Hydatid disease *(Echinococcus granulosus)* in Australian Wildlife
FACT SHEET

Introductory Statement

Echinococcus granulosus is widespread in Australian wildlife where its reproductive potential may be greater than in domestic animals. The parasite is zoonotic; humans may develop hydatid cysts following ingestion of eggs. 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, is found at a higher prevalence and is potentially fatal in smaller species including endangered rock-wallabies and the bridled nail-tail wallaby. Further research is required to reach a greater understanding of importance of the parasite in Australian wildlife.

Aetiology

Cestode, Family *Taeniidae*, genus *Echinococcus,* species *granulosus,* strain *G1* (common sheep strain). Originated in Europe. Earlier distinction between domestic and sylvatic strains no longer recognised. Tasmanian sheep strain now extinct.

Natural hosts

Echinococcus granulosus has an indirect lifecycle, causing hydatid disease in its larval form. Intermediate hosts for the common sheep strain in Australia include sheep, macropods, wombats, man, pigs and cattle (dead end hosts). Prevalence is reported to increase with age in domestic species (Baldock *et al.*, 1985). Macropods are considered novel 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. Hydatid cysts are commonly found in liver and lungs of cattle and sheep and eggs laid by adult tapeworms are frequently recovered from the faeces of domestic dogs.

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 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, New South Wales and Queensland and the hills around Perth in Western Australia (Jenkins and

MacPherson, 2003). It has been reported in wombats in Victoria. Prevalences in macropods vary (0-67%); higher prevalences have been recorded in smaller species, notably swamp wallabies and black-striped wallabies (Jenkins and Morris, 2003; Banks, 1984). A recent study in south east Queensland found 15.3% threatened brush-tailed rock-wallabies were infected (Barnes *et al.*, 2008a). Marked clustering of infection has been recorded in eastern grey kangaroos but risk factors have not been identified (Barnes *et al.*, 2007b). Infection appears to be absent from central arid areas. It has been recognised in domestic stock in most other parts of the country, has never been recorded in Northern Territory and is provisionally eradicated from Tasmania.

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 prevalences between 60 and 90% have been typical in New South Wales, south-east Queensland and Victoria (Coman, 1972; Baldock et al, 1985; Jenkins and Morris, 2003). Infected foxes have been recorded in New South Wales, Victoria and the Australian Capital Territory (Reichel et al, 1994; Jenkins and Craig, 1992; Jenkins and Morris, 2003).

Epidemiology

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 (Gemmel, 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 (Gemmel and Lawson, 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, 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; 2009). Following ingestion of viable protoscoleces by definitive hosts adult worms develop in the small intestine (Thompson and Lymbery, 1995).

Clinical signs

Macropods – cysts most commonly develop in 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 – no clinical signs.

Diagnosis

Currently diagnosis in macropods is made at post mortem. It is possible to detect lung cysts radiographically but this requires general anaesthesia (Barnes *et al.*, 2007a). As yet 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 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.
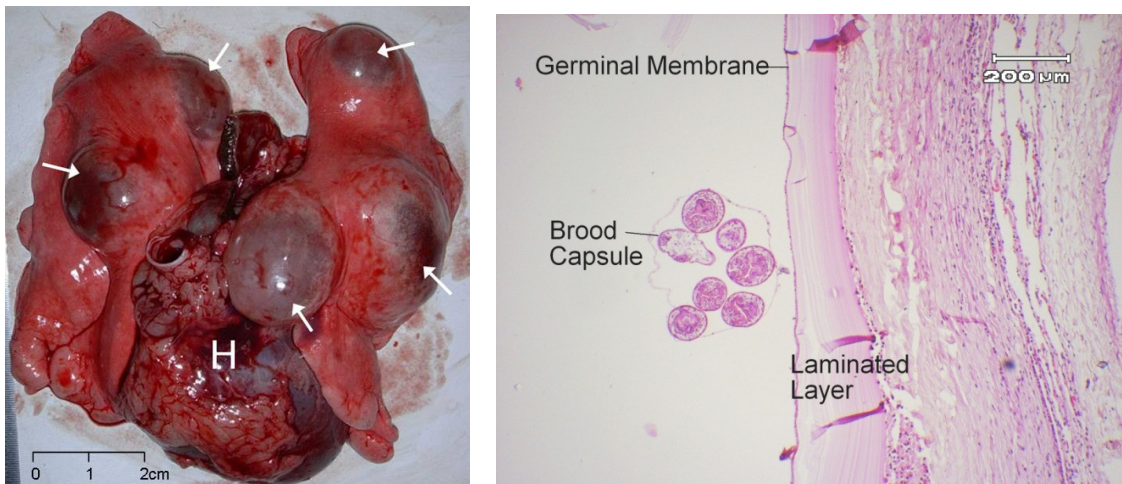
LEFT: Multiple lung cysts tammar wallaby (dorsal view, H=heart, arrows show cysts, photo: Lyn Hinds). RIGHT: Section showing laminated layer and other cyst features.

Treatment

Treatment of non-human intermediate hosts is not practical. Domestic and farm dogs should be treated with praziquantel 5mg/kg orally every 6 weeks in endemic areas, every 12 weeks elsewhere.

Prevention and control

Regular treatment of domestic and farm dogs 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 *Echinococcus 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 visit the same campsites repeatedly

A vaccine, EG95, has been developed for use in sheep (Lightowlers *et al.*, 1999). It has subsequently been shown to be effective in tammar wallabies, providing 96-100% protection against an experimental challenge (Barnes et al., 2009). It is not yet commercially available but could be supplied to those involved in translocation and captive breeding programs if requested from Marshall Lightowlers (contact details below). Users would need to obtain permission to use the vaccine in this way from the Australian Pesticides and Veterinary Medicines Authority.

Human health implications

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 therefore contact with infected dogs is an important risk factor. Infection of dogs can be prevented by prohibiting the feeding of sheep and macropod offal and can be controlled by treating dogs with praziquantel as described above.

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 macropodid 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 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, is found at a higher prevalence and is potentially fatal in smaller species including endangered rock-wallabies and the bridled nail-tail

wallaby. Further research is required to reach a greater understanding of importance of the parasite in Australian wildlife.

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Acknowledgements

To provide feedback on this fact sheet

The Australian Wildlife Health Network would be very grateful for any feedback on this fact sheet. Please provide detailed comments or suggestions to rwoods@zoo.nsw.gov.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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