How do you know if your birds are affected by mycotoxins?

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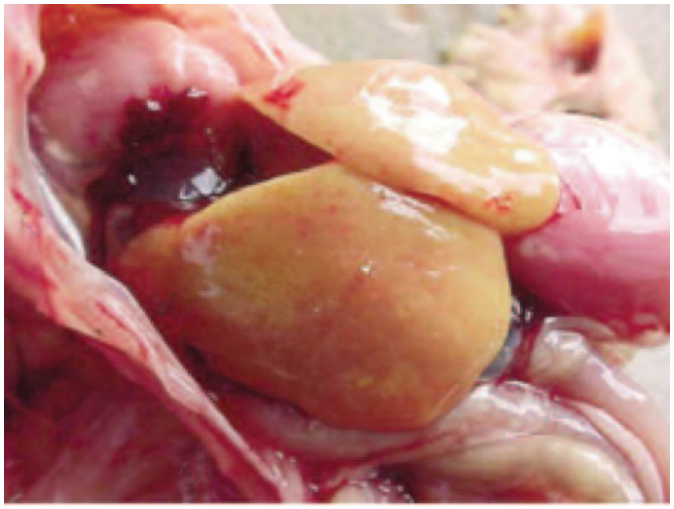
How do you know if your birds are affected by mycotoxins rather than by a viral disease? How do you interpret post mortem lesions and diagnose problems in the flock? What are the signs to look for when you suspect your birds have been exposed to mycotoxin contaminated feed?

This article will help you to allocate different symptoms and post mortem findings to specific mycotoxins, serving as a quick guide when problems arise on a poultry farm.

When you hear hoof beats, look for horses, not zebras. This is the first thing taught to students in their first year of medical training. This proverb reminds us we should look for the simplest, most common explanation first when attempting to diagnose a disease.

Differential diagnosis is especially difficult in the case of mycotoxin related problems as symptoms may vary greatly depending on interactions between toxin, animal and environmental related factors. More frequently than not commodities are contaminated with more than one mycotoxin and this factor alone leads to the occurrence of synergistic effects amongst them. For these reasons it is simply impossible to make an exact cause-effect relationship between a specific toxin concentration and its symptoms in animals.



Fatty and yellowish liver caused by aflatoxin intoxication.

Nonetheless, mycotoxins are not a new topic and they have been the subject of a lot of research within the last few years which has helped the understanding of their mode of action, epidemiology and counteraction.

Table 1 lists the major mycotoxin producing fungi, the respectively produced mycotoxins and the most important effects caused by these mycotoxins. Many other symptoms and effects have been described for poultry; however, in order to simplify their differentiation only the most distinctive ones have been chosen and will be further explained.

Table 1. The major mycotoxin producing fungi, respective mycotoxins produced and the most important effects caused.

Major mycotoxin producing fungi (amongst others)	Major mycotoxins produced (amongst others)	Most important effects
A. flavus A. parasiticus	Aflatoxin (B_1, B_2, G_1, G_2)	Fatty liver. Decreased body weight gain and impaired feed utilisation. Immunosuppression.
A. ochraceus P. verrucosum P. viridicatum	Ochratoxin (Ochratoxin A)	Renal dysfunction. Blood and meat spots in eggs. Decreased body weight gain and impaired feed utilisation. Immunosuppression.
F. verticillioides (syn. F. moniliforme) F. graminearum F. pseudograminearum F. culmorum F. poae	Type A Trichothecenes: T-2 toxin, HT-2 toxin, diacetoxyscirpenol Type B Trichothecenes: Nivalenol, deoxynivalenol, fusarenon-X Fumonisins Zearalenone	Tongue necrosis. Oral/dermal toxicity. Gizzard lesions. Decreased body weight gain and impaired feed utilisation. Immunosuppression.

Aflatoxins

Aflatoxin intoxication in poultry leads to clinical signs such as reduced general performance (reduced growth and feed intake, decreased egg production) and paleness of mucous membranes and legs (pale bird syndrome).

The most typical lesions are alterations in the liver in terms of colour and size. At post mortem examination, a friable, yellowish (due to fat accumulation) and enlarged liver with haemorrhages and anaemia can be expected in birds intoxicated with this mycotoxin.

The presence of petechial haemorrhages in the musculature of animals might result in higher susceptibility to bruising due to capillary fragility caused by aflatoxins.

However, it might be difficult to differentiate the effects of this mycotoxin from those caused by infectious bursal disease (IBD), fatty liver syndrome, malabsorption syndrome, high energy diets, amiloidosis and fish meal based diets.

Ochratoxin A

Ochratoxins are among the mycotoxins most toxic to poultry. Renal dysfunction caused by this mycotoxin may be noticed during the bird's life by increased water consumption, thus increased moisture in manure.

Similar to aflatoxins, ochratoxin A may also cause enlargement and colour alteration in the liver of affected animals.

However, in the case of the latter mycotoxin, liver will present itself pale rather than yellowish.

Nonetheless, the most characteristic change caused by ochratoxin A can be found in the kidneys as they will be severely enlarged and pale in post mortem evaluation. In laying hens the presence of ochratoxin A in diets has been related to the increase of blood and meat spots incidence in eggs.

However, blood spots inside the egg are also known to be caused by genetic factors, as well as by sudden environmental temperature changes and by aging birds. For both aflatoxin and ochratoxin A intoxication reduction of bursa is often observed which explains the severe immunosuppressive effects of these mycotoxins.

In general ochratoxin A post mortem lesions which might be confounded with those caused by aflatoxins, visceral gout, infectious bronchitis, infectious bursal disease, citrinin toxicity, sodium intoxication,

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calcium nephropathy, water deprivation, vitamin A deficiency, malabsorption syndrome, bacterial pyelonephritis and coccidiosis.

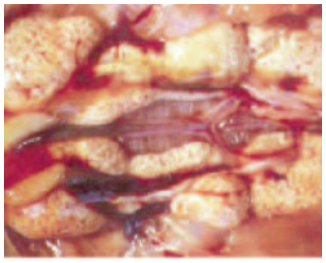
Fusarium toxins

Regardless of the mycotoxin (deoxynivalenol, T-2 toxin, fumonisins) general effects following ingestion of fusariotoxins-contaminated diets include reduced weight gain and impaired feed conversion rate along with an increased susceptibility to diseases.

Type-A trichothecenes such as T-2 toxin and diacetoxyscirpenol (DAS) produce caustic and radiomimetic patterns of disease which include ulceration, crusting and necrosis of epithelium which comes in contact with the toxins (usually oral mucosa but sometimes feet and legs).

Consequently, at post mortem lesions can vary from reddened gastro-intestinal mucosa to thickened and roughened ventricular lining, digestive ulcers, gizzard erosion and intestinal haemorrhages.

Unfortunately the negative effects of trichothecenes are much more extensive as these toxins are known to primarily inhibit protein synthesis



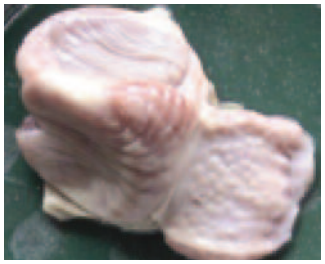
Damage to kidneys and liver caused by ochratoxin A.

followed by a secondary disruption of DNA and RNA synthesis.

These facts further explain the severe toxicity of both type A and type B trichothecenes in actively dividing cells thus their severe negative impact in the gastrointestinal tract, mucosa, feathering, and immune function.

Especially in terms of oral lesions and gizzard erosion a proper diagnostic is of crucial importance as other agents may cause similar symptoms.

Gizzard lesions alone can be caused by several different factors, namely the use of copper sulphate in diets at inclusion levels higher than 1kg/t; the inclusion of improperly processed fishmeal at levels higher than 2%; vitamin B6 deficiency and



Gizzard lesions caused by T-2 toxin.

adenoviruses. Furthermore, some amino acids found in ingredients of animal origin may degrade into biogenic amines which have been associated with the occurrence of gizzard erosion and poor performance in broilers.

Diagnostic tools

Although difficult to diagnose, the onset of mycotoxicosis on a farm is often related to a new batch of feed. Mycotoxin analysis of the feed (by high performance liquid chromatography – HPLC) or to commodities (by ELISA or HPLC) must be performed in case of suspicion of the presence of mycotoxins.

This will provide valuable information which can be gathered to that collected by observation of clinical signs and by necropsy examinations. If other diseases are to be ruled out then histopathology, bacterial and viral cultures and serology should be performed.

Prevention is key

Quite frequently the effects of mycotoxins in animals are subclinical and are therefore overlooked by farm technicians. If money losses are already existent in case of subclinical mycotoxicosis, they escalate when symptoms are observed.

These include not only the loss of genetic potential but the investment which must be done to treat symptoms or underlying illnesses.

Prevention can be done by the use of a proper mycotoxin risk management tool which adsorbs and/or biotransforms mycotoxins, thus eliminating their toxic effects for the animals, while guaranteeing liver and immune protection.

Mycofix Product Line combines the three strategies – adsorption, biotransformation and bioprotection – which work together to prevent the hazardous effects of mycotoxins hitting your flock! ■

Lesions in the tongue caused by T-2 toxin.

Poor feather development caused by trichothecenes.