Fumonisins – a critical factor in swine health and productivity

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Fumonisins are toxic secondary metabolites produced mainly by toxinogenic strains of Fusarium moniliforme. The greatest production of fumonisins take place in substrates with water activity greater than 0.91 and temperatures between 15-25°C.

There are six types of fumonisins, but the most toxic and frequently found as natural contaminants of cereals, especially corn and corn by-products, are fumonisin B1 (FB1) and fumonisin B2 (FB2). They can resist temperatures as high as 150°C, depending on exposure time and pH of the substrate.

Fumonisins alter sphingolipids biosynthesis, induce hepatotoxicity and elevate serum cholesterol concentrations in all species studied. Other effects are species specific and include pulmonary oedema and cardiovascular changes in pigs.

Fumonisins in animal feeds

Today, fumonisin is probably the biggest problem of mycotoxins in feed. The World Health Organization (WHO) working group found that globally 59% of corn and corn product samples were contaminated with FB1.

According to Mallmann and Dilkin (2007) from 50 samples of hybrid corn in Argentina, all reported concentrations of FB1 from 0.185 to 27.05ppm and FB2 from 0.04 to 9.95ppm. In Brazil, from a total of 11,814 samples of feedstuffs, the average fumonisins, adding both the FB1 and FB2 together, was 0.995ppm.

Corn samples represented 55.62% of all samples and its average contamination was 1.01ppm in 58% of this cereal. Mixed feed had an average of 1.426ppm in 55% of the analysed samples. In addition, the natural contamination of FB1 and FB2 in corn coming from several countries of Europe were positive but low, showing contaminations between 0.055 and 5ppm.

In a survey carried out from 1996 to 1999 in South Georgia, up to 91% of the samples contained fumonisins; 30% of samples had concentrations above 1ppm, and the highest contamination was 33ppm. Japan's official government report of 2005 indicates that imported corn (80% coming from the USA) showed that 75.9% was contaminated with fumonisin at levels from 0.22 to 6.8ppm with an average of 2.1ppm.

Pulmonary oedema caused by fumonisin.

In approximately 106 feed samples, using corn as a main grain, associated with confirmed cases of swine pulmonary oedema in Brazil and the USA, between 2 to 333ppm of FB1 were seen.

Toxicity in swine

Fumonisin toxicity in swine is associated with pulmonary oedema, hepatotoxicosis, cardiovascular and immunosuppression problems and alterations in the biosynthesis of sphingolipids.

Besides these damages, fumonisin toxicity obviously has negative effects on performance, reducing feed consumption and body weight gain; affecting the efficiency of feed utilisation and carcase quality.

Pulmonary oedema

Physiologically, the two main causes of pulmonary oedema are left ventricular failure, which increases pulmonary capillary hydrostatic pressure and increased vascular permeability after injury to alveolar capillary endothelium or alveolar epithelium. High concentrations of FB1 in feeds usually cause pulmonary oedema problems in pigs; but in several instances, at lower levels of contamination, the animal needs a longer period of exposure to present the syndrome.

Several symptoms of pulmonary oedema were detected in weaned pigs which received contaminated feeds with FB1 in concentrations of 10, 20 and 40ppm for a short period of four weeks. Results showed mild lesions at 10ppm; moderate lesions in the group receiving 20ppm; and severe lesions in the group with the contamination of 40ppm.

On the other hand, weaned pigs, receiving low contaminations of 1, 5 and 10ppm of FB1 for an extended period of eight weeks resulted in pathological alterations of the lungs and pulmonary oedema lesions.

PPRS and circovirus

The presence of more than 20ppm of FB1 has been associated with the occurrence of the PRRS (Porcine Reproductive and Respiratory Syndrome) disease in swine.

Out of 21 groups studied, 12 presented clinical symptoms of that disease and eight of those 12 were consuming rations with more than 20ppm of FB1.

The role of mycotoxins as co-factors for the development of circovirus (PCVD) has been speculated. The effects of FB1 intoxication, such as liver damage and jaundice, pulmonary oedema, poor growth and immunosuppression, are similar to PCV2 infection.

Due to lack of knowledge and/or good histopathology, most of the time when these symptoms are seen, they are only attributed to PCV2. In vitro treatment of previously PCV2 infected SK-6 cells with FB1 stimulates initial PCV2 replication, showing that there is an interaction between them. Also, PCV2 and FB1 induce the appearance of apoptotic cells, suggesting a regulation of cell death and proliferation. The cellular and molecular mechanisms of this interaction is still unclear.

Other immunosuppressive related problems caused by fumonisins include:

1Increase in susceptibility to Escherichia coli.

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1Susceptibility to Pseudomonas aeruginosa infection and reduction of macrophages in the lungs.

Sphingolipids biosynthesis

The toxic effects of fumonisins are the result of its interference with the sphingosine and sphinganine metabolism, which result in a disturbance in the metabolism of sphingolipids.

Alteration of sphingolipids metabolism is due to inhibition of ceramide synthesis by fumonisin and results in accumulation of sphinganine, which has been used as a biomarker for fumonisin exposure.

The resulting effects are changes in cellular growth, differentiation, morphology, permeability and apoptosis. In swine, the partial or total inhibition of the sphingosine and the N-acyltransferase enzyme is responsible for the hepatotoxicosis problems.

High concentrations of FB1 cause pulmonary oedema that, in turn, produces hepatic hydrothorax and icterus. Chronic low concentrations of FB1 produces progressive degeneration and necrosis of the liver, affecting protein synthesis and pig performance.

Barrows receiving one and 10ppm FB1 contaminated feed for two weeks presented an increase in the level of blood cholesterol; had heavier pancreas and suprarenal glands.

An increment of the sphinganine and of the sphinganine/sphingosine ratio was observed. Pulmonary oedema was observed in the animals consuming the highest level of FB1.

Other investigations by Mallmann and Dilkin (2007) reported that hepatic alterations like, aleatory hepatocellular necrosis, nuclear pleumorphism, mitosis increment, deformed cells, hepatomegalocytosis and focal hepatocytic necrosis are observed frequently in pigs suffering chronic intoxication of low levels of fumonisin, insufficient to develop clinical pulmonary oedema.

Three trials were carried out in the swine facilities of the Universidade Federal de Santa Maria, Departamento de Medicina Veterinaria Preventiva, Laboratorio de Análises Micotoxicológicas (LAMIC), in order to study the efficacy of purified and modified phyllosilicate clay in preventing the deleterious effects of fumonisin in pigs.

In the first trial, a total of 20 pre-pubertal gilts were fed 30ppm of FB1 contaminated feeds, during a 28 day period. There were no significant alterations in feed consumption, body weight gain and feed conversion or in the relative weights of liver or heart in the contaminated treatments when compared with the control group.

The only significant alteration was in the macroscopic lesions and relative weight of the lungs: 0.79g (CV=6.89%) in the control group (non-contaminated feed) and 1.22g (CV=30.26%) in the contaminated group.

The addition of 0.5% of a purified modified phyllosilicate to the 30ppm FB1 contaminated feed resulted in a significant (P<0.10) reduction of the relative lung weight 0.87g (CV=6.38%) and the absence of lungs haemorrhages which were present in the contaminated group.

The other two experiments were conducted with finishing pigs using a similar experimental design but different length of time (28 and 56 days). Twelve male pigs averaging 58.5kg initial body weight were used in each experiment.

Pigs were individually housed and randomly distributed into three dietary treatments with four replications and fed corn-soybean meal diets meeting or exceeding NRC recommendations.

All ingredients used tested free of mycotoxin contamination.

Treatments were:

- Control diet.
- Control + 25ppm fumonisin.
- Control + 25ppm fumonisin + 0.4% of purified modified phyllosilicate.

Fumonisin was obtained from a culture material containing 72% FB1 and 28% FB2 produced in LAMIC. Performance and organs (lungs, heart and liver) relative weights (g/kg body weight) were evaluated in experiment 1 (EXP1). Performance, lungs relative weight and serum sphinganine/sphingosine ratio (SA:SO) were determined in experiment 2 (EXP2).

Results from experiments

Results from both experiments showed that pigs fed 25ppm fumonisin had significantly (P≤0.05) poorer performance; increased lungs, heart and liver relative weights; and increased serum SA:SO than pigs fed the control diet. The addition of the purified modified phyllosilicate to the contaminated diet significantly (P≤0.05) improved performance parameters and relative organ weight: feed intake (2615 vs 2315g EXP1) (2948 vs 2810g EXP2), daily gain (861 vs 722g EXP1) (1084 vs 996g EXP2), feed efficiency (2.70 vs 3.08 EXP1) (3.21 vs 3.46 EXP2), lungs (6.68 vs 9.69 EXP1) (5.94 vs 6.34 EXP2), heart (3.75 vs 4.87 EXP1), and liver (18.65 vs 20.89 EXP1).

Serum SA:SO was significantly (P≤0.05) increased in pigs fed fumonisin compared to the control and the purified modified phyllosilicate fed pigs (0.78 vs 0.38 and 0.49).

The results from these three experiments indicate that this purified and modified phyllosilicate clay was very effective in preventive the toxic effects of fumonisin in pigs. ■

References are available from the authors on request.