

# Mycotoxins problems in broiler breeders

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**M**ycotoxins are toxic secondary metabolites produced by toxigenic strains of some genera of moulds. In particular, mycotoxins are polyketones compounds resulting from condensation reactions produced under specific physical, chemical and biological conditions that occur when the reduction of the ketone groups in the biosynthesis of the fatty acids, carried out by the moulds, is interrupted.

These fatty acids are primary metabolites used by moulds as an energy source.

Mycotoxins are usually formed at the end of the exponential phase or at the beginning of the stationary phase of the mould's growth. Mycotoxins can cause diseases and disorders in animals and humans, called mycotoxicoses.

The most important mycotoxins which can produce mycotoxicosis in broiler breeders are aflatoxin B1 and ochratoxin A. Other mycotoxins belonging to the trichothecene group, such as deoxynivalenol (DON) or vomitoxin, T-2 toxin and diacetoxyscirpenol are also important, mainly T-2 toxin and diacetoxyscirpenol.

Broiler breeders are resistant to zearalenone and fumonisin B1, but DON can cause some deleterious effects.

## Aflatoxin B1 (AFB1)

The AFB1 is produced by toxigenic strains of moulds of the genus *Aspergillus* and can be found as a natural contaminant in cereals (mainly in corn, wheat, sorghum and rice) and cereal by-products, oilseed meals, cassava and other animal feeds. This mycotoxin has high carcinogenic, teratogenic and mutagenic activity. The main toxic effect is hepatotoxicosis, but can also produce kidney problems. The AFB1 is immunosuppressive, since aflatoxin inhibits phagocytosis and protein synthesis (antibody production), interrupting DNA, RNA and ribosome protein synthesis, as well. Amino acid absorption is altered leading to the rise of amino acid hepatic retention.



According to scientific studies, mycotoxicosis problems can affect breeder as follows:

- Broiler breeder hens were fed diets contaminated with 0, 200, 1000, 5000, or 10000ppb (micrograms/kg) of AFB1. All concentrations resulted in embryonic mortality and reduction in hatchability compared to the control. In progeny chicks the AFB1 exposure resulted in immune dysfunction. The suppression of humoral and cellular immunity, imply that progeny chicks from broiler breeders hens consuming diets contaminated with AFB1 may be increasingly susceptible to diseases. Mycotoxin residues were found in fertile eggs collected during 14 days, at levels between 0.05 and 0.60ng/g (nanograms/g) of AFB1; and between 0.19 and 1.20ng/g of aflatoxicol.

- Diets contaminated with 0, 5000, or 10000ppb of AFB1 were fed to mature broiler breeder hens for four weeks. There was no reduction in fertility.

However, hatchability of fertile eggs collected during the first week of feeding the toxin declined significantly from 95% in the control to 68.9 and 48.5% in 5000 and 10000 ppb AFB1 fed groups, respectively.

With 10000 and 5000 ppb AFB1, egg production decreased significantly during weeks three and four after initiation of feeding the toxin, respectively.

Necropsies of breeder hens after four

weeks of consuming the contaminated feed exhibited typical symptoms of aflatoxicosis, such as enlarged, fatty and friable livers, and enlarged spleens, whereas no latent effects of AFB1 or its metabolites were observed on the performance of surviving chicks, with the mycotoxin concentrations previously mentioned.

- Levels of 250, 500, or 750ppb of AFB1 in broiler breeder hen (49-53 weeks of age) diets had no significant effect ( $P>0.05$ ) on egg production, specific gravity, percentage of shell and albumen of eggs and body weight gain.

Reduced body weight and body weight gain were observed in the chicks (seven days of age) originated from broiler breeder hens fed with the mycotoxin concentrations previously mentioned, compared to the control group. The contamination with AFB1 in the broiler breeder diets significantly affected the broiler chick mortality ( $P<0.05$ ) and a linear effect was observed at seven and 21 days of age.

## Ochratoxin A (OTA)

Ochratoxin A is produced by toxigenic strains of moulds of the genus *Aspergillus* and *Penicillium*. This mycotoxin can be found as a natural contaminant in cereals (mainly in barley and rice) and cereal by-products, flour and peanut meal and other animal feeds.

The major toxic effect produced by OTA is the nephrotoxicity, but can also produce a liver disorder which produces an accumulation of glycogen in hepatic and muscular tissue. OTA is also immunosuppressive.

According to scientific studies, significant undesirable effects can affect breeders, as follows:

- Breeder hens were fed diets contaminated with OTA at 100, 500, 1000, 3000, 5000, or 10000ppb (micrograms/kg), for three weeks. All OTA concentrations resulted in a significant decrease in feed intake, body weight and egg mass production compared to the control ( $P<0.05$ ).

Undesirable effects such as diarrhoea, unthriftiness, water intake and depression were increased with increasing levels of

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dietary OTA. The enlargement of liver and kidney, and the presence of haemorrhages were more severe in breeders fed the higher levels of OTA.

With all OTA concentrations, the creatinine, alanine aminotransferase, urea, and total protein levels in serum were significantly higher compared to the control.

With increasing dietary levels of OTA, the pathological alterations, serum biochemical changes, and production performance were more severely affected. The OTA concentrations were immunosuppressive in the progeny chicks obtained from breeder hens consuming diets contaminated with OTA.

## Residues in edible tissues

White Leghorn breeder hens were fed diets contaminated with 5000ppb (micrograms/kg) of AFB1 and 5000ppb of OTA, individually and combined. With the individual administration, the residues of AFB1 in liver, kidney and breast muscles were,  $1.44 \pm 0.21$ ,  $0.25 \pm 0.01$  and  $0.03 \pm 0.01$  ng/g and the residues of OTA were,  $22.54 \pm 1.48$ ,  $4.22 \pm 0.93$  and  $0.56 \pm 0.06$  ng/g, respectively.

When the mycotoxins were fed in combination, the residues of AFB1 and OTA were significantly lower in the tissues of breeder hens compared with those from the individual administration.

The combined administration of AFB1 and OTA decreased the residue concentrations of those mycotoxins in the tissues and eggs.

Residues of AFB1 and OTA in eggs appeared between three and five days after feeding the contaminated diets; and they disappeared between five and six days after removing the toxins from the feed.

## Trichothecene mycotoxins

The trichothecene mycotoxins are produced by toxigenic strains of moulds from the genus *Fusarium*. There are over 40 derivatives of trichothecenes, but the most important mycotoxins in broiler breeders are T-2 toxin (T-2), diacetoxyscirpenol (DAS) and DON due to its toxic effects. DON has few undesirable effects in breeders.

Trichothecene mycotoxins can be found as natural contaminants in cereals (corn, barley, sorghum, oats, wheat, rice, rye and millet), cereal by-products, hay and silage.

The trichothecene mycotoxins have potent immunosuppressive activity. Some of these described negative effects are found in broiler breeders.

The main damage produced by trichothecenes is in the gastrointestinal tract; however, depending on the species, the following symptoms can be observed:

- Vomiting, diarrhoea, tachycardia.
- Bleeding, oedema, necrosis of skin tissues.

- Haemorrhages of the epithelial mucosa of the stomach and intestine.
- Hematopoietic tissue destruction.
- Decrease in circulating white cells and platelets.
- Haemorrhagic meninges (brain).
- Nervous system disorder.
- Rejection of the feed.
- Necrotic lesions in different parts of the mouth.
- Pathological degeneration of cells in the bone marrow, lymph nodes, and intestine.

## Deoxynivalenol (DON)

This mycotoxin usually is an indicator that T-2 toxin or other *Fusarium* mycotoxins may also be present. DON alone has few negative effects in poultry; however, in field conditions, high levels of DON are sometimes associated with reduced feed consumption in broiler breeders.

In addition, Yegani et al in 2006 showed that diets contaminated with a high concentration of DON (12600ppb) can affect performance, especially the immune response of broiler breeders.

Also, Awad et al in 2008 showed that DON has significant undesirable effects in chickens, especially as an immunotoxic substance, affecting hematocrit values, total numbers of white blood cells, CD4+ and CD8+ cells, T-lymphocytes and B-lymphocytes, and biliary IgA concentration. Gut functions were also affected with a decrease in the glucose and amino acid absorption.

DON concentrations of 2500, 3100, or 4900ppb (micrograms/kg) in breeder hens feed, provided for 10 weeks, did not produce any negative effects on egg production, feed intake, fertility, hatchability or embryonic mortality.

However, there were significant development of abnormalities in progeny chicks, with unabsorbed yolk sac and delayed ossification.

Low levels of zearalenone, ochratoxin A, 3-Acetyl-DON and nivalenol were found in the experimental diets; however the possible toxic contributions from these mycotoxins were considered negligible.

## T-2 toxin (T-2)

Feeds containing 1000, 5000, or 10000ppb (micrograms/kg) of T-2 toxin given to breeder hens for a period of 28 days resulted in a decrease in egg production of 12.5, 68.0, and 78.9%, respectively, and a decrease in hatchability of these same eggs.

With a concentration of 500ppb of T-2 toxin in feed, breeder hens already developed oral lesions after consuming the feed for a period of three weeks.

Contamination levels of 2000, 4000 and 8000 ppb, negatively affected hatchability of the fertile eggs which was significantly lower ( $P < 0.05$ ) compared to those from the breeder hens receiving the control diet.

In addition, a decrease in feed intake, egg production, and egg-shell thickness was observed with the contaminated feeds.

Fertility and the relative weights of liver, heart, gizzard and spleen were not influenced by T-2 toxin. Serum levels of alkaline phosphatase, lactate dehydrogenase, serum glutamic pyruvic transaminase and uric acid of breeder hens fed 8000ppb of T-2 toxin were higher than in breeders receiving the uncontaminated diet. Oral lesions were evident in the breeders after the second week of receiving 4000 and 8000ppb T-2 toxin concentrations.

## Diacetoxyscirpenol (DAS)

Caged broiler breeder hens were fed diets contaminated with 0, 5000, 10000, or 20000ppb (micrograms/kg) of DAS from 24-25 weeks of age. The mycotoxin decreased body weight and feed consumption, indicating feed refusal. These mycotoxin concentrations resulted in oral lesions. The salivary glands and the tip of the tongue were the areas of the mouth most sensitive to DAS.

Individually caged male and female broiler breeders were fed diets containing 0, 5000, 10000, or 20000ppb of DAS from 25-27 weeks of age. All levels of mycotoxin reduced body weight and feed consumption in hens; but the only effect in males was a reduction in feed intake at 10,000 and 20,000ppb levels.

Male broiler breeders on litter were fed diets contaminated with 0 and 10,000ppb of DAS from 23-25 weeks of age. Feed consumption was measured, even though they were on a restricted feed intake program. The high level of DAS increased the amount of unconsumed feed at 23 weeks of age.

In summary, the experiments provided evidence that DAS decreased body weight and feed consumption in broiler breeders with the presence of cytotoxic injuries, including oral lesions.

## Comments

It is clear that the most important mycotoxins producing significant undesirable effects in broiler breeders are AFB1, OTA, T-2 and DAS. DON, also can produce serious problems in the progeny.

Some of the mycotoxin concentrations shown in this article can be found as natural contamination in feeds. In field conditions, T-2 and DAS can be found frequently in the litter material and because broiler breeders, especially males, always consume some litter due to the feed restriction program, the incidence of oral lesions in breeders is more common than in broilers. In addition, broiler breeders can be exposed to mycotoxins for a longer period of time. ■

*References are available from the author on request*