

A need for new analytical approach for assessment zearalenone exposure of pigs

Abbreviations: ZEA, Zearalenone; PMTDI, provisional maximum tolerable daily intake.

Editorial

Mycotoxins are secondary metabolites produced by wide variety toxigenic fungi that can occur in all agricultural commodities causing great economic losses at all levels of food and feed production, including crop yields and animal production.¹ Mycotoxin contaminated diets can reduce performance in ruminants and non-ruminants and can have detrimental effects on both finishing animals and reproducing males and females.² Despite that there are over than 500 of mycotoxins described in the literature, only the number of known mycotoxins that pose a measurable health risk to farm animals and humans are covered by EU and world legislation (aflatoxins, ochratoxinA, deoxinivalenol, zearalenone and fumonisins).³

In contrast to many other mycotoxins, zearalenone (ZEA) exhibits a very low acute toxicity, but it possesses a powerful estrogenic activity exceeding that of most other naturally occurring non-steroidal estrogens.⁴ Therefore, the presence of ZEA is of the greatest concern for reproducing animals, especially pigs which are the most susceptible species.⁵ This is supported by the fact that JECFA⁶ concluded that the safety of zearalenone could be evaluated on the basis of the dose that had no hormonal effects in pigs. JECFA established a provisional maximum tolerable daily intake (PMTDI) for zearalenone of 0.5µg/kg of body weight.

Zearalenone is a macrocyclic β-resorecylic acid lactone produced by several *Fusarium* species.⁷ ZEA and its derivatives have been detected in many important crops, such as corn, wheat, sorghum, barley, oats, sesame seed, hay and corn silage.^{8,9} Several studies carried out in Europe have reported a high incidence of ZEA in cereals and feeds, most often in Central (Slovakia, Czech Republic), Eastern (Serbia, Romania, Croatia) and Southern (Greece, Italy, Spain, Portugal) parts.¹⁰ Although in most cases the concentrations of ZEA and other mycotoxins remained below the maximum tolerable levels set by the EU (100 µg/kg for feedingstuffs for piglets and gilts),¹¹ it is perceivable that long term co-exposure to various mycotoxins will impair animal health and productivity.^{12,13} The presence of ZEA in pigs feed often can have detrimental impact on production and reproduction, leading to great economic losses for producers. According to Visconti¹⁴ pig producers are affected by increased costs because of higher mortality rates, reproductive failures (abortion), reduced feed efficiency (higher feed costs, lower live weight, infertility syndrome, increased susceptibility to disease), overall quality loss and analytical testing. It was estimated that a 10 or 20% reduction in farrowing rate combined with a 10 or 20 % reduction in growth (in cases of co-contaminated with zearalenone and deoxinivalenol feed) would result in a 17 to 44% reduction in profit margins, due to searching for uncontaminated feed, Increased health care and veterinary costs per head, and loss of markets.¹⁵

There are well established analytical techniques for determination and quantification of ZEA from almost every commodity, mainly

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based on the GS, GS-MC, HPLC, LC-MC and ELISA for the initial screening. However, the problem with these powerful tools for micotoxicological investigations remains obtaining representative samples.¹⁶ Furthermore, mycotoxins can be “masked” by certain nutrients making them undetectable. This can be possible explanation for the observed discrepancy between reported data of the prevalence of contamination with ZEA in feeds, and the number of described, proven cases of field outbreaks of zearalenone-toxicoses.¹⁷ In most cases suspected zearalenone-toxicoses detected by nutritionists and veterinarians remain unreported and the etiological agent responsible for the observed signs is not confirmed when health and production problems improve or disappears following removal of the suspected diets. Sometimes, conclusions could be drawn from herd observations rather than planned experiments, and ZEA is often not measured in the feeds involved.¹⁸

Under field conditions, ZEA, like other mycotoxins usually occur in concentrations leading to reduced animal performance without causing any obvious clinical symptoms. Moreover, clinically observed symptoms of ZEA exposure could not only be result of the actual (measured) toxin concentration in a given feed, but may also be induced or modulated as the consequence of previous exposure. This is of practical relevance, because the ‘historical exposure’ of an animal is usually unknown but should be recognized as a possible contributor to disease expression.¹⁹ For the reasons described above it is difficult to make a link between suspected mycotoxicoses of livestock and the presence of mycotoxins, using standards analytical techniques. Therefore, there is an increasing demand for developing innovative, modern, easy and fast tools for correct diagnostic of zearalenone-toxicoses in pigs.

A possible approach to achieve this goal is so called “omics” technologies and especially transcriptomics that use the ability of living cells to respond to the presence of toxins altering many parameters (including thousands of mRNAs and recently found microRNAs) and leaving specific fingerprints in the form of gene expression, according to their type. The effects on gene expression alterations precede clinical effects. Therefore, mechanisms of action can be detected using transcriptional biomarkers provides information for gene signatures, specific for certain compounds and for mode of action shared by groups of compounds.²⁰ One major promise of the transcriptomics is that it could increase our knowledge about toxic

mechanisms on basis of which the hazard and potentially the risk of a toxic compound can be assessed.

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Conflicts of interest

Author declares that there is no conflict of interest.

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