International Poultry Production

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Avian influenza VI

Pathogenesis

Infection starts with the inhalation or ingestion of avian influenza viruses and then multiple replications occur in the intestinal or respiratory tracts with the release of many viruses. In chickens the nasal cavities are an important site of initial viral multiplication.

After this phase the viruses invade the tissues and enter the blood capillaries where further viral replication occurs and the viruses are taken to the various organs of the body which the viruses then attack. Clinical signs and death are as a consequence of multiple organ failure.

For LPAI viruses viral multiplication is often limited to the intestinal or respiratory tracts and illness/death is usually associated with respiratory tract/lung damage. Secondary bacterial infections will aggravate this scenario.

Immunity

Infection or vaccination initiates both humoral and cellular antibody responses. Typically an IgM response occurs five days after infection and this is followed by an IgG response.

The antibody response is strongest in chickens and weakest in ducks. Both neutralising and protective antibodies are produced.

Protection has been shown to be as long as 7-8 months in layers.

Diagnosis

Diagnosis of avian influenza depends upon:

- Detection of avian influenza virus, viral genes or viral proteins in the tissue(s) of the bird.
- Isolation and identification of the virus.
- Detection of antibodies to the avian influenza virus.
- Clinical signs and lesions.

Differential diagnosis

For HPAI, other causes of high mortality should be considered, such as Newcastle disease, avian pneumovirus infection, acute pasteurellosis, acute Gumboro disease, heat stress and some poisons/toxins.

For LPAI, other causes of respiratory disease and egg drop must be considered including Newcastle disease, avian pneumovirus, ILT, infectious bronchitis, mycoplasma and various bacterial entities. Concurrent infections are often seen.