatus* conidia by alveolar macrophages and increased severity of experimental aspergillosis. It is possible that gliotoxin, T-2 toxin, or other mycotoxins that suppress immunity may be a trigger to increased infectivity by the fungus; ultimately resulting in HBS or other fungal infections. If this is true, then reducing animal exposure to mycotoxins may be a key to control of mycoses such as HBS. A commercial feed additive with anti-fungal and adsorbent properties appears to reduce HBS (Puntenney et al., 2003), although these additives can have other functions including the reduction of mold growth.

Toxicity of Individual Mycotoxins

Aflatoxin

Aflatoxins are a family of extremely toxic, mutagenic, and carcinogenic compounds produced by *Aspergillus flavus* and *A. parasiticus* (Deiner et a1., 1987; Kurtzman et al., 1987). Toxigenic *A. flavus* isolates produce

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aflatoxins B1 and B2, and toxigenic *A. parasiticus* isolates produce aflatoxins B1, B2, G1, and G2 (Cotty et al., 1994). Aflatoxin B1 is a carcinogen and is excreted in milk in the form of aflatoxin M1. Table 3 provides the Food and Drug Administration (**FDA**) action levels for aflatoxin in feeds and milk. 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% 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. In Europe the regulatory levels of aflatoxin are 20 ppb for dairy feeds and 0.05 ppb in milk; therefore, an illegal milk residue can occur when feed contains more than 3 ppb of aflatoxin. Figure 1 shows the clearance and appearance of aflatoxin in milk over a 16 day period in association with the feeding of clean or aflatoxin-contaminated corn, in diets with and without clay products added at 1%.
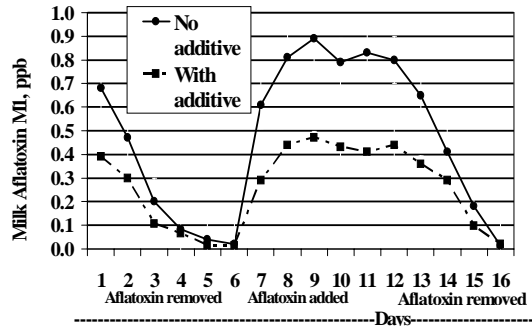
Table 3. U.S. Food and Drug Administration action levels for total aflatoxins in food and feed[a].

Food or Feedstuff	Concentration (ppb)
All products, except milk, designated for humans	20
Corn for immature animals and dairy cattle	20
Corn and peanut products for breeding beef cattle, swine, and mature poultry	100
Corn and peanut products for finishing swine (>100 lb)	200
Corn and peanut products for finishing beef cattle	300
Cottonseed meal (as a feed ingredient)	300
All other feedstuffs	20
Milk[b]	0.5

[a] Wood and Trucksess, 1998.
[b] Aflatoxin M1.

Symptoms of acute aflatoxicosis in mammals include: inappetance, lethargy, ataxia, rough hair coat, and pale, enlarged fatty livers. Symptoms of chronic

Figure 1. Concentration of aflatoxin M1 in milk of dairy cows over a 16 day period associated with consumption of aflatoxin-contaminated or clean corn in diets with and without clay products added at 1% of the diets. Milk aflatoxin concentrations are reduced by dietary addition of clay products. Appearance and clearance of aflatoxin in milk are rapid and unaffected by dietary clay additions. (Diaz et al., 2004).

aflatoxin exposure include reduced feed efficiency and milk production, jaundice, and decreased appetite (Nibbelink, 1986). Aflatoxin lowers resistance to diseases and interferes with vaccine-induced immunity in livestock (Diekman and Green, 1992). In beef cattle, Garrett et al. (1968) showed an effect on weight gain and intake with diets containing 700 ppb aflatoxin, but if increases in liver weights are used as the criteria for toxicity, 100 ppb would be considered toxic to beef cattle. Production and health of dairy herds may be affected at dietary aflatoxin levels above 100 ppb, which is considerably higher than the amount that produces illegal milk residues (Patterson and Anderson 1982, and Masri et al., 1969). Guthrie (1979) showed when lactating dairy cattle in a field situation were consuming 120 ppb aflatoxin, reproductive efficiency declined and when cows were changed to an aflatoxin free diet, milk production increased over 25%. Applebaum et al. (1982) showed that impure aflatoxin produced by culture reduced production, while equal amounts of pure aflatoxin did not.

Aflatoxin is more often found in corn, peanuts and cottonseed grown in warm and humid climates. Aflatoxin can be found in more temperate areas, as was seen in the drought year of 1988 when aflatoxin was found in 5% of

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corn grain in the Midwestern U.S. (Russell et al., 1991). The General Accounting Office (GAO, 1991) concluded that industry, federal and state programs are effective in detecting and controlling aflatoxin and that it is doubtful that additional programs or limits would reduce the risk of aflatoxin in the food supply.

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 (Whitlow et al., 1994). Dairy cattle consuming diets contaminated primarily with DON (2.5 ppm) have responded favorably (1.5 kg milk, P<.05) to the dietary inclusion of a mycotoxin binder, providing circumstantial evidence that DON may reduce milk production (Diaz et al., 2001). Field reports help substantiate this association (Gotlieb, 1997 and Seglar, 1997). Results from a Canadian study using 6 first-

Table 4. U.S. Food and Drug Administration advisory levels for deoxynivalenol in wheat and wheat derived products[a].

Product	Concentration, ppm
All finished wheat products, e.g. flour, bran and germ, for human consumption	1
Wheat and wheat by-products destined for ruminating beef cattle and cattle and cattle in feedlots older than 4 months and for chickens (these ingredients should not exceed 50% of the diet)	10
Wheat and wheat by-products destined for swine (these ingredients should not exceed 20% of the diet)	5
Wheat and wheat by-products for all other animals (these ingredients should not exceed 40% of the diet)	5

[a]Wood and Trucksess, 1998

lactation cows per treatment during mid-lactation (average 19.5 kg milk) showed that cows consuming

DON contaminated diets (2.6 to 6.5 ppm) tended (P<0.16) to produce less milk (13% or 1.4 kg) than did cows consuming clean feed (Charmley et al., 1993). DON had no effect on milk production in 8 cows fed over a 21 day period (Ingalls, 1994). Beef cattle and sheep have tolerated up to 21 ppm of dietary DON without obvious effects (DiCostanzo et al., 1995).

Like other mycotoxins, pure DON added to diets, does not have as much toxicity as does DON supplied from naturally contaminated feeds (Foster et al., 1986). This is thought to result from the interaction of multiple mycotoxins in naturally contaminated feeds. These mycotoxins can interact to cause symptoms that are different or more severe than expected. For example, it is now known that fusaric acid interacts with DON to cause the vomiting effects earlier attributed to DON alone and resulted in use of the trivial name of vomitoxin for DON (Smith and MacDonald, 1991). It is believed that DON serves as a marker, indicating that feed was exposed to a situation conducive for mold growth and possible formation of several mycotoxins.

T-2 Toxin (T-2)

T-2 toxin is a very potent *Fusarium* produced mycotoxin that occurs in a low proportion of feed samples (<10%). Russell et al. (1991) found 13% of Midwestern corn grain contaminated with T-2 toxin in a survey of the 1988 drought damaged crop.

T-2 is associated with reduced feed consumption, loss in yield, gastroenteritis, intestinal hemorrhage, reduced reproductive performance, and death. Effects are less well established in cattle than in laboratory animals (Wannemacher et al., 1991). T-2 toxin is associated with gastroenteritis, intestinal hemorrhages (Petrie et al., 1977; Mirocha et al., 1976) and death (Hsu et al., 1972 and Kosuri et al., 1970). Dietary T-2 toxin at 640 ppb for 20 days resulted in bloody feces, enteritis, abomasal and ruminal ulcers, and death (Pier et al., 1980). Weaver et al. (1980) showed that T-2 was associated with feed refusal and gastrointestinal lesions in a cow, but did not show a hemorrhagic syndrome. Kegl and Vanyi (1991) observed bloody diarrhea, low feed consumption, decreased milk production, and absence of estrous cycles in cows exposed to T-2. Serum immunoglobulins and complement proteins were lowered in calves receiving T-2 toxin (Mann et al., 1983). Gentry et al. (1984) demonstrated a reduction in white blood cell and neutrophil counts in calves. McLaughlin et al. (1977) demonstrated that primary

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basis of T-2 reduced immunity is reduced protein synthesis.

Zearalenone (ZEA)

Zearalenone is a *Fusarium* produced mycotoxin that has a chemical structure similar to estrogen and can produce an estrogenic response in animals. Zearalenone is associated with ear and stalk rots in corn and with scab in wheat (Christensen et al., 1988).

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. A controlled study with non-lactating cows fed up to 500 mg of ZEA (calculated dietary concentrations of about 25 ppm ZEA) showed no obvious effects except that corpora lutea were smaller in treated cows (Weaver et al., 1986b). In a similar study with heifers receiving 250 mg of ZEA by gelatin capsule (calculated dietary concentrations of about 25 ppm ZEA), conception rate was depressed about 25%; otherwise, no obvious effects were noted (Weaver et al., 1986a).

Several case reports have related ZEA to estrogenic responses in ruminants including abortions (Kallela and Ettala, 1984; Khamis et al., 1986; Mirocha et al., 1968; Mirocha et al., 1974; and Roine et al., 1971). Symptoms have included vaginitis, vaginal secretions, poor reproductive performance, and mammary gland enlargement of virgin heifers. In a field study, (Coppock et al., 1990) diets with about 660 ppb ZEA and 440 ppb DON resulted in poor consumption, depressed milk production, diarrhea, increased reproductive tract infections, and total reproductive failure.

New Zealand workers (Towers et al., 1995) have measured blood ZEA and metabolites ("zearalenone") to estimate ZEA intake. Dairy herds with low fertility had higher levels of blood "zearalenone". Individual cows within herds examined by palpation and determined to be cycling had lower blood "zearalenone" levels than did cows that were not cycling. The reproductive problems in dairy cattle were associated with dietary ZEA concentrations of about 400 ppb.

Fumonisin (FB)

Fumonisin B_1 produced by *F. verticillioides*, was first isolated in 1988. It causes leucoencephalomalacia

in horses, pulmonary edema in swine, and hepatoxicity in rats. It is carcinogenic in rats and mice (NTP, 1999) and is thought to be a promoter of esophageal cancer in humans (Chu and Li, 1994 and Rheeder et al., 1992). 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_1 is much less potent in ruminants than in hogs, it has now been shown toxic to sheep, goats, beef cattle, and dairy cattle. Osweiler et al. (1993) fed 18 young steers either 15, 31, or 148 ppm of fumonisin in a short term study (31 days). With the highest feeding level, there were mild liver lesions found in two of six calves, and the group had elevated liver enzymes indicative of liver damage. Lymphocyte blastogenesis was significantly impaired at the end of the feeding period in the group having the highest dose.

Dairy cattle (Holsteins and Jerseys) fed diets containing 100 ppm fumonisin 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 (Figure 2, Diaz et al., 2000). Increases in serum enzyme concentrations suggested mild liver disease. Because of greater production stress, dairy cattle may be more sensitive to fumonisin than are beef cattle. Fumonisin carryover from feed to milk is thought to be negligible (Scott et al., 1994).

A USDA, APHIS survey of 1995 corn from Missouri, Iowa, and Illinois found that 6.9% contained more than 5 ppm fumonisin B_1 (Anon., 1995). Fumonisin was prevalent in Midwestern corn from the wet 1993 season as well. Corn screenings contain about 10 times the fumonisin content of the original corn.

Other Mycotoxins

Many other mycotoxins may affect ruminants but they are thought to occur less frequently or be less potent. Diacetoxyscirpenol, HT-2 and neosolaniol may occur along with T-2 toxin and cause similar symptoms. Ochratoxin has been reported to affect cattle, but it is rapidly degraded in the rumen and thus thought to be of little consequence except for pre-ruminants. Tremorgens, such as fumigaclavine A and B, produced by *Aspergillus*

fumigatus are thought to be common in silages of the southeastern US and were toxic to beef cattle in a field case. In Georgia (Cole et al., 1977) tremorgens caused anorexia, diarrhea, unthriftiness, and irritability. Mycotoxins such as rubratoxin, citrinin, patulin, cyclopiazonic acid, sterigmatocystin, and ergot alkaloids may also be of importance. Mycotoxins in forages have been reviewed by Lacey (1991).

Mycotoxin Testing

Analytical techniques for mycotoxins are improving. Several commercial laboratories are available and provide screens for a large 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. Newer immunoassays have reduced the cost of analyses.

Collection of representative feed samples is a problem, primarily because molds can produce very large amounts of mycotoxins in small areas making the mycotoxin level highly variable within the lot of feed (Whittaker et al., 1991). Core sampling of horizontal silos shows mycotoxins can be highly variable throughout the silo. Because mycotoxins can form in the collected sample, samples should be preserved and delivered to the lab quickly. Samples can be dried, frozen, or treated with a mold inhibitor before shipping.

Figure 2. Daily milk production (31.2 vs 24.2, P ≤ .05) for dairy cows n = 26 (Holsteins and Jerseys) consuming control diets (< 1 ppm fumonisin) or fumonisin-contaminated diets (100 ppm fumonisin) respectively for about 7 days prior to parturition and for 70 days in lactation. (Diaz et al., 2000).
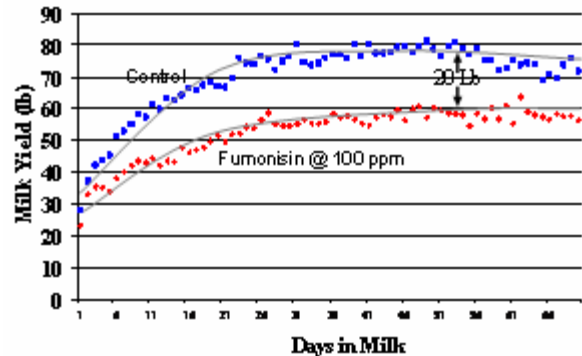

Table 5. U.S. Food and Drug Administration Guidance for industry on fumonisin levels in human foods and animal feeds[a].

	Total Fumonisins ($FB_1+FB_2+FB_3$) Concentration (ppm)
Human Foods	
Product	
Degermed dry milled corn products (e.g., flaking grits, corn grits, corn meal, corn flour with fat content of < 2.25%, dry weight basis)	2
Whole or partially degermed dry milled corn products (e.g., flaking grits, corn grits, corn meal, corn flour with fat content of ≥ 2.25 %, dry weight basis)	4
Dry milled corn bran	4
Cleaned corn intended for masa production	4
Cleaned corn intended for popcorn	3
Animal Feeds	
Corn and corn by-products intended for:	
Equids and rabbits (no more than 20% of diet)[b]	5
Swine and catfish (no more than 50% of diet)[b]	20
Breeding ruminants, breeding poultry and breeding mink and including lactating dairy cattle	

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and hens laying eggs for human consumption (no more than 50% of diet)[b]   30
Ruminants ≥ 3 months old being raised for slaughter and mink being raised for pelt production
                 (no more than 50% of diet)[b]   60
Poultry being raised for slaughter (no more than 50% of diet)[b]   100
 All other species or classes of livestock and pet animals (no more than 50% of diet)[b]   10

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[a] Federal Register, 2001.
[b] Limits on ingredients are on a dry weight basis

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Concentrations of mycotoxins, that are considered as acceptable and of no consequence, should be conservatively low due to non-uniform distribution, uncertainties in sampling and analysis, the potential for multiple sources in the diet, and interacting factors affecting toxicity (Hamilton, 1984).

Prevention and Treatment

Prevention of mycotoxin formation is essential since there are few ways to completely overcome problems once mycotoxins are present. Drought and insect damage are most important in instigating molding and mycotoxin formation in the field. Varieties chosen to have some resistance to fungal disease, and resistance to insect damage (Bt hybrids) have fewer field produced mycotoxins. Varieties should be adapted to the growing area. Irrigation can reduce mycotoxin formation in the field. When harvesting, avoid lodged or fallen material, because contact with soil can increase mycotoxins. Mycotoxins increase with delayed harvest, and with late season rain and cool periods. Damaged grains have increased mycotoxin levels; thus for dry grain storage, harvesting equipment should be maintained to avoid kernel damage. Mycotoxin concentrations are greatest in the fines, in broken and damaged kernels; thus cleaning can greatly reduce mycotoxin concentrations in the feedstuff. After harvest, grains should not be allowed to remain at levels of moisture greater than 15 to 18%. While there is little mold growth in grain at moisture levels below 15%, drying to levels below 14% and preferably to <13% help to compensate for non-uniform moisture concentrations throughout the grain mass. The high ambient temperatures of Texas also dictate that grain must be dried to the lower levels because higher temperatures increase the amount of free moisture (water activity) in the grain; which is the primary cause of mold growth in storage. Storage should be sufficient to eliminate moisture migration, moisture condensation, or leaks. Grain stored for more than two weeks should be kept aerated and cool. Aeration is important because as molds start to grow in isolated spots, the moisture produced by metabolism is sufficient to stimulate spread of the mold growth. Aeration reduces moisture migration and non-uniform moisture concentrations. Commodity sheds should protect feedstuffs from rain or other water sources. They should be constructed with a vapor barrier in the floor to reduce moisture. If wet feeds are stored in commodity sheds near dry feeds, a method must be devised to prevent moisture contamination of the dry feed. Bins, silos and other storage facilities should be cleaned to eliminate source 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.

It can be difficult to make hay at moisture levels low enough to prevent mold growth. Mold will grow in hay at moisture levels above 12 to 15%. As molds and other microorganisms grow they produce heat and cause deterioration. Heating can become so intense as to cause spontaneous combustion and hay fires. Feeding moldy hay can reduce intake and performance and the deterioration results in reduced nutritional value. Hay harvested at high moistures will tend to equilibrate to moisture contents of 12 to 14%, but rate of moisture loss is dependent on moisture at harvest, air movement, humidity, air temperature, bale density, and the storage facility. Rate of dry down is enhanced by ventilation, creation of air spaces between bales, reduced size of stacks, alternation in the direction of stacking, and avoidance of other wet products in the same area.

Prevention of mycotoxins in silage includes following accepted silage making practices aimed at preventing deterioration, primarily by quickly reducing pH and elimination of oxygen. Generally accepted silage making practices are to harvest at the proper moisture content; chop uniformly at the proper length; fill the silo rapidly; pack the silage sufficiently; use an effective fermentation aide; and cover 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 somewhat acid tolerant and may grow if any air is present. Some additives are beneficial in reducing pH very rapidly; therefore they can reduce mold growth and mycotoxin formation. Ammonia, propionic acid, sorbic acid, and microbial or enzymatic silage additives are shown to be at least partially effective at inhibiting mold growth. Ammonia may prevent silage from reaching a low pH, but it can reduce mold growth through direct inhibition of the mold. Organic acids provide the acidity for preservation without relying solely on acids produced in the ensiling process. Organic acids may be used to treat the entire silage mass, or to selectively treat the outer layers of the silo. Organic acids are also used during feedout to treat the silo feeding face and/or the TMR in an effort to reduce continuous deterioration of the feeding face and to 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

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deterioration. In warm climates 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 storage removal. Spoilage should not be fed and feed bunks should be cleaned regularly.

As with silage, high moisture grains or byproduct feeds must be stored at proper moisture contents in a well maintained structure and managed well to prevent mold. Wet feeds must be handled in quantities which allow them to be fed out within 7 to 10 days. Organic acids are very helpful in preventing mold in wet commodity feeds and can extend storage life. Discard any spoilage.

Obviously moldy feed should be avoided. Spoilage or deteriorated silage can reduce feed consumption, fiber digestibility, and production. If unacceptably high levels of mycotoxins occur, dilution or removal of the contaminated feed is preferable; however, it is often impossible to completely replace some feeds in the ration, particularly the forage ingredients. Ammoniation of grains can destroy some mycotoxins, but there is no practical method to detoxify affected forages. Cleaning grains can be helpful. Dietary strategies to counteract the effects of mycotoxins have been reviewed (Galvano et al., 2001). Increasing dietary levels of 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. Acidic diets seem to exacerbate effects of mycotoxins; therefore adequate dietary fiber and buffers are recommended. 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. Strategic use of mold inhibitors can be beneficial.

When animals are exposed to mycotoxins, favorable results have been seen when absorbent materials such as complex indigestible carbohydrates (glucomannans or mannanoligiosaccharides) or clays (bentonites and others), are added to mycotoxin contaminated diets of rats, poultry, swine, and cattle. Some of these products have been reviewed by Huwig et al. (2001), and yet many studies have been published since this review. Responses in dairy cattle to some of these products have been very encouraging. Overall results 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 generally recognized as safe **(GRAS)** feed additives and are used in diets for other purposes such as flow agents, pellet binders, etc. Figure 3 shows the effects of some feed additives on reducing aflatoxin in milk; theoretically as a result of binding the aflatoxin and therefore reducing intestinal absorption.
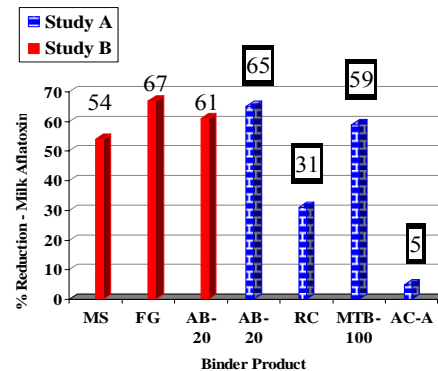

Figure 3. Effect of feed additives on reduction of milk aflatoxin residues in two studies. MS, mycrosorb, a sodium bentonite fed at 1% of DM (American Colliod Co.); FG, flowguard, a sodium bentonite fed at 1% of DM intake (La Port Biochem.); AB-20, a sodium bentonite fed at 1% of DM intake (Prince AgriProducts, Inc.); RC, Red Crown, a calcium bentonite fed at 1% of DM intake (Prince AgriProducts, Inc.); and MTB-100, a modified glucomannan product fed at 0.05% of DM intake (Alltech, Inc.) significantly reduced (P < .0001) AFM1 residues in milk. AC-A, an activated charcoal fed at 0.25% of DM intake had no effect (Diaz, et al. 2004).

Summary

- Mycotoxins are prevalent in feedstuffs.
- Many different mycotoxins exist.
- Mycotoxins affect dairy cattle in many ways, and the most important is perhaps immunosuppression.
- Mycotoxins can cause acute toxicity, but they are more likely to cause chronic problems of increased disease and decreased milk production.
- Diagnosis of a mycotoxicosis is difficult and indirect, but mycotoxins should be considered as a potential cause of increased disease and loss of production.

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- Contamination of milk by aflatoxin can cause huge economic losses.
- Management of crops and feeds is important to reduce mycotoxin contamination.
- Certain feed additives are proved to be helpful in treatment.

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