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Poultry
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Practical information for progressive poultry professionals

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A practical guide to differential diagnosis in poultry

Correctly identifying problems on the farm and their influencing factors goes a long way to finding a workable solution.

A practical guide to differential diagnosis

1 – Oral lesions

by Inês Rodrigues, technical manager, Biomin Singapore Pte Ltd and Luca Vandi and Simone Schaumberger, Biomin Holding GmbH.

Oral lesions are particularly prevalent in laying hens or breeders and may arise from different etiological agents. Alkaline acting mycotoxins, such as type-A trichothecenes may cause lesions to the epithelium and increase the speed of epithelial cell renovation (see table right). Also related to feed factors is feed granulometry in which small particles of feed may obstruct saliva ducts thus causing oral lesions. Other frequently ignored factors are, for example, an excess of organic acids or copper sulphate and/or a mismanagement of injectors which may produce points of high concentration of methionine and other components in the feed, ultimately leading to similar lesions.

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 if the presence of mycotoxins is suspected. This will provide valuable information which can be gathered in addition to observing clinical signs and necropsy examinations.

If other diseases are to be ruled out, then histo-pathology, bacterial and viral cultures and serology should be performed. Quite frequently the effects of mycotoxins in animals are subclinical and are therefore overlooked by farm technicians. If there are already financial losses in the case of subclinical mycotoxicosis, these losses escalate when symptoms are observed.

These include not only the loss of genetic potential but the investment required to treat symptoms or underlying illnesses.

Prevention can be undertaken through 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.

Biomin's Mycofix product line combines the three strategies – adsorption, biotransformation and bioprotection – which work together to prevent the hazardous effects of mycotoxins in poultry flocks. ■

Check list	Corrective action
Potential cause: MYCOTOXINS: T-2 toxin (T-2) or Diacetoxyscirpenol (DAS)	
• Positive for T-2 and/or DAS in raw materials (ELISA) or feed (HPLC). • Origin of raw materials from supplier/region with history of T-2/DAS contamination. • Histopathology: Proliferating epithelial cells. Hepatic vacuolization. • Decline in the overall performance of the flock.	• Check average contamination levels. • Use Mycofix at a correct dosage level. • Avoid contamination of feed bins or feed/water lines by stale, wet or mouldy feed.
Potential cause: NUTRITION: Feed granulometry	
• Pelletised feed: fine particles >20%. • Mashed feed: Check geometric mean particle diameter. • Histopathology: Presence of inflammatory cells and bacteria. • No decline in overall flock performance.	• Adjust the pelleting process. • Increase in sieve diameter. • Use of pellet binders to improve pellet quality.
Potential cause: MANAGEMENT: Liquid methionine	
• Methionine injector dripping inside masher. • Histopathology: Infiltration of inflammatory cells. Necrotic lesions. • No decline in overall flock performance.	• Clean/replace methionine injectors.
Potential cause: MANAGEMENT: Organic acids	
• Acids injector dripping inside masher. • Histopathology: Infiltration of inflammatory cells. Necrotic lesions. • No decline in overall flock performance.	• Clean/replace acid injectors. • Adjust dosage of organic acids.
Potential cause: MANAGEMENT: High temperatures	
• Histopathology: Infiltration of inflammatory cells. Necrotic lesions. • Possible decline in overall flock performance. • Increased mortality.	• Apply vitamins in water. • Apply organic acids in water. • Increase chlorine level in water.
Potential cause: MANAGEMENT: Copper sulphate	
• Check concentration of CuSO4 in premix. • Check concentration of CuSO4 in water. • Check if water dosing system is working correctly (if applicable).	• Apply group B vitamins and K3 vitamin in water. • Correct set up of the water dosing system

Note: Pathogens were excluded from the table due to space constraints but may be important to consider.

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2 – Impaired feathering/feather loss

by Inês Rodrigues, technical manager, Biomin Singapore Pte Ltd, Luca Vandi and Simone Schaumberger, Biomin Holding GmbH.

In poultry, feathers serve important roles in terms of protection and insulation of the body.

Whilst moulting, or renovation of older feathers by new ones, is a natural process occurring in mature layers upon completion of a laying cycle (which itself can be influenced by many factors), feather loss or impaired feathering may be indicative of other problems in the farm.

Feather-related problems in poultry can be roughly divided into two groups, either:

- They are not properly developed (linked to feather formation) which is often related to nutrition or the presence of mycotoxins.
- They are pulled off by birds (feather pecking), which is a management-related issue.

In each case it is critical to understand the foundation of the problem so that it can be properly solved (see table right).

Stressful conditions in the barn, especially during brooding, such as heat, cold and existence of air currents, amongst others, can result in feather loss and poor feather quality in the birds. In this case, it is crucial that the behaviour and interaction of animals is observed.

Often, feather pecking and pulling can also be triggered by inadequate intake of nutrients. Due to the high protein content in feathers, higher protein levels in feed may encourage faster feather development and shedding.

Imbalance of amino acids in the feed, particularly sulphur amino acids cysteine and methionine, may cause feather abnormalities and/or rough feather appearance.

The dermotoxic effect of trichothecene mycotoxins, such as T-2 toxin and others, may also contribute to low feather quality along with other negative effects, such as oral lesions and decreased performance.

Overall, excessive feather loss or impaired feathering adversely affects feed conversion as birds have to allocate extra energy from the diet to compensate for heat loss.

As such, management, housing and nutrition should be optimised to reduce this occurrence. In terms of mycotoxins, prevention can be undertaken through 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.

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References are available from the authors upon request.

Check list	Corrective action
Potential cause: MANAGEMENT: Temperature of barn	
• Temperature of barn. • Humidity of barn. • Ventilation system.	• Improve management of barn. • Correct temperature, ventilation rate and humidity according to management manuals.
Potential cause: MYCOTOXINS: T-2 toxin (T-2)/Deoxynivalenol (DON)/ Other trichothecenes	
• Positive for trichothecenes in raw materials (ELISA) or feed (HPLC). • Raw materials originating from supplier/region with a history of trichothecenes contamination. • Histopathology: Check other target organs for trichothecenes (ex. liver, for hepatic vacuolisation). • Decline in overall flock performance.	• Check the average contamination levels. • Use Mycofix at the correct dosage level. • Avoid contamination of feed bins or feed/water lines by stale, wet or mouldy feed.
Potential cause: NUTRITION: Amino acid (AA) deficiency/unbalance	
• Level of Total Sulphur Amino Acids (TSAA) in diet. • Ratio TSAA/Lys/Arg/Thr. • AA scale at feed mill.	• Increase level of synthetic Amino Acids (AA) in low digestible diets (high levels of by-products).
Potential cause: MANAGEMENT: Red mites	
• Presence of red mites in the barn during the night.	• Flame cages during withdrawal period. • Clean egg belts during withdrawal period. • Increase biosecurity level. • Use plastic egg belts whenever possible.

Note: Pathogens were excluded from the table due to space constraints but may be important to consider.

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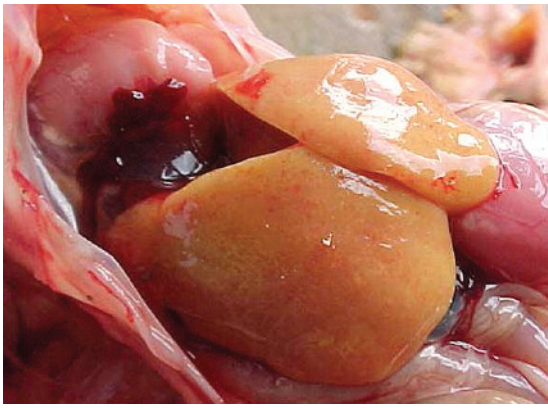

3 – Fatty liver

by Inês Rodrigues, technical manager, Biomin Singapore Pte Ltd, and Luca Vandi and Simone Schaumberger, technical managers, Biomin Holding GmbH.

Fatty liver syndrome is a non-infectious condition that affects laying or breeding hens, especially caged animals. Some strains appear to be more susceptible than others, higher producing hens are the most susceptible within a flock and outbreaks of the problem are often associated with hot weather and a period of extensive egg laying.

Afflicted animals present general obesity (on the average 20% overweight) with enlarged, fat-infiltrated livers, which are soft and easily damaged. Variable low mortality (2-5%) may be observed, caused by internal liver haemorrhage. In that case, birds will be found suddenly dead with pale head skin.

Usually, the cause is related with high calorie intake (high energy diets or incorrect energy:protein ratios) but other factors, such as intake of the hepatotoxin aflatoxin or mismanagement of layer birds must not be disregarded.

In case exposure to mycotoxins is to blame, commonly known as lean bird fatty liver, livers are yellowish with petechial haemorrhages but not swollen; microscopic lesions of centrilobular necrosis and bile duct hyperplasia are present, but no excessive abdominal fat is found.

Depending on the potential cause for fatty liver, different corrective actions are proposed (please consult table on the right).

In terms of mycotoxins, prevention can be undertaken through 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. The Mycofix product line from Biomin combines the three strategies – adsorption, biotransformation and bioprotection – which work together to prevent the hazardous effects of mycotoxins in poultry flocks. ■

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Check list	Corrective action
Potential cause: NUTRITION: Energy/protein ratio	
• Carbohydrate level in diet. • Energy/protein ratio in diet.	• Avoid high carbohydrate diets, especially in summer. • Adopt proper energy/protein ratio. • Apply AA to drinking water.
Potential cause: NUTRITION: Rancid fats	
• Quality of fats in term of: peroxide value, rancidity and free fatty acids.	• Avoid low quality fats. • Replace animal fats with vegetable fats. • Apply choline chloride + vitamin B to feed or water.
Potential cause: MYCOTOXINS: Aflatoxins (Afla)	
• Positive for Afla in raw materials (ELISA) or feed (HPLC). • Raw materials originating from supplier/region with history of aflatoxin contamination. • Histopathology: Check other target organs of Afla (ex. liver). • Decline in overall flock performance.	• Check average contamination levels. • Use Mycofix at the correct dosage level. • Avoid contamination of feed bins or feed/water lines by stale, wet or mouldy feed.
Potential cause: MANAGEMENT: Hormone status	
• Management of laying birds (excessive oestrogen stimulation).	• Improve management of laying birds. • Correct lighting program.
Potential cause: PATHOGENS: Viral hepatitis (IBH - Inclusion Body Hepatitis)	
• Clinical symptoms include immunosuppression, diarrhoea, anorexia, depression, ruffled feathers which appear only several hours prior to death. • Necropsy: Macroscopic lesion in the enlarged, dystrophic liver with yellowish colour and crumbly texture. Enlarged kidneys. • Serology: Isolation of serotype I, II or III from lesions.	• Use inactivated vaccines (existing only for Group I). • Check breeding stock and eliminate affected birds.
Note: Pathogens were excluded from the table due to space constraints but may be important to consider.	

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4 – Gizzard lesions

by Inês Rodrigues, technical manager, Biomin Singapore Pte Ltd, and Luca Vandi and Simone Schaumberger, technical managers, Biomin Holding GmbH.

The muscular stomach or gizzard is located immediately after the proventriculus in poultry. Unlike the proventriculus, which produces a number of juices or enzymes that are used in the digestion or breaking down of food into its constituent nutrients, the gizzard serves a more mechanical purpose, aiding digestion by particle size reduction and regulation of feed flow. It consists of a number of layers of tissues, some of which contain straight tubular glands. The innermost layer is a strong, flexible skin that is able to withstand the potentially damaging effects of the muscular action grinding the food, often in the presence of stones or other insoluble material. The glands of the gizzard produce a keratinised liquid material which hardens when in the surface to replace tissue worn away by the grinding action of the organ.

In spite of being a fairly strong organ, the occurrence of erosion or lesions in the mucosal lining (koilin) of the gizzard is often reported by field veterinarians in broiler and commercial layers operations. In some cases, these lesions are already observed in day-old chicks before placement in the broiler house and prior to feed consumption. For young chicks, studies point to post-hatch stress or the presence of mycotoxins in breeder diets (which then carry-over into the egg) as possible factors. For older animals, a lot more potential causes are worth consideration. The table, right, gives an overview of some of those.

In terms of mycotoxins, prevention can be undertaken through 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. The Mycofix product line from Biomin combines the three strategies – adsorption, biotransformation and bioprotection – which work together to prevent the hazardous effects of mycotoxins in poultry flocks. ■

References are available upon request

Check list	Corrective action
Potential cause: MYCOTOXINS: Cyclopiazonic acid (CPA) Deoxynivalenol (DON) and/or T-2 toxin (T2)	
• Positive for CPA, DON and/or T-2 in raw materials (ELISA) or feed (HPLC) • Raw materials originated from supplier/region with history of mycotoxin contamination • Histopathology: Proventriculus hyperplasia of mucosa with heavy infiltration of lymphocytes • Decline in flock performance	• Check average contamination levels • Use Mycofix at a correct dosage level • Avoid contamination of feed bins or feed/water lines by stale, wet or mouldy feed
Potential cause: MANAGEMENT: Copper sulphate ($CuSO_4$)	
• Concentration of $CuSO_4$ in premix • Concentration of $CuSO_4$ in water • Water dosing system is working correctly (if applicable)	• Apply group B vitamins and K3 vitamin in water • Correct set up of the water dosing system
Potential cause: NUTRITION: Biogenic amines (gizzerosine)	
• Level of gizzerosine in raw materials (especially fish meal)	• Lower level of fish meal in diet • Avoid use of low quality fishmeal • Replace standard fish meal with low temperature (LT) fishmeal
Potential cause: NUTRITION: Rancid fats	
• Quality of fats in term of peroxide value, rancidity and free fatty acids	• Avoid low quality fats • Use low quality fats in the grower/finisher phases • Replace animal fats with vegetable fats
Potential cause: NUTRITION: Tannins	
• Level of tannins in some raw materials (sorghum) and in tannin-based products	• Use high quality tannin-based product (chestnut better than quebracho) • Reduce % of sorghum in high-tannin diets
Potential cause: MANAGEMENT: Acetylsalicylic acid and sodium salicylate	
• Dosage of salicylates used (check over estimation of feed intake in feed restricted animals) • Mixability of commercial product in water	• Avoid low quality products (low mixability, low homogeneity in water) • Correct feed intake assumption in feed restricted animals
Potential cause: PATHOGENS: Adenovirus serotype I	
• Isolation of serotype I or II or III from the lesions by serological assays	• Use inactivated vaccines (only available for group I) • Check the breeding stock and eliminate the affected birds
Potential cause: PATHOGENS: Infectious bursal disease (IBDV/ Gumboro)	
• Maternal antibody titres are very low in day-old chicks	• Implement/correct vaccination program in breeders • Change from mild to strong-reaction vaccine • Correct vaccination age (Deventer formula) • Increase biosecurity level

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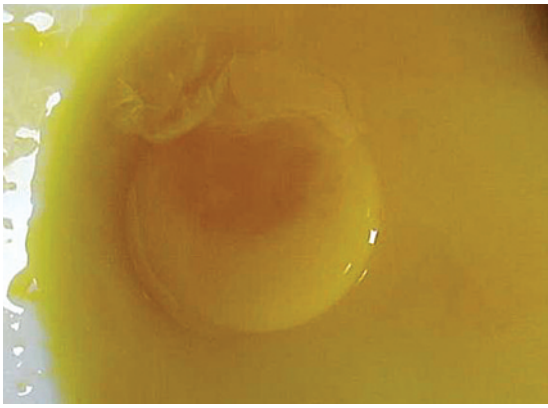

5 – Egg production/quality problems

by Inês Rodrigues, technical manager, Biomin Singapore Pte Ltd, and Luca Vandi and Simone Schaumberger, technical managers, Biomin Holding GmbH.

Good and stable egg production and good egg quality are of utmost importance to farmers depending on their layer's output for a living. Bad management practices, feed and environment-related issues and diseases are some of the factors which may cause a negative impact in egg production and egg quality. Besides these, animal-related factors, such as age and strain of layer birds must not be disregarded. Older birds and birds after moulting are known to produce bigger eggs with thinner shells and indigenous strains cannot quite compete with commercial layers in terms of number of eggs produced.

Several management issues (see table) may lead to nervous birds and/or traumatic lesions in the ovary which cause poor egg quality (fragile shell/bloodspots/meat spots). Nutrition wise, improper balance of calcium, phosphorus and vitamin D may lead to thin egg shells. Also, large amounts of lucerne/alfalfa meal in the diet can lead to blood spots caused by vitamin K antagonists in this feed ingredient. Interestingly, the use of the drug sulphaquinoxaline may have the same effect as mineral imbalance. In terms of pathogens, infectious bronchitis (IB) causes respiratory disease and kidney damage in growers and oviduct infection in adult hens, which can cause wrinkled egg shells as well as a reduction in eggs laid.

Due to the liver and kidney toxicity mycotoxins may negatively impact egg and shell formation, leading to poor egg and shell quality (pale eggs/small, fragile shell/bloodspots/meat spots). For mycotoxin-related problems, prevention can be undertaken through 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. The Mycofix product line from Biomin combines the three strategies – adsorption, biotransformation and bioprotection – which work together to prevent the hazardous effects of mycotoxins in poultry flocks. ■

Check list	Corrective action
Potential cause: MANAGEMENT: Nervous birds/traumatic lesions in the ovary	
• Lighting program • Temperature of the barn • Presence of frights and disturbances in the barn that may get birds nervous	• Correct lighting program • Correct temperature of the barn • Improve management of laying birds
Potential cause: MYCOTOXINS: Aflatoxins (Afla), Cyclopiazonic acid (CPA), T-2 toxin (T-2), Ochratoxin A (OTA)	
• Positive for Afla, CPA, T-2 and/or OTA in raw materials (ELISA) or feed (HPLC) • Raw materials originating from supplier/region with history of mycotoxin contamination • Histopathology: Check other target organs of these mycotoxins (for example kidneys, liver) • Decline in overall flock performance	• Check average contamination levels • Use Mycofix at the correct dosage level • Avoid feed bins or feed/water lines becoming contaminated by stale, wet or mouldy feed
Potential cause: NUTRITION: Mineral/vitamin imbalance	
• Calcium/phosphorus balance in diets • Calcium carbonate particle size	• Correct mineral and vitamin balance • Correct calcium carbonate particle size
Potential cause: NUTRITION: Vitamin K antagonists	
• Content of lucerne/alfalfa meal in diets • Presence of sulphaquinoxaline in diets	• Correct amount of lucerne/alfalfa meal in diets • Correct medication program
Potential cause: PATHOGENS: Infectious bronchitis (IB)	
• Laboratory tests to confirm the presence of the coronavirus in a swab or tissue sample	• Vaccination program must be adapted to the demands of the field situation in each particular area/epidemiology
Potential cause: GENETICS: Bird strain	
• Check with genetic supplies (some strains more susceptible to blood spots)	• Replace genetics if necessary

References are available from the authors on request

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6 – Avian gout /kidney failure

by Inês Rodrigues, technical manager, Biomin Singapore Pte Ltd, and Luca Vandi and Simone Schaumberger, technical managers, Biomin Holding GmbH.

Avian gout is a consequence of kidney damage which can occur from a number of potential causes leading to the accumulation of uric acid/urates in the renal tubules and serous coats of the heart, the liver, the mesentery, the air sacs or the peritoneum. Due to its complex aetiology, it is difficult to diagnose; however, the most common signs are dehydration, pale combs, depression and swelling and reddening of the feet which impair bird movement. In layers, where it is mainly observed, avian gout can lead to mortalities up to 50%, with 19-35 week-old hens mostly affected.

The causes for this condition are varied (see table right), ranging from management- and/or nutrition-related, to pathogens and/or the presence of mycotoxins in feed. In terms of nutrition, special attention must be paid to the calcium/phosphorus balance, sodium and vitamin D3.

In general, any condition favouring an increase of uric acid in blood favours precipitation in tissue and, as a consequence, development of gout. Excess dietary calcium with low available phosphorus results in the precipitation of sodium-urate crystals and calcium pyrophosphate (pseudogout). In younger birds, gout due to sodium intoxication may be observed at sodium levels exceeding 0.4% in water and 0.8% in feed.

Likewise, high levels of vitamin D3 can increase calcium absorption from the intestine favouring the formation and deposition of urate crystals. Also nutrition-related is the protein level in feed which in excess of 30% causes uric acid production leading to excretory loads in kidneys. Concurrently, sulphates decrease calcium resorption causing excess calcium secretion through the urine. This favours gout, as well as any other factor contributing to urine alkalinity. Water deprivation falls in this category as it leads to increased concentrations of uric acid and other minerals in the blood and later on in the kidneys and urine.

Viruses such as infectious bursal disease (IBDV) and/or infectious bronchitis can enhance mortalities in the presence of pre-existing kidney damage.

In terms of mycotoxin contamination of feeds, the nephrotoxic aflatoxins (Afla), ochratoxin A (OTA) and citrinin are of major concern. The impairment of the kidney function which results from the action of these mycotoxins reduces uric acid excretion and results in the accumulation of uric acid in the body.

For mycotoxin-related problems, prevention can be undertaken through a proper mycotoxin risk management tool which adsorbs and/or biotransforms mycotoxins, thus eliminating their toxic effects for animals, while guaranteeing liver and immune protection. The Mycofix product line from Biomin combines the three strategies – adsorption, biotransformation and bioprotection – which work together to prevent the hazardous effects of mycotoxins in poultry flocks. ■

Check list	Corrective action
Potential cause: MYCOTOXINS: Ochratoxin A (OTA), citrinin, aflatoxins (Afla)	
• Positive for Afla, citrinin and/or OTA in raw materials (ELISA) or feed (HPLC) • Raw materials originating from supplier/ region with history of mycotoxin contamination • Histopathology: Check other target organs of these mycotoxins (e.g. kidneys, liver) • Decline in overall flock performance	• Check average contamination levels. • Use Mycofix at the correct dosage level. • Avoid feed bins or feed/water lines becoming contaminated by stale, wet or mouldy feed.
Potential cause: NUTRITION: Calcium, sodium, vitamin D3	
• Level of minerals and vitamins in diets	• Correct level of minerals and vitamin D3. • Control fish meal usage (rich in salt). • Control total sodium chloride content in feed (<0.3%).
Potential cause: NUTRITION: Protein	
• Protein level in feeds	• Correct protein level in feeds.
Potential cause: MANAGEMENT: Water deprivation	
• Observe animal behaviour to understand the cause of water deprivation • Transportation and vaccination procedures • Drinkers in terms of number, position and blockages that may impede access. • Chemicals added to water (disinfectants, copper sulphate, etc) may result in water refusal, dehydration and gout	• Improve transportation condition of birds (access to water). • Adjust number, position and access to drinkers. • Avoid overcrowding. • Correct blockages in nipples.
Potential cause: PATHOGENS: Infectious bursal disease (IBDV/Gumboro)	
• Maternal antibody titres are very low in day-old chicks	• Adapt vaccination program to the demands of the field situation in each particular area/epidemiology. • Increase biosecurity level.
Potential cause: PATHOGENS: Infectious bronchitis (IB)	
• Laboratory tests to confirm the presence of the coronavirus in a swab or tissue sample	• Adapt vaccination program to the demands of the field situation in each particular area/epidemiology.

References are available from the authors on request

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7 – Carcase bruising

by Inês Rodrigues, technical manager, Biomin Singapore Pte Ltd, and Luca Vandi and Simone Schaumberger, technical managers, Biomin Holding GmbH.

The consumer's decision making process when purchasing poultry products mainly takes into account appearance, hygiene and flavour. To help guarantee that the best quality product reaches consumers, several procedures should be in place.

Veterinary inspection at the time of slaughter aims to guarantee that poultry carcases are free from disease or faecal contamination. In the presence of one (or both) contaminations, carcases are condemned and withdrawn from the food chain.

Carcase bruising/haemorrhage is one of several reasons leading to carcase downgrading (reduced quality) or condemnation in the slaughterhouse. It is caused by the breakage of blood vessels and subsequent leakage of blood into tissues without skin rupture.

It is difficult to determine whether they occur at the farm, during transport or at the plant; therefore, any major financial losses that result are usually absorbed by the slaughterhouse.

According to scientific literature, the colour of the bruise may be indicative of the age of the injury with red to dark red being recent bruises (≤ 12 hours) and light green, yellow-orange and yellow ones being older (≥ 24 hours).

Some 90% of bruising occurs within 12-24 hours before processing, with breast, wings and legs the most frequently affected parts. The potential causes for that are inadequate flock density in the grow-out house and/or the failure to properly adjust pickers at catching.

At the abattoir, inadequate stunning (voltage and time) can lead to haemorrhagic petechiae usually occurring in the breast and legs. The presence of pathogens in the farm, such as IBDV (Gumboro disease) may increase capillary weakness which leads to carcase bruising.

Mycotoxins such as aflatoxins (Afla) work in a similar way, by reducing the force required to produce bruises due to increased capillary fragility. Usually these occur in the thighs.

For mycotoxin-related problems, prevention can be undertaken through the use of a proper mycotoxin risk management tool which adsorbs and/or biotransforms mycotoxins, thus eliminating their toxic effects in the animals, while guaranteeing liver and immune protection. Biomin's Mycofix product line combines the three strategies – adsorption, biotransformation and bioprotection – which work together to prevent the hazardous effects of mycotoxins in poultry flocks. ■

Check list	Corrective action
Potential cause: MANAGEMENT: Stunning system	
• Voltage of stunning system • Duration of electric shock	• Correct voltage and timing of electric shocks
Potential cause: PATHOGENS: Infectious bursal disease (IBDV/Gumboro)	
• Blood spots mainly located in the legs and the breast • Necropsy: Bursa of Fabricius is swollen, enlarged and bloody • Maternal antibody titres are very low in day-old chicks • Implement/correct vaccination program in breeders	• Change from mild to strong-reaction vaccine • Correct vaccination age (Deventer formula) • Increase biosecurity level
Potential cause: MYCOTOXINS: Aflatoxins (Afla)	
• Positivity for Afla in raw materials (ELISA) or feed (HPLC) • Animals present jaundice-like symptoms, are dehydrated and emaciated. They present purple-reddish areas in the carcase • Raw materials originating from supplier/region with history of aflatoxin contamination • Histopathology: Check other target organs of Afla (for example liver) • Decrease in overall performance of the flock	• Check average contamination levels. • Use Mycofix at a correct dosage level. • Avoid feed bins or feed/water lines becoming contaminated by stale, wet or mouldy feed
Potential cause: MANAGEMENT: Animal density	
• High flock density at grow-out house	• Adjust flock density
Potential cause: MANAGEMENT: Catching	
• Poor catching procedures	• Adjust picking machines and/or catching procedure

References are available from the authors on request

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8 – Lameness conditions (nutrition)

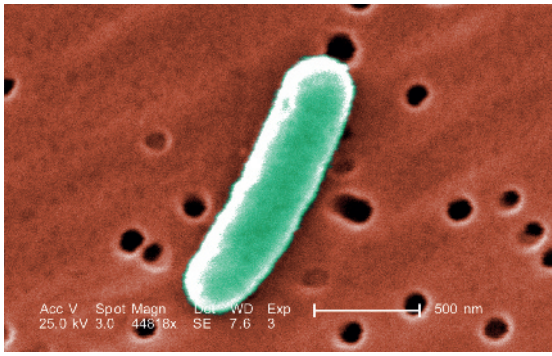
by Chasity Pender, Technical Manager, and Raj Murugesan, Technical & Marketing Director, Biomin America.

Chickens raised for commercial meat production are selectively bred to reach marketweight quickly. This rapid growth, however,can place increasing demands on the bird'sskeletal system resulting in impaired loco-motion. Lameness and gait abnormalities in poultry are conditions of high significancenot only because of their implications interms of animal welfare, but also due to thefinancial losses caused by increased mortal-ity, reduced feed utilisation and growth rate,and downgrading in the processing plant. Nutrition plays a significant role in skeletalhealth and development, thus a multitude ofnutritional factors can lead to musculo-skeletal diseases, which are commonly characterised by lameness. It is important to identify and understand these risk factors inorder to develop a prevention or mitigationstrategy to reduce incidence of lameness inpoultry flocks. The purpose of this table isto outline several nutritional factors that cancontribute to increased incidence of lameness in poultry and offer approaches tohelp mitigate the damage caused by these conditions. ■

Condition	Corrective action
RICKETS Potential cause: Vitamin D3 deficiency, Ca/P imbalance	
• **Symptoms:** Enlargement of the ends of tibia and femur, with widened epiphyseal plate • **Lesions**: Disorganised cartilage matrix, irregular vascular penetration	• Feed vitamin D3 with balanced calcium and phosphorus
PEROSIS/CHONDRODYSTROPHY Potential cause: Manganese deficiency	
• **Symptoms:** Thickened and shortened legs, shortened wings • **Lesions**: Enlargement and malformation of the tibio-metatarsal joint, twisting and bending of tibial distal end and the proximal end of tarso-metatarsus, slippage of the gastrocnemius tendon from its chondyles	• Feed appropriate manganese as per the production stage • Maintain Mn/Ca/P balance
OSTEOPOROSIS/CAGE LAYER FATIGUE Potential cause: Impaired calcium flux in laying hens	
• **Symptoms:** Soft and rubbery bones, birds on their sides in the back of the cage • **Lesions**: Vertebral fracture affecting spinal cord	• Feed appropriate calcium as per the production stage • Care must be taken to feed ~50% of the dietary calcium in the form of coarse limestone, with the remaining half as fine particle limestone
IONOPHORE TOXICITY Potential cause: Monensin	
• **Symptoms:** Legs extended backward • **Lesions**: No specific lesions	• Mix feed properly • Withdraw the ionophore
PODODERMATITIS/FOOTPAD DERMATITIS/FOOT BURN/AMMONIA BURN Potential cause: Biotin deficiency, poor quality litter	
• **Symptoms:** Ulceration of the metatarsal and digital footpads • **Lesions**: Necrotic lesions on the plantar surface of the footpads	• Improve gut integrity by feeding multi-species, poultry-specific, live probiotics • Supplement biotin in the feed • Lower litter moisture with proper ventilation and avoid water spillage
TIBIAL DYSCHONDROPLASIA/OSTEOCHONDROSIS Potential cause: Ca/P ratio, excess chloride in feed ⇒ metabolic acidosis, acid/base balance, mycotoxins	
• **Symptoms:** Swelling and bowing in the region of the knee joints, angulations of legs, typically in birds >35 days • **Lesions**: Plug of cartilage in proximal end of tibia, distal tibia, and proximal metatarsus, in decreasing order of frequency	• Correct the nutritional imbalances • Add an effective mycotoxin deactivator in the feed

References are available from the authors on request

A practical guide to differential diagnosis



9 – Lameness conditions (bacterial pathogens)

by Chasity Pender, Technical Manager, and Raj Murugesan, Technical & Marketing Director, Biomin America.

In 50 years, broiler growth rates have increased dramatically due to intense genetic selection and enhanced nutritional programs. Fast growth places great demands on birds' musculoskeletal systems, which can result in impaired locomotion and lameness.

Lameness reduces animal well-being and has severe economic consequences from poor growth, increased culling and mortality, and increased carcase condemnation and downgrading at slaughter.

Lameness is often a multifactorial condition. Understanding the various causes can help producers identify areas for improvement and develop effective strategies to reduce the incidence of lameness in their flocks. Conditions responsible for lameness can be of infectious or non-infectious origin. This table focuses on lameness conditions caused by bacterial pathogens and suggests solutions that can help prevent or alleviate lameness caused by these conditions. ■

Condition	Corrective action
Bacterial Chondronecrosis with Osteomyelitis (BCO)	
• **Etiology:** Enterococcus cecorum, Streptococcus spp., Staphylococcus spp, E. coli • **Symptoms:** Bird will be sitting on its breast/keel, with the legs directed forward, use of wings for walking support and hip flexion • **Lesions**: Necrotic degeneration and microbial infection, primarily within the proximal heads of the femur and tibia	• **Prevention:** Improve gut integrity by feeding multi-strain poultry-specific live probiotic • **Treatment:** Antibiotics depending on severity, but mostly birds are euthanised
Vertebral Osteomyelitis/Spondylitis/Spondylopathy/Spondylolisthesis/Kinkyback	
• **Etiology:** Enterococcus cecorum, Staphylococcus spp., E. coli • **Symptoms:** Typically starts from day 22, bird will be sitting on its breast/keel, with the legs directed forward, posterior paralysis due to spinal cord compression • **Lesions**: Abscess and/or necrosis in T4-T7 vertebrae, dorsal buckling of spinal cord (kyphosis), interstitial oedema, atrophy, degeneration of muscle fibres	• **Prevention:** Improve gut integrity by feeding multi-strain poultry-specific live probiotic • **Treatment:** Antibiotics depending on severity, but mostly birds are euthanised
Bumble Foot	
• **Etiology:** Staphylococcus spp. • **Symptoms:** Swelling above the hock and around the hocks and feet. • **Lesions**: Abscess on hock joint, infected joints may have clear exudate with fibrin clots	• **Prevention:** Improve gut integrity by feeding multi-strain poultry-specific live probiotic • **Treatment:** Antibiotics
Fowl Cholera	
• **Etiology:** Pasteurella multocida • **Symptoms:** Swollen hock joints, swollen wattles and comb, greenish diarrhoea • **Lesions**: Necrotic foci on liver, petechiae in the epicardial fatty tissues	• **Prevention:** Vaccines only if endemic • **Treatment:** Antibiotics
Osteomyelitis Complex	
• **Etiology:** Bacterial, but no specific pathogen has been identified • **Symptoms:** None • **Lesions**: Green discolouration of liver, inflammatory lesions in bones and joints	• **Prevention:** Improve gut integrity by feeding multi-strain poultry-specific live probiotic • Only identified at slaughter
Bacterial arthritis/Airsacculitis (MS)	
• **Etiology:** Enterococcus faecalis, Mycoplasma synoviae • **Symptoms:** Ruffled feathers, swollen hock joints and feet, bilaterally asymmetrical legs • **Lesions**: Joints and tendon sheaths have viscous grey to yellow exudate, caseous exudate from the lesions	• **Prevention:** Improve gut integrity by feeding multi-strain poultry-specific live probiotic • **Treatment:** Antibiotics and eradication of infected breeding stock

References are available from the authors on request

A practical guide to differential diagnosis

10 – Lameness conditions (viral pathogens)

by Chasity Pender, Technical Manager, and Raj Murugesan, Technical & Marketing Director, Biomin America.

In 50 years, broiler growth rates have increased dramatically due to intense genetic selection and enhanced nutritional programs. Fast growth places great demands on birds' musculoskeletal systems, which can result in impaired locomotion and lameness. Lameness reduces animal well-being and has severe economic consequences from poor growth, increased culling and mortality, increased carcase condemnation and downgrading at slaughter.

Lameness is often a multifactorial condition. Understanding various causes can help producers identify improvements and develop effective strategies to reduce the incidence of lameness in their flocks.

Conditions responsible for lameness can be of infectious or non-infectious origin.

This table focuses on lameness conditions caused by pathogenic factors, namely viruses, and suggests solutions that can help prevent or alleviate lameness caused by these conditions. ■

Condition	Corrective action
Tenosynovitis/Viral arthritis	
• **Aetiology:** Avian reovirus • **Symptoms:** Soft swelling of the joints with turbid fluid in the capsule, swollen shanks • **Lesions**: Swelling and petechiae in the synovial membranes, small erosions on the articular cartilage, adhesions between the tendons and fibrosis of tissues	• **Prevention:** Live vaccine followed by inactivated vaccine • **Treatment:** Euthanise the infected flock
Amyloidosis	
• **Aetiology:** Corona virus • **Symptoms:** Swollen hock joint containing orange-yellowish material, muscular shivering • **Lesions**: Extracellular build up of amyloid protein in joints and internal organs	• **Prevention:** Live vaccine • **Treatment:** Sodium salicylate 1g/litre (acute phase). Antibiotics to control secondary colibacillosis
Infectious bronchitis (IB)/Infectious laryngo tracheitis (ILT)	
• **Aetiology:** Corona virus, Herpes virus • **Symptoms:** Sudden death, muscular shivering • **Lesions**: Oedema of skeletal and pectoral muscles	• **Prevention:** Live vaccine • **Treatment:** Sodium salicylate 1g/litre (acute phase). Antibiotics to control secondary colibacillosis
Marek's disease	
• **Aetiology:** Avian Herpes virus 2 • **Symptoms:** One leg stretched forward and the other backward • **Lesions**: Tumors in internal organs, unilateral enlargement of peripheral nerves	• **Prevention:** Live vaccine • **Treatment:** Eradication of infected flock
Avian encephalomyelitis (AE)	
• **Aetiology:** Picornavirus • **Symptoms:** Trembling of the head, neck, and wings, paralysis of both legs extended out to one side • **Lesions**: Gross lesions are mild or absent, focal white areas in gizzard muscle	• **Prevention:** Vaccination of breeders • **Treatment:** None
Newcastle disease	
• **Aetiology:** Avian paramyxovirus serotype 1 • **Symptoms:** Twisting of neck and paralysis of wings and legs, cyanosis of comb, facial oedema, green diarrhoea, drop in egg production, sudden death • **Lesions**: Haemorrhage in intestine, petechial haemorrhage in proventriculus, congestion and mucoid exudates seen in the respiratory tract, especially in trachea	• **Prevention:** Live vaccine • **Treatment:** None. Antibiotics to control secondary bacterial infections
Eastern equine encephalitis	
• **Aetiology:** Arbovirus • **Symptoms:** Flaccid neck, staggering, paralysis • **Lesions**: No gross lesions	• **Prevention:** Vaccination. Control mosquito population • **Treatment:** None

References are available from the authors on request

A practical guide to differential diagnosis

11 – Lameness conditions (management)

by Chasity Pender, Technical Manager, and Raj Murugesan, Technical & Marketing Director, Biomin America.

Due to intense genetic selection for increased growth and feed efficiency, lameness has become a growing issue in today's broiler industry on a global scale.

Lameness is not only a concern in terms of animal welfare: it also poses a serious financial threat to poultry producers as it is a significant cause of culling, mortality, and condemnations. Overall, the economic cost associated with lameness problems in poultry can add up to several hundred million dollars each year.

Many risk factors, including both non-pathogenic and pathogenic causes, could be associated with the occurrence of lameness in broilers and the condition is usually multi-factorial. It is important to differentiate the multiple causes of lameness in order to develop proper prevention and treatment strategies as these strategies will change based on the causative agent identified.

Management factors, such as litter quality and stocking density, can play a major role in the development of leg issues and lameness.

This table highlights several management factors that are commonly associated with increased occurrence of lameness and offers solutions to help mitigate the consequences of these conditions. ■

Condition	Corrective action
Pododermatitis/Footpad dermatitis/Foot burn/Ammonia burn	
• **Causation:** Poor litter quality, biotin deficiency • **Symptoms:** Ulceration of the metatarsal and digital footpads • **Lesions**: Necrotic lesions on the plantar surface of the footpads	• Lower litter moisture with proper ventilation and avoid water spillage • Improve gut integrity by feeding poultry-specific, live probiotics • Supplement biotin in the feed
Tibial dyschondroplasia/Osteochondrosis	
• **Causation:** Genetic selection, late rapid growth rate, calcium-phosphorus ratio, excess chloride in feed ⇒ metabolic acidosis, acid/base balance, mycotoxins • **Symptoms:** Swelling and bowing in the region of the knee joints, angulations of legs typically in birds >35 days • **Lesions**: Plug of cartilage in proximal end of tibia, distal tibia, and proximal metatarsus, in decreasing order of frequency	• Lower the energy and protein density of feed to slow down the growth • Correct the nutritional imbalances • Add an effective mycotoxin deactivator in the feed
Twisted leg	
• **Causation:** Genetic selection, stocking density • **Symptoms:** Distortion at hock, valgus/varus, various angulations of legs • **Lesions**: Linear twisting of tibia and femur, Changed angulation of tibial condyles	• Euthanise affected bird
Degenerative joint disease	
• **Causation:** Developmental defects, physical damage • **Symptoms:** Imbalanced walking, huddling • **Lesions**: Damaged epiphyseal articular cartilage, especially of femoral anti-trochanter, but also other leg joints, resulting in erosions and cartilage flaps	• Euthanise affected bird
Ionophore toxicity	
• **Causation:** Monensin • **Symptoms:** Legs extended backward • **Lesions**: No specific lesions	• Mix feed properly • Withdraw the ionophore

References are available from the authors on request

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