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MYCOTOXINS
A NATIONWIDE PROBLEM

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Toxic fungal metabolites are chemically diverse and occur in a wide variety of feeds and foods. They impair human health and cause economic losses in livestock through disease and reduced production efficiency.

The fungal organisms that produce mycotoxins can invade the feed supply, grow, and produce toxins when conditions are proper. This can occur during production in the field, transportation, processing, or storage. Some are produced on the farm in feed mixers or in feed bunks. The three major genera of mycotoxin-producing fungi are *Aspergillus*, *Fusarium* and *Penicillium*. Several factors which influence mycotoxin production are: strain of fungus, substrate, moisture, humidity, temperature, pH, growth of other fungi or microbes, and stresses such as drought. The major crops affected in the United States are corn, peanuts and cotton.

The aflatoxins, the most important group of mycotoxin discovered to date, are produced primarily by *Aspergillus flavus* and *A. parasiticus*. Sterigmatocystin is a precursor in the biosynthesis of aflatoxins but can also be produced as an end product by several species of *Aspergillus*. The large family of mycotoxins known as trichothecenes (T-2, the most notable) are produced by *Fusarium*. Zearalenone is produced by *F. graminearum*. The ochratoxins are produced by *A. ochraceus* and several *Penicillium spp*. Some species of *Penicillium* and *Aspergillus* are capable of producing either citrinin, citreovirdin, or cyclopiazonic acid. Fumonisins (the newest described mycotoxins) are produced by *F. moniliforme* and *F. proliferatum*. Tremorgens (literally 'tremor producing') are produced by species of *Penicillium*, *Aspergillus*, *Claviceps*, and *Acremonium*.

The toxicity of mycotoxins to animals range from acute death to chronic disease and interference with reproductive efficiency. Aflatoxins (notably B1 and its metabolite M1) can cause liver damage or cancer, decreased rate of gain, decreased milk and egg production, reduced feed efficiency, abortion and immune suppression. The young are most susceptible to the effects of aflatoxins, which may be expressed as gastrointestinal disturbances, anemia, jaundice, and reduced feed intake and efficiency. Nursing animals may be affected by exposure to aflatoxin metabolites secreted in milk.

The trichothecenes primarily cause necrosis and hemorrhage throughout the gastrointestinal tract, depression of blood regenerative processes in the bone marrow and spleen, and changes in the reproductive organs. Signs of disease include: weight

loss, reduced feed consumption and utilization, vomiting, diarrhea, abortion and death. Immune suppression may also be important in trichothecene affected animals.

Ochratoxin A causes kidney damage and renal fibrosis at concentrations usually found in feeds. However, higher concentrations may cause liver damage, as well as intestinal necrosis and hemorrhage. This mycotoxin is also immunosuppressive and carcinogenic. It does not seem to be a common problem across the United States.

Zearalenone is an estrogenic fungal metabolite which induces feminization in swine at dietary concentrations of less than 1 ppm. Higher concentrations can interfere with conception, ovulation, implantation, fetal development and the viability of newborn.

The fumonisins cause a wide variety of problems depending upon the species. Affected horses may have leukoencephalomalacia and/or liver damage while swine may have pulmonary edema and/or liver damage.

A wide variety of other effects in animals have been attributed to mycotoxins, including embryonic death, inhibition of fetal development, and abortions attributed to aflatoxins, zearalenone, rubratoxin, and ergot toxins. Teratogenicity has been described for aflatoxins, ochratoxin, T-2, zearalenone, sterigmatocystin and rubratoxin. Nervous system function is adversely altered by at least nine mycotoxins, including the tremorgenic mycotoxins. Clinical signs include tremors, uncoordinated movements, weakness in the legs, staggering and sudden muscular collapse. The neurological signs may be complicated with seizures, hemorrhage, diarrhea, profuse salivation, feed refusal and gangrene of the extremities.

Human mycotoxicoses are documented for relatively few of the many mycotoxins. Of considerable importance are those mycotoxins such as aflatoxins, that are potentially carcinogenic to humans. Major epidemiological studies regarding the aflatoxins have been conducted, primarily in Asia and Africa. Some have shown a positive association, while others have indicated a lack of positive association, between exposure of aflatoxins and disease outcome. Major criticism of many of these studies surround the role of hepatitis B virus as an agent for liver cell cancer in the populations studied. Therefore, the exact relationship of aflatoxins with liver cell cancer in humans has not been fully established. Nevertheless, in 1988, the International Agency for Research on Cancer placed aflatoxin B1 on their list of human carcinogens.

Unlike Africa and Asia, the incidence of liver cell cancer in the United States is relatively low as is the exposure to aflatoxins. Although there have been no complete investigations conducted in the United States, a limited retrospective population study demonstrated no apparent association between consumption of aflatoxins and liver cell cancer. New epidemiology studies in the United States, utilizing recently developed techniques, could provide a better determination of the extent of exposure to populations. They also would have the potential to aid in clarifying the role of aflatoxins in liver cell cancer and other disease states in the relative absence of alternate risk factors. However, conduct of these studies may not be practical. The liver cell cancer incidence in the United States is so low that such studies would require undeniably large numbers of participants to yield meaningful statistics. In contrast, cases of acute aflatoxicosis leading to death or disease involving gastrointestinal disturbances,

hemorrhage, vomiting and hepatic changes have been well documented in humans.

Mycotoxins can contaminate crops before harvest, in transport and in storage. Thus, raw or processed foods and feeds can become contaminated. With the exception of the aflatoxins, the frequency of contamination by mycotoxins is unknown. The aflatoxins are frequently detected in a variety of feeds and foods produced in the United States, as well as in imported commodities and products. Contamination of milk, eggs and meat can result from animal consumption of mycotoxin contaminated feed. Aflatoxins, ochratoxin and some trichothecenes have been given considerable attention, because they are either carcinogenic or of economic concern in animal health.

Additional mycotoxins produced by various *Aspergillus, Penicillium, Fusarium* and other fungal genera can contaminate foods; however their importance to animal and human health has not been established.

The economic losses due to mycotoxins are multifaceted involving direct crop and livestock losses through reduced health and production efficiency, regulatory programs, processing and diagnostic expenses. The incidence of mycotoxins varies among commodities, climatic conditions and regions. For these reasons, the economic importance of mycotoxins is difficult to quantify. Product losses likely occur, but except for certain corn and peanut products, milk, and eggs, there is no documentation. Increased costs resulting from mycotoxin-contaminated commodities are likely passed on to the buyer or consumer. Such costs have significant direct or indirect effects on the control of mycotoxins and international trade of commodities and processed feeds and foods.

Because mycotoxins are unavoidable, naturally occurring compounds, regulations provide an important means to control the quality of the food and feed in which they may occur. Presently, in the United States, only the aflatoxins are regulated. To accomplish such controls, the concentration of mycotoxins in foods and feeds must be accurately assessed. Such accuracy involves adequate sampling of the food or feed, chemical extraction, c1eanup and quantitative analysis of the mycotoxins. Although most of the procedures are developed for the laboratory and often utilize sophisticated equipment, field-practical screening tests are available for rapid detection of selected mycotoxins in certain commodities.

Regardless of our best efforts to control the occurrence of mycotoxins in commodities, they do occur, and therefore strategies have been developed to decontaminate or detoxify the commodity. Presently, this is true primarily for the aflatoxins. These methods for decontamination/detoxification include physical separation, thermal inactivation, irradiation, microbial degradation and chemical treatment. Ammoniation has been safely and effectively used for aflatoxins in some commodities used for animal feed, but has not yet been sanctioned by the U.S. Food and Drug Administration. A new approach using selective adsorption of aflatoxins by dietary compounds has resulted in reducing or preventing some adverse effects of aflatoxins. Continued efforts are needed to establish these kinds of methods to practically, safely, and effectively control mycotoxins in foods and feeds.


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