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Diagnosis and Treatment of Duck Plague

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Perspective

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DESCRIPTION

Anatidae aquatic species with the common name duck are all members of this family. Swans and geese which are heirs of ducks are often larger and have longer necks than ducks. They are a form taxon that is divided into multiple subfamilies. However, they constitute a monophyletic group (the group of all descendants of a single common ancestral species) as swans and geese are not recognised as ducks. The majority of ducks are aquatic and they can be found in both fresh and salt water. The biological order Anseriformes which includes ducks, geese and swans as well as screamers and the magpie goose includes all duck species. The biological family *Anatidae* includes all save the screamers. Ducks are divided into a number of subfamilies and "tribes" within their own family. Taxonomists have a great deal of dispute over the number and together of these subfamilies and tribes. Some people base their decisions on morphological traits while others rely on social norms or genetic research. There are between two and five subfamilies that are thought to contain ducks. The high degree of hybridization that occurs in wild ducks makes it difficult to determine the links between different species.

Anatid alphaherpesvirus 1 (AnHV-1) of the *Herpesviridae* family causes the acute disease with significant fatality rates in flocks of ducks, geese and swans which is known as duck plague (also called as duck viral enteritis). Through polluted water and close communication, it spreads both vertically and horizontally. Due to the fact that they are frequently asymptomatic disease carriers, migratory waterfowl play a significant role in the spread of this illness. A three to seven day incubation time is required. One week old birds are susceptible to infection [1].

Clinical signs and diagnosis

The incubation time for symptoms after DVE (Duck Viral Enteritis) infection is 3 days-7 days for domesticated fowl and up to 14 days for wildfowl. Often, the first sign of DVE is a sudden and steady increase in flock mortality [2].

Appetite loss, decreased egg production (nearing 20%–40% falls), nasal discharge, increased thirst, diarrhoea, ataxia, tremors, a drooped wing look and in males, a prolapsed penis are among the symptoms that might affect a bird individually. DVE mortality rates could be as high as 90% [3]. Symptoms normally start to manifest within 5 days of death. Clinical DVE symptoms will be vary with host immune system status, species, sex and viral strain pathogenicity [4].

With the possible exception of coots, Anatid alphaherpesvirus 1 can only infect birds belonging to the family *Anatidae* in the group Anseriformes. Similarities to DVE lesions were discovered in a study of lesions seen in coots. The fact that DVE can cross to different orders and families or adapt to new hosts may be demonstrated by this. Different species of waterfowl are more or less susceptible to DVE with wild fowl typically being more resilient. It has not been demonstrated that non-waterfowl can contract duck plague. Mallards are among the least susceptible species whereas blue-winged teal are among the most. In a different study, it required 300,000 more virus particles to infect northern pintails than blue-winged teal [5].

Treatment and control

In the United States, vaccination against duck viral enteritis is now common place. The only vaccines that work are attenuated ones. When DVE is present, an outbreak must be controlled through depopulation, relocation and thorough disinfection. Birds who have recovered naturally have a strong immunity. Although there is no cure for DVE, resveratrol has demonstrated to have some antiviral action [6].

Pathogenesis

DVE is regarded as pantropic since it has the capacity to multiply and spread throughout the host's various organs. The liver, spleen, thymus and Fabricius bursa all have lesions and haemorrhaging as a result of increased vascular permeability brought on by viral replication. The two main entry points for the virus, the bird's oesophagus and cloaca both include mucus surfaces where DVH-1 can multiply. The way an infection is spread affects which tissues are impacted first and how long it takes for symptoms to manifest. Typically, viral replication starts in the gastrointestinal tract and progresses to the thymus, spleen and liver.

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