Pathogens in invasive animals of Australia

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Cover images (left to right): Leptospira interrogans (Centers for Disease Control and Prevention USA), Echinococcus granulosis tapeworm (SJ Upton, Kansas State Uni), and Brucella suis granuloma (Centers for Disease Control and Prevention USA).

Pathogens in invasive animals in Australia
Summary

This review provides an overview of diseases or pathogens that have been identified in invasive animals in Australia; particularly feral pigs, foxes and wild dogs, feral cats, feral goats, rabbits, cane toads, rodents (feral mice and ship rats) and European carp. Australian research published between 1990 and 2009 is reviewed. A range of bacterial, viral, fungal, helminth and protozoan pathogens have been identified: many of these have broad host specificity, so could affect a wide range of species.

Pathogens that could significantly impact on native species include *Toxoplasma gondii* (causing toxoplasmosis), *Echinococcus granulosis* (causing cystic hydatidosis) and Asian fish tapeworm *Bothriocephalus acheilognathi*. Pathogens with serious zoonotic potential include *Coxiella burnetti* (causing Q fever), *Brucella* (causing brucellosis), *Leptospira* (causing leptospirosis), Murray Valley encephalitis virus, *Angiostrongylus cantonensis* (causing neurological disease) and various gastrointestinal organisms. Pathogens particularly significant to livestock include *Neospora caninum*, porcine parvovirus, *T. gondii* and *Brucella* (all causing reproductive failure) and *E. granulosus*.

While the greatest potential threat of disease from invasive animals may be from future exotic outbreaks such as foot-and-mouth disease, it is clear many other pathogens of concern currently occur in these animals. The occurrence of such a wide range of pathogens emphasises the need to effectively manage populations of invasive animals to avoid the spread of disease into livestock, native species or humans.
1. **Introduction**

Invasive animals in Australia have the potential to harbour or transmit many diseases that could seriously harm livestock, domestic animals, native fauna or people. Feral animals can carry the same diseases as domestic animals — as such, they are a constant source of reinfection for livestock and wildlife, working against often costly control efforts and threatening Australia’s trade reputation. The threat of invasive animals transmitting exotic disease is also very real and requires constant vigilance.

This review focuses on diseases or pathogens that have been identified in invasive animals in Australia; particularly those that could have a significant impact on the health of Australian agricultural, domestic or native animals. The review is a companion to a previous IA CRC review ‘Research on wildlife disease preparedness’, which primarily focussed on the potential of invasive animals to harbour or transmit exotic disease — covering activities related to preparedness for an exotic disease outbreak in wildlife (eg training exercises, diagnostic tests, sampling for exotic diseases).

This review includes reports on the identification of disease (ie pathological condition), isolation of disease agents (potential or actual, dependent on host and environmental circumstances) and evidence of exposure to pathogens (seroconversions). The majority of the published literature described here dates between 1990 and 2009. It includes incidental findings from surveillance or research activities as well as endemic disease and outbreaks in populations.

The review does not cover details of the epidemiology of diseases. It is intended to provide a simple overview of potentially infectious agents identified in key invasive species, highlighting possible threats to Australian fauna, livestock and society. The likelihood of pathogen transmission is beyond the scope of this review.

The current report addresses two IA CRC milestones under the goal of ‘Reduced risk of disease transfer from invasive animals to livestock and humans’:

- ‘Current information relating to invasive animal diseases (exotic and endemic) collated, published and disseminated.’
- ‘Recommendations developed for improved practices to reduce the impact of endemic disease infections carried by invasive animals.’

2. **Scope and search methodology**

This literature review concentrates on diseases or disease agents that have been identified in the IA CRC’s key pest species, namely: feral pigs, foxes and wild dogs, feral cats, feral goats, rabbits, cane toads, rodents (feral mice and ship rats) and European carp. It includes research on diseases with very specific or broad host range, and zoonotic diseases (which can cause illness in humans). Some reports on diseases identified in domestic or farmed populations (eg commercial piggeries, pet cats and dogs) have been included, since these diseases may have implications for transmission to feral animals.

Published articles from 1990–2009 were searched using the Google Scholar search engine and BiblioLineSM: Wildlife & Ecology Studies Worldwide, BIOSIS Previews and the Australian Wildlife Health Network literature databases. Searches used ‘Australia+disease +feral’ as key words, and ‘Australia+disease +foxes’ etc for individual key pest species. Experts in wildlife health or feral pest control were also contacted by email to ask for relevant literature.

Other sources of information included:

- published management guides (from Bureau of Rural Sciences)
- reports from Northern Australia Quarantine Survey (NAQS)
3. Results

Table 1 lists the main diseases or pathogens found in key invasive animals (cane toads, carp, feral cats, feral goats, feral pigs, foxes, rabbits, rodents and wild dogs) in Australia. Summaries of the results found for each animal species are provided below.

<table>
<thead>
<tr>
<th>Animal</th>
<th>Disease agent</th>
<th>Reference</th>
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<tbody>
<tr>
<td>cane toad (Bufo marinus)</td>
<td>Basidiobdus*</td>
<td>Zahari et al 1990</td>
</tr>
<tr>
<td></td>
<td>Batrachochytrium dendrobatidis</td>
<td>Berger et al 2000</td>
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<td></td>
<td>Salmonella*</td>
<td>Thomas et al 2001</td>
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<td></td>
<td>Mucor amphibium</td>
<td>Speare et al 1994, 1997</td>
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<td></td>
<td>ranaviruses</td>
<td>Zupanovic et al 1998</td>
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<td></td>
<td>ectoparasite: mite (Lawrencarus domrowi)</td>
<td>Speare 1990</td>
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<tr>
<td></td>
<td>various endoparasites: 'parasites of local anurans', 'nematodes', Spirametra mansoni cestode, 'helminth parasites'</td>
<td>Barton 1997, Speare 1990</td>
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<tr>
<td>feral cat (Felis catus)</td>
<td>Bartonella henselae*</td>
<td>Branley et al 1997</td>
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<td></td>
<td>Campylobacter upsaliensis* and C. jejuni* (campylobacter enteritis)</td>
<td>Baker et al 1999</td>
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<td></td>
<td>Cryptococcus gatti*</td>
<td>Malik et al 1992</td>
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<td></td>
<td>Dirofilaria immitis*</td>
<td>Kendall et al 1991</td>
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<td></td>
<td>feline coronavirus</td>
<td>Bell et al 2005</td>
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<td></td>
<td>feline foamy virus</td>
<td>Winkler et al 1999</td>
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<td></td>
<td>feline immunodeficiency virus</td>
<td>Winkler et al 1999, Norris et al 2007</td>
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<td></td>
<td>feline leukaemia virus</td>
<td>OIE 2001</td>
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<td>feline panleucopenia virus</td>
<td>OIE 2001</td>
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<td></td>
<td>periodontal disease</td>
<td>Clarke and Cameron 1998</td>
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<tr>
<td></td>
<td>Toxoplasma gondii*</td>
<td>O’Callaghan and Beveridge 1996, Adams 2003, O’Callaghan and Beveridge 2005</td>
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<td>‘faecal coliforms’*</td>
<td>Ferguson 2005, Cox et al 2005</td>
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<td>Animal</td>
<td>Disease agent</td>
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<tr>
<td>feral goat</td>
<td>Caprine arthritis-encephalitis virus</td>
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<td>(Capra hirsus)</td>
<td>Corynebacterium ovis*-/+</td>
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<td>Coxiella burnetti*</td>
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<td>Eimeria</td>
<td>Main and Creeper 1998</td>
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<td>faecal coliforms*</td>
<td>Ferguson 2005, Cox et al 2005</td>
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<td></td>
<td>Leptospira*</td>
<td>Parkes et al 1996</td>
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<td></td>
<td>Mycobacterium paratuberculosis</td>
<td>South Australian Goat Advisory Group *</td>
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<td>various endoparasites: helminths (22 nematodes, 2 cestodes, 2 trematodes), protozoan and arthropod (eg lice) parasites</td>
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<tr>
<td></td>
<td>Yersinia*/-/+</td>
<td>Parkes et al 1996</td>
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<tr>
<td>feral pig</td>
<td>Actinobacillus</td>
<td>Pavlov et al 1992</td>
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<tr>
<td>(Sus scrofa)</td>
<td>Aeromonas hydrophilla</td>
<td>Pavlov et al 1992</td>
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<td></td>
<td>arboviruses</td>
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<td></td>
<td>Bacillus sp.</td>
<td>Pavlov et al 1992</td>
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<td></td>
<td>Burkholderia pseudomallei*</td>
<td>Pavlov and Edwards 1995, AHSQ 2001</td>
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<td></td>
<td>Chromobacterium freundii</td>
<td>Pavlov et al 1992</td>
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<td>Chromobacterium violaceum</td>
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<td></td>
<td>Entrobacteria agglomerans</td>
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<td></td>
<td>Entrobacteria cloacae</td>
<td>Pavlov et al 1992</td>
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<tr>
<td></td>
<td>Eschereschia coli*</td>
<td>Pavlov et al 1992</td>
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<td></td>
<td>Japanese encephalitis virus*</td>
<td>AHSQ 2006</td>
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<td></td>
<td>Klebsiella pneumonia</td>
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<td></td>
<td>Murray Valley encephalitis virus*</td>
<td>Pavlov et al 1992, Choquenot et al 1996</td>
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<td></td>
<td>Mycobacterium bovis*</td>
<td>Pavlov et al 1992, McInerney et al 1995</td>
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<td></td>
<td>porcine parvovirus</td>
<td>Pavlov et al 1992, Caley et al 1994</td>
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<td>Proteus sp.</td>
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<td></td>
<td>Providencia alcifacens</td>
<td>Pavlov et al 1992</td>
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<td></td>
<td>Pseudomonas pseudomallei*</td>
<td>Pavlov et al 1992</td>
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<td></td>
<td>rickettsiae Spotted Fever Group *</td>
<td>Li et al 2007</td>
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<td></td>
<td>Ross River virus</td>
<td>Pavlov et al 1992</td>
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<tr>
<td></td>
<td>Sindbis virus*</td>
<td>Johansen et al 2005</td>
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<td></td>
<td>Serratia liquifaciens</td>
<td>Pavlov et al 1992</td>
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<td></td>
<td>Serratia marcesens</td>
<td>Pavlov et al 1992</td>
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<td></td>
<td>Staphylococcus aureus*</td>
<td>Pavlov et al 1992</td>
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<tr>
<td>fox</td>
<td><em>Clostridium perfringens</em>/*+</td>
<td>Ferguson 2005, Cox et al 2005</td>
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<td></td>
<td><em>Giardia</em></td>
<td>Ferguson 2005</td>
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<td></td>
<td>faecal coliforms*</td>
<td>Ferguson 2005, Cox et al 2005</td>
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<td></td>
<td>Trubanaman virus*</td>
<td>Johansen et al 2005</td>
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<td></td>
<td>various ectoparasites: ticks, lice, mange mite, fleas*</td>
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<td>rabbit</td>
<td><em>Cryptosporidium</em></td>
<td>Cox et al 2005</td>
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<td></td>
<td>faecal coliforms*</td>
<td>Ferguson 2005, Cox et al 2005</td>
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<td></td>
<td>myxoma virus</td>
<td>Williams et al 1994</td>
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<td>rabbit calivirus A (RCV-A1— endemic calicivirus)</td>
<td>Strive et al 2009</td>
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<td></td>
<td>rabbit haemorrhagic disease or calicivirus</td>
<td>Williams et al 1994</td>
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<td></td>
<td>Sindbis virus</td>
<td>Johansen et al 2005</td>
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<tr>
<td></td>
<td>Trubnanaman virus*</td>
<td>Johansen et al 2005</td>
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<tr>
<td></td>
<td>various ectoparasites: lice, fleas*, mites</td>
<td>Williams et al 1994</td>
</tr>
<tr>
<td></td>
<td>various endoparasites: various worms (liverfluke, dog tapeworm, gastrointestinal nematodes), protozoans (coccidia)</td>
<td>Williams et al 1994</td>
</tr>
<tr>
<td>rats (Rattus rattus) and wild house mice (Mus musculus, Mus domesticus)</td>
<td><em>Angiostrongylus cantonensis</em>—rats</td>
<td>Spratt 2005, Stokes et al 2007</td>
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<tr>
<td></td>
<td><em>Capillaria hepatica</em>—mice and rats</td>
<td>Singleton et al 1991</td>
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<td></td>
<td><em>Cryptosporidium parvum</em>—mice</td>
<td>Singleton et al 2005</td>
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<tr>
<td></td>
<td><em>Escherichia coli</em>—mice</td>
<td>Singleton et al 2005</td>
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<td></td>
<td>Gan Gan virus—rats</td>
<td>Vale et al 1991</td>
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<td></td>
<td><em>Leptospira</em>—mice and rats</td>
<td>O’Neill 2003, AB CRCb</td>
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<td></td>
<td>mouse mammary tumour virus*—mice</td>
<td>Faedo et al 2007</td>
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<td><em>Mycoplasma pulmonis</em></td>
<td>Singleton et al 2005</td>
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<td></td>
<td><em>Neospora caninum</em>—mice</td>
<td>Barratt et al 2008</td>
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<tr>
<td></td>
<td><em>Streptobacillus moniliformis</em>/*+</td>
<td>Singleton et al 2005</td>
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### Animal Disease agent Reference

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<tr>
<th>Animal</th>
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<tr>
<td>wild dog/dingo</td>
<td>Anaplasma platys</td>
<td>Brown et al 2006</td>
</tr>
<tr>
<td>(Canis lupus familiaris)</td>
<td>Babesia canis vogeli</td>
<td>Brown et al 2006</td>
</tr>
<tr>
<td></td>
<td>canine adenovirus</td>
<td>McFarlane 1998, Fleming at al 2001</td>
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<td></td>
<td>canine distemper virus</td>
<td>McFarlane 1998, Fleming at al 2001</td>
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<tr>
<td></td>
<td>canine coronavirus</td>
<td>McFarlane 1998</td>
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<tr>
<td></td>
<td>canine parvovirus</td>
<td>McFarlane 1998</td>
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<tr>
<td></td>
<td>Echinococcus granulosus*</td>
<td>Jenkins and Morris 2003, Brown and Copeman 2003</td>
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<td></td>
<td>Giardia*</td>
<td>Allen 2006</td>
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<tr>
<td></td>
<td>Isospora sp. coccidia</td>
<td>Allen 2006</td>
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<tr>
<td></td>
<td>Leptospira*</td>
<td>Zwijnenberg et al 2008</td>
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<td></td>
<td>Neospora caninum</td>
<td>Allen and Fleming 2003</td>
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<td></td>
<td>parainfluenza virus</td>
<td>McFarlane 1998</td>
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<td></td>
<td>Salmonella sp.*</td>
<td>Allen 2006</td>
</tr>
<tr>
<td></td>
<td>various ectoparasites: ticks, lice, fleas*</td>
<td>Fleming at al 2001</td>
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<tr>
<td></td>
<td>* zoonotic pathogens; *-/+ potentially zoonotic</td>
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</table>

### 3.1 Cane toads

The list of pathogens found in cane toads in Australia is given in Table 1.

**Bacteria:**
Cane toads from northern New South Wales have been found with hepatic lesions caused by bacteria 'morphologically similar to' *Fusobacterium necrophorum* (Speare 1990). In a survey for Salmonella bacterial species in wildlife in Queensland, Salmonella serotype Virchow was isolated.
from a cane toad (Thomas et al 2001). Salmonellosis in cane toads was reported in the 2007 annual report of the Working Group on Wildlife Diseases for OIE. Toads are also known to carry 14 serovars of *Leptospira* in the United States, although no specific mention of this in Australia has been made (Speare 1990).

**Viruses:**
Antibodies to ranaviruses have been found in *Bufo marinus* throughout its range (Zupanovic et al 1998).

**Parasites:**
Some parasites, including helminths acquired from local anurans, have been found in cane toads in Australia (Barton et al 1997, Speare 1990). Natural infections with the intermediate stage of the cestode *Spirametra mansoni* (causal agent of sparganosis) were found in toads from Queensland (Speare 1990). Cane toads often eat animal faeces, so could spread gastro-intestinal parasites such as human-infesting worms (*Trichuris trichiura*, *Schistoma mansoni* and possibly human hookworms) and canine *Uncinaria* hookworms (Freeland 1984, Speare 1990).

The only mention of a parasitic arthropod in cane toads in Australia was of a mite, *Lawrencarus dornrowi* (Speare 1990).

No pathogenic protozoa have been identified in toads in Australia, although they are known to carry *Toxoplasma* and other protozoa in other countries (Speare 1990).

In Australia, recent research on pathogens in cane toads has been on searching for a biological control. *Rhabdias pseudosphaerocephala*, a South American species of lungworm, was identified in cane toads in Australia: it has not been found in any Australian frogs sympatric with cane toads, and so may offer potential as a biocontrol agent (Dubey and Shine 2008).

**Fungi:**
The chytrid fungus *Batrachochytrium dendrobatidis* (causal agent of chytridiomycosis) has been identified in cane toads in Australia (Berger et al 2000). This fungus has been responsible for the serious decline of a range of frog species.

*Basidiobolus haptosporus* Drechsler fungus, which causes human and animal disease in the tropics, has been found in faecal samples of cane toads (Zahari et al 1990). The isolated strains were pathogenic to suckling mice, causing high levels of mortality (Zahari et al 1990).

Another fungus, *Mucor amphibiorum*, has been found in cane toads in Queensland, New South Wales and Northern Territory (Speare et al 1994, 1997), probably sourced from ingested soil. This fungus infects amphibians and platypuses, disseminating through the internal organs and skin and causing a severe ulcerative condition (Speare et al 1994, Connolly et al 1998, Stewart and Munday 2005). Interestingly, mucormycosis or ulcerative mycosis causes high morbidity and mortality rates in platypuses in Tasmania but not in mainland states (Obendorf et al 1993, Connolly et al 1998). Pathogenicity trials with cane toads infected with different isolates of *M. amphibiorum* showed that Tasmanian isolates of *M. amphibiorum* were more likely to cause a serious, long-term infection than were isolates from Queensland or Western Australia (Stewart and Munday 2005). These results suggest that either an endemic strain has mutated and become pathogenic in Tasmania, or that a pathogenic strain has been introduced into Tasmania (Stewart and Munday 2005).
3.2 **European carp**
The list of pathogens found in European carp in Australia is given in Table 1.

*Bacteria:*
The bacterium *Aeromonas salmonicida* has been found in farmed carp in Australia (Wiklund and Dalsgaard 1998). *A. salmonicida* is recognised internationally as a serious pathogen of fish; commercial (e.g. goldfish farms) and recreational fish industries can be affected by the ulcerative disease it causes ((Wiklund and Dalsgaard 1998). The disease agent was likely introduced to Australia from infected goldfish imported from Japan, and has since established and spread via fish distribution (to other goldfish farms) and bait use (Humphrey and Ashburne 1993). Feral goldfish could therefore also potentially carry this disease agent.

*Viruses:*
Koi Herpes virus is a significant disease of carp industries overseas; it is currently being researched as a possible biological agent for naïve European carp populations in Australia (McColl et al 2007).

*Parasites:*
Carp in Australia have been found to carry the Asian fish tapeworm, *Bothriocephalus acheilognathi* (Dove et al 1997, Dove and Fletcher 2000, Koehn et al 2000) which could seriously threaten native fish. Carp are suspected to have introduced this low host-specific parasite to Australian waters: the distribution of *B. acheilognathi* matches the presence of carp in the Murray-Darling Basin (Dove and Fletcher 2000). Young fish are particularly susceptible to infection with the tapeworm (Dove et al 1997), which causes reduced growth and death. The parasite is a threat to endangered native fish species (Dove and Fletcher 2000). It also has the potential to infect commercially important fish species such as Murray cod, golden perch, and silver perch (Dove et al 1997). About a third of juvenile carp tested at Ginninderra Falls in New South Wales were found to be infected (Dove et al 1997).

3.3 **Feral cats**
The list of pathogens found in feral cats in Australia is given in Table 1. A major survey of the diseases and parasites of domestic cats in Australia (Moodie 1995, unpublished report referenced in Dickman 1996) revealed over 100 species of pathogens. While many of these are cat-specific, at least 30 of the pathogens have also been found in native animals (Dickman 1996).

*Bacteria:*
Pathogenic bacteria reported in feral cats include *Bartonella henselae* (Branley et al 1997), *Campylobacter upsaliensis* and *C. jejuni* (Baker et al 1999), and *Clostridium perfringens* (Cox et al 2005, Ferguson 2005). *B. henselae* is the causative agent of cat scratch disease in humans and was identified in 24 out of 59 feral cats studied in Sydney; more than twice the prevalence found in domestic cats tested (Branley et al 1997). While this bacterium generally only produces a mild infection in people who have been bitten or scratched, it can result in skin or eye complications. *C. upsaliensis* and *C. jejuni* were identified from faecal samples of stray cats in South Australia (Baker et al 1999): these bacteria can cause Campylobacter (gastro)enteritis — the most frequently notified infectious disease in Australia in 1996 — and *Campylobacter* species have also been associated with purulent arthritis and Guillian-Barré syndrome (Baker et al 1999). In investigations of potential contamination of Sydney’s water supply, Ferguson (2005) and Cox et al (2005) identified *C. perfringens* in feral cats. The only other report of pathogenic bacteria in feral cats was an article on periodontal disease (Clarke and Cameron 1998).
Viruses:
Viruses identified in feral cats include feline immunodeficiency virus (Winkler et al 1999, OIE 2001, Norris et al 2007), feline coronavirus (Bell et al 2005), feline foamy virus (Winkler et al 1999), feline leukaemia (OIE 2001) and feline panleucopenia virus (OIE 2001). While these viruses are unlikely to impact species other than cats, they may pose a health threat to domestic cats.

Parasites:
Feral cats have been found to be a significant reservoir of pathogenic intestinal parasites. Some studies show extremely high occurrence of parasites in cats (eg 91% of cats in the study by O’Callaghan and Beveridge 1996, 75% in Adams 2003). High prevalences of zoonotic species have been recorded; for example in Tasmania, 84% of tested cats had Toxocara cati roundworms, 50% had Toxoplasma gondii protozoa and 21% had Giardia duodenalis protozoa (Milstein and Goldsmid 1997).

Many helminth parasites have been identified in feral cats in studies in Western Australia (Adams 2003), Northern Territory (Barton and McEwan 1993, O’Callaghan and Beveridge 1996), Tasmania (Milstein and Goldsmid 1997), New South Wales (Kendall et al 1991), Kangaroo Island (O’Callaghan et al 2005) and Christmas Island (Adams et al 2008). The full list is given in Table 1. Most common helminths include Abbreviata hastaspicula, Ancylostoma spp., Cylicospirura felineus, Onciola pomatostomi, Spirometra erinaceieuropaei, Taenia taeniaeformis, Physaloptera praeputialis and Toxocara cati (Barton and McEwan 1993, O’Callaghan and Beveridge 1996, Adams 2003, 2008; O’Callaghan et al 2005). Some parasites were found to have transferred from native birds, reptiles and mammals eaten by cats; for example, O. pomatostomi is picked up from birds and Abbreviata hastaspicula from varanid lizards (O’Callaghan and Beveridge 1996, O’Callaghan et al 2005).

Many of the helminths identified could affect survival and reproductive capacity of native animals (Adams 2003). Zoonotic helminths associated with feral cats include Taenia taeniaeformis tapeworms, Ancylostoma hookworms, Toxocara cati roundworms and Dirofilaria immitis heartworms. Physaloptera praeputialis also has a wide host range, and can cause severe gastric disease (O’Callaghan et al 2005). Spirometry erinacei tapeworm, common in feral cats in eastern Australia, can also severely affect a wide range of native species (Dickman 1996).

The most common protozoan parasites found in feral cats are Isospora rivolta, I. felis, Giardia duodenalis and Toxoplasma gondii — as mentioned above, the latter two are zoonotic (O’Callaghan and Beveridge 1996; Milstein and Goldsmid 1997; Adams 2003, 2008). Cryptosporidium, considered a pathogen of mammals, birds and reptiles, has been identified in feral cats of the Northern Territory (O’Callaghan et al 2005) and Western Australia (Adams 2003).

$T. gondii$ is probably the most significant parasite occurring in feral cats. Members of the cat family (Felidae) are the only known definitive hosts for $T. gondii$. The parasite can cause significant disease or death in humans, dogs, marsupials and other mammals, through consumption of uncooked meat or exposure to contaminated cat faeces (Canfield et al 1990, Dickman 1996). Toxoplasmosis is considered to be the third leading cause of death (in humans) attributed to foodborne illness in the United States (ScienceDaily 2008).

Toxoplasmosis is also known to result in abortion and congenital defects in livestock. Two Animal Health Surveillance Quarterly reports in Tasmania attributed late abortions, stillbirths and perinatal lamb mortality in ewes to $T. gondii$ transmission from feral cats (AHSQ 1998 Vol 3 and AHSQ 2004 Vol 9). O’Callaghan and Beveridge (1996) concluded that feral cats are responsible for the high prevalence of Toxoplasma in sheep on Kangaroo Island.
Symptoms of toxoplasmosis in native fauna include poor coordination, blindness, lethargy, respiratory and enteric distress, and often sudden death (Canfield et al 1990). Signs of \textit{T. gondii} infection have been recorded in at least 30 species of native mammals including macropods (eg Bennett’s wallaby), eastern barred bandicoots, quokkas, dasyurids, possums and wombats, and in several species of native birds (Dickman 1996, Obendorf et al 1996, Eyman et al 2006, OIE 2007). Pademelons (\textit{Thylogale billardieri}) in Tasmania were reported with blindness due to severe chorioretinitis associated with \textit{Toxoplasma} sp. infection (OIE 2001). Several reports were found of eastern brown bandicoots (\textit{Perameles gunnii}) and southern brown bandicoots (\textit{Isoodon obesulus}) being severely affected by toxoplasmosis, contributing to population decline (Lenghaus et al 1990, Obendorf et al 1996, OIE 2001). Obendorf et al’s study (1996) found evidence of \textit{T. gondii} infection (by seroconversions analysis) in 10 out of 150 eastern barred bandicoots in Tasmania. \textit{T. gondii} oocysts can be transmitted to bandicoots through consumption of earthworms and other invertebrates in contaminated soil (Obendorf et al 1996, Bettiol et al 2000).

Marine animals, including beluga whales, dolphins, sea lions, sea otters and seals are also susceptible to toxoplasmosis — freshwater runoff contaminated with cat faeces has been blamed (ScienceDaily 2008).

\textit{T. gondii} was identified in half of 39 feral cats studied in Tasmania (Milstein and Goldsmid 1997), in 89\% of the cats tested on Kangaroo Island (O’Callaghan et al 2005) and it was the most common parasite detected in feral cats on Christmas Island (Adams et al 2008). However, in Western Australia, a study of 379 cats identified \textit{T. gondii} in only 4.9\% (Adams 2003). However, even if only a few cats are shedding \textit{T. gondii} oocysts at any given time, enormous numbers are produced and their resistance to destruction makes widespread contamination of the environment likely (Eyman et al 2006). At present there is no vaccine to control toxoplasmosis in humans, cats, or wild animals, and treatment options are very limited.

\textit{Fungi:}
A significant fungus found in feral cats is \textit{Cryptococcus gattii}: this zoonosis causes cryptococcosis, a systemic fungal disease affecting a large range of native mammals, birds and reptiles (Krockenberger et al 2005). \textit{C. gattii} can lead to life-threatening infections of the pulmonary and central nervous systems. It can be carried by cats, dogs, horses and goats (Krockenberger et al 2005). Cryptococcosis in cats is also described by Malik et al (1992) and O’Brien et al (2004).

### 3.4 Feral goats

The list of pathogens found in feral goats in Australia is given in Table 1. Feral goats are prone to a number of diseases currently in Australia, including Q fever, tetanus, leptospirosis, brucella melitensis, hydatids, pulpy kidney, blackleg, and various parasitic worms (Biosecurity Queensland 2007).

\textit{Bacteria:}
Of significant concern to human health is the bacterium \textit{Coxiella burnetti}, causing Q fever, which is widespread among feral goats (seroprevalence of 52\% in one study; Parkes et al 1996). Although usually non-pathogenic in goats, Q fever can cause pneumonia, hepatitis and death in humans, and is considered the most infectious disease in the world, with people being capable of becoming infected from a single cell (Maurin and Raoult 1999, OIE 2006). An outbreak of Q fever was reported in Victorian abattoir staff involved in the slaughter of feral goats (Buckley 1980). A more recent case occurred in Waikerie in South Australia, where a cluster
of Q fever cases (including one death) were thought to be linked to inhalation of contaminated dust from the local abattoir, affecting townsfolk not involved in meat preparation (Pedler 2007, ABC News 10/9/2007).

Melioidosis, caused by the bacterium *Pseudomonas pseudomallei*, is considered to be endemic in tropical Australia, with sheep and goats particularly susceptible (Choya et al 2000). It is likely to be responsible for the absence of feral goats in the Northern Territory’s top end (Parkes et al). Another bacterium reported in feral goats is the zoonosis *Corynebacterium ovis*, which causes caseous lymphadenitis (abscesses on the lymph nodes) (Bateyet al 1985, Parkes et al 1996).

Other non-specific bacteria, ‘faecal coliforms’, have been identified from feral goats in studies of possible sources of water supply contamination (Ferguson 2005). The first report of *Mycobacterium bovis* bacteria in a goat was also found in this literature search, but described infection of a domestic goat (Cousins et al 1993).

Parkes et al (1996) comment that other important diseases of livestock (such as yersiniosis, leptospirosis and mycobacterial diseases such as Johne’s disease and bovine tuberculosis) ‘appear to be rare’ in feral goats. Johne’s disease and tuberculosis are national notifiable animal diseases. While no specific reports of occurrence Bovine Johne’s Disease (BJD) and Ovine Johne’s Disease were found, risks from these chronic wasting diseases being endemic in feral goats in South Australia are mentioned (South Australian Goat Advisory Group meeting¹). The Goat Industry Council of Australia recently introduced a national goat health statement that includes a risk rating system for Johnes’s disease (also known as paratuberculosis), to help the 8000 goat producers Australia-wide provide information about the health status of their goats for sale². This disease is clearly of concern to the goat industry. There is also controversy regarding Johne’s disease having potential association with human Crohn’s disease (eg Greenstein).

**Viruses:**
Caprine arthritis/encephalitis virus infection has been found in goats in South Australia (Surman et al 1987), and was also reported by the OIE Working Group (OIE 2006). A retroviral infection of goats, caprine arthritis/encephalitis incidence is low and sporadic. It can lead to chronic disease of the joints and, on rare occasions, encephalitis in goat kids.

**Parasites:**
Feral goats are known to carry 22 nematode, 2 cestode, 2 trematode, 4 arthropod, and 3 protozoan parasites (Parkes et al 1996 and references therein). Many of these can infect domestic sheep and all can infect domestic goats. The most common health problem causing death in feral goats in the Northern Territory is reportedly worms (Rural ABC May 2008³). A link has been suggested between feral goats and the occurrence of hydatid tapeworms in cattle in the Kimberley region of Western Australia, where populations were previously uninfected (Lymbery et al 1995).

Enteric coccidiosis, an economically important parasitic disease particularly of neonatal domestic goats, has been found in feral goats (Main and Creeper 1998). Coccidiosis of Brunner’s (duodenal) glands in feral goats that died during overseas transport to the Middle East was described by Main and Creeper (1998). The researchers concluded the condition was likely caused by the protozoan parasite *Eimeria* spp, and that stress associated with transport contributed to severe coccidiosis and death (Main and Creeper 1998).

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³ see http://www.abc.net.au/rural/news/content/200805/s2242964.htm
3.5 Feral pigs

Feral pigs host a wide range of pathogens; the full list from the literature is given in Table 1. Some of these are specific to pigs, such as classical swine fever, and others can affect a wide range of species. Many of the pathogens are significant zoonoses, including *Leptospira*, *Brucella*, *Mycobacterium*, Ross River Virus and Murray Valley encephalitis virus (Pavlov et al 1992). Analyses of feral pig populations by Hampton et al (2004; Cowled 2006, 2008a) have shown that pigs are likely to play a significant role in spreading endemic or exotic disease, particularly around major river catchments.

**Bacteria:**

Leptospirosis is considered the most common bacterial disease in feral pigs (Choquenot et al 1996). It is caused by *Leptospira interrogans* and results in infertility and birth disorders in pigs and other animals. The bacterium causes influenza-like Well’s disease in humans, also known as ‘canecutter’s disease’ from when sugar was harvested manually and infections occurred from contact with contaminated pig or rodent urine in the cane. Complications include jaundice and bleeding disorders. A national notifiable disease in people, leptospirosis continues to be a significant cause of ill health in people, with high hospitalisation rates (46%) recorded in Australia (Smythe et al 2000).

At least 11 different *L. interrogans* serovars have been found in feral pigs in Australia (Pavlov and Edwards 1995, Heise-Pavlov and Heise-Pavlov 2003). Serovar *pomona* is the most common in New South Wales, found in up to half the pigs that have been examined (Choquenot et al 1996, Mason et al 1998). This serovar is a threat to livestock, and hunters and other outdoor recreational groups (Mason et al 1998). A *pv pomona* infection leading to a ‘bovine abortion storm’ in a New South Wales property was attributed to feral pigs (AHSQ 2000 Vol 5.4). Another serovar, hardjo, is more predominant in wildlife, but is also found in pigs (Mason et al 1998). This serovar is the predominant serovar infecting people in temperate regions such as Sydney (Eymann et al 2006). Leptospirosis was considered to be a significant contributor to a lower-than-expected pig population density found in tropical rainforest in northeastern Australia (Heise-Pavlov and Heise-Pavlov 2003).

The bacterium *Brucella suis* is considered endemic in feral pigs in central Queensland (Mason and Fleming 1999), serving as a source of infection of domestic/commercial pigs and also cattle herds. These authors demonstrated that *B. suis* has not spread beyond Queensland to New South Wales: only 1 out of 256 hunter-killed pigs tested from New South Wales was seropositive (Mason and Fleming 1999). It has also not been detected in Western Australia or Northern Territory (Choquenot et al 1996). Brucellosis is a national notifiable animal disease (caused by *B. abortus*, *B suis*, *B canis* and *B. melitensis*). Since successful eradication programs against *B. abortus* in cattle, the most significant causal agent in humans is *B. suis*. In humans, brucellosis can be serious and long lasting, resulting in fever, muscle/joint aches and abortion. It is strongly linked to workers associated with handling, hunting or butchering pigs (Choquenot et al 1996, Mason and Fleming 1999). Human brucellosis cases are on the rise in Queensland (Robson et al 1993), and a recent case also occurred in the Hunter Valley region of New South Wales (Communicable Diseases Bulletin 2006, 160): all these people were involved with killing feral pigs. The threat to people is increasing with the growth of the lucrative pig hunting industry (Robson et al 1993).

*Streptococcus suis* is another bacterium causing occupational hazard for piggery workers, with recent cases reported in New South Wales (AB CRC newsletter 19 Dec 2008). Other bacteria associated with feral pigs include Spotted Fever Group rickettsia in ticks, found to be endemic in...
south west Western Australia (Li et al 2007). *Salmonella anatum* and *S. typhimurium* were found in a third of 154 feral pig carcasses processed for human consumption (Bersink et al 1990). Meliodosis was found in two thirds of feral pigs tested in north Queensland (Pavlov and Edwards 1995); this disease appears more commonly in wetter weather in the Top End (AHSQ 2001, 6.4).

Tuberculosis caused by *Mycobacterium bovis* has rarely been found in feral pigs since the national bovine tuberculosis eradication campaign (BTEC) in cattle and buffalo, completed in 1997 (Choquenot et al 1996). *M. bovis* was identified in only 2 out of 790 feral pigs examined in Northern Territory — a significant drop since the early 1970's, before the BTEC (McInerney et al 1995).

**Viruses:**
Arboviruses and parvoviruses were also reported in the literature (Pavlov et al 1992, Caley 1993, Caley et al 1994). Porcine parvovirus (PPV) antibodies were found in over half of 298 feral pigs tested in the Douglas Daly district of Northern Territory, and PPV was concluded likely to be endemic in Australia (Caley 1993, Caley et al 1994). This parvovirus is a common cause of reproductive failure in piggeries.

The highly infectious zoonotic Menangle virus was reported in pigs at a commercial piggery in the Northern Territory, although it was not detected in 190 feral pigs tested by Kirkland et al (2001). The disease causes reproductive disorders in pigs, and flu-like symptoms in people. Originating from bats, the virus becomes amplified in pigs, making the transmission threat much greater (similarly to Nipah virus in Malaysia, Hooper 2001). According to Animal Health Australia, there has been only a single outbreak of Menangle disease in pigs in Australia, occurring in 1997\(^4\). The disease is a national notifiable animal disease.

Another virus that is amplified in pigs is Japanese encephalitis virus (JEV). Antibodies to JEV were detected in sentinel pigs on Cape York Peninsula in 1998, and a fisherman in that area contracted an infection (Exotic Animal Diseases Bulletin 2003). These were the only known cases of this notifiable disease occurring on the Australian mainland, until JEV was again isolated from sentinel pigs in the northern peninsula in 2004, and feral pigs in western Cape York showed serology patterns consistent with exposure to the virus (AHSQ 2006, 11.2). There has been no evidence of transmission of JEV since 2005. note: Van den Hurk et al (2003): mosquito host-feeding patterns in northern Australia showed pigs NOT automatically involved in JEV disease transmission despite presence as possible host- also despite them being the main amplifier of Japanese encephalitis disease in south east Asia, mosquitoes were found to preferentially feed from marsupials in this study (only 9% of mosssies fed from pigs)- particularly Agile wallabies, so pigs less involved-ironically/fortunately marsupials are poor hosts for JEV, thus diminishing the threat of the disease to humans despite the abundance of pigs.

Another virus identified in feral pigs by seroconversion is Trubanaman virus (Johansen et al 2005). This mosquito-borne virus was identified in 3.5% of feral pigs tested in south-western Western Australia (Johansen et al 2005). It is suspected as causing polyarthritic symptoms in people, similar to Gan Gan virus (Boughton et al 1990).

**Parasites:**
Cysts from the tapeworm *Echinococcus granulosus* were found in 9% (Banks et al 2006) and 31% (Lidetu and Hutchinson 2007) of feral pigs studied in northern Queensland — between 50 and 70% of these lung and liver cysts were viable. Viable cysts were also found in about half the pigs examined in the Kosciuszko region of New South Wales, although the viability of these cysts was lower (less than 22%; Jenkins and Morris 2003). These results show that feral pigs can contribute a significant part in the sylvatic cycle of this parasite (Lidetu and Hutchinson 2007).

*E. granulososus* was also found in feral pigs in Western Australia, where they were involved in an


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unusual cycle involving kangaroos and domestic dogs (Thompson et al 1988). This parasite also infects humans and native wildlife, often with serious consequences (see wild dogs section below).

Other endoparasites, such as stomach worm, lung worm and kidney worm, have also been found at high infection rates in feral pigs (Pavlov and Edwards 1995, Heise-Pavlov and Heise-Pavlov 2003). Sparganosis disease, caused by zoonotic Spirometra tapeworms, was found in high prevalence in pigs of northern Queensland (Pavlov et al 1992): this parasite can also infect people who eat inadequately cooked pork.

Toxoplasmosis occurs in feral pigs, with the national serological prevalence for Toxoplasma protozoa estimated at 9% (AHSQ 1998 Vol 3.1). Other protozoan parasites that present human health risks, such as Giardia, Cryptosporidium, Balantidium and Entamoeba, were detected in faeces from feral pigs caught in metropolitan drinking water catchment areas in Western Australia (Hampton et al 2006). The pigs not only aid in transmission of diseases directly through faecal contamination of water; turbidity from pig wallowing may also protect waterborne pathogens from chemical disinfection treatment (Hampton et al 2006).

Fungi:
Feral pigs also carry and spread the fungus Phytopthera cinnamomi that causes dieback disease in native flora (Pavlov et al 1992): P. cinnamomi has been declared a key threatening process under the Environment Protection and Biodiversity Conservation Act 1999.

3.6 Foxes

Foxes in Australia do not carry many significant pathogens; the exceptions are the agents for mange and canine distemper, and the recent emergence of Echinococcus granulosus (causing hydatidosis) (Saunders et al 1996). The full list of pathogens identified in foxes is given in Table 1.

Bacteria:
Clostridium perfringens and various ‘faecal coliforms’ (causal agents of gastroenteritis) have been identified in foxes in a study of water catchment quality near Sydney (Ferguson 2005, Cox et al 2005).

Viruses:
A low prevalence of canine herpes virus has been found in foxes (Reubel et al 2004). Trubanaman virus has been identified by serology (Johansen et al 2005). Canine distemper virus has also been identified in foxes (K. Rose, Taronga Zoo, personal communication).

An interesting overseas study on predator-mediated spread of viral disease found H5N1 avian influenza virus in foxes that had eaten infected bird carcasses (Reperant et al 2008). The foxes could excrete the virus even though remaining free of severe disease (Reperant et al 2008): this report shows the potential role for carnivores in spreading such disease in Australia too.

Parasites:
Echinococcus granulosus tapeworm has been widely found in up to half the foxes studied in southeastern Australia, although the worm burdens are usually much lower than found in dogs (Jenkins and Craig 1992, Reichel et al 1994, Jenkins and Morris 2003, Jenkins 2006). Disease from E. granulosus infection affects agricultural production, human health and wildlife in Australia. Some wildlife species are particularly susceptible. This is discussed further below (see Wild dogs section). People collecting fox tails as part of a bounty program in Victoria were deemed to be at high exposure risk to hydatids (AHSQ 2002, 7.2). Accidental contact with foxes or their contaminated faeces presents a public health risk (Jenkins and Morris 2003), with encroachment of foxes into urban areas presenting a new threat (Jenkins 2006, Jenkins et al 2008).
Other parasites identified in foxes include heartworm, found in a Melbourne study (Marks and Bloomfield 1998), and the zoonotic *Dirofilaria immitis* heartworm found in 9% of 68 foxes studied near Sydney (Mudley and Starr 1984). *Cysticercus ovis* cysts in lambs, causing condemnation of up to 70% of 107 abattoir lambs in one report, were assumed to have come from tapeworms from foxes and/or dogs (AHSQ 2002, 7.2).

Cysts of zoonotic *Giardia* protozoa have also been found in foxes, in a water catchment area near Sydney (Ferguson 2005). Foxes have also been identified with *Toxoplasma* protozoa (K. Rose, Taronga Zoo, personal communication).

Various ectoparasites, including ticks, lice, mange mite and fleas also occur on foxes (Saunders et al 1996). Dog ticks may be responsible for the low fox density in southern New South Wales (Saunders et al 1996). The mite *Sarcoptes scabiei*, causing sarcoptic mange, is commonly carried by foxes, and seriously affects wombats and possibly other native wildlife (Skerratt 2004, 2005).

**Fungi:**
The only report of fungi in foxes was of ringworm (*Microsporum*) occasionally being found (Saunders et al 1996).

### 3.7 Rabbits

The full list of pathogens found in rabbits in Australia is listed in Table 1. Most search results returned recent literature on rabbit haemorrhagic disease. The majority of the pathogens only present a disease threat to domestic or commercially bred rabbits, although some zoonoses were identified, as discussed below.

**Bacteria:**
Zoonotic faecal coliforms have been isolated from rabbits in water quality studies near Sydney (Ferguson 2005, Cox et al 2005).

**Viruses:**
Myxoma virus (casual agent for myxomatosis) and rabbit calicivirus (RCV, causing rabbit haemorrhagic disease) were introduced biocontrol viruses and are now considered endemic in rabbits in Australia (Williams et al 1994). Recently a benign endemic strain of RCV (‘RCV-A1’) has been identified, possibly conferring immunity to the biocontrol strain, compromising its effectiveness (Strive et al 2009).

Zoonotic arboviruses Sindbis virus (causing Sindbis fever: fever and malaise in people) and Trubanaman virus (possibly causing polyarthritic symptoms in people, Boughton et al 1990) have been identified in rabbits, at 0.8% and 2.4% prevalence respectively (Johansen et al 2005).

**Parasites:**
*Cryptosporidium* and *Eimeria* protozoa have been observed in rabbits (Cox et al 2005). Various other helminth parasites including liver fluke (*Fasciola hepatica*), dog tapeworms (*Taenia pisiformis* and *T. serialis*) and gastrointestinal worms (*Graphidium strigosum* and *Trichostrongylus retortaeformis*) are carried by rabbits (Williams et al 1994).

### 3.8 Rats and mice

A variety of pathogens and parasites have been reported in mice and rats in recent literature; the full list is given in Table 1. The majority of murine viruses were identified in a search for biological controls of mice plagues (eg Singleton et al 1991, 2005). The main zoonotic pathogens found in black rats (*Rattus rattus*) and mice (*Mus musculus* and *Mus domesticus*) are discussed below.
Bacteria:
A study of polyarthritis reported the bacterium *Streptobacillus moniliformis* in wild mice in Victoria (Taylor et al 1994). *S. moniliformis* is found in many rodents and various mammals and is the cause of rat-bite fever, transmitted to humans who have been bitten or scratched by an infected animal. Without treatment, people can develop serious infections of the lining of the heart (endocarditis), or other complications such as pericarditis, meningitis, or pneumonia. In many areas of the world, rat-bite fever has a mortality rate of 13%.

The zoonotic bacterium *Leptospira* has been reported in both mice and rats (CSIRO factsheet5, O’Neill 2003). Leptospirosis can affect a wide range of domestic and wild animals, and is a notifiable disease in humans (see section above on Feral pigs). Workers in banana and sugarcane industries are the most common to become infected from rats: 58% of leptospirosis notifications in Queensland in 1998–1999 reported exposure to rats (Smythe et al 2000).

The zoonotic bacterium *Escherichia coli* and another possible zoonose, *Streptobacillus moniliformis*, were also reported in mice (Singleton et al 2005).

Viruses:
Mouse mammary tumour virus (MMTV) was found in mice in southeastern Australia by Faedo et al (2007), who described it as enzootic in northern Victoria. The possibility that this virus is a causative agent in human breast carcinogenesis has recently been raised, with reports of MMTV infection of cultured human mammary cells, and MMTV DNA sequences being detected in human breast cancerous tissue but not healthy tissue (Indik et al 2007 and references therein).

Of the murine viruses reported, lymphocytic choriomeningitis virus (LCMV) carried by mice is of particular concern because it is transmissible to humans during pregnancy (Moro et al 2003). It can cause meningitis, abortion and foetal abnormalities in humans. LCMV was found by serology in 9.6% of mice tested in northeastern New South Wales — its presence in a plague-prone area is of public health concern (Smith et al 1993). Smith et al (1993) also identified a number of other viruses normally found in laboratory mice, but little is known about the susceptibility of native Australian rodents to these viruses.

Gan Gan virus, which can cause polyarthritis in people, has been serologically identified in rats in a New South Wales study (Vale et al 1991).

One report was also found of introduced Asian rodents being suspected as the source of a lethal retrovirus in koalas (from repeated incursions over thousands of years, AB CRC newsletter 19 Dec 08).

Parasites:
One of the most significant parasites found in rats (*R. rattus* and *R. norvegicus*) is the zoonotic lung worm *Angiostrongylus cantonensis* (Spratt 2005, Stokes et al 2007). It causes neurological disease (neural angiostrongyliasis) and death, which has been reported in a variety of Australian mammals and birds, and in domestic dogs (Spratt 2005, Stokes et al 2007). Three human infant fatalities have also occurred in Australia from *A. cantonensis* infection (OIE 2001, Stokes et al 2007). The current known distribution of *A. cantonensis* in Australia is northern (prevalence 11.8%) and southeastern Queensland (prevalence 6.5%) and around the Sydney–Jervis Bay region (prevalence 4.4%, Stokes et al 2007). Rodents become infected by ingesting infected snails or slugs and are the hosts for further development of this parasite. The presence of rats in bushland

close to campgrounds and rural homes has human health implications: the disease may be transmitted from rat faeces or by ingestion of infected snails and slugs or their slime (eg in improperly washed salad).

*Capillaria hepatica* has been reported in mice and both black and Norway rats (Singleton et al 1991). It is a zoonotic parasitic nematode that causes hepatic capillariasis (liver lesions) in rodents and numerous other mammal species. Although rare in people, the disease can be fatal.

Another zoonotic protozoan parasite found in mice is *Cryptosporidium parvum* (Singleton et al 2005). It can cause cryptosporidiosis, a parasitic disease of the mammalian intestinal tract. Moro et al (2003) also recognised mice as a reservoir for pathogens such as *Cryptosporidium* and *Giardia* for transmission to people and other mammals.

*Neospora caninum*, a protozoan parasite causing neosporosis (a significant cause of reproductive failure in cattle — see Wild dog section below) is considered common in rodents, and was identified in 28 of 104 feral mice serologically tested by Barratt et al (2008).

A recent report by Wyatt et al (2008) proposed that the historic extinction of the endemic Christmas Island rat (*Rattus macleari*) is likely to have been partly or wholly caused by a pathogenic trypanosome (*Trypanosoma lewisi*) carried by fleas hosted on introduced black rats.

### 3.9 Wild dogs

The full list of pathogens and parasites found associated with wild dogs is given in Table 1. The most significant of these are discussed below.

**Bacteria:**
Zoonotic *Campylobacter upsaliensis*, *C. jejuni* and *C. coli* (causal agents for campylobacter enteritis, one of Australia’s most notified infectious diseases) have been identified in stray and wild dogs (Baker et al 1999, Allen 2006). Zoonotic *Salmonella* sp. was also observed in urban dingoes in southeast Queensland (Allen 2006). *Leptospira interrogans* (causing canine leptospirosis) was identified in dogs held in animal shelters across Australia (Zwijnenberg et al 2008). *Anaplasma platys* rickettsia, which can cause bleeding disorders in dogs, was found on free-roaming dogs associated with Aboriginal communities (Brown et al 2006).

**Viruses:**
Wild dogs have been found with canine parvovirus, canine adenovirus and canine distemper virus, providing a reservoir for domestic dogs (McFarlane 1988, Fleming et al 2001). Canine distemper also had a devastating effect on dingoes in northern Australia in the 1970’s (Corbett 1995).

**Parasites:**
The most significant parasite carried by wild dogs (the definitive hosts) is probably *Echinococcus granulosus*. This tapeworm is widely found in wild dogs, particularly in eastern Australia, with worm burdens in extraordinarily high numbers (Jenkins and Morris 1991, 2003; Reichel et al 1994; Brown and Copeman 2003; Jenkins 2006). Prevalences of 25-100% have been recorded in Victoria and New South Wales, and 40-100% in eastern Queensland (Allens 2006, Jenkins 2006). Worm burdens of more than 10,000 worms are common, with 100,000 regularly encountered (Jenkins 2006), so only a few such dogs can maintain a high level of infection in a region (Jenkins and Morris 1991).
High infection rates have also been found in native species, especially macropods: for example New South Wales studies found 40% of macropods in the Bondo State Forest (Jenkins and Morris 1991), and up to 69% of wallabies in Kosciuszko National Park (Jenkins and Morris 2003) were infected. The main wildlife transmission cycle of \( E.\ granulosus \) is perpetuated from through a predator-prey relationship between wild dogs and macropods, although foxes, feral pigs and other wildlife may be involved (Jenkins 2006). The wildlife cycle spills over into a domestic cycle, affecting livestock, domestic dogs and people. Wildlife reservoirs hamper domestic hydatid control campaigns; to date, \( E.\ granulosus \) has only been successfully eradicated from Tasmania (Jenkins 2005).

Disease from \( E.\ granulosus \) infection affects wildlife, livestock and human health in Australia. Hydatids disease has serious consequences for wildlife, particularly macropods (Jenkins 2005, 2006). Infected animals often have multiple cysts in the lungs, particularly affecting their ability to run from predators (Jenkins and Morris 1991). Viable cysts were identified in eastern grey kangaroos, red-necked wallabies, swamp wallabies and wombats (Grainger and Jenkins 1996, Jenkins and Morris 2003).

A high prevalence of hydatid cysts in sheep in Victoria was transmitted by wild dogs from adjacent Crown land: transmission was believed to be by direct contact with dogs, and via contaminated faecal matter carried in wind, rain, by birds, insects (Grainger and Jenkins 1996). Wild dogs/dingoes have also been implicated in transmission to cattle (refs within Grainger and Jenkins 1996). Hydatids in livestock results in production losses (Fleming et al 2001); it can for instance lead to condemnation of infected organs, affecting the live cattle trade with southeast Asia (Lymberry et al 1995). Economic loss due to condemnation of cattle organs was estimated to be $6 million in northern Queensland in 2004 (Banks et al 2006).

Sylvatic isolates of \( E.\ granulosus \) are not genetically distinct from human/domestic isolates, and this has public health implications (Hope et al 1992). Hydatidosis can cause mortality or high morbidity in humans (Fleming et al 2001). Accidental contact with wild dogs or contaminated faeces presents the main public health risk (Jenkins and Morris 2003), with encroachment of wild dogs into Aboriginal communities and urban areas presenting a threat (McFarlane 1988, Lymberry et al 1995, Allen 2006, Jenkins 2006, Jenkins et al 2008). \( E.\ granulosus \) can also be passed to domestic dogs through feeding infected offal (Jenkins 2006), perpetuating the cycle.

\( Taenia\ ovis \) (\( Cysticercus\ ovis \) in the cyst-forming stage) is a tapeworm carried by wild dogs that causes sheep measles and condemnation of sheep and goat meat (Fleming et al 2001): one report claimed condemnation of up to 70% of lambs sent for slaughter, assumed from tapeworms from dogs and/or foxes (AHSQ 2002, 7.2). Lungworm (\( Oslerus\ osleri \)) and whipworm (\( Trichurus\ vulpis \)) are other helminths found that can seriously affect young dogs (Fleming et al 2001).

The protozoan parasite \( Neospora\ caninum \), causing major reproductive failure in cattle, has been shown to be transmitted from oocytes in wild dogs’ faeces (Reichel 2000, Allen and Fleming 2003, Innes et al 2005). An infection prevalence of 15% was found in Queensland beef cattle and corresponded to wild dog distribution (Landmann and Taylor 2003). \( N.\ caninum \) is also widespread in dairy cattle in Australia, causing abortion storms (Reichel 2000). It has been estimated to cost the dairy and beef industry $110 million per year (Reichel 2000).
Zoonotic Giardia protozoa were identified in 3% of urban dingoes studied by Allen (2006) in southeast Queensland. Zoonotic parasites found in high prevalences in wild dogs in a study near Townsville include: Dirofilaria immitis heartworm (at 75% prevalence), Ancylostoma caninum hookworms (90%) and Dipylidium caninum (59%) (Brown and Copeman 2003). Amblyomma sp, Spirometra erinacei and Haemaphysalis sp. were other zoonoses found in the study (Brown and Copeman 2003).

Sarcoptic mange is widespread among dingo and wild dog populations (Fleming et al 2001, Allen 2006), and although not usually debilitating to dogs, the Sarcoptes scabiei mite can seriously affect native wildlife such as wombats (Skerratt 2004, 2005).

4. Concluding remarks

This report has demonstrated a large variety of pathogenic bacteria, viruses, and parasitic helminths and protozoa are carried by invasive animals in Australia. Many of these agents present significant health threats to wildlife, domestic animals and people. According to Gortazar et al (2007): ‘One area that causes severe concern to authorities is diseases largely under control in domestic populations but still existing as a reservoir in wildlife.’

Although the pathways and likelihood of disease transmission are beyond the scope of this review, transmission could occur under the ‘right’ circumstances: from feral populations in high-risk areas (eg in close proximity to livestock, or in coexistence in high densities with wildlife species), or where other environmental conditions become favourable (eg from a change in climate, land use, cultural behaviour, etc).

Various tools have been developed to examine disease occurrence or transmission by looking at interactions between neighbouring herds (Ward et al 2007), comparing different disease control strategies (Smith and Cheeseman 2002) and assessing complexities of disease emergence and spread including biological, ecological and societal factors (Bridges et al 2007). Overseas studies that may contain relevant lessons for Australia include the role of feral mammals on wildlife infectious disease prevalence in nature reserves (Suzan and Ceballos 2004) and the potential of treating foxes in urban areas with anthelmintics to control the spread of Echinoccus tapeworms (Eckert and Deplazes 2004).

Although management of disease in invasive animals of Australia was not comprehensively searched for this review, some relevant studies were found on feral pig population genetics and behaviour (Hampton et al 2004; Cowied et al 2006, 2008a) and on potential use of baits for vaccine delivery (Cowied et al 2008b). Data on abundance and distribution of key invasive animals have also been collected nationally (West 2008). Risk factors were assessed with swine dysentery in piggeries in a Western Australian study, highlighting preventative measures and management practices on farm (Robertson et al 1992). Mason and Fleming (1999) looked at the use of hunters in exotic disease surveillance and recommended tapping into existing exotic disease surveillance programs to educate groups who are in regular contact with wildlife. This approach may be useful for endemic diseases as well. Existing programs relevant to disease surveillance in Australia include the:

- Wildlife Event Investigations Team — supporting a national approach to disease investigation that should enhance Australia’s surveillance capability
• National Significant Disease Investigation Program — standardising reporting of disease investigations by private practitioners
• National Animal Health Surveillance Strategy — covering surveillance requirements to demonstrate Australia’s animal health status and prioritising areas where there may be human health impacts.

Further research should improve disease contingency measures by defining operational management units and targeting control strategies to identified high-risk areas. It will need the integration of veterinary, ecology and wildlife management expertise and should not be limited to reporting disease: management options should also be proposed (Gortazar et al 2007). It may support conservation measures such as eradication or control of feral animals in high-risk areas, vaccination programs or other strategies. Surveillance, research and education will need to be continued and expanded for best disease management outcomes.
5. References


Caley P (year?). Masters Thesis.


