Mycotoxin Contamination of Feedstuffs - An Additional Stress Factor for Dairy Cattle

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Mycotoxins are toxic secondary metabolites produced by fungi (molds). These fungal toxins are chemically diverse -- representing a variety of chemical families – and range in molecular weight from ca. 200 to 500. There are hundreds of mycotoxins known, but few have been extensively researched and even fewer have good methods of analysis available. Fungi on crops produce them in the field, during handling, and in storage. A practical definition of a mycotoxin is a fungal metabolite that causes an undesirable effect when animals or humans are exposed. Usually, exposure is through consumption of contaminated feedstuffs or foods. Mycotoxicoses are diseases caused by exposure to foods or feeds contaminated with mycotoxins (Nelson et al., 1993). Mycotoxins exhibit a variety of biological effects in animals such as: liver and kidney toxicity, central nervous system abnormalities, estrogenic responses and others.

Mold Growth and Mycotoxin Formation

Perhaps the major mycotoxin-producing fungal genera, in terms of research in North America, are *Aspergillus, Fusarium, and Penicillium*. Many species of these fungi produce mycotoxins in feedstuffs. Molds are fungi that grow in multicellular colonies, as compared with yeasts that are single cellular fungi. Molds can grow and mycotoxins can be produced either pre-harvest or post-harvest, during storage, transport, processing, or feeding. 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. In general, environmental conditions– heat, water, and insect damage –cause plant stress and predispose plants in the field to mycotoxin contamination. Because feedstuffs can be contaminated pre-harvest, control of additional mold growth and mycotoxin formation is dependent on storage management. After harvest, temperature, moisture content, and insect activity are the major factors influencing mycotoxin contamination of feed grains and foods (Coulumbe, 1993). Molds grow over a temperature range of 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). While yeasts require free water, molds can grow on a dry surface (Lacey, 1991). Mold can grow on feeds containing more than 12% to 13% moisture. In wet feeds such as silage, molds will grow if oxygen is available and the pH is suitable. Because most molds are aerobic, high moisture concentrations that exclude adequate oxygen can prevent mold growth. The conditions most suitable for mold growth may not be the optimum conditions for mycotoxin formation in the laboratory. For example, the *Fusarium* molds associated with alimentary toxic aleukia have been reported to grow prolifically at 25 to 30° C without producing much mycotoxin, but at near-freezing temperatures, large quantities of mycotoxins were produced with minimal mold growth (Joffe, 1986). Field applications of fungicides may reduce mold growth reducing production of mycotoxins, however, the stress or shock of the fungicide to the mold organism may cause increased mycotoxin production (Boyacioglu et al., 1992 and Gareis and Ceynowa, 1994).

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Aspergillus species normally grow at lower water activities and at higher temperatures than do the *Fusarium* species. Therefore, *Aspergillus flavus* and aflatoxin are more likely in corn grown in the heat and drought stress associated with warmer climates. Aflatoxin contamination is enhanced by insect damage before and after harvest. *Penicillium* species grow at relatively low water activities and low temperatures and are widespread in occurrence. Because both *Aspergillus* and *Penicillium* can grow at low water activities, they are considered storage fungi (Christensen et al., 1977).

The *Fusarium* species are generally considered to be field fungi and were thought to proliferate before harvest (Christensen et al., 1977). However, *Fusarium* species may also grow and produce mycotoxins under certain storage conditions. In corn, *Fusarium* molds are associated with ear rot and stalk rot, and in small grains, they are associated with diseases such as head blight (scab). In wheat, excessive 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 (Trenholm et al., 1988).

Mycotoxin Occurrence

Worldwide, approximately 25% of crops are affected by mycotoxins annually (CAST, 1989). In North Carolina (Table 1), feed samples submitted by North Carolina farmers over a nine-year period indicate that mycotoxins in feeds including corn silage and corn grain occur commonly at unsuitable concentrations (Whitlow et al., 1998). Occurrence and concentrations were variable by year, which is expected because of the annual variation in weather conditions and plant stresses known to affect mycotoxin formation (Coulumbe, 1993). It is concluded that mycotoxins occur frequently in a variety of feedstuffs and are routinely fed to animals.

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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 (Whitlow et al., 1998).

Aflatoxin >10 ppb			Deoxynivalenol >50ppb			Zearalenone >70ppb			T-2 Toxin >50ppb			Fumonisin >1ppm	
n	% Pos	mean ± s.d.	n	% Pos	mean ± s.d.	n	% Pos	mean ± s.d.	n	% Pos	mean ± s.d.	n	% Pos
CORN SILAGE													
461	8	28 ±19	778	66	1991 ± 2878	487	30	525 ± 799	717	7	569 ± 830	63	37
CORN GRAIN													
231	9	170 ± 606	362	70	1504 ± 2550	219	11	206 ± 175	353	6	569 ± 690	37	60
ALL FEEDS													
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 given concentrations

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

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Mycotoxin Effects

Although the potentially harmful effects of feeding moldy grain and foods has been known for many years (Matossian, 1989), mycotoxicology, the study of mycotoxins, began in earnest in 1960 with the outbreak of Turkey-X disease in the United Kingdom. This outbreak was linked to peanut meal imported from Brazil (Sargeant et al., 1961). Because of an intensive mulitidiciplinary research effort a blue-fluorescent toxin was isolated and mycelia of *Aspergillus flavus* was observed. *A. flavus* was soon shown to produce the same toxic compound(s) found in the toxic peanut meal. The toxin was characterized chemically and biologically and was given the trivial name, aflatoxin. Aflatoxin was shown to be very toxic and carcinogenic in some of the test animal species used and that it resulted in a toxic metabolite, aflatoxin M_1, in milk of dairy cows (Allcroft and Carnaghan, 1962; 1963).

The discovery of aflatoxin and elucidation of some of its effects led to research on other livestock health and production problems linked with moldy feedstuffs and to the discovery of additional mycotoxins produced by other fungi. In dairy cattle, swine, and poultry, mycotoxin contamination of feeds reduces growth, milk production, egg production, lower reproduction, and immunity (Diekman and Green, 1992). Mycotoxins have also been involved in outbreaks of human diseases (CAST, 1989; Hayes, 1980; Joffe, 1986).

Mycotoxins exert their effects through three primary mechanisms:

(1) A reduction in amount of nutrients available for use by the animal. This occurs in a multifactorial process. First, an alteration in nutrient content of feed may occur during the molding process. Mold growth can reduce the content of nutrients such as vitamins and amino acids such as lysine in feedstuffs (Kao and Robinson, 1972). The energy value of feeds is usually reduced by mold growth. Second, some mycotoxins reduce feed intake which lowers nutrient intake. Third, a mycotoxin-produced irritation to the digestive tract can reduce nutrient absorption, and forth, certain mycotoxins interfere with normal nutrient metabolism such as the inhibition of protein synthesis by T-2 toxin.
(2) Effects on the endocrine and exocrine systems. An example is the effect of zearalenone on reproductive performance due to its estrogenic effects. Zearalenone's estrogenic effect results from the affinity of zearalenone and its derivatives to bind with the animal's estrogen receptors (Klang et al., 1978).

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(3) Suppression of the immune system. The effects of mycotoxins on immunity have been reviewed (Sharma, 1993). Trichothecenes such as DON and T-2 toxin reduce immunity by inhibiting protein synthesis and thus cell proliferation. Some mycotoxins are cytotoxic to lymphocytes in vitro. Corticosteriods produced in response to stress also reduces immune function.

Mycotoxins can increase incidence of disease and reduce production efficiency. In the field, animals experiencing a mycotoxicosis may exhibit a few or many symptoms including: digestive disorders, reduced feed consumption, unthriftiness, rough hair coat or abnormal feathering, undernourished appearance, subnormal production, impaired reproduction, and/or a mixed infectious disease profile. Some of the symptoms observed with a mycotoxicosis may be secondary, resulting from an opportunistic disease which is present because of immune suppression. Therefore, the progression and diversity of symptoms are confusing and diagnosis is difficult (Hesseltine, 1986; Schiefer, 1990). Diagnosis is further complicated by a lack of research, by a lack of feed analyses, by nonspecific symptoms, by interactions with other stress factors. A definitive diagnosis of a mycotoxicosis cannot be made directly from symptoms, specific tissue damage, or even feed analyses. Previous experience with mycotoxin-affected herds or flocks increases the probability of recognizing a mycotoxicosis. A process of elimination of other factors, coupled with feed analyses and responses to treatments can help identify a mycotoxicosis. Regardless of the difficulty of diagnosis, mycotoxins should be considered as a possible cause of production and health problems when such symptoms exist and problems are not attributable to other typical causes (Schiefer, 1990).

Safe Levels of Mycotoxins

Some of the same factors that make diagnosis difficult also contribute to the difficulty of establishing levels of safety. These include lack of research, sensitivity differences by animal species, imprecision in sampling and analysis, the large number of potential mycotoxins, interactions with other mycotoxins and interactions with the stresses of environment and production (Hamilton, 1984, and Schaeffer and Hamilton, 1991). Mycotoxin effects are also moderated by factors such as gender, age, diet, and duration of exposure. It is therefore impossible to provide guidelines about definitive levels of mycotoxins that will result in a field mycotoxicoses. Recommendations that provide mycotoxin levels of concern are intended to present the lower levels of mycotoxins that have been associated with mycotoxicoses.

Naturally contaminated feeds are more toxic than feeds with the same level of a pure mycotoxin supplemented into the diet. A generally accepted explanation for this observation is the possible

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presence of more than one mycotoxin contained in a naturally moldy feedstuff. 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 a diet containing pure added DON was less toxic than diets with similar concentrations of DON, which were supplied from naturally contaminated feeds. Smith and MacDonald (1991) have suggested that fusaric acid, produced by many species of *Fusarium*, occurs along with DON to produce more severe symptoms. Lillehoj and Ceigler (1975) give an example where penicillic acid and citrinin were innocuous in laboratory animals when administered alone but were 100% lethal when given in combination. Jones et al. (1982) demonstrated that productivity losses in commercial broiler operations can occur when aflatoxin concentrations are below those shown by controlled research to be of concern in laboratory situations. These studies strongly suggest the presence of other unidentified mycotoxins in naturally contaminated feeds. 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 produced multiple mycotoxins. Because animals are fed a blend of feedstuffs and because molds produce an array of mycotoxins, many mycotoxin interactions are possible.

Interactions with other stress factors make recommendations difficult. Animals under environmental or production stress may show the more pronounced symptoms. It is clearly shown that there is a temperature interaction with fescue toxicity such that more pronounced symptoms are expressed during heat stress (Bacon, 1995). Fumonisin at 100 ppm has been shown to reduce milk production in dairy cattle (Diaz et al., 2000), and in a separate study to not affect average daily gain in beef cattle fed 148 ppm (Osweiler et al., 1993). While this contrast may reflect a difference in the duration of feeding or otherwise, it may also suggest differences due to greater stress in early lactation dairy cattle as compared with beef cattle.

Because of partial degradation in the rumen, mycotoxins are less toxic to cattle than to most other animals. However mycotoxins are not completely degraded and some of the degradation products remain toxic (Kiessling et al., 1984). Extent of ruminal degradation appears to be variable. It is speculated that feeding situations resulting in a faster rate of ruminal feed passage or a low population of protozoa in the rumen may reduce mycotoxin degradation in the rumen. Ruminal degradation of mycotoxins appears to be more dependent on protozoal than bacterial activity (Kiessling et al., 1984).

The known dietary factors which interact with mycotoxins include nutrients such as fat, protein, fiber, vitamins and minerals (Brucato et al., 1986, Coffey et al., 1989, Smith et al., 1971). Dietary pellet binders (clay) and other additives such as glucomannans bind some mycotoxins

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reducing exposure of the animal (Diaz et al., 1999). Galvano et al., 2001, has recently reviewed the interaction of mycotoxins with dietary factors such as antioxidants, food components, medicinal herbs, plant extracts, and mineral and biological binding agents. Certain of these dietary factors alter the response of animals to mycotoxins, resulting in variable responses in the field but also offering some opportunity to modulate the toxicity to animals. Dietary approaches hold great potential for protecting animals against the effects of mycotoxins and for minimizing the possibility of mycotoxins in the human food supply.

Economic losses

Economic losses from mycotoxicoses in agriculture are due to effects on livestock productivity, losses in crops, and the costs and effects of regulatory programs directed toward mycotoxins. It was estimated in 1989 that about 2% of the U.S. corn crop is affected by mycotoxins, and the impact may be devastating in some areas (CAST, 1989). The estimated 25% contamination of the world's annual crops (CAST, 1989) would extrapolate to billions of dollars (Trail et al., 1995a). However, these estimates were generated before information about the widespread occurrence of fumonisin in corn (Anon., 1995).

Sometimes mycotoxins occur at concentrations high enough to cause major losses in health and performance of animals. However, mycotoxins are more usually at lower levels that result in interactions with other stressors to cause subclinical losses in performance, increases in incidence of disease and reduced reproductive performance. To the animal producer, these subclinical losses are of greater economic importance than losses from acute effects.

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). Aflatoxin contamination of corn, peanuts, tree nuts, cottonseed, and other commodities is a continuing worldwide problem. Toxigenic *A. flavus* isolates produce aflatoxins B1, and B2 and toxigenic *A. parasiticus* isolates produce aflatoxins B1, B2, G1, and G2 (Cotty et al., 1994).

Under certain conditions, *A. flavus* also produces sclerotia, or resting bodies, which contain indole alkaloids such as aflatrem (Wicklow, 1983). Cyclopiazonic acid (CPA), a toxic indole tetramic acid, is also produced by *A. flavus* (CAST, 1989). The role of these and other toxins produced by *A. flavus* in aflatoxicoses is not known.

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A. flavus is the predominant fungus in aflatoxin-contaminated corn and cottonseed while *A. parasiticus* is probably more common in peanuts than on corn (Davis and Diener, 1983). *A. flavus* and *A. parasiticus* are temperature-tolerant fungi (Davis and Diener, 1983) and can be selectively isolated on a high salt culture medium incubated at 37 C.

Before the 1970s, most of the aflatoxin in corn was generally believed to originate after harvest. Improperly stored corn can and does become contaminated with aflatoxin (Lillehoj and Fennell, 1975; Shotwell et al., 1975). However, after aflatoxin was identified in corn before harvest, it has become clear that most of the aflatoxin problem in corn originates in the field. Growth of *A. flavus* can occur at 86-87% equilibrium relative humidity (RH) (Davis and Diener, 1983). Field infection of corn with *A. flavus* (Wicklow, 1983) is expected when temperatures, including nighttime temperatures, are high and there is drought stress. Growth conditions in the southern U.S. result in routine aflatoxin contamination of crops, but aflatoxin can be found in crops grown in other regions in years when weather conditions are conducive. For example, 8% of samples of Midwestern U.S. corn grain from the 1988 drought season contained aflatoxin (Russell et al., 1991).

Corn is susceptible to *A. flavus* infection via the silks (Marsh and Payne, 1984) and these stress conditions at the time of anthesis (pollination) lead to preharvest aflatoxin contamination in corn. *A. flavus* spores as inoculum are plentiful at this time. In North Carolina, insect activity appears less important in the events leading to aflatoxin contamination of corn than it appears to be in Georgia (Payne, 1983).

Aflatoxin is a greater problem in cottonseed grown in the Southwestern U.S. than in the Southeastern U.S. (Ashworth et al., 1969). The complex effects of relative humidity, temperature, precipitation, and their daily variations may interact to produce conditions conducive to *A. flavus* infection and aflatoxin production in the Southwest (Ashworth et al., 1969). Early harvest and a decrease in late-season irrigation may reduce contamination (Russell et al., 1976). Experimentally, the use of spores of nontoxigenic *A. flavus* isolates in Southwestern cotton fields has resulted in greatly reduced aflatoxin levels in cottonseed (Cotty et al., 1994). Improperly stored cottonseed are susceptible to mycotoxin contamination if mold activity is allowed.

Aflatoxin lowers resistance to diseases and interferes with vaccine-induced immunity in livestock (Diekman and Green, 1992). Suppression of immunity by aflatoxin B1 has been demonstrated in turkeys, chickens, pigs, mice, guinea pigs, and rabbits (Sharma, 1993). Swine, turkeys, ducks,

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and rainbow trout are very susceptible to aflatoxin. Broiler chickens are resistant compared to these, but much more susceptible to aflatoxin than layer-type chickens. Pale, friable, fatty livers may be evident in acute aflatoxicosis in poultry.

Symptoms of acute aflatoxicosis in mammals include: inappetance, lethargy, ataxia, rough hair coat, and pale, enlarged fatty livers. Symptoms of chronic aflatoxin exposure include reduced feed efficiency and milk production, icterus, and decreased appetite (Nibbelink, 1986). If problems are found and analysis shows aflatoxin, the feed should immediately be replaced with fresh feed. (Nibbelink, 1986). Reduced growth rate may be the only clue for chronic aflatoxicosis and other mycotoxicoses (Raisbeck et al., 1991; Pier, 1992). The mechanism by which aflatoxins reduce growth rate is probably related to disturbances in protein, carbohydrate and lipid metabolism (Cheeke and Shull, 1985).

Several reports have shown that differences in resistance to aflatoxin exist in different breeds and strains of chickens (Smith and Hamilton, 1970; Washburn et al., 1978; Lanza et al., 1982). Marks and Wyatt (1980) using Japanese quail demonstrated the feasibility of breeding for resistance to aflatoxin. Manning et al. (1990) developed a line of chickens resistant to acute and chronic dietary aflatoxin exposure. This was accomplished after five generations of selecting for resistance to a single oral dose of aflatoxin.

Depending on interactions with other factors, aflatoxin concentrations as low as 100 ppb may be toxin to beef cattle, however the toxic level is generally considered to be between 300 to 700 ppb. Garrett et al. (1968) showed an effect on weight gain and intake with diets containing 700 ppb aflatoxin, but not with 300 ppb. Trends in the data suggest that toxicity may occur at the lower concentrations of aflatoxin. If increases in liver weights are used as the criteria for toxicity, then 100 ppb would be considered toxic to beef cattle. Guthrie (1979) showed a decline in reproductive efficiency when lactating dairy cattle in a field situation were consuming 120 ppb aflatoxin. When cows were changed to an aflatoxin free diet, milk production increased over 25%. Patterson and Anderson (1982) and Masri et al. (1969) also suggest that 100 ppb may reduce milk production. Applebaum et al. (1982) showed that impure aflatoxin produced by culture reduced production while equal amounts of pure aflatoxin did not.

Humans are exposed directly to aflatoxin and other mycotoxins through consumption of contaminated foods. Handling contaminated feed can result in exposure of mycotoxins through the skin and by inhalation (Schiefer, 1990). Indirect exposure of humans to aflatoxins occurs through foods -- primarily milk, liver, and eggs -- derived from animals that consume contaminated feeds (Hayes, 1980).

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Aflatoxin B1 is excreted into milk of lactating dairy cows primarily in the form of aflatoxin M_1 with residues approximately equal to 1.7% of the dietary level with a range of 1% to 3% (Van Egmond, 1989). Aflatoxin appears in the milk within hours of consumption and returns to baseline levels within two to three days after removal from the diet (Frobish et al., 1986). A concentration of 20 ppb of aflatoxin B1, in the TMR dry matter of lactating dairy cattle, will result in M1 levels in milk below FDA's action level of 0.5 ppb. However, the European Union and several other countries presently have an action level of 0.05 ppb in milk and milk products. It is our understanding that some milk processors in the U.S. reject milk containing aflatoxin at concentrations below the FDA's action level, 0.5 ppb. Assumed safe feeding levels may result in milk concentrations above the FDA action level because absolute concentrations of mycotoxins in the feed are difficult to determine, concentrations may not uniform throughout a lot of feed and concentrations can change over time.

The Food and Drug Administration has established nonbinding action levels as informal guidelines for their enforcement of aflatoxin in feedstuffs (Wood and Trucksess, 1998). Feedstuffs designated for immature animals and dairy cattle always have an action level of 20 ppb. With the following exceptions, all feedstuffs have an action level of 20 ppb. For cottonseed meal used as a feed ingredient for beef cattle, swine and poultry, the action level is 300 ppb. For corn grain and peanut products, action levels are tiered by usage and targeted at interstate shipments. When designated for breeding beef cattle, swine and mature poultry, the action level is 100 ppb. When designated for finishing swine (100 lb or greater) the action level is 200 ppb. When designated for finishing beef cattle the action level is 300 ppb. Blending contaminated ingredients with uncontaminated ingredients with the purpose of reducing aflatoxin concentrations is not allowed.

Zearalenone

Zearalenone and zearalenol are estrogenic metabolites of several species of *Fusarium*. Chemically, zearalenone (ZEN) is a resorcylic acid lactone which does not have actual toxicity. Zearalenone is the cause of hyperestrogenism, the estrogenic syndrome, in swine. *Fusarium graminearum* is the major ZEN-producing fungus of the *Fusarium* species that cause corn ear and stalk rots, but other species of *Fusarium* produce ZEN, as well as other mycotoxins. (Christensen et al., 1988). Zearalenone has been reported to occur in corn, other grains, and silage in many areas of the world. Weathered soybeans have also been reported to be contaminated with ZEN (Hagler et al., 1989). ZEN is also found in wheat, barley, oats, sorghum,

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sesame seed, hay, and silages. Conditions exacerbating ZEN accumulation in corn include weather which holds moisture contents at 22-25%, or delayed harvest (Abbas et al., 1988).

Swine appear to be most susceptible to ZEN (Diekman and Green 1992). In pre-pubertal gilts, swollen vulvae appear; this can progress to vaginal or rectal prolapse (Friend et al., 1990); internally, enlarged, swollen, distorted uteri, and shrunken ovaries are observed. (Friend et al., 1990). Litter size may also be reduced. Hyperestrogenism occurs when contamination of ZEN is as low as 0.1 ppm (Mirocha et al., 1977). Young male pigs exposed to ZEN undergo symptoms of "feminization", such as enlarged nipples, testicular atrophy, and swollen prepuce (Newberne,1987).

Broiler chicks and laying hens are apparently not susceptible to ZEN, even at very high dietary concentrations. Turkeys, on the other hand, at the high dietary levels of 300 ppm, develop greatly enlarged vents within four days with no other gross effects noted (Christensen et al., 1988).

ZEN is rapidly converted to ∀- and ∃-zearalenol in rumen cultures (Kiessling et al., 1984). ∀-Zeararalenol is ca. four-fold more estrogenic in rats than ZEN, while ∃-zearalenol is about equal in strength to ZEN (Hagler et al., 1979). However, ZEN has been considered of less importance to ruminants. Ruminal conversion of ZEN was found to be about 30% in 48 hours (Kellela and Vasenius, 1982). A controlled study with nonlactating cows fed up to 500 mg of ZEN (dietary concentrations of about 40 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 250 mg of ZEN by gelatin capsule (dietary concentrations of about 40 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 measured urinary ZEN and its metabolites (ZEN, zearalanone, ∀- and ∃-zearalenol and ∀- and ∃-zearalanol) to successfully estimate ZEN intake. ZEN intake (and urinary ZEN) was predictive of reproductive disorders in sheep and dairy cattle. In sheep, "zearalenone" was related to lower conception, reduced ovulation, increased twinning rates and a 10 to 20 % decline in fertility of ewes. With dairy cattle, herds with low fertility had higher

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levels of blood and urinary "zearalenone" originating from pastures containing higher ZEN. Individual cows within herds examined by palpation and determined to be cycling had lower blood ZEN levels than did cows that were not cycling. The reproductive problems in dairy cattle were associated with ZEN concentrations of about 400 ppb in the pasture samples.

Trichothecenes

Trichothecenes are a family of 200 - 300 related compounds that apparently exert their toxicity through protein synthesis inhibition at the ribosomal level. Several species of *Fusarium* and related genera produce trichothecenes. T-2 toxin, diacetoxyscirpenol (DAS), and deoxynivalenol (DON), are commonly found in agricultural commodities. (Desjardins et al., 1993). However, except for DON, it appears that most contamination with T-2 toxin and DAS occur post-harvest.

The toxic effects of trichothecenes include gastrointestinal effects such as vomiting, diarrhea, and bowel inflammation. Anemia, leukopenia, skin irritation, feed refusal, and abortion are also common. The trichothecenes, as a group, are immunosuppressive (Sharma, 1993).

Deoxynivalenol (DON, vomitoxin) causes feed refusal in swine. *F. graminearum* is a major producer of DON (Marasas et al., 1984). Wet, rainy, and humid weather at flowering promotes infection by *Fusarium*. The results are ear rot in corn and scab or head blight in sorghum, barley, wheat, oats and rye (Tuite et al., 1974). Minimum tillage and no tillage production are believed to increase the amount of disease in small grains and corn/wheat rotations because of increased inoculum survival on crop residue (Trenholm et al., 1988). DON occurs in cereal grains worldwide and can increase in stored grain with kernel moisture contents of 22 –25%.

One ppm or more of DON results in reduced feed intake in swine, resulting in lower weight gains. Two independent Midwestern field studies (Vesonder et al., 1978 and Côté et al., 1984) showed DON to be the primary mycotoxin associated with swine disorders including feed refusals, diarrhea, emesis, reproductive failure, and deaths. Vomiting has been reported in some outbreaks. Diets containing pure DON decrease feed consumption on a dose related basis (Marasas et al., 1984). Other mycotoxins co-occur with DON and Foster et al. (1986) found that DON concentration was not a good predictor of grain toxicity. Smith and McDonald (1991) have indicated that fusaric acid interacts with DON to produce the symptoms previously attributed to just DON.

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Chickens and turkeys apparently are not very susceptible to the effects of DON. Leghorn chickens showed no effect on weight gain from dietary levels of DON at 18 ppm (Kubena et al., 1987). A two-week study with poults fed 75 ppm DON revealed no effect on feed consumed or growth (McMillan and Moran, 1985). No residues of DON were found in meat or eggs in birds fed high levels of DON in several experiments (El-Banna et al., 1983; Kubena et al., 1987; Lun et al., 1986).

The impact of DON on dairy cattle is not established, but clinical data appear to show an association between DON contamination of diets and poor performance in dairy herds, but without establishing a cause and effect (Whitlow et al., 1994). DON may therefore be a marker for low-quality mycotoxin-contaminated feeds in these herds. Other field reports help substantiate an association of DON with poor performing dairy herds (Gotlieb, 1997 and Seglar, 1997). DON has been associated with reduced feed intake in nonlactating dairy cattle (Trenholm et al., 1985). There was a trend ($P<0.16$) for a 13% loss in 4% fat corrected milk in a study 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 approximately 0, 2.7 and 6.5 ppm in treatment diets (Charmley et al.,1993). Noller et al.(1979) utilized 54 lactating dairy cows in a 3 X 3 Latin Square experiment with 21 day feeding periods. *Gibberella zeae* (*F. graminearum*) infected corn was utilized to provide estimated concentrations of 0, 1650 and 3300 ppb DON and 0, 65 and 130 ppb of ZEN in three experimental diets. While neither intake nor milk production (22.9 kg/d) were affected, cows that received contaminated grain, gained significantly less weight. In a binder study that used over 150 cows consuming 2500 ppb DON and 270 ppb ZEN for six months, those consuming diets containing a mycotoxin binder produced significantly more milk (1.5 kg/cow/day), suggesting that the binder reduced the toxicity of DON or provided other beneficial activity (Diaz, et al., 2001). Conversely, Ingalls (1994), fed lactating cows diets containing 0, 3.6 10.9 or 14.6 ppm of DON for 21 days, without an apparent effect on feed intake or milk production (30 kg/d). DiCostanzo et al., (1995) in a review of several individual studies, concluded that beef cattle and sheep can tolerate up to 21 ppm of DON.

The FDA advisory guidelines for DON are as follows: one ppm on finished wheat products such as flour, bran, and germ for human consumption; 10 ppm in grain and grain byproducts for ruminating beef cattle, cattle in feedlots older than four months, and for chickens (the ingredients should not exceed 50% of the diet); five ppm of DON in grain and byproducts for swine (the ingredients should not exceed 20% of the diet); and five ppm in grain and byproducts for all other animals (the ingredients should not exceed 40% of the diet) [Wood and Trucksess, 1998].

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T-2 toxin (T-2) is produced primarily by *F. sporotrichioides* and *F. poae*, but is also produced by other species of *Fusarium* (Marasas et al., 1984). T-2 (and DAS) is often found in barley, wheat, millet, safflower seed, and in mixed feeds. Unthriftiness, reduced feed intake, reduced gain, low milk production, reproductive failure, gastrointestinal hemorrhage, and increased mortality can occur when cattle consume rations contaminated with these trichothecenes.

Effects of T-2 on swine include infertility accompanied with some lesions in the uteri and ovaries. Drastic and sudden decreases in egg production in laying hens have been shown to be caused by T-2 toxin in the parts per million range. Other effects in chickens include decreased shell quality, abnormal feathering, mouth lesions, and reduced weight gain. Pier et al. (1980) reported that egg production and shell quality were decreased at 20 ppm of dietary T-2 toxin. Turkeys fed T-2 exhibited reduced growth, beak lesions, and reduced disease resistance (Christensen et al., 1988). Mouth lesions were caused by DAS and other trichothecene toxins in broiler chickens (Ademoyero and Hamilton, 1991). In cattle, dietary T-2 toxin at 0.64 ppm for 20 days resulted in death and bloody feces, enteritis, and abomasal and ruminal ulcers (Pier et al., 1980).

T-2 toxin is a very potent mycotoxin and in cattle 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). 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 estrus cycles in cows exposed to T-2. 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). Our observations in dairy herds affected with T-2 toxin at dietary levels of 300 to 500 ppb suggest that T-2 toxin reduces milk production, hinders adjustment of fresh cows to the lactation diet, causes diarrhea, and intestinal irritation and increases culling and death rates.

Fumonisins

Fumonisin B_1 was first isolated in South Africa where *Fusarium moniliforme* has long been associated with animal problems (Gelderblom et al., 1988). Fumonisin has been shown to cause leucoencephalomalacia in horses (Marasas, et al., 1988), pulmonary edema in swine (Harrison et

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al., 1990) and hepatoxicity in rats (Gelderblom et al., 1991). This family of mycotoxins is produced by the species of *Fusarium* in the Liseola section. *F. verticilloides* (formerly *F. moniliforme*), a species that is almost ubiquitous in corn, and *F. proliferatum* are the main species producing high yields of fumonisins. Fumonisins B1, B2, and B3 (FB1, FB2, and FB3) are fumonisins in fungal cultures or found in naturally contaminated corn samples (Cawood et al.,1991). Feed infected with *F. verticilloides* has long been associated with outbreaks of blind staggers, equine leucoencephalomalacia (ELEM), in equines (Wilson et al., 1985). Fumonisins are structurally similar to sphingosine, a component of sphingolipids. Sphingolipids are in high concentrations in myelin and in certain nerve tissues. Fumonisin toxicity is thought to result from blockage of sphingolipid biosynthesis (Diaz and Borermans, 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). Murphy et al. (1993) reported fumonisin concentrations in corn for the Iowa, Wisconsin, and Illinois crops. Incidence of contamination was greater than 60% and concentrations ranged from 0 to 37.9 ppm. Corn screenings contained ca. 10 times the fumonisin content of the original corn.

Equine leucoencephalomalacia is characterized by facial paralysis, nervousness, lameness, ataxia and inability to eat or drink (Marasas et al., 1988). In a study conducted by Wilson et al. (1990), 14 of 18 horses fed with a corn-based feed with 37 to 122 ppm of FB1 died of equine leucoencephalomalacia. The macroscopic diagnostic lesion in ELEM is the liquefaction of the interior of the cerebral hemispheres; this lesion is not known to occur in other species exposed to fumonisin. It has been reported that if the fumonisin dose is high enough, horses will die of liver toxicity before the grossly observable lesion develops (Wilson et al., 1990; Wilson et al., 1992). Equidae are apparently the most sensitive species and can tolerate no more than ca. 5 ppm in corn.

Fumonisins cause pulmonary edema in swine (Motelin et al., 1994; Ross et al. l990). In swine, lower doses of FB, resulted in a slowly progressive hepatic necrosis; higher doses resulted in acute pulmonary edema coincident with hepatic toxicity (Haschek et al., l992). The symptoms in swine have been referred to as PPE and as “mystery swine disease” (Hollinger and Ekperigin, 1999).

Poultry are apparently more resistant to fumonisins than are swine and equines. Relatively high doses are required to induce measurable effects. Chicks fed 450 and 525 ppm of fumonisin for 21 days exhibited lowered feed consumption and weight gains. At 75 ppm free sphingosine levels were elevated (Weibking et al.1993a). Disruption of sphingolipid metabolism through

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inhibition of ceramide synthase activity is thought to be the mechanism of action (Norred, 1993). In another study, Weibking (1993b) found that day-old poults fed with rations containing 199 and 200 ppm of fumonisin B1, for 21 days had lower bodyweight gains and feed efficiency when compared to the controls. There were also differences in organ weights and blood parameters. He concluded that F. moniliforme culture material containing fumonisin, is toxic to young turkey poults and that the poult appears to be more sensitive to the toxin than the broiler chick (Weibking et al., 1993a,b).

While FB_1 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) fed young steers 15, 31 or 148 ppm of fumonisin in a short term study (31 days). There were no effects on feed consumption or gain; however, calves on the clean diet gained 1.44 kg/day, while those receiving 148 ppm fumonisin gained 097 kg/day. In the six calves consuming 148ppm fumonisin, two were found to exhibit mild liver lesions and the group had elevated liver enzymes indicative of liver damage and also had impaired lymphocyte blastogenesis.

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. Increases in serum enzymes concentrations suggested liver disease (Diaz et al., 2000). Dairy cattle may be more sensitive to fumonisin than are beef cattle, perhaps because of greater production stress. Fumonisin carryover from feed to milk is thought to be negligible (Richard et al., 1996 and Scott et al., 1994).

Fumonisin has been shown carcinogenic in rats and mice (NTP, 1999), and has been associated with esophageal cancer in humans in China (Chu and Li,1994) and South Africa (Rheeder et al., 1992). Therefore, fumonisin contamination has implications for human health, at least from a regulatory perspective. The FDA has recently released draft guidelines for fumonisin in human food and animal feed (Federal Register, 2000). In the draft guideline, it is recommended that human food products should contain no more than 2 to 4 ppm of total fumonisins. For animal feeds, the draft guideline recommends that contaminated corn or corn-byproducts be limited to no more than 20% of the diet for equids and rabbits and no more than 50% of the diet for other animals. Furthermore, the recommended maximum concentration of total fumonisins in corn and corn by-products are: 5 ppm for equids and rabbits; 20 ppm for swine and catfish; 30 ppm for breeding ruminants, breeding poultry, breeding mink, lactating dairy cattle, and hens laying eggs for human consumption; 60 ppm for ruminants three months of age or older that are raised

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for slaughter and for mink raised for pelt production; 100 ppm for poultry being raised for slaughter; and 10 ppm for pets and for all other animal species or classes of livestock. (Federal Register, 2000).

Penicillium molds

Ochratoxin A (OTA) is produced by species of *Penicillium* and *Aspergillus*, and is a causative agent of kidney disease in pigs that has been referred to as mycotoxin porcine nephropathy (Krogh, 1979). OTA can reduce weight gains and performance in swine (Cook et al., 1986) and poultry (Huff et al., 1988). Other symptoms include diarrhea, increased water consumption, diuresis and dehydration (Krogh et al, 1979). OTA 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).

Patulin is produced by *Penicillium*, *Aspergillus*, and *Byssochlamys* 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).

Dicoumarol is produced from natural plant compounds when *Penicillium* or *Aspergillus* molds grow on sweet clover or sweet vernal grass. Dicoumarol interferes with the function of vitamin K, resulting in a hemorrhagic syndrome. Moldy sweet clover poisoning is discussed by Radostits et al. (1980).

Mycotoxins in Forages

Many different mycotoxins have been found to occur on forages either in the field, or in storage as hay or silage (Lacey, 1991). Some mycotoxicoses in cattle resulting from contaminated forages have been reviewed (Lacey, 1991; Gotlieb, 1997; Seglar, 1997; and Whitlow, 1997). The limiting factor for mold growth in hay is moisture. Therefore, mold is most likely in hay stored too wet. The limiting factor for mold growth in silage is pH. However, if silage is stored too dry, or insufficiently packed and covered, infiltration of air allows for microbial activity which depletes silage acids, allowing pH to rise and molds to grow.

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It appears that *Aspergillus flavus* does not grow well in hay or silage, however, aflatoxin concentrations up to 5 ppm have been reported (Kalac and Woolford, 1982). We have detected low levels of aflatoxin (<100 ppb) in corn silage and alfalfa. Table 1 shows that the frequency of aflatoxin in corn silage is not different from the frequency of aflatoxin in corn grain, but the concentrations are lower. The frequency and concentrations of some Fusarium-produced mycotoxins are also compared in Table 1. There is trend toward a higher frequency of ZEN in corn silage than in corn grain and the concentrations of DON were higher in corn silage than in corn grain (Table 1).

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. Animal 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. *Aspergillus ochraceus* was implicated as producing OTA associated with abortions in cattle consuming moldy alfalfa hay (Still, et al, 1971). OTA has also been implicated in cattle deaths (Vough and Glick, 1993).

The most important pasture-induced toxicosis in the U.S. is tall-fescue toxicosis caused by endophytic alkaloids (Bacon, 1995). Other forage toxicoses of fungal origin include slobbers syndrome, perennial ryegrass staggers, and *Fusarium*-induced unthriftiness and impaired reproduction (Cheeke, 1995).

Mycotoxin Testing

The accurate determination of mycotoxin concentrations in grain and feeds depends on a number of factors. First, a statistically valid sample must be drawn from the lot (Whittaker et al., 1991). Because mycotoxins are not evenly distributed in grains and other feedstuffs, most of the error in a single analysis is due to sampling – as much as 90% of the error is associated with the taking of the initial sample. Proper collection and handling of representative feed samples is essential. Since molds grow in "hot" spots, mycotoxins are not uniformly distributed within a feed, making it difficult to obtain a representative sample especially from whole seed, course feeds or feeds not adequately mixed. Once collected, samples should be handled properly to prevent further mold growth. Wet samples may be frozen or dried before shipment and transit time should be minimized.

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The sample must then be finely ground and subsampled for analysis; this step is the second largest source of error in an analysis. Finally, the subsample is extracted, extract purified using one of several techniques, and measurement of the toxin is done. Toxin determination may be by thin-layer chromatography plates (TLC), high-performance liquid chromatography (HPLC), gas-liquid chromatography (GLC), enzyme-linked immunosorbent assays (ELISA), spectrophotometrically, or by other techniques.

Blacklighting for bright-greenish-yellow fluorescence (BGYF) is often used as a screening technique for aflatoxin, but it is very inaccurate; newer and better methods should be used. As far as we are aware, blacklighting is completely inappropriate for other mycotoxins.

Generally, laboratories provide analysis for only a limited number of mycotoxins perhaps including aflatoxin, ochratoxin, deoxynivalenol, ZEN, fumonisin, and T-2 toxin. Minimum detection levels may be directed at finding high levels which cause serious animal disease, rather than low levels which are associated with production losses, impaired immunity and significant economic losses. However, analytical techniques for mycotoxins are improving (Chu, 1992), costs are decreasing and several commercial laboratories are available which provide screens for an array of mycotoxins. The Federal Grain Inspection Service provides a list of approved mycotoxin tests.

Mold spore counts may not be very useful and are only a gross indication of the potential for toxicity, but 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 DON, diacetoxyscirpenol, T-2 toxin and nivalenol, because other *Fusarium* mycotoxins seldom occur without one of these four also present. Feeds could then be tested for other mycotoxins.

Mycotoxin prevention and treatment

Pre-harvest control has involved using agronomic practices, which minimize mycotoxin accumulation in the field. These include proper irrigation, pesticide application in some cases, resistant or adapted hybrids, tillage type, and proper fertilization. Unfortunately, breeding for mycotoxin-resistant hybrids has been only partially successful. Fungicides have shown little efficacy in controlling pre-harvest aflatoxin contamination in corn (Duncan et al., 1994).

Post-harvest approaches for management of mycotoxin contamination include: mycotoxin analysis of feedstuffs and diversion of contaminated lots; ammoniation of corn and cottonseed to

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destroy aflatoxin; dilution; and storage technology (Trail et al., 1995b). Mycotoxin-contaminated grains can be used for ethanol production, and in some cases mycotoxin-contaminated grains can be diluted with clean feeds (Desjardins et al.,1993). The FDA does not allow dilution of aflatoxin-contaminated feeds, which is considered adulteration. The best strategy for post-harvest control of mycotoxins is proper storage and handling of feed grains.

The potential for effective treatments has improved. Certain feed additives can reduce mycotoxin exposure of animals and thus minimize their negative effects. Some additives may be beneficial in reducing mycotoxin formation 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 can 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-face. If unacceptably high levels of mycotoxins occur, dilution or removal of the contaminated feed is preferable; however, it is usually impossible to replace all of a major forage ingredient. 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, Coffey et al., 1989, Smith et al., 1971).

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; Kubena et al., 1993; Lindemann et al., 1991; Scheideler, 1993; and Smith, 1980 and 1984). In most cases, clay has been added to the diet at about 1%. Activated carbon at 1% of the diet effectively reduced aflatoxin in milk (Galvano et al., 1996). Activated carbon fed at 0.1% of the diet did not reduce aflatoxin levels in milk (Diaz et al, 1999). A glucomannan fed at 0.05% of diet dry matter or bentonites at 1% of diet dry matter were effective in reducing aflatoxin concentrations in milk (Diaz et al., 1999).

Practical Recommendations for Prevention and Management of Mycotoxins

Prevention in Silages and Wet Feeds

Choose varieties that have resistance to fungal diseases.
Prevent silage exposure to air by following accepted ensiling practices: harvest at the proper moisture content, fill silos rapidly, pack silage well, and cover with plastic.

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Use an effective silage additive or mold inhibitor.
Match silo size with herd size to ensure daily removal of silage at a rate faster than deterioration which is considered to be at least 15 cm daily during warm weather and 8 to 10 cm during cold weather.
High moisture grains should be stored at proper moisture content and in a well-maintained structure.
Wet feeds should be fed within 10 to 14 days of delivery.

Prevention in Dry Feeds

Moisture is the single most important factor in determining if and how rapidly molds will grow in feeds.
Moisture must be monitored and controlled to remain below 15 percent.
Keep feed fresh, equipment clean, control insects and use mold inhibitors.
Avoid moisture migration by proper aeration.
Pelleted feeds must be properly cooled and dried prior to shipping.
Obvious sources of moisture in the feed handling and storage equipment such as leaks in feed storage tanks, augers, and compartments in feed trucks must be eliminated.
The humidity in confinement housing should be controlled with adequate ventilation.
Prepared feeds should generally be consumed within 10 to 14 days of delivery.
Manage the feed delivery system to ensure that feeds are uniform in freshness.
Routinely clean all feed handling equipment tanks, augars, feeders and bunks.

Use of Mold Inhibitors

Mold inhibitors are effective tools to reduce mold growth but should be not be relied on as the exclusive method of mold control.
The most commonly used mold inhibitors are either individual or combinations of organic acids (for example, propionic, sorbic, benzoic, and acetic acids), or salts of organic acids (for example, calcium propionate and potassium sorbate.
Mold inhibitors must be completely and thoroughly distributed throughout the feed, or for silage properly distributed on the area where mold is to be inhibited.
Many factors influence the effectiveness of mold inhibitors, and proper attention to these factors can enhance the benefits they provide.
Certain feed ingredients such as protein or mineral supplements (for example, soybean meal, fishmeal, poultry by-product meal, and limestone) tend to reduce the effectiveness of acid preservative, while added fat tends to enhance the activity of organic acids.

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Long-term mold inhibition requires a high initial rate of application or repeated applications.

Animal Management

Observe symptoms and then consider and eliminate other possible causes for those problems.
Analyze feeds for the more common mycotoxins such as DON, ZEN, T-2 toxin and fumonisin.
If unacceptable mycotoxin levels occur, removal of the contaminated feed is best but not always possible.
If a mycotoxicosis is suspected, add mycotoxin binders to the diet and observe the herd for responses.
Reduce stress including environmental and nutritional stresses.
Manage the feeding program to maximize feed intake.
Use buffers to maintain normal rumen pH and to avoid acidosis.
Raise protein and energy while maintaining adequate dietary fiber.
Increase antioxidant nutrients such as vitamin E and selenium.
Dry cows and springing heifers should consume no moldy, mycotoxin-contaminated feed.
Use proper dry cow and transition rations.
Make judicious use of mold inhibitors.

Mycotoxin Sampling and Testing

Mold spores counts are not of much valuable because many molds are not toxigenic and amount of mold does not correlate well with amount of mycotoxins.
Mold identification can be useful to suggest the possible mycotoxins produced by those molds.
It is difficult to take an accurate sample for mycotoxins because molds and mycotoxins are unevenly distributed.
About 90 percent of the error associated with mycotoxin assays can be attributed to how the original sample was collected.
For whole kernel grains, a properly taken composite sample of at least ten pounds is required for a reasonably accurate analysis.
Silage samples might best be taken after blending a normal feeding amount in a blender wagon.
Avoid submitting silage grab samples from the silo feeding face.
Analyze feeds for those mycotoxins known to be a concern in your geographic area, to include DON, ZEN, T-2, and fumonisin.
Black light screening of feed for aflatoxin is inappropriate and should not be used.
Dry or freeze the feed sample and ship for 2-day delivery.

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Dry feeds should be shipped in paper bags and frozen samples in plastic bags in an insulated shipping bag.
Identify laboratories in your area that are fast accurate and cost effective.

Areas of Needed Information

More information is needed about why mycotoxins occur, when to expect them, how to prevent their occurrence and how to deal with their presence. More data are needed about animal toxicity, about interactions with other mycotoxins, with nutrients, and with stress factors such as disease organisms or environmental stress. Improved screening techniques are needed for monitoring mycotoxin occurrence, for diagnosing toxicities, and for prevention and treatment (CAST, 1989).

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