



# Rabbit Diseases

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## Rabbit Haemorrhagic Disease

Rabbit Haemorrhagic Disease - RHD (also known as Viral Haemorrhagic Disease – VHD and Rabbit Calicivirus Disease) is a highly contagious disease of the European rabbit (*Oryctolagus*) caused by a calicivirus (RHDV). The disease has a high morbidity and mortality rate in susceptible animals. After its emergence in China in the 1980s it has spread naturally, and rapidly throughout most of the rest of the world. As a consequence of its worldwide spread, RHDV differentiated into many genetically closely-related strains, all highly virulent. In addition, other non-virulent caliciviruses have been identified, both in commercial and wild rabbits, that are less closely genetically related and provide varying degrees of cross-protection against RHD. More recently, a new variant strain of virulent RHDV has emerged in Europe which seems capable of evading the high degree of protection provided by 'classical' RHDV vaccines. This new variant is designated as RHDV2.

### Etiology and Pathogenesis

The etiological agent is a small RNA calicivirus which is very resistant to inactivation, particularly when protected by organic material, and may persist in decomposing carcasses in the environment, for some months. It is sensitive to phenol or formalin based disinfectants.

RHD affects both wild and domesticated members of the species *Oryctolagus cuniculus*, the European rabbit, although other rabbit species are not susceptible. Rabbits of all ages can be infected but animals younger than 40–50 days of age are refractory to disease. The morbidity rate varies from 30% to 100%, and the mortality rate is 40–100%.

Rabbits can become infected via oral, nasal or conjunctival routes following either direct contact with infected animals, exposure to an infected carcass, by means of fomites, including contaminated food, bedding and water and via mechanical transmission from flies and other insects. Rabbits which recover from disease may remain infectious for some weeks and continue to shed infection in their faeces.

Following infection the virus spreads and multiplies in splenic histiocytes and hepatocytes, in which it induces apoptosis and cell death. Substances released by the damaged cells are thought to initiate disseminated intravascular coagulation (DIC). It is this DIC and also the fulminant liver failure that causes rapid death in rabbits succumbing to acute disease.

## Clinical and Pathological Findings

The course of the disease can be peracute, acute, subacute or chronic. Peracute RHD is characterised by sudden death, sometimes with signs of terminal haemorrhagic nasal discharge, and so clinical signs are normally only observed in the acute or subacute forms. The incubation period varies between 1 and 3 days and is followed by pyrexia ( $>40^{\circ}\text{C}$ ), anorexia, apathy, dullness, collapse, nervous signs (convulsion, ataxia, paralysis, opisthotonos, paddling) vocalisation, respiratory signs (dyspnoea, frothy and bloody nasal discharge), and cyanosis of mucous membranes. In these animals death often occurs within 12–36 hours of the onset of signs. During an outbreak 5–10% of rabbits may show signs of more chronic disease, characterised by generalised jaundice, lethargy and loss of weight. These animals usually die of liver failure within 1 or 2 weeks.

Due to the rapid course of this disease, most affected animals are normally found in good condition at necropsy. There is evidence of splenomegaly and primary liver necrosis, and a massive and widespread disseminated intravascular coagulopathy which results in the presence of clotted blood in vessels and petechial haemorrhages in all organs and tissues. The most severe lesions are in the liver, trachea and lungs. In subacute and chronic disease widespread icteric discoloration of the carcass is evident.

## Diagnosis

A presumptive diagnosis can readily be made when there are multiple cases of sudden death in amongst unvaccinated animals, in some cases following a short period of lethargy and fever, and with characteristic hepatic necrosis and haemorrhages visible at necropsy.

The presumptive diagnosis can be confirmed by isolation and identification of virus or detection of viral antigen in samples of fresh liver, spleen or blood. A PCR test is available commercially which can detect viral RNA in a many organs, urine, faeces or serum.

Serum antibodies arising from natural infection or from immunisation appear within 4-6 days of infection in surviving animals. These can be measured using HI or ELISA tests.