Deoxynivalenol predisposes for the development of necrotic enteritis in broilers

<u>Gunther Antonissen^{1,2}</u>, Filip Van Immerseel¹, Frank Pasmans¹, Richard Ducatelle¹, Freddy Haesebrouck¹, Leen Timbermont¹, Marc Verlinden¹, Jeroen Dewulf³, Mia Eeckhout⁴, Sarah De Saeger⁵, Evelyne Delezie⁶, An Martel¹, Siska Croubels²

 Dept. of Pathology, Bacteriology and Poultry Diseases 2) Dept. of Pharmacology, Toxicology and Biochemistry 3) Dept. of Reproduction, Obstetrics and Herd Health, Faculty of Veterinary Medicine 4) Dept. of Food Science and Technology, Faculty of Biosciences and Landscape Architecture 5) Dept. of Bioanalysis, Faculty of Pharmaceutical Sciences, Ghent University 6) Institute for Agricultural and Fisheries Research (ILVO) Animal Sciences Unit, Belgium <u>E-mail:</u> Gunther.Antonissen@UGent.be

Clostridium perfringens induced subclinical necrotic enteritis (NE) causes important economic losses in the broiler industry. The *Fusarium* mycotoxin deoxynivalenol (DON) may affect the intestinal epithelial integrity, subsequently inducing protein leakage into the intestinal lumen. The objective of this study was to examine whether DON at contamination levels below the maximum guidance level in poultry feed is a predisposing factor for NE in broilers.

In this study we used a highly reproducible *in vivo* infection model mimicking subclinical NE (Gholamiandehkordi et al., 2007). A total of 360 one-day-old Ross 308 broilers were randomly divided into four groups of three replicates with 30 birds per replica. All birds were fed a starter diet during the first eight days of the experiment, subsequently a grower diet for eight days, followed by a finisher diet until euthanasia. Throughout the entire experiment, groups 1 and 4 received a blank diet while groups 2 and 3 received a diet experimentally contaminated with DON. All birds in group 1 and 2 were challenged orally with *C. perfringens* strain 56 containing approximately 4×10^8 cfu/ml for four consecutive days starting at day 17. The remaining groups received sterile medium.

The blank feed contained DON at 75 ± 22 μ g/kg (starter), 83 ± 24 μ g/kg (grower) and 100 ± 29 μ g/kg (finisher). The contaminated feed contained DON at 3761 ± 1100 μ g/kg (starter), 4281 ± 1300 μ g/kg (grower) and 4384 ± 1300 μ g/kg (finisher).

At 1, 2 or 3 days after the final challenge with *C. perfringens*, chickens were euthanized and scored macroscopically for intestinal NE lesions. Chickens that received DON and *C. perfringens* had significantly (alpha=0.05, P<0.001) more lesions than chickens that received only *C. perfringens*, with 46.6% and 19.5% of chickens positive for NE lesions, respectively. In non-inoculated groups no NE lesions were present.

In conclusion, the presence of DON in the feed in concentrations lower than the maximum guidance level of 5000 μ g/kg is a predisposing factor for the development of NE in broilers.

References

Gholamiandehkordi, A.R.; Timbermont, L.; Lanckriet, A.; Van Den Broeck, W.; Pedersen, K.; Dewulf, J.; Pasmans, F.; Haesebrouck, F.; Ducatelle, R.; Van Immerseel F., 2007. Quantification of gut lesions in a subclinical necrotic enteritis model. Avian Pathology 36, 375-382.

Acknowledgements

This work was financially supported by BIOMIN GmbH, Austria.