**Tammar wallaby sudden death syndrome**

**Fact sheet**

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**Introductory statement**

Tammar Wallaby Sudden Death Syndrome (TSDS) is caused by viruses of the Orbivirus genus. Blindness in kangaroos is also caused by an orbivirus, but one from a different serogroup than the orbivirus causing TSDS. Orbiviruses are usually spread by biting insects (often Culicoides and similar species) and outbreaks usually occur in early summer following periods of very heavy rain, when insects were abundant. TSDS is the subject of on-going research.

**Aetiology**

Viruses of the *Orbivirus* genus (family *Reoviridae*), from the Eubenangee serogroup (Kirkland 2005; Rose *et al.* 2012). The syndrome was first reported by Rose *et al.* (1999).

**Natural hosts**

TSDS has only been reported in the tammar wallaby (*Macropus eugenii*). Tammar wallabies are small, nocturnal macropods that originally ranged throughout south-western Australia, southern South Australia and offshore islands of these states. Free-ranging tammars are now restricted to small areas of Western Australia, a small reintroduced colony at the tip of the Yorke Peninsula in South Australia and several offshore islands. Tammars are maintained at a number of mainland captive breeding centres, zoological parks and research centres where they are an important model species for the study of marsupial biology, behaviour and physiology (Kirkland 2005).

The susceptibility of other macropod species is not known.

**World distribution**

This syndrome has only been reported in tammar wallabies in Australia.
**Occurrences in Australia**

In October 1998, a research facility in Sydney observed a sudden increase in deaths in its tammar collection. Over a six week period 85 of the 234 tammars died. Sudden deaths were observed in six other research facilities and zoological gardens in NSW between October and December 1998 and in one research facility in Queensland in March 1999. In total 120 deaths were confirmed although as many as 230 tammars may have died, the majority without premonitory signs (Rose et al 2000, Kirkland 2005).

Virus (designated NSW’98) was isolated from tissue samples collected from affected tammars from the Sydney outbreak and a similar Orbivirus, with 99% homology to the NSW ’98 virus (designated Z842) was isolated from a tammar that died suddenly at the Queensland research facility (Rose et al. 2012).

A suspected outbreak occurred in western Sydney in 2007 (eWHIS 1432), however no virus was isolated.

Between November 2010 and March 2011, 35 out of 70 tammars died in the enclosures at Tidbinbilla Nature Reserve, ACT, with peak mortalities occurring in December and January. During this same period two Canberra research and breeding facilities reported deaths in 28 out of 60 and 60 out of 200 animals. Approximately 40 deaths were also reported from a Newcastle research and breeding facility (eWHIS 3473 and 3522). A single death was recorded in April 2011 from a small captive tammar wallaby colony in Queensland.

TSDS is suspected as the cause of these recent mortality events based on history, environmental conditions, clinical signs and necropsy findings consistent with previous incidents. The timing of these events was also consistent with past outbreaks of TSDS, which occurred in early summer following periods of very heavy rain, when insects were abundant (Animal Health Australia 2011).

Detailed case records are held in the Australian Registry of Wildlife Pathology Database (www.arwh.org). Summary data are available in the National Wildlife Health Information System database (www.wildlifehealthaustralia.com.au). Both of these information sources have controlled access.

**Epidemiology**

Orbiviruses are arthropod borne viruses, usually spread by biting midges (Culicoides spp.). The epidemics of TSDS commenced in the early summer months following periods of very heavy rain and when insects were abundant. As TSDS has only been described from Queensland, NSW and the ACT it is possible that the disease vector is not present in other parts of Australia. Therefore, it could be argued that these epidemics may have been the result of human intervention through the movement of a susceptible macropod species into a geographical area that is outside its usual habitat and into the potential range of a pathogenic orbivirus (Kirkland 2005).

Mortality rates have been in the range of 38% - 60% at the different sites where disease has occurred. There is no information in the literature on: morbidity rate; incubation period; transmission; sources of agent; shedding (when, route); which tissues/ fluids are infectious; when and how the new host become infected; if there a carrier state; and, if there is any age and/or sex-linked predisposition. Whether other macropod species are susceptible is also unknown.
Clinical signs

In all cases reported, sick animals died within 12 hours of being noticed ill (Kirkland 2005). The majority of animals died without premonitory signs. However, a small proportion exhibited lethargy, depression, increased respiratory rate, an inability to hold the head up, ataxia, and were in lateral recumbency with muscle fasciculations (Rose et al. 1999, Rose et al 2000). In the 2010-11 outbreaks, clinical signs reported included sudden death, moribund animals and paddling, which was rare.

Diagnosis

Diagnosis is by signalment, gross pathology, histology and virus isolation.

Clinical pathology

Clinical pathology of TSDS has not been described.

Pathology

Post mortem findings consisted of extensive congestion and haemorrhage in a range of thoracic and abdominal organs, major muscle groups and in subcutaneous tissues (Kirkland 2005). In one event, gross post mortem findings consistently included massive pulmonary congestion, mottled hepatic parenchyma, and subcutaneous oedema throughout the hind limbs and inguinal region. Approximately 30% of the animals examined also had extensive haemorrhage within the fascial plains and skeletal muscle of the hind limb adductors, inguinal region, ventral thorax, dorsal cervical region, and peri-renal retroperitoneal area. Marked and diffuse pulmonary congestion, hepatic congestion, and necrosis of lymphoid germinal centres were consistent findings upon microscopic examination of the tissues. Additional findings in approximately 30% of the animals examined included gastric ulceration and acute, periacinar hepatocellular necrosis. The tissues of affected animals became autolytic within a short period after death (Rose et al. 1999, 2000 and 2012). Gross pathology noted in the 2010-11 outbreak also included some animals with no lesions. It was also noted that many animals were in excellent body condition.

Detailed case records, blocks and glass slides are held in the Australian Registry of Wildlife Health (www.arwh.org), which should be contacted for more detailed information on pathology, diagnosis and specimen collection.

Differential diagnoses

Bordetella bronchiseptica infection (K Rose pers. comm. 2007).

Brodifacoume or other anticoagulant rodenticides (Rose et al. 1999).

Laboratory diagnostic specimens

Procedures for specimen collection should follow those presented by Rose (2007). Key organs to target for virus isolation include heart, liver, intestine, cerebral cortex and cerebrospinal fluid.

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1 Toxicological examination of gastric and colonic contents.
Laboratory procedures

In previous events, Orbiviruses have been isolated from: cerebral cortex, cerebrospinal fluid, myocardium, lung, spleen, liver, and intestine (Rose et al. 1999, Rose et al. 2000).

Treatment

No information is available on treatment of TSDS.

Prevention and control

If this is a vector-borne disease, then exclusion of vectors during and after periods of heavy rain may be worth attempting. Suggested preventive methods include improved drainage of soils around macropod holding yards, regular treatment of tammar wallabies with long-acting pyrethrin insecticides during the summer months and treatment of animal shelter areas with dichlorvos impregnated strips (Rose et al. 2012).

Techniques for control during an outbreak have not been described for this disease. Information on management of bluetongue, which could be used as a model is available at: http://www.animalhealthaustralia.com.au/programs/emergency-animal-disease-preparedness/ausvetplan/ (Animal Health Australia 2008).

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. Please contact admin@wildlifehealthaustralia.com.au.

There is no targeted surveillance program for TSDS in Australia. However, cases of TSDS are usually captured as part of Australia’s general wildlife surveillance system (www.wildlifehealthaustralia.com.au).

Statistics


Research

The Australian Registry of Wildlife Health and the staff of the Virology Laboratory at the Elizabeth Macarthur Agricultural Institute (EMAI) in New South Wales are currently investigating the cause of mortalities in tammar wallaby (Macropus eugenii) populations in a number of locations in Australia. Key research questions have not been published for TSDS. However, a priority is likely to be a better understanding of the

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2 AUSVETPLAN is a series of technical response plans that describe the proposed Australian approach to an exotic animal disease incursion. The documents provide guidance based on sound analysis, linking policy, strategies, implementation, coordination and emergency-management plans.
epidemiology and host range of the disease. Case material from the most recent event (2011) is being reviewed by the Australian Registry of Wildlife Health and will be published in due course. Publication of this information may shed light on the range of susceptible species.

**Human health implications**

The human health implications of TSDS are not known.

**Conclusions**

Orbiviruses are arthropod borne viruses, usually spread by biting midges (*Culicoides* spp.). Bluetongue is another orbivirus and some aspects of TSDS in wallabies resemble aspects of peracute bluetongue in sheep as is found overseas (Kirkland 2005). More work is required on the epidemiology and impact of TSDS in the Australian context.

**References and other information**


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3 Bluetongue and several other orbiviruses infecting ruminants are present in northern Australia, but despite intensive surveillance there have not yet been any reported occurrences of any of these viruses causing clinical disease in livestock in Australia under natural conditions (Geering et al., 1995).
Acknowledgements

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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. If you can help, 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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