

# Neurological syndromes in brushtail possums (“wobbly possum”)

## Fact sheet

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### Introductory statement

“Wobbly possum” is a term used to describe neurological disease in brushtail possums (*Trichosurus vulpecula*; BTP). It may be applied to either:

- “wobbly possum disease (WPD),” caused by wobbly possum disease virus (WPDV)
- neurological disease of unknown origin, seen sporadically in BTP in Australia (eastern areas of mainland Australia).

Wobbly possum disease virus was first discovered in BTP in NZ and was previously thought to be unique to possums in NZ. Recent evidence suggests that a similar virus may be the cause of at least some of the cases of wobbly possum seen in Tasmania.

This Fact Sheet provides information on the situation in New Zealand and in Australia.

### Aetiology

Wobbly possum disease was first identified in a research facility in NZ in 1995 (Mackintosh et al. 1995) and has been extensively studied in free-living possums in NZ (Perrott et al. 1999; Perrott et al. 2000a). In recent years the aetiological agent of WPD in NZ has been confirmed to be a novel Nidovirus (wobbly possum disease virus; WPDV), most closely related to the family *Arteriviridae* (Dunowska et al. 2012; Giles et al. 2016; Gulyaeva et al. 2017). The nidoviruses associated with neurological disease in BTP are only very distantly related to arteriviruses known to cause disease in eutherian mammals (Kuhn et al. 2016).

Although the aetiology of wobbly possum in BTP in Australia is not confirmed, recent investigations into wobbly possum cases in BTP in Tasmania have identified a nidovirus (very similar to the one in NZ) as the likely causative agent of the disease in this state. It is not known at this stage if this virus is responsible for all cases of wobbly possum seen in Australia, or if the virus is present in BTP from the mainland of Australia.

The epidemiological relationship between the nidovirus in NZ and the nidovirus in Tasmania is unclear. Brushtail possums were originally translocated from Tasmania (and mainland Australia) to NZ. Arteriviruses are known to mutate rapidly, which may explain the apparent genetic differences between the Tasmania and NZ nidoviruses.

The aetiology of wobbly possum cases in BTP in mainland Australia is not currently known. Limited diagnostic investigations have not implicated nidovirus in these cases, however the involvement of a nidovirus remains a possibility. An alternative viral cause (undetermined) has also been hypothesised (Bender 2019).

## Natural hosts and distribution

Wobbly possum disease and WPDV have been described in brushtail possums in NZ. Recent cases in BTP in Tasmania, consistent with WPD, have been linked to a similar nidovirus.

The syndrome of “wobbly possum” (unconfirmed aetiology) has been described in BTP in the eastern areas of mainland Australia. It is reported on a sporadic basis from wild BTP in these areas (Ladds 2009). No other possum species (or other species) are thought to be affected.

## Epidemiology

### WPBV in New Zealand and Tasmania

Early studies of WPD in NZ showed a high case fatality rate with an incubation period between five and 20 days when possums were inoculated intra-peritoneally with tissue suspensions (Mackintosh et al. 1995). In more recent experimental studies, possums that received inoculum from WPD-affected possums, and possums that received purified viral isolate developed neurological disease and histopathological lesions consistent with WPD (Giles et al. 2016).

Studies in a controlled setting indicate close contact is required for transmission. Virus is likely transmitted via direct contact or fomites, but aerosol or droplet transmission is unlikely. It is hypothesised that under natural conditions the virus is spread through ingestion of contaminated food, fighting, contamination of wounds with urine or transfer via mites (Perrott et al. 2000b). Infection is likely to be persistent and presence of antibodies does not indicate resistance to infection or disease (Giles et al. 2018a).

An earlier study in NZ showed the presence of WPD in wild possums 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 (Thompson and McLeod 1999). 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 (Giles et al. 2018a).

The agent remains infective after freezing at -75°C and can pass through a 0.22 micron filter (O'Keefe et al. 1997). The virus is considered to have limited survival in the environment and is rapidly degraded by most disinfectants.

The infectious agent is thought to spread slowly through natural means, as close contact between individuals is required.

In 2019, a cluster of cases (n>20) of wobbly possum was detected in Tasmania, primarily from the greater Hobart region. Previously, occasional cases had been reported from other regions of Tasmania. Most cases involved individual animals, with one event involving several animals from the same property. Investigation confirmed (for the first time in Australia) the involvement of a nidovirus, similar to the one found to cause WPD in BTP in NZ. Archived samples from BTP in northern Tasmania (collected in 2015 and 2016) also detected the virus.

The findings to date (although based on limited data) suggest that WPD in Tasmania is similar to the disease seen in New Zealand. Further investigations into the epidemiology of the disease in Tasmania are continuing, however current data indicates that there is no detectable population level effect.

### **Wobbly possum syndrome in mainland Australia**

Little is known about the epidemiology of the neurological syndrome in Australia. Cases tend to occur sporadically (Rose 1999; Ladds 2009).

### **Clinical signs**

**WPD in New Zealand and Tasmania:** signs consistent with multifocal neurological disturbances, clinically 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 (Perrott 1998; Perrott et al. 2000b; Giles et al. 2016). The disease progresses over several weeks.

**Wobbly possum syndrome in mainland Australia:** signs include mental depression, ataxia, blindness and persistently dilated pupils. The disease course progress over weeks to months (Rose 1999; Ladds 2009; Hufschmid and Holz 2012). 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 (Ladds 2009).

### **Diagnosis**

**WPD in New Zealand and Tasmania:** wobbly possum disease should be suspected in cases of neurological disease in BTP in areas where the disease is known to be present. Initial diagnosis is based on presenting clinical signs in conjunction with typical histological changes (see 'Pathology' below).

A diagnosis of WPD can be confirmed with laboratory testing. Immunohistochemistry can be used for the detection of nidovirus antigen in formalin-fixed tissues. The ideal samples for agent detection are fresh and formalin fixed liver, kidney, spleen and brain. A RT-qPCR is able to detect the virus in a range of fresh tissues (Dunowska et al. 2013). The CSIRO Australian Animal Health Laboratory (AAHL) is developing a novel real-time PCR assay for the rapid detection of nidovirus from fresh tissue samples.

A recombinant ELISA has been developed in NZ to detect antibodies to the virus in serum (Giles et al. 2018a).

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

### **Pathology**

**WPD in New Zealand and Tasmania:** Pathology presents histologically as 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 (Ladds 2009; Giles et al. 2018b).

**Wobbly possum 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 (Rose 1999; Ladds 2009; Hufschmid and Holz 2012). This contrasts with the NZ and Tasmanian 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 Tasmanian cases (Ladds 2009).

## Differential diagnoses

Infection with nidovirus (WPD) should remain a differential diagnosis for BTP presenting with wobbly possum in mainland Australia.

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.

## Laboratory diagnostic specimens

A complete necropsy should be performed in all suspected cases of wobbly possum.

Fresh samples of liver, kidney, spleen (and brain, if possible) should be collected for PCR testing. Additional samples of liver, kidney, spleen and brain should be collected into 10% neutral buffered formalin for histopathology. Both fresh and fixed samples should be submitted to the relevant state or territory veterinary laboratory.

## 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 is being investigated as a possible biological control agent.

Known or suspected cases of wobbly possum in Australia, if in care, should be held in isolation from other possum species. General biosecurity practices including personal hygiene should be adopted when handling known or suspect cases (see National Wildlife Biosecurity Guidelines [www.wildlifehealthaustralia.com.au/Portals/0/Documents/ProgramProjects/National\\_Wildlife\\_Biosecurity\\_Guidelines.PDF](http://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 nidoviruses, as a precautionary approach, it is recommended that the carcasses of clinically affected BTP are not fed to other marsupial species (e.g. Tasmanian devils 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 WPD.

There are no recommendations for control of the Australian neurological syndrome of unknown cause because the aetiology is unknown and it appears to exist at a low level in the possum population.

## Surveillance and management

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. See the WHA website for more information:

[www.wildlifehealthaustralia.com.au/ProgramsProjects/eWHISWildlifeHealthInformationSystem.aspx#requests](http://www.wildlifehealthaustralia.com.au/ProgramsProjects/eWHISWildlifeHealthInformationSystem.aspx#requests).

Suspected cases of WPD should be reported to your WHA coordinator (see [www.wildlifehealthaustralia.com.au/AboutUs/ContactDetails.aspx](http://www.wildlifehealthaustralia.com.au/AboutUs/ContactDetails.aspx)).

Wildlife Health Australia is also interested in receiving reports of wobbly possum or other neurological disease in possums in Australia. Contact [admin@wildlifehealthaustralia.com.au](mailto:admin@wildlifehealthaustralia.com.au).

## Statistics

There a small number of reports in the National Wildlife Health Surveillance Database (eWHIS) including the cluster of 2019 cases from Tasmania. There is a report of a wild, blind, ataxic BTP from NSW which was euthanased in 2005 and diagnosed with “wobbly possum” syndrome.

Between 1985 and 1993, 540 BTP were submitted to the wildlife clinic at Taronga Zoo. Thirty of these had clinical signs of depression and blindness. Tissues from these animals, along with an additional 12 possums from NSW, Victoria and Tasmania were examined histologically. Twenty-three had chronic non-suppurative meningoencephalitis (Rose 1999). Of 31 BTP with neurological disease, sampled from 1998 to 2010 in the Sydney region, wobbly possum syndrome was diagnosed in 21 (68%), via histological examination (Ma et al. 2013).

A survey in 1999 of 887 BTP in NZ found histopathological evidence of wobbly possum disease in 39 (4.4%) of the possums sampled (Thompson and McLeod 1999). A second survey in 2005 found one positive possum out of six that were necropsied (McLeod 2007). A survey of 230 archived BTP serum samples in NZ found almost 21% positive via ELISA for exposure to WPD virus (Giles et al. 2018a).

## Research

Research is underway in NZ to better understand the epidemiology of WPD and ascertain the potential for this disease to be used as a possum control agent.

Research on the cases of neurological disease in Australia is necessary to determine/ confirm the aetiological agent(s) and to understand the epidemiology of the disease.

## Human health and domestic animal implications

There are no concerns about zoonotic potential or transmission to domestic animal species.

## Conclusions

Wobbly possum disease, caused by a nidovirus, has been detected in NZ and Tasmanian brushtail possums. The origin of the viruses is unknown and the epidemiological links between the disease in NZ and the disease in Tasmania remains unclear. In both cases, it is not known what factors have contributed to the emergence of this disease in these distinct geographical locations. The disease in wild possums in NZ and Tasmania does not appear to be exerting a significant population level effect.

The syndrome in mainland Australia appears to occur sporadically and to affect low numbers of wild possums. The cause is not determined, however involvement of WPDV or a related nidovirus cannot be excluded at this stage. There is no evidence of a population level impact from these cases.

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## To provide feedback on this fact sheet

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. Negative data are also valuable. If you can help, please contact us at [admin@wildlifehealthaustralia.com.au](mailto: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](mailto: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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