Differential Diagnosis for Mycotoxicoses in Poultry



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Differential Diagnosis for Mycotoxicoses in poultry, 2023

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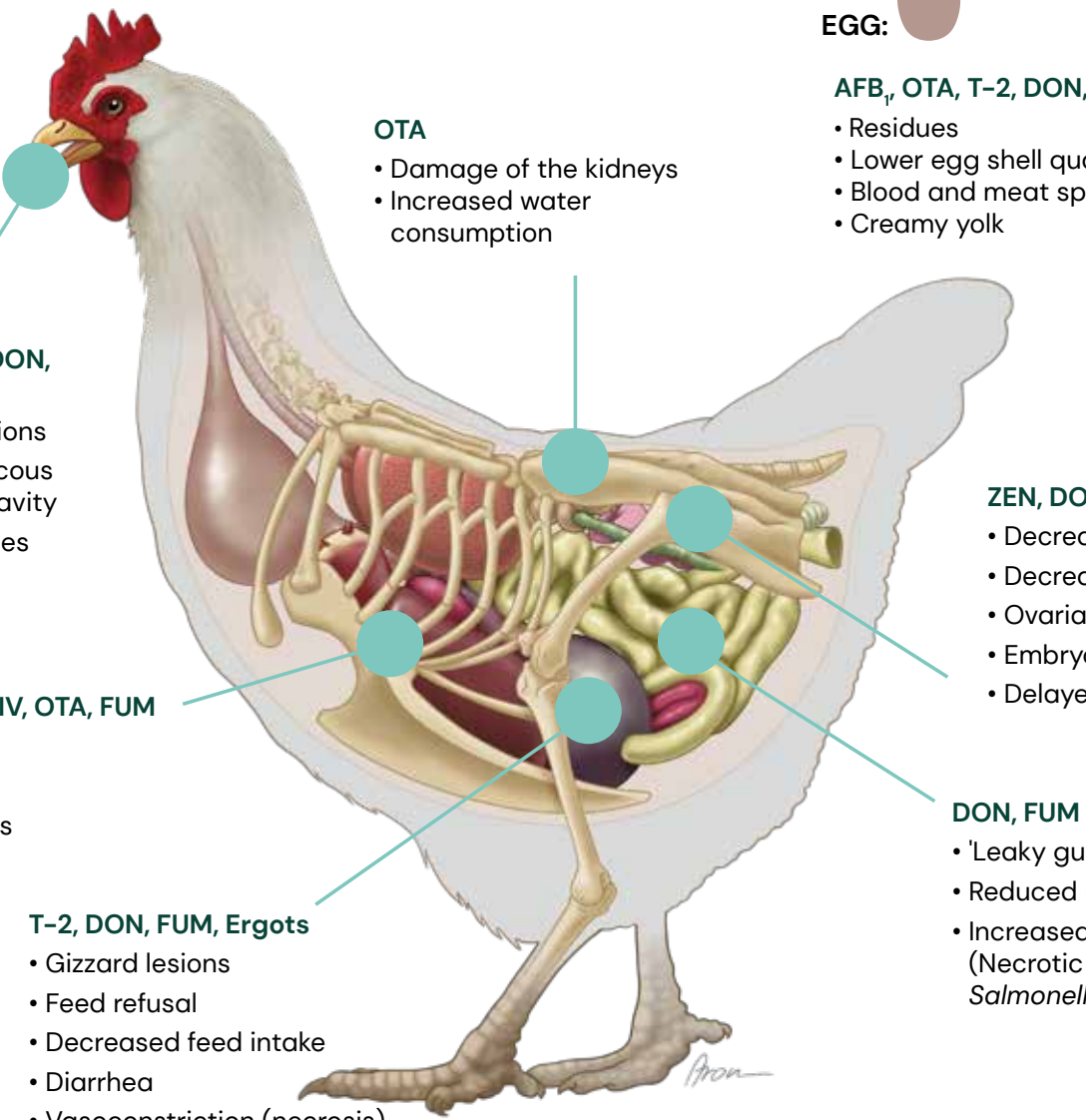
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Meaning of abbreviations:

DON: Deoxynivalenol
ZEN: Zearalenone
AFB_1: Aflatoxin B_1
FUM: Fumonisins
T-2: T-2 Toxin
HT-2: HT-2 Toxin
NIV: Nivalenol
DAS: Diacetoxyscirpenol
OTA: Ochratoxin A
Ergots: Ergot Alkaloids



EGG:

AFB_1, OTA, T-2, DON, ZEN
- Residues
- Lower egg shell quality
- Blood and meat spots
- Creamy yolk

OTA
- Damage of the kidneys
- Increased water consumption

T-2, HT-2, NIV, DAS, DON, AFB_1, Ergots
- Oral and dermal lesions
- Inflammation of mucous membrane of oral cavity
- Respiratory difficulties
- Bad feathering

ZEN, DON, T-2, DAS, Ergots
- Decreased hatchability
- Decreased egg production
- Ovarian cysts
- Embryonic loss
- Delayed sexual maturation

AFB_1, T-2, DON, DAS, NIV, OTA, FUM
- Fatty liver
- Immunosuppression
- Inhomogeneous flocks
- Impaired feathering
- Nervous syndrome

DON, FUM
- 'Leaky gut' condition
- Reduced nutrients absorption
- Increased gut health issues (Necrotic Enteritis, Coccidiosis, *Salmonella, E.coli*)

T-2, DON, FUM, Ergots
- Gizzard lesions
- Feed refusal
- Decreased feed intake
- Diarrhea
- Vasoconstriction (necrosis)
- Decreased performance

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Immunomodulation

Different factors can lead aimals for an imune suppressed condition. Chronical inflammation or infection by pathogens can persist in the flocks, reducing the uniformity, imparing vaccinal efficiency and consequently the performance of the birds.

Signs
Reduced flock uniformity, persistent infections by pathogens

We listed some potential causes that can contribute to these lesions.





Figures 1 & 2: Weak birds and reduced flock uniformity.

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Mycotoxins

Potential cause	Description of problem	Checklist	Corrective actions
Aflatoxins (Afla) and Fumonisins (FUM)	• Reduces the funcionality of hepatic cells and/or necrosis. • Impairment in the production of immunoglobulines and vaccinal antibodies	• Histopathology of the liver/fabricius bursa/thymus/kidney/spleen and bone marrow • Origin of raw materials from supplier/region with history of Afla and Fum contamination. • Check average contamination levels in feed. • Positive for Afla and Fum in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.
Ochratoxins (OTA)	• Mainly nephrotoxic and hepatotoxic • Reduction and inhibition of lymphocyte proliferation, hypotrophy of fabricius bursa • Reduction of IgA, IgG and IgM in serum	• Histopathology of the liver/fabricius bursa/thymus/kidney/spleen and bone marrow • Origin of raw materials from supplier/region with history of Afla and Fum contamination. • Check average contamination levels of Ota in feed. • Positive for (Ota) in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.
Tricothecenes (DON, T-2, DAS)	• T2 toxin induce caustic injuries to the mucosa, destroying cells on the top of villi (Hemorrhagy, necrosis, and inflammation of the intestinal epithelium). DON reduces the expression of the tight junction proteins. These effects in the intestine helps to estabilish an infamatory condition which challenges the Gut-associated lymphoid tissue (GALT) is a component of the mucosa-associated lymphoid tissue (MALT). • Trichotecenes can cause lymphoid and other hematopoietic tissue necrosis causing reduced immunoglobuline syntesis and antibody response. In a vaccination program can be compromissed by the presence these mycotoxins.	• Histopathology of the liver/fabricius bursa/thymus/kidney/spleen and bone marrow • Origin of raw materials from supplier/region with history of trichotecenes contamination. • Check average contamination levels in feed. • Positive for trichotecenes in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.

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Pathogens

Potential cause	Description of problem	Checklist	Corrective actions
Viral hepatitis (IBH – Viral inclusion body hepatitis)	• Adenovirus causes yellow/hemorrhagic • liver and focal necrosis. Symptoms include immunosuppression, diarrhea, anorexia, depression, ruffled feathers, especially in the region of head and neck.	• Necropsy: Macroscopic lesion in the enlarged, dystrophic liver with yellowish color and crumbly texture; enlarged kidneys. • Histopathology: Detection of intranuclear inclusion bodies. • Serotype identification by serological assays.	• Usage inactivated vaccines. • Check the breeding stock and eliminate affected birds.
Infectious Bursal Disease (IBD)	• IBD is very immunosuppressive and leads to kidney damage.	• Histopathology of the liver/fabricius bursa/thymus/kidney/spleen and bone marrow • Serology tests for antibody level in day old chicks and at the end of the cycle	• Adapt vaccination program to the demands of the farm situation in each particular area/epidemiology.
Aviary Reovirus	• "Malabsorption syndrome", also called reuging syndrome, affects young birds, from one to three weeks, and is characterized mainly by apathy and lack of mobility of birds, disuniformity of lots, deficiency of pigmentation, bone problems, birds with bristling feathers and wet plumage, in addition to intense diarrhea and the presence of partially digested food in the feces of birds.	• Histopathology of the liver/fabricius bursa/thymus/kidney/spleen and bone marrow • PCR test to identify the virus	• The most effective way to control reovirus is the immunization of breeders aiming at high levels of passive immunity transfer to cut birds"
Avian Infectious Anemia	• It is usually observed in young birds and is characterized by the presence of anemia and generalized atrophy of lymphoid tissues, such as thymus and bursa of Fabricius, leading to a state of immunosuppression in birds and, eventually, death.	• Histopathology of the liver/fabricius bursa/thymus/kidney/spleen and bone marrow • Serology tests for antibody level in day old chicks and breending	• Use of vaccines against Avian Infectious Anemia in breeders in rearing phase
Mycoplasma synoviae	• M. synoviae can lead not just to arthritis but also spread to other organs like the ovaries, lungs causing also airsacculitis, synovitis, bursitis and septicaemia . • It can be immunosupressive. Inside the cell, the virus can escape the immune system by implementing several mechanisms by causing excessive release of pro-inflammatory cytokines, inhibit phagocytosis, and affect B-cells and T-cells function.	• Control flocks with sorological tests	• High standarts of biosecurity and prevention • Do not mix flocks • Possible vaccination • Treatments aren't effective
Mycoplasma gallisepticum	• MG-infection induces respiratory distress, profound inflammatory response in mucosal tissues and immune dysregulation in chickens	• Control flocks with sorology tests	• High standarts of biosecurity and prevention • Do not mix flocks • Possible vaccination • Treatments aren't effective

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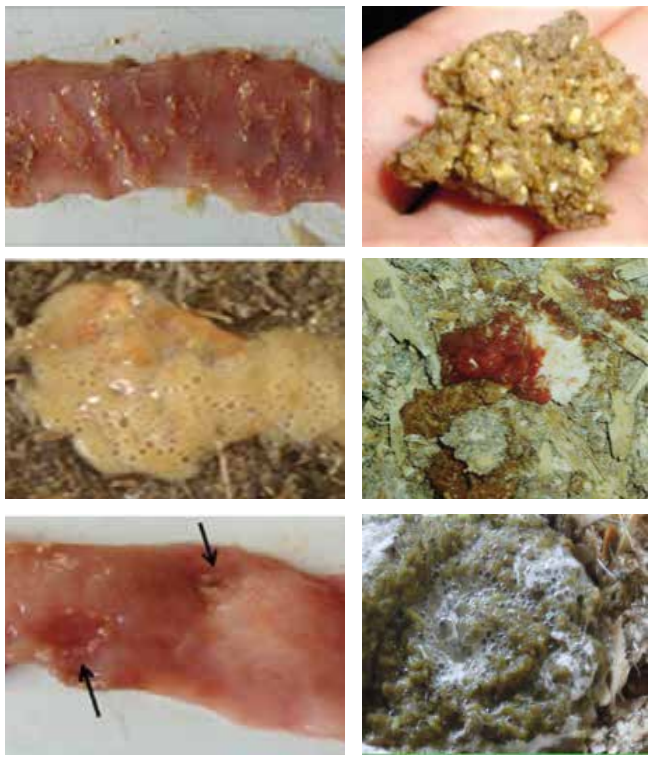
Gut Lesions

Gut is responsible for nutrient adsorption, as well as it plays an important role in the immune system. Changes in its structure can reduce the intestinal integrity leading to a ‚leaky gut' condition. When this condition is estabilished there is a reduction in nutrient absorption, increased patogenic bacteria proliferation and translocation leading to a reduced immune status and the overall performance of the birds.

Signs

Wet litter, diarrhea with undigested feed in the excreta compromised performance. Pos-mortem observations may show higher amount of mucus, liquid content and inflammatory aspect within gut lumen.

We listed some potential causes that can contribute to these lesions.



Figures 3 – 8: Gut inflamation and lesions, reduced excreta quality

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Mycotoxins

Potential cause	Description of problem	Checklist	Corrective actions
Fumonisins (FUM) and Deoxynivalenol (DON)	• Impairment of 'tight junctions' protein expression, increasing intestinal permeability • Negatively impact gut morphology, reducing absorption area in the gut • Co-factor of secondary diseases as: *Coccidiosis, Necrotic enteritis, Campylobacter, Salmonella*	• Histopathology of the gut • Origin of raw materials from supplier/region with history of FUM and DON contamination. • Check average contamination levels in feed. • Positive for Fum and DON in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.

Nutrition

Potential cause	Description of problem	Checklist	Corrective actions
Poor quality of the diet	• Reduced villus high, due to phisical agression raises the paracellular permeability and induce higher mucus amount	• Check the quality and inclusion of winter cereals (wheat, barley) in the diet • Careful usage of alternative ingredients as: rice, wheat bran, sunflower seeds	• Avoid the utilization of bad quality ingredients • Add of nutrional enzymes in the formulation
Non-starch polysaccharides (NSP)	• Impairment of amino acid digestibility can lead to na over production of mucus and higher availability of protein in the lumen for patogenic bacteria proliferation	• Check NSP levels. Check feed composition and commodities	• Maintain the correct levels of NSPs. Adapt commodities for best feed quality. • Add of nutrional enzymes in the formulation

Pathogens

Potential cause	Description of problem	Checklist	Corrective actions
Eimeria sp. (Coccidiosis)	• Part of the parasite cycle is done in gut mucosa leading to an agression in the villus • Lesions reducing nutrient absorption surface and villus funcionality	• Check for diarrhea and whole ingredients in the excreta • Observe intestinal lesions while doing necropsies in the birds • Analyse oocyst count analysis in the excreta • In a histopathologica analysis of the gut	• Estabilish a good anti-coccidial program • Usage of vaccines, feed additives and/or chemicals to reduce the coccidiosis counts • Reduce moisture in the facilities (bed) to reduce the sporulation of oocysts in the environment
Clostidium perfringens (Necrotic enteritis)	• Proliferation of the bacteria in favorable conditions leads to gut lesions and inflamation of the mucosa	• Look for sub-clinical coccidia • Check for diarrhea, orange mucus, and whole ingredients in the excreta • Observe intestinal lesions while doing necropsies	• Estabilish a good anti-coccidial program • Use of feed additives to mantain a balanced microbiota • Usage of nutritional enzymes in the diet to reduce concentration of nutrients available in gut lumen

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Others

Potential cause	Description of problem	Checklist	Corrective actions
Heat stress	• High temperatures and humidity may lead to a higher consumption of water and lower feed intake in order to regulate body temperature. • Heat stress was proven to impair gut integrity by inducing inflammation in the organ.	• Check temperature and humidity records.	• Maintaince of equipment in good conditions • Well trained people to ensure frequent check of the enviroment conditions • Set corrective measures if necessary (open curtains, fan, etc.)
High intake of salts or water	• High intake of potassium, magnesium, sodium, sulfates or chlorine through feed or water may promote an increased water consumption to maintain electrolyte embalance. It can result in more liquid excreta.	• Evaluation of salt levels in feed and drinking water	• Ensure correct concenration of salts and its mixing. • Analysis for the concentration of minerals in the water might be necessary.

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Reduced Fertility

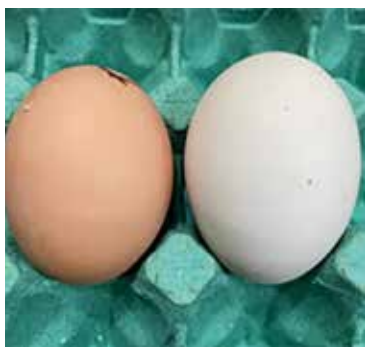
To produce fertile eggs breeders and roosters need a healthy and functional reproductive tract. Poultry fertility can be influenced by internal factors, the ones which compromise spermatogenesis in roosters and in breeders the oocite formation and egg production. External factors are related with the animals copule, bad management or inappropriate conditions of the hatchery.

Signs
Lower hactability, higher percentage of breakable eggs and embryo mortality.

We listed some potential causes that can contribute to these lesions.







Figures 9 – 11: Policistic ovaries and inflammation, testicles atrophy, lower egg quality.

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Mycotoxins

Potential cause	Description of problem	Checklist	Corrective actions
Zearalenone (ZEN)	• Similarity with hormone oestrogen ZEN influences female reproductive tract (cystic oviduct, inflammation) leading toeggsheall quality, reduced hatchability, and poor chick quality.	• Origin of raw materials from supplier/region with history of ZEN contamination. • Check average contamination levels n feed. • Positive for Zen in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.

Pathogens

Potential cause	Description of problem	Checklist	Corrective actions
"Infectious Bronchitis (IBV) "	• IB causes respiratory disease and kidney damage in growers and oviduct infection in adults.	• PCR and serology tests for virus typing.	• Adapt vaccination program to the demands of the field situation in each particular area/epidemiology. • Increase biosecurity level.
Mycoplasma	• Mycoplasmosis causes respiratory disease, synovites, testicular atrophy and oviduct infection in adults.	• PCR and serology tests	• Increase biosecurity level • Constant monitoring of long-cycle bird batches

Others

Potential cause	Description of problem	Checklist	Corrective actions
Hormone status	• An over-stimulation of egg production may lead to excessive levels of estrogen in blood impairing fertility	• Management of laying birds (excessive estrogen stimulation).	• Improve management of laying birds. • Correct lighting program.

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Avian Gout/Kidney Failure

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, liver, mesentery, air sacs or peritoneum.

Signs
Due to its complex etiology, avian gout is difficult to diagnose; however, the most common signs are dehydration, pale combs, depression, swelling and reddening of the feet, which impairs bird movement. Affected chicks appear dull with ruffled feathers and moist vent. Mortality among young chicks is high. There is irregular and excessive enlargement of kidney lobules and cutting the kidney reveals urate crystals. The deposition of urate crystals also can be seen on the visceral organs.

In layers, where it is mainly observed, avian gout can lead to mortalities up to 50%, with 19 – 35-week-old hens most affected.

We listed some potential causes that can contribute to these lesions.





Figures 12 & 13: Kidneys are enlarged and filled with urates.

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Mycotoxins

Potential cause	Description of problem	Checklist	Corrective actions
Ochratoxin A (OTA) Citrinin, Aflatoxins (Afla)	• Due to kidney damage, uric acid excretion is reduced resulting in the accumulation of uric acid in the body. At the farm, leg problems and increased water excretion may be observed.	• Positive for OTA, citrinin or Afla in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS). • Raw materials originated from supplier/region with history of OTA, citrinin or Afla contamination. • Histopathology: Check other target organs of OTA and citrinin (e.g. kidneys). • Overall decline in flock performance. • Check average contamination levels.	• Add an effective mycotoxin deactivator in the feed. • Avoid feed bins or feed/water lines becoming contaminated by stale, wet or moldy feed. • Prevent mold growth, purchase clean raw materials.

Nutrition

Potential cause	Description of problem	Checklist	Corrective actions
Protein	• >30% of protein in feed causes uric acid production leading to excretory loads in kidneys. Concurrently sulphates decrease calcium resorption causing excessive calcium secretion through urine, which favors gout.	• Protein levels in feeds.	• Correct protein levels in feeds.
Excess Ca in chick diets	• Precipitates in the kidney and surrounding tissues.	• Check feed formulation.	• Ensure correct level of Ca in diet.
High Na	• Gout due to sodium intoxication at >0.4% in water and >0.8% in feed.	• Check water quality for salinity and feed formulation to maintain correct levels of salt.	• Ensure water and feed are safe. • Control total sodium chloride content in feed (<0.3%).
Excess Vit D	• Increases Ca absorption from the blood.	• Check premix formulation.	• Correct level of minerals and vitamin D3 in diets.

Pathogens

Potential cause	Description of problem	Checklist	Corrective actions
Infectious Bronchitis (IB) especially nephropathgenic strains (QX)	• IB causes respiratory disease and kidney damage in growers and oviduct infection in adults.	• PCR and serology tests for virus typing.	• Adapt vaccination program to the demands of the field situation in each particular area/epidemiology. • Increase biosecurity level.
Infectious Bursal Disease (IBD)	• IBD is very immunosuppressive and leads to kidney damage.	• Serology tests for antibody level in day old chicks.	• Adapt vaccination program to the demands of the field situation in each particular area/epidemiology.

Others

Potential cause	Description of problem	Checklist	Corrective actions
• Water deprivation	• Water deprivation leads to concentration of uric acid and other minerals in the blood and later in the kidneys.	• Observe animal behavior to understand the cause of water deprivation. • Transportation and vaccination procedures • Drinkers in terms of number, position and blockages that may be impeding animal's access to them. • Chemicals added into 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.

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Black Tongue

Black tongue is a common occurrence in the field originated by inflammation and necrosis of the mouth and upper throat, due to a deficiency of some vitamins or others causes.

Signs
Black tongue is characterized by inflammation of the tongue and mouth cavity. Beginning at about two weeks of age, the entire mouth cavity, as well as the esophagus, becomes distinctly inflamed, growth is retarded and feed consumption is reduced. The lesions can vary from the so-called 'black tongue' in which usually the tip of the tongue is necrotized, to raised, yellow necrotic plaques.

We listed some potential causes that can contribute to these lesions.

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Mycotoxins

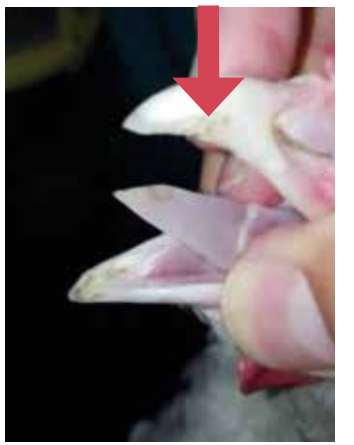
Potential cause	Description of problem	Checklist	Corrective actions
T-2 toxin (T-2) or Diacetoxyscirpenol (DAS)	• Histopathology: Necrosis of the tip of the tongue. Often associated with oral lesions. • Decline in the overall performance of the flock.	• Origin of raw materials from supplier/region with history of T-2/DAS contamination. • Check average contamination levels. • Positive for T-2 and/or DAS in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.

Nutrition

Potential cause	Description of problem	Checklist	Corrective actions
High sunflower inclusion	• Tongue generally totally black without additional oral lesions. • No decline in overall flock performance.	• Check by removing some birds and provide a corn/ soy diet.	• Reduce levels of sunflower in the diet.
Niacin deficiency in diet (niacin hypo-vitaminosis)	• Histopathology: Blackening of the tongue accompanied by inflammation of the oral cavity and esophagus.	• Check formulation of premix.	• Adapt premix as necessary. • Apply group B vitamins and K_3 vitamin in water.
Copper sulphate	• Concentration between 0.05 to 0.2% in feed and excess in drinking water can promote oral lesions.	• Check concentration of $CuSO_4$ in premix. • Check concentration of $CuSO_4$ in water. • Check if water dosing system is working correctly (if applicable).	• Correct set up of the water dosing system.







Figures 14 – 16: Tongue totally or partially black. Can appear associated with inflammation of the oral mucosa.

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Carcass Bruising

The consumer's decision-making process when purchasing poultry products mainly takes into account appearance, hygiene and flavor. Poultry carcass damage or chicken carcass bruising affect a producer's profits.

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 carcasses are free from disease or fecal contamination. In the presence of one (or both) contaminations, carcasses are condemned and withdrawn from the food chain. Carcass bruising/ hemorrhage is one of several reasons leading to carcass 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.

Signs
It is difficult to determine whether bruises 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 color 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.

We listed some potential causes that can contribute to these lesions.



Figures 17 & 18: Hemorrhages on the breast, legs and wings.

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Mycotoxins

Potential cause	Description of problem	Checklist	Corrective actions
Aflatoxin B_1 (Afla)	• Increases capillary fragility leading to bruising. • Animals present jaundice-like symptoms and are dehydrated and emaciated. They present with purple-reddish areas in the carcasses.	• Histopathology of the liver • Origin of raw materials from supplier/region with history of Afla contamination. • Check average contamination levels. • Positive for Afla in raw materials (ELISA , HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.
Fumonisins (FUM)	• Liver cells are affected, reducing the coagulation factors which induces to difused hemorrhages	Evaluation of the liver functionality: hepatic enzymes (AST and ALT) • Origin of raw materials from supplier/region with history of Fum contamination. • Check average contamination levels. • Positive for Fum in raw materials (ELISA , HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.

Nutrition

Potential cause	Description of problem	Checklist	Corrective actions
Vitamin K deficiency	• Impairment of blood coagulation is the major clinical sign of vitamin K deficiency. With a severe deficiency, subcutaneous and internal hemorrhages can prove fatal. • Hemorrhages may appear on the breast, legs, wings, in the abdominal cavity and on the surface of the intestine.	• Vitamin levels in the diet • Presence of sulfur drugs • Stress factors (e.g., coccidiosis and other intestinal diseases).	• Inclusion of menadione at 1 – 4 mg/ton of feed. • Correct drug inclusion. • Intestinal disease control.

Pathogens

Potential cause	Description of problem	Checklist	Corrective actions
Infectious bursal disease (IBD)	• Blood spots mainly in the legs and breast muscles.	• Check maternal antibody levels at placement necropsy: bursa of Fabricius is swollen and bloody.	• Adjust vaccination program to meet challenge conditions. • Increase biosecurity level.

Others

Potential cause	Description of problem	Checklist	Corrective actions
Rough handling at outloading	• Rough handling can result in carcass bruising.	• Handling procedures	• Correct or train where necessary.
Stunning system	• Stunning voltages can result in increased hemorrhagic petechial in legs and breast.	• Voltage and timing of the electric shock.	• Correct the voltage.
Catching	• Poor catching procedures.		• Adjust picking machines and/or catching.
Animal density	• High flock density at grow-out house.		• Correct the density.

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Comb Lesions

Several diseases can cause comb lesions, especially in chronic infections.

Signs
Characteristic gross lesions of affected chickens are swelling and inflamation of the comb. Pale and shrunken comb, depression, loss of appetite, dark comb and wattles, drop in egg production.

We listed some potential causes that can contribute to these lesions.





Figures 19 & 20: Cutaneous lesions on the featherless skin and/or diphtheritic lesions of mucous coats of the upper alimentary and respiratory tract. The lesions can vary according to the stage of development: papules, vesicles, pustules or crusts.

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Mycotoxins

Potential cause	Description of problem	Checklist	Corrective actions
T-2 toxin (T-2), Diacetoxyscirpenol (DAS) or Deoxynivalenol (DON)	• Histopathology: Necrosis of the tip of the comb. Often associated with necrosis of the wattle. Comb often pale in color. • Decline in the overall performance of the flock.	• Origin of raw materials from supplier/region with history of T-2/DAS or DON contamination. • Check average contamination levels. • Positive for T-2 and/or DAS or DON in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.

Pathogens

Potential cause	Description of problem	Checklist	Corrective actions
Fowl pox	• Lesions found on the comb and wattle and in severe cases in the mouth. • Conjunctivitis • Keratitis	• Clinical signs • Poxvirus: virus isolation, fluorescent antibody, electron microscopy, agar gel immunodiffusion	• Correct vaccination program. • Control invertebrate vectors.
Mosquito bites	• Usually found all over the comb and wattle rather than just at the tip of the comb.		• Bioprotection • Control invertebrate vectors.
Leucocytozoonosis	• Severe anemia, leukocytosis.	• Testing by blood screening. • PCR tests.	• Control invertebrate vectors.
Newcastle disease	• The comb may become blue as the bird becomes cyanotic.	• Virus isolation and PCR.	• Biosecurity • Correct vaccination program.
Avian Influenza	• Necrotic foci, hemorrhage and cyanosis of the non-feathered skin have been reported, especially wattles and combs (H5N2).	• Virus isolation and PCR.	• Biosecurity • Correct vaccination program.

Others

Potential cause	Description of problem	Checklist	Corrective actions
Stocking density	• Physical damage to the comb.	• Check areas affected.	• Improve management conditions to remove aggression, improve beak tipping to prevent additional damage.
Percentage of males in breeders		• Check the males number per breeders	• Improve management conditions
Fighting/aggression			• Improve management conditions

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Egg Quality Problems

Bad management practices, feed, and environment-related issues and diseases are some of the factors that may have a negative impact on egg production and egg quality. Additionally, animal-related factors, such as age and strain of layer birds must not be disregarded. Older birds and birds after molting 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 may lead to nervous birds and/or traumatic lesions in the ovary which causes poor egg quality. In addition, nutrition issues, such as improper balance of calcium, phosphorus and vitamin D, may lead to thin eggshells. Further, due to the liver and kidney toxicity, caused by substances like mycotoxins, egg and shell formation can be negatively impacted.

Signs
Shell-less eggs, dirty eggs, blood stained eggs, cracks, calcium deposits, bloodspots/meat spots, wrinkled eggs, mottled yolks and discolored yolk, rotten eggs, etc.

We listed some potential causes that can contribute to these lesions.



Figure 21: Ovaries, severe inflammatory and dystrophic changes in the mucous coat.

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Mycotoxins

Potential cause	Description of problem	Checklist	Corrective actions
Aflatoxin B_1 (Afla)	• Thin shelled eggs due to the disruption of the vitamin D pathways through the liver and kidneys resulting in a reduction of the calcium transporting proteins in the blood.	• Origin of raw materials from supplier/region with history of Afla contamination. • Check average contamination levels. • Positive for Afla in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or mouldy feed. • Add an effective mycotoxin deactivator in the feed.
Ochratoxin A (OTA)	• Increased numbers of blood and meat spots in the eggs.	• Origin of raw materials from supplier/region with history of OTA contamination. • Check average contamination levels. • Positive for OTA in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.
Trichothecenes (T-2 toxin, HT-2, DAS)	• Histopathology: Proliferating epithelial cells. Hepatic vacuolization. • Decline in the overall performance of the flock.	• Origin of raw materials from supplier/region with history of trichothecenes (T-2, HT-2, DAS) contamination. • Check average contamination levels. • Positive for trichothecenes (T-2, HT-2, DAS) in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or mouldy feed. • Add an effective mycotoxin deactivator in the feed.
Cyclopiazonic acid (CPA)	• Potent inhibitor of the Ca_2+-activated ATPase activity of the calcium pump. • Focal necrosis can be found in the liver, spleen, kidneys, pancreas and myocardium. In broiler chicks given CPA, skeletal muscle degeneration characterized by myofibular swelling and fragmentation.	Origin of raw materials from supplier/region with history of Zen contamination. • Check average contamination levels. • Positive for Zen in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.
Zearalenone (ZEN)	• Influence female reproductive tract (cystic oviduct, inflammation) leading to deficient formation of the egg	Origin of raw materials from supplier/region with history of Zen contamination. • Check average contamination levels. • Positive for Zen in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.

Nutrition

Potential cause	Description of problem	Checklist	Corrective actions
Ca presentation	• Layers particularly and also breeders need to store Ca in the gizzard for the period when egg shell production is taking place, throughout the dark period. Small particle Ca can pass through the gizzard making it unavailable for egg shell production.	• Check if the correct particle size Ca is incorporated into the feed.	• Ensure the correct ratio of 65% coarse limestone and 35% limestone flour is used.
Ca:P ratio	• Too high phosphorus can have a negative effect on eggshell quality.	• Check if the levels of Ca and P in the formulation are correct for the age of the birds.	• Increase Ca:P ratio with the flock aging to improve the requirements for Ca as the eggs increase in size.

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Pathogens

Potential cause	Description of problem	Checklist	Corrective actions
Infectious bronchitis virus (IBV)	• Many serotypes cause drops in egg production accompanied by reduced shell quality and albumen quality. Loss of shell color in brown egg layers is a very common effect of IBV, although it may also cause the production of thin shelled, misshapen and corrugated eggs as well as more elongated eggs.	• Isolation in embryonated eggs • Virus neutralization assay • Immunodiffusion in agar gel • Hemagglutination inhibition test (HI).	• Correct vaccination plan.
Newcastle disease	• Many soft shelled eggs with membranes visible on the manure belts. Misshapen eggs produced by commercial layers previously vaccinated with live vaccines. Eggs were produced 30 days after challenge with vNDV as the only clinical sign of ND.	• Virus isolation and PCR.	• Correct vaccination plan.
Mycoplasma	• Cause problems in the respiratory tract and within commercial layer flocks, it has also been responsible for salpingitis, reduced egg production and poor egg quality.	• Clinical signs and post mortem lesions followed by confirmation in the laboratory using blood (serum) samples for serology or organ swabs for identification by PCR or mycoplasma isolation.	• Correct vaccination plan.



Figure 22: De-formed and pigmentless eggs.



Figure 23: Reduction in eggshell thickness.

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Others

Potential cause	Description of problem	Checklist	Corrective actions
Red mites	• Birds are stressed (older hens) and a reduction in feed intake might be observed. The decrease in Ca intake leads then to worse eggshell quality.	• Monitor the feed intake.	• Flame cages during downtime period. • Clean egg belts during downtime period. • Increase biosecurity level.
Nervous birds	• Can cause 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.
Heat Stress	• Reduction in eggshell thickness and color during periods of heat stress due to a reduction in feed consumption, and changes in the blood chemistry, thereby reducing the transportation of calcium.	• Check the birds for panting.	• Increase air movement over the birds, add electrolytes to the water during the period of heat stress.

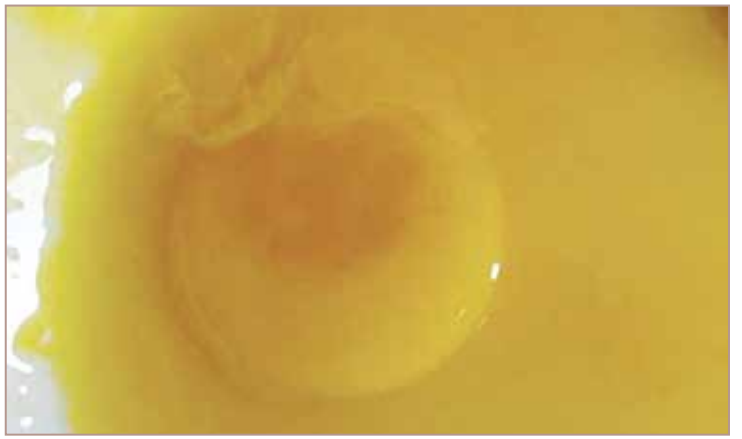


Figure 24: Pigmentless, watery yolk and egg white.

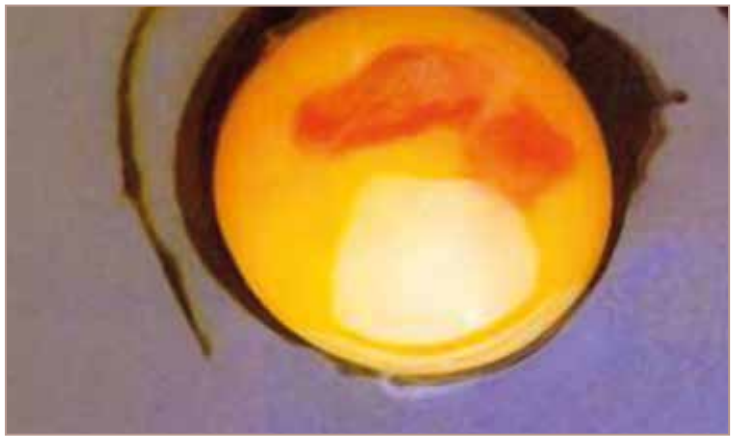


Figure 25: Blood and meat spots in yolk and egg white.

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Fatty Liver Syndrome and Liver Lesions

The liver is a vital organ and is responsible for multiple metabolic roles. Fatty liver syndrome is a non-infectious condition that affects broilers, laying or breeding hens, especially caged animals. When a chicken develops this syndrome, it means that their normal liver cells are gradually accumulating fat which causes them to no longer work efficiently. Over time these liver cells may be destroyed. Some strains appear to be more susceptible than others.

Signs
The liver is usually enlarged, putty colored and friable, showing varying degrees of hemorrhage. The abdominal cavity often contains large amounts of oily, unsaturated fat. Nonspecific signs include lethargy, weight loss, reduced appetite, increased water consumption and poor feather condition can be observed. Other signs are pale comb greenish diarrhea or yellow droppings, a quick drop in egg-laying in a non-implanted hen, lethargy or a refusal to move and enlarged or distended abdomen.

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

We listed some potential causes that can contribute to these lesions.

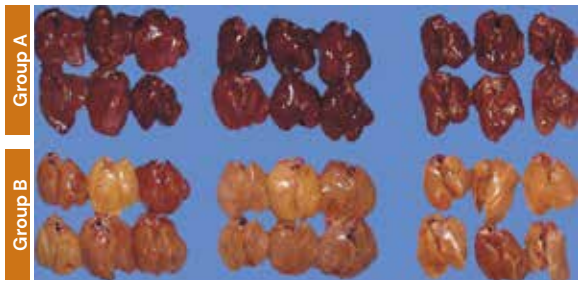


Figure 26: (Group A) Normal liver. (Group B) Fatty liver caused by chronic aflatoxin exposure.



Figure 27: The liver is enlarged, yellow, greasy and soft.

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Mycotoxins

Potential cause	Description of problem	Checklist	Corrective actions
Aflatoxin B_1 (Afla)	• Young animals: Fibrosis of liver leads to hardening of the organ. • Older animals: Hepatic lipidosis, with softening of the organ. • Pale friable livers seen at slaughter.	• Histopathology of the liver • Origin of raw materials from supplier/region with history of Afla contamination. • Check average contamination levels. • Positive for Afla in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.
Fumonisins (FUM)	• Causes oxidation in hepatic cells reducing their functionality and/or necrosis. • It results in accumulation of fat in the liver	Histopathology of the liver • Origin of raw materials from supplier/region with history of Fum contamination. • Check average contamination levels. • Positive for Fum in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines • by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.

Nutrition

Potential cause	Description of problem	Checklist	Corrective actions
Energy-protein balance	• Excessive energy in diets can cause lipidosis and fatty liver problems.	• Carbohydrate level in diet. • Energy-protein ratio in diet.	• Avoid high-carbohydrate diets, especially in summer. • Adopt proper energy-protein ratio.
Poor quality fat in the diet	• Peroxides can impair the liver activity, creating fatty liver.	• Quality of fats in term of peroxide value, rancidity and free fatty acids.	• Avoid low-quality fats (e.g., Avoid low-quality fats if possible, or only use low-quality fats in the grower/finisher phases.) • Replace animal fats with vegetable fats.
Choline/biotin deficiency	• Pale friable livers seen at slaughter.	• AA scale at feed mill.	• Increase level of synthetic amino acids in the diet.

Pathogens

Potential cause	Description of problem	Checklist	Corrective actions
Viral hepatitis (IBH – Viral inclusion body hepatitis)	• Adenovirus causes yellow/hemorrhagic liver and focal necrosis. Symptoms include immunosuppression, diarrhea, anorexia, depression, ruffled feathers, especially in the region of head and neck.	• Necropsy: Macroscopic lesion in the enlarged, dystrophic liver with yellowish color and crumbly texture; enlarged kidneys. • Histopathology: Detection of intranuclear inclusion bodies. • Serotype identification by serological assays.	• Use inactivated vaccines. • Check the breeding stock and eliminate the affected birds.

Others

Potential cause	Description of problem	Checklist	Corrective actions
Full fed broilers at slaughter	• High lipid concentrations in the blood at slaughter can lead to paler livers.		• Fast birds before slaughter.
Hormone status	• An over-stimulation of egg production may lead to excessive levels of estrogen in blood that facilitate fat storage in the liver and occurrence of fatty liver, especially in layers and breeders.	• Management of laying birds (excessive estrogen stimulation).	• Improve management of laying birds. • Correct lighting program.

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Gizzard Lesions

The muscular stomach or gizzard serves a more mechanical purpose, aiding digestion by particle size reduction and regulation of feed flow. The gizzard 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.

In spite of being a fairly strong organ, the occurrence of gizzard 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 whole set of potential causes are worth consideration.

Signs
Gizzard erosion and ulceration, sporadic increase in mortality, gizzard condemnation at slaughterhouse, etc.

We listed some potential causes that can contribute to these lesions.

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Mycotoxins

Potential cause	Description of problem	Checklist	Corrective actions
Type A trichothecenes particularly T-2 toxin and Diacetoxyscirpenol	• Mild to severe disruption of the koilin layer, which in severe cases causes lesions in the underlying tissues.	• Standard post mortem examinations. • Origin of raw materials from supplier/region with history of type A trichothecenes contamination. • Check average contamination levels. • Positive for A-trichothecenes in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.

Nutrition

Potential cause	Description of problem	Checklist	Corrective actions
Biogenic amines	• Low-quality/overly processed fish meal can result in high levels of gizzerosine. • Hyper-production of HCl in the proventriculus causes erosions in the gizzard.	• Toxicity, rancidity and peroxide tests.	• Lower level of fishmeal in diet. • Avoid use of low-quality fishmeal. Replace standard fish meal with low temperature (LT) fishmeal.
High levels of copper sulphate in diet	• $CuSO_4$ can promote gastric lesions especially at proventriculus and gizzard level.	• Check formulation to ensure that levels of copper sulphate are at the correct level in premixes.	• Correct set up of the water dosing system.
Rancid fats	• Low-quality fats (long storage, overheated) can have high levels of superoxide radicals and hydroxyl radicals.	• Quality of fats in term of peroxide value, rancidity and free fatty acids.	• Avoid low-quality fats (e.g., Avoid low-quality fats if possible, or only use low-quality fats in the grower/finisher phases.) • Replace animal fats with vegetable fats.
Acetylsalicylic acid and sodium salicylate	• Use of salicylates may induce proventriculus and gizzard ulceration.	• Dosage of salicylates used (check overestimation of feed intake in feed restricted animals). • Mixability of commercial product in water.	• Avoid low-quality products (low mix ability, low homogeneity in water). • Correct feed intake assumption in feed restricted animals.
Tannins	• Toxic levels of tannins in the feed cause esophageal and gastric edema, hemorrhagic ulceration, necrosis and sloughing of the mucosal lining.	• Level of tannins in some raw materials (sorghum) and in tannin-based products.	• Use high-quality tannin-based product (chestnut is better than quebracho). • Reduce % of sorghum in diet when high in tannins.



Figure 28: Gizzards without alterations.

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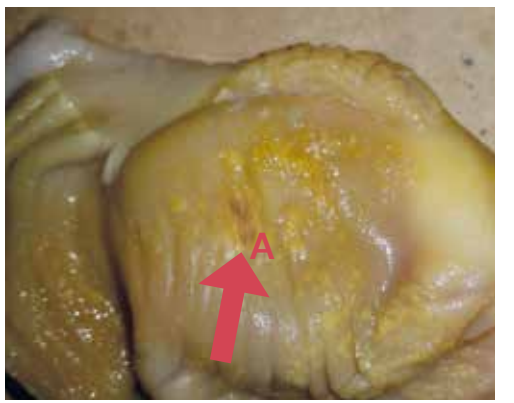
Pathogens

Potential cause	Description of problem	Checklist	Corrective actions
Adenovirus contamination	• Adenovirus is the causal agent of inclusion body hepatitis (IBH) and other avian syndromes that affect the production. • Vertically transmitted, usually sub-clinical but provides more exposure to secondary bacterial infection.	• Check for high titre level in the blood (ELISA). • The serum neutralization test.	• Biosecurity • Use inactivated vaccines. • Check the breeding stock and eliminate the affected birds.
Newcastle disease	• Slight wounds, hemorrhage and necrosis of proventriculus - more often then generalized lesions.	• Virus isolation and PCR.	• Correct vaccination plan. • Biosecurity
Infectious bursal disease (IBDV/ Gumboro)	• IBD is very immunosuppressive and causes lesions at the junction of the proventriculus-gizzard.	• Atrophy of the bursa of Fabricius. Immunofluorescence or immunohistochemistry tests.	• Biosecurity. • Implement/correct vaccination program in breeders. • Change from mild to strong-reaction vaccine.
Candida albicans (Candidiasis)	• Thickening of the mucosa in the crop with white to off-white, raised circular or rugose lesions.	• Observation of gross lesions. Diagnosis can be confirmed by demonstrating tissue invasion histologically and by culture of the organism.	• Improve sanitation and minimize antibiotic use in poultry.



Figure 29: Gizzards without problems.





Figures 30 & 31: (Arrow A) Erosions and ulcers in gizzard cuticulum.

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Others

Potential cause	Description of problem	Checklist	Corrective actions
Prolonged periods without feed	• Hyper-production of HCl in the proventriculus causes erosions in the gizzard.	• Check that feeder systems are working correctly	• Reduction of the transport time.
Delay in pulling chicks	• Can result in a long period in the hatcher.	• Check egg setting times and egg ages.	• Adapt setting times to ensure a short hatch window.
High temperatures in the hatcher (especially between 17 - 20 days)	• Hyper-production of HCl.	• Check the temperature during the incubation period.	• Adjust the temperature and properly calibrate the incubation machines.



Figure 32



Figure 33

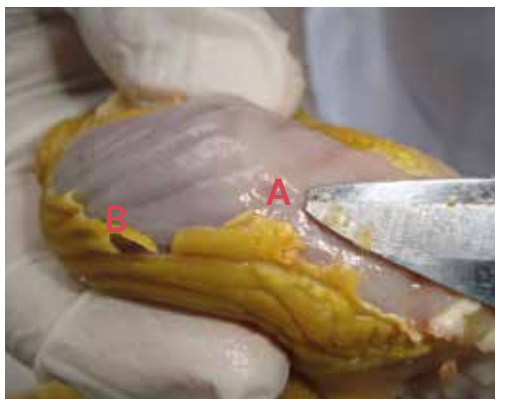


Figure 34: (Arrow B) Mucosal surface (koilin layer) peels away easily.

Figures 32 – 34: (Arrow A) Erosions and ulcers in gizzard cuticulum.

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Impaired Feathering or Feather Loss

Impaired feathering or feather loss can manifest in various ways, such as birds losing feathers on the neck, back, bottom, around the neck or chest. It may be more or less extensive and result from a variety of factors.
In poultry, feathers serve important roles in terms of protection and insulation of the body. While molting, or renovation of older feathers by new ones, is a natural process occurring in mature layers upon completion of a laying cycle, 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 of causes, either:
1. Feathers are not properly developed (linked to feather formation) which is often related to nutrition or the presence of mycotoxins.
2. Feathers are pulled off by birds (feather pecking), which is a management-related issue.

Causes of losing feathers
In each case, it is critical to understand the foundation of the problem so that it can be properly solved. Stressful conditions in the barn, especially during brooding, such as heat, cold and existence of air currents, among others, can result in feather loss and poor feather quality in the birds. In this case, it is crucial that the behavior and interaction of animals are observed. Often, feather pecking and pulling can also be triggered by inadequate intake of nutrients.

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 optimized to reduce this occurrence.

We listed some potential causes that can contribute to these losses.





Figures 35 & 36: Poorly feathered skin areas and impaired feathering growth.

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Mycotoxins

Potential cause	Description of problem	Checklist	Corrective actions
T-2 toxin (T-2)/ Deoxynivalenol (DON)/ Other trichothecenes	• Feathers are narrow due to radiomimetic injury to the developing barbs.	• Origin of raw materials from supplier/region with history of T-2/DON contamination. • Check average contamination levels. • Positive for T-2/DON in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.

Nutrition

Potential cause	Description of problem	Checklist	Corrective actions
Non-starch polysaccharides (NSP)	• Impairment of amino acid digestibility can affect gut health and leads to impaired gut integrity. Rough raw materials, overcooked soya and enzyme quality formulation together with NSPs can result in impaired feathers.	• Check NSP levels. Check feed composition and commodities.	• Maintain the correct levels of NSPs. Adapt commodities for best feed quality.
Amino acid (AA) deficiency/ imbalance	• Impaired feather formation and feather pecking.	• Check levels of total sulphur amino acids (TSAA) in the diet. • Ratio TSAA/Lys/Arg/Thr. • AA scale at feed mill.	• Maintain the correct levels of sulphur amino acids • Increase level of synthetic amino acids in the diet.

Pathogens

Potential cause	Description of problem	Checklist	Corrective actions
Mites	• Poor feathering, ruffled feathers and pecking.	• Presence of red mites in the barn during the night.	• Flame cages during downtime period. • Clean egg belts during downtime period. • Increase biosecurity level.
Infectious bursal diesease (IBD)	• IBD is very immunosuppressive and leads to impaired feathering.	• Check maternal antibody levels.	• Adapt vaccination program to meet the risks of infection as per field challenge.

Others

Potential cause	Description of problem	Checklist	Corrective actions
High house temperatures	• Pecking when associated with low humidity, also possibility of nutritional deficiencies.	• Check temperature and ventilation system.	• Maintain correct temperature and ventilation.
Overstocking	• Direct competition for feeders and drinkers leading to pecking, or nutrient deficiencies.	• Maintain correct ratio of feeders and drinkers.	• Reduce placement numbers.

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Lameness

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 wellbeing and has severe economic consequences from poor growth, increased culling and mortality, carcass 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. Conditions responsible for lameness can be of infectious or non-infectious origin. It is important to identify and understand these risk factors in order to develop a prevention or mitigation strategy to reduce the incidence of lameness in poultry flocks.

The table below focuses on lameness conditions and suggests solutions that can help mitigate the damage caused by these conditions.

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Mycotoxins

Potential cause	Description of problem	Checklist	Corrective actions
Deoxynivalenol (DON)	• Histopathology with translocation of bacteria from the intestine due to disruption of the tight junctions to the femoral head, where ongoing pressure of growth results in micro fractures and eventually degeneration of the joint. • Reduced growth with increased culling at out-loading of damaged birds; Increased further processing due to the poor quality of the femur head.	• Origin of raw materials from supplier/region with history of DON contamination. • Check average contamination levels. • Positive for DON in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.
Fumonisins (FUM)	• Reduce the expression of the tight junctions, reducing the calcium absorption and bone mineralization. It leads to more fragile bones and leg problems	Origin of raw materials from supplier/region with history of Fum contamination. • Check average contamination levels. • Positive for Fum in raw materials (ELISA, HPLC or LCMS/ MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed

Nutrition

Potential cause	Description of problem	Checklist	Corrective actions
Rickets - Vitamin D3 deficiency, Ca/P imbalance	• Enlargement of the ends of tibia and femur.	• Check the feed composition.	• Feed vitamin D3 with balanced calcium and phosphorus.
Ionophore toxicity-Potential cause: Monensin	• Legs extended backward.	• Mix feed properly.	• Withdraw the ionophore
Osteoporosis/cage layer fatigue – Impaired calcium flux in laying hens	• Soft and rubbery bones, birds on their sides in the back of the cage. • Lesions: Vertebral fracture affecting spinal cord.	• Check the feed composition.	• Feed appropriate calcium according to stage of production.
Pododermatitis/footpad dermatitis/foot burn/ ammonia burn – Biotin deficiency, poor quality litter	• Ulceration of the metatarsal and digital footpads. • Lesions: Necrotic lesions on the plantar surface of the footpads.	• Check the feed composition.	• 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 – Ca/P ratio, excess chloride in feed metabolic acidosis, acid/base balance, mycotoxins	• Swelling and bowing in the region of the knee joints, angulations of legs, typically in birds >35 days. • Plug of cartilage in proximal end of tibia, distal tibia and proximal metatarsus, in decreasing order of frequency.	• Check the feed composition.	• Correct the nutritional imbalances. • Add an effective mycotoxin deactivator in the feed.
Perosis/chondrodystrophy - Manganese deficiency	• Thickened and shortened legs, shortened wings. • 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.	• Check the feed composition.	• Feed appropriate manganese according to the stage of production. • Maintain Mn/Ca/P balance

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Pathogens, Bacterial

Potential cause	Description of problem	Checklist	Corrective actions
Bacterial chondronecrosis with osteomyelitis (BCO) (Causes include: *Enterococcus cecorum, Streptococcus spp., Staphylococcus spp, E. coli*)	• Bird will be sitting on its breast/keel, with the legs directed forward, use of wings for walking support and hip flexion. • Necrotic degeneration and microbial infection, primarily within the proximal heads of the femur and tibia.		• Improve gut integrity by feeding multi-strain, poultry-specific, live probiotic. • Antibiotics depending on severity, but mostly birds are euthanized.
Vertebral Osteomyelitis/Spondylitis/ Spondylopathy/Spondylolisthesis/ Kinkyback (Causes include: *Enterococcus cecorum, Staphylococcus spp., E. Coli.*)	• 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. • Abscess and/or necrosis in T4-T7 vertebrae, dorsal buckling of spinal cord (kyphosis), interstitial edema, atrophy, degeneration of muscle fibers.		• Improve gut integrity by feeding multi-strain, poultry-specific, live probiotic. • Antibiotics depending on severity, but mostly birds are euthanized.
Bumble Foot (Causes include: *Staphylococcus spp.*)	• Swelling of affected feet. • Abscess may have clear exudate with fibrin clots.		• Improve gut integrity by feeding multi-strain, poultry-specific, live probiotic. • Antibiotics
Fowl Cholera (Causes include: *Pasteurella multocida*)	• Swollen hock joints, swollen wattles and comb, greenish diarrhea. • Necrotic foci on liver, petechiae in the epicardial fatty tissues.		• Vaccines only if endemic. • Antibiotics
Bacterial arthritis/Airsacculitis (MS) (Causes include: *Enterococcus faecalis, Mycoplasma synoviae*)	• Ruffled feathers, swollen hock joints and feet, bilaterally asymmetrical legs. • Joints and tendon sheaths have viscous grey to yellow exudate, caseous exudate from the lesions.		• Improve gut integrity by feeding multi-strain poultry-specific live probiotic. • Antibiotics and eradication of infected breeding stock.
Osteomyelitis Complex (Causes include: Bacterial, but no specific pathogen has been identified.)	• Green discoloration of liver, inflammatory lesions in bones and joints.	Only identified at slaughter.	• Improve gut integrity by feeding multi-strain poultry-specific live probiotic.

Pathogens, Viral

Potential cause	Description of problem	Checklist	Corrective actions
Tenosynovitis/Viral arthritis (Causes include: *Avian reovirus*)	• Soft swelling of the joints with turbid fluid in the capsule, swollen shanks. • Swelling and petechiae in the synovial membranes, small erosions on the articular cartilage, adhesions between the tendons and fibrosis of tissues.		• Live vaccine followed by inactivated vaccine. • Euthanize the infected flock.
Amyloidosis (Causes include: *Corona virus*)	• Swollen hock joint containing orange-yellowish material, muscular shivering. • Extracellular buildup of amyloid protein in joints and internal organs.		• Live vaccine. • Sodium salicylate 1g/liter (acute phase). • Antibiotics to control secondary colibacillosis.
Infectious bronchitis (IB)/Infectious laryngo tracheitis (ILT)	• Respiratory symtpom complex. • Difficulty in breathing, respiratory sounds.		• Live vaccine. • Sodium salicylate 1g/liter (acute phase). • Antibiotics to control secondary colibacillosis.
Marek's disease	• One leg stretched forward and the other backward. • Tumors in internal organs, unilateral enlargement of peripheral nerves.		• Live vaccine. • Eradication of infected flock.
Avian encephalomyelitis (AE)	• Trembling of the head, neck, and wings, paralysis of both legs extended out to one side. • Gross lesions are mild or absent, focal white areas in gizzard muscle.		• Vaccination of breeders.
Newcastle disease	• Twisting of neck and paralysis of wings and legs, cyanosis of comb, facial edema, green diarrhea, drop in egg production, sudden death. • Hemorrhage in intestine, petechial hemorrhage in proventriculus, congestion and mucoid exudates seen in the respiratory tract, especially in trachea.		• Live vaccine. • Eradication of infected flock.

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Others

Potential cause	Description of problem	Checklist	Corrective actions
Tibial dyschondroplasia	• Swelling and bowing in the region of the knee joints, angulations of legs, typically in birds >35 days. • 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.
Twisted leg	• Genetic selection, stocking density. • Distortion at hock, valgus/varus, various angulations of legs. • Linear twisting of tibia and femur, changed angulation of tibial condyles.		• Euthanize affected bird.
Pododermatitis	• Ulceration of the metatarsal and digital footpads. • Necrotic lesions on the plantar surface of the footpads.	Check biotin in the feed.	• Lower litter moisture with proper ventilation and avoid water spillage. • Improve gut integrity by feeding poultry-specific, live probiotics. • Supplement biotin in the feed.
Degenerative joint disease	• Developmental defects, physical damage. • Imbalanced walking, huddling. • Damaged epiphyseal articular cartilage, especially of femoral anti-trochanter, but also other leg joints, resulting in erosions and cartilage flaps.		• Euthanize affected bird.

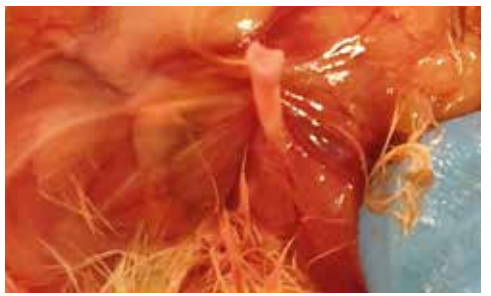


Figure 37: Day one - normal proximal femoral head.

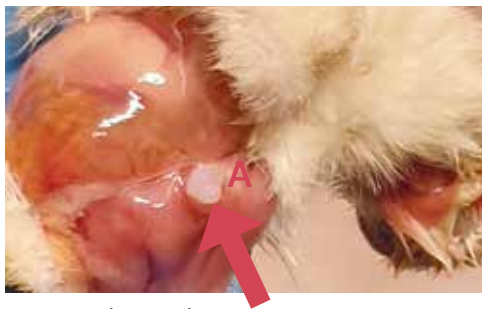


Figure 38: (Arrow A) normal proximal femoral head.

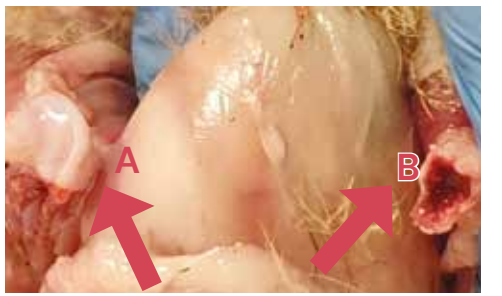


Figure 39: (Arrow A) normal proximal femoral head. (Arrow B) perforation.

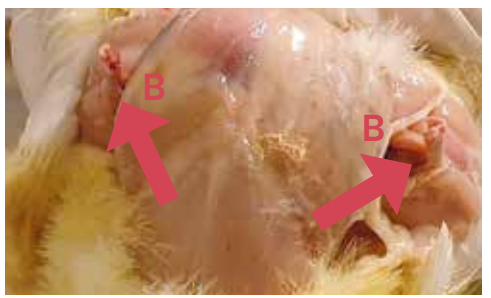


Figure 40: Perforation, fracturing and osteomyelitis of the femoral head (Arrow B).

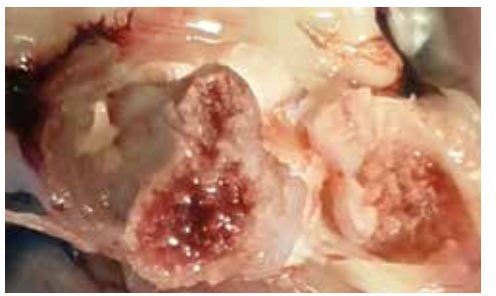


Figure 41: Femoral head necrosis.

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Oral Lesions

Oral lesions or cankers are particularly prevalent in laying hens, breeders or broilers, 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.
Also related to feeding 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 mismanagement of injectors which may produce points of high concentration of methionine and other components in the feed, ultimately leading to similar lesions. These lesions induce not only the loss of genetic potential, but required a lot of investments to treat symptoms or underlying illnesses.

Signs
The common pathological lesions are crusts on the beak, yellow caseous ulcers at the margin of the beak, mucous of the hard palate and the angle between mouth and tongue; ulcers may extend to the esophagus and larynx, gizzard erosion. Additional symptoms commonly found are decreased feed intake, including feed refusal or prolonged feed finish, growth depression, inhomogeneous flocks and impaired FCR.

We listed some potential causes that can contribute to these lesions.



Figure 42: Small particles of feed block saliva ducts, which may result in oral lesions.

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Mycotoxins

Potential cause	Description of problem	Checklist	Corrective actions
T-2 toxin (T-2) or Diacetoxyscirpenol (DAS)	• Histopathology: Proliferating epithelial cells. Hepatic vacuolization. • Decline in the overall performance of the flock.	• Origin of raw materials from supplier/region with history of T-2/DAS contamination. • Check average contamination levels. • Positive for T-2 and/or DAS in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.

Nutrition

Potential cause	Description of problem	Checklist	Corrective actions
Feed granulometry	• Pelletized feed: fine particles >20%. • Histopathology: Presence of inflammatory cells and bacteria. • No severe decline in overall flock performance.	• Check geometric mean particle diameter.	• Adjust the pelleting process. • Increase in sieve diameter. • Use of pellet binders to improve pellet quality.
Feed structure	• Feed structure too hard or soya overcooked, besides others. Histopathology: Presence of inflammatory cells and bacteria. • Decline in overall flock performance.	• Check feed structure and composition of commodities.	• Add electrolytes together with vitamins, increasing chlorine levels beyond the standard recommendation but taking care on water intake.
Liquid methionine	• Histopathology: Infiltration of inflammatory cells. Necrotic lesions. • No decline in overall flock performance.	• Methionine injector dripping inside masher.	• Clean/replace methionine injectors.
Organic acids	• Histopathology: Infiltration of inflammatory cells. Necrotic lesions. • No decline in overall flock performance.	• Acids injector dripping inside mixer.	• Clean/replace acid injectors. • Adjust dosage of organic acids.
Copper sulphate ($CuSO_4$)	• Concentration between 0.05% to 0.2% in feed and excess in drinking water can promote oral lesions.	• Check concentration of $CuSO_4$ in premix. • Check concentration of $CuSO_4$ in water. • Check if water dosing system is working correctly (if applicable).	• Correct set up of the water dosing system.

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PATHOGENS & PARASITES

Potential cause	Description of problem	Checklist	Corrective actions
Candida albicans (Candidiasis)	• The yeast *C. albicans* can lead to lesions detected in the crop but can extend to other parts, including the proventriculus and mouth. • More common in birds with longer lifespans, such as layers and breeders.	• Histopathology: Fungal hyphae present in affected mucosa.	• Antifungal treatment, supportive therapy.
Fowl pox (Avian pox)	• Viral disease caused by Poxviridae (Avipoxvirus) often leads to cutaneous lesions on head, neck, legs and feet. • Dry pox: Raised, wart-like lesions on feathered areas (head, legs, vent, etc.) which heal in about two weeks. Unthriftiness and retarded growth are typical symptoms. • In laying hens, infection results in a transient decline in egg production. • Wet pox: Canker-like lesions in the mouth, pharynx, larynx and trachea. May also cause respiratory distress by obstructing the upper air passages.	• Flock history and presence of typical lesions • Laboratory diagnosis by tissue or transmission studies	• Use preventive vaccination depending on prevalence and season (typically fall). • Treat affected birds with antibiotics to reduce secondary infection, although the disease has to run its course.
Protozoans	• Protozoans are more prevalent in birds with a longer lifespan, such as layers, breeders and turkeys, game birds and/or free-range birds.	• Histopathology: Microscopic examination of a smear of mucus or fluid from the throat demonstrates the presence of trichomonas.	• Separate chronically infected birds from breeding birds.
Trichomonas gallinae	• First lesions appear as small, yellowish areas on the oral mucosa.	• Cankers referred as “yellow buttons” – yellow, rounded areas with central caseous necrotic foci.	• Use nitroimidazoles (not approved in US by FDA and prohibited in the EU).
Histomonas meleagridis	• Also known as histomoniasis or blackhead disease. Common in commercial turkeys and chickens.	• Cecal inflammation. ulceration, thickening of wall, ceca containing yellowish cheese-like exudate.	• Use nitroimidazoles (not approved in US by FDA and prohibited in the EU).

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OTHERS

Potential cause	Description of problem	Checklist	Corrective actions
High temperatures	• Possible decline in overall flock performance. • Increased mortality.	• Calibration of the temperature control system.	• Apply vitamins in water. • Apply organic acids in water. • Provide fresh water.



Figure 43



Figure 44



Figure 45

Figure 43 – 47: Ulcers, necroses, crusts and extensive epithelial erosion of the buccal mucosa.



Figure 46



Figure 47

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Proventriculitis

Lesions in the proventriculus have been reported worldwide in poultry production. Proventriculitis is a condition characterized by enlargement of the proventriculus and fragility of the gastric isthmus, predisposing birds to organ rupture during the evisceration of the carcass. Many agents have been implicated with proventriculitis either alone or in combination. They may be infectious or dietary agents. Despite the widespread occurrence and equally large efforts to understand the pathogenesis of these syndromes, the causative agent(s) has not been established. The major economic problem caused by proventriculitis is its ability to affect overall flock performance. Broilers suffering from this disease have increased feed conversion rate and poor growth rate, which translates to large amounts of feed needed to reach target weights.

Signs
Dilated proventriculus, its wall is thickened and in its mucous coat, fibrinous necrotic and hemorrhagic lesions are detected.

We listed some potential causes that can contribute to these lesions.







Figures 48 – 50: Hemorrhagic lesions at the glandular part of proventriculus. Microscopically, degeneration and necrosis of the proventricular glandular epithelium is accompanied by marked lymphocytic infiltration.

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Mycotoxins

Potential cause	Description of problem	Checklist	Corrective actions
Cyclopiazonic acid (CPA)	• Enlarged with a noticeable thickening of the walls of the organ. May be accompanied with gizzard erosion.	• Origin of raw materials from supplier/region with history of CPA contamination. • Check average contamination levels. • Positive for CPA in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.
T-2 toxin	• Necrosis of the mucosa in the proventriculus and gizzard. Considered the main cause for gizzard erosion.	• Origin of raw materials from supplier/region with history of T-2 contamination. • Check average contamination levels. • Positive for T-2 in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.
Oosporein	• Isthmus circumference is enlarged with possible necrosis of the mucosa. Can be accompanied with pseudomembranous exudate.	• Origin of raw materials from supplier/region with history of oosporein contamination. • Check average contamination levels. • Positive for oosporein in raw materials (ELISA, HPLC or LC-MS/MS) or feed (HPLC or LC-MS/MS).	• Prevent mold growth, purchase clean raw materials. • Avoid contamination of feed bins or feed/water lines by stale, wet or moldy feed. • Add an effective mycotoxin deactivator in the feed.

Nutrition

Potential cause	Description of problem	Checklist	Corrective actions
Biogenic amines	• Low-quality/overly processed fish meal can result in high levels of gizzerosine. • Hyper-production of HCl in the proventriculus causes erosions in the gizzard.	• Ensure only high-quality fishmeal or animal by-products are incorporated into the diet.	• Ensure only good quality animal by-products and fishmeal are purchased.
High levels of copper sulphate in diet	• Copper sulfate can promote gastric lesions especially at proventriculus and gizzard level.	• Check formulation to ensure that levels of copper sulphate are at the correct level in premixes.	• Correct set up of the water dosing system.
Low fiber diet	• Fine granulometry stimulates hyperplasia and decreases the size of the gizzard.	• Bromatological analysis	• Check the formulation and modify where necessary.

Pathogens

Potential cause	Description of problem	Checklist	Corrective actions
Velogenic Newcastle disease infection	• Thickening of the proventriculus, often associated with hemorrhages in the proventricular mucosa.	• Virus isolation and PCR.	• Correct vaccination plan.
Reo virus	• Inflammation, dilatation and thinning of the organ, decreasing the speed of feed passage.	• Histopathology and PCR.	• Correct vaccination plan.
Infectious proventriculus virus associated with IBD	• IBD is very immunosuppressive and causes lesions at the junction of the proventriculus-gizzard.	• Atrophy of the bursa of Fabricius. • Immunofluorescence or immunohistochemistry tests.	• Biosecurity • Implement/correct vaccination program in breeders. • Change from mild to strong-reaction vaccine.

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