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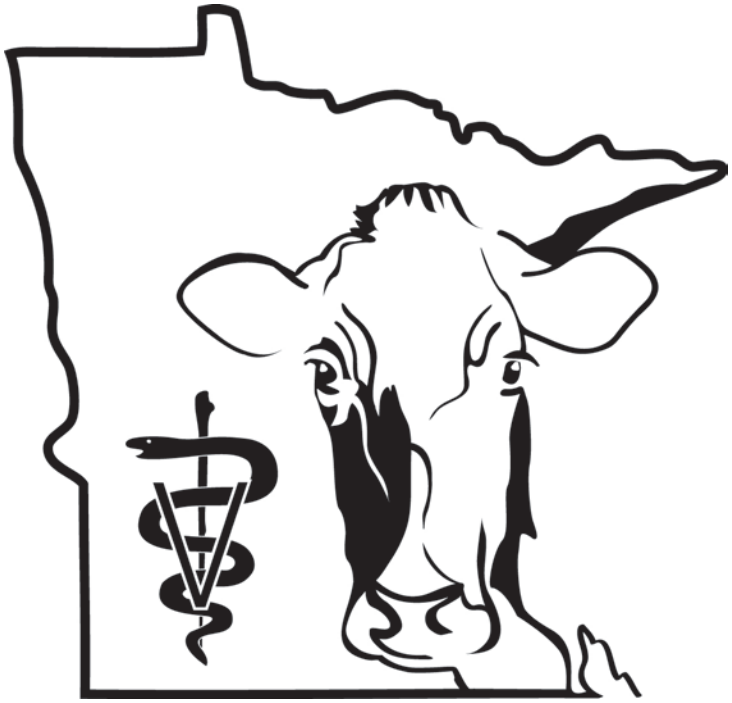

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Moldy Feed

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Mold growth, mycotoxin formation

Molds can grow and mycotoxins can be produced pre-harvest or post-harvest during storage, transport, processing or feeding. Many species of fungi produce mycotoxins in feedstuffs, yet feed can be moldy without the presence of mycotoxins. Mold growth and mycotoxin production are related to plant stress caused by weather extremes, insect damage, inadequate storage practices and faulty feeding conditions. Temperature, water activity and insect activity are the primary determinants of mold growth (Coulombe, 1993). Computer models that predict mycotoxin concentrations in pre-harvest corn are based on temperature, rainfall and insect pressure (Dowd, 2004). Molds grow over a temperature range of 10-40°C (50-104°F), a pH range of 4-8 (*Penicillium* grows at a low pH) and above 0.7 aw (equilibrium relative humidity expressed as a decimal instead of a percentage). Molds grow best at moisture contents of >12-15%. In feeds such as silage, moisture helps exclude air, but molds will grow at a high moisture content if sufficient oxygen is present.

Aspergillus species normally grow at lower water activities and at higher temperatures than the *Fusarium* species and therefore, aflatoxin in corn is favored by the heat and drought stress associated with warmer climates. The individual *Penicillium* species have variable growth requirements, but are more likely to grow under post-harvest conditions, in cooler climates, in wet conditions and at a lower pH. *Penicillium* molds are a major contaminant of silage, probably because they are acid tolerant. The *Fusarium* species are important plant pathogens that can proliferate pre-harvest, but continue to grow post-harvest. 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, *Fusarium* is associated with excessive moisture at flowering and early grain-fill stages. In corn, *Fusarium graminearum* is referred to as a red ear rot and is more commonly associated with a cool, wet growing season and with insect damage. Fusarium ear rots that produce fumonisins are referred to as pink ear rots and vary in their environmental requirements, but are often associated with dry conditions in mid-season followed by wet weather (CAST, 2003).

Mycotoxins

Mycotoxins are toxic secondary metabolites produced by toxigenic filamentous fungi that cause an undesirable effect (mycotoxicosis) when animals are exposed. Exposure is usually by consumption of contaminated feeds, but may also be by contact or inhalation. Biological effects include liver and kidney toxicity, central nervous system effects, immune suppression and estrogenic effects, to name a few. The fungal toxins are chemically diverse — representing a variety of chemical families — and range in molecular weight from about 200 to 500. There are hundreds of mycotoxins known, but

few have been extensively researched and even fewer have routine methods of analysis available. The primary classes of mycotoxins are aflatoxins, zearalenone, trichothecenes, fumonisins, ochratoxin A and the ergot alkaloids.

Molds Can Cause Disease

A mold (fungal) infection resulting in disease is referred to as a mycosis. Fungal pathogens include: *Aspergillus fumigatus, Candida albicans, Candida vaginitis,* certain species of *Fusarium* and others. *Aspergillus fumigatus* is known to cause mycotic pneumonia, mastitis and abortions and has been proposed as the pathogenic agent associated with mycotic hemorrhagic bowel syndrome (**HBS**) in dairy cattle (Puntenney et al., 2003).

Aspergillus fumigatus is a fairly common mold in both hay (Shadmi et al., 1974) and silage and produces a number of mycotoxins (Cole et al., 1977). Gliotoxin is also shown to affect rumen fermentation, reducing digestibility and VFA production *in vitro* (Morgavi et al., 2004). It is theorized that with a mycosis, mycotoxins produced by the invading fungi can suppress immunity; therefore increasing the infectivity of the fungus. While healthy cows with an active immune system are more resistant to mycotic infections, dairy cows in early lactation are immune suppressed during which HBS is more likely (Puntenney et al., 2003). Gliotoxin was shown to be produced in mice associated with *A. fumigatus* (Eichner et al., 1988) and present in ruminants infected with *A. fumigatus* (Bauer et al., 1989). In humans, gliotoxin results in apoptosis of monocytes suppressing cellular immune response by targeting antigen presenting cells (Stanzani, et al., 2004). Gliotoxin increased the virulence of *A. fumigatus* using an insect model (Reeves et al. 2004). Niyo et al. (1988a, b) demonstrated that in rabbits, T-2 toxin decreased phagocytosis of *A. fumigatus* conidia by alveolar macrophages and increased severity of experimental aspergillosis. It is possible that gliotoxin, T-2 toxin, or other mycotoxins suppress immunity and provide the trigger to increased infectivity by the fungus; ultimately resulting in HBS or other fungal infections. Feeding a commercial mycotoxin adsorbent with anti-fungal properties stimulates immunity and reduced HBS (Puntenney et al., 2003).

Mycotoxin occurrence

Mycotoxins occur frequently in a variety of feedstuffs and are routinely fed to animals. Mycotoxin occurrence and concentrations are variable by year, because of the annual variation in weather conditions and plant stresses known to affect mycotoxin formation (Coulombe, 1993). Worldwide, approximately 25% of crops are affected by mycotoxins annually (CAST, 1989), which would extrapolate to billions of dollars associated with losses in livestock productivity, losses in crops and the costs of regulatory programs (Trail et al., 1995). Annual economic costs of mycotoxins to the U.S. agricultural economy is estimated to average $1.4 billion (CAST, 2003). Table 1 provides mycotoxin analyses of feed samples submitted by North Carolina farmers over a nine-year period indicating that mycotoxins in feeds including corn silage and corn grain occur commonly at unsuitable concentrations (Whitlow et al., 1998).

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.					
Mycotoxin	Feedstuff	Number of samples	Positive above limits, %	Mean	Standard deviation
Aflatoxin, >10 ppb	Corn Silage	461	8	28	19
	Corn Grain	231	9	170	606
	All Feeds	1617	7	91	320
Deoxynivalenol, > 50 ppb	Corn Silage	778	66	1991	2878
	Corn Grain	362	70	1504	2550
	All Feeds	2472	58	1739	10880
Zearalenone, > 70 ppb	Corn Silage	487	30	525	799
	Corn Grain	219	11	206	175
	All Feeds	1769	18	445	669
T-2 toxin, > 50 ppb	Corn Silage	717	7	569	830
	Corn Grain	353	6	569	690
	All Feeds	2243	7	482	898
Fumonisin, > 1 ppm	Corn Silage	63	37	--	--
	Corn Grain	37	60	--	--
	All Feeds	283	28	--	--

Because cattle consume forages and byproduct feeds, they may be exposed to a broader array of mycotoxins than are monogastric animals. Reviews are available on mycotoxins in forages (Lacey, 1991; Scudamore and Livesay, 1998) and by-product feeds (Lillehoj, et al., 1991).

In Germany, molds were found in 206 of 233 grass or corn silage samples collected during 1997-98 (Schneweis et al., 2000). In 25 hay and silage samples collected in Minnesota, Wisconsin and Illinois, there was a high incidence of cyclopiazonic acid, DON, FB, PR toxin and alternaria TA toxin (Yu et al., 1999).

Mycotoxin effects

The potentially harmful effects of feeding moldy grain and foods has been known for many years (Matossian, 1989), however mycotoxicology, the study of mycotoxins, really began in 1960 with the outbreak of Turkey-X disease in the U.K. This outbreak was linked to peanut meal imported from Brazil (Sargeant et al., 1961). A blue-fluorescent toxin was isolated and mycelia of *A. flavus* were observed. *A. flavus* was shown to produce the toxic compound(s) found in the toxic peanut meal, which was given the trivial name aflatoxin. Aflatoxin was shown to be very toxic and carcinogenic in test animals, and it resulted in a toxic metabolite 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.

Mycotoxins, in large doses, can be the primary agent causing acute health or production problems in a dairy herd. A more likely scenario is to find mycotoxins at lower levels interacting with other stressors and contributing to chronic problems including a higher incidence of disease, poor reproductive performance, or suboptimal milk production. To the animal producer, these chronic losses are of greater economic importance than losses from acute effects, and more difficult to diagnose.

Mycotoxins exert their effects through several means including 1) reduced intake or feed refusal; 2) reduced nutrient absorption and impaired metabolism; 3) altered endocrine and exocrine systems; 4) suppressed immune function; 5) altered rumen microbial growth, and 6) cellular death.

Ruminal degradation of mycotoxins helps to protect the cow against acute toxicity, but may contribute to chronic problems, associated with long term consumption of low levels of mycotoxins. Ruminal degradation of mycotoxins may have helped mask mycotoxin effects in dairy cows. In recent years, as production stresses increased, the dairy industry has placed more attention on management details and the significance of chronic mycotoxin effects has been more widely recognized (Jouany and Diaz, 2005).

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

A diagnosis of a mycotoxicosis is difficult or impossible because of the complex clinical scenario resulting from a cascade of events producing a wide diversity of nonspecific symptoms (Schiefer, 1990). The difficulty of diagnosis is increased due to limited research, occurrence of multiple mycotoxins, non-uniform distribution, problems of sampling and analysis and interactions with other stressors. Because of the difficulty of diagnosis, the determination of a mycotoxin problem becomes a process of elimination and association. Certain basics observations can be helpful (Schiefer, 1990): 1) Mycotoxins should be considered as a possible primary factor resulting in production losses and increased incidence of disease; 2) Documented symptoms in ruminants or other species can be used as a general guide to symptoms observed in the field; 3) Systemic effects as well as specific damage to target tissues can be used as a guide to

possible causes; 4) Post mortem examinations may indicate no more than gut irritation, edema, or generalized tissue inflammation; 5) Because of the immune suppressing effects of mycotoxins, increased incidence of disease or atypical diseases may be observed; 6) Responses to added dietary adsorbents or dilution of the contaminated feed may help in diagnosis; 7) Feed analyses should be performed, but accurate sampling is a major problem and only a few mycotoxins are analyzed.

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 and interactions with stress factors or other mycotoxins (Schaeffer and Hamilton, 1991). The FDA has established action, guidance and advisory levels, in part, to protect public health. Grains with mycotoxin(s) that exceed the appropriate action, advisory or guidance levels may be considered by CVM as adulterated and may be considered by CVM as unfit for use in animal feed (Henry, 2006).

Toxicity of Individual Mycotoxins

Aflatoxin

Aflatoxins are extremely toxic, mutagenic, and carcinogenic compounds produced by *Aspergillus flavus* and *A. parasiticus*. Aflatoxin B1 is excreted in milk in the form of aflatoxin M1. The FDA limits aflatoxin to no more than 20 ppb in lactating dairy feeds and to 0.5 ppb in milk. A thumb rule is that milk aflatoxin concentrations equal about 1.7% (range from 0.8 to 2.0%) of the aflatoxin concentration in the total ration dry matter. Cows consuming diets containing 30 ppb aflatoxin can produce milk containing aflatoxin residues above the FDA action level of 0.5 ppb. Aflatoxin appears in the milk rapidly and clears within three to four days (Diaz et al., 2004).

Symptoms of acute aflatoxicosis in mammals include: inappetance, lethargy, ataxia, rough hair coat, and pale, enlarged fatty livers. Symptoms of chronic aflatoxin exposure include reduced feed efficiency and milk production, jaundice, and decreased appetite. Aflatoxin lowers resistance to diseases and interferes with vaccine-induced immunity (Diekman and Green, 1992). In beef cattle, Garrett et al. (1968) showed an effect on weight gain and intake with diets containing 700 ppb aflatoxin, but if increases in liver weights are used as the criteria for toxicity, 100 ppb would be considered toxic to beef cattle. Production and health of dairy herds may be affected at dietary aflatoxin levels above 100 ppb, which is higher than the 30 ppb that is expected to produce illegal milk residues. Guthrie (1979) showed when lactating dairy cattle in a field situation were consuming 120 ppb aflatoxin, reproductive efficiency declined and when cows were changed to an aflatoxin free diet, milk production increased over 25%. Applebaum et al. (1982) showed that milk production was reduced more by impure aflatoxin produced by culture, but than by equal amounts of pure aflatoxin.

Aflatoxin is more often found in corn, peanuts and cottonseed grown in warm and humid climates. Aflatoxin can be found in more temperate areas, as was seen in the drought year of 1988 when aflatoxin was seen in 5% of corn grain in the Midwestern U.S. (Russell et al., 1991). The US General Accounting Office has concluded that industry, federal and state programs are effective in detecting and controlling aflatoxin and that it is doubtful that additional programs or limits would reduce the risk of aflatoxin in the food supply. FDA actions levels for aflatoxin are presented in table 2 (Henry, 2006). Aflatoxin regulations worldwide have been reviewed by Van Egmond and Jonker (2005).

Table 2. Action levels for total aflatoxins in livestock feed, (Henry, 2006)

Class of Animal	Feed	Aflatoxin Level
Finishing beef cattle	**Corn and peanut products**	**300 ppb**
Beef cattle, swine or poultry	**Cottonseed meal**	**300 ppb**
Finishing swine over 100 lb.	**Corn and peanut products**	**200 ppb**
Breeding cattle, breeding swine and mature poultry	**Corn and peanut products**	**100 ppb**
Immature animals	**Animal feeds and ingredients, excluding cottonseed meal**	**20 ppb**
Dairy animals, animals not listed above, or unknown use	**Animal feeds and ingredients**	**20 ppb**

Deoxynivalenol (DON) or Vomitoxin

Deoxynivalenol is a *Fusarium* produced mycotoxin, commonly detected in feed. It is sometimes called vomitoxin because it was associated with vomiting in swine. Surveys have shown DON to be associated with swine disorders including: feed refusals, diarrhea, emesis, reproductive failure, and deaths. The impact of DON on dairy cattle is not established, but clinical data show an association between DON and poor performance in dairy herds (Whitlow et al., 1994). Dairy cattle consuming diets contaminated primarily with DON (2.5 ppm) have responded favorably (1.5 kg milk, $P<.05$) to the dietary inclusion of a mycotoxin binder, providing circumstantial evidence that DON may reduce milk production (Diaz et al., 2001). Field reports help substantiate this association (Gotlieb, 1997 and Seglar, 1997). Results from a Canadian study using 6 first-lactation cows per treatment during mid-lactation (average 19.5 kg milk) showed that cows consuming DON contaminated diets (2.6 to 6.5 ppm) tended ($P<0.16$) to produce less milk (13% or 1.4 kg) than did cows consuming clean feed (Charmley et al., 1993). DON had no effect on milk production in 8 cows fed over a 21 day period (Ingalls, 1996). DON has been associated with altered rumen fermentation (Seeling et al., 2006) and reduced flow of utilizable protein to the duodenum (Danicke et al., 2005). Deoxynivalenol decreases humoral and cell-mediated immunity and reduces host resistance (Bondy and Pestka, 2000). Beef cattle and sheep have tolerated up to 21 ppm of dietary DON without obvious effects (DiCostanzo et al., 1995).

The presence of DON may serve as a marker, indicating that feed was exposed to a situation conducive for mold growth and possible formation of several mycotoxins. Like other mycotoxins, pure DON added to diets, produces less toxicity than does DON from naturally contaminated feeds, perhaps due to the presence of multiple mycotoxins in naturally contaminated feeds. In many cases, mycotoxins interact to produce symptoms different or more severe than expected. For example, fusaric acid interacts with DON to cause the vomiting effects, which earlier was attributed to DON alone (Smith and MacDonald, 1991). Advisory levels for DON are provided by FDA as shown in table 3, (Henry, 2006).

Table 3. Advisory levels for deoxynivalenol (vomitoxin) in livestock feed, (Henry, 2006)

Class of Animal	Feed Ingredients & Portion of Diet	DON Levels in Grains & Grain By-products and (Finished Feed)
Ruminating beef and feedlot cattle older than 4 months	**Grain and grain by-products not to exceed 50% of the diet**	**10 ppm (5 ppm)**
Chickens	**Grain and grain by-products not to exceed 50% of the diet**	**10 ppm (5 ppm)**
Swine	**Grain and grain by-products not to exceed 20% of the diet**	**5 ppm (1 ppm)**
All other animals	**Grain and grain by-products not to exceed 40% of the diet**	**5 ppm (2 ppm)**

T-2 Toxin (T-2)

T-2 toxin is a very potent *Fusarium* produced mycotoxin that occurs in a low proportion of feed samples (<10%). Russell et al. (1991) found 13% of Midwestern corn grain contaminated with T-2 toxin in a survey of the 1988 drought damaged crop. Effects of T-2 are less well established in cattle than in laboratory animals. In dairy cattle, T-2 has been associated with gastroenteritis, intestinal hemorrhages (Petrie et al., 1977; Mirocha et al., 1976) and death (Hsu et al., 1972 and Kosuri et al., 1970). Dietary T-2 toxin at 640 ppb for 20 days resulted in bloody feces, enteritis, abomasal and ruminal ulcers, and death (Pier et al., 1980). Weaver et al. (1980) showed that T-2 was associated with feed refusal and gastrointestinal lesions in a cow, but did not show a hemorrhagic syndrome. Kegl and Vanyi (1991) observed bloody diarrhea, low feed consumption, decreased milk production, and absence of estrous cycles in cows exposed to T-2. Serum immunoglobulins and complement proteins were lowered in calves receiving T-2 toxin (Mann et al., 1983). Gentry et al. (1984) showed a reduction in white blood cell and neutrophil counts in calves. McLaughlin et al. (1977) found that the primary basis of T-2 reduced immunity is reduced protein synthesis. Guidelines for T-2 toxin are not established, but avoiding levels above 100 ppb may be reasonable. Diacetoxyscirpenol,

HT-2 and neosolaniol may occur along with T-2 toxin and cause similar symptoms. The FDA has established no guidelines for T-2 toxin in feedstuffs.

Zearalenone (ZEA)

Zearalenone is a *Fusarium* produced mycotoxin that has a chemical structure similar to estrogen and can produce an estrogenic response in animals. Zearalenone is associated with ear and stalk rots in corn and with scab in wheat. Controlled studies with ZEA at high levels have failed to reproduce the degree of toxicity that has been associated with ZEA contaminated feeds in field observations. A controlled study with non-lactating cows fed up to 500 mg of ZEA (calculated dietary concentrations of about 25 ppm ZEA) showed no obvious effects except that corpora lutea were smaller in treated cows (Weaver et al., 1986b). In a similar study with heifers receiving 250 mg of ZEA by gelatin capsule (calculated dietary concentrations of about 25 ppm ZEA), conception rate was depressed about 25%; otherwise, no obvious effects were noted (Weaver et al., 1986a). Field reports have related ZEA to estrogenic responses in ruminants including abortions (Kallela and Ettala, 1984; Khamis et al., 1986; Mirocha et al., 1968; and Mirocha et al., 1974). Symptoms have included vaginitis, vaginal secretions, poor reproductive performance, and mammary gland enlargement of virgin heifers. In a field study, (Coppock et al., 1990) diets with about 660 ppb ZEA and 440 ppb DON resulted in poor consumption, depressed milk production, diarrhea, increased reproductive tract infections, and total reproductive failure. New Zealand workers (Towers et al., 1995) have measured blood ZEA and metabolites ("zearalenone") to estimate ZEA intake. Dairy herds with low fertility had higher levels of blood "zearalenone". Individual cows within herds examined by palpation and determined to be cycling had lower blood "zearalenone" levels than did cows that were not cycling. In this study, reproductive problems in dairy cattle were associated with dietary ZEA concentrations of about 400 ppb. The FDA has established no guidelines for zearalenone in feed, such that any contamination issue is dealt with on a case by case basis (Henry, 2006).

Fumonisin (FB)

Fumonisin B1 produced by *F. verticillioides*, was first isolated in 1988. It causes leukoencephalomalacia in horses, pulmonary edema in swine, and hepatotoxicity in rats. It is carcinogenic in rats and mice and may be a promoter of esophageal cancer in humans (Rheeder et al., 1992). Fumonisins are structurally similar to sphingosine, a component of sphingolipids, which are in high concentrations in certain nerve tissues such as myelin. Fumonisin toxicity results from blockage of sphingolipid biosynthesis and thus degeneration of tissues rich in sphingolipids.

While FB1 is much less potent in ruminants than in hogs, it has now been shown toxic to sheep, goats, beef cattle, and dairy cattle. Osweiler et al. (1993) fed 18 young steers either 15, 31, or 148 ppm of fumonisin in a short term study (31 days). The 148 ppm level resulted in mild liver lesions in two of six calves, along with lymphocyte

blastogenesis and mild liver damage. Dairy cattle (Holsteins and Jerseys) fed diets containing 100 ppm fumonisin for approximately 7 days prior to freshening and for 70 days thereafter demonstrated lower milk production (6 kg/cow/day), explained primarily by reduced feed consumption (Diaz et al., 2000). Increases in serum enzyme concentrations suggested mild liver disease. Because of greater production stress, dairy cattle may be more sensitive to fumonisin than are beef cattle.

Fumonisin carryover from feed to milk is thought to be negligible (Scott et al., 1994). A USDA, APHIS survey of 1995 corn from Missouri, Iowa, and Illinois found that 6.9% contained more than 5 ppm fumonisin B1. Fumonisin was prevalent in Midwestern corn from the wet 1993 season as well. Corn screenings contain about 10 times the fumonisin content of the original corn. Fumonisin guidance levels are in table 4, (Henry, 2006).

Table 4. Guidance levels for total fumonisins in animal feeds, (Henry, 2006)

Class of Animal	Feed Ingredients & Portion of Diet	Levels in Corn & Corn By-products	Levels in Finished Feeds
Equids and Rabbits	**Corn and corn by-products not to exceed 20% of the diet ****	**5 ppm**	**1 ppm**
Swine and Catfish	**Corn and corn by-products not to exceed 50% of the diet****	**20 ppm**	**10 ppm**
Breeding Ruminants, Breeding Poultry and Breeding Mink*	**Corn and corn by-products not to exceed 50% of the diet****	**30 ppm**	**15 ppm**
Ruminants ≥3 Months Old being Raised for Slaughter and Mink being Raised for Pelt Production	**Corn and corn by-products not to exceed 50% of the diet****	**60 ppm**	**30 ppm**
Poultry being Raised for Slaughter	**Corn and corn by-products not to exceed 50% of the diet****	**100 ppm**	**50 ppm**
All Other Species or Classes of Livestock and Pet Animals	**Corn and corn by-products not to exceed 50% of the diet****	**10 ppm**	**5 ppm**

***** Includes lactating dairy cattle and hens laying eggs for human consumption.
** Dry weight basis.

Ergot alkaloids, including fescue toxicity

One of the earliest recognized mycotoxicoses is ergotism caused by a group of ergot alkaloids. They are produced by several species of *Claviceps*, which infect the plant and produce toxins in fungal bodies called sclerotia or ergots, which are small black colored

bodies similar in size to the grain. Ergotism primarily causes a gangrenous or nervous condition in animals. Symptoms are directly related to dietary concentrations and include reduced weight gains, lameness, lower milk production, agalactia and immune suppression (Robbins et al., 1986). Sclerotia levels above 0.3% are related to reproductive disorders.

Fescue grass infected with *Neotyphodium* or *Epichloe* can contain ergot alkaloids and cause "fescue toxicity" (Bacon, 1995). Animal symptoms are lower weight gains, rough hair coat, elevated body temperature, agalactia, reduced conception, and gangrenous necrosis of the extremities such as the feet, tail and ears. Fescue is a major pasture grass throughout the lower Midwest and upper South and over half is thought to be infected. More than 20% of US beef cattle graze fescue, making this a serious problem for cattle producers.

Ochratoxin A

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). The primary toxic effect is inhibition of protein synthesis (Creppy et al., 1984). In cattle, OTA was shown to be degraded in the rumen and thus thought to be of little consequence unless consumed by young pre-ruminant calves (Sreemannarayana et al., 1988), however, chronic exposure and acute toxicities are thought to occur is cattle. With high-grain diets, less of the dietary ochratoxin may be degraded in the rumen and may be more toxic (Hohler, et al., 1999). Moldy forage containing OTA was implicated in cattle abortions (Still et al., 1971) and deaths (Vough and Glick, 1993). The FDA has no guidelines for ochratoxin in feed, but deals with contamination issues on a case by case basis (Henry, 2006).

PR toxin

PR toxin is one of the several mycotoxins produced by *Penicillium* molds. Penicillium grows at a low pH and in cool damp conditions and has been found to be a major contaminant of silage. PR toxin, produced by *P. roquefortii*, is highly toxic and has been suggested as the causative agent associated with moldy corn silage problems (Seglar 1997 and Sumarah et al., 2005). Surveys of grass and corn silage in Europe have found an occurrence of *P. roquefortii* in up to 40% of samples (Auerbach, 2003) and associated with cattle disorders (Boysen et al., 2000). PR toxin, caused acute toxicity in mice, rats and cats by increasing capillary permeability resulting in direct damage to the lungs, heart, liver and kidneys (Chen et al., 1982) and was the suspected vector in a case study with symptoms of abortion and retained placenta (Still et al., 1972). Other *Penicillium* produced mycotoxins in silages, such as roquefortine C, and mycophenolic acid have been associated with herd health problems (Auerbach, 1998; Scudamore and Livesay, 1998, and Sumarah et al., 2005).

Patulin

Patulin is produced by *Penicillium*, *Aspergillus* and *Byssochlamys* (CAST, 2003). Patulin is most likely to occur in moldy fruits (apples), but may also be found in grains, especially wet grains, and silage. Patulin is antibiotic against gram-positive bacteria. Added to rumen continuous cultures at 0, 20, 40 or 80 mg per day, patulin reduced VFA production, fiber digestion and bacterial yield (Tapia et al., 2005). The potential for patulin toxicity of livestock is probably low, but there are field reports of toxicity (Sabater-Vilar et al., 2004).

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