Do you need help with mycotoxins and the risk they pose to food safety?

The requirements for raw materials used in animal feed are increasing day by day, and these raw materials like cereals are also significant for human nutrition. The food and feed raw materials can encounter many different risks in terms of food and feed safety. At that point, mycotoxins are among the most important safety risk factors in the feed industry.

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Mycotoxins are low molecular weight secondary metabolites produced by fungi that exert adverse effects at low concentrations in animals. The major fungal genera producing mycotoxins are Fusarium, Aspergillus, Penicillium, and Alternaria. These can produce mycotoxins on the growing plants before harvesting or during storage and transportation. Several environmental factors, especially high temperatures, and high moisture contribute to the presence of mycotoxins in feeds.

At that point, the impact of climate change on the presence of mycotoxins in feed is a topic of great concern. According to climate modelling studies, temperatures will continue to increase gradually over time, and the distribution of rainfall is also expected to change, increasing the number of extreme precipitation events. For this reason, everyone involved in every step of animal production should be even more careful about mycotoxins in the future.

The most common mycotoxins in feeds

The most well-known mycotoxins found in animal feed are aflatoxins (AF), ochratoxins (OTA), fumonisins (FUM), deoxynivalenol (DON), T-2 and HT-2 toxins, and zearalenone (ZEA). Fig. 1 shows molecular structures of the main mycotoxins. Aflatoxins are produced by fungi of the genus Aspergillus spp.

The following four types are the most abundant: aflatoxin B1 (AFB1), aflatoxin B2 (AFB2), aflatoxin G1 (AFG1), and aflatoxin G2 (AFG2). Ochratoxins are mainly produced by Aspergillus ochraceus.

However, they can also be produced by other species of Aspergillus (A. carbonarius) and Penicillium (P. verrucosum, P. nordicum). Fumonisins are commonly classified as Fusarium toxins since they can be produced by several species of this genus. The most important toxins are fumonisin B1 (FB1), fumonisin B2 (FB2), fumonisin B3 (FB3), and fumonisin B4 (FB4). Specifically, FB1 has been reported as the predominant and most toxic member of the FB family.

Trichothecenes (TCs) are produced by a wide variety of Fusarium species, and they have chemically subdivided into four types (from A to D).

Types A and B are the most important from the toxicological point of view. Type A comprises mainly T-2 toxin and HT-2 toxin, while type B is frequently represented by DON. The most toxic type A TCs are HT-2 and T-2. The DON is one of the least acutely toxic TCs, however, it is reported at high incidences worldwide. Zearalenone is an oestrogenic mycotoxin produced by several species of Fusarium particularly F. graminearum, and also F. culmorum, F. cerealis, F. equiseti, among others.

Mycotoxin	Feed materials	Max. levels (mg/kg)
[a]AFB1	All feed materials	20
	Complete feedstuffs for poultry (except young animals)	20
	Other complete feedstuffs	10
	Complementary feedstuffs for poultry (except young animals)	20
	Other complementary feedstuffs	5
[b]DON	Cereals and cereal products with the exception of maize by-products	8,000
	Maize by-products	12,000
	Complementary and complete feedstuffs	5,000
[b]ZEA	Cereals and cereal products with the exception of maize by-products	2,000
	Maize by-products	3,000
[b]OTA	Cereals and cereal products	250
	Complementary and complete feedstuffs for poultry	100
[b]FB1 – FB2	Maize and maize products	60,000
	Complementary and complete feedstuffs for poultry	20,000
[b]T2 – HT2	Cereal products	500
	Compound feed	200

Table 1. The EU regulatory levels[a] and established guidelines[b] on mycotoxins in raw materials and in poultry diets.

What must the limits of mycotoxins be in feeds?

Most countries base their regulations on the guidelines established by the European Union (EU) or by the United States Food and Drug Administration (FDA). Guidelines sometimes differ from each other; in most cases, the maximum allowed content of mycotoxins is lower in the regulations given by the EU than in those granted by the FDA.

Table 1 shows the EU regulatory levels and established guidelines on mycotoxins in raw materials and poultry diets.

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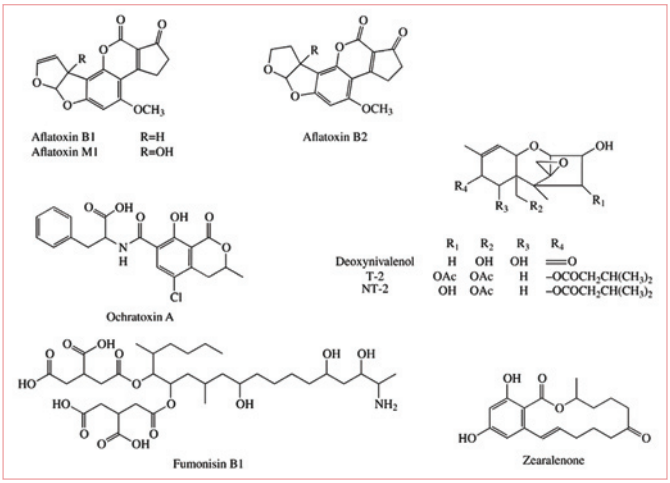
Fig. 1. The molecular structures of the main mycotoxins.



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Mycotoxins and their detrimental effects

The consumption of mycotoxin-contaminated feedstuffs by animals leads to adverse effects on animal health and the effects are more serious in monogastric animals depending on the species and the susceptibility to toxins within the species. Mycotoxins often cause chronic intoxication characterised by growth retardation and decreased productivity in animals with long-term exposure in low amounts.

This often leads to an increase in susceptibility to various infectious diseases, and some frequently encountered parasitic diseases. Mycotoxins may also lead to carcinogenic, mutagenic, teratogenic, immunosuppressive, and hormonal effects in humans and animals.

On the one hand, the occurrence of mycotoxins in feeds may destroy or reduce the nutritional value and palatability of feeds, and thus make the animals refuse to eat. On the other hand, mycotoxins may transfer to the human body through the food chain, such as milk, eggs, and other products, which will cause a severe threat to human health.

When animals are fed with contaminated feedstuffs, they are subjected to enzymatic and microbial transformations leading to the formation of metabolites in the gut. The resulting metabolites can be absorbed into the bloodstream of the animal and later may be excreted through urine and faeces. The toxins that are not excreted from the organism generally remain as residues in edible organs and muscles. For instance, AFB1 is first absorbed in the small intestine, transferred to the blood, and transported by red blood cells and plasma proteins to the liver. AF metabolites in the liver have been used as biomarkers of exposure in animals, especially when evaluating mycotoxin-mitigating agents, where a decrease in liver residues indicates the efficacy of the adsorbent agent tested.

Zearalenone has structural similarities to the female sex hormone oestradiol. This chemical characteristic gives it the capability of binding to oestrogen receptors, causing adverse effects associated with reproductive disorders.

Fig. 2 shows the metabolic process of ZEA in animals. Fig. 3 and Fig. 4 show the liver and kidneys of broilers fed with OTA contamined feeds.

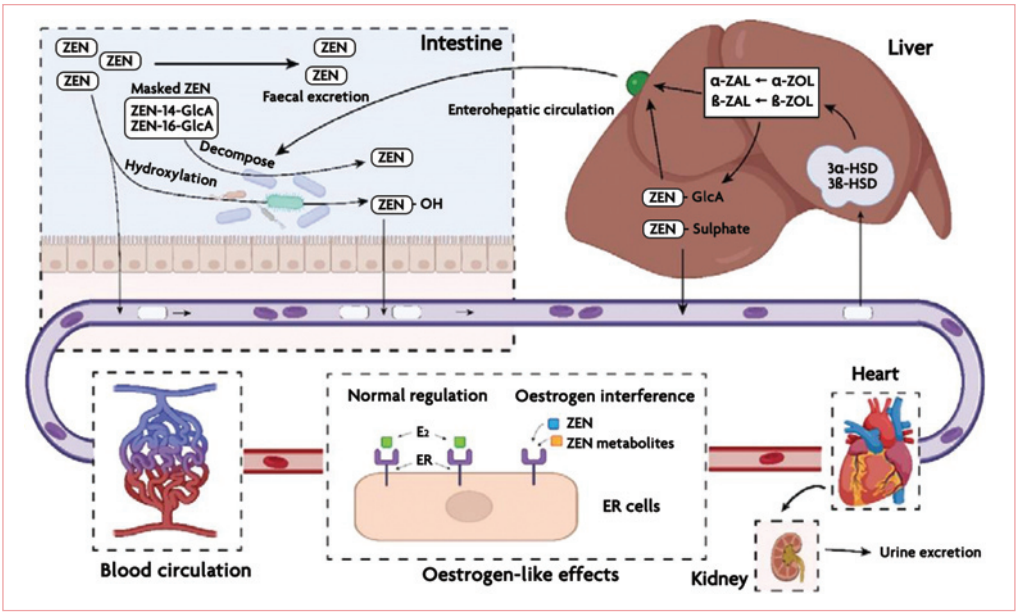

Fig. 2. The metabolic process of zearalenone in animals.
Intestine: After intestinal absorption of ZEA, some part of ZEA is affected by intestinal microbes and is hydroxylated, and the remaining ZEA is excreted with faeces. Liver: Some ZEA metabolism and detoxification products are excreted into the intestines through bile, entering the enterohepatic circulation. The metabolism and detoxification products are absorbed into the blood circulation, and part is excreted in urine and milk. ER cells: ZEA and its metabolites compete for the binding site of E2 and interfere with the normal function of oestrogen. ZAL: zearalano. ZOL: zearalenol. HSD: hydroxysteroid dehydrogenase. GlcA: glucuronic acid. E2: 17-b oestradiol. ER: oestrogen receptor.

How can you detect the mycotoxin types in feeds?

Before dealing with which types of mycotoxins, the feeds should be analysed and determined to mycotoxin types. Various analytical techniques have been developed for their detection. However, chromatographic techniques are usually used as reference methods for mycotoxin analysis.

Approaches for helping the elimination of mycotoxins

Preventing mould growth and mycotoxin contamination in feeds are very significant; however, when contamination cannot be prevented, detoxification is needed before these materials are used. Many approaches such as physical, chemical, and biological methods, have been used to remove, destroy or reduce mycotoxins.

Several technologies have been developed for mycotoxin removal including adsorption, biodegradation, extrusion cooking, and ozonation. Because of the low cost of adsorbents, adsorption has gradually become an economical and feasible method.

Adsorbents have been used to mitigate mycotoxicosis by directly decreasing the mycotoxin bioavailability, and consequently, indirectly reducing the inflammatory response, improving intestinal health, and preventing oxidative stress.

The efficacy of binding mycotoxins is dependent on the crystal structure and physical properties of the adsorbent as well as on the physical and chemical properties of the

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Fig. 3. The liver from broilers fed 0.4mg/kg OTA for 21 days from one day of age. Liver is pale, enlarged and haemorrhagic.

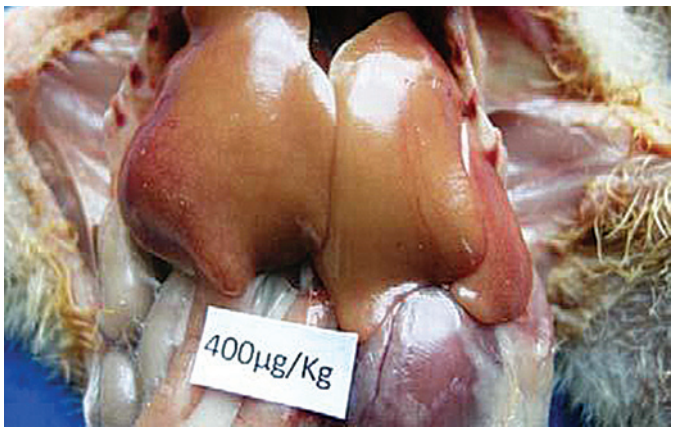
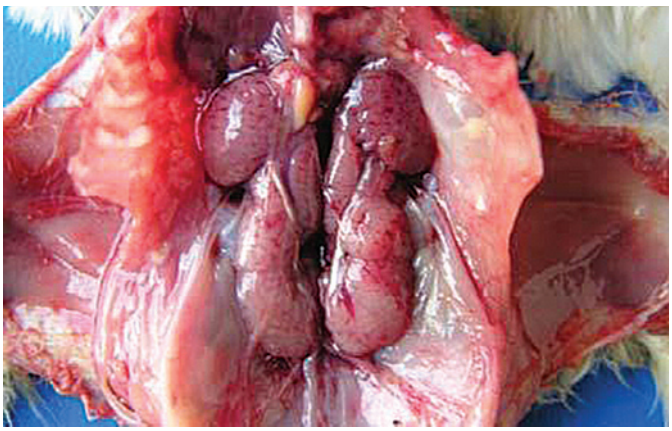


Fig. 4. The kidneys from broilers fed 0.8mg/kg OTA for 21 days from one day of age. Kidneys are swollen, enlarged and bulging out from sockets.



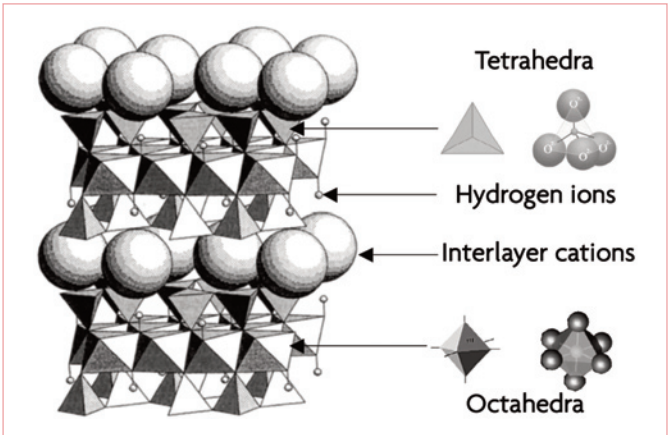


Fig. 5. The basic structure of a clay mineral, showing tetrahedral and octahedral arrangements of atoms in sheets, separated by cations (large spheres) and water in the interlayer between the silicate sheets. Small spheres are H+.

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mycotoxins. Various inorganic adsorbents, such as bentonite, sepiolite, montmorillonite, and clinoptilolite, have been tested and used as toxin binders. Fig. 5 shows the basic structure of a clay mineral.

Using clay minerals for mycotoxin binding involves physical and chemical adsorption.

This feature relies on basic chemistry principles: hydrophobic bonding (complex process involving more than one type of bond), hydrogen bonding, electrostatic interaction of attraction or repulsion, Van der Waals interactions, coordination bonds, and cation exchange.

Other physical characteristics, like specific surfaces, can also play a role in the binding capacity of clay mycotoxin binders. An alternative to inorganic adsorbents for the detoxification of mycotoxins is the use of organic binders, such as yeast cell wall components. Yeast cell wall has shown that their β-D-glucans composition and tridimensional network were able to chemically adsorb mycotoxins in vitro, reduce the absorption of mycotoxins in the small intestine, decrease the accumulation of mycotoxins in specific organs, and increase their clearance, thus protecting the vital organs against mycotoxin exposure.

Fig. 6 shows the yeast cell wall composition.

To help protect the liver health from the detrimental effects of mycotoxicosis, some plant extracts can be used in the toxin binder formulations. In the light of all this information, BAF offer six different toxin binder products from their toxin binder family, according to requirements.

After determining which mycotoxins to fight against, you can choose one of their toxin binding products: Keytox B, Keytox BS, Keytox, Keytox Ultra, Keytox Plus and Keytox Select.

The company conducted research with Selcuk University's Faculty of Veterinary Medicine to determine Keytox's and Keytox Select's in vitro binding capability.

Keytox is specially designed with four different types of clay and Keytox Select is specially designed with the yeast cell wall, three different types of clay, and plant extracts. In this study, different types of mycotoxins were used at pH 3.0 and 6.8, which represent the poultry stomach and intestine environments.

Table 2 shows the mycotoxin binding rates (%) of Keytox and Keytox Select (0.2%) in vitro at pH 3.0 and 6.8. ■

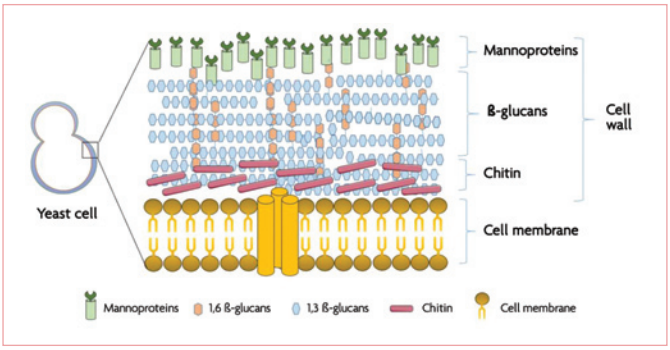

Fig. 6. Structure of yeast cell wall.

References are available from the author on request

Table 2. The mycotoxin binding rates (%) of Keytox and Keytox Select (0.2%) in vitro at pH 3.0 and 6.8.

Toxin binders	Keytox		Keytox Select	
pH	3.0	6.8	3.0	6.8
AF	91.83	84.91	86.20	79.77
OTA	56.41	53.14	74.20	51.70
ZEA	30.74	41.09	31.47	29.57
DON	47.63	31.13	31.47	52.94
FUM	42.73	18.33	54.61	32.21
T2	18.68	27.72	28.11	44.12
HT2	19.31	49.32	41.29	42.33