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SCIENCE in Practice

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Safe or Sorry? How Much of Your Livelihood Are You Willing to Gamble?

Animal producers, nutritionists, veterinarians, and university scientists turn to juried scientific sources for answers about things possibly harmful to livestock. So, you would be in good company referring to quality research to guide you about these common feed-borne problems we call mycotoxins. But, you have to understand the nature of such research to make field-applicable interpretations. To illustrate, a dairyman milking 500 cows learns his herd is at risk from a mycotoxin called citrinin in his corn silage. His local university dairy scientist checks the literature and finds two relevant published papers. In the first, the researcher gave a single oral dose of citrinin to several older culled cows and reported post-mortem findings of kidney failure. This study used an amount of citrinin equivalent to feeding 15 parts per million (ppm) for 10 days, but was given all at once. The second study used contaminated corn, which when added to a total mixed ration (TMR) produced a dose of 4.5 ppm. The TMR was fed to heifers for two weeks. Blood tests showed that the six animals dosed with citrinin had significant elevation of certain enzymes and blood constituents suggesting mild kidney disease. The farmer's own corn silage tests at 1.25, 0.35, and 0.85 ppm citrinin in three samples. "Whew!", he says; "my cows are safe since the highest test is well below the 4.5 ppm in the second study."

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Molds – Why Production Animals Are Faced With Mycotoxins

To answer "Why," we need to be familiar with three things: what molds are and where they come from, how they grow, and why mycotoxins occur in animal feedstuffs.

First, let's look at the molds. Scientists use an extensive system of classifications to identify and group various living organisms. The kingdom **Fungi** includes mushrooms (such as the deadly *Amanita* and the common edible mushroom), mildews, and molds. Basidiomycetes are mushrooms, puffballs, smuts, and rusts. Ascomycetes are fungi with a readily identifiable sexual reproductive stage; many plant pathogens are in this class. Deuteromycetes, also called the "Imperfect Fungi" because they don't have readily identifiable sexual reproduction, are the "molds" with which we're most concerned. Molds are organisms which reproduce most often via spores, have cell walls composed mainly of chitin (the same material that forms the exoskeleton of insects), and have nucleated cells. People who study molds and other kinds of fungi are called *mycologists* from the Greek *'mycos'* which means cap and refers to the well known structural characteristic of the mushroom. More to the point here is that the same root word is the origin of the term *mycotoxin*, a poisonous chemical compound produced by a fungus. Modern mycotoxin specialists tend to further limit this definition to those toxic compounds produced by molds and causing demonstrable harm to one or more other living organisms.

If you take a close look at a moldy piece of bread, cheese, or fruit, you are seeing the item covered by a brightly colored material (see example, right). If you magnified that material, you would find yourself looking at a bed of thousands upon thousands of mold spores. The actual living organism is often obscured by the covering of spores!

Under all those spores, the mold's vegetative cells (called hyphae) are most often colorless (they will look white to us). Fragments of a vegetative cell will grow into new mold colonies. Spores, given the right environmental conditions, will germinate to form new mold colonies. Whether in the corn or wheat field or in a grain storage bin or silo, dispersal of such huge numbers of 'colony forming units' means many, many new sites of mold infection of plant material.

Next, let's take a look at mold growth. Molds will grow on almost any substrate that has a source of carbon and nitrogen. The mold needs two additional items, appropriate available water and oxygen. Consider the situation of ensiled feedstuffs. Carbon, nitrogen, and water are abundant. Usually we think of silage as being an anaerobic environment; however, leaks in silos, face cuts on bunks or bags, etc. all provide areas for some silage to be exposed to air, and thus, oxygen. But that also means that molds will flourish only in those discrete areas exposed to air. Ultimately then, the distribution of mold growth in that feedstuff is not uniform; rather, it is heterogeneously distributed.

Finally, why are production animals faced with mycotoxins? We now have mold actively growing in feedstuffs, but growth conditions do not remain static. A change in the mold's environment signals the need to form spores for survival. That could mean loss of available water to support growth, but more often is a shift in the relative proportions of nutrients. For example, a change in the C:N ratio (an increase) triggers sporulation in *Aspergillus flavus*,

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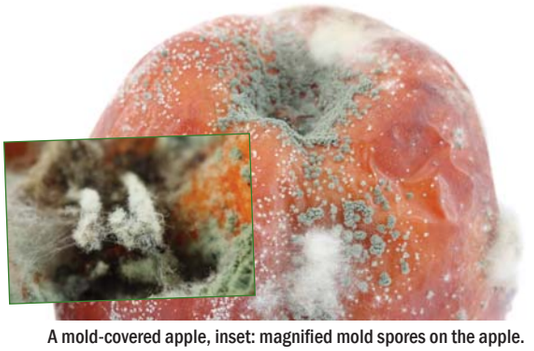

A mold-covered apple, inset: magnified mold spores on the apple.

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a mold responsible for aflatoxin synthesis. Secondary metabolism, the biochemical process through which mycotoxins form, occurs with the sporulation phase of a mold's life cycle. We don't really know the purpose of secondary metabolism, just that it occurs. And when the mycotoxin(s) has been made, some may remain inside mold cells, some may be released, and when the mold cells die, remaining toxin will continue to contaminate the feed since most of those chemical compounds are very stable.

For those feeding animals, this process means that status of available basic nutrients (C and N) plus water and air dictate where and how a mold will infect a feedstuff. Numbers, dispersal, etc. dictate how the inoculating mold spore or hyphal fragment will reach the feedstuff. Shifts in factors in the mold's environment will then initiate synthesis and deposition of mycotoxins. Combined these factors all tend to produce very random hot-spots of contamination within typical pre-harvest and post-harvest feedstuff situations. The consequence is that as mold produces mycotoxin(s), only some portions of the feedstuff will be contaminated, only some animals will actually be exposed, and, for the producer, the ability to sample and detect toxins is made infinitely more challenging.

In another edition we'll explore just how the mycotoxin reaches its taget of choice within the animal. ✤

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On the surface that certainly seems reasonable. Consider this. Controlled research studies attempt to remove or account for as many influencing factors as possible. In that idealized environment and using a very small number of test animals, large (acute) doses of a single mycotoxin are usually needed to obtain statistically significant results. However the dairy farm is not the research lab. There are more animals potentially exposed. Exposure may occur for longer periods. Other stressors are present. Also, while the researcher attempted to screen all feedstuffs to insure only the one toxin under investigation would be given to the cows, the farm is likely to see more than a half dozen different mycotoxins at once, and citrinin is not one of the more commonly found dairy feed contaminants. Now, take into account that cows in various stages of the life cycle are exposed, that there are different ages of animals, that health is not necessarily uniform for all animals, or just that each animal responds a little differently to a toxic challenge and you now have the conditions for a totally different level of citrinin to have a negative effect.

Which raises the next question: what was happening to the dairyman's cows? As it turns out, conception rate was down and many early lactation cows were showing signs of calcium insufficiency. Citrinin is primarily a kidney toxin. One function of the kidney is the hydroxylation (activation) of vitamin D to the form which regulates calcium absorption from the intestine. So, quite possibly a very small amount of citrinin fed over a long period of time was slowly reducing bone density – thus the issues with early lactation cows – and influencing reproductive performance. But the farmer had three samples tested. If the 4.5 ppm from the scientific paper is not a safe level, is 1.25 ppm or 0.85 ppm or 0.35 ppm safe?

The example is not real, but citrinin is, and the amounts and the possible action in an active dairy herd were derived from real reports for purposes of illustration. However, consider the mycotoxins about which much more information is known in dairy (e.g., deoxynivalenol, zearalenone, aflatoxin, etc.). Which reputable producer, nutritionist, veterinarian, or research scientist claims to know all ramifications of all possible interactions within a herd and can, therefore, provide reliable estimates of "safe" levels of any particular mycotoxin? Not one who has any understanding of these mold poisons! About the only reliable authority on what is safe for a particular herd or flock are the animals comprising that herd or flock. If positive test results suggest mycotoxin contaminated feed and animals are not performing optimally, it is wise to accept the fact that those mycotoxins known through the testing process (plus probably many more for which practical tests do not exist) have some role in that sub-optimal performance. Act accordingly and avoid the temptation to assume there is some arbitrary threshold for safety.

Interested in learning more about mycotoxin management? Contact Agrarian Marketing Corporation® at (888) 254-1482, or e-mail at amc801@aol.com. ✤