From research to application – proving the efficacy of ZEN*zyme*® in swine

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Introduction

Mycotoxins, secondary toxic metabolites from filamentous fungi contaminating food and feed, cause a wide range of adverse health effects. One major mycotoxin is zearalenone (ZEN), which shows strong estrogenic effects in humans and animals even at low levels due to its estrogen-like structure. One strategy to mitigate the negative impacts of ZEN involves using the zearalenone hydrolase ZenA (ZEN*zyme*®) as a feed additive for enzymatic detoxification of ZEN. The enzyme cleaves the lactone ring of ZEN to form the non-estrogenic metabolite hydrolyzed zearalenone (HZEN), which can convert spontaneously to decarboxylated hydrolyzed zearalenone (DHZEN) (figure 1). Both analytes exhibit less toxicity compared to ZEN.

The aim of this study was to prove the efficacy of ZEN*zyme*® degrading ZEN in the gastrointestinal (GI) tract of pigs, consequently resulting in reduced levels of systemically absorbed ZEN.

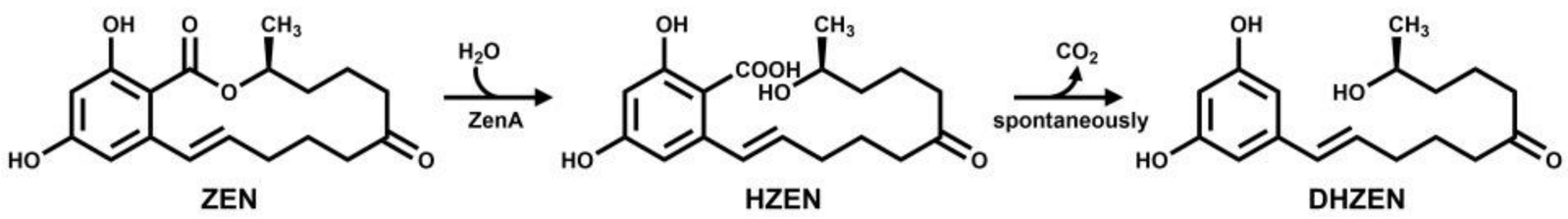

Figure 1: Enzymatic detoxification of zearalenone (ZEN) into less toxic hydrolyzed zearalenone (HZEN) and decarboxylated hydrolyzed zearalenone (DHZEN) using the zearalenone hydrolase ZenA (ZEN*zyme*®).

Materials & Methods

In a feeding trial 72 weaned piglets were exposed to feed contaminated with 200 µg ZEN/kg feed for 42 days. In one group the feed was additionally supplemented with 10 Units ZEN*zyme*®/kg feed. Performance parameters along with exposure based biomarkers in plasma, urine and feces using LC-MS/MS were assessed at different time points. Applied methods covered the quantification of ZEN, α-zearalenol (α-ZEL) and β-zearalenol (β-ZEL) as well as the enzymatic degradation products HZEN and DHZEN.

Results

Different diets had no influence on performance parameters like body weight, body weight gain or the feed conversion rate of the piglets. In plasma the mean ZEN concentration was reduced from 0.31 ng/mL to 0.22 ng/mL, if ZEN*zyme*® was added to the feed. Compared to blood, results in urine showed higher variability, even if urinary ZEN concentrations were adjusted by their creatinine (crea) values. Nevertheless, ZEN*zyme*® reduced the mean ZEN concentration in urine from 16.19 µg/mmol crea to 10.88 µg/mmol crea. In feces, in the presence of ZEN*zyme*®, both ZEN and α-ZEL levels were reduced while HZEN levels were elevated proving the enzymatic degradation of ZEN in the GI tract.

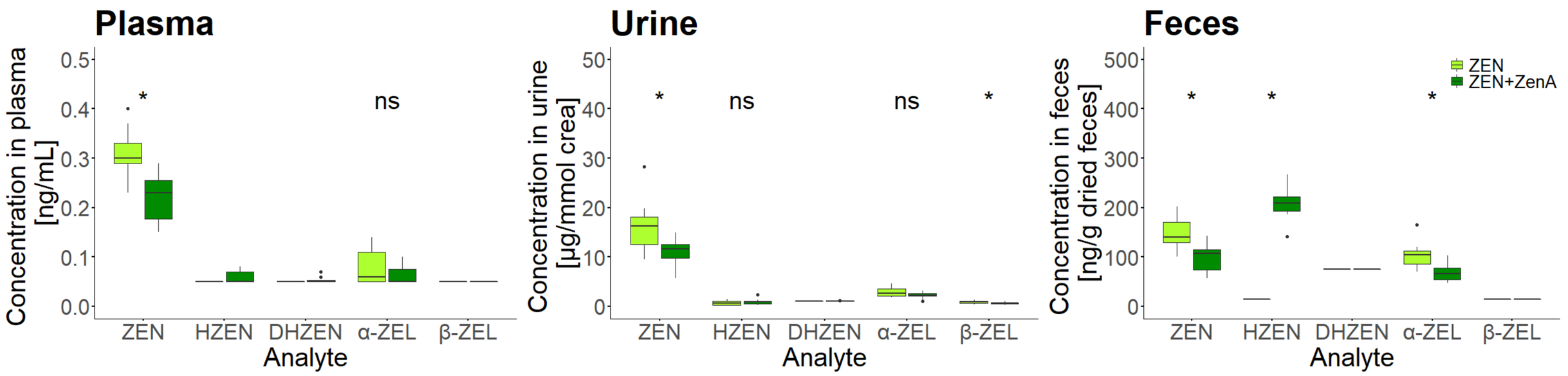

Figure 2: Concentrations of zearalenone (ZEN), hydrolyzed zearalenone (HZEN), decarboxylated hydrolyzed zearalenone (DHZEN), α-zearalenol (α-ZEL) and β-zearalenol (β-ZEL) in plasma, urine, and feces of piglets. Piglets were exposed to feed contaminated with 200 µg ZEN/kg (ZEN) or feed contaminated 200 µg ZEN/kg supplemented with 10 U ZenA/kg (ZEN + ZenA) for 42 days. Significant differences between treatments are indicated with an asterisk ($p < 0.05$), while ns describes non-significant comparisons ($p > 0.05$). If no symbol is shown, evaluation was not possible due to an insufficient number of values above the limit of quantification.

Overall, the present study proves the efficacy of ZEN*zyme*® to degrade ZEN in the GI tract of pigs. Systemic ZEN levels were decreased if ZEN*zyme*® was present in the feed at 10 U/kg feed in all tested matrices (plasma, urine and feces). Furthermore, especially in feces, increased levels of the enyzmatic detoxification product HZEN were detected.

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