CYSTIC ECHINOCOCCOSIS IN AUSTRALIA: THE CURRENT SITUATION

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INTRODUCTION

The genus *Echinococcus* consists of a group of cyclophylidean cestodes that utilize domestic and wild canids as their definitive hosts and a range of wildlife and domestic livestock species as their intermediate hosts. Humans may also act as an accidental intermediate host. There are no demonstrable ill effects associated with infection in the definitive host but pathology associated with infection in the intermediate host commonly causes considerable morbidity and occasionally death.

Echinococcus granulosus is the only member of the genus *Echinococcus* present in Australia, having been introduced into the country with domestic livestock and dogs during European colonization. In 2002, the island state of Tasmania announced "provisional eradication" of *E. granulosus* (Middleton, 2002) following about 30 years of intensive hydatid control (Beard *et al*, 2001). This impressive achievement was due, mainly, to the fact that wildlife never became involved in transmission and, as an island, Tasmania had control over animal movements. Following the introduction of *E. granulosus* to the mainland of Australia, the parasite rapidly infiltrated the *E. granulosus*-naïve native wildlife, being perpetuated through a predator/prey interaction between definitive hosts (dingoes) and intermediate hosts, macropodid marsupials (kangaroos and wallabies) (Jenkins and Macpherson, 2003; Jenkins and Morris, 2003). Currently, domestic and wildlife transmission patterns are operating in Australia, with wildlife providing an important reservoir for infection of domestic livestock and humans (Hope *et al*, 1992; Banks, 1994; Grainger and Jenkins, 1996). The geographic distribution of *E. granulosus* in Australia is strongly influenced by rainfall, optimal transmission occurs in regions with ambient temperatures of less than 30^{o}C and at least 25mm rainfall monthly for 6 months of the year (Gemmell, 1959). *Echinococcus granulosus* occurs most commonly along the eastern side of the continent in eastern Victoria, New South Wales and Queensland, in areas associated with the Great Dividing Range, and in the south-western corner of Western Australia (Jenkins and Macpherson, 2003).

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ECHINOCOCCUS GRANULOSUS IN HUMANS AND DOMESTIC ANIMALS

Human hydatid disease is a notifiable disease in all States and Territories in Australia, except New South Wales, the state where most cases arise each year. In 1999, the New South Wales health authorities decided to remove hydatid disease from their list of notifiable diseases. Human hydatidosis has always been notoriously underreported leading to official figures greatly under representing the real situation (Stein and McCully, 1970; Beard, 1979; Schreuder, 1992; Jenkins and Power, 1996). Between 1989 and 1992, Australian Capital Territory and New South Wales Hospitals treated 321 patients for hydatid disease. Trace-backs of these cases revealed that in some communities, the annual index of infection was as high as 23.5 cases/ 100,000 population (Jenkins and Power, 1996). Of the 321 cases (195 were new), only 17 had been reported. In a retrospective study of human cases in Victoria in the 12 months up to July 1992, only 2 of 50 cases had been reported (Taylor, 1993).

The decision by the New South Wales health authorities to stop reporting cases of human hydatidosis has greatly compounded the already difficult task of monitoring occurrence of human infection in Australia. McCullagh (1996) was scathing in his assessment of the situation suggesting that even if notification of all cases of human infection in Australia could be achieved, it is unlikely to alter the, apparently, disinterested official attitude to this disease because "the disease fails to conform to a pattern to which the health care systems are designed to respond". McCullagh (1996) went on to say that for successful

prevention/control of human hydatidosis, the collaboration of various agencies such as agriculture, health, education and conservation is needed to solve this regional health problem but it does not occur. Nevertheless, between 80 and100 new cases of human hydatidosis are diagnosed annually in Australia (Jenkins and Power, 1996) with some patients being young children (Thompson, 2002; Jenkins, unpublished data; Walker, Center for Infectious Diseases and Microbiology, Westmead Hospital, Sydney, personal communication).

Hydatid infection rates in Aboriginal people are largely unknown, but in the few data available from Western Australia, Aboriginal people are over represented, compared to the non-Aboriginal population (Walters *et al* 1996: Stein and McCully, 1970). Between 1985 and 1999, 90 cases of human hydatidosis were treated in Western Australian hospitals. The prevalence of infection in rural Western Australians was higher than in urban dwellers, 0.9/100,000 and 0.6/100,000, respectively. The annual prevalence index in rural Western Australian Aboriginal people was 12.2 times higher than in the equivalent non-Aboriginal rural population (5.5/100,000 compared to 0.45/100,000) (Walters, *et al* 1996). This striking disparity in hydatid infection between Aboriginal and non-Aboriginal people identified by Walters *et al* (1996) is little changed from the situation of 30 years ago. Stein and McCully (1970) reported an annual index of 0.4/100,000 in the rural non-Aboriginal population compared with 6.9/100,000 in the rural Aboriginal population. Jenkins and Power (1996) reported 1.1/100,000 cases of hydatid disease in the New South Wales Aboriginal population but were unable to present annual index data for the rural Aboriginal population of New South Wales for comparison with the non-Aboriginal rural population whose annual index was 2.6/100,000 population.

There are no recently published accounts of *E. granulosus* prevalence in Australian rural farm dogs. The most recent data consist of four surveys undertaken between 1985 and 1992 (summarized in Jenkins, 1996) where a maximum prevalence of just over 10% was reported. These surveys relied on purging with arecoline hydrobromide and examining the purge sample for *E. granulosus* or the detection of serum antibodies in blood. Both methods are unreliable and purging also has some important negative side effects (Lightowlers and Gottstein 1995; Eckert, *et al* 2001). False negative purges commonly occur in dogs with low worm burdens, some dogs do not purge, purging pregnant dogs causes abortion and purging puppies and old dogs may be fatal. The unreliability of antibody detection may involve cross-reactions (false positives) with antibodies generated against other taeniid cestode species, false positives through detection of residual antibody after an infection has been lost and non-detection of infected dogs (false negatives) (Jenkins *et al*, 1990). The presence of *E. granulosus* has also been reported in one out of 15 New South Wales Aboriginal community dogs (post mortem data) (Jenkins and Andrew, 1993) and "evidence of exposure" was reported from Western Australian Aboriginal community dogs (Kimberly area) following a serological survey (Lymbery and Thompson, 1995).

An immunodiagnostic test has been devised for detecting antigens of *E. granulosus* (coproantigens) in dog feces (Allan *et al*, 1992; Deplases *et al*, 1992; Jenkins *et al*, 2000). These antigens have recently been partially characterized and found to consist of a protein core associated with large amounts of carbohydrate (Elayoubi *et al*, 2003). These coproantigens are thought to be glycocalyx-derived. Coproantigens of *E. granulosus* are stable, remaining readily detectable in dog feces left in the environment, in direct sun, for up to one week or frozen for up to one year (Jenkins *et al*, 2000). The coproantigen ELISA has been found to be more reliable in identifying *E. granulosus*-infected dogs than either arecoline purging or serodiagnosis (Craig *et al*, 1995) and the test has been recently used to survey farm dogs in New South Wales and Victoria (Jenkins, unpublished data). In these studies, about 26% of sheep dogs in each survey were found to be *E. granulosus*-coproantigen positive. The data revealed that infection in these groups of dogs was not wide spread, but restricted to a few farms often having numerous infected dogs. The farmers on these farms were feeding offal to their dogs, commonly kangaroo lungs, still contained in carcasses from which the intestines and liver had been removed.

For a historical review of the status of hydatidosis in livestock in Australia, the reader is referred to Schantz *et al* (1995). Little recent published data are available on the prevalence of infection in domestic livestock because infection prevalence in livestock killed in abattoirs is not recorded. Nevertheless, anecdotal data from abattoir quality control inspectors indicate hydatid cysts are still seen regularly in Queensland, New South Wales, and Victoria and periodic reports of hydatid infection in livestock appear in the rural press with anecdotal data provided by farmers and researchers (Patterson, 2002; Thompson, 2003).

A recent report from a survey of 4,348 cattle slaughtered in the Northern Territory, identified hydatid

infection in 26 animals none of which were bred in the Northern Territory (Small and Pinch, 2003). From the condition and size of the cysts in these cattle, the lack of cysts in 790 feral pigs examined from the same region and the fact that the infected cattle had been imported from south-eastern Queensland (a region where hydatid disease is common in cattle (Banks, 1994); Small and Pinch (2003) concluded that *E. granulosus* is not endemic in cattle in the Northern Territory. They suggest the reasons for this are the hot dry climate in the Northern Territory and an absence of suitable wildlife intermediate hosts. In a region adjacent to the Northern Territory, Lymbery *et al* (1995) reported a high prevalence of hydatid disease in cattle in northern Western Australia (Kimberley region) (78% of 236 animals inspected) and lower prevalence, 7-25% in the south eastern part of the state. The source of the infected Kimberley cattle remains controversial. Lymbery *et al* (1995) maintain natural transmission is occurring in the Kimberly whilst Small and Pinch (2003) suggest this may not be the case, maintaining, hydatid disease in cattle in the Kimberley only occurs in cattle imported from Queensland.

Little recent data are available regarding the economic losses to the Australian meat industry, through the downgrading or condemnation of offal due to hydatid disease but losses are occurring and available evidence suggests they could be considerable. It has been observed that in some lines of older Queensland cattle, up to 50% or more of livers have been condemned due to the presence of hydatid cysts (Lee Taylor, Queensland Department of Primary Industries and Mines, personal communication). A liver is worth about $7.20 and if 5% of livers are condemned annually for hydatid infection (a conservative estimate) with approximately 7.5 million cattle killed each year in Queensland abattoirs, that represents an annual loss of $2.7 million to the Queensland meat industry (Thompson, 2003; Lee Taylor, personal communication).

WILDLIFE RESERVOIRS

Dingoes did not evolve in Australia, they are not marsupials, but placentals, introduced into Australia about 5,000 years ago from south eastern Asia (Breckwoldt, 1988; Corbett, 1995). Dingoes out competed and replaced the thylacine, the indigenous marsupial, top order predator. Following the arrival of domestic dogs into Australia, with European colonists, cross-breeding with dingoes began, and today in many parts of Australia, particularly in the south-east, domestic dog genes can be detected in 75-80% of dingoes (Corbett, 1995; Nesbitt *et al*, 2000; Wilton, 2001).

Wild dogs (dingoes and dingo/domestic dog hybrids) are numerous in the bush in Australia and play an important role in the transmission of *E. granulosus* (Schantz *et al*, 1995) through a predator/prey interaction with a range of potential intermediate hosts, both native and introduced (Jenkins and Macpherson 2003; Jenkins and Morris, 2003). Wild dogs are highly susceptible to infection with *E. granulosus* (Jenkins and Morris, 2003) with infections occasionally in excess of 300,000 worms (Jenkins and Morris, 1991) and in south-eastern, Australia the prevalence of infection in wild dog populations may be 100% (Jenkins and Morris, 2003; Reichel *et al*, 1994). Not all worms are at the same stage of development in these heavily infected wild dogs (Jenkins and Morris, 1991) ensuring a fairly constant release of large numbers of eggs into the environment. Compared to domestic dogs or foxes, there is no doubt that wild dogs in Australia currently represent the most important definitive host in terms of prevalence of infection, the large areas inhabited by infected wild dogs and the biomass of parasite they carry. Wild dogs provide an important source of hydatidosis for wildlife, domestic livestock and under certain circumstances, humans (Hope *et al*, 1992; Taylor, 1993; Grainger and Jenkins, 1996; Jenkins and Morris, 2003).

A worrying new development in wild dog behavior in Queensland has seen wild dogs entering recently constructed outer suburbs in some major urban centers. These suburbs have been constructed in established wild dog home ranges and once the disturbance of construction has subsided the wild dogs are reoccupying their home ranges and in some cases hunting domestic pets. Brown and Copeman (2003) examined the intestines of 20 adult and 7 wild dog pups trapped in areas fringing the outer suburbs of Townsville. Six of the adult wild dogs were infected with *E. granulosus* with worm burdens of less than 1,000 worms. In a second study, three of four wild dogs from areas fringing outer suburbs of the Gold Coast were found infected, with worm burdens ranging between 660-1,600 *E. granulosus*. Many towns in Queensland are expanding their borders into wild dog home range and encroachment of *E. granulosus*-infected wild dogs into these new urban developments presents a potentially important new public health issue.

Foxes were introduced into Australia in the 1860s and are now found living in all rural areas of the continent, except the tropical north, and also in many

urban centers. Foxes infected with *E.granulosus* have been found in a number of locations in south-eastern Australia (Gemmell, 1959; Thompson *et al*, 1985; Obendorf *et al*, 1989; Jenkins and Craig, 1992; Reichel *et al*, 1994; Grainger and Jenkins, 1996; Jenkins and Morris, 2003). Few individuals in fox populations are usually infected with *E. granulosus* and worm burdens are commonly less than 50 worms, although there are some exceptions (Reichel *et al*, 1994; Jenkins and Morris, 2003). For these reasons, foxes are of minimal importance in the transmission of eggs of *E. granulosus* in the bush. However, *E. granulosus*-infected foxes infiltrating urban areas from the bush are of public health importance (Jenkins and Craig, 1992).

Spotted-tailed quolls (*Dasyrurus maculatus*) are the largest native carnivore in Australia and include swamp wallabies (*Wallabia bicolour*), a highly susceptible intermadiate host for *E. granulosus*, in their diet. A recent survey of *E. granulosus* coproantigens in wild, spotted-tailed quoll feces suggest these animals are not involved in the transmission of *E. granulosus* (Jenkins, unpublished data).

Macropod marsupials are the major wildlife intermediate hosts for *E.granulosus*. In eastern Australia these consist of eastern grey kangaroos (*Macropus gigantea*), red-necked wallabies (*Macropus rufogriseus*) and swamp wallabies (Jenkins and Morris, 2003) and in south-western, Western Australia, western grey kangaroos (*Macropus fuliginosus*) are the main intermediate host (Thompson *et al*, 1988). In Queensland several other species of macropodid may be locally important intermediate hosts (Jenkins and Macpherson, 2003). In eastern Australia swamp wallabies are pivotal in wild life transmission of *E. granulosus* because they have been recorded with a high prevalence of fertile hydatid cysts (65.5%) (Jenkins and Morris, 2003) and they are a favored dietary item of wild dogs (Coman, 1972; Newsome *et al*, 1983; Robertshaw and Harden, 1985, 1986). Wombats (*Wombatus ursinus*) may also act as intermediate hosts (Grainger and Jenkins, 1996), and like swamp wallabies, are also a popular dietary food item for wild dogs. The site of predilection for hydatid cysts in macropodids and wombats is the lungs and it is thought this leads to compromised lung function rendering infected animals more susceptible to predation by wild dogs, thus facilitating transmission of the parasite.

Australian feral pigs arose from escapee domestic pigs bred by early settlers. They have reverted back to look similar to wild boar with coarse black and/or bown hair with some individuals exhibiting white patches. Feral pigs act as intermediate hosts for *E. granulosus* in both eastern and western Australia (Thompson *et al*, 1988; Lidetu, 1992; Banks, 1994; Jenkins and Morris, 2003). Prevalence levels in some populations may be close to 50% but only a small proportion of the cysts are fertile (Jenkins and Morris, 2003). Low cyst fertility in pigs, combined with the fact that wild dogs find larger adult pigs difficult to subdue, preferring to prey on small piglets that are likely to be uninfected, makes pigs an unimportant intermediate hosts for transmission of *E. granulosus* in wildlife. In some areas of North Queensland, pig cyst fertility was reported up to 70.1% (Lidetu, 1992), in these areas pigs may be contributing more to the transmission cycle than is currently recognized.

CONCLUSIONS

Echinococcus granulosus occurs widely in Australia, with high prevalence in wildlife, hydatid cysts occurring commonly in domestic livestock, echinococcosis occurring in rural dogs and almost 100 new cases of human hydatidosis diagnosed annually [(some in children as young as 26 months old (Jenkins unpublished data)]. Despite this situation, there is little official interest in this important zoonosis. Human cases are notoriously under reported, hydatid disease is not monitored in abattoirs and prevalence surveys of infection in rural domestic dogs and wildlife are rarely undertaken. Schreuder (1992) suggested that the reason hydatid disease was not perceived as public health or economic problem in Australia is because of a lack of detailed information.

An improved reporting system should be instigated for human infection and a national awareness campaign aimed particularly at rural children should be incorporated into the school curriculum. More attention needs to be paid to infection in livestock to determine accurately the degree of economic losses to the Australian meat industry. The wildlife reservoir provides a constant potential source of infection for humans and domestic animals. Increasing numbers of people are visiting Australian national/state parks, especially those in eastern Australia frequented by populations of *E. granulosus*-infected wild canids and *E. granulosus*-infected wild dogs are also infiltrating new areas of urban developments in Queensland. These scenarios present new potential public health risks that need to be addressed by the Australian state and federal health authorities. The transmission of hydatid disease from wild dogs to livestock is most common on farms at the interface of Crown Land (national/state parks and forests), this transmission could be effectively

controlled by implementing a livestock vaccination program using the new, highly effective, recombinant vaccine against hydatid disease (Lightowlers *et al*, 2003).

The current level of public awareness of hydatid disease in Australia and an appreciation of the financial losses to the Australian meat industry due to hydatid disease are poor and urgently need to be improved.

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Echinococcosis is a parasitic disease of tapeworms of the Echinococcus type. The two main types of the disease are cystic echinococcosis and alveolar echinococcosis. Less common forms include polycystic echinococcosis and unicystic echinococcosis. The disease often starts without symptoms and this may last for years. The symptoms and signs that occur depend on the cyst's location and size. Alveolar disease usually begins in the liver, but can spread to other parts of the body, such as the lungs or