Common mycotoxins, their interactions and effects on poultry production

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Mycotoxins are secondary metabolites of fungi that grow on a variety of grains, feedstuffs and animal feed.

Feed containing moderate to high levels of mycotoxins cause clinical symptoms in most animals, ranging from organ damage to acute mortality. Consumption of low levels of contaminated feed may result in subclinical problems such as immunosuppression.

More than 500 mycotoxins have been identified to date. However, only a few are regularly tested for in the field. Most poultry operations regularly test for aflatoxin B1 and less frequently ochratoxin A, deoxynivalenol (DON or vomitoxin), T-2 toxin, zearalenone and fumonisins. Thin layer chromatography (TLC) and ELISA are methods commonly used for these tests at feed mills and laboratories.

Since one mould can produce several mycotoxins and several moulds often exist simultaneously in any given feedstuff, often there are more mycotoxins present than are being tested. If a sample contains DON for example, it is likely that several other mycotoxins including 3-acetyl DON, 15-acetyl DON, fusarenon-X and DON-3-glucoside may also be present. These toxins can contribute to the toxicity of DON so failure to test for them will give an inaccurate picture of mycotoxin contamination levels. The same applies for aflatoxins, T-2 toxin related compounds, fumonisins and ergot toxins.

There are only a few laboratories in the world capable of broad spectrum analysis. Commercially accessible multiple mycotoxin analysis assists in greater understanding of the overall risk to animals as well as allowing for the development of improved mycotoxin management solutions.

Alltech's 37+ Program uses UPLC-MS/MS technology to analyse more than 37 mycotoxins from a single sample and considers the mycotoxin challenge present in each sample as a whole, rather than looking at the individual mycotoxins present. This way it more closely reflects commercial production and the challenges facing producers around the world. Since all species and life stages of birds are susceptible to mycotoxins to varying degrees, samples analysed through the 37+ Program are also given a Risk Equivalent Quantity (REQ), one number which takes into account the cumulative effect of mycotoxins and measures the overall risk to the target animal group.

Through its 37+ 2013 global survey of poultry feed, Alltech has found that multiple mycotoxin contamination is the reality facing today's poultry producers (Fig. 1).

Globally, 87% of samples tested were contaminated with fumonisins, followed by Type B trichothecenes, aspergillus mycotoxins (gliotoxin, sterigmatocystin and verruculogen) and ergot toxins – 73%, 66% and 51% respectively. Aflatoxins, Type A trichothecenes and zearalenones were less prevalent (Fig. 2).

Mycotoxins	Interaction	References
Aflatoxin and Ochratoxin A	Synergistic	Huff and Doerr (1981), Raju & Devegowda (2000)
Aflatoxin and DAS	Synergistic	Kubena et al. (1993)
Aflatoxin and DON	Additive	Huff et al. (1986)
Aflatoxin and T-2 toxin	Synergistic	Huff et al. (1986)
Ochratoxin A and T-2 toxin	Additive/ Synergistic	Kubena et al. (1989a)
Fumonisin B1 and Moniliformin	Additive	Javed et al. (1993)
Fumonisin B1 and T-2 toxin	Additive	Kubena et al. (1995), Kubena et al. (1997)
Fumonisin B1 and DON	Additive	Kubena et al. (1997)
DON and T-2	Synergistic	Kubena et al.(1989b)

Table 1. Mycotoxin interactions in poultry.

Mycotoxin interactions

Mycotoxin growth can be attributed to factors such as geographical location, climatic conditions, agricultural practices and trade of feed ingredients. Interaction of these mycotoxins in the animal often leads to a more adverse case of mycotoxicosis than is anticipated and can alter clinical signs of individual mycotoxins making field diagnosis very complex. The interactions can be additive, synergistic or antagonistic (Table 1).

- Reduced feed intake
- Reduced weight gain
- Increased feed conversion
- Reduced egg production and reduced egg weight
- Poor egg shell quality
- Poor internal egg quality
- Poor fertility
- Reduced hatchability
- Increased mortality
- Reduced immune response (including poor antibody titers)
- Organ damage (liver, kidney, spleen, gizzard, thymus, bursa Fabricius etc)
- Meat decolouration
- Skeletal abnormalities (tibial dyschondroplasia, articular gout etc)

Table 2. Common production problems and symptoms in poultry production caused by mycotoxins.

Effects in poultry

One of the biggest challenges with mycotoxicoses is the non-specific nature of symptoms in poultry. These can be similar to those arising as a result of poor management,

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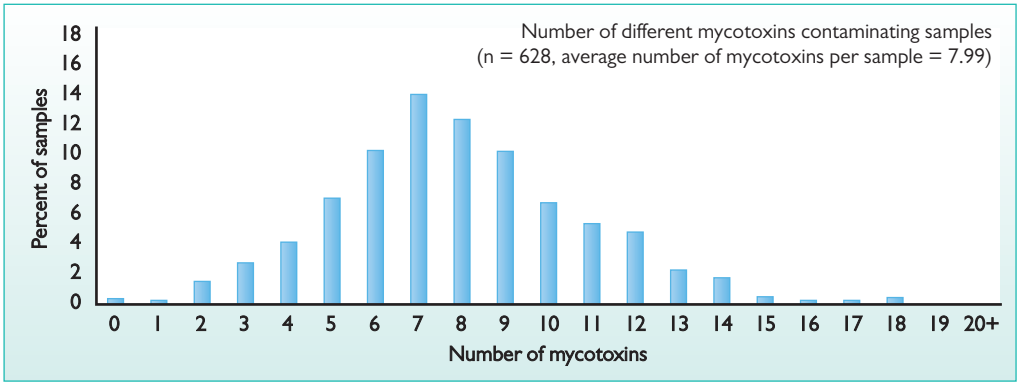
Fig. 1. Results of Alltech's 37+ 2013 global survey of poultry feed.



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nutrition and health status. Hence it is quite common under commercial conditions to compare the results of mycotoxin testing in poultry feed with the symptoms on the farm to confirm mycotoxicoses.

Some of the most common symptoms of mycotoxins in three classes of birds are shown in Table 2.

Turkey poults are more sensitive to fumonisins when compared to chicks. Experimental fumonisin toxicity resulted in enlargement of the kidney and liver and variation in the size of the pancreas, proventriculus and gizzard, as well as atrophy of lymphoid organs.

Multifocal necrosis in liver, hyperplasia of bile ductus, intestinal villus size reduction and hyperplasia of goblet cells can be observed upon histological examination of the gastrointestinal tract. Also it has been reported that Fumonisin B1 causes depletion of thymic lymphoids, toxicity of macrophages and a reduction in mitogenic responses.

Trichothecenes mycotoxicosis can result in caustic and radiomimetic effects. Contact with these mycotoxins can result in feed refusal and necrosis of oral mucosa and skin.

Trichothecenes can cause acute diseases of the digestive tract characterised by reddening of gastrointestinal mucosa, immunosuppression and bone marrow alteration. Spleen atrophy, gall bladder distention, mottling liver and visceral haemorrhages are other common symptoms caused by trichothecenes.

Intake of contaminated feed also results in reduced growth, digestive disorder, pigmentation problem, rickets, feathering abnormality and nervous disorders.

In layer hens T-2 and HT-2 toxins can result in a rapid decrease in egg production as well as thin-shelled eggs shortly after consumption of contaminated feed.

Feed refusal, depression, comb and wattle cyanosis as well as atrophy of the ovary and oviduct are common. Yellowish, thick crusts may be seen on ulceration of mouth mucosa. Rough and thickened gizzard lining, urates deposits in ureters, swollen kidneys and friable livers may also occur in the flock.

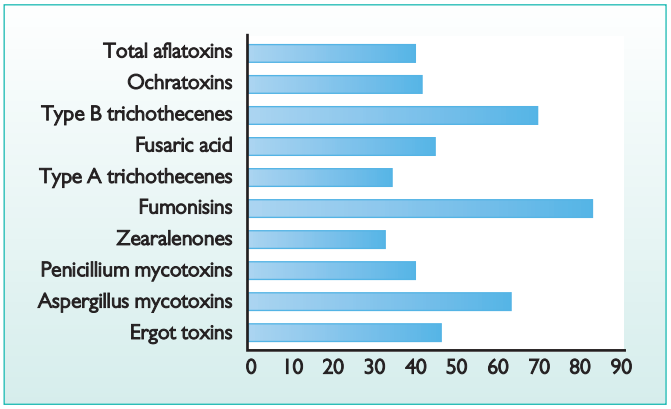

Fig. 2. Results of Alltech's 37+ 2013 global survey of poultry feed.

		OA				
		0ppm		2ppm		
		T-2		T-2		
		0ppm	3ppm	0ppm	3ppm	
AF	0ppb	1.31^{ax}	1.14^{ax}	1.10^{ax}	1.22^{ax}	0.04
	300ppb	1.10^{ax}	1.11^{ax}	1.19^{ax}	0.84^{by}	0.05
	SEM	0.06	0.08	0.06	0.07	

Table 3. Additive interaction among aflatoxin B1 (AF), ochratoxin A (OA) and T-2 toxin (T-2) for their effects on bursa weight in commercial broilers at five weeks of age.

Toxicosis associated with ergot alkaloids can be characterised by decreased feed intake and growth, necrosis of the comb, beak and toes and diarrhoea. Vesicular dermatitis on the wattle, comb, face and eyelids may also be evident especially in layers. Permanent atrophy of combs and wattles are other symptoms of ergotism in poultry.

Aspergillus mycotoxins including sterigmatocystin, verruculogen and gliotoxins are less toxic than aflatoxin but can be produced in higher concentrations. In layer hens feed intake and egg production can be reduced and irregular pigmentation may occur on brown eggs. Other clinical findings of Aspergillus toxins are pale and fatty livers containing haemorrhages.

Experimental toxicosis with sterigmatocystin in leghorn chicks resulted in liver, pancreas, lymphoid organs and kidney damage. In breeders it caused decreased weight of embryo, malformation and embryonic mortality.

Patulin is a mycotoxin produced by several species of Aspergilius moulds, Penicillium and Byssochlamys.

It has comparatively lower toxicity for young poultry however it causes haemorrhage in the proventriculus, gizzard and intestinal lumen. It also increases the number of thin shelled misshapen eggs in layer hens. The immunosuppressive effects of mycotoxins are well known, particularly the reduction of immune organ weights and immune response (including antibody titers) against important viral disease in poultry.

It has been reported that aflatoxin B1 and ochratoxin and their combination have a negative effect on cell mediated immune response and haemagglutination inhibition (HI) titers against sheep red blood cells in broilers.

Raju and Devegowda (2002) demonstrated that aflatoxin B1 (AF), ochratoxin (OA) and T-2 toxin (T-2) led to a reduction of bursa of Fabricius weights when fed individually. Additionally they observed significant further reduction when birds were given feed with a combination of these three mycotoxins (Table 3).

Immune response to vaccinations for Newcastle disease (ND) and infectious bursal disease (IBD) was significantly reduced in five week old commercial broilers when fed the same toxins. Esterified glucomannan significantly improved reduced antibody titers against ND and IBD vaccines (Figs. 3-4).

Conclusions

Poultry are sensitive to mycotoxins and sensitivity varies from toxin to toxin. Understanding mycotoxicoses in poultry is complicated by the occurrence of multiple mycotoxins in finished poultry feed, mycotoxin interactions and non-specific symptoms. Broad spectrum mycotoxin detection that is commercially available offers producers a more accurate understanding of the overall risk to their birds and the steps they can take to minimise its impact on animal health and performance.

The use of a proven, broad spectrum mycotoxin binder such as yeast cell wall based esterified glucomannans is highly recommended to negate the damaging effects of mycotoxins on bird health. ■

Fig. 3. Main effects of aflatoxin B1 (AF), ochratoxin A (OA), T-2 toxin (T-2) and esterified glucomannan (E-GM) on antibody titers against Newcastle disease in commercial broilers.

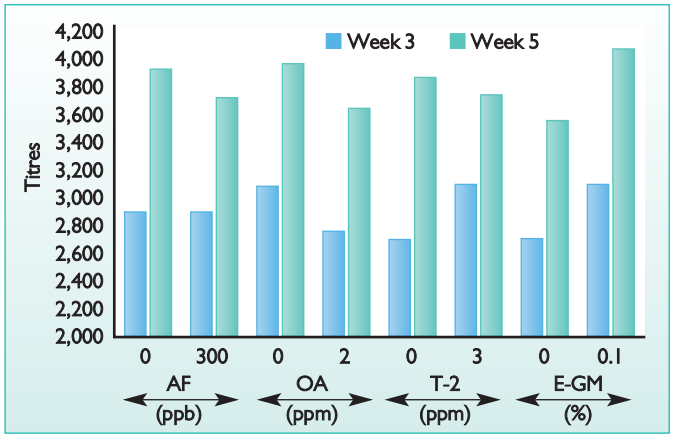

Fig. 4. Main effects of aflatoxin B1 (AF), ochratoxin A (OA), T-2 toxin (T-2) and esterified glucomannan (E-GM) on antibody titers against infectious bursal disease in commercial broilers.