MYCOTOXINS IN SILAGE

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Introduction

Mycotoxins are secondary metabolites secreted by molds mostly belonging to the Aspergillus, Penicillium and Fusarium genera (Yiannikouris and Jouany, 2002). The ubiquitous nature of mycotoxins and the severity of their effects on human health make them a major food safety concern. The Food and Agriculture Organization (FAO) estimates that 25% of all crops are contaminated with mycotoxins (CAST, 1989). Direct costs of disposal of condemned food and feed ingredients and indirect costs of regulatory enforcement and quality control measures caused by fungal toxin contamination in the US were estimated at approximately $1.4 billion (CAST, 2003). The social importance of mycotoxins are evident from the 25,000 to 166,000 new causes of liver cancer annually caused by aflatoxins (Liu and Wu, 2010).

Mycotoxins also negatively affect domestic animals causing suppression of the immune system, imbalance of hormonal function, and reduction of nutrient utilization, which result in decreased performance and eventually death (Whitlow and Hagler, 2005). For the farm system, the costs associated with feeding animals contaminated diets occur in the form of poor animal performance and milk disposal due to high concentration of toxins (Applebaum et al., 1982; Masoero et al., 2007; Kutz et al., 2009). Milk with aflatoxin concentrations above 0.05 µg/kg are considered illegal to be marketed in USA, Brazil, Argentina, and countries belonging to the southern common market while on Europe the action level is 0.05 µg/kg (FDA, 2000; ANVISA, 2002; EFSA, 2004a).

Silage making is based on the principle of forage conservation under anaerobic conditions. Epiphytic or inoculated lactic acid bacteria naturally ferment carbohydrates in the forage and produce acids, which reduce the pH and consequently reduce the growth of undesirable microorganisms (Garon et al., 2006). Anaerobiosis and low pH inhibit the growth of most of fungi, thus ensiling is an effective strategy to prevent the growth of and mycotoxin production from many field and storage fungi. However, the conditions that prevent the growth of the majority of fungi in silage also provide a more conducive environment for the growth of acid and low oxygen-tolerant species, such as *Penicilium roqueforti, Aspergilus*

fumigatus, *Byssochlamys nivea*, and various Fusarium species (Pahlow et al., 2003). Gonzales-Pereyra et al. (2008) also noted that inadequate management of silage can impair fermentation, promote aerobic conditions within the silage mass, and favor the growth of fungi that are normally less tolerant of acidic or anaerobic conditions such as *Aspergillus flavus*. Furthermore, once silos are opened, the ensuing aerobic conditions often allow the growth of spoilage-causing, yeasts, which metabolize lactate to CO_2 and thereby increase the silage pH. This elevated pH predisposes toxigenic fungi to grow actively, particularly in poorly managed silages during the feed-out phase.

Mycotoxin contamination is a ubiquitous problem due to the high adaptive capacity of toxigenic fungi, which allows them to proliferate in all stages of feed production such as on the field and at harvest, storage, and feed-out. In spite of their adaptive capacity, various techniques can be used to prevent or at least minimize contamination of feeds with mycotoxins and effective detoxification methods are also available. This review intends to briefly discuss some of the mycotoxins frequently found in silages and to discuss strategies for preventing mycotoxin contamination or decontaminating mycotoxin-infested feed ingredients.

Mycotoxins

Mycotoxins cause several undesirable effects in humans and animals. More than 400 mycotoxins are known to occur naturally, however, only a few of them have been extensively studied (Whitlow and Hagler, 2005). Mycotoxins that are frequently present in silages include deoxynivalenol (DON), zearalenone (ZEA), fumonisin, roquefortine C. Aflatoxin can also be present in silages made in hot and humid environments.

Deoxynivalenol

Deoxynivalenol (DON) is a toxin produced by Fusarium species (F.graminearum, F. sporotrichioides, F. culmorum, F. poae, F. roseum, F. tricinctum) it is also known as vomitoxin because it tends to cause emesis in swine. Other than vomiting, DON causes feed refusal, diarrhea, reproductive problems, and eventually death in non-ruminant animals. The effect of DON in dairy cattle is not well established but decreases in animal performance in dairy herds have been associated with the toxin (Whitlow et al., 1994). Deoxynivalenol has also been related to altered rumen fermentation (Seeling et al., 2006) and reduced flow of protein to the duodenum (Danicke et al., 2005). In a survey on the presence of mycotoxins in

feed ingredients, Driehuis et al. (2008) observed that corn silage was the main source of DON and zearalenone (ZEA) in diets of dairy cattle. These authors reported an average concentration of DON in corn and grass silage samples of 550 µg/kg and a maximum concentration of 1,250 µg/kg. They estimated that these high DON concentrations could result in intakes of 8.4 µg of DON /kg of BW or 5 mg per cow/day. After reviewing several studies, DiCostanzo et al., (1995) concluded that beef cattle are able to tolerate up to 21 mg/kg of DON. Charmley et al. (1993) demonstrated that a contamination level of 6 mg of DON /kg of diet DM did adversely affect on milk yield or cause carry-over of the toxin into milk. Data available on adverse effects of DON on dairy cattle is limited, and insufficient to allow the establishment of a maximum tolerance level, however the guidance value is 5mg/kg (European commission, 2006, Driehuis et al., 2008). The Food and Drug Administration (FDA) advisory guideline for DON are 10 and 5 mg/kg DM of diet for beef and dairy cattle respectively (FDA, 2010). The concern about the intake of DON by dairy cattle is related to its' potential negative effect on animal health and production, however due to the low transfer of this toxin to milk, DON is not considered a contaminant that reduces the safety of dairy products (EFSA, 2004b).

Fumonisin

Fumonisins are mainly produced by two species of Fusarium, *F. verticilloides* (formely *F. moniliforme*) and *F. proliferatum* (Whitlow and Hagler, 2005). The structural formula of fumonisin is similar to sphingosine, which is a component of sphingolipids that abound in nerve tissue. The toxicity of this mycotoxin results from interruption of sphingolipid biosynthesis, therefore it causes paralysis, nervousness, and ataxia in horses, and pulmonary edema in swine (Marasas et al., 1988; Ross et al., 1990; Diaz and Borermans, 1994). The toxin tends to be less aggressive in ruminants than monograstrics, however fumonisin has been shown to be hepatoxic and nephrotoxic to calves receiving 1 mg/kg body weight of the toxin (Mathur et al., 2001). The latter authors reported hepatocellular apoptosis and renal tubular necrosis within 7 days of dosing the toxin. Similar results were found in a study with beef calves supplemented with 148 mg/kg of total fumonisin in the diet for 31 days (Osweiler et al., 1993). Diaz et al. (2000) demonstrated that dosing of 100 mg/kg of fumosin reduced feed intake and milk yield in dairy cattle. Nevertheless, carry-over of the toxin into milk is minimal (Scott et al., 1994).

Gonzalez-Pereyra et al. (2008) reported that fumonisin levels in corn silage varied from 340 to 2490 µg/kg DM. These authors reported that samples from different locations in the silo had different concentrations of the toxin and those from the top layer and side walls had high pH values, which could favor development of the toxin.

Fusarium species tolerate low winter temperatures and colonize crop residues such as maize stalks and rice and wheat stubble, and these become major sources of inocula as temperatures increase in early spring (Binder, 2010). Fungal spores also become are airborne during rainy season and can travel long distances causing mycotoxin contamination epidemics (Binder, 2010; Lanier et al., 2010). The Food and Drug Administration (FDA) advisory guideline for fumonisim is 15 mg/kg DM of diet for lactating dairy cows (FDA, 2001).

Zearalenone

Zearalenone is an estrogenic metabolite produced by several species of Fusarium such as F. graminearum, F. culmorum, f. crookwellense (Saeger et al., 2003). Despite of the structural dissimilarity between zearalenone and steroidal estrogens, ZEA can cause numerous reproductive problems including, hyperestrogenism, mammary gland enlargement, and vaginitis (Diekman and Green, 1992). Ruminants are less susceptible than pigs or chickens due to conversion of ZEA to its' hydroxyl-metabolites, α alpha and β zearalenol, by ruminal flora (Kiessling et al., 1984; Kennedy et al., 1998). Although α-zearalenol is three to four times more estrogenic than ZEA, its lower rate of absorption and interconversion to β zearalenol in the liver helps to decrease negative effects of ZEA (Fink-Gremmels, 2008). Despite their lower susceptibility to this toxin, studies show that high levels of the toxin may negatively affect dairy cattle. For instance, conception rates were decreased by 25% in dairy heifers receiving 250 mg of ZEA (Weaver et al., 1986).

Whitlow and Hagler (2005) reported a 30% incidence of ZEA contamination in 461 corn silage samples and the average contamination level was 525 µg/kg DM. Driehuis et al. (2008) reported ZEA contamination in 13 and 50% of grass and corn silage samples with average concentrations of 180 and 146 µg/kg respectively. Reed and Moore (2009) reported that sorghum silage contained 660 µg/kg of ZEA and concentration up to 79.80 mg/kg in Medicago sativa. The main concerns with this toxin focus on its' negative effect on animal health and reproduction because like fumonisin the level of transfer into milk is considered negligible (Seeling et al., 2005). There are no zearalenone action limits, guidance, or advisory

levels established by FDA at this time. The guidance value for zearalenone in Europe is 500 µg/kg (European commission, 2006).

Aflatoxin

Aflatoxin is a mutagenic, carcinogenic, and toxic secondary metabolite produced *Aspergillus flavus*, *A. parasiticus and A. nomius* (Creppy, 2002). Due to the ability of Aspergillus to grow over a broad range of temperatures and humidities, aflatoxin is a ubiquitous contaminant of food and feed ingredients worldwide (Phillips, 1999). Feeding aflatoxin -contaminated diets to lactating cows reduces their health and performance and also causes transfer of the toxin to milk and dairy products (Diaz et al., 2004).

The symptoms associated with aflatoxin ingestion or inhalation include: inappetence, lethargy, ataxia, enlargement of the liver, decreased rumen motility, and reduced feed efficiency and milk production (Whitlow and Hagler, 2005; Guthrie and Bedell, 1979; Mathur et al., 1975). Aflatoxin also reduces the immune response to opportunistic diseases and interferes with the efficiency of vaccines (Diekman and Green, 1992; Marin et al., 2002). However, these symptoms are not specific to aflatoxicosis, which makes precise diagnosis of the condition difficult (Columbre, 1993). Garret et al. (1968) demonstrated increased liver weight in beef cattle fed a diet containing 100 µg/kg of aflatoxin. Similar concentrations also cause decrease in animal production and compromise animal health (Patterson and Anderson, 1982). Applebaum et al. (1982) showed that high-producing animals are more sensitive to impure aflatoxin contaminated diet than diets contaminated with pure aflatoxin B1. Queiroz et al. (2010) observed increase concentrations of plasma adhesion molecules and haptoblogin, an acute phase protein when dairy cattle were fed an aflatoxin-contaminated diet.

The incidence of aflatoxin is relatively low in silages compared to other mycotoxins, probably due to low tolerance of *Aspergillus flavus* and *A. parasiticus* to the acidic and anaerobic silage environment. However, data in literature report high levels of aflatoxin in poorly made or managed silages or in silages made with diseased corn plants (Queiroz et al., 2009). Gonzalez-Pereyra et al. (2008) reported an aflatoxin B_1 (AFB_1) concentration of 156 µg/kg DM in corn silage stored in a trench type silo without proper sealing. Richard et al. (2009) observed aflatoxin concentrations of up to 60 µg/kg DM in corn silage.

The rate of aflatoxin transfer to the milk varies from 1 to 6% (EFSAa, 2004). Due to the severity of aflatoxin effects on human health, it is the only one of the 400 known mycotoxins that has an action level regulated by governmental agencies. The aflatoxin Action

Level established by the FDA for fluid milk is 0.5 µg/kg and it is 20 µg/kg for feed ingredients offered to dairy cattle. Therefore, their high aflatoxin concentrations would render the corn silages mentioned above unsafe for feeding to dairy cattle.

Factors Affecting the Extent of Mycotoxin Contamination

Mycotoxins are produced through different pathways involved in secondary metabolism by fungi (Steyn, 1998). Pathways involved in secondary metabolism are not essential for cell growth in microorganisms and are not the main synthetic or catabolic pathways. Rather, secondary metabolism processes that result in mycotoxin contamination are caused by interaction and response of fungi to environmental conditions (Yiannikouris and Jouany, 2002). Thus, the extent of feed contamination by mycotoxins is related to environmental stresses and physicochemical parameters such as availability of free-water, prevailing temperature, oxygen, and pH and availability of substrate (Nelson, 1993). Common environmental problems associated with mycotoxin contamination of feed ingredients are: insect or animal damage, plant stress caused by extreme weather conditions such as hail and the attendant lodging, or by contamination with opportunistic fungal diseases (Queiroz et al., 2010). Mycotoxin contamination of silage is frequently associated with inadequate silage management practices.

Preventing Mycotoxin Contamination of Feeds

Preventing mycotoxin contamination is a complex task because many interplaying factors contribute to establishing and predisposing fungi to produce mycotoxins. Risk-management concepts can help to identify control points within the feed production chain that can be monitored to minimize mycotoxin contamination in feed ingredients. As mentioned earlier, infestation by toxigenic fungi can occur in the field, during storage of crop products or when feed is offered to animals. The following section outlines the different phases of feed production and corresponding strategies to prevent mycotoxin contamination.

Field or Pre-harvest stage

Prevention of mycotoxin contamination on the field is desirable, but not always achievable. Agronomic practices to minimize environmental stress and maximize plant performance can significantly decrease mycotoxin contamination (Binder, 2010). Some of

such practices include the use of varieties or hybrids that are adapted to the growing area and resistant to fungal diseases, control of weeds, application of fungicides and pesticides, use of irrigation, timely crop rotation, proper fertilization (Edwards, 2004; Whitlow and Hagler, 2005). Despite their theoretical potential, Duncan et al. (1994) noted that fungicides have shown little efficacy at preventing the growth of *A. flavus* and thereby preventing aflatoxin contamination. It is important to note that no matter how good the management at the field is, no technique completely eliminates the possibility of mycotoxin contamination of crops on the field.

Harvest phase

Crop and forage harvests should be carefully timed to coincide with the recommended maturity for optimizing yield, DM concentration, and nutritive value for each crop. Excess moisture in harvested crops and forages can predispose to spoilage of grains and prevent efficient silage fermentation.

Harvesting equipment should be properly maintained and calibrated to avoid lodging of or physical damage to crops at harvest because contact with soil and kernel damage can predispose to fungal growth and mycotoxin production (Whitlow and Hagler, 2005). Immediate storage of harvested feeds in appropriate storage facilities is also necessary to minimize the risk of mycotoxin contamination.

Post-harvest stage

Proper storage of feeds is critical to prevent the development of mycotoxins. Special attention should paid to the moisture concentration of stored feeds. Moisture levels should be kept under 15% for dried feeds and artificial or natural drying should be employed where necessary. Drying feeds to moisture contents below 13% compensates for uneven moisture concentrations throughout the feed or forage mass (Whitlow and Hagler, 2005).

When poor storage conditions allow fungal growth, it is not uncommon to detect infestation with more than one species because during storage, grains are colonized by a succession of fungi, which result in contamination with different toxins (Binder, 2010). Other factors that should be considered to prevent mycotoxin contamination during storage are appropriate temperature and adequate aeration (Shapira and Paster, 2004). High temperatures increase the water activity leading to mold growth, while inadequate aeration of grains allows moisture accumulation, which increases the chances for spreading of fungi. Special attentions

should be paid to the physical structure of silo to avoid rehydration of the grains by rain or water sources and to prevent damage caused by insects or rodents. As mentioned above, damaged grains favor mycotoxin development thus mechanical separation of broken kernels is recommended.

Strict adherence to excellent silage management principles and practices at all stages of silage making and feeding are essential to prevent mycotoxin contamination of silages. Such principles and practices include the following: Choosing disease resistant hybrids, ensuring that appropriate agronomic practices are employed to minimize lodging, disease infestation or stressing of crops, harvesting at the appropriate maturity stage that optimizes nutritive value, yield and DM concentrations, chopping forages to lengths that facilitate compaction without reducing the effective fiber concentration, filling silos rapidly, packing or consolidating silage to achieve a density of $\geq$ 200 kg/m^3 to reduce porosity and prevent air pockets in the silage, proper management of the silo face and feeding the silage at a rate that minimizes spoilage during feed-out.

The low pH and anaerobic conditions in silage inhibit the growth of most spoilage and toxigenic fungi (Pahlow et al., 2003). Bacterial inoculants and chemical additives have been successfully used to enhance the acidity by promoting a rapid drop in pH at ensiling. Inoculants and additives are also used to increase aerobic stability, and reduce the spoilage fungal population in silages (Huisden et al., 2009; Mari et al., 2009; Pedroso et al., 2010). However, few trials have evaluated if such additives and inoculants can prevent or reduce mycotoxin contamination of silage. Queiroz et al. (2009) reported when corn plants infested with Southern rust disease were ensiled, that aflatoxin concentration was 200 times above the FDA Action Level. However, inoculation of the diseased plants with an inoculant containing *L. buchneri* and *P. Pediococcus* resulted on silages with no aflatoxin. *Lactobacillus buchneri* is a heterofermentative bacteria, which can convert lactic acid to acetic acid and 1,2-propanodiol (Oude Elferink et al., 2001). Acids such as acetate and propionic acid have proven antifungal effects (Moon, 1983) and as such, they are added to silage directly or indirectly through the use of bacterial inoculants to inhibit the growth of yeasts and molds (Kung et al., 2003). Numerous studies demonstrating the antifungal effects of acids, mold inhibitors and inoculants can be found on the literature, however mold inhibitors and inoculants often have no effect on mycotoxins that have been already synthesized (Binder, 2010).

Another strategy to reduce the growth of toxigenic fungi in silage involves limiting oxygen ingress into silos. Amaral et al. (2010) evaluated the efficacy of using traditional

plastic or a film that limits oxygen infiltration for covering silos. They reported that unlike traditional plastic, the new oxygen barrier film inhibited synthesis of aflatoxin in corn silage in Brazil. This experiment emphasizes the importance of maintaining anaerobioc conditions during and after ensiling to avoid mycotoxin contamination of silage.

Mycotoxin Detoxification Methods:

Unquestionably, the feed industry and feed producers should target prevention of mold growth to prevent mycotoxin contamination of feeds. Despite the existence of several notable advances in pre and post-harvest fungal inhibition technologies, feed contamination with mycotoxins is still very common.

Silage traditionally represents a significant proportion of the diet of dairy cows and its' year-round, widespread use in some countries and seasonal use at times of low pasture availability in others, emphasize the importance of ensuring that mycotoxin contamination of silage is minimized. Yet no method effectively or completely detoxifies silages contaminated with mycotoxins. However, numerous products that improve aerobic stability via their antimycotic action may indirectly decrease the risk or level of mycotoxin contamination of silage.

Dilution of contaminated ingredients is an effective decontamination strategy for contaminated grains. However, it is less appropriate for contaminated silage because it would probably reduce the feedout rate and may necessitate partial utilization of the silage on the silo face. These factors would lead to mold growth and intensification of the contamination problem (Whitlow and Hagler, 2005). The other post-harvest methods for detoxifying contaminated feed ingredients can be classified into the following groups:

Biological methods

Microorganisms can reduce mycotoxin production by competition for nutrients and space with toxigenic molds (Bhatnagar et al., 1994). Nontoxigenic strains of *A. flavus* and *A. parasiticus* have been successfully used to compete with wild toxigenic strains and decrease aflatoxin contamination in cottonseed (Cole and Cotty, 1990). Microorganisms also can be used to metabolize and reduce mycotoxins within the environment. Kiessling et al. (1984) reported that ruminal microbes can metabolize ochratoxin A, zearalenone, T-2 toxin, and diacetoxyscirpenol but had no effect on aflatoxin and deoxynivalenol. Binder et al., 2000)

were the first to isolate a pure strain of Eubactarium, referred as BBSH 797 that is able to transform the epoxy group of DON.

Physical Methods

Manual removal of contaminated grains is the simplest method of physical removal of mycotoxins, but it is extremely time consuming and in many cases, impractical. Automated physical removal of moldy, damaged, or inadequately developed kernels and grains is one of the widely used decontamination techniques. Partial removal of mycotoxins is also achieved by cleaning the grain or milling, however this can lead to increased toxin levels in the bran though levels in the flour are decreased (Bata and Lastity, 1999). During milling or dehulling, the external layers of grains, which are more susceptible to fungal action are removed (Siwela et al., 2005). This process results in a toxin concentrated dust by-product (Sorenson et al., 1981), and requires a significant input of energy, which in turn, increases costs and makes the method less suited for on-farm use. Also, because mycotoxins diffuse away from the mycelia region, physical segregation of moldy material does not guarantee efficient removal of aflatoxin (Park et al., 1981).

Mycotoxins can be relatively heat stable; therefore thermal detoxification produces variable results. Rater and Matissek (2008) noted that aflatoxin B_1 inactivation by thermal treatment was affected by the type of contaminated matrix, time of heat exposure, and heat intensity. The authors reported that after 30 min under 150 ^{o}C, 100% of pure AFB_1 had been degraded, however, when the toxin was mixed with carbohydrates, the inactivation efficacy was only 50%. The same authors reported that increasing the temperature to 180 ^{o}C caused a 70% reduction in AFB_1 concentration when the toxin was mixed with carbohydrates during the same time of exposure.

Irradiation is a physical method that reduces infestation by undesirable *Aspergillus* species and decreases contamination with AFB_1. Irradiation of contaminated feed with ultraviolet light, microwaves, or gamma rays have decreased aflatoxin contamination (Yousef, 1986). Corn grain samples contaminated with AFB_1 and AFB_2 were totally decontaminated after exposure to 10 kGV of gamma radiation (Aquino et al., 2005). Ultraviolet light is unquestionably the cheapest method of irradiation. Reduction of AFB_1 and total aflatoxin varied from 60.5 to 75% when contaminated feed samples were subjected to an extensive period (30 h) of sunlight exposure (Herzallah et al., 2008). However most irradiation methods are unaffordable to producers and some are inaccessible. The sunlight

treatment is cheap but only effective to treat grains on the surface of a batch. Therefore only a small amount of feed can be treated at a time. This and the long exposure period make this method impracticable for large quantities of feed. In fact, none of these physical methods is practicable or effective for decontaminating silage on dairy farms due to the large quantities involved.

Chemical methods

About 100 compounds are known to inhibit to some extent aflatoxin production (Zaika and Buchanan, 1987). Most of them work by inhibiting the growth of fungi, inhibiting aflatoxin synthesis, or both. Dichlorvos is an organophosphate insecticide which inhibits aflatoxin synthesis but not fungal growth (Buchanan et al., 1983). Caffeine is also capable of decreasing aflatoxin biosynthesis and it negatively affects mold growth (Buchanan et al., 1983). Rodriguez and Mahoney (1994) reported that non ionic surfactants such as polyoxyethylene 10 lauryl ether reduced the synthesis of aflatoxin from *A. flavus*. Practical use of these chemicals on farms may be restricted due to their costs and harmfulness to human health.

Ammoniation is one of the most widely used methods of decontamination of toxins in commodities and it is accepted as a decontamination practice in many countries including Brazil (CAST, 2003). Ammonia can modify the molecular structure of aflatoxin and decrease aflatoxin concentration in feed ingredients by about 99% (Hoogenboom et al. 2001). The adoption of this technique on farms is highly dependent on training and investment due to the risks associated with this procedure. Ammoniation of forages, though effective and feasible for forage conservation, is not as effective at decontaminating mycotoxins as ammoniation of grains under high pressure and temperature. Also, under high temperatures, ammonia reacts with sugars in forages to form 4-methylimidazole, which causes hyperexcitability in cattle (Kerr et al., 1987).

Ozonation is a very effective method to detoxify contaminated feed. Reduction of 92% of corn contamination with aflatoxin was reported when samples were treated with ozone gas (12% of total weight) for 96 h (Prudente and King, 2002). Total degradation of aflatoxin in corn samples was reported when 20% of ozone was applied (Mckenzie, 1997). The same authors noted that AFB_2 and AFG_2 were more resistant to degradation than the AFB_1 and AFG_1 forms, which are rapidly degradable when ozone is applied at 2% of the sample weight. Ozonation destroys the structure of AFB_1 by interacting with a double bond of the

furane ring in the toxin molecule (Puzyr et al., 2010). This procedure has also been used to destroy DON, moniliformin, cyclopiazonic acid, ochratoxin A, patulin, secalonic acid D, and zearalenone (Young et al. 1986; Zhang and Li, 1994; McKenzie et al. 1997). Despite the advantages of ozonation over most other chemical treatments, drawbacks include loss of nutritive compounds and potential formation of fat-soluble reaction products with some mutagenic potential (Prudent and King, 2002).

Most of the decontamination strategies described above are mostly unsuitable for silages and other feeds on dairies due to the large quantities that would be treated.

Enterosorbents

Enterosorbents are substances that can bind to toxins in the gastrointestinal tract of animals, reducing their bioavailability and associated toxicities (Philips, 1999). They represent an alternative method to avoid the inaccessibility or high cost of physical methods and the hazards of chemical methods. Hydrated sodium calcium aluminosilicates (HSCAS) are clay-based products that form a stable complex with AFB_1, which cannot cross the luminal membrane in the gastrointestinal tract (Spotti et al., 2005). In vitro evaluation of the binding capacity of HSCAS demonstrated that 80 to 99 % of aflatoxin present in various solutions (buffer, water, or rumen fluid) was bound to the clay (Moschini et al., 2008). In vivo trials with HSCAS have shown less complete, but nevertheless useful levels of detoxification. Diaz et al. (2004) evaluated different sequestering agents and reported a wide range of efficacy at reducing AFM_1 in milk (31 to 65%) when a diet containing 100 µg/kg of AFB_1 was fed to lactating dairy cows. Kutz et al. (2009) reported 44 to 48 % of toxin reduction in milk when mycotoxin binders were used in the diet. These results emphasize the importance of carefully selecting and screening potential sequestering agents before evaluating them in animals under practical farming conditions. Satisfactory decontamination has been achieved with the use of inorganic sequestering agents such as HSCAS but these have discriminating affinity to aflatoxin, having no effect on other toxins. For instance, the inclusion of HSCAS in diets has not changed the estrogenic effect of zearalenone (Bursian et al., 1992). When used at 0.5 or 1% of the diet, HSCAS did not mitigate the negative effects of DON on the daily gain of nonruminat animals.

Other types of enterosorbents such as activated carbon, glucomannan, and peptidoglycans have also been evaluated. Diaz et al. (2004) studied the effect of bentonites, esterified glucomannan (yeast cell wall), and activated carbon as sequestering agents to

reduce AFB_1 absorption and transfer to milk. The authors reported that esterified glucomannan fed at 0.05% of diet DM was similarly effective as sodium bentonites fed at 1.2%. Both products decreased milk aflatoxin concentration by 58.5% and 64.6%, respectively. In the same experiment activated carbon showed no significant reduction in aflatoxin M1. The results of using these binders are fairly dependent on the extent of aflatoxin contamination in the diet. For instance, the same esterified glucomannan product that reduced milk AFM_1 concentration when dietary AFB_1 concentration was 55 µg/kg of diet DM (Diaz et al., 2004) had no effect on milk aflatoxin when dietary concentration of AFB_1 was 100 µg/kg of diet (Kutz et al., 2009). Cholestyramine, an insoluble quaternary ammonium anion exchange resin was used (2.5g/kg) to decrease the toxic effect of 6 mg/kg of zearalenone in mice (Underhill et al. 1995)**.** Avantaggiato et al. (2005) observed that cholestyramine effectively binds to zearalenone and fumonisins. The authors also reported that activated carbon was the only absorbent, out of 21 products, capable of binding deoxynivalenol and nivalenol.

Conclusions

Mycotoxins are a major food safety concern due to their effects on animal and human health and their ubiquitous presence in food and feed ingredients. Despite of all the scientific efforts towards development of techniques to prevent mycotoxin contamination, none of the existing techniques is foolproof. Most of the detoxification techniques focus on elimination of mycotoxins present in grains. Special attention needs to be taken when considering mycotoxin contamination in silages because there are no effective detoxification methods for silage. Fortunately, promising contamination prevention strategies have been obtained with fermentation aids, including acids and inoculants, better silo covering films, and the use of enterosorbents. These techniques do not eliminate the problem, but can ameliorate it by inhibiting the growth of toxigenic fungi or by reducing absorption of the toxin by the animal.

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