MOLDY FEEDSTUFFS AND POTENTIAL TOXINS

COOPERATIVE EXTENSION SERVICE-GREAT PLAINS STATES

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At times, the feeding of moldy feedstuffs can have a markedly deleterious effect on livestock while, at other times, it has no discernable ill-effects whatsoever.

When is mold spoilage harmful? Any spoilage that causes an animal to reduce its intake of feed that upsets the metabolic processes, that contains toxic substances or that reduces the quality of edible products is harmful.

Mycotoxins

Certain conditions of mold growth cause certain molds to produce toxins. The toxins are called mycotoxins and the effects of the toxins on animals are called mycotoxicoses. More than 200 toxic compounds produced by molds have been identified. Fortunately most of these occur infrequently and, when they are produced, are low to moderate in toxicity.

Several of the more potent mycotoxins are listed in Table 1, along with the parent molds and the recorded symptoms of the mycotoxicoses produced. Of these, the most common mycotoxins found to date are aflatoxin, F-2 group (Zearalenone), sterigmatocystin, T-2 toxins, ochratoxin and rubitoxin in that order.

Acute Mycotoxicoses

The ingestion of a highly toxic level of mycotoxin results in symptoms similar to severe poisoning. The onset of vomiting, diarrhea, prostration and death can be both sudden and drastic. Acute mycotoxicoses have occasionally been mistaken for acute viral or bacterial diseases because of the rapidity and severity of the symptoms. When this happens, failure of therapeutic measures indicates the error of diagnosis.

Full recovery is rarely obtained from acute mycotoxicoses because of the damage to the liver and other vital organs.

Chronic Mycotoxicoses

Intake of sub-acute levels of mycotoxins is a common occurrence: When this happens, symptoms may be absent entirely or be evidenced by reduced growth rate and appetite. Clinical examination of these animals might show areas of liver necrosis, vascular changes, pinpoint hemorrhages, and gastric lesions.

Chronic mycotoxicoses can also reduce the natural body resistance to disease and infection and thereby become a complicating factor in diagnosis and treatment of a disease.

Animals showing chronic symptoms of aflatoxin, ochratoxin or rubitoxin mycotoxicoses recover slowly and incompletely when switched to feed free of toxins.

Long-Term Aflatoxin Intake

Chronic symptoms resulting from continued intake of low levels of aflatoxin by livestock are summarized in Table 2. Note the wide range of tolerance to the toxin.

Reduced growth rate and lowered feed efficiency are the first signs of toxicity. As intake continues, animals become less active and show reduced response to stimuli. Mortality may increase for young, rapidly growing animals, but not change for more mature animals.

Lactating animals will secrete aflatoxin in their milk.

Ruminants

Generally, ruminants appear to tolerate higher levels of mycotoxins and longer periods of low-level intake as compared to simple stomach animals. It is postulated that the rumen microorganisms interact with the toxins before they are absorbed into the body proper.

Sheep appear to excrete significant amounts of aflatoxin in their urine with a minimum of damage to liver and kidneys. Accordingly, they tolerate higher levels and longer period of intake of aflatoxin than can other livestock.

TABLE 1. Certain Mycotoxins, Their Sources and Their Symptoms

Toxin	Fungi Involved	Symptoms*
Aflatoxins B1, B2, G1, G2	*Aspergillus flavus, A. parasiticus, A. ruber, A. wenti A. oryzae, A niger, A. ostianus, A. ochraceus, Penicillium puberulum, P. variable, P. citrinum, P. frequentans, Rhizopus* sp	Vascular hemorrhages, hepatic necrosis, emesis, diarrhea, prostration, death. A carcinogen
Zearalenone (F-2 F-3 F-5)	*Fusarium graminearum, F. moniliforme, F. roseum F. tricinctum, G. zeae, F. roseum "Graminearum*"	Vulvovaginitis, vaginal or rectal prolapse, enlarged mammary glands, emesis, anorexia, restlessness, involved in feed refusal
Sterigmatocystin	*Aspergillus versicolor, A. flavus, A. parasiticus, A nidulans, A. ruber, P. luteum, A. chevalieri, A amstelodami*	Diarrhea, Nephritis, hepatic necrosis carcinogen-usually occurs with aflatoxin about 1/16 toxicity of aflatoxin
T-2 Trichothecenes	*F. tricinctum, F. scirpi, F. equiseti, F. roseum, F. oxysporum, F. nivale, F. episphaeris, F. lateritium, F. rigidiusculum, F. solani, F. poae*	Neural disturbances. dermatitis, oral lesions, diarrhea, anorexia, GI hemorrhages, death emesis, feed refusal
Ochratoxin	*A. ochraceus, A. sulphureus, A. sclerotiorum, A. alliaceus, A. melleus, A. ostianus, A. petrakii P. viridicatum, P. commune, P. cyclopium, P. variable, P. purpurresces -*	Impairment of kidney functions, some liver damage anorexia, diarrhea, prostration, death (estimated10x toxicity of aflatoxin to chickens).
Rubitoxin	*Penicillium rubrum*	Similar to aflatoxin
Ergot	*Claviceps purpurea*	Gangrene, blood clots, neurologic seizures.

Penicillic Acid	*P. puberulum P. viridicatum, P. cyclopium, P. thomii, P. baarnense, A. ochraceus, A sclerotiorum, A. alliaceus, A. melleus, A. sulphureus*	Cirrhosis, nephritis, convulsions, coma.
Kojic Acid	*A. flavus, A. oryzae. A. tamarii*	Convulsions, edema, prostration
Oxalic Acid	*A. niger, P. oxalicum*	Gastric irritation, lowered calcium, neurological disorders.
Fusarium roseum	*F. roseum*	Cirrhosis, nausea
Alimentary Toxic Aleukia	*F. sporotrichioides, F. tricinctum*	Leukopenia, hemorrhages, diathesis

*Some symptoms are those reported for field cases and may represent effects of more than one toxin or a co-existing malady.

Younger ruminants are more susceptible to aflatoxin toxicity than are older ruminants. This is particularly noticeable in the period before rumen function is established.

Other Toxicities

Toxic levels have not been clearly defined for ochratoxin or rubitoxin. However, South Dakota research indicates that ochratoxin may be more toxic (estimated 10X) than aflatoxin in poultry feeds.

F-2 Toxins (zearalenone or resorcinylic acid lactones) cause estrogenic responses in livestock. Symptoms include swollen vulva in females, shrunken, inactive testes in males, enlarged mammary glands in the young of both sexes, and abortion or resorption of fetuses in pregnant females. High intake will produce symptoms of severe poisoning and sudden death.

Continued intake of F-2 toxins at moderate levels can and will result in prolapses of vulva in gilts and rectal prolapses of both sexes in swine. Rectal prolapses have also been observed in cattle consuming corn that was suspected to have F-2 toxins.

Sterigmatocystin appears to be a precursor of aflatoxin and can usually be detected anytime anatoxin is present. It is about 1/16 the toxicity of anatoxin B1 but is produced in greater quantities. The effects and symptoms are similar to those of aflatoxin when the intake is toxicologically equal.

Carcinogenic Potential

Aflatoxin has been clearly demonstrated to be a carcinogen. Tumors have been found in all experimental animals receiving dosages of aflatoxin with liver tumors being most commonly observed.

Ochratoxin and rubitoxin are not known to be carcinogenic at the present time even though their toxic action is similar to that of aflatoxin. Patulin, sterigmatocystin and islandotoxin are known carcinogens and are subject to zero tolerance just as aflatoxin.

Prevalence

The exact prevalence of mycotoxins in grains and feedstuffs is not known. It is probably more than has been expected. Minnesota scientists have isolated toxin- producing molds from every

sample of suspect feedstuffs sent to their laboratory. Even with the skewed nature of the samples (coming from cases that baffled practicing veterinarians), this indicates the seriousness of the mycotoxin problem and the vast effect it could have on present day animal agriculture. The condition or the grain and feed fed should be a routine question for case histories of livestock problems.

Beneficial Toxins

Zearalenol (one of F-2 group or closely related resorcinylic acid lactone) is being commercially produced as a growth promoting hormone for beef steers and non- replacement heifers.

Identifying Molds

What kind of mold do I have in my feedstuffs? Since the same mold may have different colors at different stages of growth, we cannot use color for identification. Mold count analysis in a laboratory is the only way to identify the molds.

The most prevalent molds are the *Aspergilli*, followed by *Penicillii*, *Fusaria*, *Alternaria*, and *Cladospori*. Based on this prevalency, we may expect aflatoxin to be the most common toxin found followed by F-2 toxins, ochratoxin and rubitoxin. Laboratory identification of the molds and tests for the presence of toxins are necessary for positive diagnosis of a mycotoxicoses.

Table 2. Toxic Symptoms From Continued Intake of Aflatoxin

ANIMAL	Level of Aflatoxin PPB*	SYMPTOMS
Rainbow Trout	0.4	Liver Tumors
Ducklings	30	Liver Tumors
Turkey Poults	250	High Mortality
Chickens (early growth)	610	High Mortality
Chickens (late growth and mature)	1834	Mortality, Reduced Growth, Low Egg
Beef Cattle (450 lbs.)	700	Liver Damage
Beef Cattle (450 lbs.)	1000	Reduced Growth and Lower Feed Efficiency
Pigs (50 lbs.)	280	Reduced Growth and Lower Feed Efficiency
Pigs (80 lbs.)	450	Liver Damage
Pigs (80 lbs.)	615	Reduced Growth Rate
Pigs (80 lbs.)	810	Reduced Growth Rate and Lower Feed Efficiency
Sheep (mature)	1750	Lower Fertility

*PPB is parts per billion

Black Light

A long wave (365 nanometers) ultraviolet light may be used as a preliminary test on broken corn kernels. A bright greenish gold fluorescence is an indicator that mold metabolites are present and that aflatoxin may also be present (about 3 of every 4 times it is not present). If the bright greenish gold fluorescence (very close to the color emitted by a firefly) is absent, the grain is presumed to be free of aflatoxin.

Laboratory Analysis

A minicolumn test should be run on the "positives" from the black light test. A positive minicolumn should be followed by thin layer chromatography so that aflatoxin is separated from zearalenone, ochratoxin and sterigmatocystin and the amounts of these toxins determined. A method for T-2 toxins is being developed by Minnesota researchers.

Remove Or Deactivate

Most of the mycotoxins and aflatoxin in particular, are difficult to remove from feedstuffs. Ultraviolet irradiation and anhydrous ammonia under pressure will reduce the toxicity of aflatoxins and, if continued long enough, will deactivate them entirely. Aflatoxin can be extracted from a feedstuff with a water-isopropyl alcohol solution. These methods are not economically feasible at present.

Dry heat can be used to reduce aflatoxin levels in contaminated corn according to preliminary tests by the USDA Northern Regional Research Laboratories. Reductions from 60 to 90 percent in aflatoxin levels were observed when corn kernels were heated to 325³F for approximately 1 1/2 minutes.

Continued metabolism of the molds under conditions not fully understood, result in diminishing levels or mycotoxins, often reducing them to undetectable traces. Mixed populations of molds are less apt to generate mycotoxins than are single species. This is fortunate since most cases of spoilage involve a number of mold species. Neither effect can be counted on to alleviate a potential mycotoxicoses problem in spoiled feed.

Feeding Trial

One way to determine the potential toxicity of a spoiled feedstuff is to feed it to some expendable animals. The test period should be at least two weeks in length. Observe the animals daily for any sign of toxicity such but not absolutely) safe to feed the suspect feedstuff to other livestock. If ill effects are noticed, switch the animals to good feed immediately. A consultation with your veterinarian is suggested at this point. Dispose of the suspect feedstuffs by spreading it on land and plowing it under.

General Comments

Fungi are found everywhere on earth. Fortunately, not all of them form toxic metabolites; however, and in field or storage environmental conditions are capable of forming toxins and must be considered as potentially hazardous.

Freshly harvested grain has relatively low mold spore counts. However, the trip through elevating and handling equipment insures that the grain is well "seeded" with mold spores when it goes into the storage bin. Most spores are unable to grow at grain moisture levels below 14.5 percent. *Aspergilli* grow best at moisture levels of 17.5 to 21 percent and temperatures of 70° to 100 °F. Once spores begin to grow, they produce heat and moisture as a by-product. This triggers the growth of more spores. This snowball effect is well-known to grain men who monitor.