Understanding gut health and mycotoxin interaction

Although mycotoxins are an unavoidable problem and can have a detrimental effect on gut health and the microbiome, some key management practices can help reduce the risk of such occurrences. Alltech believes that effective mycotoxin management is about seeing the whole challenge, from the farm to the feed mill and from risk assessment to feed management.

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The gastrointestinal tract represents the first barrier against ingested chemicals, feed contaminants and natural toxins. Following ingestion of mycotoxin-contaminated feed, intestinal epithelial cells can be exposed to high concentrations of toxins. This is especially important when considering toxins with poor intestinal absorption, such as fumonisin B1. The biological actions of mycotoxins can cause direct intestinal damage. Trichothecenes affect actively dividing cells, such as those lining the gastrointestinal tract. They directly affect protein synthesis in eukaryotic cells due to the interaction with the ribosomal units, preventing either initiation of protein synthesis or elongation of the polypeptidic chains (Ueno, 1984). It should be noted that the gastrointestinal tract (GIT) is also sensitive to trichothecene-induced apoptosis, affecting mainly the gastric mucosa, gastric granular epithelium and intestinal crypt cell epithelium. The toxic action of trichothecenes results in extensive necrosis of oral mucosa and gizzard lesions. The T-2 toxin inhibits DNA, RNA and protein synthesis in eukaryotic cells, affecting the cell cycle and inducing apoptosis both *in vivo* and *in vitro*. It should also be mentioned that the primary effect of T-2 toxin occurs through contact with the mouth epithelium (beak cavity and tongue).

Direct impact of mycotoxins on the gastrointestinal barrier

Another important effect of some mycotoxins, such as FB1 and ochratoxins A (OTA), is that they alter the barrier function of the intestinal epithelium which is measured as a decrease in the transepithelial electrical resistance. It is likely that the environment surrounding the tight junctions is somehow altered by continuous exposure to FB1. Poults fed grains naturally contaminated with *Fusarium* mycotoxins showed decreased villus height in the duodenum and decreased villus height and villus surface in the jejunum during the starter period. In turkeys fed the same diet contaminated with *Fusarium* mycotoxins, the width and villus surface of the duodenum, villus height and surface of the jejunum and submucosal thickness of the ileum decreased during the grower phase. Broilers fed diets contaminated with 0.5mg deoxynivalenol (DON)/kg had shorter and thinner villi which resulted in lighter small intestines compared to birds fed control diets.

To effectively manage the inevitability of feed mycotoxin contamination, it is crucial to understand the level of mycotoxin challenges.

Research by Weaver in 2020 looked into the impact of chronic intake of naturally multi-mycotoxin-contaminated feed (aflatoxin B1, DON, 3-DON, 15-DON, HT-2 toxin, fusaric acid and fumonisins) with or without titers of yeast cell wall extract (YCWE; Mycosorb A+) on day-old male Cobb chicks. This study suggests that broiler performance and intestinal health are impacted by chronic intake of naturally occurring multi-mycotoxin-contaminated feed. Broilers fed YCWE during the mycotoxin challenge showed improved performance, gut health and efficiency over 42 days. These results indicate that YCWE, particularly at 0.2%, had a protective effect against mycotoxins and improved production efficiency metrics *(Figures 1–4)*.

Mycotoxin impact on gut biochemistry

Aflatoxins fed to broiler chickens decreased pancreatic secretions, whereas aflatoxins fed to layers increased the production of pancreatic enzymes. Intestinal morphology (intestinal crypt depth) and the specific activity of intestinal disaccharidase and maltase were also altered by feeding AFB1. This generates a change in the chemistry of the luminal environment. The bacteria that are most suitable to the new luminal environment will have more chance of successfully multiplying. In addition to the morphological changes induced in the intestinal villi by DON, it is suggested that this mycotoxin inhibits Na+ transport and Na+-D-glucose co-transport in the jejunum of layers. This results in reduced glucose uptake when the intestine is exposed to DON concentrations of 10mg/L. Similarly, in layers, DON affects the intestinal absorption of the amino acids that are co-transported with sodium, such as L-proline. So, the quality and quantity of nutrients available in the intestinal lumen changes as the absorption capacity is altered. It is likely that species of bacteria that can successfully ferment the new 'luminal diet' will predominate in the lumen.

Sub-chronic ingestion of DON, comparable with concentrations occurring in contaminated food and feed, was reported to impair the intestinal transfer and uptake of nutrients.

Black, sticky diarrhoea was reported in a flock of 6,700 laying hens in India after they consumed a feed batch contaminated with FB1 (6.5mg/kg feed) and AfB1 (0.1mg/kg). Hemorrhages of the proventriculus and accumulations of fluid in the intestine were commonly seen in the postmortem examinations. The disease was then experimentally reproduced in day-old chicks and laying hens by feeding the contaminated diet.

Figures 1–4 - Effects of Fusarium mycotoxins on villus height (1), crypt depth (2), villus depth to crypt ratio (3) and goblet cell count in broiler chicks (4) (day 21 and 42).

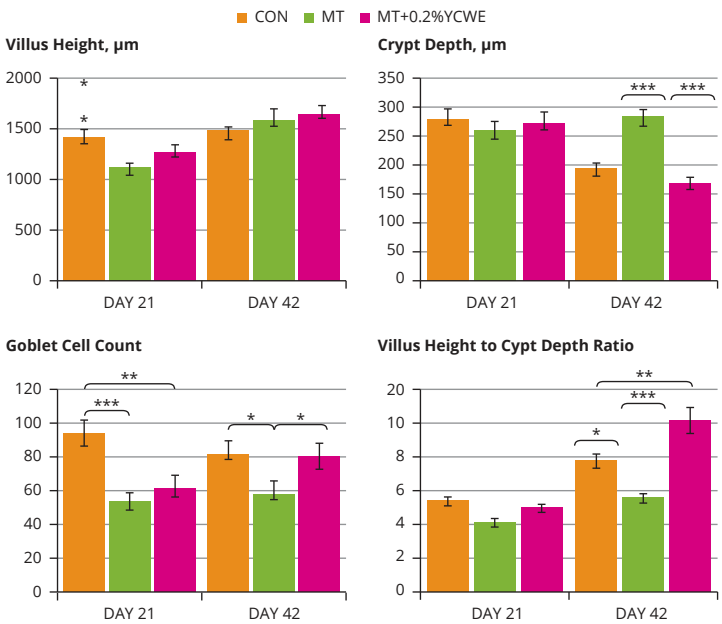

PHOTO: PETER ROEK

To effectively manage the inevitability of feed mycotoxin contamination, it is crucial to understand the level of mycotoxin challenges in the raw materials.

Mycotoxins and infectious agents

In an experimental necrotic enteritis (caused by *Clostridium perfringens*) infection model, broiler chickens fed a diet contaminated with 5mg DON/kg of feed were more prone to developing necrotic enteritis lesions compared to chickens on a control diet. In that particular case, DON acted as a predisposing factor by damaging the intestinal mucosa, leading to leakage of nutrients into the intestinal lumen, therefore providing the necessary growth substrate for extensive proliferation of *C. perfringens*. Another predisposing factor to necrotic enteritis is mucosal damage caused by coccidial pathogens. The interaction between toxins from *Fusarium* with strains of *Eimeria* responsible for coccidiosis in poultry has been investigated. Realistic and occasional doses of *Fusarium* mycotoxins have also shown delayed intestinal recovery, up-regulation of IFN-γ and delayed recruitment of CD4+ and CD8+ cells after *Eimeria* challenges in chickens. Similarly, chickens challenged with strains of *Eimeria* and fed with either individual doses of DON and fumonisins (FUM) or in combination (1.5mg DON/kg and 20mg FUM/kg diet) showed a higher occurrence of lesions in the GIT and more oocysts in the jejunum and excreta compared to only *Eimeria*-challenged birds on the control diet. Further, high (and unrealistic) doses of OTA to broilers and turkeys have resulted in more severe lesion scores and greater incidence of bloody diarrhoea following an *Eimeria* challenge. In addition, typical upregulation of pro-inflammatory cytokines following coccidial infection was stronger in the jejunum of birds fed DON and fumonisin in combination, suggesting an exacerbation of the inflammatory response that might lead to tissue damage.

Holistic solution

To effectively manage the inevitability of feed mycotoxin contamination, it is crucial to understand the level of mycotoxin challenges so that the right steps can be taken to mitigate any adverse effects on animal performance, production efficiency and food safety. Using a combination of powerful management tools, the Alltech Mycotoxin Management Program provides a complete holistic solution to help producers take control of mycotoxin contamination and protect their business. The programme is built around next-generation risk identification technology, data analysis and insights, along with mycotoxin binder solutions designed to reduce the damaging effects of mycotoxins on animal health and production potential.

References available on request.