Hydatid disease in
Australian wildlife

Fact sheet

Introductory statement

Hydatid disease is caused by infection with the intermediate stage of the tapeworm *Echinococcus granulosus*. *E. granulosus* is widespread in mainland Australian wildlife and its reproductive potential may be greater in wildlife than in domestic animals. The parasite is zoonotic; humans may develop hydatid cysts following accidental ingestion of eggs (released by the tapeworms passed into the environment in faeces of the definitive hosts). Infected definitive hosts (dogs, dingoes, their hybrids and foxes) may pose a public health risk but even heavy parasite loads are of no significance to the animals’ health. The cystic intermediate stage of the parasite is pathogenic in macropods. It is found at a higher prevalence and is potentially fatal in smaller macropods including endangered rock-wallabies and the bridled nail-tail wallaby. Wild macropods are an important sylvatic reservoir for this parasite in Australia and provide a pathway of transmission of disease to domestic animals and humans. Ongoing research is required to better understand the epidemiology and importance of infection in Australian wildlife.

Aetiology

Cestode, Family *Taeniidae*, genus *Echinococcus*, species *granulosus*, strain G1 (common sheep strain). Originated in Europe. The distinction between domestic and sylvatic strains is no longer recognised. *E. granulosus* has a two-host or indirect lifecycle and causes hydatid disease in its larval form, in the intermediate host.

Natural hosts

Intermediate hosts for the common sheep strain in Australia include sheep, macropods, wombats, humans, pigs and cattle (dead end hosts). Prevalence in domestic species is reported to increase with age (Baldock et al. 1985). Macropods are considered naive intermediate hosts as it is thought they have only been recently exposed to the parasite. Dogs, dingoes and their hybrids and foxes are the recognised definitive hosts.
**World distribution**

*Echinococcus granulosus* is found almost worldwide. It is most prevalent in parts of Eurasia, North and East Africa, Australia and South America (McManus et al. 2003).

**Occurrences in Australia**

*Echinococcus granulosus* is thought to have been introduced to Australia with domestic dogs and sheep at the time of European settlement (Jenkins and Macpherson 2003). The first report of the parasite in Australian wildlife was of a black-striped wallaby (*Macropus dorsalis*) with multiple lung cysts (Bancroft 1890). It is now widespread in macropods and domestic stock along the Great Dividing Range in Victoria, NSW and Qld and the hills around Perth in WA (Jenkins and Macpherson 2003). It has been reported in wombats in Vic (Grainger and Jenkins 1996). Reported prevalence in macropods varies (0-67%); higher prevalence has been recorded in smaller species, notably swamp wallabies (*Wallabia bicolor*) and black-striped wallabies (*Petrogale penicillata*) were infected (Barnes et al. 2008a). Marked clustering of infection has been recorded in eastern grey kangaroos (*Macropus giganteus*) but risk factors have not been identified (Barnes et al. 2007b). Infection has been reported in Lumholtz’s tree-kangaroos (*Dendrolagus lumholtzi*) in the Atherton Tablelands of north Qld (Shima et al. 2018). Infection appears to be absent from central arid areas of Australia. Rangeland (feral) goats do not appear to act as intermediate hosts for *E. granulosus* in Australia (Andrewartha 2014). It has been recognised in domestic stock in most other parts of the country. *E. granulosus* is present at very low levels in domestic animals in Tasmania, where it has never been reported in wildlife (Andrewartha 2014).

Dingoes and their hybrids have been recognized as important definitive hosts in maintaining the sylvatic cycle since 1952 (Durie and Riek 1952). Sample sizes of most surveys have been small but prevalence between 60 and 90% has been typical in NSW, south-east Qld and Vic (Coman 1972; Baldock et al. 1985; Jenkins and Morris 2003). Infected foxes have been recorded in NSW, Vic and the ACT (Jenkins and Craig 1992; Reichel et al. 1994; Jenkins and Morris 2003).

**Epidemiology**

The life cycle is indirect. Eggs are shed by the definitive host following a prepatent period of 40-48 days. Dingoes and their hybrids tend to have high worm burdens; over 1,000 is common and over 300,000 have been recorded. This results in heavy environmental contamination. Foxes usually have less than 50 worms and thus play a smaller role in transmission. Parasite survival is thought to require a temperature of less than 30°C and rainfall greater than 25mm for six months per year (Gemmell 1958). In addition to localized faecal contamination of the environment, eggs may be dispersed by wind, rain, herbivores and insects. Eggs of *E. granulosus*, under Australian conditions, have been reported to remain viable for up to one year (Gemmell et al. 1986). However, more recently, eggs extracted from dog faeces and kept outside, directly exposed to the environment (in Patagonia, Argentina) for 41 months were still infective to sheep. The faeces were subjected to temps of 3 - 37°C, desiccating wind and less than 300mm rainfall per year (Thevenet et al. 2005). These data suggest that eggs of in the Australian environment may survive longer has been previously indicated.

Eggs are ingested by intermediate hosts and cysts develop in various organs. Susceptibility of intermediate hosts appears to vary but is poorly understood. For example, cattle infected with the sheep strain produce predominantly sterile cysts, but they are the main intermediate hosts for the cattle strain found in Europe.
Smaller macropodid species appear to be more susceptible, possibly due to behavioural differences affecting exposure to eggs or differences in immune response. Cysts are infective to definitive hosts when protoscoleces are produced by budding from the germinal membrane which lines the hydatid cysts. Following experimental infection of tammar wallabies (*M. eugenii*), fertile cysts were found after eight months, whereas in sheep protoscoleces are only occasionally seen after two years. This finding, coupled with higher burdens of adult worms commonly seen in dingoes compared to domestic dogs suggests that the importance of the sylvatic cycle to the epidemiology of *E. granulosus* in Australia may be greater than previously thought (Barnes et al. 2007a). Following ingestion of viable protoscoleces by definitive hosts, adult worms develop in the small intestine (Thompson and Lymbery 1995).

### Clinical signs

In macropods, hydatid cysts most commonly develop in the lungs. Mortalities of endangered small wallabies have occurred in association with stress of handling/human contact in cases with large or multiple cysts. As a result of compromised pulmonary function, infection may lead to poor condition and increased susceptibility to predation or hunting. Pneumothorax, atelectasis and fatal anaphylaxis have also been recorded (Barnes et al. 2007a). Although only one third of experimentally infected tammar wallabies developed cysts, of these 64% either died or were euthanased as a result of cyst pathology within 14 months of infection. However, as with human cases, smaller cysts and early stage infection are likely to be asymptomatic, particularly in larger macropods.

Definitive hosts have no clinical signs.

### Diagnosis

Diagnosis in macropods is made at post mortem examination. It is possible to detect lung cysts radiographically but this requires general anaesthesia (Barnes et al. 2007a). No serodiagnostic test has proven reliable in non-human intermediate hosts. Attempts to optimize two immunoblot-based serodiagnostic tests, which have high sensitivity and specificity in humans, for use in macropods were unsuccessful (Barnes et al. 2008b). Identification of infection of the definitive host (dog) was previously undertaken by arecoline purging. Coproantigen ELISAs have recently proved more reliable and practical, particularly for use in wildlife (Jenkins et al. 2000).

### Pathology

**Intermediate host**: Cysts are found in various organs. In macropods the lungs are most commonly affected. Cysts may be single or multiple. They are usually soft, fluid filled and fertile containing protoscoleces but may show signs of caseation or calcification if the host mounts a significant immune response. Cyst growth rate in macropods may be much greater than has been recorded in sheep. The features of the adventitial layer differ markedly between the two species, indicating differing immune responses which have yet to be characterized. Histological examination of cysts demonstrates diagnostic laminated layer even in absence of protoscoleces.

**Definitive host**: Worms may carpet the small intestine in heavily infected individuals. Histologically, there is little or no inflammatory response.
Treatment

Treatment of non-human intermediate hosts is not practical. Domestic and farm dogs should be treated with praziquantel orally every 6 weeks in endemic areas, every 12 weeks elsewhere.

Figure 1: LEFT: Multiple lung cysts tammar wallaby (dorsal view, H=heart, arrows show cysts, photo: Lyn Hinds). RIGHT: Section showing laminated layer and other cyst features.

Prevention and control

Infection of dogs can be prevented by prohibiting the feeding of sheep and macropod offal and can be controlled by treating dogs with praziquantel. Regular treatment of domestic and farm dogs is recommended to reduce transmission in domestic stock.

Frequent baiting of Australian wild definitive hosts with baits containing praziquantel is unlikely to be practical because of the vast areas involved. However, such a regime has successfully been trialled in foxes in Germany to control *E. multilocularis*. In defined areas in Australia (e.g. around popular camping areas in national parks), the use of medicated baits may be practical, since dingoes and their hybrids have defined home ranges (Claridge et al. 2009) and the same animals would repeatedly visit the same campsites.

A vaccine, EG95, has been developed for use in sheep (Lightowlers et al. 1999) but is not currently commercially available. It was shown to be effective in tammar wallabies, providing 96-100% protection against an experimental challenge (Barnes et al. 2009).

Human health implications

Hydatid infection causes serious disease in humans. Human cases of hydatid disease are under-reported nationwide. Most cases have been linked to infection though domestic sheep-dog cycle. However, a few cases have been directly attributed to infection through the sylvatic macropod-dingo cycle. Humans become infected by accidental ingestion of the parasite eggs and contact with infected dogs is an important risk factor.
**Research**

**Key research questions:**

1) Is hydatid disease a significant cause of mortality and reduced fitness to endangered macropods?
2) Is it practical to vaccinate translocated or re-introduced small macropods to prevent infection? If so, how often would booster vaccinations need to be given?
3) Does continued high prevalence of *E. granulosus* in Australian wildlife pose a significant public health risk through direct contact with wild canids or their faeces?
4) Are there fundamental differences in macropod immune responses that explain the differences in cyst growth rates and pathology compared to other intermediate hosts?

**Research activities/future directions:**

1) To determine the efficacy of the EG95 hydatid vaccine administered intra-nasally to tammar wallabies when followed by immediate challenge.
2) Identify features of the macropod immune system that may explain the differences in cyst growth and host response compared to other intermediate hosts.
3) To model the transmission dynamics of *E. granulosus* to investigate relative significance of sylvatic and domestic cycles in Australia.

**Conclusions**

*Echinococcus granulosus* is widespread in Australian wildlife where its reproductive potential may be greater than in domestic animals. Infected definitive hosts, dingoes, their hybrids and foxes may pose a public health risk, but even heavy parasites loads are of no significance to the animals’ health. The parasite is pathogenic in macropods. Ongoing research is required to better understand the importance of the parasite in Australian wildlife.

**References and other information**


**Acknowledgements**

We are extremely grateful to Tamsin Barnes and David Jenkins who provided the initial draft of this fact sheet and to those individuals, agencies and organisations that provided comment and external review. Without their ongoing support production of these fact sheets would not be possible.

Updated: December 2018

**To provide feedback on this fact sheet**

We are interested in hearing from anyone with information on this condition in Australian wildlife or feral species, 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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