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Mycotoxins are secondary metabolites produced by over 100 different molds that can cause reduced feed intake and milk production, disease infestation, reproductive problems and death in livestock. In addition, certain mycotoxins can be transmitted from livestock diets to animal products and therefore pose a food safety hazard. Mycotoxins may be carcinogenic, mutagenic (cause mutations), neurotoxic, immunotoxic, oestrogenic, teratogenic (embryotoxic or fetotoxic agent) or neurotoxic (Yiannikourisa and Jouany, 2002).

Mycotoxins can act by altering nutrient content, absorption and metabolism, modifying enzyme, endocrine and neuroendocrine function, and suppressing immunity (CAST, 1989). Diseases caused directly by molds are called mycoses, while those caused by mycotoxins are mycotoxicoses. It is typically difficult to diagnose mycotoxicoses because of the difficulty of representatively sampling feedstuffs, limited knowledge about the role of mycotoxins in disease incidence, similarity between mycotoxin-induced symptoms and those from other pathogens, interaction between mycotoxins and the cost and complexity of mycotoxin analysis (Whitlow, 1993). Mycotoxins should not be implicated in causing a disease unless the disease: 1) is feed related; 2) is not contagious, transferable or infectious; 3) can not be associated with a pathogenic microorganism; 4) can not be cured by therapeutic drugs and antibiotics; 5) symptoms disappear when the contaminated feed is withdrawn; and 6) feed analysis confirms the presence of mycotoxins that are known to cause the disease symptoms (Robb, 1990).

Figure 24: Appearance of Common Forage Molds



Molds occur in a variety of colors (Figure 24), but the color or level of mold infestation does not dictate the type or level of mycotoxin contamination; mycotoxins can be present even when molds are not

visible. However, visibly moldy forages should not be fed due to the possible presence of mycotoxins and since most of the feed value has already been lost. Molds thrive in the presence of oxygen, therefore mycotoxin production can occur during plant growth in the field, or during the stages of silage making or storage that allow air ingress into the silo. Delayed harvesting, slow or delayed filling, inadequate packing and sealing, slow feedout rates and damaged bunker or bag plastic can lead to pockets of mycotoxin production where the presence of oxygen and a conducive microclimate allow mold proliferation (Whitlow, 1993).

The most common mycotoxins in forages include aflatoxin, deoxynivalenol (DON), zearalenone (ZEN), T-2 toxin and fumonisin. Their occurrence in various feeds in a nine-year North Carolina survey is shown in Table 1. These mycotoxins are mainly produced by *Aspergillus* molds which require warm, humid conditions, and *Penicillium* and *Fusarium* molds which require moist, humid, cool conditions. *Fusarium* ear and stalk rot and head blight is also common in corn grown in warm, humid climates. Other factors that predispose to mold growth and mycotoxin production include insect, rodent, rain, hail and lodging damage, drought and floods. These factors create entry points for fungal spores that produce mycotoxins.

Ruminants are more tolerant to some mycotoxins due to detoxification in the rumen, however the increased rumen passage rates of today's high producing dairy cattle may reduce the detoxifying influence of the rumen (Seglar, 2001). Generally, mycotoxins that have a cyclic lactone ring (Figure 2) are quite susceptible to hydrolysis in the rumen.

The following section classifies mycotoxins based on their mold source, and outlines their debilitating effects on livestock, toxic levels in feed and where known, their fate in silage and animals.

ASPERGILLUS TOXINS

Aflatoxins

Aflatoxins are some of the most common and potent forage mycotoxins. Their production is favored by high humidity (>80%) and temperature (>90 °F), insect damage and drought stress (DeWolf et al., 2005). These carcinogenic toxins are mainly produced by *A. flavus*, *A. parasiticus* and *A. fumigatus*, soil-borne molds that thrive in

nutrient dense environments, particularly after a drought or in warm conditions. Symptoms include inappetance due to reduced digestion, reduced rumen motility and fermentation, liver damage, ataxia, rough hair coat, delayed blood clotting and reduced immunity (Deikman and Green, 1992). Aflatoxins are classified based on the blue and green fluorescence that develops when they are viewed under ultraviolet light into B1, B2, B2a, G1, G2 and Ga. Aflatoxins do not have a lactone ring and generally are poorly degraded in the rumen and low concentrations can inhibit rumen bacterial growth (Yiannikourisa and Jouany, 2002). The most toxic and widespread group is B1, which is excreted in milk as aflatoxin M1. Milk M1 concentrations are usually 1.7% of the aflatoxin B1 concentration in the total ration dry matter (Whitlow, 2005). Levels of B1 above 100 ppb can compromise the performance of dairy cattle, and cause kidney damage in beef cattle (Garett et al., 1968; Whitlow, 2005). Aflatoxin is the only mycotoxin with Food and Drug Administration (FDA) action levels in the US. These are 20 ppb in feeds and 0.5 ppb in milk.

Other potent *Aspergillus* mycotoxins whose roles in the aetiology of mycotoxicoses in animals are not understood include:

Fumitremoregens e.g. fumigaclavine A and B from *A. fumigatus*, common in southeast silages cause anorexia, diarrhea, unthriftiness and irritability (Cole et al., 1977, cited by Whitlow, 1993). They are called tremorgens because the toxins cause trembling due to neurotoxicity.

Sterigmatocystin, produced by *A. versicolor*, has been associated with bloody diarrhea and death in cattle (Whitlow and Hagger, 2004).

Glitoxin, produced by *A. fumigatus* and some other *Penicillium* molds has been associated with gastroenteritis and Hemorrhagic Bowel Syndrome in dairy cows.

FUSARIUM TOXINS

Fusarium mycotoxins include several toxins that infest plants in the field and survive during ensiling. They include tricothecenes, which are about 150 structurally related compounds produced by several fungi including *F. sporotrichioides* and *F. graminearum*. Some of the most potent tricothecenes produced in conserved forages are deoxynivalenol and T-2 toxin, though diacetoxyscirpenol (DAS), nivalenol, neosolalaniols and hydroxyl-T-2 can also occur.

Deoxynivalenol (DON)

Also known as vomitoxin because it often causes vomiting, DON is one of the most commonly found mycotoxins in conserved forages, and is often produced along with other mycotoxins. Consequently it is often used as a marker for the presence of other mycotoxins. It is produced by *Fusarium roseum* or *graminearum* (*Gibberella zeae*) often when a cold, wet spell is followed by a short, dry period (Diekman and Green, 1992). It is also prevalent when wet conditions coincide with warm days and cool nights. Symptoms include feed refusal, reduced milk production, emesis (vomiting), unthriftiness, immunosuppression or immunoexcitation, diarrhea, emaciation, reproductive failure and death (Whitlow, 1993; Rotter et al., 1996). DON inhibits protein synthesis and alters brain chemicals involved in serotonin production (Rotter et al., 1996).

DON is extensively degraded in the rumen (50% in 24 h) into much less harmful products. It is excreted mainly through the urine and is there is little transfer to milk (Cote et al., 1986 and Prelusky et al., 1984; Yiannikourisa and Jouany, 2002). The FDA stipulates advisory levels of 1 ppm for finished wheat products for human consumption, and 5 -10 ppm for grains and grain by-products destined for cattle consumption, provided the contaminated feed is less than 50% of the diet. Beef cattle have tolerated feeds with up to 20 ppm DON, but research on effects of low levels (2 - 6 ppm) on milk production in dairy cows is not conclusive.

Zearalenone (ZEN)

Zearalenone is an estrogen-like compound that is mainly produced by *F. graminearum* and *F. sporotrichioides*. Moist conditions with alternating low (53-57 °F) and moderate (81 °F) temperatures favor its production (De Wolf et al, 2005). Its structural similarity to estrogen, and ability to mimic this hormone, leads to infertility, prolonged oestrus, reduced conception rates, decreased litter size, rectal or vaginal prolapse and malformed offspring and abortions (Mirocha and Christensen, 1974; Whitlow, 1993). Other symptoms include reduced feed intake and milk production, and diarrhea. About a third of the ZEN intake is ruminally degradable, however metabolites of ZEN can be more, or less, toxic than the parent toxin. A diet to milk carry-over rate of 0.06% of the dietary dose has been reported when 544 mg of ZEN was ingested daily for 21 days. However ingestion of

much higher, single doses produced negligible transfer to milk (Yiannikourisa and Jouany, 2002). Levels of 12 and 50 ppm can reduce conception in virgin heifers and dairy cows, respectively (DeWolf et al., 2005).

Fumonisin

Fumonisin B1 (FB1) and B2 (FB2) are the most important of a group of at least 15 recently discovered silage toxins. Produced by *F. moniliforme* and *F. proliferatum*, they are estrogenic and carcinogenic in humans and act by blocking biosynthesis of important membrane lipids, resulting in cell dysfunction and death (Yiannikourisa and Jouany, 2002). Hot, dry periods followed by humid conditions and insect damage favor their production of this toxin (DeWolf et al. 2005). Symptoms include leucoencephalomalacia in horses, and inappetance and liver damage in ruminants (DeWolf et al., 2005). Excretion of fumonisin in milk is thought to be negligible (Scott et al., 1994, cited by Whitlow, 2005), though a carryover rate of 0.05% was reported when the diet contained 3 ppm of FB1 toxin (Yiannikourisa and Jouany, 2002). The FDA stipulates guidance levels for total fumonisin concentration in contaminated corn and corn by-products of no more than 2 - 4 ppm in human food, 30 ppm in feed for breeding ruminants, and 60 ppm in feed for calves over 3 months that are raised for slaughter. Levels of contaminated corn and corn by-products in ruminant rations should also not exceed 50% (dry matter basis) of the ration.

T-2 Toxin

T-2 is a common contaminant of feeds and is more prevalent in wet, warm (60° -89°F) conditions. Symptoms include feed refusal, perineal and pharyngeal irritation, reduced immunity, gastroenteritis, hemorrhage of the gastrointestinal tract, diarrhea, infertility and death. It is potentially harmful to cattle at levels of 0.7 - 1.5 ppm in the ration (DM basis, Adams, 1995). T-2 is degraded in the rumen to metabolites that are less toxic, but still poisonous (Whitlow, 1993). Between 0.05 and 2% of dietary T-2 can be excreted in milk and the lethal dose in cattle is more than 13 mg/kg body weight (Yiannikourisa and Jouany, 2002).

Fusaric acid

Fusaric acid is produced by *F. moniliforme*, *F. proliferatum* and several

other *Fusarium* species. It inhibits plant growth and blocks Dopamine β-hydroxylase in the nervous system which is integral for the flight or fight response in animals (Angel, 1998). It is often found with tricothecenes like DON, and it increases the toxicity of such toxins.

PENICILLIUM TOXINS

Ochratoxin

This toxin is produced by *Aspergillus ochraceus*, *A. clavatus* and *Penicillium verrucosum* particularly when temperatures range between 68 - 77°F and moisture content exceeds 16%. Ochratoxins A and B occur naturally but the former is more widespread. It is carcinogenic and immunotoxic and it impairs enzyme and kidney function, inhibits cellular respiration and glucose metabolism and therefore increases the incidence of fatty liver syndrome and retards growth (Whitlow, 1993; Yiannikourisa and Jouany, 2002). Nevertheless, the toxin can be ruminally degraded into a less toxic product (Hult, et al., 1976). However, when the detoxifying capacity of the rumen is exceeded, (>1.7 mg/kg body weight), the toxin can be detected in milk and symptoms have included diarrhea, kidney damage and reduced milk production (Whitlow, 1993; Yiannikourisa and Jouany, 2002).

Other potent *Penicillium* mycotoxins which have poorly understood effects on animal performance and health include:

Secalonic acid, produced by *P. oxalicum* is a tetratoxin, causes malformations at birth.

Patulin is produced by *P. expansum*, *P. urticae*, *A. clavatus* and *Byssoschlamys nivea* (Yiannikourisa and Jouany, 2002). It is commonly found in deteriorating silage and can reduce digestion of protein, fiber and organic matter, alter rumen volatile fatty acid profile and kill cows (Seglar, 2001). It is carcinogenic and mutagenic, and also causes lack of coordination of motor organs, gastric paralysis and death (Yiannikourisa and Jouany, 2002).

PR toxin and Roquefortin are tremorgens produced by *P. roqueforti* that cause rumen stasis, digestive upsets, abortion and retained placenta (Whitlow, 1993). However they have relatively low stability in the silo (Yiannikourisa and Jouany, 2002). There is some evidence from Europe that *P. roqueforti* is more predominant in silage than other mold species.

PREVENTING MYCOTOXICOSES

Complete detoxification of silage contaminated with mycotoxins is not practical. Since mycotoxins are produced by molds which depend on oxygen for growth, any steps that ensure and accelerate oxygen removal from silage at filling or minimize oxygen ingress into silos are very important. The following agronomic and silage management practices should be ensured to minimize mold growth and mycotoxin production:

- ▶ Planting insect and disease-resistant varieties and practicing crop rotation.
- ▶ Avoiding or minimizing the effects of plant stressors (e.g. inadequate fertilization, insect, bird or hail damage, lodging, flooding and drought) that predispose to mold infestation and mycotoxin production.
- ▶ Timely harvesting that avoids ensiling mature, dry forages that are difficult to pack.
- ▶ Using proven additives to minimize mold growth such as *Lactobacillus buchneri* inoculants or organic acids like propionic acid. It is important to follow manufacturer's recommended application rates when using organic acids or the situation can actually be made worse (Figure 25).
- ▶ Using sharp knives at harvest to enhance packing, aiming for a fill rate of 1 minute/ton and a packing density of at least 14 lb/ft³.
- ▶ Cleaning bunkers prior to use and sealing silos promptly on the day they are filled.
- ▶ Weighing down plastic adequately (> 20 tires/100 square feet, Bolsen and Bolsen, 2004)
- ▶ Inspecting bag or bunker plastic for holes regularly and sealing these promptly with proper silage tape.
- ▶ Feeding at least 6 inches of silage per day to minimize exposed faces that are undisturbed for days.
- ▶ Maintaining a straight silo face with shavers or block cutters.
- ▶ Cleaning feedbunks and discarding contaminated silage.

SUGGESTED TREATMENTS FOR MYCOTOXICOSES

- ▶ Withdraw the problem silage from the ration where possible.
- ▶ Dilute the amount of the problem silage fed.
- ▶ Use a proven mycotoxin absorbent such as clays, aluminosilicates, glucomannans or mannanoligosaccharides. However, such commercial products differ in efficacy, and do not bind all mycotoxins equally well. Certain binders may reduce bioavailability of minerals and vitamins in the diet. Only products that have been shown to be effective in independent research trials should be used.
- ▶ Ensure the levels of dietary antioxidants (vitamins A and E, selenium and zinc) are adequate.
- ▶ Ensure the ration is balanced to provide adequate nutrients.

Figure 24: The Influence of Propionic Acid on Aflatoxin

