

Mycotoxins In Silage: A Silent Loss In Profits

The Vermont Crops and Soils Home Page

INTRODUCTION

In this article I am going to present information on mycotoxins and spoilage and what they mean to you as a producer. I am going to talk about the following:

- What is the known history of mycotoxins and their seriousness as a food and feed contaminant
- Why, where, and under what conditions mycotoxins are produced
- What practices worsen contamination by mycotoxins
- How mycotoxin contamination can be reduced
- The future of mycotoxin control and prevention

MYCOTOXINS ARE PRODUCED BY MOLDS

While most fungi only reduce the yield or nutritive value of the feed they infest, some fungi have the ability to produce toxic chemicals called mycotoxins. Mycotoxins are complex organic compounds that are produced by a fungus to increase its virulence as a plant pathogen by reducing the ability of the plant's resistance. In contrast, saprophytic (mold or rot) fungi reduce the competitive ability of other fungi or bacteria that are competing for the same food source through the release of these toxins. As these fungi grow, the nutritive value of the plants they infect or the stored feed they infest is depleted. Available carbohydrates and other nutrients are converted to carbon dioxide and other fungal metabolites not readily available as animal nutrients (DiCostanzo). The toxins they produce have probably been in the environment for millions of years.

When tissue is rotting (spoilage), thousands of bacteria and fungi are competing for the nutrients in the now dead tissues. Any fungus that can reduce competition, through production of a toxin, will become the dominant organism. These toxin-producing fungi did not have animals in mind as part of their environmental competition. The affect on animals and humans is purely coincidental due to the similarity of affected metabolic systems.

Throughout history, the damage due to mycotoxins has been recorded as ailments and death to humans and animals. Rye and wheat infected with ergot has caused major human illness due to contaminated bread with ergotamine.

In Europe, Napoleon's defeat in Russia may not have been due as much to cold

but rather to ergotized grain fed to their animals which resulted in a catastrophic loss in horses. In England, over 100,000 turkeys were killed (turkey X disease) due to an Aspergillus mold in grain which produced a toxin. Aflatoxin, produced by the fungus Aspergilli, is one of the most powerful carcinogens known (natural or synthetic). Ten to 20 parts per billion in the diet of a susceptible animal can cause liver cancer. This toxin is closely monitored by federal agencies in grain and peanuts to keep serious levels from reaching the human or animal population.

In Russia, during World War II, thousands of civilians died due to a fall grain harvest delayed by war until spring. The result was a grain crop contaminated with T-2 toxin, produced by a Fusarium mold in the grain heads. T-2 is also the primary toxin in the biological weapon known as yellow rain. These are only a few of the serious worldwide reports of toxins causing catastrophic toxicity in humans and livestock (Bullerman).

My own worst experience with a mycotoxin in Vermont was the toxic reaction of a dairy herd to a mycotoxin contamination in citrus pulp. Toxicity resulted in the destruction of a 100-cow herd over an eight month period. Major organs were damaged and eventually the entire herd was destroyed. This type of catastrophic event is highly unusual in dairy cows with most toxic reactions appearing as poor weight gain, reproduction problems, reduced feed intake, and reduced milk production.

Many people have asked me if toxin problems in dairy and other livestock are increasing. My answer to that is yes, I believe it is. Keep in mind that the dose makes the toxin. Over the past 25 years we have nearly doubled the intake of the dairy cow, yet we have not doubled the size of the animal. The significant increase of feed intake of the dairy cow, which has aided in the major increases accomplished in milk production, has also caused a significant increase in toxin dosage per pound of animal when consuming contaminated feeds. Higher intake of toxins, combined with the increased stress of higher milk production, could account for the perceived increase in reported mycotoxin problems.

TOXINS OF CONCERN AND THEIR CONDITIONS FOR GROWTH

In the United States, some of the primary toxin producing fungi found in silage includes Fusarium, Penicillium, and Aspergillus (Shurtleff). Several toxins of great concern are produced by Fusarium and include vomitoxin (DON), fumonisin, zearalenone, and T-2. As discussed above, Aspergillus is known to produce aflatoxin while the fungus Penicillium produces several serious toxins in silage and other stored feeds. All of these fungus toxins have been associated with acute, chronic, and sub chronic diseases of livestock.

Various genera of fungi can produce toxins and literally hundreds (probably thousands) of fungus species have the capacity to produce toxins. All these fungi have three critical environmental requirements.

- temperatures above freezing,

- moisture above 20%, and
- oxygen.

Limiting any one of these requirements will reduce or prevent the production of toxins. When considering silage, it is neither practical nor desirable to limit temperature or moisture. Limiting oxygen is the key to successfully limiting toxin production during ensiling. Oxygen is like a light switch. It can turn toxin production on and off during storage.

Aflatoxin, the most serious carcinogen, has been found in high levels in peanuts, corn, cotton seed, and grain and can contaminate milk. This toxin is a serious problem for human and animal health and can contaminate corn in warmer growing regions. Aflatoxin requires warm (85o F) and moist conditions. Where fall conditions are cool, aflatoxin is rarely found. For example, in Vermont, our fall conditions are often wet but temperatures normally average between 50 and 60 degrees. We can find the fungus, *Aspergillus*, but the toxin it produces is not produced under our cool conditions. The further south one goes, the greater the potential of aflatoxin contamination in corn and other feeds. For those in cool growing regions, however, keep in mind that aflatoxin can occur in grain shipped in from out of state. Government and private industry have reputable testing programs to control the entry of this toxin into the feed and food systems. Always be wary of special deals from unknown suppliers and be sure to ask about their mycotoxin testing programs.

In contrast, the fungi in the genus *Fusarium* produce their toxins efficiently between 45 and 75 degrees Fahrenheit. This is just the right temperature for corn, haylage, and silage contamination in the more northern temperate regions. I don't know where this group of fungi would fit in the Florida production with higher temperatures but *Penicillium* may fit just fine. Several genera of *Fusarium* and *Penicillium* are known to be plant pathogens and attack corn, grasses, and legumes. For example, a primary *Fusarium* disease of corn is ear rot, stalk rot, and root rot. Corn ears can be infected through the silks at flowering or through any type of damage such as insect feeding in ears, stalks, or roots (Farar). As the fungus grows in the plant tissue, it may or may not form toxins in high enough levels to cause contamination problems in feeds. However, a common scenario for high levels of *Fusarium* toxin infection in corn starts with wet conditions during silking accompanied by insect damage (thrips) to silks. The fungus infects the silks directly or through insect damage and grows down the silks and infects the kernels and cob. As the season progresses, further damage to the stalk or ear by other pests such as the European corn borer and birds can result in an increase of *Fusarium* rot in damaged tissues. *Penicillium* rots can also invade through insect or bird damaged tissues. Rot continues to increase as the season progresses and toxin production begins to accelerate as the crop begins to mature (Lepon). The longer corn is allowed to stand in the field after maturity, the greater the likelihood of significant toxin development. Levels of *Fusarium* toxins can be the result of a continuous accumulation of toxin over time during the growth

period and continuing after maturity and into storage until oxygen becomes limiting or, in the case of grain, moisture is reduced to less than 20%. In the case of silage, corn harvested after frost is at even greater risk of toxin contamination. When the corn is chopped and placed in a silo, the frosted and now drier silage is difficult to pack properly. The same could occur in field dried leaves and stems left too long without frost conditions. The oxygen level in the silo takes longer to deplete during filling and the fungus can continue to grow and produce toxin for several days. In one study at the University of Wisconsin (Park), moldy ear corn increased in toxin concentration by 10 times when left in contact with air for 8 days after harvest. Our studies in Vermont show that molded ears left in the field will increase in toxin production significantly in just two weeks. The level of toxin in cobs was greater than the levels found in the kernels. Fusarium toxins in corn are probably not reduced by ensilement (Lepon).

WHICH SILO IS BEST?

At the University of Vermont, eight different mycotoxins have been found contaminating Vermont feeds including vomitoxin, ochratoxin A, patulin, penicillic acid, T-2 toxin, verrucaric acid, zearalenone, and kojic acid. These toxins were found to be present (in varying combinations) in haylage, corn silage, dry hay, grains, and all commodities. In one survey, the amount of mycotoxin in contaminated silage samples increased as the ensilement method changed from airtight, upright silos to concrete capped and uncapped silos. The highest forage concentrations of toxins were found in horizontal storage methods such as bunker silos and feed piles which were left open to oxygen. In all cases, where greater amounts of toxins were found, poor management of the upright or bunker silo resulted in oxygen getting into the stored feed. Well managed bunker silos, covered with plastic and weighted with tires, had no significantly greater levels of toxin than well managed upright silos. In any fermentation storage system, temperature and the presence of moisture is sufficient for toxin production. But, oxygen will act as the switch which turns toxin production on or off during storage. In a plastic covered storage system, oxygen penetration is slowed but not eliminated. The longer the silage is stored, the greater the opportunity for significant fungus growth and toxin contamination. In one observation by Trenholm in Canada, the levels of DON increased in the silo slowly over time even when properly covered.

HOW CAN I MEASURE THE EFFECTIVENESS OF MY FORAGE MANAGEMENT SYSTEM?

Over the past three years we have made more than 1500 tests for Fusarium toxins in all types of silage using a variety of ensilement methods. The highest levels of toxin are consistently associated with areas of spoilage. In one survey of 85 farms, 38% of the spoiled areas in bunker silos tested greater than 3

ppm DON. It is critical that we always dispose of spoilage as it can contain some of the highest concentrations of toxin. While DON is not yet shown to cause toxicity in research trials using pure DON (Charmley), it is the most common Fusarium toxin we find in Vermont. Approximately 42% of silage tested in Vermont shows positive for DON, with the majority of samples below 1ppm. However, 15% of the samples test at above 3ppm. The finding of DON at this level indicates to me that there may well be some management strategies some farmers can implement to reduce the threat of toxin development. We used the ELISA DON test as an indicator to determine if conditions for Fusarium toxin development have been favorable. Much like the canary in the mine shaft warning miners of the threat of gas, a high ELISA reading for DON tells us conditions were favorable for any toxin to develop. This warning encouraged the producer to take a more critical look at his forage production and storage practices. We now have numerous anecdotal reports of farmers who have improved management and gained satisfactory results. This approach was highly successful in getting farmers to improve storage management practices.

WHAT ABOUT PLASTIC AND OXYGEN PENETRATION?

There is no such thing as an oxygen proof silo. We would all like to think this is true, but in practical terms our current technologies are not perfect. When one examines a plastic layer under a microscope, you will find tiny holes through which oxygen slowly flows. This is especially true of plastic that is stretched for wrapping bales. Any damage to the plastic further increases the flow of oxygen from a trickle to a river and must be repaired as quickly as possible. Our current system only slows the damage from spoilage over time to an acceptable level until we can utilize the feed within months of harvest.

WHAT IF I SEE MOLD?

Just as you would not eat moldy food, a good rule to follow is not to feed moldy hay, haylage, or corn silage to your animals. There is no way to distinguish between toxic and non-toxic fungi by their presence in the feed or the discoloration of the feed. The fungi that produce toxins are present in all feeds since they are naturally occurring in the fields where the crops are grown. We can see the result of these fungi when hay is cut and does not dry quickly and rots in the field or in a wet bail. Fortunately, the presence of these fungi does not automatically mean toxins are present in the feed. However, the absence of visible molds does not guarantee that a feed is safe. Dangerous levels of mycotoxins may accumulate earlier during growth of the crop and often will not be visible. This level of toxin can then continue to increase during poor harvest conditions and on into storage. Whenever possible, use the following adage. "When in doubt, throw it out."

DETOXIFYING CONTAMINATED FEEDS.

Presently there are no economical means to detoxify contaminated feeds. However, a steady number of dairy consultants and farmers in Vermont and

other states continue to report that the use of sodium bentonite, and other adsorbent materials, added to feed suspected to be contaminated with mycotoxin, has resulted in benefits in milk production, feed intake, and reduced reproductive problems. Sodium bentonite is a complex clay-like material commonly used as a flow agent to reduce caking in feeds. These products are mined from ancient deposits of a combination of sea life and volcanic eruptions. Therefore the purchased ingredient can vary by the geographical region from which it is obtained. Such materials have been reported to reduce the harmful effects of aflatoxins in pigs and rats by binding to the mycotoxin and making it less available for absorption in the digestive tract (Carson). In dairy cows, there is no direct scientific evidence to support this claim. However, there is now a large body of anecdotal reports supporting the use of binding agents. In Vermont, we have observed a significant number of herds that have benefited by feeding 4 to 8 ounces of adsorbent when mycotoxin effects were suspected. Increased feed intake and milk production were sometimes noted within a matter of days. On the other hand, we have also noted herds with similar symptoms that have had no beneficial response. The number of adsorbent sources and types do vary between reporting farms. Thus there can be no general claims made as to the use of a specific product. However, in practical terms, a local successful experience may be as good a recommendation as any at this time. We have also found that if one adsorbent does not work, try another. In specific cases, up to three products were tried before one was found to work.

CURRENT RESEARCH

The United States Government has placed a high priority on developing technology to reduce toxin contamination in the food and feed supply (CAST). Presently there is a flurry of research dealing with the reduction of aflatoxin in grains and peanuts since it impinges directly on human health and especially children. Much of this research is devoted to the development of plant resistance to invasion of *Aspergillus* and *Fusarium* and the cleaning up of grains once contaminated with toxin. There is also great interest in *Fusarium* toxins and their control due to their devastating affect on poultry and pork production. However, dairy production has not been a high priority for mycotoxin research in this country. Mycotoxin damage in dairy production is insidious in that it generally causes a sub-acute or chronic result such as a herd not meeting production expectation of the managers. In Vermont, several of our herd consultants estimate that 20% of their clients have mycotoxin like problems at any one time and that almost all farms experience the problem in a 5 year period. Farm losses can be anywhere from 2 to 10% of milk production. This level of loss is difficult to measure and pin down to a single cause. In Europe, there is currently research activity looking at mycotoxins in both silages and pasture. Both *Fusarium* and *Penicillium* appear to be the major players in silage mycotoxin problems there. In Vermont we are now concentrating on *Penicillium* toxins as these are primary spoilage fungi found in

our silages. Other than documenting the presence and toxicity of such toxins, there is as yet little in the way of new technology to reduce contamination. However, a lot can be accomplished by using best storage and harvest management practices available.

In the United States, we need to know at what point the greatest levels of toxin are being produced in our forages and silages to efficiently focus our management strategies. This will require the dissection of the forage management system. We are currently doing just this in Vermont and looking at environmental and biotic factors involved in increased toxin contamination. A major effort currently underway is to develop greater resistance of corn to ear rot as this is a prime entrance site for infection and toxin contamination later in storage. We also need increased resistance in stalk tissue as this represents half the dry weight of silage. In Vermont, we have shown that stalk tissue contributes up to 50% of DON toxin levels found in corn silage. Resistance is needed to silk infection as well as resistance to insect attack through which the fungus can infect the plant. Some of the newer genetically engineered corn varieties have resistance to insects such as the European corn borer. Scientists have recently transferred the gene that produces Bt toxin from the bacterial pathogen to the corn plant. The bacterial toxin, which is toxic to specific insects, is now produced by the corn plant to defend against the European Corn Borer. We are finding that the use of Bt transgenic corn results in decreased corn borer injury. This has resulted in decreased fungus discoloration in stalks and ears with a significant reduction in toxin as well.

A CHECK LIST OF PRACTICES WE ADVISE TO PREVENT TOXIN CONTAMINATION IN SILAGE

- Purchase corn and other feed varieties resistant to foliar, ear rot, and stalk rot diseases.
- Purchase varieties resistant to ear and stalk boring insects.
- Harvest corn and haylage at the recommended maturity and moisture level for your storage system. DO NOT let corn stand in the field after completed maturity or killing frost.
- Be sure chopper knives are sharp and cutting at the correct length to improve packing.
- Harvest forages as quickly as possible and pack tightly with the proper weight of tractor matched to the right number of packing hours and filling rate.
- Be sure the silo is sealed to exclude oxygen. Use plastic cover secured by tires on bunkers.
- Patch any holes in plastic covers, bags, or wrapped bails as soon as possible.
- Discard obviously spoiled feed or layers of feed.
- Since mycotoxins are highly soluble in water, do not allow rain to wash through upper layers of spoiled feed.

- Clean out leftover feed from feeding bunks regularly.
- Consider the use of an inoculant in silage or acid additive in high moisture corn to enhance fermentation and storageability.
- Match the rate of feed removal from the silo face to the size of the herd. In the north, bunker silo face should be removed at 4 to 6 inches and upright silo face at 3 to 4 inches per day. Use the higher rate during the warm seasons.
- When confronted with a toxicity problem, stop feeding the contaminated feed or dilute with a known clean feed.
- With your veterinarian or nutritionist, consider the use of a toxin adsorbent to be mixed with the feed such as sodium bentonite or a similar material.

Selected References

- Bullerman, Loyd B. "Mycotoxins and food safety." *Food technology*. May 1986. Volume 40. p.59-66.
- Carson, M.S., and T.K. Smith. 1983. Role of Bentonite in Prevention of T-2 Toxicosis in Rats. *Journal of Animal Science*. 57: 1498-1506.
- CAST. 1989. *Mycotoxins, Economic and Health Risks*. Council of Agricultural Science and Technology (CAST), Task Force Report No. 116. CAST Ames IA.
- Charmley, E., et al. 1993. Influence of level of deoxynivalenol in the diet of dairy cows on feed intake, milk production, and its composition. *Journal of Dairy Science*, 76: 3580-3587.
- DiCostanzo A., L. Johnston. L. Felice and M. Murphy. 1995. Effects of molds on nutrient content of feeds reviewed. *Feedstuffs*, January 16, 1995. pp17-20 & 52-54.
- Farrar, J.J. and Davis, R.M. Relationships among ear morphology, western flower thrips, and Fusarium ear rot of corn. *Phytopathology*. June 1991. v. 81 (6) p. 661-666.
- Lepon, P, et al. Occurrence of Fusarium species and their mycotoxins in maize. 7. Formation of DON in a maize plot artificially inoculated with *F. culmorum* and the influence of ensilaging on the stability of DON formed. *Archives of animal nutrition*. 1990. Volume 40. pp 1005-1012.
- Shurtleff, Malcolm. *Compendium of corn diseases*. 1986. p43-63.
- Park, J.J. Smalley, E.B. Chu, F.S. Natural occurrence of Fusarium mycotoxins in field samples from the 1992 Wisconsin corn crop. *Applied and environmental microbiology*. May 1996. v. 62 (5) p. 1642-1648.
- Alan Gottlieb, Professor, Plant & Soil Science Department, University of Vermont
alan.gottlieb@uvm.edu