Utah State University
DigitalCommons@USU

All Archived Publications | Archived USU Extension Publications

1-1-1997

Mycotoxins

Clell V. Bagley

Warning: The information in this series may be obsolete. It is presented here for historical purposes only. For the most up to date information please visit The Utah State University Cooperative Extension Office

Recommended Citation
Bagley, Clell V., "Mycotoxins" (1997). *All Archived Publications.* Paper 98.
http://digitalcommons.usu.edu/extension_histall/98

This Article is brought to you for free and open access by the Archived USU Extension Publications at DigitalCommons@USU. It has been accepted for inclusion in All Archived Publications by an authorized administrator of DigitalCommons@USU. For more information, please contact digitalcommons@usu.edu.

Utah State University Extension
electronic publishing

Animal Health Fact Sheet

MYCOTOXINS

Clell V. Bagley, DVM, Extension Veterinarian
Utah State University, Logan UT 84322-5600

July 1997 AH/Beef/20

Certain metabolites of some fungi (molds) are poisonous (toxic). Fungal poisons have been known for many years, but they were not considered a major factor in animal disease until fairly recently. Because the prefix myco- refers to fungi, these toxins are termed mycotoxins. Penicillin is derived from a fungus and could be termed a mycotoxin, in relation to bacteria which are sensitive to it.

There are thousands of species of fungi but relatively few of these grow on agricultural products and only a fraction are capable of producing mycotoxins. It is known that about 100 fungi which grow on standing crops or stored feeds, produce toxic substances and approximately 20 of these have been associated with naturally occurring diseases.

A primary inoculum of the fungus onto the feed or food product is necessary for the fungus to develop, but abundant spores are usually present in soil, air and water. The toxigenic fungi spores are present almost everywhere and they can germinate, grow and elaborate their toxins into a variety of substrates when conditions of moisture, temperature and aeration are favorable. The optimal conditions for toxin production by different fungi may be quite variable.

A great portion of the mycotoxin problem is associated with stored grains and other concentrate rations, especially high-moisture corn, silage, cottonseed, peanuts and, to some extent, soybeans. Because of the involvement of stored feeds, the mycotoxicoses are especially important in intensively raised animals in confinement such as feedlot cattle, dairy cattle, market swine and poultry. Many of the mycotoxins are heat stable and are not inactivated by pelleting or even by ensiling. They are also non-antigenic, so immunity is not developed against them.

PRODUCTION OF MYCOTOXINS

Several factors influence mold development, including moisture, temperature, aeration and substrate. Moisture is probably the most important of these factors but the type of mold and whether or not a toxin is produced will depend on the interplay of all these factors.

Physical damage, such as breakage and stress cracks in grain, will increase the likelihood of fungal growth. High moisture corn, especially if ground, is highly susceptible to fungal invasion and toxin formation. To prevent production of the toxin on harvested products, care should be taken to prevent physical damage at harvest and to reduce the moisture level soon after harvesting.

Rapid ensiling or the addition of organic acids will aid in preventing the formation of additional mycotoxin.

DIAGNOSIS

Fungal growth may occur, without the production of mycotoxins. A "blacklight" can be used to detect the presence of mold growth on grain and is satisfactory for use as an initial test (for aflatoxin). It must be realized, however, that this checks only for the presence of mold growth and even if this test is positive, it doesn't mean that a toxin is present. Culture of mold or even isolation of specific molds from feeds means very little. The specific toxin must be isolated and/or its toxicity demonstrated.

Mycotoxin assays are generally directed toward specific compounds. Toxins that are not specifically looked for in the testing will be missed. The diagnostic problem is further aggravated by wide variations among samples of the same feed and sensitivities of animals. There are also interactions between the mycotoxins and other stresses on the animals. These factors make it very difficult to recommend "safe" concentrations in the feed.

Some mycotoxins can be identified in the rumen contents or urine. But often guidelines for diagnostic levels have not been established. The suspect feed is a better source for sampling but recognize that mold growth (and toxin production) can be very spotty.

SPECIFIC MYCOTOXINS THAT AFFECT CATTLE

Aflatoxin

Aflatoxins are a group of chemically related mycotoxins and are classified as B1, B2, G1, G2. The most common and toxic of the aflatoxin group is B1. Aflatoxin was first identified in stored grain, as a product of the fungus Aspergillus flavus, or A. flavus, hence the prefix afla-. It was later found that similar toxins were produced by toxigenic strains of Aspergillus parasiticus. Aflatoxin has been found in wheat, corn, barley, oats, coconut oil and meal, cottonseed, cassava, dry peameal and other raw food products. The molds which produce aflatoxin usually do not grow in silage, but aflatoxins already present can survive the acids produced during the ensiling process.

The main effects of aflatoxin are related to liver damage. If the dose is sufficient to produce an acute toxicity, it results in an increased clotting time and hemorrhage, especially in the intestinal lumen. There is also edema of the gall bladder. Acute poisoning causes hepatitis and necrosis of liver cells, resulting in prolonged blood clotting time, with affected animals dying from severe hemorrhages.

In subacute cases of poisoning, the liver lesions are those of regeneration and repair rather than cell necrosis. The bile duct cells proliferate and scar tissue forms. The rate of protein formation and the growth rate are depressed but the animal may not die.

In chronic cases, the lesions are those of chronic liver dysfunction. These usually include icterus, fibrosis or cirrhosis of the liver, ascites and pulmonary edema. The changes may be so subtle that they are overlooked, but it can result in decreased appetite, poor feed conversion, reduced growth rate and decreased resistance to disease. It may also cause diarrhea, bloody diarrhea, abortion, or deformities of the fetus.

Immunity is impaired by aflatoxins but the mechanism by which this occurs is still unclear. The species of animal involved and the time of intake relative to immunization both result in variations in the affect of aflatoxins. Aflatoxins may also be carcinogenic and result in tumor formation in some animals after prolonged consumption of feed containing as little as 0.5 to 15 parts per billion (ppb). This is evident in laboratory animals and especially in trout.

Young animals are usually more sensitive than are older ones. In calves, 150–200 ppb (or 0.5 mg/day) results in unthrifty animals. It would require 300–400 ppb to cause first calf heifers to lose weight and 2400 to 3100 ppb to reduce the appetite and markedly decrease production of

adult dairy cows. However, 10,000–12,000 ppb (10–12 ppm) would cause the death of an adult cow in about five days. Even lower levels may have an effect in the farm situation, compared to when a purified toxin is used in the laboratory. This is evidently due to the synergistic effects of other molds and their products in the farm situation.

Human exposure to aflatoxin can result from direct consumption of aflatoxin contaminated foods, whether processed or unprocessed. Aflatoxin ingested by food producing animals may also be transferred within the animal's body into meat, milk, or eggs and these would be potential sources for human exposure. Aflatoxin contaminated foods are deemed adulterated.

Residues of aflatoxin B1 have been found in the musculature and certain organs of poultry and swine after they were given aflatoxins in their feed. Aflatoxin B1 in the dairy ration is transformed to a metabolite, aflatoxin M1, in the cow's milk. The metabolite is as potent a carcinogen as is the parent toxin. When aflatoxin B1 is present in the cow's feed at a concentration of 100 ug/kg (ppb) it can result in aflatoxin M1 in the milk at a concentration of 1 ug/L.

Because the aflatoxins have been shown to be carcinogenic, federal law requires that the FDA establish a zero tolerance level for them in food. However, in practical application this is limited by the methods of measurement, so guidelines are provided to define the baseline for sampling and assay procedures. These guidelines apply to primary agricultural products because complete freedom of contamination cannot be achieved with present methods and technology. In 1965, the original limits were set at 30 ppb (ug/kg) in raw or finished products. In 1969 this was lowered to 20 ppb, and in the future it may be lowered further as technology improves. If there is evidence that the contamination could have been avoided, the zero tolerance level can be used by the regulatory officials for enforcement.

Ergot

Ergotism is caused by several mycotoxins from molds which invade the seed head of numerous grass and cereal species, especially rye. The ergot sclerotia are often removed from cereal grains during cleaning, but these screenings may be put into livestock feeds. Once grinding or milling has occurred the ergot cannot be recognized without microscopic examination or chemical analysis.

The primary clinical sign is dry gangrene with sloughing of the hooves, the tip of the tail and ears. It results from persistent constriction of the small arteries. Early cases may show only mild tissue swelling. A nervous form of ergotism has also been recognized. It results in hyperexcitability and tremors that are intensified by forced movement.

Rubratoxin

This toxin also affects the liver and accentuates the effects of aflatoxin when they occur together. It causes reduced liver function and rate of gain. It may result in hemorrhage and death. There may be a yellow, mottled discoloration of the liver. Suspect feed may show areas of red pigmentation.

Trichothecenes

This group of mycotoxins contains over 50 compounds. The toxins tend to form in temperate climates when wet weather delays the grain harvest. The fungus may even be destroyed later on, but the stable toxin remains.

Ingestion often results in reduced feed intake and eventually feed refusal. This in turn may cause unthriftiness and nutritional deficiencies. There may be lesions on the muzzle, lips, tongue and pharynx. Defects of the immune system are common.

Slaframine (Slobber Factor)

The clinical signs from this mycotoxin include profuse salivation, frequent urination, loss of appetite and watery diarrhea. The fungus and toxin are found on forages, especially red clover and red clover hay.

Facial Eczema

A photosensitivity due to liver damage may be caused by a fungus and mycotoxin that occur on a wide variety of dead plant litter of grasses and legumes. The animals appear as if "sunburned" on all areas of their skin that lack pigmentation (white).

Tremorgenic Toxins

The animals affected with one of these six mycotoxins show no signs until disturbed or excited. Then the apparent signs vary from muscular tremors to severe incoordination. They may become "paddling-downers."

TREATMENT

The basic efforts of treatment are directed to removal of the contaminated feed and good nursing care of the animals. All surgical procedures should be delayed until the liver function and blood clotting mechanisms have returned to near normal.

PREVENTION

Don't feed products that are obviously contaminated with mold unless they have been tested. The laboratory must be aware of the history, reasons for concern, and the specific mycotoxin analysis desired. Negative analytical results do not guarantee safe feed. The problem feed can also be fed to 2–3 test animals and their status closely monitored for any ill effects. Use of other feeds along with the contaminated products may aid to further dilute any ill effects.

Utah State University Extension is an affirmative action/equal employment opportunity employer and educational organization. We offer our programs to persons regardless of race, color, national origin, sex, religion, age or disability.

Issued in furtherance of Cooperative Extension work, Acts of May 8 and June 30, 1914, in cooperation with the U.S. Department of Agriculture, Robert L. Gilliland, Vice-President and Director, Cooperative Extension Service, Utah State University, Logan, Utah. (EP/DF/07-97)