

Fumonisin, Vomitoxin, and Other Mycotoxins in Corn Produced by *Fusarium* Fungi

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Certain *Fusarium* fungi are capable of causing a variety of diseases in corn, including seedling disease, stalk rots, and ear rots. In addition to their direct effect on corn yields, *Fusarium* fungi sometimes produce mycotoxins (poisonous substances produced by fungi) in the infected ears and kernels. The mycotoxins produced by *Fusarium* spp. in cereal grains are second only to the aflatoxins in attracting the attention of scientists and farmers.

Three mycotoxin-producing *Fusarium* fungi are common in Kentucky corn fields:

1. *Fusarium verticillioides* (formerly called *Fusarium moniliforme*). This fungus is also known by the name of the sexual stage, which is *Gibberella moniliformis*, formerly known as *Gibberella fujikuroi*.
2. *Fusarium proliferatum*.
3. *Fusarium graminearum* (also called *Gibberella zeae*).

Under certain conditions, kernels infected by *F. verticillioides* or *Fusarium proliferatum* can become contaminated with fumonisins. Fumonisin B₁ and related fumonisins have been implicated in equine leukoencephalomalacia (ELEM), also called blind staggers and moldy corn disease, and porcine pulmonary edema. Studies have also established the cancer-promoting activities of fumonisins. Of the mycotoxins produced by *F. graminearum*, two are common: vomitoxin—also called deoxynivalenol (DON)—and zearalenone. In vomitoxin-sensitive animals, such as swine, the presence of vomitoxin can cause feed refusal, and zearalenone can cause estrogenic syndrome. In addition to the fungi listed above, *Fusarium subglutinans* can also produce toxins in corn, including fusaproliferin.

Occurrence of *Fusarium* Toxins

Studies show that fumonisins are the most frequent mycotoxin in corn for our region and in the United States. For example, scientists at Purdue University surveyed mycotoxin occurrence in Indiana corn harvested from 1989 to 1993. The researchers began surveying for fumonisin in 1991, and they found that fumonisins were the most frequently detected mycotoxins in Indiana corn harvested during the period 1991 to 1993. However, fumonisin contamination varied greatly from year to year. For example, in 1991, almost all (96 percent) of 328 corn samples evaluated had some *Fusarium* ear rot. Of the

113 most severe samples, 44 percent had fumonisin levels above 5 ppm (parts per million). In contrast to 1991, all lots tested were below 5 ppm in 1992. More recent surveys from various regions of the United States show that fumonisins are commonly present in corn, although usually at levels below those known to pose a health risk.

In the Purdue study cited above, vomitoxin (DON) and zearalenone were detected less frequently than fumonisins. Vomitoxin was found above 1 ppm in 10 to 23 percent of fields sampled in 1990, 1992, and 1993 but not in any fields in 1989 and 1991. Zearalenone was not found above 1 ppm in any field sampled during the period from 1989 through 1993.

Data on the occurrence of mycotoxins in Kentucky indicate that the Purdue survey is representative of the general trends observed in the commonwealth. Preharvest mycotoxin contamination of Kentucky corn at levels that warrant concern is generally not common; however, mycotoxin contamination often occurs postharvest when corn is stored improperly.

Key Features of Biology

Fusarium verticillioides

F. verticillioides is widespread in the midwestern and southeastern United States. A closely related fungus—*F. proliferatum*—also occurs regularly, but much less is known about the biology of this fungus. Corn seeds and plants showing no symptoms are commonly infected with *F. verticillioides*. Usually, the majority of plants in a given field have symptomless infections of *F. verticillioides*. *F. verticillioides* can sometimes also be detected in crop debris on the soil surface.

Kernels may become infected in several ways. Probably the most common pathway is infection via the silk channel. Airborne spores of *F. verticillioides* are produced on corn residue and can land on corn silks. Once there, the spores germinate and infect the silks, especially as they turn green-brown and brown. The fungus then grows down the silk channel and among the developing kernels. Green silks are relatively resistant to infection and

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colonization, whereas green-brown and brown silks can be colonized by the fungus. Once *F. verticillioides* is in contact with kernels, it may enter them through the silk scars or through cracks and breaks in the seed coat. Another manner of spread of *F. verticillioides* is by the European corn borer and other insects, such as the corn earworm. The feeding activities of insects may spread spores to silks or directly to kernels and can create wounds in kernels, which are then colonized by the fungus. Kernels may also become infected via internal, systemic infection of the corn plant. *F. verticillioides* can internally colonize plants grown from infected seed. As plants grow from infected seed, internal colonization occurs in the stalk, the ear shank, the cob, and ultimately the kernels themselves.

Under certain conditions that are stressful for the corn plant, the fungus becomes pathogenic and causes disease of infected tissues. Infected seeds usually germinate normally and produce healthy seedlings, although *F. verticillioides* can cause damping off of seedlings under certain conditions, especially in supersweet corn hybrids. Stalk rot, ear rot, and kernel rot can occur in infected tissues, although many times infected tissues have no symptoms. There are no diagnostic symptoms or signs of Fusarium stalk rot, and it can be difficult to distinguish this from Gibberella stalk rot. The pith of the lower internodes becomes spongy as maturity approaches, taking on a whitish-pink to salmon discoloration. Distinctive fungal structures in tissues are rare, and laboratory analysis is required for definitive diagnosis.

Where Fusarium kernel or ear rots occur, symptoms often occur on individual kernels or groups of kernels scattered over the ear (Figure 1). Kernels often exhibit salmon-pink to reddish discoloration on the kernel cap, and the damage is sometimes associated with insect injury. White streaks radiating down from the tops of kernels (“starbursting”) is another symptom associated with *F. verticillioides*; however, other fungi can cause these symptoms as well. Once symptoms develop, the pathogen may continue to spread on the ear and form a heavy cottony mycelial growth that can consume the entire ear.

Rots caused by *F. verticillioides* and contamination by fumonisins appear to be favored by drought stress prior to and during silking. While infection of intact kernels may be symptomless, rotting and fumonisin contamination often occur when

the same kernels are damaged during maturation by birds or lepidopterous insects (corn earworms, European corn borers, or fall armyworms). Damaged, *Fusarium*-rotted kernels typically contain higher fumonisin levels than intact, healthy grain. This explains why corn screenings are often associated with animal toxicoses attributed to fumonisins. Fumonisin contamination is typically highest in lots with moderate to high levels of Fusarium ear rot. Fumonisin levels are often (but not always) roughly correlated with the level of Fusarium ear rot damage. However, fumonisin contamination can occur even in visibly healthy corn, although at much lower levels than in diseased kernels.

As a group, high-lysine hybrids have a higher incidence and severity of Fusarium kernel rot than do normal varieties.

The minimum moisture for growth of *F. verticillioides* in corn kernels is 18 percent. Thus, timely harvest, prompt drying (within 24 hours of harvest), and moisture management in storage are all important in reducing the risk of fumonisin contamination.

Fusarium graminearum

F. graminearum is common on Kentucky farms where corn and small grains are grown. In addition to causing stalk and ear rots in corn, *F. graminearum* causes head scab of small grains.

The fungus survives in soil on residues of small grains and corn. As an aggressive colonist of dead plant tissues, the fungus can also grow on and survive in soybean residue even though it does not cause disease on growing soybean plants. *F. graminearum* sometimes can continue to colonize crop residues for several weeks after harvest. The fungus best survives in residues on the soil surface. Burial by plowing reduces but does not eliminate survival and production of inoculum (spores that can initiate infection).

During cool, wet weather, spores of *F. graminearum* are produced on infested residue for one to three years following harvest of the susceptible crop. These can be spread by rainsplash or air movement to corn silks where they germinate and colonize the silks during wet weather. In contrast to *F. verticillioides*, *F. graminearum* requires succulent silk tissue, less than eight to 10 days old, for infection. By growing down the silk channel, the fungus is able to attack the developing ear and cause an ear rot. A reddish-brown discoloration of kernels progresses from the tip downward in affected ears (Figure 2). A whitish-red mold growth may be present on highly affected ears.

Stalks affected by Gibberella stalk rot, caused by *F. graminearum*, have a pink to reddish discoloration of the pith and vascular strands. An identifying feature is the presence of tiny, round, bluish-black perithecia (fungal fruiting bodies) that form on the surface of infected stalks in the fall or the following spring. These fruiting bodies are easily scraped off with a thumbnail. Laboratory diagnosis is recommended for positive diagnosis.

Wet weather and moderate temperatures around the time of silking favor ear rot development. The fungus can continue to grow and rot ears when corn is left standing in the field. Most cases of mycotoxin contamination of corn caused by *F. graminearum* are associated with excessively postponing harvest and/or storing high-moisture corn. Contamination of corn

Figure 1. Fusarium kernel rot. Note randomly scattered occurrence of damaged kernels. (Disregard the missing kernels on the lower ear.)



Figure 2. Gibberella ear rot. Note how rot typically progresses from the tip of the ear downward. (Disregard the missing kernels on the lower two ears.)



with vomitoxin is most abundant at temperatures of 70° to 85°F and grain moisture levels above 20 percent; optimal temperatures for zearalenone accumulation are 65° to 85°F.

Clinical Effects of *Fusarium* Toxins

At least three chemical classes of mycotoxins are produced by *Fusarium* fungi. Fumonisin is a recently discovered class of mycotoxin and have been associated with damage to brain function in horses and pulmonary disease in swine. A number of *Fusarium* toxins fall into the chemical class trichothecenes. These include T-2, vomitoxin (deoxynivalenol, or DON), diacetoxyscirpenol (DAS), monoacetoxyscirpenol (MAS), and nivalenol. The trichothecene that is generally of concern in Kentucky is vomitoxin. The trichothecenes as a group are strong irritants and have been associated with naturally occurring outbreaks of vomiting, feed refusal, and possibly gastric ulcers when consumed. Zearalenone represents a third chemical class of *Fusarium* mycotoxin. It possesses estrogenic activity and, when consumed by animals, has been associated with reproductive problems, such as abortions, false heat, recycling, reabsorption of fetuses and mummies, and vulval-uterine prolapse.

Fumonisin

Of the identified fumonisins produced by the fungus *F. verticillioides*, B1, B2, and B3 are the most abundant in contaminated food and feed. Fumonisin B1 is the most common, comprising approximately 75 percent of the total fumonisin content. This mycotoxin has been associated with equine leukoencephalomalacia (ELEM) and pulmonary edema in swine. It has also been associated with esophageal cancer in humans in certain regions of South Africa and China where consumption of fumonisin-contaminated corn has been common. In addition to their adverse affect on the brain, liver, and lungs, fumonisins also affect the kidneys, pancreas, testes, thymus, gastrointestinal tract, and blood cells. There is also concern that consumption of fumonisins during early pregnancy could result in elevated risk of neural tube defect in the developing fetus.

Horses

Horses consuming diets contaminated with fumonisins develop ELEM. This condition is characterized by a sudden onset of one or more of the following signs: frenzy, aimless circling, head pressing, paresis, ataxia, blindness, depression, and hyperexcitability. The primary pathologic feature of ELEM is softening and liquefying of the cerebral hemispheres of the brain. In conjunction with ELEM, muscular melting and cardiac failure have been reported in horses.

Current evidence suggests that horses consuming diets containing levels as low as 8 ppm fumonisin B may be at risk to develop ELEM. Symptoms will develop in about seven to 35 days. Horses are the most sensitive species to this toxin. Horses developing symptoms of ELEM usually die; recovery is rare. There could be a wide variability in individual susceptibility, especially in field cases due to variable length of exposure with different levels of fumonisins.

Poultry and Swine

Studies with chicks and pigs have indicated that lymphocyte viability is reduced by fumonisins, which can have a negative impact on the immune system. As mentioned earlier, this toxin has also been associated with pulmonary edema in swine. The mycotoxin fusaric acid (another toxin associated with *F. verticillioides*) has been reported to interfere with normal bone development of pigs and chickens.

Ruminants

Steers have been fed diets with 150 ppm fumonisins, resulting in some liver lesions. Overall, cattle appear to be less susceptible to fumonisins compared to other species.

Vomitoxin

Swine

Vomitoxin is a metabolite of *F. graminearum* that usually causes pigs to vomit following consumption of feed with high concentrations of the toxin. Swine are the most sensitive livestock species to vomitoxin. The most common effect of vomitoxin is reduced feed intake or feed refusal. Diets containing 5 ppm can reduce feed intake by 30 to 50 percent. Vomiting has been reported in swine ingesting finishing diets containing greater than 10 ppm vomitoxin. The reason pigs refuse feed containing vomitoxin is not known. There is little evidence that smell, taste, or a combination of the two is responsible. The lowered weight gain associated with the toxin is due directly to the reduction in feed intake. This can be somewhat compensated for by increasing the nutrient concentration of the diet, depending on the magnitude of feed reduction, which is positively correlated to the level of toxin in the diet. In studies conducted at the University of Kentucky, gain decreased approximately 8 percent and feed intake 10 percent for each 1 ppm increase of vomitoxin in the diet.

There are few reports available on the effect of vomitoxin on pregnant and lactating females. In one study, gilts fed diets containing 3.5 ppm vomitoxin had decreased feed consumption and growth rate and had a trend toward reduced fetal weight and length, but no macroscopic signs of abnormal fetal development were evident between 50 and 54 days of pregnancy.

Poultry

Experimental studies with vomitoxin-contaminated grains have been conducted in broilers and layers. Adverse production effects were not observed in broilers and layers ingesting feed contaminated with up to 5 ppm in the diet for 168 days. Effects on a hen's egg production, egg weight, feed efficiency, fertility, or chick weights at hatching were not detected in layers ingesting feed containing 18 ppm vomitoxin in the diet. Also, reports have indicated that no ill effects were detected in turkey poults given a diet containing 5 ppm of vomitoxin.

Ruminants

Ruminants appear to be less sensitive to dietary vomitoxin concentrations than are monogastrics (particularly swine), perhaps due to the presence of rumen microorganisms. It is postulated that these microorganisms partially degrade the toxin prior to absorption into the blood stream and vital organs of the animal.

In trials with dairy cattle, up to 6.5 ppm vomitoxin has been fed to dry cows and 12 ppm to lactating cows in the concentrate dry matter for 42 and 70 days, respectively, with no effect on milk production, body weights, or milk composition. Experiments with cattle and sheep also indicate that the effect of vomitoxin on animal health or performance is negligible. Diets containing vomitoxin, but negative for other prevalent toxins, indicate that up to 15 or 10 ppm vomitoxin in the diet dry matter were tolerated by sheep and cattle, respectively, without any adverse effects on animal health or performance.

Zearalenone Swine

Zearalenone is another common toxic metabolite produced by *F. graminearum*. This estrogenic mycotoxin affects reproductive efficiency but generally not feed intake. Swine are the most susceptible to the effects of zearalenone. In the prepubertal female, classic field observations of animals ingesting 1 to 5 ppm zearalenone include vulval reddening and/or swelling, which may progress to vaginal or rectal prolapse. The teats and mammary glands also become enlarged. These outward changes are accompanied by an enlarged, potentially twisted uterus and shrunk ovaries. On withdrawal of contaminated feed, clinical signs disappear within three to four weeks. There is little evidence of permanent changes interfering with subsequent reproductive processes.

Compared with the immature female, zearalenone ingestion causes more disruptive changes in the mature female. Levels of 3 to 10 ppm ingested by mature females have induced pseudopregnancy. The females do not cycle and cannot be mated successfully, and the breeding program is disrupted. Also, placental membrane weights can potentially be reduced, affecting fetal development. In view of present knowledge, giving pregnant and lactating sows feed containing zearalenone at levels exceeding 4 to 5 ppm would be risky if reproductive efficiency were expected to remain at optimum level.

In young males, levels exceeding 10 ppm of zearalenone in the diet have reduced libido, resulting in atrophy of the testes and enlargement of the mammary gland. Mature boars appear

to be able to tolerate high levels of zearalenone without any effect on the reproductive system.

Poultry

Broiler chicks and laying hens are not greatly affected by zearalenone, even when consuming large quantities of the toxin. Turkeys, on the other hand, when eating feed containing 300 ppm, developed enlarged vents within four days. However, no other gross effects were noted.

Ruminants

When sheep were fed a level of 12 ppm zearalenone in diet dry matter for 10 days prior to introduction of the ram, they exhibited prolonged estrous behavior, reduced ovulation rate, and reduced fertility. There were no effects of the toxin on embryo survival or lambing performance when this level was fed for 10 days immediately post-mating. However, zearalenone may exert its toxic effects by accumulating in the body when low levels are fed long-term or as a result of short-term exposure to high concentrations. For instance, reduced ovulation rate was evident in ewes exposed to 1.5 ppm of the toxin in diet dry matter content for only 10 days pre-mating, while ovulation rate was reduced in ewes exposed to only 0.5 ppm for 20 to 40 days pre-mating. Apparently, only ewes are affected by zearalenone when dosed prior to mating. Rams fed 2.5 ppm zearalenone in the dry matter portion of the diet for 30 days had normal semen production and fertility.

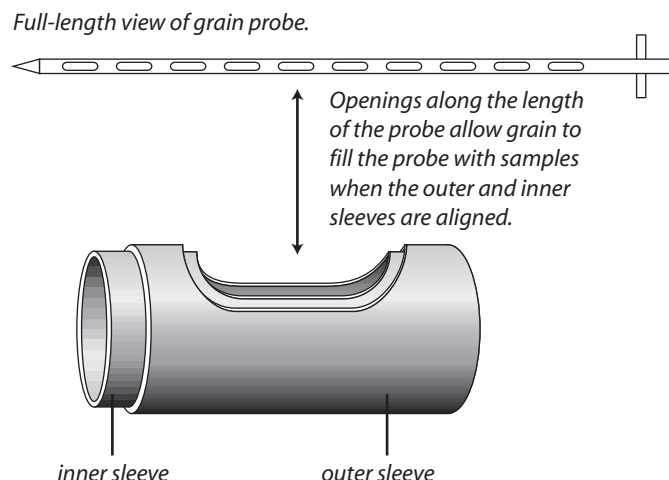
The effects of zearalenone on reproductive performance of cattle is not clearly defined. Recommendations in the literature would indicate not to exceed 5 ppm in the dry matter portion of the diet fed to growing heifers prior to their first breeding. Heifers fed 15 ppm zearalenone in the dry matter portion of the diet had reduced ovulation rates. Reported effects of zearalenone on production and reproduction of mature cows have not been consistent enough to suggest acceptable levels. Some studies have exposed either dry or lactating cows to levels of 100 ppm in the dry matter portion of the diet for up to 42 days with no adverse physiological effects, although some swollen genitalia were observed. It would appear that mature cows may be more resistant to zearalenone toxicosis than heifers.

Sampling and Detection

Collecting a representative sample is critical to obtaining accurate estimates of mycotoxin concentration. Studies show that up to 90 percent of the variability in test results comes from sampling variability.

For stationary shelled corn, sample using a grain probe, sometimes referred to as a "trier" (Figure 3). Don't collect a sample just from the most convenient place, such as the top of the truck or storage bin. The odds are good that this will give a misleading result, since mycotoxins are distributed very unevenly in a lot of corn. Details on sampling patterns appropriate to different grain carriers are provided in the publication *Grain Fungal Diseases and Mycotoxin Reference*. Take a minimum of 4 to 5 probefuls (preferably 10 probefuls) and collect 10 pounds of corn. For a moving stream of grain, use a diverter-type mechanical sampler (Figure 4). If one is not available,

Figure 3. Double-tube grain probe or “trier” in top image. The separate components are shown in the bottom image. The outer tube blocks the openings to the inner tube until the probe is inserted into the grain; the outer tube is then rotated, exposing the openings of the inner tube so that they may fill with grain.



cautiously grabbing fistfuls can also be suitable; take care to avoid personal injury.

If the test is not to be performed within 12 to 24 hours, dry the corn to below 16 percent moisture. If high-moisture corn is held for an extended period of time before testing, the test results may not be accurate, as *Fusarium* fungi can continue to grow and produce mycotoxins in the sample.

Grind the aggregate sample and mix it very well. A 1- or 2-pound subsample should be drawn from that; a riffle divider is the best way to obtain a representative subsample. Blend this subsample by lifting or rolling the ends of the bag to the opposite side and repeating at least ten times. A final subsample can be drawn from this for testing. Be sure to thoroughly clean grinding and sampling equipment between samples. Remember you are working with potentially toxic materials. For personal protection, wear a dust mask when grinding, mixing, and dispensing the sample. Minimize dust at all times.

Analysis of samples by an analytical laboratory is the most reliable way to determine the presence of *Fusarium* toxins in grains. A list of laboratories that conduct mycotoxin assays is available in the University of Kentucky Cooperative Extension



Figure 4. Diverter-type sampler installed in spout. (Courtesy, USDA Grain Inspection, Packers, and Stockyard Administration, Technical Services Division)

sion publication *Laboratories for Mycotoxin Analysis* (PPFS-MISC-1).

Rapid detection test kits are also available for detecting the *Fusarium* toxins discussed in this publication. A list of commercially available test kits for the detection of fumonisin, vomitoxin, and zearalenone are listed in the University of Kentucky Cooperative Extension publication, *Commercially Available Test Kits for the Detection of Mycotoxins in Corn* (PPFS-AG-C-3). Be sure the test used is approved by the USDA Grain Inspection, Packers, and Stockyard Administration.

Reducing Risk of Contamination from *Fusarium* Toxins

Production Practices

Preharvest contamination of corn with fumonisins is typically associated with drought stress prior to and during silking. Thus, production practices that minimize stress should reduce the risk of preharvest fumonisin contamination.

Be sure the hybrids sown are adapted to local climatic and soil conditions. Hybrids grown outside of their range of adaptability tend to accumulate higher levels of fumonisins than adapted hybrids. Although heritable differences exist among certain corn inbreds in susceptibility to *Fusarium* infection and mycotoxin contamination, most commercial hybrids are susceptible. Since *F. verticillioides* only colonizes silks as they turn from green to green-brown and finally brown, use of hybrids with tight husks that maintain silk integrity for longer periods may help reduce the incidence of fumonisins in some cases. Tight husks could enhance the risk of field outbreaks of Gibberella ear rot by retaining moisture inside the ear, although typically *Fusarium* ear rot is a much greater concern in Kentucky.

High levels of ear-feeding insects can enhance fumonisin levels; therefore, controlling insects that can damage kernels can help reduce fumonisin contamination. The Bt endotoxin is very active against both the European corn borer and southwestern corn borer, both of which can create wounds on kernels that allow *F. verticillioides* to invade. If a producer expects high levels of activity from corn borers (either European corn borer or southwestern corn borer), use of a corn hybrid that expresses high levels of the Bt endotoxin in kernels and provides good late-season corn borer control (MON810 and BT11 events, for example) may result in lower levels of fumonisin contamination in the harvested grain. As a guideline, the greatest threat from these borers is in late-planted corn (past May 10 through May 15 in Western Kentucky). Use of Bt corn does not assure the producer freedom from kernel-feeding insects. The Bt toxin only suppresses the corn earworm and provides no control at all of fall armyworm. However, use of Bt corn hybrids in fields where high insect pressure is expected may reduce fumonisin contamination in some cases. Understand that the use of Bt corn that expresses the endotoxin in green tissues and pollen only would not be expected to consistently reduce fumonisin levels.

Avoid excessive plant populations; consult with your seed supplier when deciding on planting density. Maintain adequate levels of nitrogen and other nutrients for good growth. If soil is compacted, subsoiling can alleviate stress and may reduce the risk of fumonisin contamination.

A rotation away from corn and small grains for two to three years can reduce inoculum levels of *F. graminearum* in a field under conservation tillage. However, recognize that in an epidemic year, airborne spores of *F. graminearum* are abundant and widespread. Rotation is thus a supplement to other practices for reducing the risk of mycotoxin contamination due to *F. graminearum*.

Harvesting, Drying, and Storage

The potential always exists for harvested corn to be decayed by and contaminated with mycotoxins from *Fusarium* fungi, if conditions permit growth of the fungi. Symptomless corn plants and kernels are commonly infected by *F. verticillioides*. Spores of *F. graminearum* are also commonly present at some level in many Kentucky corn fields. Contamination of corn by toxins produced by *F. graminearum* usually is the result of excessively postponing harvest and/or storing high-moisture corn. Thus, timely harvest and proper storage can reduce the risk of contamination of corn by *Fusarium* toxins, particularly those produced by *F. graminearum*.

Generally, the best moisture level to start harvest is between 25 and 27 percent. Leaving the corn in the field for a long period after maturity increases the risk of contamination from mycotoxins. The combine should be adjusted to harvest the grain with a minimum of kernel damage. Shelled corn should be dried to 16 percent or less within a day or two of harvest, as *Fusarium* fungi grow readily in shelled high-moisture corn. Stored shelled corn should be aerated regularly and monitored during storage. More information on grain storage is provided in the University of Kentucky Cooperative Extension publication *Principles of Grain Storage* (AEN-20). Drying of corn with heat does not alter the fumonisin content of the grain.

Feeding Contaminated Grain

Recognize that there may be many other unidentified compounds produced by *Fusarium* fungi that are toxicologically important. For example, in one study with swine, feed amended with 5 ppm of purified vomitoxin was readily eaten by the animals, whereas they refused to eat feed naturally contaminated with 5 ppm vomitoxin. Predicting the effects of utilizing feeds of known analytical composition is still difficult.

When corn is contaminated with fumonisins, FDA recommendations presented in Table 1 provide guidelines for the utilization of the grain.

FDA recommendations for the use of grains contaminated with vomitoxin are provided in Table 2. These guidelines apply to the interstate movement and utilization of vomitoxin-contaminated grain and grain by-products.

When Kentucky producers have a grain source contaminated with either vomitoxin or zearalenone, Table 3 provides acceptable guidelines for the use of that source in livestock feed. The recommendations listed in Table 3 are considered acceptable based on the research published to date. The FDA guidelines listed in Table 2 would, of course, apply to all interstate shipment of contaminated corn.

Table 1. Maximum levels of fumonisins in corn and corn by-products recommended by the U.S. Food and Drug Administration (November 9, 2001).

Human Foods	
Product	Total fumonisins (FB1 + FB2 + FB3)
Degermed dry-milled corn products (e.g., flaking grits, corn grits, corn meal, corn flour with fat content of < 2.25% dry weight basis)	2 ppm
Whole or partially degermed dry-milled corn products (e.g., flaking grits, corn grits, corn meal, corn flour with fat content of > 2.25% dry weight basis)	4 ppm
Dry-milled corn bran	4 ppm
Cleaned corn intended for mass production	4 ppm
Cleaned corn intended for popcorn	3 ppm
Animal Feeds	
Corn and corn by-products intended for:	Total fumonisins (FB1 + FB2 + FB3)
Equids and rabbits	5 ppm (no more than 20% of diet)**
Swine and catfish	20 ppm (no more than 50% of diet)**
Breeding ruminants, breeding poultry, and breeding mink*	30 ppm (no more than 50% of diet)**
Ruminants ≥ 3 months old being raised for slaughter and mink being raised for pelt production	60 ppm (no more than 50% of diet)**
Poultry being raised for slaughter	100 ppm (no more than 50% of diet)**
All other species or classes of livestock and pet animals	10 ppm (no more than 50% of diet)**

* Includes lactating dairy cattle and hens laying eggs for human consumption.

** Dry weight basis.

Table 2. Advisory levels for maximum concentration of vomitoxin in grains and grain by-products (U.S. Food and Drug Administration, September 16, 1993).

Concentration	Product or Use
1 ppm	Finished wheat products (e.g., flour, bran, and germ) that may potentially be consumed by humans. FDA is not stating an advisory level for wheat intended for milling because normal manufacturing practices and additional technology available to millers can substantially reduce DON levels in the finished wheat product from those found in the original raw wheat. Because there is significant variability in manufacturing processes, an advisory level for raw wheat is not practical.
10 ppm	Grains and grain by-products destined for ruminating beef and feedlot cattle older than 4 months and for chickens with the added recommendation that these ingredients not exceed 50 percent of the diet of cattle or chickens.
5 ppm	Grains and grain by-products destined for swine with the added recommendation that these ingredients not exceed 20 percent of their diet.
5 ppm	Grains and grain by-products destined for all other animals with the added recommendation that these ingredients not exceed 40 percent of their diet.

Be aware of the risks of feeding corn screenings to livestock. Fumonisin levels can be 50 to 500 times higher in screenings than in intact, healthy corn. Diseased kernels appear to be more brittle than healthy kernels, so they break apart more easily and end up in the screenings. Plus, some scientists believe that *F. verticillioides* grows more readily in broken than intact kernels. If screenings are being fed (which is not advisable), each lot should be tested for the presence of mycotoxins using commonly available test kits approved by the USDA Grain Inspection, Packers, and Stockyard Administration. Screenings may also be a source of toxic weed seeds for which test kits are not available.

Table 3. Recommended maximum concentrations of vomitoxin and zearalenone in livestock diets for use of contaminated grain lots on Kentucky farms.

Species	Vomitoxin	Zearalenone
	ppm	ppm
<i>Swine</i>		
Nursery	1.0	1.0
Grower/Finisher	1.0	1.0
Sow herd	1.0	2.0
Breeding males	1.0	2.0
<i>Poultry</i>	ppm	ppm
Broilers	5.0	no effect
Layers	5.0	no effect
Turkeys	5.0	no effect*
	(% dietary DM basis, ppm)	(% dietary DM basis, ppm)
<i>Ruminants</i>		
Sheep	15.0	0.5
Feeder cattle	15.0	5.0
Cows, mature	15.0	10.0**
Dairy cows	5.0	10.0**
Heifers	15.0	5.0

* In one study with 300 ppm zearalenone (greatly exceeds naturally occurring levels), turkeys developed enlarged vents within four days with no other gross effects noted.

** Recommendations for mature cattle are not well defined, but one study indicated 100 ppm fed for 6 weeks had no adverse effect; therefore, 10 ppm appears to be a safe guideline.

Other Uses of Contaminated Grain

When corn is wet-milled, the majority of the fumonisins end up in the gluten, fiber, germ, and steep water fractions. Thus, products derived from the starch fraction should be relatively low in fumonisins. Fumonisin are generally not detected in cornstarch. Likewise, fumonisins are typically not detected in ethanol or corn oil. Heating by frying or extrusion has been reported to reduce fumonisin levels. Fermentation has no effect on fumonisin contamination.

At low levels of fungal invasion, DON and zearalenone are mostly present in the nonstarch fractions during wet-milling. Thus, products derived from the starch fraction contain the lowest toxin concentration. Zearalenone has been reported to be destroyed at temperatures of 250°F or more.

Additional References

The Cooperative Extension Service (CES) publications listed below are available through county Extension offices. Many are also available on the World Wide Web at <<http://www.ca.uky.edu/agc/pubs/pubs.htm>>.

- *A Comprehensive Guide to Corn Management in Kentucky* (ID-139). M. Bitzer and J. Herbek, eds. CES publication.
- *Aflatoxins in Corn* (ID-59). CES publication available at <<http://www.ca.uky.edu/agc/pubs/id/id59/id59.htm>>.
- *Commercially Available Test Kits for the Detection of Mycotoxins in Corn* (PPFS-AG-C-3). Available at <<http://www.ca.uky.edu/agcollege/plantpathology/PPAExten/PPFShtml/ppfsagc3.htm>>.
- Grain Fungal Diseases and Mycotoxin Reference. USDA Grain Inspection, Packers, and Stockyards Administration. Available at <<http://www.usda.gov/gipsa/pubs/mycobook.pdf>>.
- *Head Scab of Small Grains in Kentucky* (PPA-38). CES publication.
- *Laboratories for Mycotoxin Analysis* (PPFS-MISC-1). Available at <<http://www.ca.uky.edu/agcollege/plantpathology/PPAExten/PPFShtml/ppfmisc1.htm>>.
- *Principles of Grain Storage* (AEN-20). CES publication.