

# Feeding mycotoxin contaminated grain to swine



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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

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

## Take Home Message

- 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.

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

“Determining the optimal feeding strategy is dependant on which mycotoxins are presence and their concentration”

status are important considerations. Table 1 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 which is representative of the entire lot. When sampling grains or feeds, subsamples from 12-20 locations should be collected, composited 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

stock may help to reduce overall contamination, it also means that these fractions are typically heavily contaminated. Because mycotoxins and mould spores can concentrate in grain dust it is very important that inhalation is avoided and dust masks are worn when handling, as they will affect human health also. Soaking, dehulling, cleaning and/or roasting may be beneficial in some cases, as are some dietary additives.

The Canadian regulatory guidelines for feeding mycotoxins are summarized in Table 2 (see page 9). Diets must not contain more than what is listed in these guidelines. If mycotoxin contamination is suspected; dilution can mitigate the problem, but because of the issues discussed with sampling, even when diluted, the grain should be fed to the least susceptible group (for example, keep ergot and ZEN out of your breeding herd).

**Table 1: Major effects of mycotoxins on swine performance**

<i>Mycotoxin</i>	<i>Primary Effect</i>	<i>Stage Affected</i>	<i>Clinical Signs</i>
<i>Deoxynivalenol (DON, vomitoxin)</i>	<i>Affects serotonin receptors and cytokine production</i>	<i>All stages (younger pigs may be more susceptible)</i>	<ul style="list-style-type: none"> <li>- <i>Reduced ADFI and ADG<sup>1,2</sup></i></li> <li>- <i>Vomiting<sup>2</sup></i></li> <li>- <i>Diarrhea (soft or watery feces)<sup>3</sup></i></li> <li>- <i>Reduced immune function<sup>3</sup></i></li> <li>- <i>Mild changes to kidney, thyroid, blood<sup>4</sup></i></li> </ul>
<i>Aflatoxins</i>	<i>Mutagenic and carcinogenic</i>	<i>All stages</i>	<ul style="list-style-type: none"> <li>- <i>Reduced ADFI and ADG<sup>5</sup></i></li> <li>- <i>Reduced milk production<sup>5</sup></i></li> <li>- <i>Lethargy<sup>6</sup></i></li> <li>- <i>Ataxia (lack of coordination)<sup>6</sup></i></li> <li>- <i>Rough hair coat<sup>6</sup></i></li> <li>- <i>Hemorrhage<sup>6</sup></i></li> <li>- <i>Fatty liver<sup>6</sup></i></li> </ul>
<i>Zearalenone</i>	<i>Estrogenic</i>	<i>Pre-pubertal gilts, sows and pre-pubertal boars</i>	<ul style="list-style-type: none"> <li>- <i>Swelling and reddening of the vulva<sup>7</sup></i></li> <li>- <i>Vaginal and/or rectal prolapse<sup>7</sup></i></li> <li>- <i>Anestrus<sup>8</sup></i></li> <li>- <i>Reduced litter size<sup>8</sup></i></li> <li>- <i>Fetal resorption<sup>8</sup></i></li> <li>- <i>Implantation failure<sup>8</sup></i></li> <li>- <i>Decreased libido and testosterone<sup>9</sup></i></li> <li>- <i>Feminization<sup>9</sup></i></li> </ul>
<i>Ochratoxin A</i>	<i>Disrupts phenylalanine (an amino acid) metabolism</i>	<i>All stages</i>	<ul style="list-style-type: none"> <li>- <i>Kidney damage<sup>10</sup></i></li> <li>- <i>Decreased ADFI and ADG<sup>11</sup></i></li> <li>- <i>Immunosuppression, increased risk of infection<sup>12</sup></i></li> </ul>
<i>Fumonisin</i>	<i>Disrupts lipid metabolism</i>	<i>All stages, especially young pigs</i>	<ul style="list-style-type: none"> <li>- <i>Unthriftiness<sup>6</sup></i></li> <li>- <i>Low ADFI and ADG<sup>6</sup></i></li> <li>- <i>Reproductive failure<sup>6</sup></i></li> <li>- <i>Gastric upset (diarrhea)<sup>6</sup></i></li> <li>- <i>Cellular necrosis<sup>6</sup></i></li> <li>- <i>Immunosuppression<sup>6</sup></i></li> </ul>
<i>T-2 and HT-2 Toxins</i>	<i>Inhibits protein synthesis</i>	<i>All stages</i>	<ul style="list-style-type: none"> <li>- <i>Pulmonary edema<sup>3</sup></i></li> <li>- <i>Reduced immunity<sup>3</sup></i></li> <li>- <i>Decreased ADFI and ADG<sup>13</sup></i></li> <li>- <i>Shortness of breath<sup>3</sup></i></li> <li>- <i>Weakness<sup>3</sup></i></li> <li>- <i>Cyanosis (blue/purple colour of skin/membranes)</i></li> </ul>
<i>Ergot Alkaloids</i>	<i>Neurological</i>	<i>All stages, especially the reproductive herd</i>	<ul style="list-style-type: none"> <li>- <i>Lameness<sup>14</sup></i></li> <li>- <i>Gangrene<sup>14</sup></i></li> <li>- <i>Decreased ADG<sup>14</sup></i></li> <li>- <i>Abortion<sup>14</sup></i></li> <li>- <i>Agalactia (absence of milk production)<sup>14</sup></i></li> <li>- <i>Ataxia<sup>14</sup></i></li> </ul>

<sup>1</sup> Decreased ADFI and feed refusals have been shown at levels as low as 0.5-1 ppm (Smith et al., 2005)

<sup>2</sup> > 2-5 ppm is for decreased ADFI and ADG, vomiting and complete feed refusal at > 20 ppm (Haschek et al., 2002)

<sup>3</sup> Pierce and Diaz, 2014

<sup>4</sup> JECFA, 2001

<sup>5</sup> Nibbelink, 1986

<sup>6</sup> Whitlow et al., 2014

<sup>7</sup> Friend et al., 1990

<sup>8</sup> Smith et al., 2005

<sup>9</sup> Osweiler, 1986

<sup>10</sup> Kidney damage occurs at levels as low as 0.5 ppm (Lippold et al., 1992)

<sup>11</sup> Performance is affected at levels of 2 ppm or greater (Lippold et al., 1992; Stoev et al., 2000)

<sup>12</sup> Can occur when levels > 2 ppm are fed for longer periods of time (Harvey et al., 1992)

<sup>13</sup> ADG reduced by 11% when 10 ppm fumonisin B1 was fed to starter pigs for 8 weeks (Rotter et al., 1996)


<sup>14</sup> Strickland et al., 2011

(Feeding Mycotoxin...Continued on page 9)

## Study Design

Twenty-eight litters were studied over four treatments (seven litters per treatment), with creep feed provided to all litters from 10 to 28 days of age (weaning). Treatments consisted of creep offered in one of two feeder designs (a standard commercial feeder, or a low edge baking tray), with or without enrichment provision, as follows: T1) creep provided in a standard feeder (SF), T2) creep provided in a standard feeder with enrichment (SF&E), T3) creep provided in a tray feeder (TF), and T4) creep provided in a tray feeder with enrichment (TF&E) (Figure 1). Enrichment treatments received strips of cotton rope suspended in the pen from day 5 until weaning. Piglet weights and creep consumption were recorded weekly, from birth up until six weeks of age, including an additional weight at day 1 post weaning. Piglet behaviour was recorded from 8am – 4pm, on days 12, 19, and 26 of age, and on days 1, 7 and 14 post-weaning. Footage was scanned at five minute intervals to determine the number of piglets interacting with the feeder (head in feeder), and the number of piglets interacting with the enrichment.

## The Bottom Line

Provision of a large tray feeder encouraged social feeding and foraging by piglets and was more effective at attracting piglets to the creep than a standard feeder, or the provision of rope enrichment. This may be because the tray feeder provided a greater opportunity for group foraging and rooting behaviour. Provision of the tray feeder before weaning led to a positive effect on piglet growth immediately after weaning. These growth benefits may have arisen from piglets more readily taking to solid feed post weaning, having had increased exploration of solid feed pre-weaning. The greater feed disappearance from the tray feeder may have been due to increased feed wastage. However, if increasing the foraging behaviour is enough to encourage feed intake immediately post-weaning then providing expensive creep feed in the tray may not be necessary – and rather any material that the piglets can forage and ingest would do, such as beet pulp. This is an area for further research. Analysis of the post-weaning data is ongoing, and results will help determine if the effects of the tray feeder pre-weaning has lasting positive effects post weaning. 

(Feeding Mycotoxin...Continued from 7)

**Table 2. Legislated maximums, regulatory guidelines and recommended maximums for different mycotoxins into swine diets (adapted from Charmley and Trenholm, 2012)\***

Mycotoxin	Commodity	Levels
Deoxynivalenol <sup>1</sup>	Diets for swine	1 ppm
Aflatoxins <sup>2</sup>	Animal feeding stuffs	20 ppb
T-2 toxin <sup>3</sup>	Swine diets	< 1 ppm
Zearalenone <sup>3</sup>	Gilt diets	< 1-3 ppm
Swine diets	< 0.25-5 ppm	
Ochratoxin A <sup>3</sup>	Swine diets (kidney damage)	0.2 ppm
	Swine diets (reduced weight gain)	2 ppm
Ergot Alkaloids <sup>3</sup>	Swine diets	4-6 ppm
Fumonisin <sup>3</sup>	Swine diets	10 ppm

\*ppm is parts per million (mg/kg) and ppb is parts per billion.

<sup>1</sup> Regulatory guidelines (Worldwide regulations for mycotoxins. FAO Food and Nutrition Paper 64, 1997)

<sup>2</sup> Legislated maximum tolerated level (Worldwide regulations for mycotoxins. FAO Food and Nutrition Paper 64, 1997)

<sup>3</sup> Recommended tolerance levels in Canada and the United States

## For Further Information:

Charmley, L. L., and H. L. Trenholm. 2012. RG-8 Regulatory Guidance: Section 1: Mycotoxins in Livestock Feed. Canadian Food Inspection Agency Regulation. <http://www.inspection.gc.ca/animals/feeds/regulatory-guidance/rg-8/eng/1347383943203/1347384015909?chap=1#s1c1>

Friend, D. W., H. L. Trenholm, B. K. Thompson, K. E. Hartin, P. S. Fiser, E. K. Asem, and B. K. Tsang. 1990. The reproductive efficiency of gilts fed very low level of zearalenone. *Can. J. Anim. Sci.* 70: 635-645.

Harvey, R. B., M. H. Elissalde, L. F. Kubena, E. A. Weaver, D. E. Corrier, and B. Clement. 1992. Immunotoxicity of ochratoxin A to growing gilts. *Am. J. Vet. Res.* 53: 1966-1970.

Haschek, W. M., K. A. Voss and V. R. Beasley. 2002. Selected mycotoxins affecting animal and human health. In: *Handbook of Toxicologic Pathology*. 2nd Ed, Vol. 1, Academic Press, p. 645-699.

JECFA 56th. 2001. Joint FAO/WHO Expert Committee on Food Additives, 56th Report. Safety evaluation of certain mycotoxins in food. Food and Agriculture Organization of the United Nations, paper 74. World Health Organization Food Additives Series 47, World Health Organization, Geneva, Switzerland.

Lippold, C. C., S. C. Stothers, A. A. Frohlich, R. J. Boila, and R. R. Marquardt. 1992. Effects of periodic feeding of diets containing ochratoxin A on the performance and clinical chemistry of pigs from 15 to 50 kg body weight. *Can. J. Anim. Sci.* 72: 135-146.

Nibbelink, S. K. 1986. Aflatoxicosis in food animals: A clinical review. *Iowa State Univ. Vet.* 48: 28-31.

Osweller, G. D. 1986. Occurrence and clinical manifestations of trichothecenes toxicoses and zearalenone toxicoses. In: *Diagnosis of Mycotoxicoses* (J. L. Richard and J. R. Thurston, eds.). Martinus Nijhoff Publishers, Dordrecht. P. 31-42

Pierce, J., and D. Diaz. 2014. Mycotoxin Management and its Importance in Feed Quality, Safety and Maintaining Reproductive and Growth Performance in Swine Production. *Proceedings of the 35th Western Nutrition Conference, Edmonton, AB.* p 23-32.

Rotter, B. A., B. K. Thompson, D. B. Prelusky, H. L. Trenholm, B. Stewart, J. D. Miller, and M. E. Savard. 1996. Response of growing swine to dietary exposure of pure fumonisin B1 during an eight-week period: growth and clinical parameters. *Nat. Toxins* 4: 42-50.

Smith, T. K., G. Diaz, H. V. L. N. Swamy. 2005. Current concepts in mycotoxicoses in swine. In: *Mycotoxin Blue Book* (D. E. Diaz ed.). Nottingham University Press, Nottingham, UK. p. 235-248.

Stoeb, S. D., D. Goundasheva, T. Mirtcheva, and P. G. Mantle. 2000. Susceptibility to secondary bacterial infections in growing pigs as an early response in ochratoxicosis. *Exper. Toxicol. Path.* 52: 287-296.

Strickland, J. R., M. L. Looper, J. C. Matthews, C. F. Rosenkrans Jr., M. D. Flythe, and K. R. Brown. 2011. St. Anthony's Fire in livestock: Causes, mechanisms, and potential solutions. *J. Anim. Sci.* 89: 1603-1626.

Whitlow, L. W., D. E. Diaz, and W. M. Hagler, Jr. 2014. Mycotoxins: Effects and Management. *Proceedings of the 35th Western Nutrition Conference, Edmonton, AB.* p 85-103. 