Veterinary aspects of the broiler breeding

Summer term 2010
Broiler chickens

Standard intensively farmed broiler chickens are reared to their slaughter weight of around 1.8 to 3 kg within just 6 weeks of being hatched (chickens are normally fully grown by 5-6 months).

By selective breeding, the length of time broiler chicks take to grow to 2 kg has been halved in the last 30 years.

As broilers are bred to grow as fast as possible this has lead to them becoming more inactive. Their frame cannot support their own weight and this affects the way they walk and puts additional stresses on their hips and legs.

At just 6 weeks old, they spend 76%-86% of their time lying down. Birds severely crippled and deformed die of starvation and thirst, unable to reach food or water. Other birds may only be able to move by using their wings to balance. The Farm Animal Welfare Council (FAWC) describes these birds as "obviously distressed".

Broilers have a mortality rate 7 times that of young laying hens of the same age.
Omphalitis, Yolk Sac Infection

A condition seen worldwide in chickens, turkeys and ducks due to bacterial infection of the navel and yolk sac of newly hatched chicks as a result of contamination before healing of the navel.

Disease occurs after an incubation period of 1-3 days.

Various bacteria may be involved, especially *E. coli*, *Staphylococci*, *Proteus*, *Pseudomonas*.

Morbidity is 1-10% and mortality is high in affected chicks.

It is seen where there is **poor breeder farm nest hygiene**, use of floor eggs, inadequate hatchery hygiene or poor incubation conditions, for example poor hygiene of hatching eggs, and poor hygiene of setters, hatchers or chick boxes.

**Inadequate incubation conditions** resulting in excessive water retention and slowly-healing navels and 'tags' of yolk at the navel on hatching also contribute to the problem.
Omphalitis, Yolk Sac Infection

Signs

• Dejection.
• Closed eyes.
• Loss of appetite.
• Diarrhoea.
• Vent pasting.
• Swollen abdomen.
Omphalitis, Yolk Sac Infection

Post-mortem lesions

• Enlarged yolk sac with congestion.
• Abnormal yolk sac contents (colour, consistency) that vary according to the bacteria involved.

Diagnosis

• A presumptive diagnosis is based on the age and typical lesions. Confirmation is by isolation and identification of the bacteria involved in the internal lesions. Differentiate from incubation problems resulting in weak chicks.
Omphalitis, Yolk Sac Infection

Treatment
• Antibiotics in accordance with sensitivity may be beneficial in the acute stages, however the prognosis for chicks showing obvious signs is poor; most will die before 7 days of age.

Prevention
• Prevention is based on a good programme of hygiene and sanitation from the nest through to the chick box (e.g. clean nests, frequent collection, sanitation of eggs, exclusion of severely soiled eggs, separate incubation of floor eggs etc. There should be routine sanitation monitoring of the hatchery.
• Multivitamins in the first few days may generally boost ability to fight off mild infections.
Ascites

Associated with inadequate supplies of oxygen, poor ventilation and physiology (oxygen demand, may be related to type of stock and strain).

Ascites is a disease of broiler chickens occurring worldwide but especially at high altitude.

The disease has a complex aetiology and is predisposed by reduced ventilation, high altitude, and respiratory disease.

Morbidity is usually 1-5%, mortality 1-2% but can be 30% at high altitude.

Pulmonary arterial vasoconstriction appears to be the main mechanism of the condition.
Ascites

Signs

• Sudden deaths in rapidly developing birds.
• Poor development.
• Progressive weakness and abdominal distension.
• Recumbency.
• Dyspnoea.
• Possibly cyanosis.
Ascites

Post-mortem lesions

• Thickening of right-side myocardium.
• Dilation of the ventricle.
• Thickening of atrioventricular valve.
• General venous congestion.
• Severe muscle congestion.
• Lungs and intestines congested.
• Liver enlargement.
• Spleen small.
• Ascites.
• Pericardial effusion.
• Microscopic - cartilage nodules increased in lung.
Ascites

Prevention

Good ventilation (including in incubation and chick transport), avoid any genetic tendency, control respiratory disease.
Spiking Mortality of Chickens

This is a condition characterised by a sudden increase in mortality in young, typically 7-14-day-old, rapidly growing broiler chickens. Birds in good condition die after showing neurological signs. Mortality drops off as sharply as it started. This appears to be a multifactorial condition. Feed intake, and/or carbohydrate absorption are disturbed resulting in a hypoglycaemia. Males are more susceptible than females, probably because they are growing faster. Filtered intestinal contents from affected flocks appear to be capable of reproducing the condition, suggesting a viral component. In order to reproduce the typical condition the affected birds are subject to 4 hours without feed and then a mild physical stress such as spraying with cool water.
Spiking Mortality of Chickens

Signs

• Tremor.
• Paralysis.
• Coma.
• Death.
• Orange mucoid droppings.
Spiking Mortality of Chickens

Post-mortem lesions

• Mild enteritis.
• Excess fluid in lower small intestine and caeca.
• Dehydration.
Sudden Death Syndrome, 'Flipover'

A condition of broiler chickens of unknown cause, possibly metabolic. It can be induced by lactic acidosis and about 70% of birds affected are males.
Sudden Death Syndrome, 'Flipover'

**Signs**

- Sudden death in convulsion, most are found lying on their back.
Sudden Death Syndrome, 'Flipover'

Post-mortem lesions

- Intestine filled with feed.
- Haemorrhages in muscles and kidneys.
- The atria of the heart have blood, the ventricles are empty.
- Serum accumulation in lung (may be little if examined shortly after death).
- Livers heavier than those of pen-mates (as a percentage of bodyweight.).
Sudden Death Syndrome, 'Flipover'

**Diagnosis**
- Birds found on back with lack of other pathology.

**Treatment**
- None possible.
Sudden Death Syndrome, 'Flipover'

Prevention

Lowering carbohydrate intake (change to mash), feed restriction, lighting programmes, low intensity light, use of dawn to dusk simulation and avoidance of disturbance.
## Locomotory disease

### Viral
- Viral Arthritis (*Reovirus*)
- Malabsorption Syndrome
- Avian Encephalomyelitis, Epidemic Tremors

### Bacterial
- Infectious Synovitis
- Pullorum Disease
- Colibacillosis
- Staphylococcosis
- Streptococcosis
- Botulism
Immunosuppressive diseases
Infectious Bursal Disease
Gumboro

A viral disease, seen worldwide, which targets the bursal component of the immune system of chickens.

In addition to the direct economic effects of the clinical disease, the damage caused to the immune system interacts with other pathogens to cause significant effects.

The age up to which infection can cause serious immunosuppression varies between 14 and 28 days according to the antigen in question.

Generally speaking the earlier the damage occurs the more severe the effects.
Infectious Bursal Disease

The infective agent is a *Birnavirus* (*Birnaviridae*), serotype 1 only, first identified in the USA in 1962.

Morbidity is high with a mortality usually 0-20% but sometimes up to 60%.

Signs are most pronounced in birds of 4-6 weeks and White Leghorns are more susceptible than broilers and brown-egg layers.
Infectious Bursal Disease

The route of infection is usually oral, but may be via the conjunctiva or respiratory tract, with an incubation period of 2-3 days. The disease is highly contagious. Mealworms and litter mites may harbour the virus for 8 weeks, and affected birds excrete large amounts of virus for about 2 weeks post infection. There is no vertical transmission.

The virus is very resistant, persisting for months in houses, faeces etc. Subclinical infection in young chicks results in: deficient immunological response to Newcastle disease, Marek's disease and Infectious Bronchitis; susceptibility to Inclusion Body Hepatitis and gangrenous dermatitis and increased susceptibility to CRD.
Infectious Bursal Disease

Signs

• Depression.
• Inappetance.
• Unsteady gait.
• Huddling under equipment.
• Vent pecking.
• Diarrhoea with urates in mucus.
Infectious Bursal Disease

Post-mortem lesions

• Oedematous bursa (may be slightly enlarged, normal size or reduced in size depending on the stage), may have haemorrhages, rapidly proceeds to atrophy.

• Haemorrhages in skeletal muscle (especially on thighs).

• Dehydration.

• Swollen kidneys with urates.
Infectious Bursal Disease

Differentiate clinical disease from:

Infectious bronchitis (renal)
Cryptosporidiosis of the bursa (rare)
Coccidiosis
Haemorrhagic syndrome
Infectious Bursal Disease

Treatment

No specific treatment is available.

Use of a multivitamin supplement and facilitating access to water may help.

Antibiotic medication may be indicated if secondary bacterial infection occurs.
Infectious Bursal Disease

Prevention

Vaccination, including passive protection via breeders, vaccination of progeny depending on virulence and age of challenge. In most countries breeders are immunised with a live vaccine at 6-8 weeks of age and then re-vaccinated with an oil-based inactivated vaccine at 18 weeks. A strong immunity follows field challenge. Immunity after a live vaccine can be poor if maternal antibody was still high at the time of vaccination.

When outbreaks do occur, biosecurity measures may be helpful in limiting the spread between sites, and tracing of contacts may indicate sites on which a more robust vaccination programme is indicated.
Chicken Anaemia

A viral disease of chickens caused by Chicken Anaemia Virus or CAV. Prior to confirmation that it is in fact a virus it was known as Chicken Anaemia Agent or CAA.

Mortality is typically 5-10% but may be up to 60% if there are predisposing factors present such as intercurrent disease (Aspergillosis, Gumboro, Inclusion body hepatitis etc.) or poor management (e.g. poor litter quality).

Transmission is usually *vertical* during sero-conversion of a flock in lay, lateral transmission may result in poor productivity in broilers.
Chicken Anaemia

**Signs**

- Poor growth.
- Pale birds.
- Sudden rise in mortality (usually at 13-16 days of age).
- No clinical signs or effect on egg production or fertility in parent flock during sero-conversion.
Chicken Anaemia

Post-mortem lesions

- Pale bone marrow.
- PCV of 5-15% (normal 27-36%).
- Atrophy of thymus and bursa.
- Discoloured liver and kidney.
- Gangrenous dermatitis on feet, legs wings or neck.
- Acute mycotic pneumonia.
Chicken Anaemia

**Diagnosis**

Gross lesions, demonstration of ongoing sero-conversion in parent flock, virus may be isolated in lymphoblastoid cell line (MDCC-MSB1).

**Treatment**

Good hygiene and management, and control of other diseases as appropriate, may be beneficial. If gangrenous dermatitis is a problem then periodic medication may be required.
Chicken Anaemia

Prevention

Live vaccines are available for parents, their degree of attenuation is variable. They should be used at least 6 weeks prior to collecting eggs for incubation. Their use may be restricted to those flocks that have not seroconverted by, say, 15 weeks.

Immunity: there is a good response to field challenge (in birds over 4 weeks of age) and to attenuated live vaccines.

Serology: antibodies develop 3-6 weeks after infection, and may be detected by SN, Elisa, or IFA.
Inclusion Body Hepatitis

A disease of chickens characterised by acute mortality, often with severe anaemia, caused by an adenovirus. A number of different sero-types have been isolated from disease outbreaks but they may also be isolated from healthy chickens.

It has a course of 9-15 days with a morbidity of 1-10% and a mortality of 1-10%. Infected birds remain carriers for a few weeks.
Inclusion Body Hepatitis

Agens

Genus *Aviadenovirus*

*Fowl adenovirus serotype 1-12*

Transmission may be vertical or lateral and may involve fomites. Immunosuppression, for instance due to early IBD challenge or congenital CAV infection, may be important.
Inclusion Body Hepatitis

Since adenoviruses are commonly found in healthy poultry, isolation alone does not confirm that they are the cause of a particular problem.

Progeny of high health status breeding flocks appear to be at greater risk, perhaps because they have lower levels of maternal antibody.
Inclusion Body Hepatitis

Signs

• Depression.
• Inappetance.
• Ruffled feathers.
• Pallor of comb and wattles.
Inclusion Body Hepatitis

Post-mortem lesions

- Liver swollen, yellow, mottled with petechiae and ecchymoses.
- Kidneys and bone marrow pale.
- Blood thin.
- Bursa and spleen small.
- Microscopically - basophilic intranuclear inclusions.
Pathohistological finding
Inclusion Body Hepatitis

**Diagnosis**

A presumptive diagnosis may be made on history and lesions. Confirmation is made on finding inclusions in the liver. The virus grows well in tissue culture (CEK, CEL).

Serology: DID for group antigen, SN for individual serotypes.

Differentiate from
Chick anaemia syndrome, sulphonamide intoxication, Infectious Bursal Disease, vibrionic hepatitis, fatty liver syndrome, and deficiency of vitamin B12.
Serology investigation

- Immunodiffusion test
- ELISA
Inclusion Body Hepatitis

Treatment

None. Soluble multivitamins may help with the recovery process.

Prevention

Quarantine and good sanitary precautions, prevention of immunosuppression.
Viral Arthritis is the classic, but by no means the only, manifestation of reovirus infection of chickens; at least 5 sero-types of virus occur. Morbidity is high but mortality is usually low. Transmission is by faecal contamination, and good both laterally and vertically. Birds remain carriers for over 250 days. The virus is resistant to heat, ether, chloroform, pH and environmental factors. Reoviruses vary markedly in pathogenicity and the tissue damaged. Some can cause other disease syndromes such as early chick mortality and malabsorption syndrome. Some strains have shown severe systemic disease including pericarditis in chickens.
Viral Arthritis/Tendosynovitis

**Signs**

- Lameness.
- Low mobility.
- Poor growth.
- Inflammation at hock.
- Swelling of tendon sheaths.
- Unthriftiness.
- Rupture of gastrocnemius tendons.
Viral Arthritis/Tendosynovitis

Post-mortem lesions

• Swelling and inflammation of digital flexor and metatarsal extensor tendon sheaths.
• Foot pad swelling.
• Articular cartilages may be ulcerated.
• Haemorrhage in tissues.
• Fibrosis in chronic cases.
Viral Arthritis/Tendosynovitis

Diagnosis

Diagnosis may be based on the history, lesions, IFA and rising antibody titre. Isolation may be readily achieved in CE yolk sac and CAM and also cell cultures (CE kidney or liver cells).

Serology may be by DID, FAT or Elisa. 'Silent' infections (not associated with obvious disease) are common.

Differentiate from mycoplasmosis, salmonellosis, Marek's, Pasteurella, erysipelas.
Viral Arthritis/Tendosynovitis

Treatment
• None.

Prevention
• Vaccination is ideally carried out by administering a live vaccine in rear followed by an inactivated vaccine prior to coming into lay.
• Most vaccines are based on strain 11/33.
• Rear birds in all-in/all-out production systems.
Locomotory disease

Osteopathy
- crooket toes
- spondylolisthesis (kinky back)
- valgus and varus deformation of the intertarsal joint
- dyschondroplasia

Metabolic disease
- rickets
- encephalomalacia
- exudative diathesis
- muscular dystrophy
- vitamin B1 and B2 deficiency
- perosis
- gout, uremic poisoning
Crooked toes

• Crooked toes is a condition best described in poultry as a developmental anomaly in which the toes are bent laterally and medially (outward and inward, respectively) in a horizontal plane. This is different than curled toes in pigeons and poultry which is due to a riboflavin deficiency in the diet.

• Pathogenesis: Crooked toes occurs due to a twisting of the phalanges (toe bones).
  Etiology: The cause is unknown, however wire floors and low humidity may predispose birds to the anomaly.
Crooked toes
Valgus and varus deformation of the intertarsal joint
Rickets (hypocalcaemic)

- Vitamin D deficiency or phosphorus/calcium imbalance is seen in chickens, turkeys and ducks worldwide.

Signs
- Lameness.
- Hock swelling.
- Soft bones and beak.
- Birds go off legs.
- Poor growth.
- Birds rest squatting.
- Reduction in bodyweight.
Rickets (hypocalcaemic)

Post-mortem lesions

• Bones soft and rubbery.
• Epiphyses of long bones enlarged.
• Beading and fracture of ribs.
• Growth plates widened and disorganised.
• Beak soft.
• Parathyroids enlarged.
Rickets (hypocalcaemic)

**Diagnosis**
- History, signs, lesions.
- Differentiate from Encephalomalacia, Femoral Head Necrosis.

**Treatment**
- Over-correct ration with three times vitamin D for 2 weeks, or Vitamin D or 25-hydroxy vitamin D in drinking water.

**Prevention**
- Supplementation of vitamin D, proper calcium and phosphorus levels and ratio, antioxidants.
Rotated tibia
Spondylolisthesis, Kinky-back

A complex condition of broiler chickens with a morbidity of 1-5% and low mortality associated with rapid growth and possibly having a genetic component.
Spondylolisthesis, Kinky-back

**Signs**

- Sitting on hock or rumps, legs extended forward.
- Use of wings to help in walking.
- May walk backwards.
Spondylolisthesis, Kinky-back

Post-mortem lesions

Anterior-posterior rotation of the bodies of the last or penultimate thoracic vertebrae resulting in scoliosis just anterior to the kidney.
Spondylolisthesis, Kinky-back

Diagnosis
• Symptoms, lesions.

Treatment
• None.

Prevention
• Selection for satisfactory conformation in primary breeding.
Tibial Dyschondroplasia

A complex condition seen in chickens, turkeys and ducks. It may be associated with rapid growth and have a nutritional factor.
Tibial Dyschondroplasia

**Signs**

- There are usually no signs unless the condition is severe.
- Swelling and bowing in the region of the knee joints.
- Lameness.
Tibial Dyschondroplasia

Post-mortem lesions

- Plug of cartilage in proximal end of tibia, distal tibia, and proximal metatarsus, in decreasing order of frequency.
- Microscopically - a mass of avascular cartilage with transitional chondrocytes, small ovoid lacunae and more matrix than normal.
A1 – growing plate
A2 – hypertrophic zone of cartilage
A3 – epifysis
Tibial Dyschondroplasia

**Diagnosis**
- Gross pathology; mild lesions may require histology to distinguish from other problems. Differentiate from rickets.

**Treatment**
- None.

**Prevention**
- Genetic selection, modifications of calcium and phosphorus ratios, Vitamin D3 supplementation, chloride levels and acid/base balance.
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List of viral infection of broiler chickens

Disease
• Inclusion body hepatitis
• Avian adenovirus splenomegaly of chickens
• Hydropericardium-hepatitis syndrome
• Marek’s disease
• Infectious anaemia of chickens
• Retikuloendoteliosis
• Transmissible viral proventriculitis

Agen
• genus Aviadenovirus, FAV 1-12
• genus Siadenovirus
• genus Aviadenovirus, FAV 4 + 8
• Herpesvirus
• Gyrovirus (Circoviridae)
• Retrovirus
• genus Aviadenovirus
Bacterial and mycotic infections

- Staphylococcosis
- Streptococcosis
- Necrotic Enteritis
- Gangrenous dermatitis
- Ulcerative enteritis
- Colibacillossis
- Pullorum disease
- Paratyphoid infection
- Campylobacteriosis

- Chronic Respiratory Disease (CRD)
- Infectious synovitis

Mycotic infection
- Aspergillosis
- Candidiasis
- Dactylariosis

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