Wobbly possum disease

Fact sheet

August 2023

Key points

- "Wobbly possum" is a term used to describe neurological disease in common brushtail possums. The term may be applied to either:
  - *wobbly possum disease* caused by *wobbly possum disease virus* (or closely-related viruses) or
  - a *syndrome* of "*neurologically wobbly possums",* of unknown aetiology, seen sporadically in brushtail possums in eastern areas of mainland Australia.
- *Wobbly possum disease virus* (an arterivirus) was first discovered in common brushtail possums in New Zealand. Similar viruses have been since been found in possums in Tasmania and in mainland Australia.
- Serological evidence of exposure to these viruses has also been found in common ringtail possums and mountain brushtail possums in Australia.
- This Fact Sheet provides information on *wobbly possum disease* (caused by *wobbly possum disease virus*) and "neurologically wobbly possum syndrome", of unknown cause.

Aetiology

Wobbly possum disease (WPD) was first identified in a NZ research facility in 1995 [1] and has been extensively studied in NZ [2]. The aetiological agent of WPD in NZ is known to be *wobbly possum disease virus* (WPDV) [3, 4].

Wobbly possum disease virus is an RNA virus in the family *Arteriviridae,* subfamily *Zealarterivirinae*, order Nidovirales. WPDV is the only species of the only genus (*Kappaarterivirus*) in this subfamily. The virus may be a split into separate species in the future as there is considerable variability between genetics of WPDV isolated in NZ and Australia [5].

Wobbly possum disease syndromes in Australian possums have not been as extensively studied as in NZ. Investigations into wobbly possum cases in common brushtail possums (*Trichosurus vulpecula*; BTP) in Tas have identified an arterivirus (very similar to WPDV in NZ) as the likely causative agent of the disease in this state [6]. Two divergent WPDV sequences were identified in archived tissues from clinically affected BTP from NSW. These two newly-identified Australian WPDV viruses were 71-74% identical to each other and to the NZ variant [7]. Although it appears that a WPD-like virus is responsible for WPD cases seen in Tas in recent years, it is not known if these two viruses are responsible for all cases of "neurologically wobbly possums" seen in Australia.

WPDV appears to have separated early in the evolution of the family *Arteriviridae*, suggesting that it may have co-evolved with its possum host [7, 8]. WPDV was most likely brought to NZ when BTP were originally introduced from Tas (and mainland Australia) to NZ in the 1800s. Arteriviruses are known

to mutate rapidly, which may explain the apparent genetic differences between the Australian and NZ arteriviruses [8].

One Health implications

Wildlife and the environment: there is no evidence of a population level impact from WPD in Australia [9, 10]. In NZ, WPDV is being investigated as a tool for biological control of BTP, which are a pest species in that country. There is no evidence that wildlife species other than possums are susceptible to infection with WPDV or related arteriviruses.

Domestic animals: there is no evidence that domestic animals are susceptible to infection with WPDV or related arteriviruses.

Humans: there is no evidence that humans are susceptible to infection with WPDV or related arteriviruses.

Natural hosts and distribution

Wobbly possum disease and WPDV have been described in BTP in NZ. Recent cases in BTP in Tas, consistent with WPD, have been linked to a similar arterivirus [10]. On mainland Australia, cases histologically consistent with WPD have been reported in BTP in NSW, Vic and Qld [7, 11].

The syndrome of "neurologically wobbly possums" (of unconfirmed aetiology) has been described in BTP in the eastern areas of mainland Australia for many years. It has been reported on a sporadic basis from wild BTP in these areas [12].

Although clinical disease associated with WPDV has only been reported in BTP, antibodies to WPDV have been found in mountain brushtail (*T. cunninghami*) and common ringtail (*Pseudocheirus peregrinus*) possums, (as well as BTP), from mainland Australia with an estimated seroprevalence of 22%. It is not known if species of possums other than BTP experience disease as a result of infection with WPDV. None of the seropositive possums (where tissue samples were available) were positive for WPDV RNA [8]. This suggests that WPDV infection may have occurred at some point in the past or that tests were not able to detect the WPDV variants circulating in mainland possums. The large percentage of apparently healthy, seropositive BTP both in NZ and Australia suggests that some BTP may not develop clinical disease when exposed to WPDV [13].

Epidemiology

New Zealand: early studies of WPD in NZ showed a high case fatality rate with an incubation period between five and 20 days in experimentally-infected possums [1]. In more recent studies, BTP experimentally-infected with WPDV developed neurological disease and histopathological lesions consistent with WPD [4].

Studies indicate close contact is required for transmission. The route of transmission has not been confirmed but virus is likely transmitted via direct contact or fomites, with aerosol or droplet transmission unlikely. It is thought that under natural conditions the virus is spread through ingestion of contaminated food, fighting, contamination of wounds with urine or transfer via mites

[14]. Infection is likely to be persistent and presence of antibodies does not indicate resistance to infection or disease [15]. The infectious agent is thought to spread slowly through natural means.

An earlier study showed the presence of WPD in wild possums in NZ was not related to geographical location, sex or body weight of the animal and that the natural prevalence of WPD and rates of cross-infection within a site were low [16]. More recent studies show a variability in seropositivity across different geographic areas of NZ. Factors such as possum density, den sharing, landscape or urbanisation of the environment may influence local transmission dynamics [15].

The virus is considered to have limited survival in the environment and is rapidly degraded by most disinfectants.

Surveys in BTP in NZ found between 4 and 17% of individuals affected by the disease [16, 17]. A serological survey of BTP in NZ found 21% carried antibodies to WPDV [15].

Tasmania: in 2019, a cluster of cases (n>25) of "neurologically wobbly possums" was detected in BTP, mainly from the greater Hobart region. Previously, occasional cases had been reported from other regions of Tas. Cases mostly involved single animals. Investigation confirmed (for the first time in Australia) the involvement of an arterivirus, similar to the one found to cause WPD in BTP in NZ [10]. Archived samples from BTP in northern Tas (collected in 2015 and 2016) also detected the virus [9]. Investigations into the epidemiology of the disease in Tas are continuing. Current information indicates that the Tas arterivirus is responsible for the cases of WPD but there is no detectable population level effect [9, 10].

Factors contributing to the emergence of WPD in these distinct geographical locations are unknown.

Mainland Australia: *wobbly possum syndrome* was diagnosed via histopathology in 21 of 31 BTP with neurological disease from the Sydney region, sampled 1998-2010. Virus was not identified in these cases [18]. A study of archived samples from BTP in the greater Sydney area with neurological signs consistent with WPD found that the majority of cases occurred in adult female BTP (35 female, 9 male, 6 unknown sex, 42 adult, 3 juvenile, 4 unknown age) [7].

In an earlier study, 30 of 540 wild BTP submitted to Taronga Zoo wildlife clinic between 1985-1993 had signs of depression and blindness. The majority of these had chronic non-suppurative meningoencephalitis, suggestive of WPD, but a virus was not identified [19].

Little is known about other possible causes of *wobbly possum neurological syndrome* in BTP in Australia. Cases have been reported to occur sporadically [7, 8, 12, 19] and an alternative, undetermined viral cause has been hypothesised [20].

Clinical signs

WPD in NZ and Tas: signs are consistent with multifocal neurological disturbances, appearing to involve the vestibular system. Early signs (detectable in captivity) include decreased appetite and weight loss. Neurological signs include docility and dullness, gait abnormalities (including incoordination, loss of balance, head tilt and circling, difficulty climbing, stumbling, lameness and

abnormal hindlimb gait), aimless wandering, daytime feeding, wasting and blindness. Anaemia and hyperglobulinaemia also occur [4, 10, 14, 21]. The disease progresses over several weeks.

***Wobbly possum neurological syndrome* in mainland Australia:** signs include mental depression, ataxia, blindness and persistently dilated pupils. The disease course progress over weeks to months [7, 12, 19, 22]. Ophthalmological exam of blind individuals may show a pale optic disc with changes to fundus vascular tuft and a fully dilated, non-responsive pupil. Weight loss may be seen [12].

Differences in clinical signs and pathology (below) probably reflect the different variants of the virus [13].

Diagnosis

Wobbly possum disease should be suspected in cases of neurological disease in BTP. Initial diagnosis is based on presenting clinical signs in conjunction with typical histological changes (see 'Pathology' below).

A diagnosis of WPD due to WPDV can be confirmed with laboratory testing. Immunohistochemistry can be used for the detection of arterivirus antigen in formalin-fixed tissues. A RT-qPCR is able to detect the virus in a range of fresh tissues [23].

A recombinant ELISA is available in NZ to detect antibodies to the virus in serum [15].

***Wobbly possum neurological syndrome*:** diagnosis of wobbly possum syndrome in Australian BTP is based on clinical signs in conjunction with typical histological changes.

Laboratory diagnostic specimens and procedures

A complete necropsy should be performed in all suspected cases of wobbly possum disease, regardless of suspected aetiology. Fresh samples of liver, kidney, spleen (and brain, if possible) should be collected for PCR. Additional samples of these tissues should be collected for histopathology. Fresh and fixed samples should be submitted to the relevant state or territory veterinary laboratory.

Pathology

WPD in NZ and Tas: histologically there is perivascular infiltration of a range of tissues with mononuclear leukocytes, especially lymphocytes and plasma cells. There tends to be a mild to moderate nonsuppurative meningoencephalitis with more pronounced changes occurring in the liver and kidney, including periportal mononuclear cell infiltrates, focal hepatic necrosis, mild non-suppurative myocarditis and non-suppurative interstitial nephritis. Similar lesions have been reported from the salivary gland, spleen, lung, bladder and lymph nodes. Some animals have low body fat reserves. There are no other typical gross findings [12, 24].

***Wobbly possum neurological syndrome* in mainland Australia:** histologically there is a non-suppurative meningoencephalitis with non-suppurative perivascular inflammation throughout the brain parenchyma. In addition, non-suppurative inflammation and Wallerian degeneration in the optic tract commonly occurs along with atrophy of the cerebellar folia and retina. The eyes may also show foci of tapetal discolouration, a pale optic disc or an optic disc that lacks the normal vascular

tuft [12, 19, 22]. This contrasts with the NZ and Tas cases, in which the inflammatory infiltrate is multisystemic. In addition, in mainland Australian cases, infiltrates may be more histiocytic with concurrent vasculitis, compared to NZ and Tas cases [12].

Differential diagnoses

Other differential diagnoses include traumatic injury, toxoplasmosis and other infectious and non-infectious causes of neurological disease. Although Australian bat lyssavirus has never been reported in wildlife other than bats, testing of Australian possums with neurological disease and/or encephalitis for ABLV should be considered.

Treatment, prevention and control

There are no known treatment options. The disease is usually fatal in BTP. Control and prevention of WPD in NZ is not considered desirable and it the virus is being investigated as a possible biological control agent.

Known or suspected cases of wobbly possum disease in Australia, if animals are in care, should be held in isolation from other possum species, as spread of the virus may occur through contact or fomites. General biosecurity practices including personal and equipment hygiene should be adopted when handling known or suspect cases [10](see also National Wildlife Biosecurity Guidelines www.wildlifehealthaustralia.com.au/Portals/0/Documents/ProgramProjects/National_Wildlife_Biosecurity_Guidelines.PDF for more information).

Although there is no evidence that other marsupial species are susceptible to infection with WPDV or related arteriviruses, it is recommended, as a precaution, that carcasses of clinically affected BTP are not fed to other marsupial species in captivity. The carcasses of BTP to be used as feed for native species should be sourced from areas where there is no recent evidence of WPDV.

Research

Research continues in NZ to better understand the epidemiology of WPD and investigate the potential for WPDV to be used as a possum control agent.

Research on cases of WPD in Australia continues, to confirm the aetiological agent(s), to understand the epidemiology of the disease and to investigate the significance of viral and antibody findings in other species of possum.

Surveillance and management

Wildlife Health Australia administers Australia's general wildlife health surveillance system, in partnership with government and non-government agencies. Wildlife health data is collected into a national database, the electronic Wildlife Health Information System (eWHIS). Information is reported by a variety of sources including government agencies, zoo based wildlife hospitals, sentinel veterinary clinics, universities, wildlife rehabilitators, and a range of other organisations and individuals. Targeted surveillance data is also collected by WHA. See the WHA website for more information https://wildlifehealthaustralia.com.au/Our-Work/Surveillance and

https://wildlifehealthaustralia.com.au/Our-Work/Surveillance/eWHIS-Wildlife-Health-Information-System.

We are interested in hearing from anyone with information on this condition in Australia, including laboratory reports, historical datasets or survey results that could be added to the National Wildlife Health Information System. Suspected cases of WPD should be reported to your WHA coordinator (see https://wildlifehealthaustralia.com.au/Incidents/WHA-Coordinator-Contacts). If you are in Tas and suspect a case of WPD, please contact the Dept of Natural Resources and the Environment [10].

There a number of reports of WPD in the National Wildlife Health Surveillance Database (eWHIS) including many recent cases from Tasmania.

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Wildlife Health Australia recognises the Traditional Custodians of Country throughout Australia. We respectfully acknowledge Aboriginal and Torres Strait Islander peoples' continuing connection to land, sea, wildlife and community. We pay our respects to them and their cultures, and to their Elders past and present.

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