# Be aware of aflatoxins



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Feed should be free from toxic inclusions. A special warning should be given regarding the hidden danger of aflatoxins, which are poisonous by-products from fungi that grow on grains. Ignoring the danger may result in huge losses in bird performance and even mortalities.

By Dr Ismail Suliman, Coral Farms, Sudan

spergillus flavus and A. parasiticus are fungi commonly found in the soil. They are responsible for the decomposition of plant materials. Despite this positive function they are very dangerous for animals. The consumption of these fungi by livestock and poultry results in a disease called aflatoxicosis due to the aflatoxins (mycotoxins), which are poisonous by-products from the fungi.

The Aspergillus is a mould that, when stressed by unfavourable weather and storage conditions, infects grains including corn sorghum, peanuts and cotton. It is our experience that a dry growing environment or drought stress tends to encourage the development of aflatoxins in corn. When soil moisture is below normal and the temperature is high, the number of Aspergillus spores in the air increases during pollination. These spores infect the corn kernels either through silks (Pollination tubes) or through areas of damage caused by insects, birds and weather events. Once infected plants are stressed because of nutrient deficiency, persistent dry weather or kernel damage during harvest, aflatoxin levels may increase.

#### Zero tolerance

There are four major types of aflatoxins: B1, B2, G1 and G2. Although all of these toxins are usually found in grain at a minute level, B1 is typically more abundant. This can cause an increased protein requirement in livestock and poultry that consume it. Aflatoxins are odourless, tasteless and have no colour, and are therefore difficult to detect. For that reason testing for aflatoxins concentration should be the first step in proper feeding management.

Because of the detrimental effect, the recommended aflatoxin level in feed is 0 parts per billion (PPb). Meeting this ideal level is not always possible, but it should never exceed 20 PPb aflatoxins in the total diet. If unexpected symptoms of aflatoxicosis occur one should immediately discontinue feeding contaminated grain and return to an aflatoxin-free diet.

Aflatoxin toxicity is mainly due to the inhibition of the protein synthesis, which is why fast-growing young animals, such as broilers, are more susceptible to aflatoxin toxicity due to their fast growth rate (which is protein driven), organ development and relative high level feed consumption.

## The first signs

Aflatoxins are metabolised in the liver by all living organisms, so a high concentration can lead to acute liver disease or death within 72 hours. High levels can result in extreme mortality in a short period of time.

Lower concentrations have shown various symptoms, including feed refusal, decreased feed efficiency, impaired reproduction, haemorrhage in muscles Blue hocks are typical expressions of aflatoxicosis.

and suppression of the immune system. Soon after young birds have eaten feed containing low levels of aflatoxins one may see clear clinical symptoms, like diarrhoea, paralysis or incoordination, reduced weight gain, lower egg production, lower egg weight and hatchability, increased condemnations, pale shanks and combs, and reduced growth rates.

When diagnosing symptoms of this disease one should be aware that both clinical signs and the post-mortem lesions may vary with the breed affected,



Bird displaying recumbence with the extension of its wing to out side. The diarrhoea contains: a liquid yellowish material representing urates from the kidney; undigested feed; and fibrinous casts, which is usually caused by anything that causes severe damage to the intestinal wall for examples (clostridium, coccidia, poisons).



As soon as you discover that the feed is contaminated with a high level of aflatoxins, feed should be taken from the feed lines and mixed with an aflatoxins binder.

the mycotoxin involved, the dose ingested and the period of exposure.

When needed there is an Elisa test for the aflatoxins in both serum and feed, however, there is some concern with using Elisa assays on biological samples (serum) because of the binding of aflatoxin metabolites to protein and neucleotides. The problem is that such a small amount of the total aflatoxin that is consumed (less than 5%) can be detected in the serum. However, the 5% or so of aflatoxins that end up in the blood when the birds have been consuming contaminated feed will quickly be cleared from the blood when the birds stop eating the feed.

#### **Post-mortem lesions**

In case young birds are fed with aflatoxininfected grains mortality levels may go up rather quickly. Post-mortem examination may demonstrate a number of possible lesions, such as:

- Damage to mucosa that has been in contact with aflatoxins.
- More bruising may be seen. Firstly, there can be an increase in capillary fragility, which means that it takes less force to cause bruising. It is thought that this effect of aflatoxin is due to protein synthesis inhibition in the liver. Secondly, one may see blood clotting cascades. The result is that the blood clotting time is prolonged, similar to haemophilia. Both factors will result in petechiae and larger haemorrhage in various tissues.
- Liver and kidney lesions. Liver may be enlarged and fatty with a yellow coloration or show bile retention or tumours, while the kidney may be swollen. The yellow liver is a classic example of aflatoxicosis. Aflatoxin is primarily a hepatotoxin mainly affecting the liver through inhibition of lipid transportation from the organ. As a result of that lipid build-up the liver

becomes enlarged and appears yellow.Enteritis, which appears in various degrees.

- Hydropericardium. Although it is not a direct symptom of aflatoxicosis, aflatoxin does affect the immune function resulting in secondary infections.
- Pale bone marrow. Is thought to reflect a direct effect of aflatoxins on red blood cell development, probably due to its effects on protein synthesis. Additionally, there appears to be an affect in the bone marrow on lipid metabolism and/or transport. These effects result in a pale and fatty bone marrow.
- Gizzard erosions. This does not commonly occur during aflatoxicosis, but does occur in occasion and is thought to reflect irritation of the gizzard lining due to direct contact with aflatoxins.
- Increased intestinal fragility. This can happen in two ways, either due to its effect on protein synthesis or due to inflammation of the intestinal epithelium.
- Regression of the Bursa of Fabricius. The best guess on why the bursa regresses is that aflatoxins affect the protein synthesis. This results in a situation where the bursa makes a lot of protein generating B-cells.



This picture shows the mixing of feed with the aflatoxins binder in the feed hopper. The aflatoxins binder appears as a dark red powder scattered over the feed

#### **Prevention strategy**

Controlling the adverse effects of mycotoxins (of which aflatoxin is one of the most important) requires a multifaceted approach involving the purchase of quality grains; management practices in the feed mill and in growing facilities; adjustments in nutrient content of the diet; and the use of selected feed additives. These are common and logical elements of a strategy, but what should be done when feed is infected with mycotoxins and is in the bins or in the feed troughs.

We usually put a large amount of feed in the feed lines of our rearing cages to assure that the baby chicks have access to enough feed during the starter phase. When we discover that the feed is contaminated with aflatoxins we take it out and mix it with a toxin binder and put it back in the hopper or straight back in the troughs. In some cases blending is also an option, but dosing is more than difficult.

When confronted with a mycotoxin problem in feed for older birds one should make sure that no animals consume any level of aflatoxins in their diet for at least three weeks prior to slaughter.

Mycotoxin binders are inert and generally made of specialised chemically modified



Engorged gall bladder.



Hydropericardium - inflammation and swelling of the heart sack.



clay. They bind the toxins very tightly when they are solubilised during digestion. Good binders insure that there is little disassociation of aflatoxins from the binders so no aflatoxins can be absorbed, but rather released through the faeces.

## **Testing for aflatoxins**

In order to prevent a situation where you are faced with a serious aflatoxin problem, feed should be checked. We conducted several Coral aflatoxins laboratory tests (*Figure 1 and 2*). The first was done when the birds were 10 days old and the sample was taken directly from the feeders. The results showed that the aflatoxin level (35.4 ppb) was too high, since the acceptable level for poultry should not exceed 20 ppb. About two weeks later, when the birds were 25-days-old, we did a second test. This time we tested samples taken from the



Fatty hemorrhagic liver.

feed mill and from the storing house. The results of the two samples revealed that, although the original source was the same, the fresh feed had lower levels of aflatoxin (23.8 ppb) than the feed from the storing house (31.1 ppb).

From these tests we can conclude that the main problem was coming from storing the feed, while the season played an important factor in aggravating the



Haemorrhage & bleeding in the chest muscles. There is also an area of bleeding in the subcutaneous tissues of the chest muscles.

problem. Although the level of aflatoxins in the freshly collected sample from the mill was relatively low, it was still higher than the acceptable level for chickens.

It was a surprise to witness that the aflatoxin level in the sample taken at day 10 was much higher than the level of the sample taken at day 25. Although we do not know for sure, we guess that this is because of the rain falling one day before the chicks were set. The accompanying higher environmental temperature stimulated the rapid growth of the fungi at that specific moment.

The results from these tests suggest that it may be wise for future practice to bring in feed day by day or a little time before use. In our hot and sometimes humid climate we believe that it is better to have the grinding process of feed in the rearing farm to provide fresh feed to the birds. Establishing a grinder/mill locally at the farm instead of bringing the fresh feed from a great distance may reduce storing time and limit the possibility of aflatoxin growth in the feed. ■

#### **Proper storage facilities**

The advice given by Dr Suliman is valid for his specific situation, but it has to be noted that safe storage and milling of feed is not necessarily a privilege of the poultry farmer. It can also be done by professional feed millers who can test and store feed ingredients efficiently and secure the delivery of fresh feed. Bearing in mind that there is no difference between feed produced on the farm and feed that has been produced at another location, on farm storage facilities should be good enough to prevent the growth of fungi and thus secure the safety of the feed (ingredients) – *The editor* 

| Eiguro 1 Corol Johorator   | v results for aflatoxin tests o | n food for 10 day old | chicks (S)  |
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| rigure i - Curai laburatur | y results for analoxin lests u  | n leeu lor lo-uay-olu | CIIICKS (3) |

|                            | Group: ISMAEEL.DTA<br>Assay: feed<br>Toxin: Aflatoxin<br>Date: 09-07-2005(1 | 0:58)                                     |  |
|----------------------------|---|---|--|
|                            | SAMPLE I.D.   | O.D. (@650nm)                             | RESULT   |
| 1)<br>2)<br>3)<br>4)<br>5) | 0.0 ppb C<br>5.0 ppb C<br>15.0 ppb C<br>50.0 ppb C<br>S                     | 1.649<br>1.307<br>0.952<br>0.593<br>0.691 | 0.0 ppb<br>4.8 ppb<br>16.4 ppb<br>47.9 ppb<br>35.4 ppb |
|                            | CORR.COEFF. = 0.9977<br>SLOPE = -1.9143<br>Y-INT = 2.6390                   |   |  |

# Figure 2 - Coral laboratory results for aflatoxin tests on feed for 25-day-old chicks in the feedmill (A) and on farm (B)

|          | Assay:<br>Toxin:                     | ISMAEEL.DTA<br>FEED23/09<br>Aflatoxin<br>09-22-2005(1 | 1:11)  | 3  |
|----------|--------------------------------------|---|--|--|
|          | SAMPLE                               | E I.D.  | O.D. (@650nm)                                      | RESULT   |
| 3)<br>4) | 0.0<br>5.0<br>15.0<br>50.0<br>A<br>B | ppb C<br>ppb C  | 0.957<br>0.800<br>0.561<br>0.332<br>0.486<br>0.423 | 0.0 ppb<br>4.7 ppb<br>17.2 ppb<br>46.8 ppb<br>23.8 ppb<br>31.1 ppb |
|          | SLOPE<br>Y-INT                       | DEFF. = 0.9947<br>= -2.2548<br>= 3.<br>2005(13:04)    |  |  |