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Corn Ear Rots, Storage Molds, Mycotoxins, and Animal Health

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Corn Ear Rots, Storage Molds, Mycotoxins, and Animal Health



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Contents

<i>Toxigenic ear rots and molds</i>	3
Fusarium ear rots	3
Gibberella ear rot.....	4
Fusarium toxins	5
Aspergillus ear rot and storage mold.....	5
Penicillium molds	6
<i>Non-toxigenic ear rots and molds</i>	6
Diplodia ear rot	6
Nigrospora ear rot.....	7
Cladosporium ear rot	8
<i>Animal health effects of Fusarium toxins</i>	8
Trichothecenes	8
Zearalenone	10
Fumonisin.....	11
Aflatoxin.....	12
Ochratoxin.....	12
<i>Detection of mycotoxins</i>	13
Sampling.....	13
Analysis	13
<i>A checklist for preventing molds and toxins</i>	14
In the field.....	14
After harvest	14
<i>Utilization of affected grain</i>	15

Ear rots and storage molds occur every year on corn throughout the Midwest. These diseases are serious concerns in corn production because they cause losses in grain yield and quality, and pose potential animal health risks from feeding moldy grain, due to the presence of toxic chemicals (mycotoxins). Fungi can produce many chemical products, some of which are medically useful, while others (mycotoxins) are toxic to animals and/or people. There are many fungi that can be present on corn, and several of the common species are toxigenic. Because of losses in yield and quality and the possibility of mycotoxin production, it is important to recognize ear rots and storage molds and be aware of their toxigenic properties.

The occurrence of ear rots and storage molds is

influenced by environmental and genetic factors which play a role both in the field and in storage. In addition, production and handling procedures can have positive or detrimental effects on grain susceptibility and mold and toxin development.

Fungi often are classified as ear-rot fungi or storage fungi, depending on their ability to develop in grain at low moisture content. Ear-rot fungi usually attack plants and cause disease in the field, so the majority of damage is done prior to harvest. However, these fungi may continue to develop in storage if conditions are favorable. Ear-rot fungi typically do less damage in storage because they do not grow at low grain moisture content (less than 18%).

Storage fungi generally do not cause extensive damage to ears in the field, but some damage may

occur, especially in kernels that are wounded by insects or other animals. These fungi are associated with dead plant debris and soil; they usually contaminate kernels during harvest. Kernels may be infected at such low incidence that it is not noticeable, or the spores contaminate the surface of kernels without penetrating. The majority of the damage subsequently occurs in storage. These fungi generally can grow at much lower grain moisture content (as little as 14.5%) than the ear rot fungi.

Symptoms of mycotoxicoses in animals can range from acute illness and death to a gradual loss in productivity. Acute illness, occurring suddenly and with some death loss, is not commonly associated with the well known mycotoxins. It is more likely that animals are affected with chronic (long term)

disease such as prolonged liver or kidney damage. Even more common (and harder to detect) are subtle effects on productivity. These can include reduced feed intake or feed efficiency, weight loss or reduced gain, impaired fertility and compromised immune function. Generally, outbreaks of mycotoxicoses occur seasonally, and they are associated with a specific feed source.

Many of the ear rots and molds can be identified to some extent by their appearance to the naked eye. Characteristics to look for are the color and texture of the fungal growth, and the distribution of fungal mycelium on the ear. The following are the most commonly encountered ear and storage fungi in the Midwest.

••••• *Toxigenic ear rots and molds* •••••

► **Fusarium ear rots** ◀

The most common ear diseases in much of the Midwest are caused by fungi in the genus *Fusarium*. *Fusarium moniliforme* and the closely related *F. proliferatum* and *F. subglutinans* are common. The names “Fusarium ear rot” or “Fusarium kernel rot” refer to these and other *Fusarium* species, but do not include *F. graminearum* and *F. culmorum*, which are usually called “Gibberella ear rot” or “pink ear rot.” Most corn fields in the Midwest have some *Fusarium* ear rot every year, and many healthy-looking kernels are infected with *Fusarium* species.

Symptoms of *Fusarium* ear rots are a white to pink or salmon-colored mold, beginning anywhere on the ear or scattered throughout (Figure 1). Often the decay begins with insect-damaged kernels, but most of the ear can be affected. Infected kernels are often tan or brown colored, or have white streaks (Figure 2). *Fusarium* species also cause corn stalk rots. The fungi survive on corn residue and the residue of other plants, especially grasses. *Fusarium* spores are spread by wind and splashing rain to the silks, which are most susceptible for the first five days after they appear. Infection of kernels also can

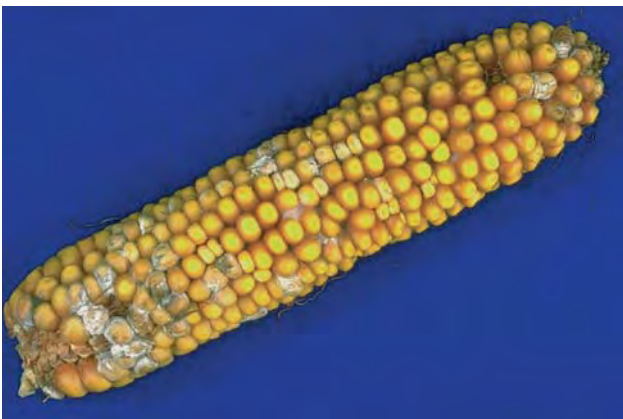


Figure 1. *Fusarium* ear rot, caused by *F. moniliforme*, *F. subglutinans*, or *F. proliferatum*, on scattered kernels.



Figure 2. *Fusarium* ear rot causes brown discoloration and white streaking of kernels.



Figure 3. Insect injuries are often infection sites for *Fusarium* species.

occur through the stalk, but this mode of kernel infection is less common. Insect-damaged kernels are very susceptible, and spores carried on the insects can infect the damaged kernels (Figure 3). *Fusarium* ear rot occurs under a wide range of weather conditions, and insect activity may be the critical factor determining *Fusarium* ear rot severity. *F. moniliforme* has been reported to grow very little when grain moisture content is below 19 percent.

▮ Gibberella ear rot ◀

Gibberella ear rot is caused by the fungus *Gibberella zeae*, also known as *Fusarium graminearum*.

It can be identified most readily by the red or pink color of the mold.



Figure 5. Gibberella ear rot sometimes lacks the pink color.

It almost always begins at the tip of the ear (Figure 4). In some cases, the color is too pale to be seen readily, so the mold appears white (Figure 5). In this case, it may not always be possible to distinguish Gibberella ear rot from *Fusarium* ear rot



Figure 4. Gibberella ear rot causes a pink rot starting at the tip of the ear.

without a microscope. Gibberella ear rot can be very destructive to the ears. This species also causes corn stalk rot and scab of wheat; it survives in corn and small grain residue. The spores of this fungus infect through silks; stalk infections are not believed to lead to Gibberella ear rot. Spores reach the silks by splashing water or they may be carried by insects. Infections may originate at insect wounds rather than at the ear tip (Figure 6). Gibberella ear rot infections occur more commonly when the weather is cool and wet during the first five days after silking. Continued development of the mold also depends on subsequent cool, wet weather. Optimum temperatures for *F. graminearum* (*Gibberella zeae*) are 65–70°F but it can grow at much lower temperatures. Grain that overwinters in the field can have severe Gibberella ear rot and high levels of mycotoxins. The fungus *Gibberella zeae* shares many characteristics with the other *Fusarium*



Figure 6. Insect injuries are sometimes infection sites for *Gibberella zeae*.



Figure 7. Discolored kernels infected with *Fusarium* or *Gibberella*.

species. In storage, *Fusarium* species (including *Gibberella*) appear as white or pink mold, with kernels that appear brown and decayed (Figure 7).

► Fusarium toxins ◀

Fusarium species produce more than 80 different mycotoxins, but only a few are consistently found in corn. Table 1 lists the most common *Fusarium* toxins in corn and the most important species that produce them. *F. graminearum* (*Gibberella zeae*) is the most important toxigenic species in corn in the Midwest, producing deoxynivalenol, zearalenone, and T-2 toxin. *F. moniliforme* and *F. proliferatum* are probably the next most important toxigenic fungi, producing fumonisins and possibly other toxins. It is common for toxic corn samples to contain more than one *Fusarium* toxin. Visible mold indicates a potential for mycotoxin problems, but there can be a poor relationship between visible mold growth and the occurrence of toxins. Therefore, the safety of grain for feeding can not be determined by its appearance.



Figure 8. *Aspergillus* ear rot.

► Aspergillus ear rot and storage mold ◀

Aspergillus flavus and *A. parasiticus* are generally known as storage fungi, but they also can cause ear rots in the field. *Aspergillus* is a gray-green or yellow-green, powdery mold (Figure 8). These fungi also survive in plant residue and soil. *Aspergillus* does not compete well in the field with other fungi and bacteria under moist conditions. In Iowa, *Aspergillus* is much more common in hot, dry years. Under these conditions, high populations of the fungus develop in plant debris and soil; the spores then infect silks or kernels usually through insect wounds. It can grow at temperatures higher than 90°F and grain moisture content as low as 15 percent. Injury by insects or other animals, as well as drought stress, predisposes kernels to infection. These fungi produce the most well-known mycotoxins in corn, aflatoxins. The fungus can be detected in corn because it produces compounds that are fluorescent under black light, but this does not directly detect the presence of aflatoxins. In the Midwest, aflatoxin outbreaks are closely associated with hot, dry weather conditions.

Table 1. The four most commonly detected *Fusarium* toxins in corn in the Midwest and the *Fusarium* species most often reported to produce each toxin.

Toxin:	Produced by:
Deoxynivalenol (vomitoxin, DON)	<i>F. graminearum</i> (<i>Gibberella zeae</i>), <i>F. culmorum</i>
Fumonisin	<i>F. moniliforme</i> , <i>F. proliferatum</i>
Zearalenone	<i>F. graminearum</i> (<i>Gibberella zeae</i>), <i>F. culmorum</i> , <i>F. sporotrichioides</i> , <i>F. equiseti</i>
T-2 toxin	<i>F. sporotrichioides</i> , <i>F. graminearum</i> (<i>Gibberella zeae</i>), <i>F. acuminatum</i> , <i>F. equiseti</i> , <i>F. poae</i>



Figure 9. *Aspergillus flavus* (right) has a yellow-green color compared to *Penicillium* fungi (left).

A. ochraceus can produce another toxin, ochratoxin. In the Midwest, *Fusarium* fungi and toxins are much more common in corn than *Aspergillus* and aflatoxins.

► Penicillium molds ◀

Penicillium oxalicum and several other *Penicillium* species attack corn kernels, primarily in storage. Like *Aspergillus*, this fungus also can cause ear damage in the field. *Penicillium* is a blue-green, powdery mold. *Cladosporium*, *Aspergillus*, and *Penicillium* can be distinguished by their color (Figure 9), and by their spores under a microscope. Some *Penicillium* species

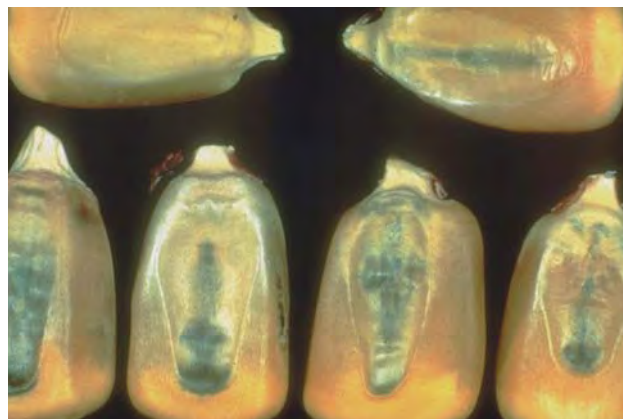


Figure 10. Embryo infections by *Penicillium* can cause “blue eye.”

can grow at grain moisture contents of 16-17 percent. Kernels with “blue eye” have embryos infected with *Penicillium* (Figure 10). Generally, *Penicillium* does not cause mycotoxin problems, but *Penicillium viridicatum* and possibly other species can produce ochratoxin, citrinin, and other toxins.

A common symptom of any storage mold is the presence of “hot spots.” When fungi grow in the bin, they give off heat and moisture to the surrounding grain. This makes the grain more conducive to further mold growth, and the spots grow, becoming warmer, wetter, and more moldy.

••••• Non-toxigenic ear rots and molds •••••

There is a wide variety of fungi that infect corn ears and kernels but do not produce toxins. Although these fungi may not be harmful to livestock, they can cause considerable damage to ears and kernels, causing reductions in yield, quality, and nutritive value.

► Diplodia ear rot ◀

Next to the *Fusarium* and *Gibberella* ear rots, *Diplodia* ear rot, caused by *Diplodia maydis*, is probably the most important. This fungus initially appears as a white mold beginning at the base of the ear (Figure 11). The mold and the kernels eventually turn a grayish brown color and rot the entire ear (Figure 12). The mold may be apparent on the outside of the husk. A very distinguishing characteristic of *Diplodia*

ear rot is the appearance of raised black bumps on the moldy husk or kernels (Figure 13). These are the pycnidia of the fungus, where new spores are produced. *Diplodia* is spread primarily by splashing water. Corn borer damage in the shank can provide an entry wound for the pathogen, but some



Figure 11. *Diplodia* ear rot usually starts at the base of the ear.



Figure 12. *Diplodia maydis* often decays the entire ear.

infections occur early in the season through the whorl and remain undeveloped until after pollination. Infections also occur through silks. Diplodia ear rot occurs most often in fields under reduced tillage where corn follows corn. It is favored by cool, wet weather during grain fill. Rainfall during August,



Figure 14. Mycelium and pycnidia of *Diplodia maydis* on infected kernels.



Figure 15. *Nigrospora* ear and cob rot usually starts at the base of the ear.



Figure 13. Spore-forming structures (pycnidia) of *Diplodia maydis*.

September, and October is correlated with *Diplodia* ear rot incidence. *D. maydis* appears as a white mold in storage, with kernels appearing brown and decayed. It can be identified by the black pycnidia, if present (Figure 14). Growth of this fungus is very slow when grain moisture is below 21 percent.

Fusarium, *Gibberella*, and *Diplodia* also cause stalk rots. They survive in crop residue from year to year. Therefore, continuous corn production favors more severe stalk rot and ear rot. This has been demonstrated for *Diplodia* and *Gibberella*, but may not be as important for the other *Fusarium* species.

▮ *Nigrospora* ear rot ◀

This fungus, *Nigrospora oryzae*, appears as a gray or black mold, often starting at the base of the ear (Figures 15, 16). It produces large black spores, which can be seen with the naked eye or a hand lens. They appear as “peppery” speckles on the surface of



Figure 16. Severe *Nigrospora* ear rot.



Figure 17. *Cladosporium* usually infects damaged kernels.



Figure 18. Dark green or black mold is typical of *Cladosporium*.

the kernels. *Nigrospora* also rots the cob, giving it a brown, tattered appearance at the shank.

Cladosporium ear rot

Cladosporium herbarum and other species often infect kernels damaged by insects, hail, or frost. This fungus appears gray to black or very dark green, and can have a powdery appearance (Figures 17, 18). It also causes black streaks in the kernels. This disease can be fairly common but usually does not cause extensive damage to the ears. In storage, it can be identified by the dark green or black powdery spores and black kernel streaks (Figure 19). Although some species of *Cladosporium* are toxigenic,

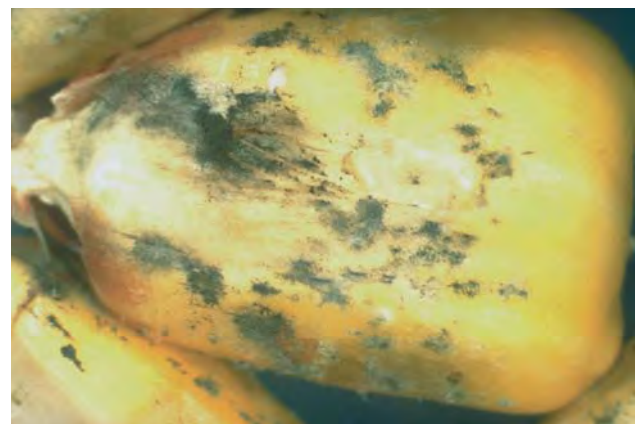


Figure 19. Close-up of *Cladosporium* on single kernel.

the development of these toxins on corn appears to be very rare.

••••• Animal health effects of Fusarium toxins •••••

Tables 2 and 3 list some concentrations of common mycotoxins that are associated with acute, chronic, or productivity effects. **Remember that these are only guidelines and exceptions may occur frequently.** In addition, mycotoxin concentrations vary widely in a grain supply and may change with time. Thus the levels determined in a particular feed are only as good as the sampling techniques and frequency of collections.

Trichothecenes

The trichothecene mycotoxins include over 60 toxins produced by several *Fusarium* species including *F. graminearum*, *F. culmorum*, *F. sporotrichioides*,

and *F. poae*. Best known are T-2 toxin, diacetoxyscirpenol and deoxynivalenol (also known as DON or vomitoxin). DON is by far the most commonly reported. Most animals refuse to consume high concentrations of trichothecenes and never achieve the full range of reported toxic effects. Forced intake of trichothecenes can cause reduced appetite, vomiting, intestinal inflammation, depletion of lymph node activity, low white blood cell counts, bone marrow depression, reduced blood clotting, and impaired immunity to infectious diseases.

Reduced feed intake in swine begins at approximately 1 ppm DON and increases proportionally until complete refusal occurs at greater than 10 ppm,

Table 2. Expected detrimental feed concentrations of common *Fusarium* mycotoxins (ppm = parts per million).

Zearalenone			
Swine	Concentration	Duration	Effect
Prepubertal gilts	1-5 ppm	3-7 days	Hyperestrogenism, prolapse
Sexually mature open gilts	3-10 ppm	Mid-cycle (day 11-14)	Anestrus, pseudopregnancy
Bred sows	15-30 ppm	1st trimester	Early embryonic death, small litters
Juvenile boars	10-50 ppm	Indefinite	Reduced libido, small testicles
Mature boars	200 ppm	Indefinite	No effect
Cattle	Concentration	Duration	Effect
Virgin heifers	12 ppm	Open heifers	Reduced conception
Dairy cows	50 ppm	Open cows	Reduced conception
Poultry	Concentration	Duration	Effect
Broilers & turkey poults	200 ppm	Indefinite	No effect
Vomitoxin (deoxynivalenol, DON)			
Swine	Concentration	Duration	Effect
Feeder pigs	1-3 ppm	1-5 days	Reduced feed intake
Feeder pigs	5-10 ppm	1-5 days	50% reduction in feed intake, vomiting
Feeder pigs	10-40 ppm	1-5 days	Complete feed refusal, vomiting
Sows	3-5 ppm	Gestation, lactation	Lower fetal weights, or no effect
Cattle	Concentration	Duration	Effect
Feeder cattle	10 ppm	Indefinite	No effect
Dairy cows	6 ppm	6 weeks	No effect or slightly reduced feed intake
Dairy cows	12 ppm	10 weeks	No effect on milk production
Poultry	Concentration	Duration	Effect
Broilers and turkey poults	50 ppm	Indefinite	No effect
Fumonisin (FB1 and/or FB2)			
Horses	Concentration	Duration	Effect
All classes and ages	>10 ppm	30 days	Liver damage, leucoencephalomalacia, death
Swine	Concentration	Duration	Effect
	>25 ppm	30 days	Reduced gain and feed efficiency, mild liver damage
	>50 ppm	10 days	Reduced gain and feed efficiency, moderate liver damage
	>100 ppm	5 days	Severe pulmonary edema, death
Cattle and sheep	Concentration	Duration	Effect
	>100 ppm	30 days	Slightly reduced gain, mild liver damage
	>200 ppm	14 days	Reduced feed intake and gain, moderate liver damage
Turkeys	Concentration	Duration	Effect
	>100 ppm	7-21 days	Reduced feed intake, liver damage, diarrhea, rickets, tibial lesions
Chickens	Concentration	Duration	Effect
	>200 ppm	7-21 days	Reduced feed intake, liver damage, diarrhea,

Table 3. Expected detrimental feed concentrations of aflatoxin (ppb = parts per billion).

Animal species	Concentration	Effect
Swine	200 ppb	Slow growth, reduced feed efficiency
	400 ppb	Liver damage and immune suppression
Feeder cattle	400 ppb	Tissue residues
	700 ppb	Mild liver damage, reduced growth and feed efficiency
	1000 ppb	Moderate liver damage, weight loss
	2000 ppb	Severe liver damage, jaundice, death
Dairy cows	20 ppb	Detectable aflatoxin in milk
	1500 ppb	Decreased milk production
Poultry	250 ppb	Turkeys have reduced growth
	210 ppb	No effect on broiler chicks
	420 ppb	Broiler chicks lose weight, moderate liver damage after 3 weeks
Horses	400 ppb	About same response as pigs

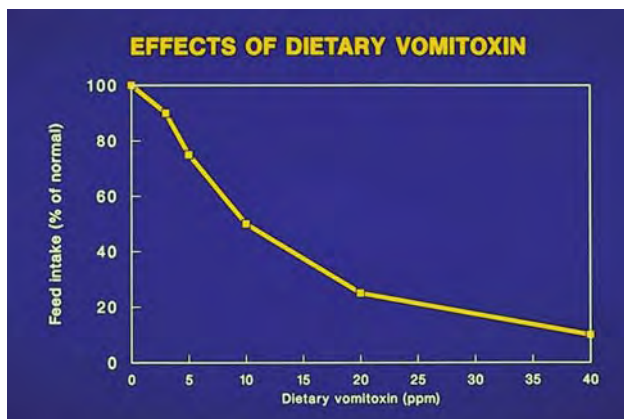


Figure 20. Example of the relationship of feed intake to vomitoxin (DON) concentrations in diet for swine. The relationship can vary considerably.

although the response is highly variable (Figure 20). An association of a specific feed with unpleasant reaction such as nausea or vomiting can result in the learned behavior of taste aversion or avoidance of the feed. The only documented clinical health effects in livestock as a result of DON are from reduced feed intake. Vomiting can be induced with high concentrations.

Beef cattle tolerate DON contaminated feed at approximately 10 ppm in the ration. Limited studies in dairy cows indicate 6 ppm may slightly reduce feed intake in some cows. Although low levels (less than 1 ppm) of DON in feed have been associated in some field cases with reduced milk production, DON itself may not be responsible. More studies are needed, and so far a cause-effect relationship has not been established. Very little DON is excreted in milk.

FDA advisory levels issued in 1993 for DON are as follows:

- 1 ppm for finished wheat, such as flour, bran, and germ.
- 10 ppm for grains and grain byproducts destined for ruminating beef and feedlot cattle older than 4 months and chickens. The total should not exceed 50 percent of the diet of either animal species (5 ppm DON in feed).
- 5 ppm on grain and byproducts destined for swine. The ingredients should not comprise more than 20 percent of the ration (1 ppm DON in feed).

T-2 toxin is a less common but more potent toxin



Figure 21. Effect of T-2 toxin on growth of broiler chickens. The two birds are the same age; the smaller one was fed a diet containing T-2 toxin.

than DON. This toxin causes hemorrhaging, inflammation of the digestive tract, vomiting, diarrhea, decrease in milk production, feed refusal, and reduced weight gain (Figure 21). These symptoms can be fatal in some cases.

▮ Zearalenone

Zearalenone is an estrogen. It is produced under low temperature conditions. A common clinical effect of zearalenone is increased estrogen levels (also known as vulvovaginitis, Figure 22) in young female swine. This includes swelling of the vulva and signs of clinical estrus sometimes accompanied by straining and protrusion of the vagina or rectum. Zearalenone will induce clinical signs of estrus or vulvovaginitis in prepubertal gilts at dietary concentrations as low as 1–5 ppm. With removal from toxic diets, pigs return to normal in 3–7 days.

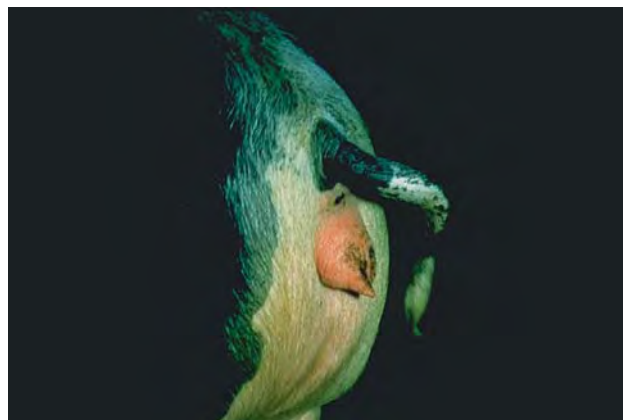


Figure 22. Vulvovaginitis in a gilt, caused by zearalenone.

Zearalenone in mature female swine aids in maintaining the corpus luteum of the ovary causing suppression of estrus as in pregnancy, and a prolonged estrus interval of 40–80 days. Affected sows may appear pregnant after breeding, only to return to estrus weeks later. The prolonged estrus interval is maintained long after zearalenone-containing feed is withdrawn from the diet. Dietary zearalenone as low as 3 ppm has caused this effect in some sows. Litter size may be affected by zearalenone due to early embryonic death before implantation, but only when zearalenone levels are high (greater than 15 ppm). Anestrus, pseudopregnancy, and fewer pigs per litter are consistent observations in sows given zearalenone. Generally, grain contaminated with zearalenone at any level is not recommended for sows or replacement gilts.

Cattle are considerably more resistant to the effects of zearalenone than swine, with dietary levels of 50 ppm zearalenone required to produce adverse effects on fertility in dairy cows. In virgin dairy heifers, conception rates are reduced when dietary zearalenone is greater than 12.5 ppm.

► Fumonisin

In 1989, corn screenings caused an outbreak of severe pulmonary edema (fluid in the lungs) and death in swine. Five to 50 percent of affected herds died and affected pigs were lethargic with difficult breathing and cyanotic (blue colored) skin indicating



Figure 23. Depressed, lethargic pig with cyanosis (dark ears and nose) due to fumonisin-induced pulmonary damage.

severe lack of oxygen (Figure 23). Deaths occurred 4–10 days after first feeding of corn screenings contaminated with a newly discovered mycotoxin known as fumonisin B₁. *Fusarium moniliforme* and *Fusarium proliferatum* were isolated from field cases, and cultures of these molds caused acute porcine pulmonary edema (PPE) when fed for 5 days. Survivors of the lung disease, or pigs that consumed lower levels of fumonisin B₁, developed moderate liver damage and had reduced weight gain. Dietary fumonisins at 100 ppm or greater are likely to cause pulmonary edema while feed concentrations above 50 ppm may cause liver damage and reduced rate of gain. Although pregnant sows affected by PPE sometimes aborted their pigs, the abortions appear due to lack of oxygen to the fetuses, since fumonisins fed to sows did not cause abortion as long as the sows were healthy.

During the same time period, horses throughout the United States were affected with a severe to fatal brain disease known as leucoencephalomalacia or “leuco” in which the white matter of the brain softened and liquified. Affected horses were uncoordinated and depressed and often could not swallow food. Fatality rate of the leuco condition was near 100 percent. This disease has been known for many years as “moldy corn poisoning” but only since 1988 has the cause been identified as fumonisin mycotoxins. As little as 10 ppm fumonisin in the diet of horses for 30 days or more can cause leuco. Feed horses only high quality corn low in fumonisins.

Cattle and sheep are comparatively resistant to fumonisins, with mild liver damage occurring at feed levels above 100 ppm and moderate feed refusal and reduced growth at 200 ppm or more. Poultry are more resistant to fumonisins than other common livestock, and signs of fumonisin poisoning do not occur until feed levels are above 100 ppm for turkey poults and 200 ppm for broiler chicks. Effects include liver damage, diarrhea, and rickets or related tibial (leg) lesions. Field outbreaks of fumonisin poisoning in poultry have not yet been reported.

The American Association of Veterinary Laboratory Diagnosticians (AAVLD) has recommended guidelines for fumonisin concentrations in livestock feed. Recommendations are that livestock feed contain fumonisin B₁ below the following concentrations:

Horse and other equine species	5 ppm
Swine	10 ppm
Beef cattle	50 ppm
Poultry	50 ppm

▮ Aflatoxin ◀

Aflatoxin refers to a closely related group of metabolites of *Aspergillus flavus* and *A. parasiticus*. Those toxins generally recognized are B₁, B₂, G₁, G₂, and M₁. The M refers to “milk” as the original source of M toxin. The most abundant member of the group present under natural contamination is Aflatoxin B₁, a potent carcinogen.

The main target organ for aflatoxin is the liver. Aflatoxin suppresses both nucleic acid and protein synthesis. This impairment of protein synthesis and related ability to mobilize fats causes growth suppression and lesions in the liver of affected animals. Effects following exposure to high dosages of aflatoxin reflect liver cell damage and hemorrhage. Affected animals are depressed and have reduced feed intake. Jaundice and hemorrhages become evident during this same time period. The most likely effects of aflatoxin in swine are reduced gain and feed efficiency, subtle liver lesions, and possible increases in incidence or severity of infectious diseases. Constant exposure to aflatoxin for more than 7–10 days can result in hemorrhages in a variety of organs. In acute to subacute poisoning, liver weight may be increased but is reduced in chronic cases where the liver becomes shrunken and filled with scar tissue.

Aflatoxin is not considered a serious threat to fertility and is not likely to cause abortions at concentrations that cause moderate disease. However, piglets nursing sows fed aflatoxin gain weight poorly.

Acute to subacute toxicosis from aflatoxin in chickens and turkeys results in severe liver damage.

Clinical signs observed from acute aflatoxicosis include hemorrhage, jaundice, anemia, depression, ruffled feathers, and poor appetite.

Horses and ponies are susceptible to aflatoxin. Clinical signs of acute poisoning include anorexia, fever, rapid heart rate, ataxia, colic, icterus, convulsions, bloody feces, and abdominal straining.

Cattle, sheep, and other ruminants appear less susceptible to mycotoxins than other animals. Among cattle, calves appear to be more susceptible than mature animals. Generally aflatoxin concentrations of 1–2 ppm in mature cattle for short periods of time result in reduced gain and decreased milk production. As little as 1 ppm aflatoxin has caused liver damage, reduced gain, and death in feedlot steers.

An “action level” is a regulatory term meaning the concentration at which regulatory agencies will take action. Aflatoxin is regulated because it is a known carcinogen. This level relates to regulatory action controlling interstate movement of grain. Action levels may also apply to animal products consumed by the public. Action levels in such products are generally lower than for grains. Recent exemptions to the long standing action level of 20 ppb have been suggested by the FDA. They are as follows:

Human food and milk	<0.5 ppb
Corn of unknown destination	<20ppb
Young animals	<20ppb
Dairy cattle	<20ppb
Breeding cattle, swine, and mature poultry	<100ppb
Finishing swine	<200ppb
Finishing cattle	<300ppb

These guidelines are based on maintaining performance and avoiding lesions or disease related to aflatoxin, except for dairy cattle where prevention of carcinogenic aflatoxin residues in milk is the concern.

Detrimental effects from aflatoxin in livestock and poultry occur at concentrations approximately twice those listed as FDA guidelines. This could vary if animals are stressed, malnourished, selenium deficient, or otherwise stressed.

► Ochratoxin ◀

Ochratoxin causes increased water consumption and excessive urination by affecting renal tubules. This condition is not commonly diagnosed in the

United States but occurs as a common and serious economic problem in northern Europe. It is an example of a mycotoxin which may be a potential or real problem in this country.

• • • • • Detection of mycotoxins • • • • •

Safety of feed can not be determined by the presence or absence of mold. Mold counts, cultures, spore counts, and other methods of determining mold infestation do not prove that mycotoxins are present. They should be used only to show that conditions for mycotoxin production exist. Whether a feed contains sufficient mycotoxin to affect animal health can be determined only by either isolating or identifying the toxin accompanied by determination of the amount present, or by appropriate well-controlled feeding studies. No one can guarantee the absolute safety of a molded feed, no matter how many chemical tests are run upon it.

► Sampling ◀

Determining of toxin content in feed depends on first obtaining an adequate representative sample. Mycotoxin contamination may vary widely within a storage facility or within a field of standing grain. Thus a single sample collected from one location within a storage unit does not give useful information about the entire bin. Sampling is often a major source of error in determining actual levels of mycotoxins in grain.

Sampling procedures for mycotoxin analysis have been developed by the U. S. Department of Agriculture. Recommendations include sampling after particle reduction such as grinding. In addition, samples are best taken after blending of grain such as in hauling or mixing for feed. The recommended procedure is to sample periodically from a moving stream, combining these samples to obtain a composite minimum sample of 10 pounds. An alternative to this is to use multiple probe sampling

through a storage unit (5 perimeter samples and one center sample for each 6 feet of bin height). When field sampling is done, a minimum of 10 to 30 locations should be selected for sampling within each field. Storage and transport for laboratory evaluation is important. Samples should be dried to 12 to 14 percent moisture in order to prevent development of mold and potential mycotoxin contamination during transport or storage. If toxin analysis is desired, drying may be at 80 to 90°C (176 to 194°F) since toxins of interest will resist degradation by heat. If mold culture is important, drying temperature should not exceed 60°C (140°F). High moisture samples are best frozen and delivered to the laboratory in the frozen state. Dried samples will maintain their quality best if shipped in cloth or paper containers.

► Analysis ◀

A variety of analytical methods are available for mycotoxins. The quality and usefulness of results often parallel the investment made. Direct fluorescence using the ultraviolet lamp (black light) is useful only for tentative identification of *Aspergillus*. Other procedures such as thin layer chromatography, mini columns, gas chromatography, or mass spectroscopy are probably necessary if quantitation and confirmation of the mycotoxin is needed. Special problems are presented by chemical analysis in mixed feeds. Additives in other feed ingredients may mask toxin which is present or may cause false positives to occur.

Samples for mycotoxin analysis can be sent to Iowa State University (ISU) or a private laboratory. The ISU Veterinary Diagnostic Laboratory will only analyze

samples submitted through a veterinarian or through the ISU Plant Disease Clinic at the following address:

Iowa State University
Plant Disease Clinic
Dept. of Plant Pathology
323 Bessey Hall
Ames, IA 50011

Currently several commercial test kits using fluorescence, radioimmunoassay, or ELISA techniques are available to test for mycotoxins. These kits provide rapid, convenient analysis on-site, but they are not as precise as the chromatography methods.

• • • • • *A checklist for preventing molds and toxins* • • • • •

► In the field ◀

- Crop rotation or tillage can reduce the risk of some ear rots, such as *Diplodia* and *Gibberella*. These practices have little effect on other ear rots.
- Control of 2nd generation European corn borers and other insect pests of corn ears can greatly reduce infection by *Fusarium* and *Aspergillus*.
- Some hybrids are more resistant to ear rots than others, but overall, resistance to ear rots is not widely available. Hybrids with ears that do not remain upright experience less ear rot. Widely grown hybrids are not excessively susceptible if they are grown within their adapted range.
- As with any crop pest, early detection through scouting can prevent serious losses and avoid crises. If extensive ear rot development is observed (10 percent or more of the ears with more than 10-20 percent mold), a field should be harvested as soon as moisture content reaches a level that can be harvested. Even if some drying costs are incurred, this is less expensive than loss of a crop due to mycotoxins. Decisions on handling moldy grain should be made before it is harvested. After harvest, spoilage can occur quickly if delays result from indecision. Very moldy grain is a poor candidate for storage. If toxin analyses indicate safe levels, selling the grain or feeding to less sensitive livestock species may be a better alternative than storage.
- Kernel damage that occurs during harvest can lead to subsequent mold development. Properly adjusted equipment can reduce the amount of damage. Poor quality, damaged, or low test-weight corn is more prone to attack by storage fungi.

- Cleaning grain before drying will remove much of the particles and damaged kernels. These are often the moldiest and most toxic component of harvested grain.

► After harvest ◀

- Corn must be dry to prevent mold development. Clean corn can be kept at 16-17 percent moisture content during the winter. Moldy corn should be dried immediately to 15 percent moisture or less. Holding moist grain for even a short time can allow significant mold and mycotoxin development. For long term storage, all corn should be dried to 13-14 percent. Fast drying will minimize mold growth, but may have certain disadvantages.
- Corn must be stored cool. Grain must be cooled after drying, and maintained at 35-40°F for the duration of storage. Aeration can be used for temperature control. Aside from moisture content, temperature is the most important factor in preventing mold development. Freezing the grain further reduces the risk of molds, but leads to other problems, so this is not recommended. Keep in mind that even clean-looking grain will develop mold problems if not dried and stored properly.
- Bins should be thoroughly cleaned before the new crop is stored.
- Storage insects cause kernel damage and carry fungi, so controlling them will help reduce the risk of molds and mycotoxins.
- Grain in storage should be checked every two weeks (more frequently if the quality is suspect) for temperature, crusting, hot spots, moisture, and mold. If any of these conditions are detected,

steps should be taken immediately to reduce the temperature, aerate the bin, break up hot spots, or remove spoiled grain.

- Antifungal agents can be applied to grain to reduce mold growth in storage. These products, such as propionic acid, do not kill the mold already present or reduce toxins already formed. **Do not use antifungal agents on stored grain unless you are certain the grain can be marketed after treatment.**
- Hydrated sodium calcium aluminosilicate (HSCAS) (Novasil) has reduced the effects of aflatoxin

when fed to swine, cattle, or poultry. HSCAS at 10 lb./ton provides substantial protection against dietary aflatoxin.

The key to preventing ear rot and storage mold problems is finding them early, in the field and in the bin. For more details on proper grain handling to prevent molds, mycotoxins, and other problems, see ISU extension publications AED-20, *Managing Dry Grain in Storage*, and Pm-589, *High-Moisture Corn Storage*, and Midwest Plan Service publication MWPS-13, *Grain Drying, Handling, and Storage Handbook*.

Utilization of affected grain

A number of factors influence the decision to use contaminated grain. Since the effects of mycotoxins are often subtle, long term, and not amenable to treatment, prevention is of great importance. **Ideally, moldy or mycotoxin-contaminated grain should not be fed to livestock.** Mycotoxins may occur for which there are no documented effects nor means of identification. Heavily molded feedstuffs also may lack normal energy and vitamin levels. If a decision is made to feed moldy grain to livestock, some considerations are important, especially livestock species. Ruminant species are much less sensitive to mycotoxins. While not entirely predictive, the type of mold infestation and extent of that infestation may be important factors to consider. Dilution of certain kinds of molded grain with good-quality feed may aid in reducing the extent of contamination. However, any blending carries the inherent risk that good feed will be contaminated and further mold growth and/or mycotoxin formation may occur. Wet grains should not be blended with dry

grains unless the mixture is quickly dried. The new mixture should be tested for toxins. Regulatory agencies do not recognize blending as an acceptable means for detoxification. Generally mature animals are less susceptible to mycotoxins. This knowledge is helpful in using limited amounts of contaminated grain. Usually the longer the duration of feeding, the higher the likelihood of creating a problem.

Management of mycotoxicoses should include removal of all known feeds containing toxins. Increased levels of high-quality protein and supplementation with selenium as well as vitamins (A, D, E, K, and B complex) should be included to counteract the effects of mycotoxins in protein and vitamin utilization. Mycotoxin exposed animals sometimes may have compromised immune systems; thus clinical signs of infectious disease should be aggressively treated with appropriate antimicrobial therapy and passive immunization if possible.

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