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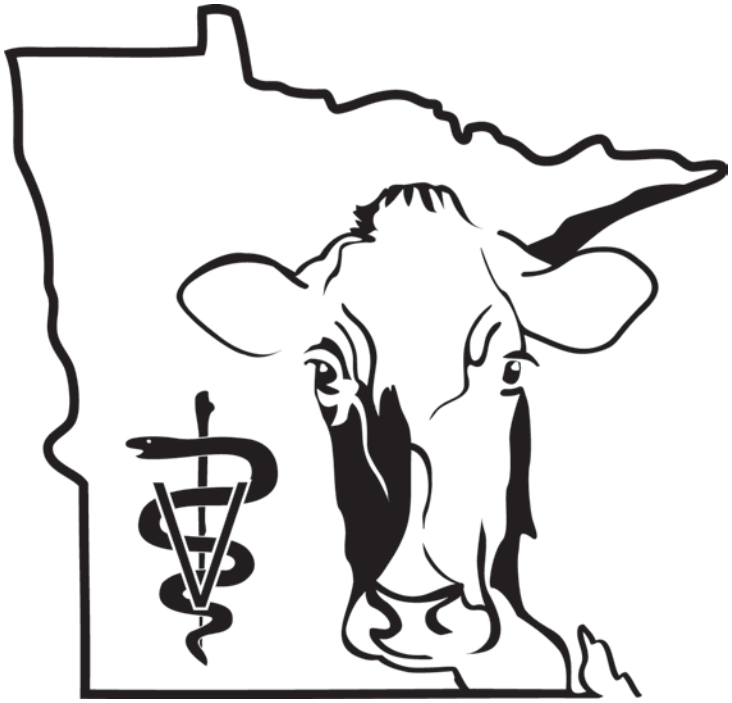

ST. PAUL, MINNESOTA
UNITED STATES OF MINNESOTA

The Potential for an Association of Mycotoxins with Problems of Production, Health and Reproduction in Dairy Cattle

L. W. Whitlow and W. M. Hagler, Jr.
Animal Science Department and Poultry Science Department
North Carolina State University
Raleigh, NC

Introduction

A priority list of mycotoxins was subjectively produced by a survey of mycotoxicologists worldwide and included: aflatoxin (AF), ochratoxin, trichothecenes (primarily T-2 toxin (T-2), zearalenone (ZEN), deoxynivalenol (DON), citrinin, sterigmatocystin, patulin, and cyclopiazonic acid (Hesseltine, 1986a). Fumonisin was identified after this list was compiled (Gelderblom et al., 1988), but undoubtedly it would be included in a current list.

The mycotoxicoses which may be most commonly associated with grazing cattle include, ergotism, paspalum staggers, fescue toxicity, sweet clover poisoning, facial eczema, and slaframine toxicity. These and other mycotoxicoses are important and have been reviewed by Lacey (1991).

This paper will concentrate on those mycotoxins which are of greatest concern for dairy cattle consuming stored feeds, and includes: AF, fumitremorgens, and sterigmatocystin, which are primarily produced by *Aspergillus* molds; DON, ZEN, T-2, diacetoxyscirpenol (DAS), and fumonisins, which are produced by *Fusarium* molds; and ochratoxin (OT), PR toxin, roquefortine primarily produced by *Penicillium* molds. Several other mycotoxins, produced by these and other molds, are known to be prevalent at times, including derivatives of those listed. It is probable that a lack of observation and simple analytical techniques have prevented us from more fully understanding the prevalence of these mycotoxins and their impact on animal production.

Mold Growth and Mycotoxin Formation

Molds occur universally in a variety of feedstuffs, including roughages and concentrates and can produce mycotoxins under certain conditions. Molds can grow and mycotoxins can be produced pre-harvest or post-harvest, during storage, processing, or feeding. Mycotoxin production is often related to extremes in weather conditions (causing plant stress or excess hydration of stored feedstuffs), to inadequate storage practices, to low feedstuff quality, and to faulty feeding conditions.

Conditions for mold growth and mycotoxin formation are dependent on the specific mold but include the presence of fungal spores, an organic substrate and the proper levels of moisture, oxygen, temperature, and acidity (Moss, 1991). Temperatures may range from -5° to 60°C. Water activity must generally be above 0.7 a_w (ratio of the vapor pressure of the product to that of pure water or equilibrium relative humidity as a percentage). Mold can begin growing when moisture exceeds about 12%. Higher levels of moisture will support mold growth up to the point where water excludes adequate oxygen. High levels of CO_2 can prevent mold growth even when O_2 is at levels high enough to support mold growth. Oxygen as low as 0.5% can support mold growth, thus there

can be pockets of adequate oxygen within silage and high moisture grain storage within the feed mass and especially near the feed surfaces. A fairly wide range of pH levels will support mold growth, although they do not grow well at extremely low or high pH levels. While silage pH is generally low enough to prevent most mold growth, yeast are active at a lower pH and can raise the pH to a point conducive for mold growth.

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

Fusarium commonly affects corn, causing ear and stalk rots, and small grains, causing 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) suggests that plowing in plant debris and crop residue left on the field after harvest may reduce fungal disease problems.

It should be noted that 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 (ATA) 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).

Mycotoxin Occurrence

The warm, humid climate of the southern U.S., results in a considerably higher incidence of AF in feeds. From 1975 to 1980, 34% of corn grain in North Carolina (NC) contained more than 20 ppb of AF. Corn gain and peanut meal have been the primary sources of AF contamination in NC. Cottonseed has seldom been a problem source of AF for dairymen in NC. Corn samples from the midwestern U.S. representing the 1988 season (severe drought) showed 8% with AF levels above 10 ppb, 3% positive for ZEN above 1 ppm, 3% positive for DON above 1 ppm and 7% positive for T-2 above 500 ppb (Russel et al., 1991). The positives for Minnesota were 4% AF, 4% ZEN, 4% DON and 9 % T-2.

Mycotoxins analyses results, from feed samples submitted by North Carolina farmers over a nine

year period and representing over 2400 samples, were summarized. Percentage of corn silage and corn grain samples testing positive were for aflatoxin ≥10 ppb, 8% and 9%; DON ≥500 ppb, 51% and 52%; ZEN ≥300 ppb, 17% and 3%; T-2 ≥200, 5% and 4% and FB ≥1 ppm, 37% and 60%, respectively (Whitlow, In press). Occurrence was highly variable by year.

Mycotoxins effects

Mycotoxins can increase disease incidence and reduce production efficiency in cattle. They 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 (CAST, 1989). The resulting nonspecific symptoms may therefore be perplexing and make diagnosis difficult. Hesseltine (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.

Our experience suggests that while a definitive diagnosis cannot be made directly from symptoms, specific tissue damage, or even feed analyses, experience with mycotoxin affected herds greatly increases the probability of recognizing the 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 as a general guide to symptoms observed in the field, however there is a lack of research data, and field observations may differ from those seen in controlled research studies, (3) Systemic effects as well as specific damage to target tissues can be used as a guide to possible causes, (4) Postmortem examinations may indicate no more than gut irritation, edema or generalized tissue inflammation, (5) Ruling out other possible causes such as infectious agents or other toxins is essential, (6) All feeds should be analyzed for common mycotoxins, (7) Responses to simple treatments such as dilution or removal of the contaminated feed are helpful, (8) Diagnosis may be impossible because the clinical situation may be complex and complicated due to interactions with other agents.

Dairy herds thought to suffer from a mycotoxicosis severe enough to experience a milk production loss, 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 many of these symptoms evident.

Aflatoxin (AF)

AF, produced primarily by *Aspergillus flavus*, is a mycotoxin of major concern, because it is carcinogenic and is commonly found in the southern US. Major efforts are directed at eliminating food residues. FDA limits AF to no more than 200 ppb in breeding cattle, 300 ppb for finishing beef cattle, 20 ppb in lactating dairy feeds and 0.5 ppb in milk. AF is excreted into milk in the form of AFM_1 with residues approximately equal to 1.7% of the dietary level (Van Egmond, 1989). Since AF residues can be found in tissues, beef cattle should not be fed AF contaminated diets for three weeks prior to slaughter. Regulatory pressures and a widespread awareness have helped minimize AF problems. The GAO (1991) concluded that industry, federal and state programs are effective in detecting and controlling AF and that it is doubtful that additional programs or limits would reduce the risk of AF in the food supply. Thus, current surveillance programs aimed at reducing food residues make it very unlikely for AF to have significant production or health effects on dairy herds.

AF can reduce performance and impair health but this occurs generally at dietary levels greater than the 25 to 50 ppb which can cause illegal milk residues. Although no level of AF is considered safe, the degree of toxicity is related to level of toxin, duration of feeding, and the amount of other stresses affecting the animal. Levels of 300 to 700 ppb are considered toxic for beef cattle depending on criteria for toxicity, and other factors affecting toxicity (CAST, 1989). 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 effects 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. Milk production increased 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, but equal amounts of pure AF did not. Several studies suggest that naturally contaminated feeds are more toxic than would be expected from the concentrations of assayed mycotoxins, suggesting the presence of unidentified toxins.

Fumonisin (FB)

Fumonisin B_1 (FB_1) was isolated by Gelderblom et al. (1988) and shown to be a cancer promoter. FB_1 has been shown to cause leukoencephalomalacia in horses (Marasas, et al., 1988), pulmonary edema in swine (Harrison et al., 1990) and hepatoxicity 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_1. 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) demonstrated that FB_1 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 (1998) has demonstrated that FB_1 is also toxic to dairy cattle. Fed for approximately 7 days prior to freshening and for 70 days thereafter, dietary FB_1 at 100 ppm significantly and dramatically reduced milk production (7 kg/cow/day) and affected serum enzymes indicative of liver disease. These results strongly suggest that FB_1 is toxic to dairy cattle and that FB_1

is less toxic to beef cattle than to dairy cattle, or perhaps FB_1 interacts with other factors to produce greatly different effects in beef and dairy cattle under different conditions.

FB_1 carryover from feed to milk is thought to be negligible. Richard et al. (1996) fed fumonisin B_1 (about 75 ppm) to dairy cows and with no fumonisin B_1 or B_2 detectable in milk (detection limit of 5 ng/ml). Scott et al. (1994) have confirmed this observation.

Deoxynivalenol (DON)

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) and lower milk production (Whitlow et al., 1994). Clinical data from 300 herds representing about 40,000 cow records showed that DON was associated with a loss in milk production but did not establish a cause and effect (Whitlow et al., 1994). 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) were affected by additions of this grain to the diet, however compared with controls, cows which received this grain at either 10% (about 1.25 ppm DON and 50 ppb ZEN) or 20% (about 2.50 ppm DON and 100 ppb ZEN) of their diet gained significantly less weight during the study (5.8 kg and 8.1 kg, less weight gain for cows consuming the 10% and 20% diets over 21 days).

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, apparently without 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. Reports from Nebraska, indicated similar feed intakes, average daily gains and feed efficiencies when sheep (8.5 dietary DON) or cattle (1 ppm dietary DON), consuming DON contaminated diets were compared with those consuming control diets containing no detectable DON (DeHaan et al., 1984). Nelson et al. (1984) fed feedlot steers and heifers, diets containing either 0.2, 2.3 or 10 ppm of DON for 126 days. The low DON diet was corn based while the other two contained wheat. Results reported for the low to high DON diets were similar for dry matter

intake (9.4, 8.7 and 7.8 kg/day), average daily gain (1.54, 1.64, and 1.34 kg/day) and feed efficiency (6.2, 5.6, and 5.7 kg dry matter intake/kg gain). Results for carcass characteristics, serum biochemistry and tissue histology were similar across treatments. DiCostanzo et al. (1995a and 1995b) indicated that feeding up to 18 ppm dietary DON did not affect intake, daily gain, feed efficiency or carcass characteristics of 415 kg steers fed for 166 days. Other recent feeding experiments with beef cattle suggest that beef cattle can tolerate large concentrations of DON in a feedlot situation without effects on dry matter intake, average daily gain, or feed to gain ratio (Boland et al., 1994, and Windels et al., 1995).

These data suggest that cattle are relatively tolerant of DON. While not compared directly, it appears that beef cattle and sheep may be less sensitive to DON than are dairy cattle. Differences could be related level of production stress, since midlactation, low-producing dairy cattle also appear to be more tolerant to DON than are high-producing dairy cattle in early lactation. Mycotoxins may interact with immune suppression in early lactation to produce more severe effects than would otherwise be expected. Heat or other environmental stresses may be involved. Thus, the early-lactation, high-producing cow which experiences greater stress, lower immunity, marginal nutrient deficiencies and a faster rumen turnover (less mycotoxin degradation in the rumen) may be more vulnerable to mycotoxin effects.

DON is but one causative agent that may be present. DON may serve as a marker for feed exposed to a situation conducive to mold growth and mycotoxin formation, and thus the possible presence of other mycotoxins or factors more toxic than DON itself. The differences in response to DON may be due to other mycotoxins. The beef cattle experiments have generally utilized DON contaminated corn or barley. DON provided by contamination of a different feed source, such as silage, could result in interactions of different mycotoxins. Mycotoxin interactions are discussed more fully in the section "Safe Levels of Mycotoxins".

T-2 Toxin

T-2 toxin, a *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, Kosuri et al., 1970). Weaver et al. (1980) demonstrated that T-2 was associated with feed refusal and gastrointestinal lesions in a cow, but failed to show a hemorrhagic syndrome. Serum immunoglobulins and certain complement proteins were lowered in calves receiving T-2 toxin (Mann et al, 1983). Other data demonstrated a reduction in white blood cell and neutrophil counts in calves (Gentry et al, 1984). A calf intubated with T-2 developed severe depression, hindquarter ataxia, knuckling of the rear feet, listlessness and anorexia (Weaver et al, 1980).

While data with cattle are limited, the toxicity of T-2 toxin in laboratory animals is well documented (Wannemacher et al, 1991). Our experience suggests that T-2 is a severe gastrointestinal irritant, which can cause hemorrhage and necrosis of the intestinal tract. Diarrhea is usually present but may not be hemorrhagic. With high levels of T-2, there can be congestion and irritation to the liver, lungs and heart. Two dairy herds were observed to receive T-2 contaminated feed from the same supplier and on similar dates. Early lactation cows were more severely affected, showing a lack of appetite, severe and prolonged weight loss, low peak milk production and higher levels of morbidity and

death. In another field case, T-2 in corn produced on the farm resulted in approximately 350 ppb in the diet. Cows exhibited diarrhea, which moved in a wave through three groups in a dairy herd of about 150 Jersey cows. Milk production was erratic for two to three days and then dropped by 15%. The addition of a clay product to the diet appeared to restore production to previous levels after about three weeks. Removal of the clay resulted in an immediate loss in milk production and the clay was again fed with a positive response.

Zearalenone (ZEN)

Zearalenone is a *Fusarium* produced mycotoxin which elicits an estrogenic response in monogastrics (Sundlof and Strickland, 1986). However, ZEN is rapidly converted to α- and β-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 (Khamis et al., 1986; Mirocha et al., 1968; and Roine et al., 1971). Large doses are associated with abortions in cattle (Kellela and Ettala, 1984; and Mirocha et al., 1974). Mirocha et al. (1968) isolated ZEN from hay associated with infertility in dairy cattle. 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, α- and β-zearalenol and α- and β-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, within herds, individual cows 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 problems in dairy cattle were noted with "zearalenone" concentrations of about 400 ppb in the pasture samples.

Our observations suggest that ZEN may be associated with poor feed intake, a loss of milk production, poor conception, early embryonic mortalities and increased reproductive tract infections. In most cases, cows have appeared well nourished with average body condition scores but poor reproductive performance. The differences may be attributed to the presence of other mycotoxins or interaction with other factors.

Other Mycotoxins

Many other mycotoxins may affect ruminants but are thought to occur less frequently or be less potent.

Fumitremorgens such as fumigaclavine A and B are produced by *Aspergillus fumigatus*, and are thought to be common in silages of the southeastern US. They can cause anorexia, diarrhea, unthriftiness and irritability (Cole et al., 1977).

Strigmatocystin 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).

Diacetoxyscirpenol is a *Fusarium* produced mycotoxin. It may occur along with T-2 toxin and causes similar symptoms.

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, a *Penicillium* produced mycotoxin associated with aerobic deterioration of silage 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 and was the suspected vector in a case study with symptoms of abortion and retained placenta (Still et al., 1972).

Roquefortine, produced by *Penicillium requefortii*, is a tremorgen which has been found in silage.

Other mycotoxins such as rubratoxin, citrinin, cyclopiazonic acid, and ergotoxins may be of some importance. Many other mycotoxins are possible.

Mycotoxin Testing

To determine toxicity, feeds should be analyzed for mycotoxins and not just mold content, however the type of mold present may suggest the mycotoxins most likely to be present. The amount or presence of mold or mold spore counts are not very indicative of mycotoxin content (Wyatt, 1991). Molds may be present which do not produce, or are not currently producing mycotoxins. A mold may have produced mycotoxins and is no longer viable resulting in mycotoxin levels without the obvious presence of mold. It is possible that opinions have been formed about the toxicity of mycotoxins, based on the presence of mold, which might suggest a low or erratic toxicity.

Analytical techniques for mycotoxins are improving (Chu, 1992). Several commercial laboratories are available and provide screens for a large array of mycotoxins. Cost of analyses has been a constraint but can be insignificant compared with the economic consequences of production and health losses related to mycotoxin contamination. Newer immunoassays have reduced the cost for analyses.

Collection of representative feed samples is a problem primarily because molds can produce very large amounts of mycotoxins in small areas making the mycotoxin level highly variable within the lot of feed. Sampling of horizontal silos show mycotoxins to be highly variable throughout the silage, however, the silo face appears to have higher and more consistent levels. Because mycotoxins can form in the collected sample, it should be preserved and delivered to the lab quickly. Samples can be dried, frozen or treated with a mold inhibitor before shipping.

Safe Levels of Mycotoxins

Guidelines for acceptable levels of mycotoxins should be conservatively low due to nonuniform distribution, uncertainties in sampling and analysis, the potential for more than one source in the diet, and the limited amount of research. All these factors make it impossible to declare levels of safety.

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 may be the best approach to determining safe levels. They state that any level of mycotoxin carries with it a risk of loss and that 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 where 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, 1998), but to not effect average daily gain in beef cattle fed 148 ppm (Osweiler et al,, 1993). AF produced from culture was more toxic to dairy cattle than pure AF added to diets (Applebaum et al., 1982). In swine, Foster et al. (1986) demonstrated that pure DON added to diets was less toxic than diets with similar concentrations of DON which was supplied from naturally contaminated feeds. Smith and MacDonald (1991) have suggested that fusaric acid may occur along with DON 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. Scott (1990) states that screening methods are needed for the *Fusarium* produced mycotoxins and that one approach is to

test for DON, DAS, T-2 and nivalenol, because other *Fusarium* mycotoxins seldom occur without one of these four also present. Feeds could then be further tested for other 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). The rat is much more sensitive to both aflatoxin and T-2 than is the mouse (Wannemacher et al., 1991). Other animal factors include sex, age, environmental and production stress. 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.

Prevention and Treatment

Prevention of mycotoxin formation is essential since there are few ways to completely overcome problems once mycotoxins are present. Prevention of mycotoxins in silage includes following accepted silage making practices aimed at enhancing proper fermentation and eliminating oxygen. Silages should be harvested at the correct moisture content, the silo filled rapidly, the silage packed tightly and the silo sealed completely. Silo size should be matched to herd size to insure daily removal of silage at a rate faster than deterioration (4 to 6 inches daily, depending on weather). The face of horizontal silos should be cut cleanly while avoiding loosening more silage than is to be fed. Secondary fermentation can occur very rapidly after loosened silage is exposed to the air. Therefore, silage should be fed directly after removal from the silo and feed bunks should be cleaned regularly. Care should be taken to ensure that high moisture grains are stored at proper moisture contents and in a well maintained structure. Grains or other dry feed such as hay should be stored at a moisture content (<14%) below which molds do not readily grow. Aeration of grain bins is important to reduce moisture migration and to keep the feedstuffs dry.

Some additives may be beneficial in reducing mycotoxins because they are effective in reducing mold growth. Ammonia, propionic acid and microbial or enzymatic silage additives have all shown effectiveness as mold inhibitors. It seems reasonable that additives, which enhance fermentation, may be added at ensiling, while those which inhibit mold growth be added as surface treatments when capping off the silo or daily after silage feed-out to reduce molding of the exposed silage 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. Ammoniation of grains can destroy some mycotoxins, but there is no practical method to detoxify affected forages. Increasing nutrients such as protein, energy and antioxidant nutrients may be advisable (Brucato et al., 1986, Chandler, 1992). Adsorbent materials are not approved by the FDA for the prevention or treatment of mycotoxicoses. However, favorable research results have been seen when adsorbent materials such as clays (bentonites) are added to mycotoxin contaminated diets of rats, poultry, swine and cattle (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%. Considerable data is also available for other absorbent materials such as charcoal, fiber, and yeast cells. Acidic diets may exacerbate effects of mycotoxins. In some situations, poultry respond to water soluble vitamins. Additional research on treatments is needed.

Areas of Needed Information

The Council for Agricultural Science and Technology published a list of major needs for research (CAST, 1989). Included the their list are surveillance of feeds for mycotoxin presence and quantity, assessment of control methods, development of resistant plants, improvement of sampling and analysis, improved understanding of effects on animals particularly on immunosuppression, toxicological evaluation of newly discovered mycotoxins and assessment of economic effects.

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