Introductory statement

Rift Valley fever (RVF) is a zoonotic arboviral disease that causes high abortion rates and fatalities in livestock. Rift Valley Fever has never been reported in Australia (OIE 2015). However, Australia has several potentially competent vector, reservoir, and free-ranging and captive host species, plus the environmental conditions currently deemed as risk factors for RVF outbreaks. It is uncertain what role Australian native fauna would play should an outbreak occur in Australia, but susceptible wild animals could pose a considerable threat to RVF control, because of their potential to harbour and spread the virus (Animal Health Australia 2013).

Aetiology

**Virus:** Rift Valley fever virus (RVFV); **genus:** Phlebovirus; **family:** Bunyaviridae.

Other notable viruses in this family include La Crosse encephalitis, California encephalitis, Crimean-Congo haemorrhagic fever and Nairobi sheep disease viruses (Geering et al. 1995; Peters 1998).

**Grouping (non-taxonomic):** Arbovirus - see separate WHA factsheet on Arboviruses for more details.

Natural hosts

RVF has a very wide host range, however the full host range of the virus is unknown. The susceptibility of Australian fauna to RVF is unknown, as is the potential role of native fauna in the ecology and epidemiology of virus, if it were to enter Australia (Animal Health Australia 2013).

**Reservoir host:** Uncertain – livestock*, Aedes spp.* mosquitoes (eggs) (CDC 2013).

* present in Australia in free-ranging/ wild situations
**Amplifying host:** Livestock*, humans* (?) (Animal Health Australia 2013; CDC 2013).

**Clinically affected hosts:** Humans*, sheep*, goats*, cattle*, buffalo*, camels* (Geering et al. 1995; Animal Health Australia 2013; CDC 2013).


**World distribution**

The disease is present in sub Saharan Africa, Madagascar, Saudi Arabia and Yemen (CDC 2013). Epidemics of RVF have occurred at irregular intervals of 5-20 years in eastern and southern Africa where sheep and cattle are raised (Geering et al. 1995).

**Epidemiology**

The epidemiology of RVFV is not fully understood. It is transmitted by direct contact (including aerosols) as well as by biting vectors and may affect a broad host range. The virus is known to survive in mosquito eggs for extended periods. Risk factors are poorly understood but are known to include heavy rainfall, flooding (resulting in increased vectors) and a susceptible livestock population (Geering et al. 1995).

Incubation period ranges from 1-30 days (OIE 2009). Direct and vector transmission are both possible. Mosquitoes (include Aedes and other species) and other biting arthropods transmit RVFV (CDC 2013), either biologically or via contamination of biting mouthparts (Geering et al. 1995).

Direct transmission to humans may occur from blood, body fluids (including milk) and tissues of infected animals (e.g. during slaughter or veterinary procedures). The virus may survive up to 3 months in dried blood. Aerosol transmission may occur (Merck Veterinary Manual 2014).

Epizootics in animals probably occur when large numbers of eggs hatch and adult vectors are abundant, such as after periods of heavy rainfall or flooding (CDC 2013). This leads to a subsequent epidemic in humans from bites from infected mosquitoes or direct transmissions from infected animals (Animal Health Australia 2013). No human to human transmission has been recorded (WHO 2010).

There is potential for intercontinental spread via infected vectors or humans (Geering et al. 1995).

Free-ringing and captive hosts, vectors and environmental conditions (such as seasonal flooding and inaccessibility) for RVFV establishment exist in Australia, particularly in the northern regions (Turell and Kay 1998). However, no host or vector competency studies, or other wild animal risk assessments for RVFV have been identified. However, it is likely that that Australian wild animals (both native and feral) would be a significant consideration in control of RVF in the Australian context, because of their likely potential to harbour and spread the virus (Animal Health Australia 2013).

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Clinical signs

Clinical signs have not been described in Australian native wildlife. In other animals signs may be non-specific, which can contribute to delays in diagnosis (Merck Veterinary Manual 2014). Infected humans typically have either no symptoms or a mild illness with fever, weakness, back pain and extreme weight loss. In some patients, the disease may progress to haemorrhagic fever, encephalitis or ocular disease (CDC 2013).

Sheep are the most severely affected species with the young being the worst affected. Signs include fever, weakness, ataxia, vomiting and mucopurulent nasal discharge. Haemorrhagic diarrhoea and other petechial or ecchymotic haemorrhage of mucous membranes may also be seen. Goats and cattle show similar but less severe signs (Geering et al. 1995).

Diagnosis

Diagnosis in both animals and humans is made on the basis of a history of possible exposure, relevant clinical signs, exclusion of differential diagnoses and confirmatory laboratory tests (see below). Antibody responses may only be detectable 4-7 days post infection (WHO 2010).

Pathology

Characteristic lesions in livestock include congested and haemorrhagic liver, due to hepatic necrosis. Inflammation and haemorrhage is commonly present in most other organs. Ascites, hydropericardium, hydrothorax, pulmonary oedema and jaundice may be present (Geering et al. 1995).

Differential diagnoses

In livestock, enterotoxaemia, hepatotoxicosis, and other causes of abortions in sheep and cattle, as well as other exotic diseases including Wesselsbron disease, Nairobi sheep disease and bluetongue, (Geering et al. 1995; Animal Health Australia 2013).

Laboratory diagnostic specimens

Whole blood, liver and spleen are preferred for virus isolation. Blood (minimum 20 ml) should be collected from febrile suspect animals and also those in the convalescent phase (Geering et al. 1995).

Specimens should initially be sent to the state or territory diagnostic laboratory. After being cleared by the jurisdictional Chief Veterinary Officer (CVO) and the Victorian CVO, samples will then be forwarded to the Australian Animal Health Laboratories (Animal Health Australia 2013).

Laboratory procedures

Laboratory tests include IgM antibody-capture ELISA and reversed-passive haemagglutination for rapid antigen detection; and virus culture, mouse inoculation, virus and plaque reduction neutralisation, complement fixation, haemagglutination inhibition, reverse transcriptase PCR and immunofluorescence (Geering et al. 1995).
Treatment

There is no known effective treatment for RVF. Anti-viral drugs, immune modulators and convalescent-phase plasma may be possible treatments (Animal Health Australia 2013; CDC 2013).

Prevention and control

There is an AUSVETPLAN control strategy for Rift Valley fever. It acknowledges that, in Australia, ‘susceptible wild animals pose a considerable threat to RVF control’ because of their ‘potential to harbour and spread the virus’ and that the ‘full host range of RVF is unknown’ (Animal Health Australia 2013).

There is a human vaccine for RVF (MP-12) which has also shown promising results in laboratory trials in domestic animals. Existing veterinary vaccines can cause birth defects and abortions in sheep and are poorly effective in cattle (CDC 2013).

Statistics

Wildlife disease surveillance in Australia is coordinated by Wildlife Health Australia. The National Wildlife Health Information System (eWHIS) captures information from a variety of sources including Australian government agencies, zoo and wildlife parks, wildlife carers, universities and members of the public. Coordinators in each of Australia’s States and Territories report monthly on significant wildlife cases identified in their jurisdictions. There are no reports of RVFV in Australian wildlife in the National Database.

NOTE: access to information contained within the National Wildlife Health Information System dataset is by application. Please contact admin@wildlifehealthaustralia.com.au.

Research

No research activity for RVFV in Australia has been identified. Research questions pertaining to Australian wildlife may include:

- What are the risks of RVFV entry into Australia and spread to wildlife?
- What role would Australian wild animals play the ecology and epidemiology of the disease should it enter Australia?
- How quickly could a RVF incursion be detected?
- Would this detection and the subsequent response be sufficiently quick and effective to contain an outbreak?
- What are the human, livestock and wildlife health, and financial implications of an incursion or RVFV becoming endemic in Australia?

Human health implications

Conclusions

**RVFV is exotic to Australia.** However, potential hosts and competent vectors for RVFV (including the reservoir mosquito *Aedes* spp.) are present in Australia. An outbreak of this virus in Australian may be difficult or impossible to contain, especially if it enters the wildlife population. Much of the ecology of RVFV is also not fully understood making disease risk estimations challenging.

References and other information


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To provide feedback on this fact sheet

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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