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Invited Review

**Echinococcus** as a model system: biology and epidemiology

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**A B S T R A C T**

The introduction of *Echinococcus* to Australia over 200 years ago and its establishment in sheep rearing areas of the country inflicted a serious medical and economic burden on the country. This resulted in an investment in both basic and applied research aimed at learning more about the biology and life cycle of *Echinococcus*. This research served to illustrate the uniqueness of the parasite in terms of developmental biology and ecology, and the value of *Echinococcus* as a model system in a broad range of research, from fundamental biology to theoretical control systems. These studies formed the foundation for an international, diverse and ongoing research effort on the hydatid organisms encompassing stem cell biology, gene regulation, strain variation, wildlife diseases and models of transmission dynamics. We describe the development, nature and diversity of this research, and how it was initiated in Australia but subsequently has stimulated much international and collaborative research on *Echinococcus*.

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**1. Introduction**

*Echinococcus* remains a major cause of zoonotic diseases of public health and economic significance (Jenkins et al., 2005a; Budke et al., 2006; Davidson et al., 2012; Hegglin and Deplazes, 2013). Despite advances in control strategies, clinical management and vaccine development, the parasite continues to thrive in countries throughout the world.

Hydatid disease, cystic and alveolar, is a typical cestodean that can be perpetuated in nature in wild animal cycles without impacting on public health but with human interference (Thompson, 2013), directly or accidentally, with spillover to domestic cycles can lead to severe clinical disease and death. It is also an important cause of economic losses to livestock industries, particularly *Echinococcus granulosus* in sheep and cattle (Table 1).

It is now well known that *Echinococcus* has a two-host life cycle with a sexual stage in the intestine of a carnivore definitive host and a unique, cystic larval stage in the tissues of non-carnivorous mammals and omnivores (Thompson, 1995). It is interesting that much of the research that revealed the unique features of the parasite’s way of life were undertaken in Australia. This is not a coincidence and relates to the impact *Echinococcus* had on a developing agricultural society following early European settlement of a continent with huge temperate areas perfect to exploit for raising livestock.

It was the upsurge in sheep farming at the end of the 19th century, with expanding exports to Europe, that contributed to the serious and largely undocumented human health problem that existed during the late 19th and early 20th centuries (Gemmell, 1990). During the first half of the 20th century, there was a high incidence of cystic hydatid disease in Australia, and to a lesser extent alveolar hydatid disease, either contracted in Australia (*E. granulosus*) or in migrants from endemic areas overseas (*E. granulosus* and *Echinococcus multilocularis*) (Dew, 1935). There was thus a need for regular surgical intervention. It was surgeons such as Harold Dew who had an interest in the basic biology of the parasite and who published in international journals such as the "British Journal of Surgery" and "British Medical Journal" that led to widespread recognition of the research being undertaken on hydatid disease in Australia. This was enhanced by Dew's contributions in the literature (Dew, 1953) and at conferences to the debate raging at the time on whether cystic and alveolar hydatid disease were caused by the same or different species of *Echinococcus* ("dualists" versus "monists") as well as his collaboration with researchers in Europe such as Félix Dévé.

Dew recognised the developmental differences of the two stages in the life cycle and studied the metacestode stage of the cystic and alveolar forms (Fig. 1) in depth, both in human cases...
and animals (Dew, 1922, 1925, 1928, 1935). He thus complemented much of Dévé's research undertaken in rodents (Dévé, 1919, 1946) and built on this. Dew demonstrated that an intact laminated layer was a fundamental and unique component of the 'healthy' hydatid cyst and considered this layer to be of host origin, and demonstrated that elements of the cyst wall (germinal layer) could regenerate (Dew, 1935). He realised that the cyst enclosed a sterile environment and was under intracystic pressure, and speculated on the reasons for this being indicative of viability and a function of the germinal layer. In observations of what we now know to be the metacestode of *E. multilocularis*, Dew described naked prolongations of the nucleated germinal membrane (layer) in direct contact with host tissues (Dew, 1935), a fundamental feature of the infiltrating metastatic metacestode of *E. multilocularis* (Fig. 1) subsequently described using electron microscopy 60 years later (Mehlhorn et al., 1983; and see Section 2.4). Thus it was Harold Dew that led to Australia being referred to as the 'home of hydatids' in terms of research. He paved the way for other researchers and did much to establish *Echinococcus* as a model organism. "In this country of Australia we all have unrivalled opportunities to investigate this disease, both in man and in animals, and it is our duty to contribute our share to future advances in its study" (Dew, 1935).

The economic impact and public health significance of cystic hydatid disease in Australia worsened over the next 30 years, which provided an opportunity for obtaining research funds from organisations supporting health and livestock research. J.D. Smyth obtained funds from these sources and took a physiological approach in his research aimed at increasing understanding of the developmental biology of *Echinococcus*. Having forged an interest in developing in vitro cultivation procedures for *Schistocephalus* with success in establishing much of the life cycle of this cestode in culture (Smyth, 1946, 1950), he turned his attention to *Echinococcus* (see Smyth, 1990) for which the ability to study and maintain the parasite in vitro would provide a great research tool for investigating methods of control.

The practical and ethical difficulties of undertaking in vivo studies in the canid or felid definitive hosts of *Echinococcus* further reinforced the need to develop in vitro systems. It was these pioneering studies of Smyth (Smyth et al., 1966 and reviewed in Smyth and Davies, 1974a; Howell and Smyth, 1995) that demonstrated the potential of *Echinococcus* as a model for studying principles of developmental biology, differentiation, host–parasite relationships and evolutionary biology (Smyth, 1969; Smyth et al., 1966), and which have influenced research and theoretical understanding far beyond parasitology (Thompson and Lymbery, 2013). Smyth saw the potential of exploiting *Echinococcus* as a novel model system for studying parasitism as distinct from a model for studies on evaluating anthelmintic or other anti-parasitic drugs (Smyth, 1969). He thus expanded the definition of a 'model' to embrace

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Table 1
Current taxonomy of *Echinococcus* spp.

<table>
<thead>
<tr>
<th>Species</th>
<th>Strain/genotype</th>
<th>Known intermediate hosts</th>
<th>Known definitive hosts</th>
<th>Infectivity to humans</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Echinococcus granulosus</em></td>
<td>Sheep/G1</td>
<td>Sheep (cattle, pigs, camels, goats, macropods)</td>
<td>Dog, fox, dingo, jackal and hyena</td>
<td>Yes</td>
</tr>
<tr>
<td></td>
<td>Tasmanian sheep/G2</td>
<td>Sheep</td>
<td>Dog</td>
<td>Yes</td>
</tr>
<tr>
<td></td>
<td>Buffalo/G3</td>
<td>Buffalo</td>
<td>Dog</td>
<td>Yes</td>
</tr>
<tr>
<td><em>Echinococcus equinus</em></td>
<td>Horse/G4</td>
<td>Horses and other equines</td>
<td>Dog</td>
<td>Probably not</td>
</tr>
<tr>
<td><em>Echinococcus ortleppi</em></td>
<td>Cattle/G5</td>
<td>Cattle</td>
<td>Wolves, dog</td>
<td>Yes</td>
</tr>
<tr>
<td><em>Echinococcus canadensis</em></td>
<td>Cervids/G8,G10</td>
<td>Cervids</td>
<td>Dog</td>
<td>Yes</td>
</tr>
<tr>
<td><em>Echinococcus intermedius</em></td>
<td>Camel/Pig/G6/G7</td>
<td>Camels, pigs, sheep Warthog (possibly zebra, wildebeest, bushpig, buffalo, various antelope, giraffe, hippopotamus)</td>
<td>Lion</td>
<td>Uncertain</td>
</tr>
<tr>
<td><em>Echinococcus feldisi</em></td>
<td>Lion/–</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Echinococcus multilocularis* variation

<table>
<thead>
<tr>
<th>Species</th>
<th>Strain/genotype</th>
<th>Known intermediate hosts</th>
<th>Known definitive hosts</th>
<th>Infectivity to humans</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Echinococcus shiquicus</em></td>
<td>–/–</td>
<td>Pika</td>
<td>Fox, dog, cat, wolf, racoon-dog, coyote</td>
<td>Yes</td>
</tr>
<tr>
<td><em>Echinococcus vogeli</em></td>
<td>None reported</td>
<td>Rodents</td>
<td>Tibetan fox</td>
<td>Uncertain</td>
</tr>
<tr>
<td><em>Echinococcus oligarthrus</em></td>
<td>None reported</td>
<td>Rodents</td>
<td>Bush dog</td>
<td>Yes</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Wild felids</td>
<td>Yes</td>
</tr>
</tbody>
</table>

studies on all the biological activities that supported the parasite life style of *Echinococcus*, as well as the potential of this model system for broader studies of a more fundamental nature in biology, for example stem cells (Smyth, 1969, 1987). As such the promotion of such a eukaryotic, metazoan model was at the time somewhat of a ‘first’.

Smyth worked closely with his Australasian colleague Michael Gemmell, who also had an interest in *Echinococcus* but who was keen to understand the epidemiology of hydatid disease, its economic impacts and impediments to control (Gemmell, 1990). As such, he was the first to apply mathematical models to *Echinococcus* and the concept of the basic reproductive rate (R0) in order to define the conditions under which transmission of *Echinococcus* and other taenids occur, and the regulatory role of immunity (Gemmell, 1990; Gemmell and Roberts, 1995).

In this review, we describe the research, particularly that undertaken in Australia, which has demonstrated the value of *Echinococcus* as a model system influencing advances in biology and epidemiology of parasites and parasitic infections.

### 2. Developmental biology

#### 2.1. Growth and maturation

*Echinococcus* has two developmental stages in its life cycle; the adult tapeworm, which is the sexual stage, and the cystic or infective larval metacestode that reproduces asexually (Fig. 1). The metacestode of *E. granulosus*, which was easily accessible from infected livestock in the abattoir or from human surgical cases, was the logical starting point for experimental manipulations. Initially, these were undertaken in rodents where the asexual proliferative nature of the parasite was first observed, a phenomenon that was subsequently exploited with the development of rodent models of secondary hydatidosis (Dévé, 1919). These provided a means to maintain the parasite virtually indefinitely in vivo by serial passage (e.g., Thompson, 1976; Whitfield and Evans, 1983; Kamiya et al., 1985).

Although a number of workers had reported vesicular/cystic development and proliferation of protoscoleces in vitro, a major goal was to provide appropriate conditions to stimulate and support development in an adult (strobilate) direction. It was not until Smyth and his colleagues refined procedures with the aim to reproduce the conditions that protoscoleces would be exposed to in the gut of the definitive host that strobilate development was obtained (Smyth et al., 1966). Smyth took a physiological approach to this research, with the cyst as a starting point. Its sterile environment provided the perfect ‘entry point’ if accessed aseptically to carefully remove essentially ‘dormant’ invaginated protoscoleces and then expose them to appropriate physiochemical conditions (Smyth and Davies, 1974a). Of particular significance was the provision, in addition to the liquid medium, of a solid proteinaceous base in the culture flask (diphasic medium) which was found to be important in stimulating proglottisation and segmentation in *E. granulosus* with maturation to the pre-fertilisation stage (reviewed in Smyth and Davies, 1974a; Howell and Smyth, 1995). Similar results were obtained with *E. multilocularis* but proglottisation without segmentation will take place without a solid proteinaceous base (Smyth and Davies, 1975; Smyth, 1979; Thompson et al., 1990). This and other developmental abnormalities observed in *E. multilocularis* were considered to reflect a difference in gene expression between the two species (Howell and Smyth, 1995 and see Section 2.3). Although the conditions developed supported reproducible strobilate development, fertilisation and egg production eluded Smyth and other workers. However, they served to emphasise the complexity of the process and to support observations made from in vivo and population genetic studies of the importance of the host intestinal ecosystem in providing the correct physical and physiochemical conditions necessary to support fertilisation (Smyth, 1982; Smyth and Davies, 1974a; Lymbery and Thompson, 1988, 2012; Lymbery et al., 1989; Howell and Smyth, 1995).

The success with cultivating *Echinococcus* and the elucidation of conditions that would support maturation stimulated studies on other taenids including *Taenia pisiformis* and *Taenia crassiceps* (Esch and Smyth, 1976; Osuna-Carrillo and Mascaró-Lazcano, 1982), as well as *Mesococoides corti* (Barrett et al., 1982; Thompson et al., 1982). These largely supported the observations made with *Echinococcus* but also served to illustrate the uniqueness of the *Echinococcus* model system.

During the 25 years Smyth and his colleagues spent studying and optimising strobilate development and maturation of *Echinococcus* in vitro, many valuable observations were made that have served to establish *Echinococcus* as a model system not only in terms of developmental biology, but also for cytodifferentiation and the host–parasite interface.

#### 2.2. Developmental plasticity

Studies on the in vitro cultivation of *Echinococcus* from the larval protoscoleces to the adult worm demonstrated the inherent plasticity of the parasite – a phenomenon eloquently described as ‘heterogeneous morphogenesis’ (Smyth et al., 1967; Smyth, 1987), unique in most metazoan organisms apart from coelenterates such as Hydra, and making *Echinococcus* an unusual model for differentiation studies. Depending upon the culture conditions, protoscoleces can either develop in a cystic or adult direction. Cystic development itself may take a variety of paths, again influenced by environmental conditions. These alternative developmental pathways have been described in detail by Howell and Smyth (1995). Protoscoleces can also develop into an adult tapeworm under different conditions to those supporting cystic development, most importantly the need for a diphasic medium incorporating a solid supporting substrate thought to provide nutrients and/or a contact stimulus (see Section 2.1). However, observations on the development of adult *E. granulosus* and *E. multilocularis* demonstrated that in addition to ‘normal’ worms, cultures often contained worms that had matured but not segmented (monozoic) and worms with more than one scolex and other malformations (reviewed in Howell and Smyth, 1995). Interestingly, adult worms that have developed from protoscoleces can ‘de-differentiate’ in a cystic direction under adverse conditions (see Sections 2.3 and 2.4; Smyth, 1969; Thompson and Lymbery, 1990; Thompson et al., 1990). These observations raised two important and fundamental questions: how is this complex and plastic development controlled and what cells have the potential to develop in such a myriad of different routes?

#### 2.3. Developmental control

Smyth developed a hypothetical yet logical model to explain how genes regulate differentiation and developmental shifts in *Echinococcus* (Smyth, 1969). This was based on the Jacob–Monod model of genetic expression and showed the complex interactions and regulatory switches that may be involved in controlling development and differentiation in *Echinococcus*. Since then a number of studies have isolated and characterised regulatory genes from *Echinococcus* (reviewed in Thompson, 1995 and Parkinson et al., 2012) but how they fit together in a control network has yet to be determined. Recently, Parkinson et al. (2012) identified long non-coding (nc)RNAs that may be involved in the regulation of gene expression in response to environmental cues in the host. They also identified...
a number of genes reflecting specificities of particular stages, including those whose expression is up-regulated by pepsin-acid activation. It is to be hoped that Smyth’s models will help to provide functional differences to the genomic data that is now available for both *E. granulosus* and *E. multilocularis* (Tsi et al., 2013; Zheng et al., 2013). However, differences in development between *E. granulosus* and *E. multilocularis* highlighted by studies in vitro are considered to reflect differences in the control of gene expression (Howell and Smyth, 1995).

### 2.4. Cytodifferentiation

Slais (1973) demonstrated that the post-oncospheral development of *Echinococcus* was initiated by the growth and division of primary germinal cells, and Swiderski (1983) described five pairs of these cells in the posterior pole of the oncosphere. Observations on the development of *Echinococcus* in vitro demonstrated that the fundamental processes of germinal and somatic differentiation, comprising cystic development, proglottisation, maturation, growth and segmentation (strobilisation), can take place independently. This not only illustrated the complexity of cytodifferentiation but also the possible existence of several primitive cell lines. However, to date all the evidence suggests that only one primitive morphological cell type exists as a pool of uncommitted, undiffer- entiated multipotent germinal, or stem, cells in both the adult and metacestode (Smyth, 1969; Gustafsson, 1976; Thompson et al., 1990; Thompson, 1995; Koziol et al., 2014). The undifferentiated germinal cells are a component of the syncytial germinal layer of the metacestode and neck region of the adult cestode. Ultrastructural studies reveal unremarkable rounded cells of variable size of approximately ? µm diameter (Gustafsson, 1976; Mehlhorn et al., 1983; Albani et al., 2010). Cell proliferation derives from the continuous replicative activity of these dividing stem cells located in the germinal layer or neck region of the adult tapeworm (Gustafsson, 1976; Galindo et al., 2003). They have considerable proliferative potential (Eckert et al., 1983; Mehlhorn et al., 1983; Galindo et al., 2003; Martinez et al., 2005), and are the only proliferating cells in *Echinococcus* (Koziol et al., 2014). This is particularly well illustrated by the capacity of the parasite for indefinite perpetuation in the larval stage by passage of protoscoleces or germinal layer material in rodents (secondary hydatidosis; Howell and Smyth, 1995). In alveolar hydatid disease caused by the metacestode of *E. multilocularis*, the proliferating larval parasite has an infiltrative capacity to establish distant foci of infection due to the distribution via blood or lymph of detached germinal cells (Ali-Khan et al., 1983; Eckert et al., 1983; Mehlhorn et al., 1983; Fig. 1).

Problems with host cell contamination dogged early attempts to establish germinal cell lines of *E. granulosus* and *E. multilocularis* (reviewed in Howell and Smyth, 1995). In addition, their isolation from the germinal layer, and their in vitro propagation, could have been hampered by the fact that the germinal layer is a syncytium. However, the establishment and long-term perpetuation of *Echin- cococcus* germinal cells has now been achieved for both species (Yamashita et al., 1997; Spiolitosis and Brehm, 2009; Spiolitosis et al., 2008; Albani et al., 2010). The germinal cells behave similarly to classical stem cells with the formation of cell aggregates and clusters with cavity formation, and there is cytological evidence of transformation (Spiolitosis et al., 2008; Albani et al., 2013). Galindo et al. (2003) found evidence of the regionalisation of DNA and protein synthesis in developing stages of *Echinococcus*, although no morphological evidence has been found of different primitive or germinal cell lines to explain the concept of heterogeneous morphogenesis (Smyth, 1965). However, such heterogeneity has now been confirmed at the molecular level. Germinal cells are in fact heterogeneous, with the existence of subpopulations with different gene expression patterns (Koziol et al., 2014), thus confirming Smyth’s predictions.

### 2.5. Host–parasite interface

#### 2.5.1. Definitive host

Observations that contact of the scolex with a proteinaceous base stimulated strobilate development in *E. granulosus* led to studies on the nature of the interface both in vitro and in vivo, and the discovery of the rostellar gland which comprises a modified group of tegumental cells situated in the apical rostellum (Smyth, 1964; Smyth et al., 1969; Thompson et al., 1979; Thompson and Eckert, 1983; Fig. 2). The rostellar gland releases secretory material by a holocrine process into the interface between parasite and host (Thompson et al., 1979). The origin and site of synthesis of the secretion has still to be determined, although large amounts occur in both the perinuclear and distal cytoplasm of the tegument as well as in the tegumental nuclei (Thompson et al., 1979; Herbaut et al., 1988). The secretion is proteinaceous, containing cystine and lipid. It is not known if there are one or more proteins secreted but Siles-Luces et al. (2000) demonstrated that the secretion contains a regulatory protein (14-3-3) that is released into the host–parasite interface.

The rostellar gland of *Echinococcus* is seemingly unique to *Echi- nococcus*. Although rostellar secretions have been described in larval *T. crassiceps* (Krasnoschekov and Pluzhmnikov, 1981), no gland has been described. Rostellar glands have been described in other adult cestodes, particularly proteocephalids, but their function is also unclear and structurally they are different to the rostellar gland in *Echinococcus* (McCullough and Fairweather, 1989; Zd-?rská and Nebesárová, 2003). There is clearly a need for more studies on the interface of attached adult cyclophyllidean cestodes (Pospekova and Bondarenko, 2014).

This intimate association of the rostellar gland and its secretions suggests a role(s) that enhances the host–parasite relationship in favour of the parasite, which may be regulatory, nutritional and/or protective. The relationship between rostellar gland activity and localised humoral and cellular reactions (Deplazes et al., 1993) is not known but such localised reactions demonstrate stimulation of host immune effector mechanisms. The secretory molecules would seem to be obvious candidates for exploitation in vaccine studies since a focus on prophylaxis of the definitive host may be more attractive than the intermediate host, particularly for the control of *E. multilocularis*, from both commercial and practical perspectives. As pointed out by Heath (1995) the scolex is in intimate contact with the systemic circulation even in the Peyers patches and would appear to maintain its privileged integrity by suppression of cytotoxic and effector cell activity in the region of the scolex.

The few studies on definitive host vaccines do not seem to have targeted rostellar gland secretions and have produced results which are controversial and open to contrasting interpretation (Zhang et al., 2006; Petavy et al., 2008; Torgerson, 2009; Kouguichi et al., 2013). The studies by Petavy et al. (2008) demonstrated a strong inflammatory response in the intestine of vaccinated compared with infected controls but did not show if this was localised to where worms were situated. More recently, Kouguichi et al. (2013) used a surface glycoprotein from *E. multilocul- laris* as a vaccine in dogs which induced significant protection.
2.5.2. Intermediate host

A unique interface is also found in the intermediate host where the laminated layer presents a physiochemical barrier with apparent multifunctionality and a structure whose biosynthesis has become a model system for carbohydrate chemistry (Diaz et al., 2011a,b; Parkinson et al., 2012). The laminated layer is a specialised extracellular matrix unique to Echinococcus (Fig. 1), whose synthesis is a major metabolic activity of the much thinner germinal layer (Diaz et al., 2011a; Lin et al., 2012; Parkinson et al., 2012). The origin of the laminated layer was controversial for some time, i.e., whether it is entirely of parasite origin or if there is a host contribution. Holcman and Heath (1997) demonstrated that it is entirely of parasite (germinal layer) origin by studying the early stages of cyst development from oncospheres in vitro.

Considerable metabolic activity in the germinal layer is required to synthesise and maintain the interfacial barrier of the laminated layer (Parkinson et al., 2012). The role of the laminated layer would appear to be one of protection since cyst survival is dependent upon its integrity (Gottstein et al., 2002). Whether this is purely physical or if there is selective permeability is not known. Montero et al. (2010) identified several molecules in hydatid cyst fluid that could play a role in host evasion. Smyth commented on the significance of the presence of a human blood group P-like substance in the laminated layer of Echinococcus and its significance as a model system in better understanding the immunological basis of the host-parasite relationship. This P1 blood-antigen motif has since attracted much attention and has been further characterised as a protein-carbohydrate, trisaccharide/mucin complex containing galactosamine, yet no biological function has been described to date (Lin et al., 2012). There is also increasing evidence of the immune-regulatory role of the laminated layer, probably in association with the immuno-modulatory activities of the glycoproteins known as Antigen 5 and Antigen B (Diaz et al., 2011b).

2.6. Taxonomy and speciation

One of the most important observations made as a result of studies on the in vitro cultivation of Echinococcus was the failure of protoscoleces collected from hydatid cysts in horses to develop in the same way as those of sheep origin. Protoscoleces from horses evaginated and increased in length but failed to undergo proglottisation or segmentation, even though they were grown in exactly the same diphasic medium (Smyth and Davies, 1974b). This fairly simple observation resulted in radical shifts in our understanding of the epidemiology of hydatid disease and transmission of the aetiological agents as well as their taxonomy and phylogenetic relationships. The results demonstrated that there were fundamental physiological differences between E. granulosus of sheep and horse origin and the coining of the term 'physiological strain differences' (Smyth and Davies, 1974b; Smyth, 1982). This had a broad influence beyond Echinococcus, and in particular the importance of combining phenotypic and genetic differences in the characterisation and description of parasites at the infraspecific level (Thompson and Lymbery, 1990, 1996; Lymbery and Thompson, 2012).

In terms of Echinococcus, the observation of physiological differences between the two parasites of sheep and horse origin complemented earlier epidemiological and taxonomic studies on Echinococcus of horse origin (Williams and Sweatman, 1963). These demonstrated morphological differences between the two forms which were considered to be taxonomically significant, and to reflect differences in host specificity and the life cycles which maintained the two parasites. This was subsequently shown to be correct with the sympatric occurrence of distinct sheep- and horse-dog cycles in several European countries (Thompson and Smyth, 1975; Thompson, 2001; Gonzalez et al., 2002). In addition,
epidemiological evidence has not only demonstrated distinct differences in intermediate host specificity but also that, unlike the sheep strain (E. granulosus), the horse strain (Echinococcus equinus) (Table 1) does not appear to be infective to humans (Thompson and Lymbery, 1988, 1991).

The concept of host-adapted strains of E. granulosus led to studies on other forms of the parasite in other species of intermediate hosts such as cattle, pigs, camels and cervids. These studies not only confirmed the existence of a number of host-adapted life cycles in different parts of the world but also provided additional data on developmental differences between strains which may impact on control (reviewed in Thompson and Lymbery, 1988; Thompson et al., 1995; Thompson, 2008).

Subsequent molecular characterisation of host-adapted strains of Echinococcus, coupled with molecular epidemiological studies in endemic areas, has confirmed their genetic and morphological distinctness and revealed phylogenetic relationships which support a robust, meaningful taxonomy based on a previously suggested nomenclature (Table 1; Bowles et al., 1994; Thompson et al., 1995, 2006; Cruz-Reyes et al., 2007; Harandi et al., 2002; Thompson and McManus, 2002; Lavikainen et al., 2003; Jenkins et al., 2005a; Romig et al., 2006; Moks et al., 2008; Thompson, 2008; Huttner et al., 2009; Saarma et al., 2009; Nakao et al., 2013). Interestingly, the nomenclature used for these ‘species’ conforms to that proposed by observational parasitologists in the 1920s–1960s, before molecular tools were available to confirm their morphological descriptions and epidemiological observations (Thompson et al., 1995; Thompson and McManus, 2002; Thompson, 2008).

3. Epidemiology of E. granulosus in Australia

3.1. Introduction

Much of the basic knowledge regarding the epidemiology and control of E. granulosus in Australia was generated by Michael Gemmell. His contribution to the field has been immense; the breadth of his studies were wide, covering the impact of climate on egg longevity, immune responses against the parasite in definitive and intermediate hosts, prevalence in domestic dogs and livestock, studies on infection in wildlife and mathematical modelling of transmission as an aid to more effective control (Gemmell and Lawson, 1986). Gemmell’s work has provided a solid platform for many of the current studies reviewed below. However, probably his greatest legacy was his contribution to the development of strategies for the control of E. granulosus transmission. Nevertheless, in the Australian situation, although control of E. granulosus, domestically, has largely been achieved (Jenkins et al., 2014), the presence of an extensive wildlife reservoir on mainland Australia ensures the complete eradication of E. granulosus is unlikely to ever become a reality. Further, unlike the situation with E. multilocularis and Echinococcus canadensis which are maintained in natural wildlife cycles, the wildlife cycle in Australia is an artificial one established as a result of anthropogenic activities and spillover from domestic cycles (Thompson, 2013).

The work of Gemmell had broad ramifications internationally in terms of the epidemiological principles of hydatid control. Many subsequent hydatid control programmes world-wide looked at Gemmell’s critical analysis of the epidemiology in Australia and New Zealand and based their programmes on his data. These served as a foundation for the development of control programmes in many countries, and the epidemiological principles he developed similarly have been used as a model (Eckert et al., 2001).

Echinococcus granulosus is the only member of the genus to occur in Australia, having been introduced with domestic dogs and livestock, mainly sheep, during European settlement (Gemmell, 1990). Domestically, the parasite spread rapidly through sheep and dog populations (Gemmell, 1990), due mainly to a complete lack of understanding of the association between taenid tapeworms in dogs and cysts in sheep. It was not until 1851, (63 years after the first European settlers and their animals arrived in Australia) that infection experiments were undertaken, demonstrating the relationship between cysts and tapeworms. Küchenmeister, working in Germany, fed metacestodes of T. pisiformis to foxes and obtained tapeworms (Küchenmeister, 1851) and in 1853 fed tapeworm eggs to sheep and obtained metacestodes. In the next few years von Siebold and Küchenmeister, working independently, fed protoscoleces of Echinococcus to dogs and obtained tapeworms (von Siebold, 1853; Küchenmeister, 1853). An additional important infection experiment, also undertaken in Germany, was that of Naunyn in 1863 who fed protoscoleces from a human cyst to dogs and obtained adult E. granulosus, reporting his results the same year. This experiment demonstrated for the first time the association between hydatid cysts of sheep and humans and the role of dogs in the life cycle of human hydatidosis. However it was not until 1876 that Leuckart demonstrated eggs of Echinococcus when fed to intermediate hosts (pigs) led to the development of hydatid cysts (Leuckart, 1876). A detailed review of the elucidation of the Taenia and Echinococcus life cycles can be found in Grove (1990). Word of these discoveries took time to reach Australia but it was not long before experimental studies were undertaken in Australia that confirmed Naunyn’s data (Thomas, 1884).

Hydatid disease soon became a major public health problem in the growing population of new Australians (Gemmell, 1990) causing serious illness and leading to the deaths of many colonists.

3.2. Wildlife hosts of E. granulosus in Australia

Data generated in studies on wildlife in Australia have worldwide relevance, an example being the work of Barnes (Barnes et al., 2007a,b, 2008, 2011) investigating the health impacts and growth rate of hydatid cysts in macropodids. Barnes showed that hydatid cysts have a major impact on respiration capacity in macropods, indicating infected animals to be more susceptible to predation by wild dogs. This work complements and validates the studies of Mech (1966) and Jolly and Messier (2004) in the USA with respect to the impact of hydatid disease on moose (Alces alces), rendering infected animals more susceptible to predation by wolves (Canis lupus). Another good example is the study on the encroachment of E. granulosus-infected foxes (Vulpes vulpes) and wild dogs into urban areas in Australia and associated public health implications (Jenkins and Craig, 1992; Jenkins et al., 2008). These studies complement and support work undertaken in Europe with the encroachment into urban centres of foxes infected with E. multilocularis (Deplazes et al., 2004) and studies in Alberta, Canada on E. multilocularis in urban coyotes (Canis latrans) (Catalano et al., 2012).

Australian native wildlife species evolved in isolation from E. granulosus; consequently, they were highly susceptible to infection with this new parasite and wildlife species were soon acting as a major sylvatic reservoir for the perpetuation of E. granulosus in Australia (Durie and Riek, 1952), a situation that prevails today (Jenkins and Morris, 2003).

Initially, transmission of E. granulosus to macropodids occurred through accidental consumption of eggs spread in the environment by the dogs of colonists. Infection in dingo’s eventuated through predation of sheep on the recently established sheep farms. However, it would not have been long before the dingo themselves began to spread eggs throughout their territories, exposing an ever increasing population of macropodids to infection with hydatid...
3.2.1. Dingoes

The Australian wildlife top-order predator coincidentally was a placental canid, the dingo (*Canis lupus dingo*), introduced from south-eastern Asia by visiting seafarers (Corbett, 1995). Dingoes were medium sized wild dogs (11–17 kg) capable of killing sheep. Settlers moving to Australia also brought their domestic dogs and it soon became evident that dingoes were able to breed with domestic dogs and produce fertile hybrid offspring. The result has been that currently in most areas of remaining suitable habitat in south-eastern and eastern Australia the resident top order predator populations no longer consist of pure-bred dingoes but mainly dingo/domestic dog hybrids with occasional pure-bred dingoes (Claridge et al., in press). Animals in these populations are referred to as wild dogs. Pure-bred dingoes are mostly restricted to remote areas, especially in northern parts of Western Australia. Nevertheless, there is no indication that hybrid dingoes are any more or less susceptible to infection with *E. granulosus* than pure-bred animals since no difference in the range of worm burdens has been noted between hybrid and pure-bred dingoes (Jenkins and Morris, 2003; Jenkins et al., 2008). The hunting and pack behaviours of dingo hybrids compared with dingoes also appear to be similar (Claridge et al., in press). The one important physiological difference between dogs and dingoes is breeding capacity: female domestic dogs come into oestrous twice each year whilst for dingoes it is only once. Data collected on the breeding behaviour of dingo hybrids does not indicate they are having more litters per year than pure-bred dingoes, but there have been reports that litter sizes in hybrids are larger than those of pure-bred dingoes and the size of wild dogs has increased approximately 20% during the last 40 years (Claridge et al., in press).

Anecdotally, in many areas wild dog population numbers appear to be increasing, a likely combination of increased litter size and increased food resources due to environment modification. Provision of more pasture and watering points for livestock has led to major increases in macropodid populations in many areas, providing increased food supply for wild dogs. With increased wild dog populations it is reasonable to assume that the biomass and rate of transmission of *E. granulosus* in wildlife is also increasing.

3.3. Echinococcus granulosus-infected wild dog encroachment into urban areas

With increasing wild dog numbers and the spread of urbanisation into areas of hitherto wild dog habitat has displaced young and old animals seeking new uncontested habitat. These animals are encroaching into outer urban areas and bringing *E. granulosus* with them (Brown and Copeman, 2003; Jenkins et al., 2008).

Reports of *E. granulosus*-infected wild dog encroachment into outer suburban areas have begun to appear; Townsville (Brown and Copeman, 2003), Maroochy Shire (Jenkins et al., 2008), the north-western suburbs of Brisbane in the Pine River Shire, Queensland and the outer suburbs of Katoomba in New South Wales (D.J. Jenkins, unpublished data). In a number of urban areas, wild dogs have become numerous and a public nuisance raiding garbage bins, killing domestic pets and menacing residents. They have become such a problem in a number of places that local authorities have employed professional wild dog controllers to remove them using lethal methods. Examination of the intestines of some of these euthanised wild dogs from several locations has revealed high prevalences and heavy worm burdens of *E. granulosus* (Brown and Copeman, 2003; Jenkins et al., 2008). The establishment of peri-urban transmission cycles are unlikely, but regular encroachment of *E. granulosus*-infected wildlife definitive hosts into outer suburbs of urban centres has been demonstrated (Brown and Copeman, 2003; Jenkins et al., 2008), which is difficult to control and likely to be an ongoing issue for local councils to manage.

Hitherto, it was supposed urban human populations in Australia were insulated from exposure to *E. granulosus*, and clearly, with the encroachment of *E. granulosus*-infected wild dogs (and foxes, see Section 3.7.1) into urban areas this is no longer the case.

3.4. Australian native carnivores as definitive hosts for *E. granulosus*

Infection with adult *E. granulosus* has never been reported from Australian native marsupial carnivores. In 2005, Jenkins et al. undertook a small study investigating the faeces from wild-captured spotted-tailed quolls (*Dasyurus maculatus*) for coproantigens of *E. granulosus* and reviewed the literature regarding experimental infection of dasyurids with *E. granulosus*. None of the quoll faeces tested were found to be positive for coproantigens of *E. granulosus* (Jenkins et al., 2005b) and in none of the attempted experimental infections of several species of dasyurid have *E. granulosus* been recovered. From these data, Jenkins et al. (2005b) concluded that dasyurids are refractory to infection with *E. granulosus*.

3.5. Wildlife intermediate hosts

Macropodid marsupials are highly susceptible to infection with the intermediate stage of *E. granulosus* (Jenkins and Morris, 2003; Banks, 2006), often carrying multiple large metacestodes (hydatid Q4 cysts). This is especially the case in a number of wallaby species, namely swamp wallabies (*Wallabia bicolor*) in south-eastern Australia (Jenkins and Morris, 2003). Swamp wallabies are commonly infected with *E. granulosus* (Jenkins and Morris, 2003) and are also a favourite dietary item for wild dogs (Claridge et al., in press), making them pivotal in the transmission of *E. granulosus* in wildlife in south-eastern Australia. Other wallaby species appear to be of similar importance in other parts of Australia (Banks et al., 2006a).

Curiously, on the island state of Tasmania macropodid marsupials never become infected with *E. granulosus* (Howkins, 1966), despite there having been high levels of transmission in domestic animals. This may have been associated with the absence of dingoes and the presence of a dasyurid top order predator, the thylacine (*Thylacinus cynocephalus*), refractory to infection with *E. granulosus* (see Section 3.4). Nevertheless, human hydatidosis became such a major public health problem in Tasmania that a program of intense control was introduced that continued for 30 years (Beard et al., 2001).

Other species of native wildlife susceptible to infection with *E. granulosus* are wombats (*Wombatus ursinus*). However, hydatid infection in wombats has only been reported once (Grainger and Jenkins, 1996). Wombats are consumed periodically by wild dogs.
but appear to only be an occasional host for *E. granulosus*, therefore they cannot be regarded as an important component of the *E. granulosus* life cycle in Australia.

### 3.6. Factors contributing to the transmission success of *E. granulosus* in Australian wildlife

Hydatid disease of macropodids is almost exclusively confined to the lungs, causing major respiratory impairment (Jenkins and Morris, 2003; Barnes et al., 2007a, 2008), which may lead to the death of the host (Johnson et al., 1998; Barnes et al., 2008). Why hydatid infection in macropods should be mainly confined to the lungs is unclear, however, this is also seen with hydatid infection in cervids (Schantz et al., 1995). Hydatid-infected macropods are rendered highly susceptible to predation by dingoes, a similar situation to that with the interaction of wolves and hydatid-infected moose (*A. alces*) in the USA (Mech, 1966; Jolly and Messier, 2004). In addition, cysts in macropodids develop quickly, becoming fertile (containing protoscoleces) in 8–9 months (Barnes et al., 2007a), compared with sheep where the earliest time to fertility is approximately 2 years (Silas, 1980). The increased susceptibility of hydatid-infected macropods to predation by wild dogs means that wild dogs are catching and consuming a disproportionately large number of infected animals which is likely to be a major contribution to the high worm burdens seen in many wild dogs. Within all wild dog populations surveyed, a proportion of the sample has been wild dogs with worm burdens in excess of 100,000 *E. granulosus* (Jenkins and Morris, 1991, 2003; Jenkins et al., 2008). These animals are the “super spreaders” of the population (Gemell and Lawson, 1986), ensuring large numbers of eggs being released into the environment. The home range of wild dogs varies depending on the sex of the animal and availability of food and water; in eastern Australia home ranges average approximately 100 km² (Claridge et al., in press). Therefore large areas can become contaminated with eggs from a single animal, but since packs of wild dogs consisting of several animals usually occupy these spaces (Claridge et al., in press), contamination of the environment with faeces can become heavy in areas used commonly by a resident pack.

The longevity of eggs of *E. granulosus* in the environment is thought to be several months to perhaps approximately 1 year, so long as the environment is not too hot and dry (Eckert and Deplazes, 2004). However, in a study in Argentina (Thevenet et al., 2005), *E. granulosus* egg-laden dog faeces were left in the environment for 41 months. After this time eggs were separated from the faeces and fed to each of four sheep. Hydatid cysts developed in each of the sheep, suggesting that the longevity of eggs of *E. granulosus* in Australia under optimal conditions may be longer than 12 months. Studies reported in Gemmell (1958) revealed the most important environmental conditions required for transmission for *E. granulosus* in Australia to be at least 25 mm of rain for 6 months of the year with temperatures up to 30 °C. Consequently, *E. granulosus* has become more prevalent in eastern Australia, in areas associated with the Great Dividing Range, and in an elevated area of Western Australia (Thompson et al., 1988; Jenkins and Macpherson, 2003) (Fig. 3).

### 3.7. Echinococcus granulosus in introduced wildlife species

#### 3.7.1. Foxes

The first creditable report of *E. granulosus* infection in foxes in Australia was by Gemmell (1959a) where a single terminal segment was recovered from the intestine of one of 41 animals collected in New South Wales. Nevertheless, based on the available experimental infection and survey data, Gemmell (1959a) felt that Australian foxes were not acting as a definitive host for *E. granulosus*. More recently, *E. granulosus* have been recovered from a number of naturally infected foxes in various studies (Thompson et al., 1985; Obendorf et al., 1989; Jenkins and Craig, 1992; Reichel et al., 1994; Jenkins and Morris, 2003). Almost exclusively, worm burdens have been small, usually less than 50 tapeworms/fox. However, in one study (Reichel et al., 1994) several thousand *E. granulosus* in each of two foxes from Victoria were reported. These data are at odds with all other existing fox *E. granulosus* worm burden data that they should be regarded cautiously. However, if corroborated then the role of foxes in the transmission of *E. granulosus* may have to be re-evaluated. The current consensus is that foxes act as definitive hosts for *E. granulosus* but are of minor importance in the transmission of the parasite in rural areas of Australia. However, foxes infected with *E. granulosus* are of potential public health importance when infected animals encroach into urban areas (Jenkins and Craig, 1992).

#### 3.7.2. Feral cats

Infection of Australian feral cats with adult *E. granulosus* has never been reported. Jenkins and Morris (2003) examined the intestines of 23 feral cats collected in an area of high *E. granulosus* transmission in the local wildlife and none was found infected. Jenkins also fed protoscoleces recovered from a wallaby to two cats and two fox cubs, and small numbers of *E. granulosus* were recovered from both foxes but nothing was found in the cats (D.J. Jenkins, unpublished data). It is generally considered that feral cats in Australia are not definitive hosts for *E. granulosus*.

#### 3.7.3. Feral pigs, goats, deer, horses and rabbits

Hydatid disease occurs in Australian feral pigs (Jenkins and Morris, 2003; Lidetu and Hutchinson, 2007). The study of Jenkins and Morris (2003) was conducted in south-eastern New South Wales; 204 pigs were examined, 22.5% contained hydatid cysts and depending on the survey area between 15 and 22% of the cysts were fertile. In contrast, the study of Lidetu and Hutchinson (2007) was conducted in tropical northern Queensland; 74 pigs were examined and 31.1% contained hydatid cysts, but the rate of fertility was almost three times that of the pigs collected in New South Wales. The reason for this difference in cyst fertility is unclear. There is no doubt feral pigs could contribute to transmission of *E. granulosus* in wildlife but the degree to which they contribute is debatable. Adult feral pigs (the animals most likely to harbour fertile cysts) are big and strong and difficult for wild dogs to catch and kill; consequently, wild dogs mainly predate on piglets, animals least likely to contain fertile cysts. Inevitably, adult pigs eventually die and their carcasses become available for scavenging by wild dogs and foxes. However, particularly in summer, scavengers would need to find the carcass soon after death before fertile cysts became non-infective due to putrefaction. Therefore, the role of pigs in the transmission of *E. granulosus* in Australian is likely to be minor.

Hydatid infection in goats has never been reported except for an anecdotal report from Western Australia (R.C.A. Thompson, unpublished data). Periodically, one of the current authors (D.J. Jenkins) has examined small numbers of feral goats, collected from various areas of south-eastern New South Wales, and has never found any infected with hydatid cysts (D.J. Jenkins, unpublished data). The absence of hydatid infection in Australian feral goats is curious because in many countries goats are important intermediate hosts (Schantz et al., 1993). Hydatid cysts have never been reported from feral horses or deer in Australia. Therefore, until data to the contrary are available, feral goats, deer and horses appear not to be part of the lifecycle of *E. granulosus* in Australia.

Rabbits naturally infected with hydatid disease have rarely been reported. The two reports, (cited in Gemmell, 1959b) are...
thought to be a mis-identification of metacestodes of *Taenia serialis*. Wild Australian rabbits have been shown to be susceptible to experimental infection with hydatid disease (Jenkins and Thompson, 1995). The cysts established in the lungs and appeared to be developing normally. However, the rabbits were given a large dose of eggs, far higher than anything they would normally be exposed to in the wild, which may have been the reason they became infected.

3.8. Current prevalence of *E. granulosus* in Australian domestic dogs, sheep and cattle on mainland Australia and the island state of Tasmania

3.8.1. Dogs

The most recently published study on intestinal helminths in Australian mainland dogs is that of Palmer et al. (2008) where 1400 faecal samples collected in vet clinics were sent to the authors for testing. These samples were from urban dog owners with a close interest in the health of their pets; unsurprisingly, no taeniid cestodes were identified in any of the samples collected. The most recent study of intestinal helminths in rural dogs (Jenkins et al., 2014) screened faeces from 1425 rural dogs living in eastern Australia (1119 mainland; 306 Tasmania) with faecal flotation, molecular methods and the *E. granulosus* coproantigen test (Jenkins et al., 2000). Surprisingly, taeniid eggs in faeces were also uncommon, being recovered from the faeces of only 11 dogs. This is likely to be because more than 98% of owners reported feeding dry commercial dog food exclusively or as a major component of their dog’s diet and approximately 50% of owners either dewormed their dog(s) 2 monthly or 4 monthly with a de-wormer containing praziquantel. Nevertheless, an additional 45 dogs were positive in the *E. granulosus* coproantigen test (1.9% of mainland samples and 7.8% of Tasmanian samples). Some coproantigen-positive faecal samples were also positive in an *E. granulosus* coproPCR (Jenkins et al., 2014).

3.8.2. Sheep

Hydatid disease prevalence data are not collected in Australian abattoirs, but in Tasmania occurrence of infection in livestock is monitored and traced back to the property of origin. Hydatid disease still occurs periodically in Australian sheep, however the prevalence has declined steadily during the last 30 years (D.J. Jenkins, personal observations), no doubt a reflection of the decline of *E. granulosus* infection in rural domestic dogs (Jenkins et al., 2014). Sheep populations now most at risk of contracting hydatid disease are those grazed on pasture abutting national and state parks and forests containing populations of wild dogs (Grainger and Jenkins, 1996). These wild dogs periodically enter pastures to predate on the sheep but whilst there also defecate, contaminating the pasture with eggs of *E. granulosus*. Nothing recent has been published regarding the prevalence of hydatid disease in mainland sheep. The most up-to-date data have been collected by the National Sheep Health Monitoring Program (Animal Health Australia, 2011, http://www.animalhealthaustralia.com.au/programs/disease-surveillance/the-national-sheep-health-monitoring-project/).

Routine monitoring of slaughtered sheep for hydatid infection is still undertaken in Tasmania. Since the declaration of provisional eradication in 1996, there have been two reports of infection in sheep; one in 2011 in sheep sent to a mainland abattoir for slaughter (Jenkins et al., in press) the other, more recently in 2013 (D.J. Jenkins, unpublished data) in a single animal, resident on a property in the midlands area of Tasmania. Currently, the origins of this animal are still being investigated.
3.8.3. Cattle

“High levels” of hydatid disease in slaughtered cattle have been reported, anecdotally, from abattoirs in south-eastern Queensland, north-eastern New South Wales and eastern Victoria. No recent data have been published regarding hydatid disease prevalence in Australian cattle, but according to abattoir management prevalence is high enough to be causing substantial financial losses to the abattoirs through condemnation of offal. An estimated loss to the northern Queensland beef industry of $6 million per year due to hydatid-infected offal was reported for 2004, with prevalence levels ranging between 20–51% in cattle from coastal areas and 3% in those from inland areas (Banks et al., 2006b). The source of infection in these cattle is likely from wildlife since cattle are commonly grazed in areas inhabited by wild dogs because predation impacts are less severe than if sheep were grazed in the same areas.

As with sheep, hydatid disease is monitored in slaughtered cattle in Tasmania. Since declaration of provisional eradication in 1996, over 200 hydatid-infected cattle have been identified. Most of these animals proved to be imports from the mainland, mainly Gippsland in Victoria, an area where hydatid infection in cattle is common. However, 31 were animals less than 3 years old and had never left Tasmania (Jenkins et al., 2014). DNA was extracted and sequenced from cysts removed from four of the infected cattle; their infections proved to be the G1 sheep strain of *E. granulosus* (Jenkins et al., unpublished data). The G1 sheep strain not only infects sheep but can also infect cattle, pigs, goats and macropodid marsupials. Less than 5% of G1 hydatid cysts in Australian cattle are fertile (Kumaratilake and Thompson, 1982), therefore cysts from infected cattle, if consumed by dogs, dingo or their hybrids, commonly do not contribute to transmission of *E. granulosus*.

The 24 Tasmanian domestic dogs that tested positive in the *E. granulosus* coproantigen ELISA and the hydatid-infection data from sheep and cattle found since declaration of provisional freedom in 1996 indicate that hydatid disease has not been completely eradicated from the island as was previously thought but continues to be transmitted at low levels (Jenkins et al., 2014).

3.9. Human prevalence

The prevalence of human hydatidosis on mainland Australia is unknown, because in most jurisdictions human hydatid disease has ceased to be notifiable. The last published data on human hydatid disease on mainland Australia was that of Jenkins and Power (1996), but these data were only collected in New South Wales and the Australian Capital Territory and are now out of date. Nevertheless, new cases continue to be identified annually on mainland Australia. A proportion of these new cases are in recently arrived immigrants from a range of countries who were infected in their country of origin. In the study of Jenkins and Power (1996), 60% of cases diagnosed in residents living in major urban centres were recent immigrants. There has been a recent publication on human hydatid disease in Tasmania (O’Hern and Cooley, 2013).

The authors described human hydatid disease in Tasmania since the declaration of provisional freedom in 1996. Transmission of hydatid disease to humans in Tasmania has now ceased, although one or two new cases are diagnosed annually. However, all of these new cases are in elderly people who were thought to have been infected pre-1996.

4. Conclusions

Australia has a long history of research on hydatid disease and the causative agent *Echinococcus*. Much of this was borne from necessity, given the impact the parasite had on public health and the agricultural economy in the late 19th and early 20th centuries in Australia. However, the research that has been conducted in Australia has contributed much to the international research effort on *Echinococcus* and hydatid disease, in particular the value of the organism as a model in studies on developmental biology and epidemiology, not just concerned with *Echinococcus* but more broadly ranging in terms of basic biology and theoretical models of transmission dynamics. We hope that Australia will continue to contribute to research on *Echinococcus* and that this will have an impact internationally and sustain the collaborations that have developed with research groups and centres worldwide.

5. Unicted references

(Gemmell et al., 1986; Harris and Revfey, 1980; Lavikainen et al., 2006).

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