The impact of mycotoxins in feed and the poultry-specific solutions

The susceptibility of poultry to the negative effects of mycotoxin ingestion have been known for more than 50 years, ever since the so-called 'Turkey X Syndrome' outbreak in 1960 was traced to the presence of the Aspergillus spp. mycotoxin aflatoxin (AFB1) in turkey feed. Since then, many additional mycotoxins have been identified, and the potential threat to bird health and production has become well accepted globally.

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For the poultry industry, AFB1 remains the greatest threat, with poultry more susceptible to AFB1 ingestion than any other livestock species. As evidence of this clear difference in vulnerability has mounted, so too has the demand for solutions to the mycotoxin problem that target the specific threat and physiological differences of each individual species.

Identifying mycotoxin threats

Of primary concern to poultry producers are the mycotoxins produced by Aspergillus moulds – including the ochratoxins (OTA) as well as AFB1 – which regularly develop during the storage of feeds and feed ingredients, as well as infecting growing crops.

Any time feeds are exposed to moisture, whether from rain, humidity or run-off, the risk of Aspergillus growth is increased. With many key monogastric feed ingredients produced in regions of the world known to suffer from high humidity, the risk of OTA and AFB1 contamination can be considered to be permanently high.

Both AFB1 and OTA are also highly toxic for poultry (Table 1).

This is in stark contrast to pigs, for example, which are at greatest risk from mycotoxins produced by Fusarium fungal diseases, such as deoxynivalenol (DON), zearalenone (ZON), fumonisin (FUM) and T2 toxin.

Toxin	Poultry	Swine	Ruminant
Aflatoxins	+++	++	+
Ochratoxins	+++	+	+
T2 toxin	++	+++	+++
Deoxynivalenol	+	++	++
Zearalenone	+	+++	++
Fumonisin	+	+++	+

Table 1. Relative toxicity of different mycotoxins on different livestock species (+ = mild toxicity, ++ = moderate toxicity, +++ = high toxicity).

However, commercially-produced poultry in modern production units can still be exposed to considerable levels of Fusarium mycotoxins in feed due to the heavy reliance on cereal grains.

In a survey of poultry diets from across Europe, the Middle East and Russia carried out by Micron Bio-Systems between 2014 and 2015, 96-97% of samples contained DON, FUM and ZON, with DON in particular found at extremely high levels that averaged over 900ppb.

Although no AFB1 or OTA were found in these particular samples, the risk may be higher in other regions of the world. It is also worth remembering that poultry diets rely heavily on human-edible ingredients – the more stringent mycotoxin limits for human consumption mean that rejected feed batches often find their way into poultry feed.

Impact on performance

The effects of these mycotoxins on poultry health and performance vary considerably (Table 2).

AFB1 is a carcinogenic compound that is known to affect gene regulation and metabolism at the cellular level, the symptoms of which include liver damage, development of fatty liver, immune suppression and reduced growth rates.

In contrast, OTA is associated with renal dysfunction and kidney damage, and is reported to affect weight gain, feed intakes and immune function.

Both can cause increased mortality where levels of exposure are high. Although poultry are considered relatively resistant to the Fusarium mycotoxins, the potential threat should not be overlooked completely, since the possible negative effects of ingestion have not been as extensively researched as those for AFB1 or OTA.

Ingestion of ZON in particular, which is an oestrogen mimic, may impact fertility and egg production in breeder and also layer units.

In addition, even low levels of DON – the mycotoxin most frequently found in feed – and T2 toxin are known to damage the lining of the small intestine and reduce nutrient absorption. This has the potential to negatively affect feed conversion efficiency, feed intakes and liveweight gain. Along with FUM, these mycotoxins also impair immune function, increasing susceptibility to disease.

Species-specific challenges

This potential impact on immune function is critical in poultry, with the breeder stage of the production cycle perhaps the most important when it comes to managing the risk of mycotoxin ingestion.

This is due to the fact that in addition to the direct effects on breeders, which include increased embryonic mortality and diarrhoea, plus reduced hatchability, feed intakes and body weight gain, the consumption of contaminated feed by breeders can cause immune dysfunction,

Continued on page 9

Continued from page 7
reduced body weight and abnormalities in their progeny.

Poultry producers also need to consider the role that mycotoxins may play in intestinal disorders. There is evidence to suggest that some mycotoxins can predispose poultry to other diseases, such as Clostridium perfringens-induced necrotic enteritis and coccidiosis, with subsequent negative effects for growth rates, feed conversion and mortality.

Symptoms	Mycotoxins				
	Aflatoxin	Fumonisin	Trichothecenes (DON/T2)	Zearalenone	Ochratoxin A
Weight gain/growth rate	✓	✓	✓	✓	✓
Egg production	✓		✓	✓	✓
Egg quality	✓		✓	✓	
Fertility				✓	
Disease susceptibility	✓		✓		✓
Liver/kidney damage	✓	✓	✓		✓
Diarrhoea			✓		
Mortality	✓				✓
Vaccine efficacy	✓				✓
Feed efficiency	✓		✓		
Feed consumption			✓		✓

Table 2. Negative effects of different mycotoxins in poultry.

Mycotoxin remediation strategies

With these mycotoxin threats now widely recognised, the use of in-feed mycotoxin deactivators and binders has increased markedly in recent years. Designed to bind, transform or degrade mycotoxins, there is a wide range of such products available commercially.

Binding (adsorption) is the most common approach within the feed industry, with clay minerals such as bentonite used to bind with polar mycotoxins (AFB1) and yeast cell walls demonstrating some efficacy against non-polar Fusarium spp. mycotoxins. The ability to maintain efficacy within the pH range typically found in the bird's gastro-intestinal tract (pH 3-7) is critical to success, and can vary considerably between different binders.

An additional strategy, particularly for non-polar mycotoxins, is to remove or modify a particular functional site on the surface of the mycotoxin.

This transformation can render the mycotoxin harmless, or expose the binding site of the molecule to mineral binding agents.

Finally, degradation is the application of multiple transformations to ensure that any mycotoxin fragments remaining after transformation – even if bound to a mineral binder – do not retain any toxic effect.

For poultry producers, the strategy of transformation and degradation is critical, since it is the most effective in eliminating the effects of DON, which is one of the most prevalent mycotoxins in poultry feeds.

Targeted poultry solutions

The most effective approach is typically a combination of all three strategies and it is clear from the information above that it is the specific nature of the mycotoxin threat that will dictate how such strategies should be prioritised.

It also highlights just how important it is to understand the differences between species, both in terms of exposure and vulnerability. Such differences are already recognised by key regulatory authorities. In the EU, for example, the maximum permitted limit for FUM in poultry is 20ppm and there is no specific limit for ZON, whereas in pig feed the limits for FUM and ZON are set at 5ppm and 0.1ppm, respectively.

The recent development of species-specific in-feed mycotoxin solutions such as Ultrasorb P therefore represents a major advance in mycotoxin remediation, and is already leading the way towards potential customised, bespoke solutions based on individual farm mycotoxin profiles.

Although reliant on effective and timely testing of feed samples for actual mycotoxin loading, the potential benefits from such a flexible, yet highly targeted, approach are likely to generate considerable interest in years to come. ■