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Incidence

Sporadic cases of avian leukosis virus tumours occur in most flocks and on occasions it can cause heavy losses. In the field erythroblastosis occurs less frequently than lymphoid leukosis.

More recently, high losses have been attributed to subgroup J avian leukosis.

Avian leukosis viruses are the most common isolated in the field although, again more recently, the incidence of subgroup J avian leukosis virus in infected broiler breeder flocks has been reported as high as almost 90%.

Leukosis viruses and antibodies against them have been reported from many parts of the world.

Transmission

Exogenous avian leukosis viruses are transmitted both vertically through the egg and horizontally from bird to bird. Only a small proportion of chicks are infected vertically but this transmission is important in maintaining infection. Horizontal transmission may have an important role to play in maintaining the rate of vertical transmission.

The virus has a relatively short life outside the bird and so rapid horizontal spread is rarely seen. With subgroup J avian leukosis virus contact exposure at hatch was an effective means of spread in broiler breeder chicks.

Four classes of avian leukosis virus infection are known.

- No viraemia, no antibody (V-A-)
- No viraemia with antibody (V-A+)
- Viraemia with antibody (V+A+)
- Viraemia, no antibody (V+A-)

Birds in an infection free flock, and genetically resistant birds in a susceptible flock, are V-A-.

Genetically susceptible birds in an infected flock fall into one of the three remaining types. Many are V-A+ and a small number (<10%) are V+A- and most of these transmit avian leukosis virus to their offspring. Congenitally infected embryos develop immunotolerance to the virus and after hatching become V+A-. By 22 weeks of age up to a quarter of broiler breeders exposed to subgroup J avian leukosis virus at hatch were found to be V+A-.

Infection of a cockerel does not appear to impact on the rate of congenital infection in the offspring. Avian leukosis virus is shed into egg albumen and then on to the developing embryo from albumen producing glands in the oviduct. Avian leukosis infection rates are closely related to avian leukosis virus production in the oviduct.

In flocks infected with subgroup A avian leukosis, only a small minority of birds develop avian leukosis – the remainder become carriers and shedders. If infection occurs after the first few weeks of life the incidence of leukosis declines significantly.

Endogenous avian leukosis viruses are transmitted vertically.