Mycotoxins in Swine Diets

Mycotoxins are toxic compounds produced by mold growth in feed ingredients. Although not all molds produce toxins, the most significant mycotoxins affecting swine are aflatoxin, vomitoxin, zearalenone, fumonisin, and ochratoxin. These mycotoxins are produced by molds that belong to the genera *Aspergillus*, *Fusarium*, and *Penicillium*. The occurrence of mycotoxins in swine diets is discussed in this fact sheet.

Mycotoxin formation

Mycotoxins are only produced by certain molds and under certain conditions (**Table 1**). Thus, the presence of molds in feedstuffs or feeds does not automatically imply presence of mycotoxins. The major factors influencing mold growth and mycotoxin formation are moisture and temperature. Mold growth requires readily available starch from grains, moisture, air, and appropriate temperatures, often 54 to 77°F. Mycotoxin formation can occur under such conditions, but typically the presence of stressors, such as drought, high environmental temperatures, excessive water, nutrient deficiency, insect damage, and harvest damage, are also necessary to predispose the formation of mycotoxins (Osweiler and Ensley, 2012; Rodrigues and Naehrer, 2012).

Mycotoxin-producing molds are classified into two categories: field and storage molds (Osweiler and Ensley, 2012). Field molds grow in grains before harvest and require high relative humidity above 70% and typically grain moisture above 22% for growth. The most concerning field molds are the *Fusarium* species, which produce vomitoxin, zearalenone, and fumonisin. Storage molds grow in grains after harvest and during storage of grains and feeds. These molds do not require high humidity and even grow in grains with 12 to 18% moisture. Storage molds include *Aspergillus* and *Penicillium* species, which produce aflatoxin and ochratoxin. However, under certain conditions, storage molds grow in grains even prior to harvest and field molds continue to grow during storage. This is often the case of *Aspergillus flavus*, a mold that produces aflatoxin. Moreover, grains and feeds may contain more than one mold and have a number of mycotoxins present at the same time.

Mycotoxin contamination

Mycotoxin contamination occurs worldwide. Certain regions are predisposed to have higher risk of mycotoxin contamination. For example, temperate regions tend to have higher field mold prevalence (*Fusarium* species), whereas tropical and subtropical regions have higher storage mold prevalence (*Aspergillus* and *Penicillium* species). When conditions are favorable for field molds, grains from a geographic location are widely affected by mycotoxins. In contrast, when conditions are favorable for storage molds, grains are not evenly affected by mycotoxins and distribution is more diverse both across and within storage bins (Jacela et al., 2010).

Mycotoxin contamination most often occurs in grains, such as corn, sorghum, wheat, and barley. Corn is the most extensively and highly contaminated grain (Rodrigues and Naehrer, 2012; Hendel et al., 2017). In addition, mycotoxins are often concentrated in co-products from grains such as corn distillers dried grains with solubles (DDGS) (Rodrigues and Naehrer, 2012). The fermentation process to produce DDGS results in removal of most of the starch in corn and, if corn is contaminated, mycotoxins are unaffected by fermentation and are concentrated by as much as three times the concentration of mycotoxins in the corn source (Jacela et al., 2010).

Types of mycotoxins

Swine are particularly susceptible to mycotoxicosis. Mycotoxicosis is the intoxication that results from the consumption of grains or feeds contaminated with mycotoxins. The degree of mycotoxicosis depends on mycotoxin type and concentration present in the feed and the category of swine consuming the diet. **Table 2** presents the effects of mycotoxicosis in each category of swine according to mycotoxin concentration in the feed. In addition, the Food and Drug Administration determines regulatory limits of aflatoxins to commercialize feed ingredients and feeds for swine to 20 ppb for grower pigs, 200 ppb for finisher pigs over 100 lb BW, and 100 ppb for breeders.

Mycotoxicosis affects many body systems with a wide variety of signs, lesions, and impaired performance. Typically, young pigs and sows are more susceptible and

the effects of mycotoxicosis are more evident (Osweiler and Ensley, 2012). Moreover, contamination with more than one mycotoxin is frequent and the combination of mycotoxins often have additive effects (Vila-Donat et al., 2018). However, while there is data available on the effects of a single mycotoxin contamination on swine performance, little is known about the effects of multiple mycotoxin contamination.

Aflatoxin

Aflatoxins are produced by molds of *Aspergillus* species before harvest and in storage. Aflatoxin B_1 is the most abundant and toxic aflatoxin and is often produced by *Aspergillus flavus*. Aflatoxins affect liver function and cause immunosuppression (Osweiler and Ensley, 2012). Acute aflatoxicosis is uncommon in swine but causes severe liver lesions and signs are a consequence of liver disfunction, such as hemorrhages, jaundice, and sudden death (Osweiler and Ensley, 2012). Aflatoxin at lower doses is cumulative. Thus, chronic aflatoxicosis is more common in swine, as a result of ingestion of lower amounts of aflatoxin for a prolonged period of time and is expressed as lower feed intake and growth rate (Devreese et al., 2013). Also, the occurrence of secondary diseases can increase as well as response to vaccination can decrease because of immunosuppression (Pierron et al., 2016). Nursery pigs are more susceptible to aflatoxicosis than grower-finisher pigs or sows. However, suckling piglets are also considered susceptible to aflatoxicosis because aflatoxin passes through milk when sows in lactation consume contaminated feed.

Vomitoxin

Vomitoxin is the term for deoxynivalenol (DON), a mycotoxin produced by *Fusarium graminearum* before harvest. Vomitoxin is the most common contaminant of corn, wheat, and DDGS in North America and Europe (Rodrigues and Naehrer, 2012; Hendel et al., 2017) and swine is the most sensitive species. Vomitoxin interferes with protein synthesis, modulation of immunity, and activity of neurotransmitters in the brain (Osweiler and Ensley, 2012). Despite what the name suggests, vomitoxin rarely induces vomiting in swine. Acute toxicity is uncommon, but in that case vomit, diarrhea, severe digestive lesions, and sudden death occur (Young et al., 1983). Chronic vomitoxin toxicity is more common and of practical importance. In most cases, a sharp decrease in feed intake is evident and, consequently, a reduction in growth rate upon first exposure (Frobose et al., 2015). The impact on feed intake is dose-dependent, with an estimation of 4% decrease in feed intake for every additional ppm of vomitoxin above the dietary concentration of 1.5 ppm (Frobose et al., 2015).

Zearalenone

Zearalenone is produced by *Fusarium graminearum* generally before harvest. Zearalenone is similar in structure and mimics the effects of the hormone estrogen (Zinedine et al., 2007). Thus, the primary effects of zearalenone are in the reproductive tract of swine (Osweiler and Ensley, 2012). In gilts, a characteristic of zearalenone is swelling and redness of the vulva. Rectal and vaginal prolapses often occur. In sows, zearalenone induces modification in heat behavior with either prolongation of standing heat or no manifestation of standing heat. In bred sows, false pregnancy and early embryo loss also occur. During lactation, zearalenone passes through milk and induces vulvar swelling and redness in newborn suckling gilts (Hennig-Pauka et al., 2018). In boars, zearalenone suppresses testosterone levels, sperm production, and libido, particularly in young boars (Benzoni et al., 2008). The normal reproductive performance of swine typically resumes after the removal of zearalenone contamination from the diet.

Fumonisin

Fumonisins are produced by *Fusarium* species before harvest. Fumonisin B_1 is the most abundant fumonisin and is more likely produced by *Fusarium verticillioides*. Fumonisins interfere with cell function and signaling in many tissues, but mainly the lungs, heart, and liver (Haschek et al., 2001). Fumonisins also cause immunosuppression (Pierron et al., 2016). Acute toxicity causes a condition called porcine pulmonary edema, which is characteristic of fumonisin intoxication and causes heart failure and fluid accumulation in the lungs (Haschek et al., 2001; Osweiler and Ensley, 2012). Pigs with acute toxicity have severe respiratory signs, with labored and openmouthed breathing, cyanosis, and death. Chronic toxicity develops as a result of ingestion of smaller amounts of fumonisins for a prolonged period of time. Pigs with chronic toxicity have lower feed intake and lower growth rate, but may also have greater susceptibility to secondary diseases and lower response to vaccination because of suppression of the immune system (Pierron et al., 2016). Fumonisins toxicity to the liver is common and is both time- and dose-dependent (Haschek et al., 2001).

Ochratoxin

Ochratoxin is mainly produced by *Aspergillus ochraceus*, *Penicillium verrucosum*, and *Penicillium viridicatum* during storage. Ochratoxin A is toxic for kidneys and liver (Osweiler and Ensley, 2012). In most of the cases of ochratoxin A toxicity, pigs have low growth rate and poor feed efficiency due to impaired kidney and liver functions. But feed intake is often unaffected (Malagutti et al., 2005). In some cases, the only effect of ochratoxin A toxicity is found at slaughter by the appearance of pale, firm, enlarged kidneys (Stoev et al., 2002). Ochratoxin A contamination is also a concern for human health because pork and pork-derived products may contain ochratoxin residues with carcinogenic potential (Malagutti et al., 2005).

Mycotoxin analysis

Sampling grain or feed for mycotoxins analysis is critical for mycotoxin detection. Mycotoxins are not evenly distributed in grains or feeds. Rather, mycotoxins are often found at high concentrations in 'hot spots', but at the same time may not be found at detectable amounts in other locations. Thus, the ideal sample should be representative of all of the grain or feed. Importantly, the presence or absence of visible mold growth is not a reliable indicator of mycotoxin contamination and should not be used as a sampling criterion (Carlson and Ensley, 2003b).

For bulk grains or feeds, samples should be collected from at least 10 evenly-spaced locations in the bulk carrier to be representative of the entire load of grains or feeds. For sampling during loading or unloading of bulk grains or feeds, samples should be collected at least 10 times at regular intervals. For sampling from bags of grains or feeds, at least 10 bags should be collected from the lot, with random selection of bags at varying locations in the lot. For sampling from feeders, at least 6 feeders should be collected with a probe or 9 feeders by hand grabbing. In either sampling procedure, sample size should be at least 1 lb and preferentially 2 lb (AAFCO, 2017). Samples should be combined in a composite sample.

Samples should be sent for analysis in paper bags rather than plastic bags to prevent condensation of moisture and further proliferation of mold growth (AAFCO, 2017).

Suggested laboratories performing mycotoxin analyses of complete feeds and feed ingredients are North Dakota State University Veterinary Diagnostic Laboratory (www.vdl.ndsu.edu) and Romer Labs, Inc. (www.romerlabs.com).

Management of mycotoxins in swine diets

In the occasion of mycotoxin contamination of grains or feeds, some strategies for mitigation of mycotoxins in swine diets are available (Dänicke, 2002). Importantly, strategic feeding should be adopted in any instance of mycotoxin contamination by preferentially feeding finishing pigs instead of nursery pigs and sows, which are typically more susceptible to mycotoxins.

At low mycotoxin concentration, grains are often used as is in swine diets as long as mycotoxin concentration in the diet is below the levels affecting health and performance (**Table 2**). At high mycotoxin concentration, different strategies can be adopted. Grains can be fed to species less sensitive to mycotoxins, such as cattle, or grains can be blended with clean grains to reduce mycotoxin concentration by dilution (Carlson and Ensley, 2003a). However, both strategies require using clean grains, which may be a challenge when mycotoxin occurrence is widespread. Furthermore, the Food and Drug Administration does not permit aflatoxin-contaminated grains to be blended for commercialization. Alternatively, grains can be screened and cleaned to remove broken kernels and reduce mycotoxin contamination in grains (Yoder et al., 2017). Misshapen and broken kernel are associated with higher mycotoxin concentrations.

The inclusion of mold inhibitors and mycotoxin binders in the feed can be used as a detoxification strategy. Mold inhibitors are used to control mold contamination and prevent mold growth, whereas mycotoxin binders or adsorbents are substances that bind to mycotoxins and prevent absorption through the gut. Mycotoxin binders are not effective against all mycotoxins and must be rather targeted to a specific mycotoxin.

Table 1. Sources and conditions for mycotoxin formation

Mycotoxin	Mold source	Grains affected	Optimal temperature	Optimal humidity	Favorable conditions
Aflatoxins (B_1, B_2, G_1, G_2)	*Aspergillus flavus, Aspergillus parasiticus*	Corn, sorghum, cotton seed, peanuts	75 to 95°F	80 to 85% relative humidity 17% moisture content	Grain damage, constant high temperature and humidity
Vomitoxin (deoxynivalenol, DON)	*Fusarium graminearum*	Corn, wheat, barley, sorghum, rye, others	79 to 82°F	88% relative humidity 22% moisture content	Alternating warm and cool temperatures during growing season, high humidity
Zearalenone	*Fusarium graminearum*	Corn, wheat, barley, sorghum	45 to 70°F	24% moisture content	Alternating warm and cool temperatures during growing season
Fumonisins (B_1, B_2, B_3)	*Fusarium verticillioides*	Corn	Likely < 77°F	Likely > 20% moisture content	Drought during growing season followed by cool, wet conditions
Ochratoxin A	*Aspergillus ochraceus*, *Penicillium verrucosum*, *Penicillium viridicatum*	Corn, wheat, barley, rye	54 to 77°F	85% relative humidity 19 to 22% moisture content	Low temperatures

Adapted from Osweiler and Ensley (2012).

Table 2. Effects of mycotoxicosis according to category of swine and mycotoxin level in the diet

Mycotoxin	Category of swine	Dietary level	Effects
Aflatoxins (B_1, B_2, G_1, G_2)[1]	Grower-finisher	< 100 ppb	No signs
		200 to 800 ppb	Low feed intake, low growth rate, immunosuppression
		800 to > 2000 ppb	Severe liver disfunction, hemorrhages, jaundice, and sudden death
	Breeders	400 to 800 ppb	No signs on breeders, slow-growing suckling pigs due to aflatoxin in milk
Vomitoxin (deoxynivalenol, DON)	Grower-finisher	< 1 ppm	No signs
		2 to 8 ppm	Sharp decrease in feed intake, low growth rate
		10 ppm	Complete feed refusal, vomit, diarrhea, severe digestive lesions, sudden death
Zearalenone	Gilts and sows	1 to 3 ppm	Vulvar swelling and redness, prolapses of rectum and vagina
		3 to 10 ppm	Anestrus, false pregnancy
		> 30 ppm	Early embryo loss
	Boars	> 40 ppm	Low libido
Fumonisins (B_1, B_2, B_3)	All swine	< 20 ppm	No signs
		50 to 100 ppm	Low feed intake, low growth rate, immunosuppression
		> 100 ppm	Severe lung lesions, labored breathing, cyanosis, and death
Ochratoxin A	Grower-finisher	200 ppb	Low growth rate, poor feed efficiency, kidney lesions at slaughter
		> 1000 ppm	Severe kidney disfunction

Adapted from Osweiler and Ensley (2012).

[1]Food and Drug Administration regulatory limits of aflatoxins to commercialize feed ingredients and feeds for swine: 20 ppb for grower pigs, 200 ppb for finisher pigs over 100 lb BW, and 100 ppb for breeders.

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