From Wikipedia

Animal feed

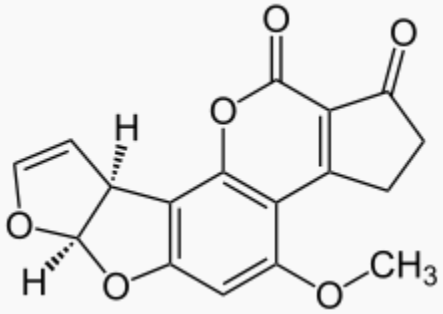
Copra meal is used as fodder for horses and cattle. Its high oil levels and protein are fattening for stock. The protein in copra meal has been heat treated and provides a source of high quality protein for cattle, sheep and deer, because it does not break down in the rumen.

Coconut oil can be extracted using either mechanical expellers, or solvents (hexane). Mechanically-expelled copra meal is of higher feeding value, because it contains typically 8-12% oil, whereas the solvent-extracted copra meal contains only 2-4% oil. Premium quality copra meal can also contain 20-22% crude protein, and <20ppb aflatoxin.

High quality copra meal contains <12% non-structural carbohydrate (NSC) which makes this product well suited for feeding to all horses that are prone to ulcers, insulin resistance, colic, tying up, and acidosis.[8]

Aflatoxin

Not to be confused with Alpha toxin.



Chemical structure of aflatoxin B_1

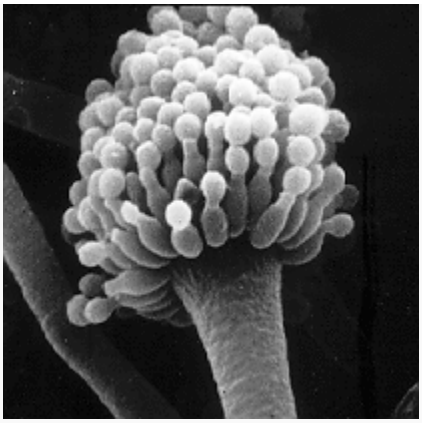
Aflatoxins are naturally occurring mycotoxins that are produced by *Aspergillus flavus* and *Aspergillus parasiticus*, species of fungi. The name was created around 1960 after the discovery that the source of turkey X disease was ***A**spergillus **fla**vus**toxins***.[1] Aflatoxins are toxic and among the most carcinogenic substances known. After entering the body, aflatoxins may be metabolized by the liver to a reactive epoxide intermediate or hydroxylated to become the less harmful aflatoxin M_1.

Major types of aflatoxins and their metabolites

At least 14 different types of aflatoxin are produced in nature.[3] Aflatoxin B_1 is considered the most toxic and is produced by both *Aspergillus flavus* and *Aspergillus parasiticus*. Aflatoxin G_1 and G_2 are produced exclusively by *A. parasiticus*. While the presence of *Aspergillus* in food products does not always indicate harmful levels of aflatoxin are also present, it does imply a significant risk in consumption. Aflatoxins M_1, M_2 were originally discovered in the milk of cows that fed on moldy grain. These compounds are products of a conversion process in the animal's liver. However, aflatoxin M_1 is present in the fermentation broth of *Aspergillus parasiticus*.

- Aflatoxin B_1 & B_2, produced by *Aspergillus flavus* and *A. parasiticus*
- Aflatoxin G_1 & G_2, produced by *Aspergillus parasiticus*
- Aflatoxin M_1, metabolite of aflatoxin B_1 in humans and animals (exposure in ng levels can come from a mother's milk)
- Aflatoxin M_2, metabolite of aflatoxin B_2 in milk of cattle fed on contaminated foods[4]
- Aflatoxicol
- Aflatoxin Q_1 (AFQ_1), major metabolite of AFB_1 in *in vitro* liver preparations of other higher vertebrates[5]

Contamination conditions



Aspergillus fumigatus as seen under an electron microscope

Aflatoxin-producing members of *Aspergillus* are common and widespread in nature. They can colonize and contaminate grain before harvest or during storage. Host crops, which include

maize, sorghum and groundnuts, are particularly susceptible to infection by *Aspergillus* following prolonged exposure to a high-humidity environment, or damage from stressful conditions such as drought, a condition that lowers the barrier to entry. In 2003, 120 people died in Kenya after eating maize with very high aflatoxin levels.[6]

The native habitat of *Aspergillus* is in soil, decaying vegetation, hay, and grains undergoing microbiological deterioration, and it invades all types of organic substrates whenever conditions are favorable for its growth. Favorable conditions include high moisture content (at least 7%) and high temperature. The Aflacontrol project, conducted by IFPRI with scientists from CIMMYT, ICRISAT and other organizations, sought to provide evidence of the cost-effectiveness of aflatoxin risk-reduction strategies along maize and groundnut value chains in Africa, and to understand what prevented adoption of these control strategies. The project found that, in both Kenya and Mali, maize drying and storage practices were inadequate in minimising exposure to aflatoxins.

The toxin can also be found in the milk of animals that are fed contaminated feed.

International sources of commercial peanut butter, cooking oils (e.g. olive, peanut and sesame oil), and cosmetics have been identified as contaminated with aflatoxin. In some instances, liquid chromatography-tandem mass spectrometry (LC-MS/MS), and other analytical methods, revealed anywhere from 48–80% of selected product samples as containing detectable quantities of aflatoxin. In many of these contaminated food products, the aflatoxin exceeded U.S. Food and Drug Administration (FDA), or other regulatory agency, safe limits.

The United States Food and Drug Administration (FDA) has established action levels for aflatoxin present in food or feed to protect human and animal health which range between 20 and 300 ppb.

Pathology

High-level aflatoxin exposure produces an acute hepatic necrosis, resulting later in cirrhosis, or carcinoma of the liver. Acute hepatic failure is made manifest by hemorrhage, edema, alteration in digestion, changes to the absorption and/or metabolism of nutrients, and mental changes and/orcoma.

No animal species is immune to the acute toxic effects of aflatoxins; however, adult humans have a high tolerance for aflatoxin exposure and rarely succumb to acute aflatoxicosis.

Chronic, subclinical exposure does not lead to symptoms as dramatic as acute aflatoxicosis. Children, however, are particularly affected by aflatoxin exposure, which leads to stunted growth and delayed development. Chronic exposure also leads to a high risk of developing liver cancer, as aflatoxin metabolites can intercalate into DNA and alkylate the bases through its epoxide moiety. This is thought to cause mutations in the *p53* gene, an important gene in preventing cell cycle progression when there are DNA mutations, or signaling apoptosis. These mutations seem to affect some base pair locations more than others — for example, the third base of codon 249 of the p53 gene appears to be more susceptible to aflatoxin-mediated mutations than nearby bases.

Medical research indicates that a regular diet including apiaceous vegetables, such as carrots, parsnips, celery and parsley, may reduce the carcinogenic effects of aflatoxin.

Moreover, aflatoxin B_1 can permeate through the skin. Dermal exposure to this aflatoxin in particular environmental conditions can lead to serious health risks.

Because aflatoxin B_1 can cause immune suppression, exposure is associated with an increased viral load in HIV positive individuals.

Pregnancy

Some studies showed significant relationship between exposure of Aflatoxin B1 (4 mg/kg, single dose) with teratogenesis in hamsters.

Microbiology

Aflatoxins are recognized as the most important mycotoxins. They are synthesized by only a few *Aspergillus* species of which *A. flavus* and *A. parasiticus* are the most problematic. The expression of aflatoxin-related diseases is influenced by factors such as species, age, nutrition, sex, and the possibility of concurrent exposure to other toxins. The main target organ in mammals is the liver, so aflatoxicosis is primarily a hepatic disease. Conditions increasing the likelihood of aflatoxicosis in humans include limited availability of food, environmental conditions that favour mould growth on foodstuffs, and lack of regulatory systems for aflatoxin monitoring and control.

A. flavus and *A. parasiticus* are weedy molds that grow on a large number of substrates, in particular under high moisture conditions. Aflatoxins have been isolated from all major cereal crops, and from sources as diverse as peanut butter and marijuana. The staple commodities regularly contaminated with aflatoxins include cassava, chillies, corn, cotton seed, millet,

peanuts, rice, sorghum, sunflower seeds, tree nuts, wheat, and a variety of spices intended for human or animal consumption. When processed, aflatoxins get into the general food supply where they have been found in both pet and human foods, as well as in feedstocks for agricultural animals. Aflatoxin transformation products are sometimes found in eggs, milk products and meat when animals are fed contaminated grains.

Detection in humans

There are two principal techniques that have been used most often to detect levels of aflatoxin in humans.

The first method is measuring the AFB_1-guanine adduct in the urine of subjects. The presence of this breakdown product indicates exposure to aflatoxin B1 in the past 24 hours. However, this technique measures only recent exposure, and, due to the half-life of this metabolite, the level of AFB_1-guanine measured can vary from day to day, based on diet, and thus is not ideal for assessing long-term exposure.

Another technique that has been used is a measurement of the AFB_1-albumin adduct level in the blood serum. This approach provides a more integrated measure of exposure over several weeks/months.

Animals

Aflatoxin has potential to lead to liver disease in dogs; however, not all dogs exposed to aflatoxin will develop liver disease. As with any toxic exposure, development of aflatoxicosis is a dose-related occurrence. Some dogs that develop liver disease will recover; those exposed to large doses for extended periods may not.

Low levels of aflatoxin exposure require continuous consumption for several weeks to months in order for signs of liver dysfunction to appear. Some articles have suggested the toxic level in dog food is 100–300 ppb and requires continuous exposure/consumption for a few weeks to months to develop aflatoxicosis. No information is available to suggest that recovered dogs will later succumb to an aflatoxin-induced disease.

Turkeys are extremely susceptible to aflatoxicosis. Recent studies have revealed that this is due to the efficient cytochrome P450 mediated metabolism of aflatoxin B1 in the liver of turkeys and deficient glutathione-S-transferase mediated detoxification. The mechanistic understanding of the susceptibility of turkeys to aflatoxin B1 is very relevant since turkeys are important from an agricultural standpoint.

There is no specific antidote for aflatoxicosis. Symptomatic and supportive care tailored to the severity of the liver disease may include intravenous fluids with dextrose, active vitamin K, B vitamins, and a restricted but high-quality protein diet with adequate carbohydrate content.

As a precautionary measure, both human and pet food recalls have occurred, casting a wide safety net to prevent exposure to potentially unsafe food. Recalled food products are subsequently sampled and tested for aflatoxin.

In 2005, Diamond Pet Foods discovered aflatoxin in a product manufactured at their facility in Gaston, South Carolina. Diamond voluntarily recalled in 23 states 19 products formulated with corn and manufactured in the Gaston facility. Testing of more than 2,700 finished product samples conducted by laboratories confirmed that only two date codes of two adult dog formulas with the "Best By" dates of April 3, April 4, April 5, and April 11 were potentially toxic.