Mycotoxin: A critical component of any feed quality program.

Duarte Diaz, Ph.D.
Associate Professor and Dairy Extension Specialist
University of Arizona
(520) 626-1221 (office)
duartediaz@arizona.edu

Introduction

Fungi create hazardous secondary metabolites called mycotoxins. *Aspergillus*, *Fusarium*, and *Penicillium* are prominent mycotoxin-producing fungus genera studied in the U.S. and worldwide. Hundreds of mycotoxins have been identified, although few have been widely studied or have commercially available analysis methods. Mycotoxins include aflatoxins, zearalenone (ZEA), trichothecenes (DON and T-2), and fumonisins. The discovery of aflatoxin and its effects in the 1960's led to research on other moldy feedstuff-related health and productivity issues and other fungi-produced toxins. Mycotoxin-contaminated feed affects growth, milk, egg, reproduction, and immunity in cattle, swine, and poultry (Diekman and Green, 1992). Long-term, low-level mycotoxin exposure can cause chronic consequences. Mycotoxins reduce intake, alter feed nutrient content and absorption and metabolism, affect the endocrine and exocrine systems, suppress the immune system, cause antibiotic effects, and kill cells.

In the field, mycotoxicosis can cause digestive disorders, reduced feed consumption, unthriftiness, rough hair coat, undernourished appearance, low production, poor production efficiency, impaired reproduction, and/or a mixed infectious disease profile. Mycotoxins can increase sickness and decrease production. Some mycotoxicosis symptoms may be due to an opportunistic disease caused by mycotoxin-induced immune dysfunction. Mycotoxin immunotoxicity has been reviewed (Oswald et al., 2005). Immune suppression can cause persistent toxicity from low mycotoxin levels, especially if many are present (Xue et al., 2010). Limited research, nonspecific symptoms, complex biomarkers, and co-occurring stressors make diagnosis complicated (Schiefer, 1990). Biomarkers have been proposed for aflatoxin and *Fusarium*-produced mycotoxins—fumonisins, DON, ZEA, and T-2 toxin—despite scant research.

Eliminating other factors, feed analyses, and treatment responses can assist in diagnosing mycotoxicosis. Despite the difficulties of diagnosis, mycotoxins should be considered a possible source of production and health problems when alternative explanations are not present (Schiefer, 1990).

Zearalenone

Several Fusarium species produce estrogenic metabolites ZEA and zearalenol. ZEA competes with 17b-estradiol, the main natural estrogen receptor ligand, for estrogen receptor sites in the uterus, mammary gland, and liver. ZEA passively penetrates the cell membrane and competes with the cytosolic estrogen receptor. ZEA quickly converts to alpha- and beta-zearalenol in rumen cultures (Kiessling et al., 1984). Alpha-zearalenol is four times more estrogenic than ZEA in rats, whereas beta-zearalenol is nearly as strong (Hagler et al., 1979). Ruminal microbial ZEA conversion was 30% in 48 hours (Kellela and Vasenius, 1982). ZEA administered to nonlactating cows at 500 mg

had no apparent effects, except for smaller corpora lutea (Weaver et al., 1986b). Comparable research with heifers ingesting 250 mg of ZEA by gelatin capsule reduced the conception rate by 25% with no additional effects (Weaver et al., 1986a). ZEA has been linked to an estrogenic response in ruminants, including abortions (Kellela and Ettala, 1984). Other cow responses include vaginitis, vaginal discharges, poor reproduction, and virgin heifer mammary gland hypertrophy. In a field case study (Coppock et al., 1990), meals with 750 ppb ZEA and 500 ppb DON caused low intake, diarrhea, decreased milk output, and reproductive failure.

By assessing urinary ZEA+M, which includes zearalanone, alpha- and beta-zearalenol, and alpha- and beta-zearalanol, New Zealand researchers calculated ZEA and its metabolites (ZEA+M) intake. ZEA+M consumption (predicted from urinary ZEA) was linked to sheep and dairy cattle reproductive problems. ZEA+M lowered sheep ovulation and conception. Low-fertility dairy herds showed greater blood and urinary ZEA+M from higher ZEA+M pastures. Palpated cycling cows had lower blood ZEA+M levels than non-cycling cows. ZEA+M values of 400 ppb caused reproductive issues in dairy calves. FDA feed ZEA guidelines are lacking (Henry, 2006).

Trichothecenes

Trichothecenes, a class of 200-300 chemicals, include T-2 toxin, DAS, and DON, which are prevalent in agricultural products (Desjardins et al., 1993). They decrease protein synthesis at the ribosome level, depress immunity, damage cell membranes, and trigger apoptosis (Shifrin and Anderson, 1999). Trichothecenes cause vomiting, diarrhea, and colon irritation. Anemia, leukopenia, skin irritation, feed refusal, growth reduction, and reproductive failure are typical. Several Fusarium species and genera produce trichothecenes. Chronic DON impacts include reduced feed consumption, growth (anorexia and impaired nutritional efficiency), immunological function (enhancement and suppression), and reproductive effects, including smaller litters. Schoevers et al. (2010) showed that DON disrupts cytoplasmic maturation and oocyte development. DON may influence serotonergic activity or receptors (Rotter et al., 1996).

DON levels above 1 ppm can impair swine feed intake and weight increase. Swine's no-observable adverse impact limit is 0.03-0.12 mg/kg of bodyweight per day, or 1-3 ppm in feed (Pestka and Smolinski, 2005). The effects of DON on dairy cattle are not well described. Clinical data suggest a link between DON contamination of diets and poor dairy herd performance, but not a cause and effect (Whitlow et al., 1994). Thus, DON may indicate low-quality, mycotoxin-contaminated diets in these animals. Other field findings link DON to poor dairy herd performance however other mycotoxins are frequently present (Gotlieb, 1997). In a study of 18 mid-lactation dairy cows (average 19.5 kg of milk), a trend ($P < 0.16$) showed a 13% loss in 4% fat-corrected milk. The diets contained no common mycotoxins, except for DON (0.0, 2.7, and 6.5 ppm) (Charmley et al., 1993). Danicke et al. (2005) found that feeding 3.1 ppm DON from contaminated wheat reduced metabolizable protein by 20%. Higher rumen ammonia levels indicated less protein synthesis. Korosteleva and Smith (2006) found higher blood urea nitrogen in dairy cows fed a Fusarium mycotoxin cocktail with DON. DON-contaminated grain alters rumen volatile fatty acid concentrations (Keese et al., 2008). DON reduces feed intake in non-lactating dairy cattle (Trenholm et al., 1985).

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A 3 x 3 Latin square experiment with 21-day feeding cycles by Noller et al. (1979) used 54 nursing dairy cows. DON and ZEA concentrations in three experimental diets were measured using Gibberella zeae (F. graminearum)-infected corn. Despite not affecting intake or milk output (22.9 kg per day), contaminated grain-fed cows gained significantly less weight. In contrast, Ingalls (1994) fed lactating cows diets with 0.0, 3.6, 10.9, or 14.6 ppm DON for 21 days without affecting feed intake or milk output (30 kg per day). DiCostanzo et al. (1995) research review found that beef cattle and sheep can withstand 21 ppm DON without harm. *Fusarium sporotrichioides, Fusarium poae*, and other *Fusarium* species produce T-2 toxin (Marasas et al., 1984). Barley, wheat, millet, safflower seed, and mixed meals contain T-2 toxin. Laboratory animals are the best model for T-2 toxin toxicity (Wannemacher et al., 1991). Li et al. (2011) found that T-2 is the most dangerous of the 190 trichothecene family members. Ironically, T-2 toxin has gotten little scientific attention.

Protein synthesis reduction, lowered immunity, cellular necrosis, and hematological consequences (mainly a decrease in circulating blood cells sometimes linked with bone marrow failure) cause toxicity. Immunosuppression is substantial (Bondy and Pestka, 2000). According to Li et al. (1997), T-2 causes stomach mucosa, gastric glandular epithelium, and intestinal crypt cell epithelial apoptosis. Animals show unthriftiness, reduced feed intake, productivity, reproductive failure, diarrhea, gastrointestinal bleeding, and illness.

T-2 toxin causes diarrhea, intestinal hemorrhages, and death in cattle (Petrie et al., 1977). In a cow, Weaver et al. (1980) found that T-2 was related to feed refusal and gastrointestinal lesions but not hemorrhagic illness. Pier et al. (1980) found that 0.64 ppm dietary T-2 caused bloody stools, enteritis, abomasal and ruminal ulcers, and mortality after 20 days. Kegl and Vanyi (1991) found bloody diarrhea, reduced feed consumption, decreased milk output, and no estrous cycles in T-2-exposed cows. In calves given T-2 toxin, immunoglobulins and complement proteins were reduced (Mann et al., 1983). Gentry et al. (1984) found calves had less white blood cells and neutrophils. A T-2-intubated calf showed severe depression, hindquarter ataxia, rear foot knuckling, listlessness, and anorexia (Weaver et al., 1980).

Fumonisins

Fumonisin B1 was first discovered in South Africa, where Fusarium-contaminated feed has long caused animal issues (Gelderblom et al., 1988). Fumonisin causes equine leukoencephalomalacia (ELEM) in horses (Marasas et al., 1988), pulmonary edema in swine (Harrison et al., 1990), and hepatoxicity in rats and cattle (Gelderblom et al., 1991). Fumonisins alter lipid metabolism and calcium homeostasis, causing animal toxicity (Riley and Pestka, 2005). Fumonisins inhibit ceramide synthase (sphinganine and sphingosine N-acyltransferase), which forms ceramide and complex sphingolipids. ELEM, porcine pulmonary edema, liver and kidney damage, and fumonisin-induced sphingolipid metabolism disturbance are strongly connected (World Health Organization, 2000). Fumonisins produce pulmonary edema in pigs (Harrison et al., 1990). Lower fumonisin B doses caused slowly progressive hepatic necrosis; higher doses caused abrupt pulmonary edema and hepatic toxicity (Haschek et al., l992).

Kriek et al. (1981) found fumonisin B1 harmful to sheep, despite its lower potency in ruminants than monogastrics. In a 31-day research, Osweiler et al. (1993) fed young steers 15, 31, or 148 ppm fumonisin and observed no impact on feed consumption or gain, but 148 ppm showed a trend toward lower intake and weight increase. At high feeding levels, two calves showed modest liver lesions and increased liver enzymes, indicating liver injury. The highest dose group had dramatically reduced lymphocyte blastogenesis after eating.

Holsteins and Jerseys fed 100 ppm fumonisin diets for seven days before freshening and 70 days afterward produced less milk (6 kg per cow per day), mostly due to reduced feed consumption. Serum enzyme elevations indicated moderate liver illness (Diaz et al., 2000). Even though rumen fumonisin destruction is minimal (Calino et al., 2000), fumonisin carryover from feed to milk is assumed to be insignificant (Richard et al., 1996). Rats and mice develop cancer from fumonisin (National Toxicology Program, 1999).

Aflatoxins

Aflatoxin doses as low as 100 ppb may be harmful to beef cattle; however, the hazardous level is usually 300–700 ppb. Garrett et al. (1968) found that 700 ppb aflatoxin feeds affected weight gain and consumption, but if liver weight increases are the toxicity criteria, then 100 ppb is hazardous to beef cattle. In steers fed 600 ppb aflatoxin, feed efficiency and gain decreased (Helferich et al., 1986). When nursing dairy cattle in the field consumed 120 ppb of aflatoxin, Guthrie (1979) found decreased reproductive efficiency. Aflatoxin-free diets improved milk output by 25%-plus. Patterson and Anderson (1982) and Masri et al. (1969) reported 100 ppb may diminish milk output. Applebaum et al. (1982) found that culture-produced impure aflatoxin lowered output, whereas pure aflatoxin did not. Aflatoxin dosages of 1 µg and 10 µg/mL reduced ruminal alfalfa digestibility by 50% and 67%, respectively (Westlake et al., 1989), but 9.5 ng/mL had no effect (Auerbach et al., 1998).

Food contamination exposes humans to aflatoxin and other mycotoxins. Mycotoxins can be inhaled and skin-absorbed from contaminated feed (Schiefer, 1990). Humans are indirectly exposed to aflatoxins through milk, liver, and eggs from animals fed contaminated diets (Hayes, 1980). Lactating dairy cows excrete aflatoxin B1 as aflatoxin M1, with residues of 1-3% of the dietary concentration (Van Egmond, 1989). Aflatoxin occurs in milk within hours of ingestion and returns to baseline levels within two to three days of diet elimination (Frobish et al., 1986). Lactating dairy cattle with 20 ppb of aflatoxin B1 in their total mixed feed dry matter will have aflatoxin M1 levels in milk below the FDA's action standard of 0.5 ppb. However, the EU and some nations have a 0.05 ppb action limit for milk and milk products. Because absolute mycotoxins in feed are hard to measure, concentrations may vary over a lot of feed, and concentrations might alter over time, assumed safe feeding levels may result in milk concentrations exceeding the FDA action level.

Other Toxins

Over one hundred different mycotoxins have been described in the scientific literature. Some of the most important due to their occurrence or toxicity are Penicillium produced toxin Ochratoxin A, Citrinin, MPA, Patulin, PR toxin, and Roquefortine C. Many of these Penicillium-produced toxins

have been commonly found in silages (Hacking and Rosser, 1981) and have shown to have strong antimicrobial properties (Tapia et al., 2005). European grass and corn silage surveys identified P. roquefortii in up to 40% of samples (Auerbach, 2003). PR toxin may be a major mycotoxin in silage toxicity (Auerbach, 2003). Schneweis et al. (2000) discovered MPA in 32% of German silages. Cole and Cox (1981) found little animal toxicity. However, MPA decreases T and B lymphocyte proliferation, antibody generation, and cytotoxic T-cell production (Eugui et al., 1991). Although the highest dose shrank thymic lobules, sheep fed 0.0, 0.5, 1.2, or 5 mg of MPA per kilogram of body weight daily for six weeks exhibited no effect on general health (Baum et al., 2005). As MPA doses increased, ileum IgG- or IgM-positive plasma cells decreased. These findings imply that silage's high MPA levels may impair sheep lymphatic organ architecture. Gallo et al. (2015) used rumen microorganisms to investigate the antibacterial capabilities of MPA and PR toxin and found that both toxins could disrupt digestion and pose a risk to ruminants.

Treatment

A guidebook on hazard analysis and important control point procedures for mycotoxin prevention and control is available from the UN Food & Agriculture Organization (2001). Mycotoxin management quality assurance was examined by the Council for Agricultural Science & Technology (2003). Prevention, sampling, sample preparation, extraction, testing, validation, documentation, supplier participation, and removal are components. Cereal safety regulations were evaluated by Heli et al. (2013), focusing on mycotoxin contamination and the EU. Predictive mathematical models and geographical occurrence data can improve cereal supply chain mycotoxin management (van der Fels-Klerx and Booij, 2010). De Wolf et al. (2003) created a U.S. model that predicts wheat Fusarium head blight risk using within-season weather data. Kabak et al. (2006) examined preventing, decontaminating, and reducing feed mycotoxin toxicity.

Agronomic approaches reduce plant stress, fungal invasion, and mycotoxin accumulation in the field for pre-harvest control. Proper irrigation, insect control, pesticide application, resistant or adapted hybrids, tillage type, fertilizer, timely planting, and harvest on time are examples. Unfortunately, mycotoxin-resistant hybrid breeding has only partially succeeded. Munkvold et al. (1999) found that Bt-transgenic corn had less corn borer damage, F. verticilloides infection, and fumonisin contamination than non-transgenic corn. Despite their ineffectiveness against pre-harvest aflatoxin contamination in corn (Duncan et al., 1994), fungicides may suppress other mycotoxins. The fungicide shock may suppress mold growth but not mycotoxin generation (Boyacioglu et al., 1992). Utilizing non-toxigenic fungi to compete with toxigenic ones has greatly reduced aflatoxins. This technology is increasingly significant for aflatoxin contamination avoidance worldwide. A recent assessment (Mwakinyali et al. 2019) identified these techniques' two main development areas. First is biocontrol by preventing fungal growth by using biocompetitive non-toxin strains to suppress toxin production. The second biocompetitive technique is to inhibit aflatoxin formation by identifying genes involved in biosynthesis and using molecular tools to find natural inhibitors.

Mycotoxin management after harvest is best achieved by properly storing and treating feedstuffs to prevent fungal growth. The main elements causing mycotoxin production in storage are temperature, water activity, and insects. Mycotoxin analysis of feedstuffs, diversion of

contaminated lots, mold growth treatments, dilution, and mycotoxin reduction are further management measures. Protecting animals against mycotoxin exposure and toxicity is being researched. By cleaning or screening grains, physical separation can aid. Milling reduces mycotoxin concentrations in human-consumed fractions but not animal feed fractions. Chieli et al. (2013) found eight-fold more mycotoxin in wheat byproducts than grain.

Some chemical and biological activities may reduce mycotoxin effects. Kabak and Dobson (2009) reviewed mycotoxin-fighting biological methods. Microorganisms or enzymes can breakdown mycotoxins into harmless metabolites. Fermentative bacteria may bind mycotoxins (Niderkorn et al., 2007). Protease A, pancreatin, carboxypeptidase A, epoxidase, and lactonohydrolase have been shown to degrade mycotoxin. Flavobacterium aurantiacum, Enterococcus faecium, Eubacterium: BSSH 797 and LS 100, and Trichosporon mycotoxinivorans can detoxify mycotoxin. Some Fusarium species produce acetytransferases that breakdown DON (Khatibi et al., 2011). Some Bacillus subtilis subspecies degrade zearalenone extensively (Cho et al., 2010).

No viable method exists for treating forages, but ammoniating grains can eliminate some mycotoxins. Aflatoxin may be reduced by adding 0.25% or 0.50% calcium propionate to detoxification diets (Bintvihok and Kositcharoenkul, 2006). Galvano et al. (2001) examined mycotoxin-fighting diets. Adding protein, energy, and antioxidants may be beneficial. Sorrenti et al. (2013) examined how antioxidant supplementation affects OTA toxicity. Silicate clays (bentonites and others), activated carbons, and beta-glucan polymers (extracted from yeast cell walls) can reduce mycotoxin effects, according to research. Reviewing in vitro mycotoxin adsorption methods suggests that while they may be useful in product screening, they may not necessarily predict in vivo findings. The mycotoxin-binding efficiency of various additives has been tested in an in vitro gastrointestinal model to better replicate in vivo settings (Avantaggiato et al., 2005). Avantaggiato et al. (2005), and Ramos and Hernandez (1997) reviewed mycotoxin enterosorbents. Magnoli et al. (2011) suggested that dietary monensin may inhibit sodium bentonite's aflatoxin binding. Similar consequences may apply to other mycotoxins, binders, and additives.

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