# Feeding Mycotoxin Contaminated Grain

Mycotoxins are chemicals (secondary metabolites) produced by moulds or fungi infecting grains. There are over 400 known mycotoxins; however only a small number of these probably affect pig performance on a regular basis. It is important to note that the presence of the mould or fungi does not guarantee the presence of mycotoxins; conversely, mycotoxins can be present in a sample with no obvious mould contamination.

The mycotoxins of major concern in Canada are listed in Table 1. Several factors contribute to the production of mycotoxins in grain, including humidity and temperature during the growing and harvest periods, oxygen availability during growth, harvest, transport or storage and insect or bird damage. Multiple mycotoxins may be present at the same time and mycotoxins may be "masked". These are mycotoxins bound to another molecule which may make them undetectable by routine assays. They will however, break down in the gut, and cause problems.

Pigs are more susceptible to the effects of most mycotoxins than other species, especially ruminants. The age of the animal and production status are important considerations. Table 2 outlines the mycotoxins of major concern, and their impact on animal performance. In order to determine optimal feeding strategies, it is critical to know which mycotoxins are present and the approximate concentration. Many commercial laboratories can analyze for the common mycotoxins.

The difficulty is obtaining a sample that is representative of the entire lot. When sampling grains or feeds, subsamples from 12-20 locations should be collected and mixed thoroughly (Whitlow et al., 2014). Once a sample is collected, it is also important to store it in a dry, cool area to impede further mycotoxin development before the analysis. Mycotoxins are often distributed unevenly throughout the load, and very small quantities can cause problems (1 part per million (ppm) is equal to 1 contaminated grain in 1 million non-contaminated grains). The more subsamples collected, the better the likelihood of obtaining a laboratory analysis which really represents what is in the feed.

The CFIA has regulatory guidelines for the feeding of mycotoxins to livestock. This document reminds us that mycotoxin contamination is typically higher in the lighter fractions (grain dust, screenings, shrivelled kernels, etc.), and that while removing these fractions from the parent stock may help to reduce overall Mycotoxins, which are produced from moulds, can contaminate all grains and grain-by-products commonly fed to swine in Western Canada. Personnel working with grains should avoid inhaling the dust and wear a mask. Dilution is (only partially) the solution.

**Table 1. Legislated maximums, regulatory guidelines and recommended maximums for different mycotoxins into swine diets (adapted from Charmley and Trenholm, 2012)***

| Low | Target | High |
|---|---|---|
| Mycotoxin | Commodity | Levels |
| Deoxynivalenol1 | Diets for swine | 1 ppm |
| Aflatoxins2 | Animal feeding stuffs | 20 ppb |
| T-2 toxin3 | Swine diets | < 1 ppm |
| Zearalenone3 | Gilt diets | < 1-3 ppm |
| | Swine diets | < 0.25-5 ppm |
| Ochratoxin A3 | Swine diets (kidney damage) | 0.2 ppm |
| | Swine diets (reduced weight gain) | 2 ppm |
| Ergot Alkaloids3 | Swine diets | 4-6 ppm |
| Fumonisins3 | Swine diets | 10 ppm |

* ppm is parts per million (mg/kg) and ppb is parts per billion.
1 Regulatory guidelines (Worldwide regulations for mycotoxins. FAO Food and Nutrition Paper 64, 1997)
2 Legislated maximum tolerated level (Worldwide regulations for mycotoxins. FAO Food and Nutrition Paper 64, 1997)
3 Recommended tolerance levels in Canada and the United States

**Table 2. Major effects of mycotoxins on swine performance**

| Mycotoxin | Primary Effect | Stage Affected | Clinical Signs |
|---|---|---|---|
| Deoxynivalenol (DON, vomitoxin) | Affects serotonin receptors and cytokine production | All stages (younger pigs may be more susceptible) | Reduced ADFI and ADG[1,2]<br>Vomiting[2]<br>Diarrhea (soft or watery feces)[3]<br>Reduced immune function[3]<br>Mild changes to kidney, thyroid, blood[4] |
| Aflatoxins | Mutagenic and carcinogenic | All stages | Reduced ADFI and ADG[5]<br>Reduced milk production[5]<br>Lethargy[6]<br>Ataxia (lack of coordination)[6]<br>Rough hair coat[6]<br>Hemorrhage[6]<br>Fatty liver[6] |
| Zearalenone | Estrogenic | Pre-pubertal gilts, sows and pre-pubertal boars | Swelling and reddening of the vulva[7]<br>Vaginal and/or rectal prolapse[7]<br>Anestrus[8]<br>Reduced litter size[8]<br>Fetal resorption8<br>Implantation failure8<br>Decreased libido and testosterone[9]<br>Feminization[9] |
| Ochratoxin A | Disrupts phenylalanine (an amino acid) metabolism | All stages | Kidney damage[10]<br>Decreased ADFI and ADG[11]<br>Immunosuppression, increased risk of infection[12] |
| Fumonisins | Disrupts lipid metabolism | All stages, especially young pigs | Pulmonary edema[3]<br>Reduced immunity[3]<br>Decreased ADFI and ADG[13]<br>Shortness of breath[3]<br>Weakness[3]<br>Cyanosis (blue/purple colour of skin/membranes)[3] |
| T-2 and HT-2 Toxins | Inhibits protein synthesis | All stages | Unthriftiness[6]<br>Low ADFI and ADG[6]<br>Reproductive failure[6]<br>Gastric upset (diarrhea)[6]<br>Cellular necrosis[6]<br>Immunosuppression [6] |
| Ergot Alkaloids | Neurological | All stages, especially the reproductive herd | Lameness[14]<br>Gangrene[14]<br>Decreased ADG[14]<br>Abortion[14]<br>Agalactia (absence of milk production)[14]<br>Ataxia[14] |

1 Decreased ADFI and feed refusals have been shown at levels as low as 0.5-1 ppm (Smith et al., 2005),
2 > 2-5 ppm is for decreased ADFI and ADG, vomiting and complete feed refusal at > 20 ppm (Haschek et al., 2002),
3 Pierce and Diaz, 2014,
4 JECFA, 2001,
5 Nibbelink, 1986,
6 Whitlow et al., 2014,
7 Friend et al., 1990,
8 Smith et al., 2005,
9 Osweiller, 1986,
10 Kidney damage occurs at levels as low as 0.5 ppm (Lippold et al., 1992),
11 Performance is affected at levels of 2 ppm or greater (Lippold et al., 1992; Stoev et al., 2000),
12 Can occur when levels > 2 ppm are fed for longer periods of time (Harvey et al., 1992),
13 ADG reduced by 11% when 10 ppm fumonisin B1 was fed to starter pigs for 8 weeks (Rotter et al., 1996),
14 Strickland et al., 2011

*(Heart Health ... cont'd from page 9)*
"Eventually, we would look in to bringing in human health experts [such as] dietitians, and use this information to develop those protocols and resources," says Columbus. "And those programs are already out there. We know that good food is essential, but we know that it can be a problem even in regular birth-weight infants. And so how do we promote that even more"?

The study's results will also give Canadian swine producers insight about whether young piglets that are less competitive at nursing and receive less nutrition from their mothers will be able to respond to nutritional therapies later in life and recover — maturing to fully-grown healthy animals.

As Columbus explains, the analysis could show whether these pigs benefit from extra management and feed, and whether they need a better diet once they are weaned.

"Or is it just they will always be small so we just have to accept that and then incorporate that into the [pigs'] production and management?" he adds.

In addition to the research being done by Columbus and Weber, several other USask researchers are working with the same group of piglets to optimize their use and the amount of knowledge gained from the project — a One Health approach to research that's a critical part of the university's research program.

"We brought in so many researchers on this one study," says Columbus. "[We] are trying to get as many samples and analyses done as possible because then it gives all these people ideas as to what can we pursue in the future — beyond the cardiovascular, beyond the muscle development."

The Natural Sciences and Engineering Research Council of Canada (NSERC) provided financial support for this USask research study.

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