Rabbit haemorrhagic disease (RHD), commonly known as calicivirus, was introduced into Australia in 1995 as a biological control for the introduced European rabbit, *Oryctolagus cuniculus*. The European rabbit has a devastating impact on Australian ecosystems and with an estimated economic impact of $200 million p.a. (McLeod 2004), rabbits cause significant damage to agricultural and pastoral industries.

**Aetiology**

Rabbit hemorrhagic disease virus (RHDV) is a member of the genus *Lagovirus*, family *Caliciviridae*. Only a single serotype is known, but two major subtypes exist: RHDV and the antigenic variant RHDVa. RHDVa does not currently occur in Australia. Nonpathogenic strains of RHDV also occur with an endemic Australian strain, RCV-A1, identified (Strive et al. 2008).

**Natural hosts**

RHDV is highly specific to rabbits of the genus *Oryctolagus*. Hares (*Lepus sp.*) and rabbits of other genera (*Sylvilagus sp*) do not appear to be susceptible (The Merck Veterinary Manual 2011). Other mammals and some rabbit predators can develop antibodies and excrete virus, but virus replication has not been observed.

Kittens (< 50 days) are much less susceptible to RHDV. The exact reason for this is unknown. Protection offered via maternal antibodies from seroconverted mothers can last up to 70 days, with rabbits older than this being fully susceptible.

**World distribution**

RHDV was first reported in China in 1984 and is now found in over 40 countries across 5 continents. It is endemic in wild populations in Europe, Australia, New Zealand, Cuba and parts of Asia. Limited outbreaks of RHDV have occurred in North America, however the disease has been eradicated each time and RHDV is not currently endemic.
**Occurrences in Australia**

RHDV occurs throughout the rabbit’s distribution in Australia, which encompasses every State and Territory.

**Epidemiology**

Morbidity and mortality estimates for RHD have a broad range as rabbits often die in their burrows, making accurate assessments difficult. In farmed rabbits the estimated morbidity rate ranges from 30–100% with mortality rates ranging from 40–100%, however the typical mortality rate is around 90% (The Merck Veterinary Manual 2011). Morbidity rates in wild populations range from exceedingly low to high in the 90% range, however the mortality rate is around 70–80%.

RHDV incubates for a period of 1-3 days and death may occur 1-2 days post-infection. Transmission is via direct contact with infected animals or via fomites. Species of blowfly (Calliphoridae) and bushfly (Muscidae) are known vectors of RHDV in Australia (Asgari et al. 1998, McColl et al. 2002), with fresh fly-spots containing 2–3 times the median lethal dose (LD_{50}). Rabbit fleas (*Sphilopsyllus cuniculi* & *Xenopsylla cunicularis*) and mosquitos (*Culex annulirostris*) may also be important (Lenghaus, 1994). RHDV is acquired through oral, nasal or conjunctival pathways, and seroconverted carriers may be infectious for up to a month depending on the climatic conditions. All excretions are thought to contain virus.

**Clinical signs**

Rabbits that have died from RHD often show no outward cause of death. A freshly dead, but otherwise healthy-looking rabbit indicates that it may have died from RHD. Occasionally there may be a small discharge of blood from the nose and/or anus; the head may be thrust back and front legs pointing forwards. In peracute infections, infected rabbits develop a fever and die within 12–36 hours of its onset. Symptomatic rabbits may display fever, squeals and lethargy. Convulsions may also be seen. In acute disease dullness, anorexia, congestion of palpebral conjunctiva and/or prostration may be observed. Neurological signs may also develop including excitement, incoordination, paddling and some rabbits may display symptoms of convulsions or mania (turning and flipping quickly in their cages). In some animals that recover from acute disease, jaundice, weight loss and lethargy develop before death occurs in a few weeks. Subacute infections show similar symptoms however the majority of rabbits may survive. Chronic infections appear to be asymptomatic.

**Diagnosis**

RHD should be suspected when a number of animals die suddenly after a brief period of lethargy and fever. Diagnosis is usually made by necropsy and can be further confirmed by ELISAs, reverse-transcriptase polymerase chain reaction (RT-PCR), western blotting or negative-staining immunoelectron microscopy.

**Pathology**

The liver of RHD-dead rabbits may appear marbled with a fine reticular pattern of necrosis. It will be friable and pale pinky-red rather than dark red-brown. The spleen will be black in colour and swollen with rounded edges. Blood clots may be found in the heart, lungs and/or kidneys. The lungs are often a bright orangey-white colour. Blood spots may be seen along the large intestine.
Differential diagnoses

Acute pasteurellosis, an atypical form of myxomatosis, poising, heat exhaustion and some causes of severe septicaemia (E. coli or Clostridium perfringens) can show similar symptoms to RHDV infection. These can often be ruled out at necropsy.

Laboratory diagnostic specimens

Samples of liver, spleen, blood sera, kidney or bone marrow can be used to confirm RHDV. The liver contains the highest viral titres in acute or peracute disease. In chronic or subacute cases, virus may be easier to find in the spleen. Samples should be stored in a freezer (-20°C) (OIE 2009).

Treatment

There is no known treatment for RHD.

Prevention and control

RHDV is extremely contagious. The virus resists degradation by chloroform and ether but can be inactivated with 10% sodium hydroxide or 1-2% formalin. 10% household bleach may also work. Carcasses should be removed immediately however restocking should not occur immediately as virus may persist in the environment depending on climatic conditions (OIE 2009).

Domestic rabbits can be protected via vaccination, and by implementing Biosecurity measures such as sanitation, disinfection and the maintenance of closed colonies. Vaccination should be repeated every 12 months (OIE 2009).

Statistics

After the escape of the virus in 1995, RHD caused a decline in rabbits of between 0–80%, with variability occurring both within sites and between sites (Mutze et al. 2010). RHDV had its greatest impact in arid and semi-arid inland Australia (Henzell et al. 2002). However, its performance in the southern agricultural areas and more temperate agricultural areas of Australia has been highly variable (Neave 1999, Mutze et al. 2010), with RHDV least effective in coastal areas, in cool moist areas, and during summer in areas of summer rainfall (Henzell et al. 2002).

Research

The effects of the original strain of RHDV (RHDV CZ 351) that was released in Australia are beginning to wane. The presence of the benign calicivirus RCV-A1 offers partial protection to infection with the pathogenic strain (Strive et al. 2010), and this, coupled with apparent rising genetic resistance, has led to the search for more virulent strains from overseas.

Human health implications

There is no indication that exposure to RHDV is associated with infection or disease in humans (Carmen et al. 1998, Greenslande et al. 2001).
Conclusions

RHD is a species specific, highly lethal disease of European rabbits, and as such, is an important biological control for the European rabbit in Australia. Indications of a developing genetic resistance, and the partial protection offered by the benign RCV-A1, has led to the search for more virulent overseas strains as biological control agents.

References and other information


Neave H (1999) Rabbit Calicivirus Disease Program Report 1: Overview of Effects on Australian Wild Rabbit Populations and Implications for Agriculture and Biodiversity. A report of research conducted by participants of the Rabbit Calicivirus Disease Monitoring and Surveillance Program and Epidemiology Research Program Bureau of Rural Sciences, Canberra.


The Merck Veterinary Manual (2011) Rabbit Calicivirus Disease

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To provide feedback on this fact sheet

We encourage those with laboratory confirmed cases of this condition in native Australian or feral animals to submit this information to the national system for consideration for inclusion in the national database. Please contact us at admin@wildlifehealthaustralia.com.au.

Wildlife Health Australia would be very grateful for any feedback on this fact sheet. Please provide detailed comments or suggestions to admin@wildlifehealthaustralia.com.au. We would also like to hear from you if you have a particular area of expertise and would like to produce a fact sheet (or sheets) for the network (or update current sheets). A small amount of funding is available to facilitate this.

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