

Why poultry breeders need to be concerned about emerging mycotoxins

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Mycotoxins are often referred as 'hidden killers', 'silent killers' or 'thieves' for several reasons. They are 'hidden killers' because it is hard to detect them although we are sure about their existence; they are 'silent killers' because it is hard to identify their negative effects; they are 'thieves' because they steal our profits. This is particularly true for breeder feeds as we believe that we always make extra efforts to provide best quality feed to the breeders.

Not too long ago, our mycotoxin testing ability was limited to aflatoxins. Later on ochratoxin A, T-2 toxin, zearalenone, deoxynivalenol (DON) and fumonisin B1 were added to the testing list, respectively.

Now we know there are 500 of them and therefore, efforts should be made to analyse as many as possible. Such elaborate testing under a commercial setting would allow us to understand the role of emerging mycotoxins in addition to those already known.

Unveiling emerging mycotoxins

There are a few laboratories in the world capable of analysing 50 or more mycotoxins. Such analyses, however, are restricted to research and regulatory purposes.

To bring such capability to the field and to assist animal producers around the world, Alltech established the 37+ Program. As the name indicates, this program makes use of

Mycotoxin group	Routine mycotoxin analysis	37+ program
Aflatoxins	Aflatoxin B ₁	Aflatoxin B ₁ , B ₂ , G ₁ , and G ₂
Ochratoxins	Ochratoxin A	Ochratoxin A and B
Type A trichothecenes/ T-2 Group	T-2 toxin	T-2 toxin, DAS, HT-2 toxin, neosolaniol
Type B trichothecenes/ DON Group	DON	DON, 3-acetyl DON, 15-acetyl DON, Nivalenol, Fusarenon-X, masked DON
Fumonisin	Fumonisin B ₁	Fumonisin B ₁ , B ₂ , and B ₃
Zearalenone group	Zearalenone	Zearalenone, α -zearalenol, β -zearalenol and zearalanone
Other penicillium and aspergillus mycotoxins	-	Patulin, roquefortine C, penicillic acid, mycophenolic acid, gliotoxin, sterigmatocystin, verruculogen, wortmannin
Ergot mycotoxins	-	2-bromo-alpha-ergocryptine, ergocornine, ergometrine, ergotamine, lysergol, methylergonovine
Alternaria toxins	-	Alternariol
Total number analysed	6	38

Table 1. Comparison of routine mycotoxin analysis versus 37+ program.

state of the art UPLC-MS/MS techniques to analyse more than 37 mycotoxins.

Table 1 compares the list of mycotoxins that are part of the 37+ Program with those analysed routinely in the poultry industry. For ease of understanding, I have considered the additional mycotoxins in the 37+ Program as the emerging mycotoxins.

Let us take an example of Type B trichothecene group/DON group to better

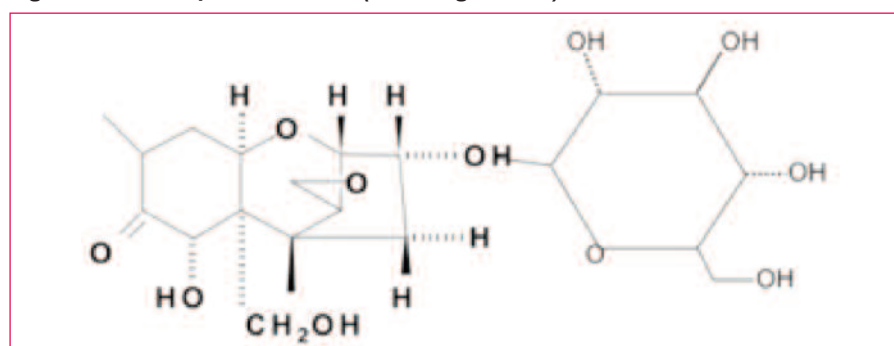
understand the topic of emerging mycotoxins (Table 1). Many studies have shown that when grains/feeds contain DON, other mycotoxins such as 3-acetyl DON and 15-acetyl DON are also present. Since we do not analyse them routinely, they can be considered as emerging mycotoxins. Masked/bound DON is another important aspect to consider as this form of DON cannot be detected using conventional methods of mycotoxin analysis, including HPLC (Fig. 1). As soon as the masked DON reaches stomach/intestine, the enzymatic activity release DON molecule from its bound form making it toxic to birds.

Toxicity of trichothecenes

In Table 1, we can see two groups of trichothecene mycotoxins – Type A and Type B. Taken together, 10 different trichothecene mycotoxins are analysed in the 37+ Program as opposed to only two in the rou-

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Fig. 1. Structure of masked DON (DON-3-glucoside).



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tine analysis. All 10 mycotoxins can interact with each other to increase overall toxicity.

The failure to analyse these eight additional mycotoxins can underestimate the potential toxicity to breeders. Moreover, DAS, which is seldom analysed, is about three times more toxic to poultry than T-2 toxin. Table 2 shows results from three different peer-reviewed research trials wherein breeders were fed varying levels of fusarium mycotoxins. In trials 2 and 3, the negative effects observed are certainly due to the combination of mycotoxins and not just DON. The same is the case in Trial 1, but the multiple mycotoxin analysis was not performed on experimental diets.

The results also indicated that the reproduction parameters are much more sensitive to fusarium mycotoxins than body weight, feed efficiency and egg production.

Fusarium mycotoxins, even at concentrations that do not result in an obvious, adverse effect on performance, can be subtle modulators of immunity. Fusarium mycotoxins have been shown to interfere with the restoration of cell-mediated immunity following coccidial infection in broiler breeder pullets.

Decreased immune response to vaccines has also been observed in breeders.

Antibodies to Newcastle disease virus (NDV) decreased in layers exposed to DON or to multiple fusarium mycotoxins. Although fumonisins are not well studied in breeders, they are capable of affecting the immunity. Despite indications that zearalenone is not very toxic to poultry, there are many field cases of reproductive disorders pointing finger at this oestrogenic mycotoxin. Table 1 show three other forms of zearalenone which are not analysed currently in the poultry industry. It is recommended that further research is conducted to determine the collective negative effects of all four forms of zearalenone.

Other emerging mycotoxins

Apart from fusarium mycotoxins, many aspergillus, penicillium, alternarium and

Sl. No.	Mycotoxin source	Mycotoxin conc. (ppm)	Period of exposure (week)	Effects
1	Corn purposely infected with <i>F. graminearum</i>	DON: 38	58-63	Decreased yolk percentage
2	Naturally contaminated oats	DON: 2.5-4.9; 3-acety-DON: 0.25-0.63; ZEN: 0-0.55	21-28	Increased incidence of unwithdrawn yolk sac, cloacal atresia, cardiac anomalies and delayed ossification
3	Naturally contaminated wheat and corn	DON: 12.6; 15-acety-DON: 1; ZEN: 0.6	27-38	Transient reduction in egg production, increased early embryonic mortality, decreased eggshell thickness, decreased antibody titers to infectious bronchitis disease

Table 2. Effects of Fusarium mycotoxins on breeder performance (Moran et al., 1987; Yegani et al., 2006; Bergsjø et al., 1993).

ergot mycotoxins, which are not routinely analysed, are also part of the 37+ Program. The toxicity of such emerging mycotoxins, although not very well understood at this stage, can certainly contribute to the total mycotoxin toxicity.

Penicillium mycotoxins can increase the toxicity of aflatoxins and ochratoxins, while ergot toxins can be an issue in countries such as Australia.

Mycotoxin management

In light of the unexpected toxicity from emerging and unknown mycotoxins, the use of a HACCP-like approach to control mycotoxins in feed mills and farms is highly recommended. Such a programme allows the control of factors such as moisture and water activity of grains, temperature and relative humidity of the environment. Better aeration of storage silo structures and frequent cleaning of feed mill equipment are also desirable to control mycotoxin production.

This is the basis of Alltech's MIKO Program, a comprehensive and integrated approach to reduce the incidence of mycotoxicoses in poultry.

Alltech's three-pronged Mycotoxin

Management Program tackles the subject of mycotoxin contamination as a whole – from identification (37+ Program) to risk/feed management (MIKO) to remediation.

Mycotoxins are ubiquitous. Their nature lends itself to growth in such a wide variety of conditions that irrespective of the lengths producers go to in terms of minimising the risk or level of contamination it is inevitable.

As such it is recommended that producers include a proven broad spectrum binder into the diets of their birds to minimise their impact on animal health and performance.

Conclusions

The role of lesser known and emerging mycotoxins, along with routinely analysed mycotoxins, should be considered in determining the total toxicity to poultry. This is particularly important in breeders as they are subjected to chronic toxicity and their reproductive system is much more sensitive to mycotoxins.

The non-specific symptoms and subtle nature of the mycotoxin challenge warrants the implementation of mycotoxin prevention steps all along the mycotoxin production chain rather than waiting for the devastation to happen. ■