

EXOTIC

Rabies in wildlife

Fact sheet

Introductory statement

Rabies virus is **not found** in Australia. Rabies is an invariably fatal viral disease that causes acute encephalitis capable of affecting all wildlife, domestic animals and humans (AHA 2011). Animals that are infected with rabies are called 'rabid'. Despite rabies not being found in Australia, it is an important disease for Australia because of the potential impacts on public health and trade should it be introduced and become established. As with other exotic diseases, Australia has very good processes for the identification and management of rabies should it enter the country. Rabies is moving east along the Indonesian island chain and ongoing vigilance is required.

Aetiology

Rabies is caused by infection with viruses of the genus *Lyssavirus*, family *Rhabdoviridae* (AHA 2011). Lyssaviruses are genetically and serologically related. The genus is classified phylogenetically into seven genotypes (Table 1). Classical rabies virus (genotype 1), which is **not** present in Australia, can be further classified into variants or biotypes that have adapted to a specific host species. A virus biotype is adapted to a single maintenance-host species, where infection and transmission by members of this species are highly efficient. Other species may also be infected by the virus biotype, but these hosts may be inefficient as vectors or may not be numerous enough to maintain a cycle (spill-over hosts). The rabies virus is genotype 1 of the *Lyssavirus* genus (AHA 2011). The RNA virus is single, negative-stranded and unsegmented (Rupprecht et al. 1995).

Example of classification:

Family: *Rhabdoviridae*, Genus: *Lyssavirus*

Genotype: for example, classical rabies = genotype 1

Table 1. Lyssavirus genotypes: common name, numerical genotype classification, geographic location, maintenance hosts and known spillover hosts (AHA 2011); * = Genera that have a worldwide distribution including Australia.

Name	Genotype designation	Locality	Maintenance hosts	Spillover hosts reported
Rabies virus	1	Worldwide (with exceptions) Bat biotypes are confined to the American continents; insectivorous bats mainly in North America, haematophagous bats in South and Central America, and the Caribbean.	American insectivorous bats: highest frequency in <i>Eptesicus</i> * <i>fuscus</i> , <i>Lasionycteris noctivagans</i> , <i>Lasiurus</i> spp., <i>Myotis</i> * spp., <i>Pipistrellus</i> * spp., <i>Tadarida</i> * <i>brasiliensis</i> Haematophagous (vampire) bats: <i>Desmodus</i> spp. Carnivores	Insectivorous bat strains: humans, foxes, skunks Vampire bat rabies: mainly cattle, horses, humans Carnivore rabies: several spillover hosts reported, including cats, dogs, humans, cattle, horses and wildlife
Lagos bat virus	2	Sub-Saharan Africa One case from France in a fruit bat imported from West Africa (1999)	Fruit bats: <i>Eidolon helvum</i> , <i>Micropteropus pusillus</i> , <i>Epomophorus wahlbergi</i> Single isolate from insectivorous bat: <i>Nycteris gambiensis</i>	Cats, dogs, water mongoose (<i>Atilax paludinosus</i>)
Mokola virus	3	Sub-Saharan Africa	Not known. Has been isolated from shrews (<i>Crocidura</i> spp.)	Rodents cats, dogs, humans (two), shrews. Mokola virus is able to infect and replicate in mosquito cells and so may be harboured by insects; Aitken et al. (1984).
Duvenhage virus	4	Southern and eastern Africa	Insectivorous bats: <i>Nycteris thebaica</i> , possibly <i>Miniopterus</i> * <i>schreibersi</i>	Humans
European bat lyssavirus 1	5	Europe (continental)	Insectivorous bats, particularly <i>Eptesicus serotinus</i>	Sheep, stone martens (<i>Martes foina</i>), cats, humans
European bat lyssavirus 2	6	Europe (continental, United Kingdom)	Insectivorous bats, particularly <i>Myotis</i> * <i>daubentonii</i> , <i>M. dasycneme</i>	Humans
Australian bat lyssavirus	7	Australia	Flying-foxes (<i>Pteropus</i> spp.) Insectivorous bat: <i>Saccolaimus flaviventris</i> . All Australian bat species are considered susceptible	Humans
	"New" and unclassified genotypes	Central Asia	Single isolates from insectivorous bats: <i>Myotis</i> * <i>blythi</i> (Aravan virus), <i>Myotis</i> * <i>mystacinus</i> (Khujand virus), <i>Murina leucogaster</i> (Irkut virus), <i>Miniopterus</i> * <i>schreibersi</i> (WCBV)	None recorded

Natural hosts

Any warm-blooded animal, including humans, may become infected with the rabies virus and develop clinical signs. Although birds have been known to develop antibodies, rabies is not considered a disease of birds (Gough and Jorgenson 1976). The maintenance hosts of rabies virus are usually members of the orders *Carnivora* (carnivores) and *Chiroptera* (bats). Maintenance hosts are generally more susceptible to rabies, shed a larger amount of virus and follow a more predictable disease course than non-maintenance species. In some areas of the world, wildlife species have become important for maintenance of the disease. This includes the red fox and raccoon dog in Europe; striped skunks, raccoons, red and grey foxes, and coyotes in North America; side-striped and black-backed jackals, various mongoose species (particularly the yellow mongooses) and bat-eared foxes in southern Africa; and the Arctic fox in the northern polar areas.

There are other animal species that are regarded as accidental hosts and have no epidemiological significance in maintaining rabies epidemics but are capable of transmitting disease. Accidental hosts include humans, other primates, horses, cattle, pigs and sheep.

In developing countries domestic dogs are recorded as the primary vector of rabies in urban cycles (Marks and Bloomfield 1999).

The susceptibility of Australian native animals is unknown. Species with a very low probability of natural virus infection include monotremes (echidna, platypus), marsupials (kangaroos, wombats, possums etc.) and cetaceans (whales and dolphins). The North American opossum (*Didelphis virginianus*) appears relatively resistant to experimental infection (McRuer and Jones 2009), also reflected by consistently low numbers of natural cases. One of the greatest sources of infection for the opossum is the raccoon, with overlap present in the urban environment. The virtual absence of substantial reports, despite the diversity of marsupials in South America, argues in part their resistance to natural infection (McRuer and Jones 2009).

World distribution

Rabies is recognised to be endemic in wildlife within North America and Europe (with the exception of United Kingdom and Ireland). In central and South America, Africa, the Indian subcontinent and Asia, rabies is primarily a disease of urban areas and the reservoir for the virus is primarily stray dogs with 95% of human deaths occurring in Africa and Asia (AHA 2011). Countries that are free of rabies include Japan, Singapore, New Zealand, Papua New Guinea and Australia. Rabies appears to be moving east along the Indonesian island chain. In recent years, there have been outbreaks of rabies on a number of the Indonesian islands including Bali, Flores, Pulau Larat and Kisar; previously, these islands had been considered rabies free. Detailed country information can be found on the OIE website (www.oie.int/en/animal-health-in-the-world/rabies-portal/).

Occurrence in Australia

Classical rabies virus is **not** present in Australia. Another *Lyssavirus*, Australian bat lyssavirus (ABLV) is found in Australian bat populations (see Wildlife Health Australia fact sheet “Australian bat lyssavirus”).

Rabies was the likely cause of a disease outbreak reported in Tasmania in 1867. It was confined to an area around Hobart and involved several dogs, a pig and a child, but was eradicated (Pullar and McIntosh 1954). Since then there have been two cases of rabies that were diagnosed; one in 1987 and the other in 1990. Both cases were in children that had been infected with the disease in other countries prior to entering Australia. These cases demonstrated a prolonged incubation period of at least several months.

Epidemiology

The incubation period in animals is highly variable. It is generally 3–8 weeks but can vary from 2 days to 6 months or even longer. Factors that may influence the length of the incubation period are the virus strain involved, viral dose, proximity of the animal bite to the central nervous system and the degree of sensory innervations in the affected location (AHA 2011).

Rabies virus is comparatively fragile and does not survive for long periods outside the host. The virus is inactivated by heat, and is susceptible to ultraviolet (UV) light, lipid solvents (soapy water, ether, chloroform, acetone), 45–75% ethanol, quaternary ammonium compounds (e.g. 0.2% cetrimide) and 5–7% iodine preparations (AHA 2011). Aerosol contamination in bat caves is well recognised.

The virus is shed in saliva from about the time of onset of clinical signs. Virus shedding 1–5 days and up to 13 days before clinical signs appear has been reported.

Recovery from infection is rare but has been recorded in wildlife such as foxes and bats. It is generally accepted that there is no carrier or latent state for rabies.

Rabies transmission occurs primarily through contamination of fresh wounds with infective saliva. Infection therefore can be established through the bite of a rabid animal or licking of cuts or abrasions in skin or mucous membranes. Transmission in a region is primarily influenced by the population density of susceptible (non-vaccinated) host species. Australia has an abundant variety and widespread population of potential wild maintenance species, such as the European red fox, feral cat, wild dogs and the dingo (AHA 2011). The density estimates from Australian studies indicate fox densities are well above those found in many parts of Europe and North America where fox rabies has been endemic (Coman et al. 1991).

Where outbreaks occur, they are locally explosive in the maintenance species and cause a rapid decrease in population. Outbreaks generally last for several months.

Clinical signs

Clinical signs in affected wildlife can be variable and subtle. A common and important feature of rabid wildlife is a change in social behaviour including a loss of fear, ataxia, subsequent aggression towards other animals and people. Wild animals will often lose their natural fear of humans. Animals may wander into urban areas and into buildings. These behavioural changes pose a threat to public health with increased contact resulting from the behaviour changes. Pica and unusual vocalisation which is often incessant, and in dogs may manifest as a low coarse howl, are other notable clinical signs. Excitation is also often seen and may manifest as ceaseless running over long distances, with or without snapping at nearby objects, animals or people. A rabid fox may charge at and bite passing people, animals and even vehicles. Paralysis, paresis and in the terminal stages coma are signs of disease.

Animals may progress rapidly between different clinical signs and there have been occasional reports of death with few prior signs.

Secondary signs noted may include unkempt coat, dehydration, red eyes, salivation, poor condition and signs of trauma due to neurological deficits. Dilated pupils, loss of the corneal reflex and squinting may also be observed.

Diagnosis

Diagnosis is often made on clinical signs in endemic countries however laboratory tests are required for definitive diagnosis. Samples should be submitted to the state or territory government veterinary laboratory from which they will be sent to the Australian Animal Health Laboratory.

Diagnostic tests carried out by laboratories include fluorescent antibody test (the preferred initial test) RT-PCR and sequencing, immunohistochemistry, viral isolation in neuroblastoma cell cultures or viral isolation in mice. A positive result must be notified immediately to the chief veterinary officer (CVO) of the state or territory concerned, who will immediately notify their public health department equivalent.

Pathology

There are no consistent macroscopic lesions in animals that die of rabies (AHA 2011). Gross lesions secondary to neurologic deficits that may be observed include dehydration, loss of condition, lesions secondary to trauma, broken teeth from chewing and biting inappropriate objects and unusual stomach or oral cavity content including soil and plant material in carnivorous species.

Microscopically, significant lesions can be located in the central nervous system and cranial and spinal ganglia (AHA 2011). Perivascular cuffing, focal and diffuse gliosis, neuronal degeneration and intracytoplasmic inclusion bodies can be seen. Occasionally, intracytoplasmic inclusion bodies may also be located in glial, ganglion, salivary gland, adrenal medulla and retinal cells.

No gross or histopathological lesions are pathognomonic for rabies and laboratory testing is required for confirmation. There is no information on clinical pathology.

Differential diagnoses

In wildlife, differential diagnoses for rabies include any disease that can produce neurological deficits, as behavioural changes are the main clinical sign.

Viral encephalitides for the species in question, bacteria such as *Listeria monocytogenes* and mycosis such as cryptococcosis should be considered. Other differentials include protozoa (*Toxoplasma* and *Babesia*) and toxicity including 1080, lead and other heavy metals, organophosphates and urea.

Laboratory diagnostic specimens

For diagnosis whole brain collection is required after natural death or from animals that are euthanased during any stage of the clinical syndrome. Fresh brain is required for all tests with the exception of immunohistochemistry for which a formalin-fixed brain is used. Samples of the head or animal carcass must be chilled and not damaged. Samples of lung, liver and stomach contents should be collected for differential toxicological investigations (AHA 2011).

Treatment

No effective treatment is described for wildlife.

Prevention and control

If an outbreak of rabies occurs, a vaccine may be used in wildlife populations to prevent further spread. Oral vaccinations can be delivered to wildlife by a bait system, using attenuated strain vaccines. Vaccines are incorporated in edible baits and distributed in rabies enzootic areas. During the US and European vaccination campaigns this was done by dropping baits from aeroplanes over the target areas. Vaccines may also be spread manually e.g. by farmers and hunters. The vaccine achieves its effect by producing immunising infection through the oral and pharyngeal route. The efficacy of the vaccines is dependent on various factors including the ability of certain wildlife species to accept the bait and absorption rates of the vaccines via the oropharyngeal mucosa.

An alternative is the 'Trap-vaccinate-release' (TVR) method. This involves capturing live wildlife in cages and administering an intramuscular vaccine injection then releasing the individual. This process can be time consuming and expensive but may be more suitable than oral vaccination strategies due to unavailable baits, areas inhabited with people or areas where there is abundant food supply for wildlife which renders baits to be less effective (AHA 2011).

In an epidemic of rabies in Australia the approach to national, state and local control and prevention is summarised in the AUSVETPLAN (www.animalhealthaustralia.com). The strategy is primarily directed towards minimising and preventing human exposure to rabies infections and preventing its establishment in an urban area. The approach will focus on identification and control of the biotype and the susceptibility of its preferred host through vaccination where possible (Forman 1993). In Indonesia, despite 70% of dogs in East Flores being culled, rabies was neither contained nor eliminated and managed to spread across the entire island. At the time vaccination was not used (Windiyarningsih et al. 2004). Recently vaccination has proven to be more successful.

Surveillance and management

The threat to Australia from rabies is taken very seriously by Australian governments. The rabies AUSVETPLAN (AHA 2011) and the rabies Series of National Guidelines (SoNG) are regularly reviewed and updated (Communicable Diseases Network 2013). A working group established by Australia's Animal Health Committee examined in detail rabies preparedness for animals in Australia (Anderson 2015).

Australia's animal health and wildlife health surveillance systems include consideration of rabies as part of general surveillance activities. During an incursion, wildlife surveillance and monitoring will focus on any potential maintenance, and to a lesser extent, spill-over hosts. The policy is for eradication.

Monitoring of wildlife should include careful observation of animals behaving abnormally and testing for the presence of rabies in animals showing neurological signs.

Statistics

An estimated 40,000 to 100,000 human deaths are caused by rabies each year worldwide; in addition, millions of persons, primarily in developing countries of the subtropical and tropical regions, undergo post exposure treatment (Rupprecht et al. 1995; OIE 2018).

In 2015, there were over 5,500 cases of animal rabies reported in the U.S, 92% of which were wildlife (Birhane et al. 2017). These animals can expose humans or pets to rabies. In the U.S., typically 1-3 cases of human rabies are reported per year (CDC 2018).

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

There have been no reports of rabies in wildlife from Australia's States and Territories in this time.

Research

An Animal Health Committee rabies preparedness working group identified key activities to improve preparedness for rabies in Australia (Anderson 2015).

Human health implications

Advice regarding human health implications of rabies should be sought from the local public health department. The public health threat of rabies as a preeminent zoonoses relates to the acute, incurable encephalitis that results from transmission of the virus by the bite of an infected animal (Rupprecht et al. 1995). Human infections have no significance in the epidemiology of rabies. In humans the most cost-effective prevention strategy is by eliminating rabies in dogs through animal vaccinations where the disease is endemic. Pre- and post-exposure prophylaxis are available for rabies in humans. In Australia, preventing the introduction of rabies is the best measure. More information on rabies can be found at the Australian Government Department of Health website (see [www.health.gov.au/internet/main/publishing.nsf/content/ED62D139B56F7B80CA257BF0001B7422/\\$File/ABLV-Rabies-SoNG.pdf](http://www.health.gov.au/internet/main/publishing.nsf/content/ED62D139B56F7B80CA257BF0001B7422/$File/ABLV-Rabies-SoNG.pdf)).

Conclusions

Australia is free of rabies virus. The introduction of rabies could have a significant impact on public health, wildlife and animal health. The most realistic risk of rabies introduction into Australia is entry of a dog-adapted virus in a dog that then comes in contact with wild dogs. This would be difficult to detect and report and may result in dissemination in the wild animal population. Surveillance of rabies in Australian wildlife is important in preventing the potential for cross transmission from wildlife into domestic and wild dogs that may subsequently be the sources of human rabies infections.

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To provide feedback on this fact sheet

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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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