A veterinary look at mycotoxin lesions

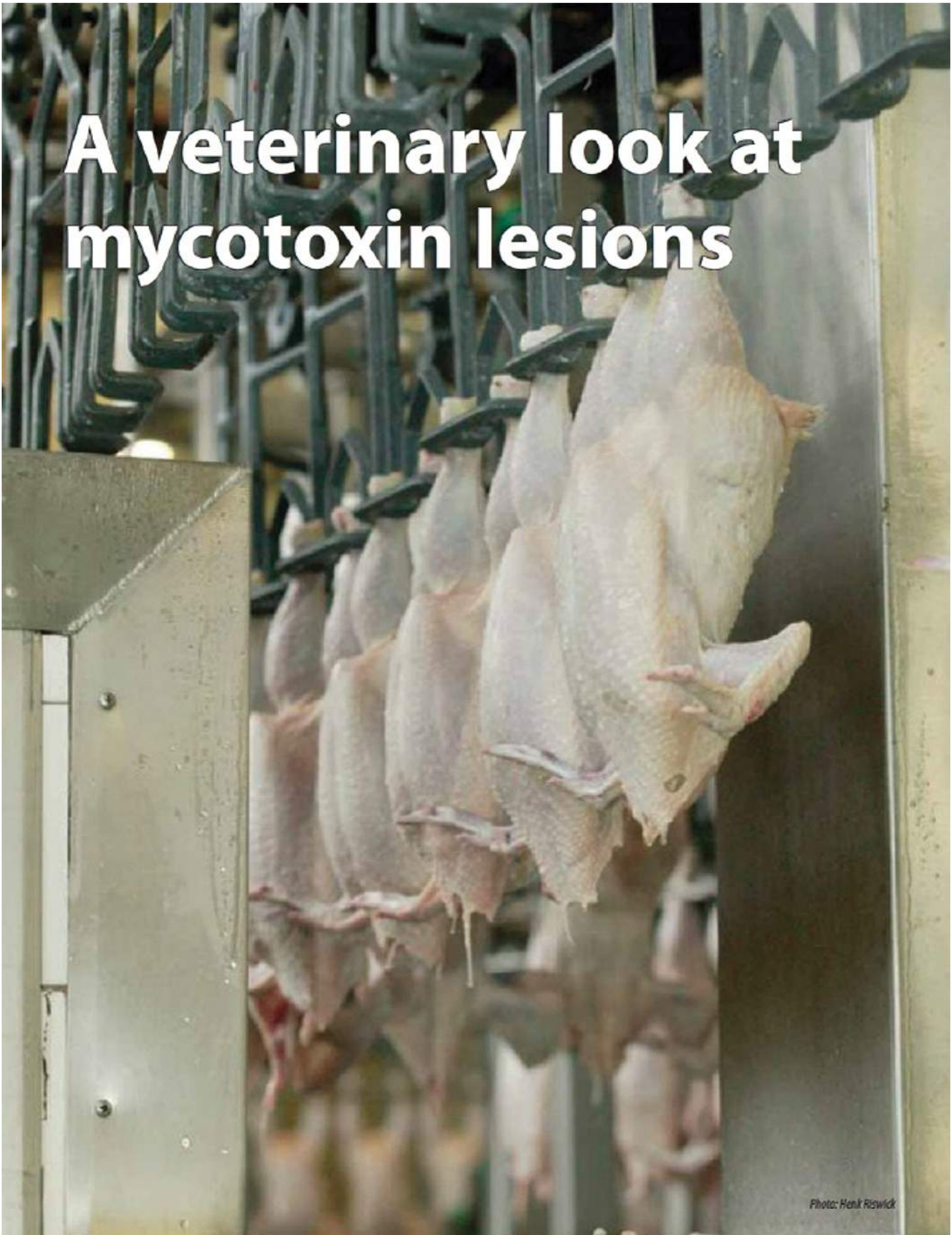

Photo: Henk Riswick

Want to know whether the mycotoxins measures made at feed level have been successful? A look at the lesions found in the slaughterhouse could hold the answer. However, the changes present in poultry affected by mycotoxins can be microcopic.

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A common concern facing feed mills and poultry production facilities around the word consist of determining if the mycotoxins present in feed ingredients or in the rations are affecting performance. Producers that have already decided to use anti-mycotoxins additives, wonder if their decision is economically wise. Considering that mycotoxins detection in feed frequently provides inconsistent results, due to their uneven distribution, one practical approach for evaluation is the identification of lesions in the slaughter houses. This search will confirm their presence for companies that may have any doubts about their existence and will also serve to demonstrate if the preventive measures taken are effective. In this article we will review some of the macroscopic changes present in the birds affected by mycotoxins with the objective of offering another tool to evaluate their incidence.

It is important to that besides evaluating these lesions, performance results must be taken into consideration in any type of evaluation.

Oral lesions

Plaques and ulcers in the oral cavity may be produced by several mycotoxins belonging to the group of Trichothecene, including T-2 toxin, MAS(monoacetoxyscirpenol) and DAS (diacetoxyscirpenol). During the first stage, the damage appears as focal with the development of yellow, oral plaques that become ulcers located in different parts of the mouth (palate, tongue, floor of the mouth, salivary duct opening, and the interior margins of the beak). Lesions in the mouth are more frequently seen after long-term feeding, generally in pullets, but if the concentration of T2 is high enough, lesions will appears sooner. Under commercial conditions it is more difficult to detect them in younger birds (4 to 5 weeks). Since the head is usually several and placed apart from the rest of the carcass at the slaughter house, meat inspectors or veterinarians usually do not evaluate it. Typically, the presence of the oral lesions already described is the beast way to reconfirm that mycotoxins are affecting a flock. Essentially, this is a lesion mainly produced by mycotoxins. The presence of wheat deposits in the corner of the mouth is a condition that must be ruled out in a differential diagnosis in areas where this grain is added to the diet.

Proventriculosis and gizzard erosion

Proventriculosis consist on the enlargement of the proventriculus, and proventriculitis compromises the inflammation of the internal surface. Here we concentrate on proventriculosis because it is easier to identify without opening the organ in the slaughter house. Cycloplazonic acid (CPA) produced by Aspergillus flavus, the predominated fungus responsible for Aflatoxin production, is the main mycotoxin associated with this pathology. The inflammation of the proventriculus or its enlargement can be the results of over stimulation of the glands responsible for hydrochloric acid secretion. Fish meal and biogenic amines (present in bone, blood, meat and feather meals) can produce some degree of proventriculitis, depending on the inclusion rate i the diet.

Pathogens like Marke's Disease, Reovirus, and the Infectious Proventriculitis Virus associated with Infectious Bursal Disease (TBD) are responsible for this lesion in infected birds. Another post mortem finding is gizzard erosion. Among other entities, it can be due to mycotoxins such as T2, MAS, or DAS. Their caustic effect it responsible for the necrotic damage observed in the mouth, gizzard and intestines. Copper sulphate, biogenic amines present in bone, feather, blood and meat meals, as well as fish meal, can cause some degree erosion.

Intestinal and liver damage

Hemorrhages and enteritis can be produced by mycotoxins, as well as many other elements including viral infections (Reovirus), bacterial pathogens (Escherichia coli, Salmonella, etc), and nutritional factors. In severe cases, mycotoxins such as T2 toxin can produce petechial hemorrhages that spread along the intestines (small and large). During the process of sacrificing birds in the farm or at the slaughter house, the presence of haemorrhages in the form of petechiae (tiny pin point, 1-2 mm in size) or ecchymoses (large bruises 2-3 cm in size) in the duodenum, is sometimes confused with lesions caused by mycotoxins (T2 toxin, DAS, MAS). One common lesion that must be ruled out when establishing of agonal haemorrhages, a normal post mortem change present in some birds, associated with death struggle.

Fatty livers

Generally, the liver of healthy young birds (broilers and pullets) is of a brown colour with a compact appearance. Yellow or pale livers can be caused by

Table 1 - A list of organs that should be evaluated weekly or monthly in the slaughterhouse to detect the effect of mycotoxins in the field.

Organ	Lesion	Mycotoxin
Oral cavity	Ulcers, plaques	T2 toxin/MRS/OAS
Proventriculus	Enlargement	Cyclopiazonic acid
Gizzard erosion	Ulcers	T2 toxin/MRS/OAS
Liver	Pale, yellowish, friable	Aflatoxin
Gall bladder	Pale content	Aflatoxin
Breast	Bruises	Aflatoxin
Wings	Bruises	Aflatoxin
Thigh	Bruises	Aflatoxin
Kidney	Urate deposits inflammation	Ochratoxin
Intestines	Intestinal haemorrhages in large areas of the intestines	T-2 toxin, DAS, MAS

Fatty Liver and Kidney Syndrome (FLKS), a condition that occurs in 1 to 4 week-old broilers and is associated with biotin deficiency. Affected chicks show growth depression and fatty infiltration of the liver, kidney and heart without the presence of the characteristic signs of a biotin deficiency. On the other hand, the presence of fatty or pale livers has been associated with the used of bad quality oil (oxidized fat) in the ration. The use of antioxidants in the feed has been considered as a positive measure to help reduce its incidence. Fatty liver haemorrhages Syndrome (FLHS) is characterized by the presence of excessive oily fat deposits and haemorrhages in the abdominal cavity with enlarged, friable, pale/yellowish livers. The liver can become so fragile that it is difficult to remove each lobe without causing its breakage. This condition is considered one of the most important metabolic disorders commonly reported during the production period in commercial layers. Typically, even healthy flocks will show layers affected by FLHS after 45-50 weeks of age. When FLS (fatty Liver Syndrome) is produced by mycotoxins (mainly Aflatoxin) birds show a yellowish appearance with petechial haemorrhages but no excessive abdominal fat is present. Another difference is that typically, abdominal haemorrhage is not necessarily present. The main target organ of Aflatoxin in poultry is the liver, causing yellow discolouration due to lipid accumulation as a result of the impairment of the liver capacity to synthesize amino acids, In one scientific trial perform in chickens where 100 ppm of Fumonisin was added to the diet, the liver size nor its colour were affected. In addition, the presence of pale bile content (bilis) has been associated with Aflatoxin. A reduction in amylase , lipase and biliary salts production by the liver is responsible for That appearance of the gall bladder.

Bruiser and Kidney lesions

This is also a somewhat fairly common lesion affecting carcasses in slaughter houses in many regions. The main mycotoxin associated with bruisers in chickens is Aflatoxin, which is characterized by increasing capillary fragility and the reduction of skeletal muscles Aflatoxin interferes with coagulation by affecting some of its components, mainly prothrombin, a key factor in the reaction cascade necessary to complete the whole process. Experimentally, Wyatt in the US has reported that concentrations as low as 100ppb of Aflatoxin cause bruising in broiler carcasses. Some recent reports from Brazil indicate that Fumonisin are associated with a higher incidence of skin haemorrhages in chickens. High density (number of birds per square meter) is a common reason for this lesion under commercial conditions. Besides mycotoxins improper stunning in the slaughter house can result in incomplete bleeding while excessive stunning can increase the presence of haemorrhages as a results of ruptured arteries and capillaries.

Urate deposits and swollen kidneys can be induced by Ochratoxin. Viral pathogens as nefropathogenic strains of infectious bronchitis (IB), excessive levels of calcium in layer diets and lack of water can be responsible for urate deposits in kidneys and other structural damage to the organ. For decades the tropism for kidneys has been recognized for strains such as Gray, Holte, and Florida 88. Lately, new Chinese strains as QX and Q1 have been associated with renal lesions in China. Europe and Latin America. Reports of the effects of Citrinin in the kidney under commercial conditions are not as common, even through some personal communications indicate the involvement of this mycotoxin in the presence of watery faeces and wet litters.

Testing of Carcasses

Table 1 includes some of the organs that should be evaluated weekly or monthly in the slaughter houses to detect the effects of mycotoxins in the field. It is import to emphasize that even the most efficient mycotoxins binders available in the market will not adsorb 100% of the mycotoxins present in the feed, so the detection of some degree of lesions is expected when mycotoxin are present in the feed. At least 200 to 300 carcases should be examined and periodically some tissue samples should be evaluated using histopatlology. The inclusion of the evaluation of the Bursa of Fabricius in the table presented, if possible, will offer important information on the status of the immune system. AAF.

References are available upon request

Poultry producers that have already decided to use anti-mycotoxins additives, wonder if their decision is economically wise. Lesions detected at the slaugterhouse might give some more insights.