



Mycotoxin Effects in Dairy Cattle

L.W. Whitlow and W.M. Hagler, Jr.

North Carolina State University, Raleigh, NC

Summary

- Molds are filamentous (fuzzy or dusty appearing) 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.
- A wide range of different molds produce poisons called mycotoxins that affect animals when they consume contaminated feeds.
- A mycotoxin poisoning is called mycotoxicosis.
- Mycotoxins are of constant concern, but some growing seasons produce increased problems.
- The U.N.'s Food and Agriculture Organization (FAO) estimated that worldwide, about 25% of crops are affected annually with mycotoxins.
- Molds are present throughout the environment and therefore, mycotoxins can be formed on crops in the field, during harvest, or during storage, processing, or feeding.
- Spores are in the soil and in plant debris and lie ready to infect the growing plant in the field.
- Fungal 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.
- *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.
- Mycotoxin-contaminated feeds often contain multiple mycotoxins.
- Management of crop production can reduce, but not prevent, the occurrence and concentrations of mycotoxins.
- Feed and feeding management practices can reduce the production and impact of mycotoxins.
- Experimentally, mycotoxin binders have been effective at reducing mycotoxin effects.

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*, certain species of *Fusarium* and others. *Aspergillus fumigatus* is known to cause mycotic pneumonia, mastitis and abortions and has been recently 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 and silage. While healthy cows with an active immune system are more resistant to mycotic infections, dairy cows in early lactation are immune suppressed and HBS is more likely in fresh cows (Puntenney et al., 2003). It is theorized that with a mycosis, mycotoxins produced by the invading fungi can suppress immunity; therefore increasing the infectivity of the fungus. *A. fumigatus* produces several mycotoxins, including tremorgens that are toxic to cattle and gliotoxin. Gliotoxin, an immune suppressant, 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. Perhaps reducing animal exposure to mycotoxins and moldy feeds 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).

Mycotoxin Effects

Mycotoxins can increase the incidence of disease and reduce production efficiency in cattle. 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.

Mycotoxins exert their effects through several means:

1. Reduced intake or feed refusal
2. Reduced nutrient absorption and impaired metabolism
3. Altered endocrine and exocrine systems
4. Suppressed immune function
5. Altered microbial growth

Recognition of the impact of mycotoxins on animal production has been limited by the difficulty of diagnosis. 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 (Schiefer, 1990):

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, increased incidence of disease or atypical diseases may be observed.
6. Responses to added dietary adsorbents or dilution of the contaminated feed may help in diagnosis.
7. Feed analyses should be performed, but accurate sampling is a major problem

Symptoms are often nonspecific and may be wide-ranging. Symptoms result from a progression of effects, or of opportunistic diseases, making a diagnosis difficult or impossible because of the complex clinical results with a wide diversity of symptoms. Symptoms 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 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.

The FDA regulatory control program for mycotoxins is discussed by Wood and Trucksess (1999).

Toxicity of Individual Mycotoxins

Aflatoxin

Aflatoxins are extremely toxic, mutagenic, and carcinogenic compounds produced by *Aspergillus flavus* and *A. parasiticus*. Aflatoxin B1 is a carcinogen and 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% 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 (Diaz et al., 2004).

Symptoms of acute aflatoxicosis in mammals include: inappetence, 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 (Diekmann 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 milk production was reduced in cows consuming impure aflatoxin produced by culture, but production was not significantly affected by equal amounts of pure aflatoxin.

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 seen in 5% of corn grain in the Midwestern U.S. (Russell et al., 1991). The General Accounting Office 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-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). DON has been associated with reduced flow of utilizable protein to the duodenum (Danicke et al., 2005), and appears

to affect rumen fermentation (Seeling et al., 2006). 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, perhaps due to the presence of multiple mycotoxins in naturally contaminated feeds. These mycotoxins can interact to produce 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, which earlier was 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.

The U.S. Food and Drug Administration advisory level for deoxynivalenol in wheat and wheat derived products destined for dairy cattle diets is for no more than 5 ppm DON and to be used at levels below 40%.

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. Effects of T-2 are less well established in cattle than in laboratory animals. In dairy cattle, T-2 has been 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 basis of T-2 reduced

immunity is reduced protein synthesis. Guidelines for T-2 toxin are not established, but avoiding levels above 100 ppb may be reasonable.

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. 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 (Kellela 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. In this study, reproductive problems in dairy cattle were associated with dietary ZEA concentrations of about 400 ppb.

Fumonisin (FB)

Fumonisin B1 produced by *F. verticillioides*, was first isolated in 1988. It causes leucoencephalomalacia in horses, pulmonary edema in swine, and hepatotoxicity in rats. It is

carcinogenic in rats and mice and is thought to be a promoter of esophageal cancer in humans (Rheeder et al., 1992). Fumonisin is 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 FB1 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 lymphocyte blastogenesis and elevated enzymes indicative of liver damage. 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 B1. 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.

U.S. Food and Drug Administration guidance for industry on fumonisin levels in human foods and animal feeds recommends that corn used for

dairy purposes should contain no more than 30 ppm of total fumonisins and limited to 50% of the diet.

PR toxin

PR toxin is one of the several 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 has been suggested as the causative agent with moldy corn silage problems (Seglar 1997 and Sumarah et al., 2005). PR toxin caused acute toxicity in mice, rats and cats by increasing capillary permeability resulting in direct damage to the lungs, heart, liver and kidneys (Chen et al., 1982) and was the suspected vector in a case study with symptoms of abortion and retained placenta (Still et al., 1972).

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 producing 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).

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Notes
