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J.D. Pagan

IMPLICATIONS OF MYCOTOXINS

JOHN C. REAGOR
Texas A & M University, College Station, Texas, USA

This paper will cover the most important mycotoxins which affect cattle and horses with emphasis on the newly discovered fumonisins. Many adult horses have consumed moldy feed or forage. In most instances, these fungal contaminated materials are consumed without harm. Feeds which contain a significant concentration of a mycotoxin can and do cause severe problems.

Aflatoxins, the most studied mycotoxins, were first discovered some 30 years ago after the death of thousands of birds in England. The most abundant and most toxic of the group is aflatoxin B1. This and its metabolite, aflatoxin M1, are the compounds most commonly referred to when aflatoxicosis is discussed. Livestock are usually poisoned when the toxins contaminate corn, peanut or cottonseed products, as these commodities may support toxin production in the field prior to harvest. These, as well as other high energy feedstuffs, may support fungal growth and toxin production by improper storage or handling. We have seen significant aflatoxin production occur in feed mixer wagons and bunks where wet feed was fed to dairy cows. The equipment was not cleaned on a regular basis and a moldy layer allowed toxins to form over a period of a few days to weeks. Aflatoxin levels can increase dramatically in harvested corn (16-18% moisture) that is allowed to stay in the wagon overnight at harvest.

Aflatoxin production requires a suitable substrate, a toxigenic strain of *Aspergillus*, the proper temperature (80-110^{o} F), adequate moisture (14-22%) and the correct relative humidity (62-99%). Widespread aflatoxin problems (like those occurring in corn from Texas, Florida and Georgia) are the result of adverse growing conditions.

Problems with aflatoxin have been far more severe on corn and peanuts when drought stress prevailed during the latter part of the growing season. Under such conditions, factors favoring infestation are ideal because temperatures are high, relative humidities around kernels are high and the kernel moisture lowered enough for infestation to occur. High grain moisture (22 to 30%) is not conducive to the infection process for this fungus. The fungus that produces aflatoxin also has less competition from other organisms when drought stress conditions prevail.

Confusion often arises when one thinks of production and storage conditions at the same time. Those oriented toward considering the aflatoxin problem to be a harvesting and storage problem find it difficult to see how drought stress (low moisture)

can cause a problem considered to be related to high moisture at harvest time. Corn, for example, is vulnerable when it reaches the dough stage when the moisture usually is too high for infection by *Aspergillus*. If severe drought stress is present, however, kernel moisture is lowered to the point where infection can occur.

Numerous field studies have documented infection of corn kernels by aflatoxin-producing fungi. Corn silks protruding from shucks are readily invaded by these fungi. Germ tubes rapidly grow down these tubes to the kernel surface where they lie until late in the development of kernels. Once the kernel reaches a moisture content of approximately 32%, the fungus enters the kernel. *Aspergillus favus* is capable of direct penetration in the absence of insect injury even though the latter may enhance the chances for organism development. It is rare indeed to find a corn ear that is not infected with a corn ear worm larva.

Extensive research shows that corn kernels may be preconditioned by a multitude of factors that may interact to influence the amount of aflatoxin that may occur in a given field at harvest. These include the potential for harvest cracks in kernels and holding high moisture corn on transport equipment an excessive period before drying.

Silage production from drought-stressed corn may also contain sufficient levels of aflatoxin to be of concern to livestock producers and dairymen. Therefore, it is advisable to evaluate the potential aflatoxin content in drought-stressed crops before harvesting for "green-chop" or silage production.

The most significant effect of aflatoxins to dairy cattle is the contamination of milk by aflatoxin M1. Of the over 200 mycotoxins identified, aflatoxin is of major concern to dairymen. The maximum level of aflatoxin allowed in dairy feed is 20 ppb (parts per billion). When aflatoxin is consumed by lactating cows, it not only can be toxic to the cow but also appears in the milk within 24 hours. Since aflatoxins also affect humans, a maximum level of 0.5 ppb of the aflatoxin is permitted in bulk tank milk. A general conversion factor of 100 to 200X reduction in the aflatoxin fed to that appearing in the milk or 0.91% of the aflatoxin consumed in the feed appearing in the milk is used. For both rules of thumb, the aflatoxin metabolite appearing in milk would be 0.1 to 0.2 ppb if feed containing 20 ppb aflatoxin is fed. Generally, levels of 50 ppb or greater in the feed produce metabolite levels over 0.5 ppb. Once the affected feed is removed, aflatoxin levels in the milk will disappear in 48 to 72 hours.

While ruminant animals, such as dairy cows, are more resistant to aflatoxins, toxicity does occur with disastrous results. Calves are particularly sensitive and exhibit reduced growth rate. The target sites of action of mycotoxins are organs such as liver, heart, kidney and adrenal. Aflatoxins are particularly damaging to the liver with noticeable reduction in milk production and appetite. Diarrhea, internal hemorrhaging, abortion and weight loss may also occur.

When we know aflatoxin problems are common to an area, it is extremely important that feeds–particularly corn, whole cottonseed and cottonseed meal–be checked for mycotoxins. This is important both for the short term and long term, with the short term being for the protection of milk supplies. Long-term consequences can be devastating, as aflatoxins may permanently reduce productive capacity due to organ

damage. This is particularly significant since contaminated corn supplies are often fed to heifers as a means of saving the commodity without regard to the long-term effects.

The federal allowable level for aflatoxin in corn for beef cattle is quite acceptable. Young calves, under 200 lb., should not receive feed containing over 20 ppb. Beef cows should not receive over 100 ppb, while finishing feedlot cattle can tolerate up to 300 ppb.

Immunosuppression occurs when cattle rations exceed 200 ppb and severe health problems (chronic liver damage, reduced growth and feed efficiency) can result from the continued intake of 600 to 700 ppb aflatoxin in the feed of 450-pound cattle.

Kidney damage, anemia, interference with the body's immune system, greater susceptibility to bruising and interference with normal protein and fat metabolism have all been reported with varying levels of intake. In acute cases, those below the level that lead to sudden death in cattle, the following signs may occur: depression, lack of appetite, nervousness, abdominal pain (animals may stretch or kick at their abdomen), diarrhea and rectal prolapse. Death of steers has been reported from an intake of 1,000 ppb of aflatoxin in feed during a 59-day trial.

The tremorgens produced in bermudagrass and on seedheads infected with *Claviceps spp*. can be a significant problem in cattle. Ergot-infected dallisgrass, rye, ryegrass, bahiagrass and other native *Paspalum spp*. seedheads can cause staggers, abortion and death in cattle or horses which consume large amounts. Most cases occur when animals are moved to a pasture that contains mature seed or when round bales containing mature plants are fed. The ergot bodies are retained in the round bales and are not lost in the feeding process, as is usually the case with small square bales.

Tall fescue grass, other than pastures planted with endophyte-free seed, contains the fungus *Acremonium coenophialum*. Cattle grazing contaminated fescue develop fescue toxicity which results in lower feed intake, reduced grazing time, lower animal performance (for example, gain, milk production, reproductive performance) and sometimes the extreme toxicity called "fescue foot" where animals develop lameness and sometimes lose portions of their feet and tails from the restricted blood circulation caused by the disease. Mares that graze contaminated stands often abort, produce stillborn foals, retain placentas and have reduced milk production

The fungus is transmitted by seed and does not affect the growth or appearance of the grass. Hence it requires a laboratory test to detect its presence. Prevention measures include planting fungus-free seed in newly established pastures or destroying old pastures before seed formation and replanting with fungus-free seed. Interseeding pastures with clover or other cool season grasses helps dilute the effect of the fungus.

Equine leukoencephalomalacia (ELEM) or moldy corn poisoning in horses has been recognized since the turn of the century. The disease has been associated with *Fusarium moniliforme* and was experimentally reproduced with *F. moniliforme* culture material in 1971. Fumonisin B1 was identified and reported to cause a similar condition following IV injection in 1988. Oral administration of purified Fumonisin B was reported to produce ELEM in 1989.

A large amount of effort has been placed on fumonisin recently. There are now assay procedures for the mycotoxin and many samples associated with ELEM have been analyzed.

ELEM may be clinically characterized by a sudden onset of one or more of the following signs: depression, aimless circling, head pressing, paresis, ataxia, blindness, hypersensitivity and frenzy. Some animals are found dead where they have run through a fence. Diagnosis has been based on the classical brain lesions. There is liquefactive necrosis of the white matter of the cerebral hemispheres. These lesions may also include the gray matter and may be located in the midbrain or brain stem. With the advent of chemical analysis of feed for fumonisin, some cases are being diagnosed where the only brain lesions are areas of hemorrhage and the "impression of edema."

ELEM is not an acute mycotoxin poisoning. Typically, affected animals have consumed toxic feed for at least 14 days and often as long as 60 days. The morbidity rarely exceeds 50% but the mortality is high.

Fumonisin appears to be limited to corn or corn products. Most cases involve horses which were consuming feed that was composed of, at least in part, corn screenings. Based on field cases, animals must consume a feed which contains at least 10 ppm fumonisin B1 for ELEM to occur.

Very high levels of fumonisin have been seen in corn which was down and had extensive water damage prior to harvest. We have seen some selected, damaged kernels that contained over 700 ppm (parts per million) fumonisin. This is an extremely high mycotoxin concentration considering that most mycotoxins are reported as ppb.

There was a significant number of ELEM cases from the 1989 corn harvest. It was not uncommon for corn screenings from export elevators in Louisiana to contain 40 to 50 ppm fumonisin B1. This was from corn which originated from the Corn Belt.

Fumonisin may well become one of the most significant mycotoxins. Unlike many of the newly discovered fungal metabolites or mycotoxins, there is no need to look for a clinical disease associated with this one. In addition to ELEM, this toxin can cause liver damage in horses and pigs as well as pulmonary edema in swine. It is also a strong tumor promoter in rats.

Much of the corn in the United States appears to be contaminated with at least 0.5 to 1 ppm fumonisin. If this is the case, the screenings from this corn could poison horses if it were fed at a high level. Horse feed should not contain corn screenings!