Mycotoxins and their impact on your breeder flock

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It is a fact that feed represents 40-70% of costs in poultry production. Even if the amount of feed needed to grow a day old chick (DOC) into a finished broiler is around 12 times higher than that needed to produce a DOC – and because of that some might say mycotoxins are no real issue in breeders – the fact is that while a broiler takes approximately 40 days to grow, a breeder will be exposed to these toxic substances found in the feed for much longer. And how valuable is a breeder in comparison to a broiler? And what are the consequences of this exposure to the offspring?

All people dealing with animal production will have heard the term mycotoxins, these ubiquitous toxic substances produced by toxigenic moulds (Table 1).

Mycotoxins have a great negative impact on the performance and health of the animals ingesting them. When mycotoxins exist in breeder flocks, several aspects must be taken into account which will have a negative impact on the final productivity of the animals.

General problems such as decreased feed intake, feed refusal, egg production, egg weight, egg shell quality, hatchability and higher mortality rates are well described in literature and are known to occur in the presence of mycotoxin contaminated feeds. However, there are some other problems which may exist in a farm in which mycotoxins are (mistakenly) not considered to have an impact.

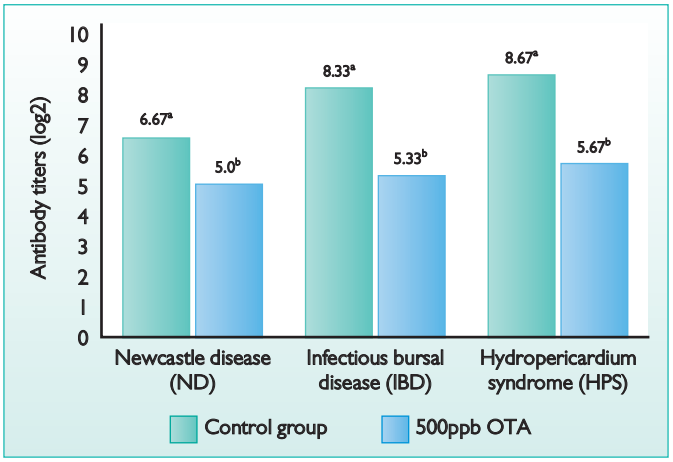

Fig. 1. Antibody titer response in broilers fed ochratoxin A contaminated diets.

Mouth and tongue lesions

Type-A trichothecenes such as T-2 toxin exert an inflammatory response in the mouth that often progresses to necrosis and invasion by normal microbial flora.

Lesions can vary from the so-called 'black tongue' in which usually the tip of the tongue is necrotised without further inflammation of the oral cavity or oesophagus, to raised, yellow necrotic plaques.

Therefore, in flocks where this condition is observed it might be worth investing in type-A trichothecene analyses as a differential diagnosis tool.

Toxin transference

Although found at very low levels to represent a danger to human health, mycotoxins might be a concern for producers who care about the quality of products they deliver, those being eggs for human consumption or DOC.

In the latter case, thoughts must be focused on the viability of the offspring. Scientific reports mention the incidence of chick development abnormalities together with unabsorbed yolk sac and delayed ossification in birds fed DON contaminated diets, although no signs are detected in the breeders. Also, in the case of aflatoxins and their metabolites, studies exist evidencing their accumulation in genitals and their transference to the egg and progeny.

Likewise, metabolites of zearalenone and T-2 toxin have also been reported to be transferred into the egg, with the same associated risks. Other occurrences, which interestingly might be connected with the ingestion of mycotoxin contaminated diets, are, for example, the increased incidence of soft-shelled eggs in hens fed diacetoxyscirpenol (DAS) alone or in combination with T-2 toxin.

Immunosuppression

Another aspect closely related to the transference of mycotoxins into eggs and malabsorption of the yolk sac is actually the immunity of the offspring as the immune system is a key target of several important mycotoxins.

Salmonellosis and candidiasis outbreaks were associated with the outbreak of aflatoxicosis in the 1960s, when aflatoxins were first identified.

Moreover, other mycotoxins such as the trichothecenes group are good modulators of the immune system due to their capacity to inhibit protein synthesis.

Regardless of the mode of action on how mycotoxins damage the immune system – either by affecting cellular responses or humoral factors, the net result is often the impaired resistance to infectious agents.

The money lost with the reduced productivity and irregular results, increased mortality and susceptibility to diseases due to mycotoxins is greater than that invested in prevention.

Proof of that are the numerous papers reporting increased susceptibility to diseases such as pasteurellosis, coccidiosis, Marek's disease, salmonellosis, infectious bursal disease (IBD), infections by staphylococcus, listeria, mycobacterium and infectious bronchitis, amongst others. Interestingly, other studies report the impact of mycotoxins in poultry antibody titers.

Fig. 1 shows the decrease of the antibody titers against Newcastle, IBD and Hydropericardium syn-

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Table 1. Major mycotoxin producing fungi and respective mycotoxins produced.

Major mycotoxin producing fungi (amongst others)	Major mycotoxins produced (amongst others)
A. flavus A. parasiticus A. ochraceus	Aflatoxin (B_1, B_2, G_1, G_2) Ochratoxin (Ochratoxin A) (OTA)
F. verticillioides (syn. F. moniliforme) F. graminearum F. pseudograminearum F. culmorum F. poae	Fumonisins (B_1, B_2, B_3) Zearalenone Type-A Trichothecenes: T-2 toxin, HT-2 toxin, diacetoxyscirpenol Type-B Trichothecenes: Nivalenol, deoxynivalenol (DON), fusarenon-X, acetyl-deoxynivalenol

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drome in broilers experimentally fed ochratoxin A in comparison with the control group.

Risk management

After the acknowledgment that mycotoxins are a major hindrance to successful production, mycotoxin risk management is crucial to eliminate the effects of mycotoxins and their toxicity.

Binders are commonly referred to as substances used in animal feeds which will adsorb mycotoxins in the gastrointestinal tract, thus impeding them to be absorbed by the animal.

However, while binders may be successful in the case of adsorbable mycotoxins, such as aflatoxins, for others, such as zearalenone, trichothecenes, ochratoxins and fumonisins their efficacy is much reduced or even zero. The use of different strategies combined in one product such as the Mycofix Product Line has proven to be very successful in vitro, in vivo and under the pressure of commercial production.

Together with adsorption, biotransformation enables the conversion of non-adsorbable mycotoxins into non-toxic metabolites by means of micro-organisms and their enzymes.

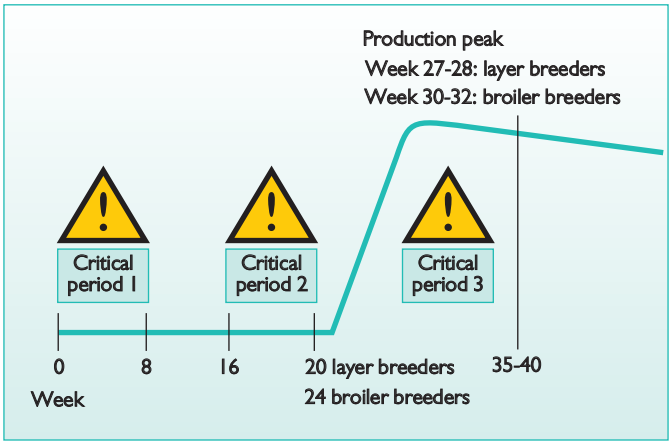

Fig. 2. High risk periods in breeders.

Furthermore, bioprotection provides the hepato-protection and the immune support needed when animals are confronted with these toxic substances.

When to act?

Whilst it is true that prevention should be done on a continuous basis since the mycotoxin threat is permanent, there are some critical periods during a breeder production cycle where their risk management should be stricter.

These periods refer to those when animals, because of their stage and largely due to the stress that they are subjected to, are mostly sensitive to the hazardous effects of toxins (Fig. 2).

Until eight weeks of age young animals are the most sensitive to mycotoxins, even though their feed intake is low.

Later on, the risk is that pullets do not maintain feed intake due to formulation changes, where mycotoxins are a threat. If the necessary laying weight is not reached then the achievement of sexual maturity might be jeopardised.

Finally, the last critical period comprises the time from start of laying until after the laying peak, just following the stress of transfer, when animals are under very high production demand and still growing to reach their mature bodyweight.

Conclusion

Management of the risk of mycotoxins must be done on a preventative basis rather than focusing on treatment.

More frequently than not, the money lost with the reduced productivity and irregular results, increased mortality and susceptibility to diseases due to mycotoxins is greater than that invested in prevention. The use of a good mycotoxin risk management tool, such as the Mycofix Product Line, is of utmost importance to guarantee a successful and stable production with fluctuating feed quality in terms of mycotoxin contamination. ■

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