

# Impact of Mycotoxins in Turkeys



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Mycotoxins are present in nearly all raw materials used to make turkey feed. They have a huge impact on the production performance of the flock, but using a mycotoxin deactivation product in the diet can mitigate these negative effects.

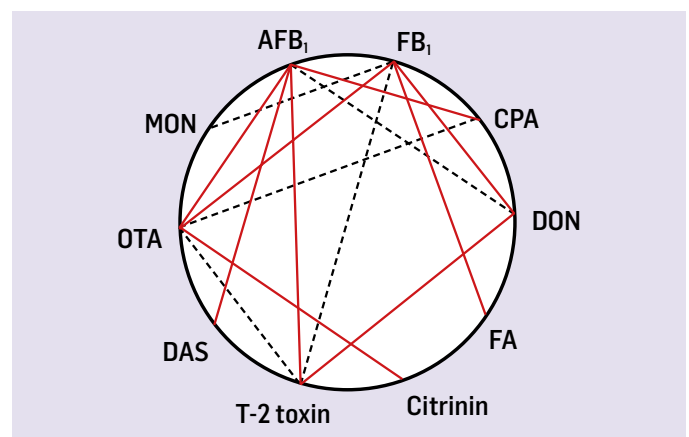
Turkey and poultry species in general are sensitive to a broad array of mycotoxins. Aflatoxins, type A trichothecenes (T-2 and HT-2 toxin), type B trichothecenes (deoxynivalenol (DON), nivalenol (NIV), or diacetoxyscirpenol (DAS)), fumonisins (FUM) and ochratoxins are among the groups that can impair production the most. Aflatoxins are potent liver carcinogens; they can influence animal production by triggering severe immunosuppression, cancer of the liver and spleen, feed refusal and carry-over into tissues and eggs. Contamination of feed with subclinical doses of aflatoxins can negatively influence intestinal histology and reduce the adsorption of crude proteins from the feed. Trichothecenes are protein synthesis inhibitors; hence, they are highly toxic to cells. Type A trichothecenes such as T-2 and HT-2

## IN BRIEF

- Mycotoxin contamination in feed can cause a myriad of performance and health problems
- Most raw materials are naturally contaminated with more than one mycotoxin
- To mitigate negative effects, a mycotoxin deactivation product with several modes of action should be included in the diet

Figure 1.

Synergistic effects of mycotoxins in poultry



FB<sub>1</sub> = Fumonisin B<sub>1</sub>, CPA = Cyclopiiazonic acid, DON = Deoxynivalenol, FA = Fusaric acid  
DAS = Diacetoxyscirpenol, OTA = Ochratoxin A, MON = Moniliformin, AFB<sub>1</sub> = Aflatoxin B<sub>1</sub>

Source: BIOMIN

produce visible lesions on the beak and in the gut, leading to feed refusal. The most detrimental effects of trichothecenes are observed in the gastrointestinal tract, where they can compromise the integrity of the gut by disrupting the tight junctions – thus favoring the passage of pathogens and other toxic entities into the bloodstream. Trichothecenes such as DON have repercussions on villi histology as well: villi atrophy, decreased villi height and crypt depth have been observed in birds fed subclinical doses (below EU regulation guidelines) of DON. The effects of DON are enhanced by the presence of FUM. These mycotoxins act synergistically, rendering the immunosuppressive and cytotoxic effects of DON and other trichothecenes more severe. Moreover, DON and FUM are predisposing factors for the development of necrotic enteritis and coccidiosis.

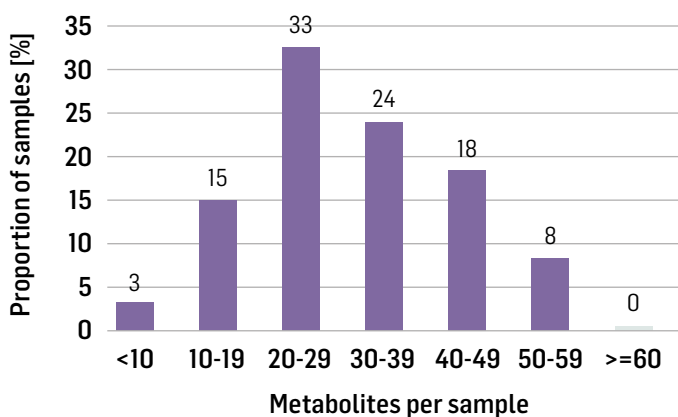
When it comes to mycotoxin exposure, it is important to bear in mind the synergistic effects. Synergism is when the toxicity of one mycotoxin is greatly increased by the presence of others. The most relevant synergistic interactions in poultry are reported in Figure 1. The toxicity of mycotoxins depends on the dosage and the exposure time. Consequences

## Contamination of feed with subclinical doses of aflatoxins can negatively influence intestinal histology and reduce the adsorption of crude proteins from the feed.

Photo: Shutterstock, Dr. Mierze

**Figure 2.**

Co-occurrence of mycotoxins in samples worldwide, Jan. - Nov. 2017



Source: BIOMIN

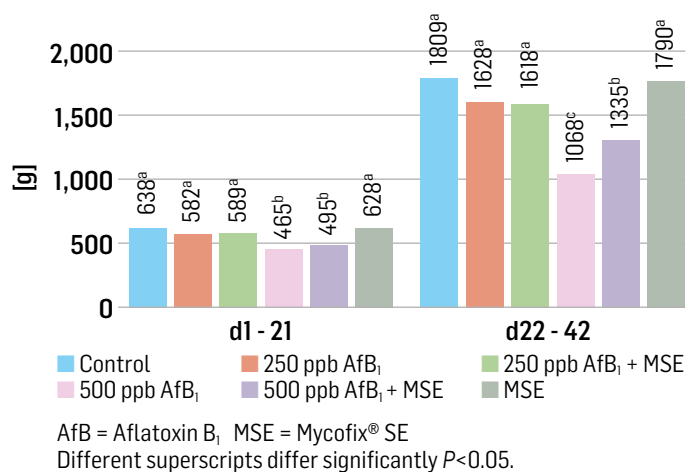
to production can be detrimental whether the animals are exposed to subclinical doses over a prolonged time, or short-term high-level exposure.

As reported by the BIOMIN mycotoxin survey, animals are always exposed to cocktails of mycotoxins – the 2017 survey data reported an average of 31 metabolites per sample (Figure 2). As mycotoxins can greatly differ in their physiochemical properties, an efficacious mycotoxin deactivation product needs to work in several different ways to counteract them all. Adsorption can only help against a small number of mycotoxins (mostly aflatoxins, ergots and ochratoxins).

One of the major challenges for mycotoxin deactivation is to prove the effectiveness *in vivo*. According to the official EU registration protocol, this has to be accomplished with biomarkers, as they are the proof of mycotoxin deactivation at a molecular level. In fact, to register a product in the EU, *in vitro* results are not enough. Mycofix® is the only EU registered multi-strategy product available on the market and its state-of-the-art mode of action, based on adsorption and biotransformation, has been tested in turkeys against aflatoxins, trichothecenes and fumonisins in three different trials.

**Figure 3.**

Body weight at day 21 and at the end of the experiment



Source: BIOMIN

## Mycofix® is able to counteract high concentrations of aflatoxin

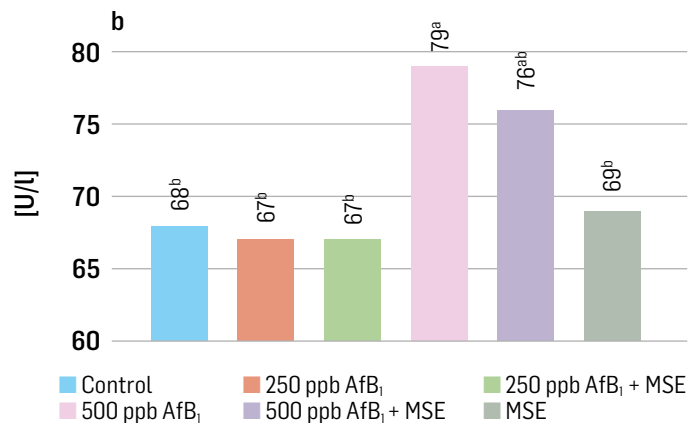
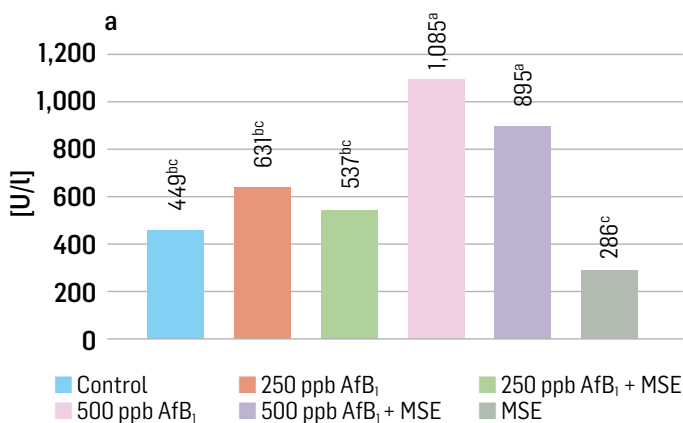
The efficacy of Mycofix® to counteract aflatoxins (Afla) was tested on 210 one-day-old turkey poults exposed to relatively high amounts of Afla for 42 days. Different parameters were measured during the experiment including performance parameters (individual weight, feed intake, feed conversion ratio (FCR)), organ health measurements (relative organ weights, liver enzymes (AST and LDH), and strength of the immune response. The results showed that Mycofix® counteracted the adverse effects on turkey performance and on selected toxicopathological parameters, and completely overcame the negative effects of mycotoxins, including mortality, which has important economic implications for the poultry producer. The results are shown in Figures 3 and 4.

## FUMzyme®, the breakthrough in FUM deactivation

The ability of FUMzyme® to detoxify FUM in the gastrointestinal tract of turkeys was assessed in a field trial.

**Figure 4.**

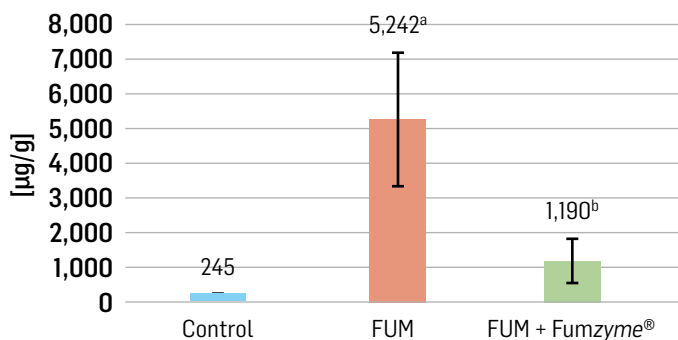
LDH (a) and AST (b) levels at day 35



Different superscripts differ significantly  $P < 0.05$ .  
Source: BIOMIN

**Figure 5a.**

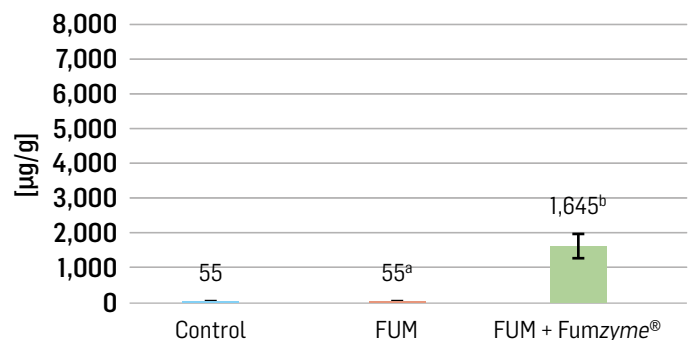
FB<sub>1</sub> in turkey feces day 14 (µg/g)



Different superscripts differ significantly  $P < 0.05$ .  
Source: BIOMIN

**Figure 5b.**

HFB<sub>1</sub> in turkey feces day 14 (µg/g)



Different superscripts differ significantly  $P < 0.05$ .  
Source: BIOMIN

Fifteen hybrid turkeys at ten weeks of age were fed 15 ppm of FUM (specifically FB<sub>1</sub> was used in the trial). FUMzyme<sup>®</sup> converts FB<sub>1</sub> into the hydrolyzed non-toxic metabolite HFB<sub>1</sub>. A way to assess the activity of the enzyme is to measure the gradual disappearance of FB<sub>1</sub> and appearance of HFB<sub>1</sub>. To do so, fecal samples were collected after 14 days. As shown in Figure 5a (green bar), FUMzyme<sup>®</sup> significantly lowered the FB<sub>1</sub> content in the feces compared to the FB<sub>1</sub> contaminated group without additive (red bar). The metabolite HFB<sub>1</sub> was significantly elevated in the FUM + FUMzyme<sup>®</sup> treatment (Figure 5b, green bar), showing effective biotransformation of FB<sub>1</sub> to HFB<sub>1</sub>.

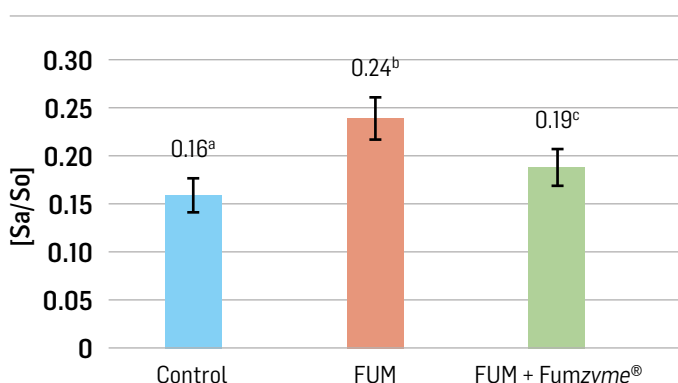
Another biomarker assay that is commonly used to assess FUM deactivation is the sphinganine (Sa): sphingosine (So) ratio. The mode of action of FUM is the inhibition of the enzyme ceramide synthase that converts free Sa and So (molecules that are precursors of sphingolipids) into complex sphingolipids, important structural components of cell membranes. Once the enzyme is inhibited, the free Sa and So molecules start accumulating in the cell with Sa being the

predominant metabolite. This accumulation is measurable; specifically the ratio between free Sa and So. The higher the ratio, the more severe the FUM intoxication. In one trial, the Sa:So ratio (Figure 6) in serum at day 14 was significantly elevated in the FUM contaminated group compared to the control group without FUM and FUMzyme<sup>®</sup>. The addition of FUMzyme<sup>®</sup> significantly lowered the ratio, indicating FUM inactivation *in vivo*.

## Trichothecenes detoxification by BIOMIN BBSH 797

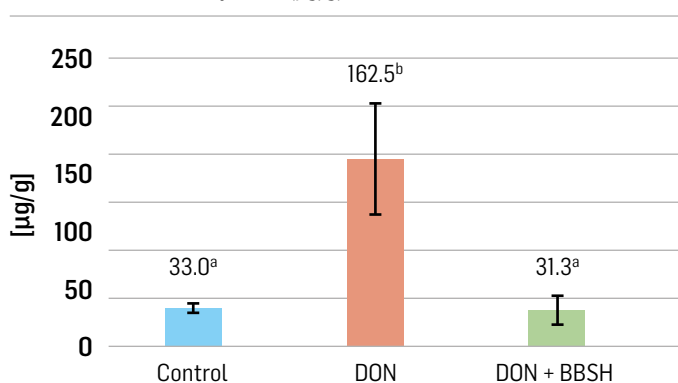
BIOMIN BBSH 797 catalyzes the cleavage of the epoxy group of trichothecenes by producing a specific enzyme called de-epoxidase during its metabolic activity in the gastrointestinal tract, which results in metabolites of no toxicological concern. The main metabolite of DON, the most prominent and prevalent mycotoxin among the group of trichothecenes, is DOM-1 (de-epoxy-deoxynivalenol). As reported in the literature (Wan *et al.*, 2014), DON-3-sulfate is the major metabolite of

**Figure 6.**  
Sa:So ratio



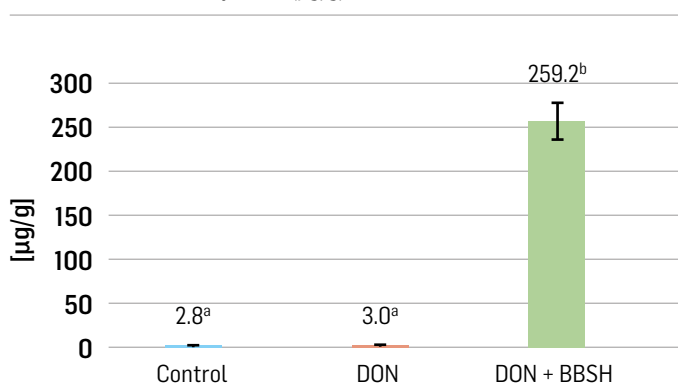
Different superscripts differ significantly  $P < 0.05$ .  
Source: BIOMIN

**Figure 7a.**  
DON-3-sulfate in turkey feces ( $\mu\text{g/g}$ )



Different superscripts differ significantly  $P < 0.05$ .  
Source: BIOMIN

**Figure 7b.**  
DOM-3-sulfate in turkey feces ( $\mu\text{g/g}$ )



Different superscripts differ significantly  $P < 0.05$ .  
Source: BIOMIN

DON in poultry. The resulting de-epoxy metabolite of BIOMIN BBSH 797 activity is DOM-3-sulfate. DON, DOM-1, DON-3-sulfate and DOM-3-sulfate were used as biomarkers in the feces.

In this trial, 15 female ten-week-old turkeys (Hybrid Converter) were randomly allocated to three experimental groups using three double pens with five birds per double pen of the poultry trial facility. Birds were kept for six days in floor pens

## Reference

Wan, D., Huang, L., Pan, Y., Wu, Q., Chen, D., Tao, Y., Wang, X., Liu, Z., Li, J. and Wang, L. (2014). Metabolism, distribution and excretion of deoxynivalenol with combined techniques of radio-tracing, HPLC-IT-TOF/MS and online radiometric detection. *Journal of Agricultural Food Chemistry* 62(2014). pp. 288-296.

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Pinton, P., Tsybulskyy, D., Lucioli, J., Laffitte, J., Callu, P., Lyazhri, F., Grosjean, F., Bacarene, A.P., Kolf-Clauw, M. and Oswald, I.P. (2012). Toxicity of deoxynivalenol and its acetylated derivatives on the intestine: differential effects on morphology, barrier function, tight junction proteins, and mitogen-activated protein kinases. *Toxicol Sci.* 2012 Nov; 130(1): 180-190.

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on wood shavings with free access to feed and water. After the first six days of accustomed, the trial period started for two consecutive days. The diets were artificially contaminated with 1.5 ppm DON, and BIOMIN BBSH 797 was administered via the feed as well. Fecal samples were taken five times per day from each pen. A pooled fecal sample per day and pen was analyzed for toxin residues and metabolites at the Christian Doppler laboratory at IFA-Tulln, Austria. The recorded parameters were the concentration of DON, DOM-1, DON-3-sulfate and DOM-3-sulfate in feces ( $\mu\text{g/day}$ ). DON was only present in small amounts below the limit of quantification and only in the group receiving the toxin without the additive (results not shown). BIOMIN BBSH 797 significantly lowered the load of DON-3-sulfate (*Figure 7a*; green bar) and significantly raised the amount of DOM-3-sulfate detected (*Figure 7b*; green bar). It was clearly demonstrated that the de-epoxidation reaction only took place in the BIOMIN BBSH 797-treated group.

To conclude, the enzymes contained in Mycofix® are an effective, state-of-the-art strategy for the deactivation of non-adsorbable mycotoxins. The fact that biomarker studies have been carried out on turkeys as well is a warranty that the product works efficiently in different animal classes. Purchasing registered products with a proven mode of action *in vivo* is a way to ensure robust production and to make sure that capital is properly invested in a product designed to get the job done!