

# **Mycotoxins: Occurrence in Feeds, Effects on Dairy Cattle, Prevention and Treatment**

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## **Introduction**

Mycotoxins are toxic compounds produced by actively growing molds. Mycotoxins occur frequently in dairy cattle feedstuffs and have been associated with chronic and acute problems of poor performance and health. With such problems, mycotoxins should be considered as a causative factor, however a mycotoxicosis is difficult to diagnose. Methods for detection of mycotoxins have improved in accuracy and cost. The potential for effective treatments has improved. Prevention practices should receive greater emphasis.

## **Mold Growth and Mycotoxin Formation**

Molds are fungi which grow in multicellular colonies, as compared with yeasts which are single cellular fungi. The great majority of molds require oxygen for growth. They grow over a temperature range of approximately 10-40° C (50-104° F), a pH range of 4 to 8, and above 0.7  $a_w$  (equilibrium relative humidity expressed as a decimal instead of a percentage). Mold can grow when moisture exceeds about 12%. Higher levels of moisture support mold growth up to the point where water excludes adequate oxygen. Almost all molds are aerobic. Molds can grow on a dry surface, while yeast require a moist surface or water layer (Lacey, 1991).

Mold growth and mycotoxin production are related to weather extremes (causing plant stress or excess hydration of stored feedstuffs), to inadequate storage practices, to low feedstuff quality, and to faulty feeding conditions. Molds can grow and mycotoxins can be produced pre-harvest or post-harvest, during storage, processing, or feeding. Because crops can be contaminated prior to harvest, excellent storage conditions are essential to prevent further mold growth and mycotoxin formation. A mycotoxicoses can result from consumption of fresh forages (Lacey, 1991). A few of the more recognizable may include ergotism, fescue toxicity, ryegrass staggers, and slaframine toxicity.

The *Aspergillus* species grow at lower water activities and at higher temperatures than do the *Fusarium* species which generally require higher water activities and grow at much lower temperatures. *Aspergillus flavus* and aflatoxin in corn are favored by the heat and drought stress associated with warmer climates. Aflatoxin seems to be enhanced by insect damage before and after harvest. *Penicillium* species grow at relatively low water activities and low temperatures and are fairly widespread in occurrence. Since both *Aspergillus* and *Penicillium* grow at low water activities, they are considered to be storage fungi. *Aspergillus* species are more likely in warm climates while *Fusarium* and *Penicillium* species are more likely in cooler climates.

The *Fusarium* species are generally considered to be field fungi and more likely to proliferate prior

to storage. *Fusarium* commonly affects corn and small grains. In corn, *Fusarium* molds are associated with ear rot and stalk rot. In small grains, *Fusarium* molds are associated with field diseases such as head blight (scab). These field diseases are characterized by yield loss, quality loss and mycotoxin contamination. 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 insect damage, warm conditions at silking, and wet conditions late in the growing season. Joffe (1986) suggests that the toxic principle in the soil spreads to the plant, first affecting the vegetative parts and then the grain. The grain provides a favorable substrate for toxin accumulation. Trenholm et al. (1988) suggest that plowing in plant debris and crop residue left on the field after harvest may reduce fungal disease problems.

The conditions most suitable for mold growth are not necessarily the optimum conditions for mycotoxin formation. For example, the *Fusarium* molds associated with alimentary toxic aleukia have been reported to grow prolifically at temperatures of 25 to 30°C without producing much mycotoxin, but at near freezing temperatures, large quantities of mycotoxins are produced without much mold growth (Joffe, 1986). Field applications of fungicides may either increase or decrease mycotoxin production (Boyacioglu et al., 1992 and Gareis and Ceynowa, 1994). Reduction of the mold presence may reduce mycotoxins, however, the stress or shock to the mold organism may cause increased mycotoxin production.

### **Storage of Grains and Forages**

In stored grains, the primary factors affecting mold growth are water activity ( $a_w$ ), aeration and temperature. Physical damage to the kernel and insect damage can also increase mold contamination. Water availability to the mold is better described by  $a_w$  than by simple moisture content. Generally mold growth is minimal when the  $a_w$  is below 0.70. As the temperature increases, more water becomes free and increases the  $a_w$ . Aeration is important in replacing moist air with drier air and in maintaining a lower temperature. Variations in temperature can cause moisture migration, creating spots of higher moisture which support mold growth. As a thumb rule, it is best to store grain with no more than 13 -15% moisture, aerate when air humidity is low, and if temperature rises in the grain.

High moisture corn should be stored at a moisture of 26 - 29%, and 1 -2 % higher for ear corn. Corn should be ground prior to storage. Avoid long delays between harvest and storage. The silo should be well maintained and as air tight as possible. Propionic acid applied at 0.5 to 1.5%, can be used as a preservative with more acid required when moisture content is high. Some cases of mycotoxicoses in dairy cattle have been associated with contaminated high moisture corn.

The primary storage methods for forage are drying (hay) and ensiling (silage). These methods of storage utilize the principles that growth of undesirable organisms is retarded in hay by low moisture content and in silage by a low pH and absence of air. In poorly stored forages, molds are potential spoilage organisms, which not only cause deterioration but can also produce mycotoxins. Therefore, maintaining proper preservative conditions are critical in ensuring quality forages. (Woolford, 1984 and McDonald, et al., 1991).

Dry matter loss during hay storage can be huge, especially for large round bales stored unwrapped and without shelter in the Eastern US. Heat tolerant molds account for much of the spoilage occurring in wet hay. With excessive moisture (>20%) molds can grow prolifically, dry matter and nutrient losses can be large and heating can be significant. With such hay, it is common to have spoilage in the outer 4 to 8 inches, and sometimes with browning throughout the bale. General recommendations suggest that large round bales should contain no more than 18% moisture, large rectangular bales <16% moisture and small rectangular bales < 20% moisture. Even at these levels of moisture, some molding can occur if moisture levels remain high for an extended period, or if spots of higher moisture are present.

In silage, molds may grow during the early aerobic phase, but as the oxygen is depleted and the lactic acid producing anaerobes prevail, the pH and oxygen levels become too low for molds to continue growing. However, some mold growth may continue near the silo surfaces, which are exposed to air and in air pockets within the silage mass. A dry silage (>35% dry matter) is more prone to mold growth than is a wet silage (<30% dry matter). A wet silage has less air infiltration and is more likely to support production of butyric acid, which contributes to aerobic stability and retards mold growth. A dry silage may allow more air to infiltrate, resulting in a prolonged aerobic phase, less production of lactate, and a higher final pH. After the primary fermentation phase, continued air infiltration and a higher than optimum pH may allow renewed aerobic activity and depletion of organic acids. Acid tolerant yeast may proliferate if silage with adequate residual sugars is exposed to air. In this case, yeast will utilize lactic acid and then raise the pH enough for molds to grow. High yeast counts, especially when in excess of  $10^5$  organisms per gram, may be indicative of aerobic instability but yeast are not the only factor (Henderson et al., 1979).

Molds may be especially prolific within areas of the silage where air is allowed to infiltrate. Problems may occur in all types of silos, for example in tower silos that may have poorly fitting doors, in bag silage where the silage is not tightly packed or plastic is not properly sealed or maintained, and in horizontal silos not properly packed or sealed. In all these cases, infiltration of air can result in spoilage. Once the silo is opened, the silage becomes aerated. Oxygen can penetrate to a depth of two feet within a day. Therefore it is important that silage be fed at a rate which allows exposed silage on the feeding face to be fed before excessive deterioration occurs. Recommended feeding rates vary from 4 to 12 inches daily with the higher feeding rates recommended in warm weather. Horizontal silos offer a special challenge. Wide horizontal silos may have feeding faces too large to allow for a sufficient feeding rate. In this case, preservatives such as propionic acid may be used on the feeding face to reduce daily deterioration and mold growth. The feeding face should be disturbed as little as possible and that silage which is removed should be fed soon after removal. Fissures opened when the loader bucket is raised against the feeding face can introduce air to a depth of several feet. Dry matter loss in horizontal silos is twice as great when using a front-end loader in comparison to a block cutter (Honig, 1975).

Excessive heating in the feed bunk is an indication of unstable silage and further deterioration. Practices to reduce deterioration and heating in the feed bunk include: immediate feeding of silage after unloading, use of propionic acid on the silage or TMR at feeding, increasing the

frequency of feeding, and properly cleaning and maintaining feed bunks.

### **Mycotoxin Occurrence**

Mycotoxins occur in most all types of feedstuffs and they occur worldwide. Table 1 shows the occurrence of five mycotoxins in corn silage, corn grain and in all feeds analyzed (Whitlow, et al., 1998). These results over a nine-year period from feed samples submitted by North Carolina farmers may contain a bias because samples are not random. Occurrence and concentrations of mycotoxins were variable by year.

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.

Aflatoxin >10 ppb n % mean ± s.d.	Deoxynivalenol >50 ppb n % mean ± s.d.	Zearalenone > 70ppb n % mean ± s.d.	T-2 Toxin >50 ppb n % mean ± s.d.	Fumonisin >1ppm n %
<b>CORN SILAGE</b>				
461 8 28 ± 19	778 66 1991 ± 2878	487 30 525 ± 799	717 7 569 ± 830	63 37
<b>CORN GRAIN</b>				
231 9 170 ± 606	362 70 1504 ± 2550	219 11 206 ± 175	353 6 569 ± 690	37 60
<b>ALL FEEDS</b>				
1617 7 91 ± 320	2472 58 1739 ± 10880	1769 18 445 ± 669	2243 7 482 ± 898	283 28

n = number of samples

% = percentage of samples positive above levels given

x = mean of the positive samples plus and minus the standard deviation

### **Mycotoxin Effects**

Mycotoxins can increase disease incidence and reduce production efficiency. Mycotoxins exert their effects through three primary mechanisms: (1) alteration in nutrient content, absorption and metabolism, (2) changes in the endocrine and neuroendocrine function, and (3) suppression of the immune system (Council for Agricultural Science and Technology, 1989). The resulting cascade of symptoms may be perplexing and make diagnosis difficult. Hesselstine (1986b) and Schilfer (1990) discussed some of the problems encountered in diagnosing a mycotoxicosis which include: (1) a lack of research reports especially concerning some mycotoxins (2) symptoms which are not specific or unique for the mycotoxin, (3) interaction of mycotoxins with other mycotoxins or other stress factors, (4) interaction of mycotoxins with immune suppression and thus infectious diseases, (5) lack of feed samples or samples improperly collected, (6) analysis which is complex and expensive.

Dairy herds experiencing a mycotoxicosis which is severe enough to reduce milk production, will usually display other symptoms. Often there is intermittent diarrhea, sometimes with bloody or dark manure. Cows may not respond well to typical veterinary therapy. Symptoms may be nonspecific and wide ranging and may include: reduced feed intake, feed refusal, unthriftiness, rough hair coat, undernourished appearance, subnormal production, increased abortions or embryonic mortalities, silent heats, irregular estrus cycles, expression of estrus in pregnant cows, and decreased conception

rates. Fresh cows perform poorly and generally have an increased incidence of disease particularly those that are most opportunistic in a dairy herd. There may be a higher incidence of displaced abomasum, ketosis, retained placenta, metritis, mastitis, and fatty livers. There may only be a few or several of these symptoms evident.

A definitive diagnosis of a mycotoxicosis cannot be made directly from symptoms, specific tissue damage, or even feed analyses, however, experience with mycotoxin affected herds greatly increases the probability of recognizing a problem.

The following guidelines may be helpful in dealing with a possible mycotoxicosis:

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 utilized to determine if a mycotoxin is possible. Note systemic effects or damage to target tissues. Postmortem examinations may be of little benefit, other than to indicate general gut irritation, edema or inflammation.
3. It is essential that other possible causes of similar symptoms be considered. These possibilities such as infectious agents or other toxins must then be methodically examined and ruled out.
4. All feeds should be analyzed for common mycotoxins.
5. Responses to simple treatments such as addition of feed additives (such as sorbants) or dilution of the contaminated feed can be helpful.
6. Diagnosis may be impossible because the clinical situation may be complex and complicated due to interactions with other agents.

### **Safe Levels of Mycotoxins**

Hamilton (1984) and Schaeffer and Hamilton (1991) have reviewed the topic of safe levels of mycotoxins. They conclude that epidemiological studies coupled with laboratory studies to elaborate the underlying principles of toxicity may be the best approach to determining safe levels. They state that any level of mycotoxin carries a risk of loss and thus it is impossible to define a safe level under laboratory conditions that will be accurate under field conditions, primarily because of three reasons: (1) difficulties in conceptualizing and executing experiments to investigate multiple interacting factors simultaneously; (2) the unappreciated fact that the frequency and level of contamination with aflatoxin and other mycotoxins vary unpredictably under field conditions; and (3) animal facilities currently available to investigators do not permit experiments under controlled conditions with the number of animals commonly at risk under field conditions. Establishing usable or tolerable levels of mycotoxins may be acceptable when all concerned parties are aware of levels and the risks associated.

Interactions with other factors make recommendations difficult. Lillehoj and Ceigler (1975) give an example where penicillic acid and citrinin were innocuous when administered alone but were 100% lethal when given in combination. Fumonisin at 100 ppm has been shown to reduce milk production in dairy cattle (Whitlow, unpublished), but to not affect average daily gain in beef cattle fed 148 ppm (Osweiler et al, 1993). Aflatoxin produced from culture was more toxic to dairy cattle than pure aflatoxin added to diets (Applebaum et al., 1982). In swine, Foster et al. (1986) demonstrated that pure deoxynivalenol added to diets was less toxic than diets with similar concentrations of

deoxynivalenol, which was supplied from naturally contaminated feeds. Smith and MacDonald (1991) have suggested that fusaric acid may occur along with deoxynivalenol to produce more severe symptoms. Many such interactions are possible since *Fusarium* molds produce many mycotoxins, and it is well documented that several mycotoxins may be found in the same feed (Hagler et al., 1984). Abbas et al. (1989) demonstrated that *Fusarium* species isolated from Minnesota corn produces an array of mycotoxins.

There are distinct species differences in tolerance to mycotoxins. Cattle are more tolerant to most mycotoxins than many other animals, probably due to some mycotoxin degradation in the rumen (Kiessling et al., 1984). However, he also demonstrated that a low rumen pH reduced degradation and that protozoa were the primary rumen microorganism with the ability to degrade mycotoxins. Thus, diet may play an important role in degree of mycotoxin degradation in the rumen. Wannemacher et al. (1991) presents data to show that even with laboratory animals such as the mouse and rat, T-2 elicits very real species differences. Mycotoxin effects are also moderated by factors such as sex, age, and stresses of the environment and production. Certainly duration of exposure is important. The known dietary factors, which interact with mycotoxins, include most nutrients for which rations are formulated including, fat, protein, fiber, vitamins and minerals. Dietary pellet binders (clay) adsorb some mycotoxins reducing exposure of the animal. Thus, many factors and interactions make it difficult to relate field observations to those from controlled research.

### **Mycotoxin Testing**

Molds can be present in a feed without the presence of mycotoxins, either because the mold is not stimulated to produce a mycotoxin or because the specific mold does not produce mycotoxins. Conversely, mycotoxins can be present without obvious mold growth. Therefore, mold spore counts may not be very useful and are only a gross indication of the potential for toxicity. Mold identification can be useful to suggest which mycotoxins that may be present.

Scott (1990) states that screening methods are needed for the *Fusarium* produced mycotoxins and that one approach is to test for deoxynivalenol, diacetoxyscirpenol, T-2 toxin and nivalenol, because other *Fusarium* mycotoxins seldom occur without one of these four also present. Feeds could then be further tested for other mycotoxins. Analytical techniques for mycotoxins are improving (Chu, 1992). The costs are decreasing and several commercial laboratories are available which provide screens for an array of mycotoxins. Generally laboratories provide analysis for only a limited number of mycotoxins perhaps including aflatoxin, ochratoxin, deoxynivalenol, zearalenone, fumonisin, and T-2 toxin. Collection and handling of representative feed samples is a problem. Since molds grow in spots, mycotoxins are not uniformly distributed within a feed, making it difficult to obtain a representative sample. This is further complicated if the feed is course, which prevents complete and adequate mixing. Once collected, samples should be handled properly to prevent further mold growth. Wet samples may be frozen or dried prior to shipment. Transit time should be minimized.

### **Aspergillus Molds**

Aflatoxin (AF) is produced primarily by *Aspergillus flavus* and is commonly found in the southern U.S., but also in other regions in some years when weather conditions are conducive. For example, 8% of samples of midwestern corn grain from the 1988 season contain aflatoxin (Russel, et al., 1991).

Aflatoxin levels in corn grain are much higher than in other parts of the corn plant, thus levels of aflatoxin in corn silage are diluted. It appears that *Aspergillus flavus* does not grow well in hay or silage, however, concentrations of aflatoxin as high as 5 ppm have been reported (Kalac and Woolford, 1982). We have detected low levels of aflatoxin in alfalfa, but it appears that levels of concern are infrequent.

The FDA limits AF in corn grain according to its intended use, which for lactating dairy cattle is 20 ppb. AF is excreted into milk in the form of AFM<sub>1</sub> with residues approximately equal to 1.7% of the dietary level (Van Egmond, 1989). The FDA limits aflatoxin M<sub>1</sub> in milk to no more than 0.5 ppb.

Levels of 300 to 700 ppb are considered toxic for beef cattle depending on criteria for toxicity, and other factors affecting toxicity. Garrett et al., (1968) showed that with beef cattle, gain and intake were affected at 700 ppb AF, but not at 300 ppb; however, levels of no effect can not be determined from such data with few animals. Trends in the data, especially for increased liver weights, would indicate potential toxicity at levels as low as 100 ppb. Guthrie (1979) showed a decline in reproductive efficiency when lactating dairy cattle in a field situation were consuming 120 ppb AF and an increase in milk production of over 25% when cows were changed to an AF free diet. Patterson and Anderson (1982) and Marsi et al. (1969) also suggest that 100 ppb may reduce milk production. Applebaum et al. (1982) showed that impure AF produced by culture reduced production while equal amounts of pure AF did not.

*Aspergillus fumigatus* has been found in both hay (Shadmi, et al., 1974) and silage (Cole, et al., 1977). The silage was found to contain fumigaclavine A and C and several fumitremorgens. Symptoms included generalized deterioration typical of protein deficiency, malnutrition, diarrhea, irritability, abnormal behavior and occasional death. The hay was fed to goats and rats and resulted in retarded growth and histopathological changes in the livers and kidneys.

Sterigmatocystin is primarily produced by *Aspergillus versicolor* and has been observed as a primary mycotoxin produced by *Aspergillus* on cereal grains in western Canada (Mills and Abramson, 1986). While it is thought to be infrequent at toxic levels in the U.S., it was detected in a grain mixture and associated with bloody diarrhea and cow deaths in a field case in Tennessee (Vesonder and Horn, 1985).

*Aspergillus ochraceus* was implicated as producing ochratoxin A associated with abortions in cattle consuming moldy alfalfa hay (Still, et al, 1971). Lacey (1991) has reviewed other cases of potential toxicities associated with *aspergillus* molds.

### **Fusarium molds**

Fumonisin B<sub>1</sub> (FB<sub>1</sub>) was isolated by Gelderblom et al. (1988) and shown to be a cancer promoter. FB<sub>1</sub> has been shown to cause leukoencephalomalacia in horses (Marasas, et al., 1988), pulmonary edema

in swine (Harrison et al., 1990) and hepatotoxicity in rats (Gelderblom et al., 1991). A USDA, APHIS (1995) survey found an average of 6.9% of 1995 corn samples from Missouri, Iowa and Illinois to contain more than 5 ppm FB<sub>1</sub>. While FB<sub>1</sub> is thought to be much less potent in ruminants than monogastrics, work by Kriek et al. (1981) suggested that fumonisin was toxic to sheep. Osweiler et al., (1993) demonstrated that FB<sub>1</sub> in large amounts (148 ppm) can cause mild liver damage in cattle even when fed for a short term (31 days), but without an effect on feed intake or weight gain. Whitlow (unpublished) has demonstrated that FB<sub>1</sub> is toxic to dairy cattle. Fed for approximately 7 days prior to freshening and for 70 days thereafter, dietary FB<sub>1</sub> at 100 ppm significantly and dramatically reduced milk production (7 kg/cow/day) and increased serum enzymes levels indicative of liver disease. These results strongly suggest that FB<sub>1</sub> is toxic to dairy cattle at levels that are less toxic to beef cattle, or perhaps FB<sub>1</sub> interacts with other factors to produce different effects in beef and dairy cattle under different conditions. FB<sub>1</sub> carryover from feed to milk is thought to be negligible. Richard et al. (1996) fed fumonisin B<sub>1</sub> (about 75 ppm) to dairy cows and with no fumonisin B<sub>1</sub> or B<sub>2</sub> detectable in milk (detection limit of 5 ng/ml). Scott et al. (1994) have confirmed this observation.

Deoxynivalenol (DON) is the proper name for a commonly detected *Fusarium* produced mycotoxin often referred to as vomitoxin. Two independent Midwestern studies (Vesonder et al., 1978 and Côté et al., 1984) showed DON to be the primary mycotoxin associated with swine problems including feed refusals, diarrhea, emesis, reproductive failure, and deaths. In cattle, DON has been associated with reduced feed intake (Trenholm et al., 1985). Clinical data from 300 herds representing about 40,000 cow records showed that DON was associated with a loss in milk production but this study did not establish a cause and effect (Whitlow et al., 1991). DON may simply be a marker for problem feeds. Field observations by others help substantiate these observations (Gotlieb, 1997 and Seglar, 1997). Charmley et al. (1993), demonstrated a 13% (2.85 kg) numerical decrease in 4% fat corrected milk production (statistics not available), utilizing 18 midlactation dairy cows (average 19.5 kg milk) consuming diets shown to contain no common mycotoxins other than DON which was at levels of 2.7 to 6.4 ppm in treatment diets. While the decrease in actual milk production (1.35 kg) was not statistically significant, the decrease in fat test (3.92% vs. 3.04%) was significant. Noller et al., (1979) utilized 54 lactating dairy cows in a 21 day feeding experiment using corn grain contaminated with *Gibberella zeae* and containing 500 ppb of zearalenone. DON was probably present, but it was not analyzed directly. Grain harvested earlier from the same field was contaminated with DON at 12 to 13 ppm. Neither dry matter intake nor milk production (average 22.9 kg) was affected by additions of contaminated grain to the diet. However, compared with controls, cows that received contaminated grain, gained significantly less weight. At 10% of the diet (about 1.25 ppm DON and 50 ppb ZEN in the diet) daily gains were 0.60 lb less and at 20% of the diet (about 2.50 ppm DON and 100 ppb ZEN in the diet) daily gains were 0.85 lb less than controls. DiCostanzo et al., (1995a) cites results by Ingalls (1994) where lactating dairy cows were fed 0, 3.6 10.9 and 14.6 ppm of DON for 21 days, without an apparent effect on feed intake or milk production which averaged about 30 kg daily. Beef cattle and sheep appear to tolerate relatively large amounts of DON without obvious deleterious effects (DeHaan et al., 1984, Nelson et al., 1984, DiCostanzo et al., 1995b, Boland et al., 1994, and Windels et al., 1995).

Zearalenone is a *Fusarium* produced mycotoxin, which elicits an estrogenic response in monogastrics (Sundlof and Strickland, 1986). However, ZEN is rapidly converted to  $\alpha$ - and  $\beta$ -zearalenol in rumen

cultures (Kiessling et al., 1984) and has been of less toxicity to ruminants. Ruminal degradation of ZEN was found to be about 30% in 48 hours (Kellela and Vasenius, 1982). A controlled study with cows fed up to 22 ppm ZEN showed no obvious effects except that corpora lutea were smaller in treated cows (Weaver et al., 1986b). In a similar study with heifers receiving about 13 ppm ZEN, conception rate was depressed about 25%; otherwise, no obvious effects were noted (Weaver et al., 1986a). Several case reports have related ZEN to an estrogenic response in ruminants and sometimes included abortions as a symptom (Kellela and Ettala, 1984, Khamis et al., 1986; Mirocha et al., 1968; Mirocha et al., 1974; and Roine et al., 1971). Other cattle responses may include vaginitis, vaginal secretions, poor reproductive performance and mammary gland enlargement of virgin heifers. In a field study, (Coppock et al., 1990) diets with about 750 ppb ZEN and 500 ppb DON resulted in poor consumption, depressed milk production, diarrhea, and total reproductive failure. New Zealand workers (Towers, et al., 1995a, Towers, et al., 1995b, Sprosen and Towers, 1995, and Smith et al., 1995) have related urinary zearalenone and zearalenone metabolites (zearalenone, zearalanone,  $\alpha$ - and  $\beta$ -zearalenol and  $\alpha$ - and  $\beta$ -zearalanol) which they refer to as “zearalenone” to intake of “zearalenone” and to reproductive disorders in sheep and dairy cattle. In sheep, “zearalenone” was related to lower conception, reduced ovulation, and increased twinning rates. With dairy cattle, herds with low fertility were found to have higher levels of blood and urinary “zearalenone” and consumed pastures containing higher levels of “zearalenone”. In addition, individual cows within herds were examined by palpation and those that were determined to be cycling had lower blood “zearalenone” levels than did cows that were not cycling. Differences in “zearalenone” levels were attributed to selective grazing behavior. The reproductive problems in dairy cattle were noted with “zearalenone” concentrations of about 400 ppb in the pasture samples.

T-2 toxin, a very potent *Fusarium* produced mycotoxin, has been associated with gastroenteritis, intestinal hemorrhages (Petrie et al., 1977 and Mirocha et al., 1976) and death (Hsu et al., 1972 and Kosuri et al., 1970). 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. Serum immunoglobulins and certain 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. A calf intubated with T-2 developed severe depression, hindquarter ataxia, knuckling of the rear feet, listlessness and anorexia (Weaver et al, 1980). Data with cattle are limited, but, the toxicity of T-2 toxin in laboratory animals is well documented (Wannemacher et al, 1991). In a field observation with Jersey cows, we observed a 7 lb decrease in milk production coinciding with diarrhea and apparently associated with 350 ppb of T-2 toxin in the dietary dry matter. Cows responded to a clay type feed additive. At similar levels in other herds, we have associated T-2 toxin with an increased incidence of disease in early lactation, poor adjustment of fresh cows to the lactation ration, excessive weight loss, increased death loss and a loss in milk production.

Diacetoxyscirpenol is a *Fusarium* produced mycotoxin. It may occur along with T-2 toxin and is thought to produce similar symptoms of toxicity.

### **Penicillium molds**

Ochratoxin, produced primarily by a *Penicillium* mold but also by certain *Aspergillus* molds, has been

reported to affect cattle (Vough and Glick, 1993), but it is rapidly degraded in the rumen and thus thought to be of little consequence unless consumed by young pre-ruminant calves (Sreemannarayana et al., 1988). However, high-concentrate diets reduce ochratoxin degradation in the rumen.

Patulin is produced by *Penicillium*, *Aspergillus*, and *Byssochlamys* molds and may be found in silage (Dutton, et al., 1984 and Hacking and Rosser, 1981). Patulin has been incriminated as a possible toxin in Europe and New Zealand (Lacey, 1991).

PR toxin, produced by *Penicillium roquefortii*, has been found in silage (Hacking and Rosser, 1981) and was the suspected vector in a case study with symptoms of abortion and retained placenta (Still et al., 1972).

*Penicillium* or *Aspergillus* molds growing on sweet clover or sweet vernal grass can cause a conversion of natural compounds in the plant to dicoumarol. Dicoumarol interferes with the function of vitamin K, resulting in a hemorrhagic syndrome. Moldy sweet clover poisoning is discussed by Radostits, et al., (1980).

### **Others**

*Stachybotrys* toxicosis has been observed when the mold occurs on hay and straw but it is thought to be rarely associated with dairy cattle problems in the U.S. This mold was associated with deaths of thousands of horses in Russia during the 1930's. The mold produces a large number of spores, resulting in sooty black spots on the forage. There have been several mycotoxins isolated and identified (Eppley, 1977).

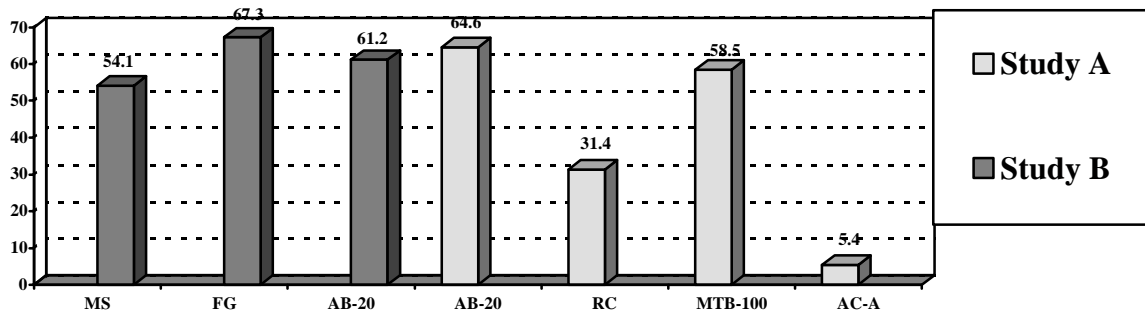
There are other mycotoxins that affect ruminants. Some are thought to occur less frequently or to be less potent, but in many cases, there is a lack of information.

### **Treatment**

Some additives may be beneficial in reducing mycotoxins because they are effective in reducing mold growth. Ammonia, propionic acid, microbial, and enzymatic silage additives have all shown some effectiveness as mold inhibitors. Additives to enhance fermentation, may be added at ensiling. Mold growth inhibitors such as propionic acid may be helpful as a surface treatment when capping off the silo or daily after silage feed-out to reduce molding of the exposed silage feeding surface. If unacceptably high levels of mycotoxins occur, dilution or removal of the contaminated feed is preferable; however, it is usually impossible to completely replace major forage ingredients. While dilution is sometimes a viable practice to reduce exposure, reduced feeding of silage could result in such a slow feedout that mycotoxin problems within the silage increase. Ammoniation of grains can destroy some mycotoxins, but there is no practical method to detoxify affected forages already in storage. Increasing nutrients such as protein, energy and antioxidant nutrients may be advisable (Brucato et al., 1986, Chandler, 1992). Adsorbent materials such as clays (bentonites) added to contaminated diets fed to rats, poultry, swine and cattle have helped reduce the effects of mycotoxins

(Diaz et al., 1997; Galey et al., 1987; Harvey, 1988; Lindemann et al., 1991; Scheideler, 1990; Hayes, 1990 and Smith, 1980 and 1984). In most cases, clay was added to the diet at about 1%. Other absorbent materials such as charcoal (Galvano, et al., 1996), and glucomannans at 0.05% of diet dry matter have shown effective in reducing aflatoxin in milk. Some of these results are shown in figure 1 (Diaz, et al., 1999).

Figure 1. Effect of Feed Additives on Reduction of Milk Aflatoxin Residues



**MS**, mycosorb, a sodium bentonite fed at 1% of DM (American Colloid 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. 1999. Journal of Dairy Science 82(6):1828

### Summary

Until recent years, it was thought that mycotoxins were relatively harmless to mature cattle because mycotoxins are partially destroyed in the rumen. It is now known that mycotoxins occur frequently in dairy cattle feedstuffs and in general affect production, reproduction, and health of dairy cattle. However, we need a better understanding of why mycotoxins occur, when to expect them, how to prevent their occurrence and how to deal with their presence. We need to better understand their toxicity to dairy cattle, their interactions with other mycotoxins and with other factors such as nutrients, or stresses such as disease organisms or environmental stress. Diagnosis of a mycotoxicosis is difficult and not highly definitive. We need better methods to monitor animal exposures to mycotoxins and to diagnose their contribution to unsatisfactory conditions. And, we must learn how to prevent toxicities and to treat those, which do occur.

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