Pathogens in vertebrate pests in Australia

Wendy Henderson
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Wendy R. Henderson

Invasive Animals Cooperative Research Centre
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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).

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Summary

This review provides an overview of diseases or pathogens that have been identified in vertebrate pests 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 fauna include *Toxoplasma gondii* (causing toxoplasmosis), *Echinococcus granulosus* (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 and *Angiostrongylus cantonensis* (causing neurological disease) and various gastrointestinal pathogens such as *Salmonella*. 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 vertebrate pests 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 in Australia. The occurrence of such a wide range of pathogens emphasises the need to effectively manage populations of vertebrate pests to avoid the spread of disease into livestock, native species or humans.
1. Introduction

Vertebrate pests 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 expensive control efforts and threatening Australia’s trade reputation. The threat of pest animals transmitting exotic disease is also very real and requires constant vigilance.

This review focuses on diseases or pathogens that have been identified in key vertebrate pests in Australia; particularly those that could have a significant impact on the health of Australian agricultural, domestic or native animals, or people. Species covered include: feral pigs, foxes and wild dogs, feral cats, feral goats, rabbits, rodents (mice and rats), cane toads and European carp. The review is a companion to a previous IA CRC review: ‘Research on wildlife disease preparedness in Australia’ (Henderson 2008), which primarily focuses on the potential of pest 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 (eg positive antibody or PCR assays). The majority of the published literature described here dates between 1990 and 2009. It includes specific scientific research, incidental findings from surveillance activities, and reports of endemic disease and outbreaks.

The review is intended to provide a simple overview of potentially infectious agents identified in the above-mentioned key invasive species, highlighting possible threats to Australian fauna, livestock and society. Invasive birds, such as starlings, sparrows and Indian mynas are not included. The review does not cover details of the epidemiology of diseases. The likelihood of pathogen transmission is also beyond the scope of this review.

Two IA CRC milestones are addressed by this review, under the goal of ‘Reduced risk of economic losses, environmental damage and social stress by forecasting and responding to potential, new or emerging invasive animal problems’:

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

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 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 or from 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 include:
- published pest animal management guides (from Bureau of Rural Sciences)
- reports from the Northern Australia Quarantine Survey
- Australian Wildlife Health Network newsletters
- Communicable Diseases bulletins

3. Results

Table 1 lists the main pathogens reported in the key pest animals (cane toads, carp, feral cats, feral goats, feral pigs, foxes, rabbits, rodents and wild dogs) in Australia.

Table 1: Pathogens and parasites identified in vertebrate pests in Australia

<table>
<thead>
<tr>
<th>Animal</th>
<th>Pathogen/parasite</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>cane toad (Bufo marinus)</td>
<td>bacteria: <em>Salmonella</em></td>
<td>Thomas et al 2001</td>
</tr>
<tr>
<td></td>
<td>fungi: <em>Batrachochytrium dendrobatidis</em></td>
<td>Berger et al 2000</td>
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<tr>
<td></td>
<td><em>Mucor amphibiorum</em></td>
<td>Speare et al 1994, 1997</td>
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<td></td>
<td><em>Basidiobolus haptosporus Drechsler</em></td>
<td>Zahari et al 1990</td>
</tr>
<tr>
<td></td>
<td>mite (Lawrencarus dornowi)</td>
<td>Speare 1990</td>
</tr>
<tr>
<td></td>
<td>viruses: ranaviruses</td>
<td>Zupanovic et al 1998</td>
</tr>
<tr>
<td>feral cat (Felis catus)</td>
<td>bacteria: <em>Bartonella henselae</em></td>
<td>Branley et al 1997</td>
</tr>
<tr>
<td></td>
<td>Campylobacter upsaliensis* and C. jejuni*</td>
<td>Baker et al 1999</td>
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<tr>
<td></td>
<td><em>Clostridium perfringens</em> +/- 'faecal coliforms' +/-</td>
<td>Ferguson 2005, Cox et al 2005</td>
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<tr>
<td></td>
<td>fungus: <em>Cryptococcus gattii</em></td>
<td>Malik et al 1992</td>
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<td></td>
<td>helminths: <em>Dirofilaria immitis</em></td>
<td>Kendall et al 1991</td>
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<td></td>
<td>viruses: 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></td>
<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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<td>Animal</td>
<td>Pathogen/parasite</td>
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<tr>
<td>feral goat</td>
<td>bacteria: <em>Corynebacterium ovis</em>*/+</td>
<td>Batey et al 1985, Parkes et al 1996</td>
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<tr>
<td>(Capra hirsus)</td>
<td><em>Coxiella burnetti</em></td>
<td>Parkes et al 1996</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><em>Leptospira</em></td>
<td>Parkes et al 1996</td>
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<td></td>
<td><em>Mycobacterium paratuberculosis</em></td>
<td>South Australian Goat Advisory Group</td>
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<td></td>
<td><em>Pseudomonas pseudomallei</em></td>
<td>Choy a 2000</td>
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<td></td>
<td><em>Yersinia</em>*/+</td>
<td>Parkes et al 1996</td>
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<td></td>
<td>ectoparasites: arthropods (eg lice)</td>
<td>Parkes et al 1996</td>
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<tr>
<td></td>
<td>helminths: (22 nematode spp, 2 cestode spp, 2 trematode spp)</td>
<td>Parkes et al 1996</td>
</tr>
<tr>
<td>feral pig</td>
<td>bacteria: <em>Actinobacillus</em></td>
<td>Pavlov et al 1992</td>
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<tr>
<td>(Sus scrofa)</td>
<td><em>Aeromonas hydrophylia</em></td>
<td>Pavlov et al 1992</td>
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<td></td>
<td><em>Bacillus</em></td>
<td>Pavlov et al 1992</td>
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<tr>
<td></td>
<td><em>Brachyspira hyodysenteriae</em>, <em>Brachyspira pilosicoli</em></td>
<td>Phillips et al 2009</td>
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<tr>
<td></td>
<td><em>Burkholderia pseudomallei</em></td>
<td>Phillips et al 2009</td>
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<td></td>
<td><em>Chromobacterium freundii</em></td>
<td>Pavlov et al 1992</td>
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<td></td>
<td><em>Chromobacterium violaceum</em></td>
<td>Pavlov et al 1992</td>
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<td></td>
<td><em>Enterobacter agglomerans</em></td>
<td>Pavlov et al 1992</td>
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<td></td>
<td><em>Enterobacter cloacae</em></td>
<td>Pavlov et al 1992</td>
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<td></td>
<td><em>Escherichia coli</em>*/+</td>
<td>Pavlov et al 1992</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><em>Klebsiella pneumoniae</em></td>
<td>Pavlov et al 1992</td>
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<td></td>
<td><em>Lawsonia intracellularis</em></td>
<td>Phillips et al 2009</td>
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<td></td>
<td><em>Mycobacterium bovis</em></td>
<td>Pavlov et al 1992</td>
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<td></td>
<td><em>Proteus</em></td>
<td>Pavlov et al 1992</td>
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<td></td>
<td><em>Providencia alcifaciens</em></td>
<td>Pavlov et al 1992</td>
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<td></td>
<td><em>Pseudomonas pseudomallei</em></td>
<td>Pavlov et al 1992</td>
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<td></td>
<td>rickettsiae Spotted Fever Group*</td>
<td>Pavlov et al 1992</td>
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<td></td>
<td><em>Salmonella anatum</em>, <em>S. typhimurium</em></td>
<td>Li et al 2007</td>
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<td></td>
<td><em>Serratia liquefaciens</em></td>
<td>Bensink et al 1990</td>
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<td></td>
<td><em>Serratia marcescens</em></td>
<td>Pavlov et al 1992</td>
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<td></td>
<td><em>Staphylococcus aureus</em></td>
<td>Pavlov et al 1992</td>
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<td></td>
<td><em>Streptococcus</em></td>
<td>Pavlov et al 1992</td>
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<td></td>
<td>ectoparasitic ticks <em>Amblyomma</em> spp and <em>Ixodes australiensis</em>, pig lice</td>
<td>Pavlov and Edwards 1995, Heise-Pavlov and Heise-Pavlov 2003</td>
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<td></td>
<td><em>Hematopinus suis</em> and mange mite</td>
<td>Heise-Pavlov and Heise-Pavlov 2003</td>
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<td></td>
<td><em>Stephanurus dentatus</em>, lungworm</td>
<td>Heise-Pavlov and Heise-Pavlov 2003</td>
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<td><em>Metastrongylus</em> sp, liver fluke <em>Fasciola hepatica</em>, thorny headed worm</td>
<td>Hampton et al 2006</td>
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<td></td>
<td><em>Macracanthorhynchus hirudinaceus</em>, and <em>Taenia hydatigena</em></td>
<td>Spratt and Pavlov 1996</td>
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<td></td>
<td>protozoa: <em>Giardia</em>, <em>Cryptosporidium</em>, <em>Balantidium</em> and <em>Entamoeba</em></td>
<td>Heise-Pavlov and Heise-Pavlov 2003</td>
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<td></td>
<td><em>Toxoplasma</em></td>
<td>AHSQ 1998</td>
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<td>Animal</td>
<td>Pathogen/parasite</td>
<td>Reference</td>
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<tr>
<td>fox (Vulpes vulpes)</td>
<td><strong>viruses</strong>: arboviruses</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>Murray Valley encephalitis virus*</td>
<td>Pavlov et al 1992, Choquenot et al 1996</td>
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<td></td>
<td>Porcine parvovirus</td>
<td>Pavlov et al 1992</td>
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<td></td>
<td>Ross River virus</td>
<td>Pavlov et al 1992</td>
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<td></td>
<td>Sindbis virus*</td>
<td>Johansen et al 2005</td>
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<td></td>
<td>Trubanaman virus*</td>
<td>Johansen et al 2005</td>
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<tr>
<td></td>
<td><strong>bacteria</strong>: <em>Clostridium perfringens</em></td>
<td>Ferguson 2005, Cox et al 2005</td>
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<tr>
<td></td>
<td>faecal coliforms-*/-+</td>
<td>Ferguson 2005, Cox et al 2005</td>
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<tr>
<td></td>
<td>ectoparasites: ticks, lice, mange mite, fleas*</td>
<td>Saunders et al 1996</td>
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<td></td>
<td><strong>protozoa</strong>: <em>Giardia</em></td>
<td>Ferguson 2005</td>
</tr>
<tr>
<td></td>
<td><strong>virus</strong>: Trubanaman virus*</td>
<td>Johansen et al 2005</td>
</tr>
<tr>
<td>rabbit (Oryctolagus cuniculus)</td>
<td><strong>viruses</strong>: <em>Murray Valley encephalitis virus</em></td>
<td>Pavlov et al 1992, Choquenot et al 1996</td>
</tr>
<tr>
<td>and mice (Mus musculus, M. domesticus)</td>
<td><strong>protozoa</strong>: <em>Cryptosporidium</em></td>
<td>Ferguson 2005, Cox et al 2005</td>
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<tr>
<td></td>
<td><strong>helminths</strong>: <em>Angiostrongylus cantonensis</em></td>
<td>Spratt 2005, Stokes et al 2007</td>
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<tr>
<td></td>
<td><em>Leptospira</em></td>
<td>O’Neill 2003, AB CRC ^</td>
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<tr>
<td></td>
<td><em>Mycoplasma pulmonis</em></td>
<td>Singleton et al 2005</td>
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<td></td>
<td><em>Streptobacillus moniliformis</em></td>
<td>Taylor et al 1994, Singleton et al 2005</td>
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<td></td>
<td><strong>protozoa</strong>: <em>Cryptosporidium parvum</em></td>
<td>Singleton et al 2005</td>
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<td></td>
<td><em>Neospora caninum</em></td>
<td>Barratt et al 2008</td>
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<td></td>
<td><strong>viruses</strong>: <em>Mouse adenovirus, coronavirus,</em></td>
<td>Smith et al 1993, Singleton et al 1993, Singleton et al 2005</td>
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<td></td>
<td><em>adenovirus (K87), hepatitis virus, parvovirus,</em></td>
<td>Singleton et al 2005</td>
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<td></td>
<td><em>roevirus, rotavirus, rota virus (EDIM), minute virus of mice and Sendai – mice</em></td>
<td>Singleton et al 2005</td>
</tr>
<tr>
<td></td>
<td>*Gan Gan virus – rats</td>
<td>Singleton et al 2005</td>
</tr>
<tr>
<td></td>
<td><em>Mouse mammary tumour virus</em></td>
<td>Barratt et al 2008</td>
</tr>
<tr>
<td>wild dog/dingo (Canis lupus familiaris and C. l. dingo)</td>
<td><strong>bacteria</strong>: <em>Anaplasma platys</em></td>
<td>Brown et al 2006</td>
</tr>
<tr>
<td></td>
<td>and <em>C. coli</em></td>
<td>Allen 2006</td>
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<td></td>
<td><em>Leptospira interrogans</em></td>
<td>Zwijnenberg et al 2008</td>
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<tr>
<td></td>
<td><em>Salmonella</em></td>
<td>Allen 2006</td>
</tr>
<tr>
<td></td>
<td>ectoparasites: ticks, lice, fleas*</td>
<td>Fleming at al 2001</td>
</tr>
</tbody>
</table>

Pathogens in vertebrate pests in Australia 8
Animal Pathogen/parasite Reference

hookworms incl *Ancylostoma caninum*, lungworm, heartworm, roundworm incl *Capillaria* sp*, tapeworm incl *Spirometra* sp* and whipworm *Trichurus vulpis*

protozoa: *Babesia canis vogeli* Brown et al 2006

*Giardia* Allen 2006

*Isospora* sp coccidia Allen 2006

*Neospora caninum* Allen and Fleming 2003


Canine distemper virus McFarlane 1998, Fleming et al 2001

Canine coronavirus McFarlane 1998

Canine parvovirus McFarlane 1998

parainfluenza virus McFarlane 1998

* zoonotic pathogens; */+ potentially zoonotic


Notes: Pathogens were identified by seropositivity (antibody assay), faecal sample analysis (eg bacterial culture or PCR assay) or direct observation of the pathogen or disease in the animals. Scientific names are as cited in the references, but organisms may have since been reclassified. For example, *Pseudomonas pseudomallei* is now called *Burkholderia pseudomallei*. Serological tests were assumed for the purposes of this review to be sensitive and specific enough to accurately detect actual exposure to the pathogen.

### 3.1 Cane toads

A list of pathogens reported in cane toads in Australia is in Table 1.

**Bacteria:**
Cane toads from northern New South Wales have been reported 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 (OIE 2007). Toads are also known to carry *Leptospira* in the United States, although this bacterium has not been reported in toads in Australia (Speare 1990).

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

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

In Australia, recent research on pathogens in cane toads has largely focussed on searching for a biological control. *Rhabdias pseudosphaerocephala*, a South American species of lungworm, was reported in cane toads in Australia (Dubey and Shine 2008).

**Protozoa:**
Some of the South American protozoa recorded in *Bufo marinus* also occur in the toad in Australia, including *Trichomititus batrachorum*, *Zelleriella antilliensis*, *Hyalodaktylethra renacauno* and *Myxidium immersum* (Delvinquier and Freeland 1988). *M. immersum* myxosporidia are gall bladder
protozoa also found in a range of Australian frog species, believed to have been introduced from cane toads (Delvinquier 1986). Cane toads are known to carry \textit{Toxoplasma} in other countries, although this parasite has not been identified in toads in Australia (Speare 1990).

**Fungi:**

The chytrid fungus \textit{Batrachochytrium dendrobatidis} (causal agent of chytridiomycosis) has been identified in cane toads in Australia (Berger et al 2000). This fungus is lethal to a broad range of amphibians, and has been responsible for the serious decline of frog species. Infected frogs have been found from 46 Australian species, including 9 of 15 threatened and 6 of 12 vulnerable species (OIE 2006). Infection of amphibians with chytrid fungus resulting in chytridiomycosis has been declared a key threatening process under the \textit{Environment Protection and Biodiversity Conservation Act 1999}.

\textit{Basidiobolus haptosporus} Drechsler fungus, which causes human and animal disease in the tropics, has been reported 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, \textit{Mucor amphibiorum}, has been reported in cane toads in Queensland, New South Wales and Northern Territory (Speare et al 1994, 1997), probably 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 \textit{M. amphibiorum} showed that Tasmanian isolates of \textit{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 indicate 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).

**Ectoparasites:** The only mention of a parasitic arthropod in cane toads in Australia was of a mite, \textit{Lawrencarus dornrowi} (Speare 1990).

### 3.2 European carp

The only pathogen reported in wild European carp in Australia is the Asian fish tapeworm (see Table 1 and discussion below).

**Bacteria:**

The bacterium \textit{Aeromonas salmonicida} has been reported in farmed carp in Australia (Wiklund and Dalsgaard 1998). \textit{A. salmonicida} is recognised internationally as a serious pathogen of fish; commercial (eg 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 carry this disease agent.

**Viruses:**

Koi Herpes virus (subsequently renamed Cyprinid herpesvirus-3, CyHV-3) is a significant pathogen of carp industries overseas; it is being researched as a possible biological agent for naïve European carp populations in Australia (McColl et al 2007). It is a highly contagious viral disease causing significant morbidity and mortality in common carp, but other related cyprinid species such as goldfish and grass carp are unaffected by the virus (Gilligan and Rayner 2007).
Helminths:
Carp in Australia carry the Asian fish tapeworm, *Bothriocephalus acheilognathi* (Dove et al 1997, Dove and Fletcher 2000, Koehn et al 2000), which could seriously threaten the health of native fish. Carp are suspected to have introduced this broad host range parasite to Australian waters: the distribution of *B. acheilognathi* matches that 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), and 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 infected with *B. acheilognathi* (Dove et al 1997).

3.3 Feral cats

A list of pathogens reported in feral cats in Australia is in Table 1. A major survey of the diseases and parasites of domestic cats in Australia revealed over 100 species of pathogens (Moodie 1995: unpublished report referenced in Dickman 1996). Although many of these are cat-specific, at least 30 of the pathogens have also been reported 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 of 59 feral cats studied in Sydney; more than twice the prevalence reported in domestic cats tested (Branley et al 1997). Although this bacterium generally produces a mild infection in people who have been bitten or scratched, infection 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 gastrointestinal disease in Australia in 1996 — and *Campylobacter* species have also been associated with purulent arthritis and Guillain-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. Another report of pathogenic bacteria in feral cats was 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 affect species other than cats, they may pose a health threat to domestic cats.

**Helminths:**

Feral cats are 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 (Milstein and Goldsmid 1997).

Many helminth parasites have been reported in feral cats 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). Most common helminths include *Abbreviata hastaspicula, Ancylostoma* spp, *Cylicospirura felineus, Oncicola pomatostomi, Spirometra erinaceieuropaei* (also called *S. erinacei*), *Taenia taeniaeformis, Physaloptera praeputialis* and *Toxocara cati* (Barton and McEwan 1993, O’Callaghan and Beveridge 1996, Adams 2003, O’Callaghan et al 2005, Adams et al 2008). Some parasites were presumed to have transferred from native birds, reptiles and mammals eaten by cats; for example, *O. pomatostomi* is
likely transferred from birds and *Abbreviata hastaspicula* from varanid lizards (O'Callaghan and Beveridge 1996, O'Callaghan et al 2005).

Many of the helminths reported 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). *S. erinaceieuropaei* tapeworm, common in feral cats in eastern Australia, can also severely affect a wide range of native species (Dickman 1996).

**Protozoa:**
The most common protozoan parasites reported 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, Adams et al 2008). *Cryptosporidium*, considered a pathogen of mammals, birds and reptiles, has been reported in feral cats of the Northern Territory (O'Callaghan et al 2005) and Western Australia (Adams 2003).

*T. gondii* is arguably 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.

Signs of toxoplasmosis in native fauna include poor coordination, blindness, lethargy, respiratory and enteric distress (such as diarrhoea), and sudden death (Canfield et al 1990). Signs of *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 (*Thylологale billardieri*) in Tasmania were reported with blindness due to severe chorioretinitis associated with *Toxoplasma* sp infection (OIE 2001). Several reports were found of eastern brown bandicoots (*Perameles gunnii*) and southern brown bandicoots (*Isoodon obesulus*) being severely affected by toxoplasmosis, contributing to population decline (Lenghaus et al 1990, Obendorf et al 1996, OIE 2001). Ten out of 150 eastern barred bandicoots in Tasmania had antibody that indicated they had been infected by *T. gondii* (Obendorf et al 1996). *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 have also been reportedly infected by *Toxoplasma* — freshwater runoff contaminated with cat faeces has been blamed (ScienceDaily 2008).

*T. gondii* was identified in about 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 in feral cats on Christmas Island (Adams et al 2008). In Western Australia, a study of 379 cats identified *T. gondii* in only 4.9% (Adams 2003). Even if only a few cats are shedding *T. gondii* oocysts at any given time, enormous numbers may be produced and their resistance to destruction enables widespread contamination of the environment (Eyman et al 2006). There is no vaccine to control toxoplasmosis in humans, cats, or wild animals, and treatment options are very limited.
Fungi:
A significant fungus reported in cats (at least in ‘rural’ and domestic cats) is *Cryptococcus gattii* (previously *C. neoformans var. gattii*) (Malik et al 1992, O’Brien et al 2004). This organism causes cryptococcosis, a fungal disease affecting people (Speed and Dunt 1995) and a large range of native mammals, birds and reptiles (Krockenberger et al 2005). *C. gattii* can lead to respiratory and central nervous system problems —often fatal in cats (Duncan et al 2006 and references therein), infection in people tends to require surgery and prolonged treatment (Speed and Dunt 1995). It is carried by animals including cats, dogs, horses and goats, probably transferred from environmental sources such as certain eucalypts (Ellis and Pfeiffer 1990, Krockenberger et al 2005). Although direct transmission from animals has not been recorded (Krockenberger et al 2005), it is possible that infected cats could provide a source of environmental contamination.

3.4 Feral goats

A list of pathogens reported in feral goats in Australia is in Table 1. Feral goats are prone to a number of diseases currently in Australia, including Q fever, tetanus, leptospirosis, hydatids, pulpy kidney and blackleg (Biosecurity Queensland 2007).

Bacteria:
Of significant concern to human health is the bacterium *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 *C. burnetti* is considered the most infective organism in the world, with people being capable of becoming infected by 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 at Waikerie in South Australia, where a cluster of Q fever cases (one of which was fatal) were linked to inhalation of contaminated dust from the local abattoir, affecting townsfolk not involved in meat preparation (Pedler 2007, ABC News 10/9/20071).

Meliodosis, caused by the zoonotic bacterium *Burkholderia pseudomallei* (reclassified from *Pseudomonas pseudomallei*), is 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). Melioidosis is an emerging disease in people, associated with a high morbidity and mortality, particularly in Indigenous Australians (Short 2002, Cheng et al 2003). Another zoonotic bacterium reported in feral goats is *Corynebacterium ovis*, which causes caseous lymphadenitis (abscesses in lymph nodes) (Batey et 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). A report of *Mycobacterium bovis* infection in a goat was found, although this involved 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 nationally notifiable animal diseases. While no specific articles were found reporting occurrence of Bovine Johne’s Disease (BJD) and Ovine Johne’s Disease, risks from these chronic wasting diseases being endemic in feral goats in South Australia are mentioned (South Australian Goat Advisory Group meeting2). 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...

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sale. This disease is a concern to the livestock industries generally. There is speculation that human Crohn's disease may be caused by *Mycobacterium avium* subspecies *paratuberculosis*, the same agent that causes Johne's disease (Greenstein 2003).

**Viruses:**
Caprine arthritis/encephalitis virus infection has been reported in feral 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 sporadic. It can lead to chronic disease of the joints and, on rare occasions, encephalitis in goat kids.

**Helminths:**
Feral goats are known to carry 22 nematode, two cestode, two trematode, four arthropod and three 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 worms (Rural ABC May 2008). A link has been suggested between feral goats and the occurrence of hydatid tapeworms in cattle in the Kimberley region of Western Australia, where cattle populations were uninfected in previous surveys (Lymbery et al 1995).

**Protozoa:**
Enteric coccidiosis, an economically important parasitic disease particularly of neonatal domestic goats, has been reported 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 *Eimeria* sp, and that stress associated with transport contributed to severe coccidiosis and death (Main and Creeper 1998).

### 3.5 Feral pigs

Feral pigs host a wide range of pathogens; a list compiled from the literature is in Table 1. Some of these pathogens are specific to pigs, such as classical swine fever, and others can affect a wide range of species. Many of the pathogens cause significant zoonosis, including *Leptospira*, *Brucella*, *Mycobacterium*, Ross River Virus and Murray Valley encephalitis virus (Pavlov et al 1992). Analyses of feral pig populations (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 infection 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 Weil’s disease in humans, also known as "canecutters’ 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 nationally 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 *L. interrogans* serovars have been reported 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, reported in up to half the pigs that have been examined (Choquenot et al 1996, Mason et al 1998). This serovar is a threat to the health of livestock, and hunters and other outdoor

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4 http://www.goatworld.com/articles/cae/cae-waddl.shtml
5 http://www.abc.net.au/rural/news/content/200805/s2242964.htm
recreational groups (Mason et al 1998). An sv 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 reported in pigs (Milner et al 1981, Mason et al 1998). This serovar is the predominant serovar infecting people in temperate regions such as Sydney (Eymann et al 2006). Leptospirosis was suspected to be a significant contributor to a lower-than-expected pig population density reported in tropical rainforest in north-eastern Australia (Heise-Pavlov and Heise-Pavlov 2003).

The bacterium *Brucella suis* is endemic in feral pigs in central Queensland (Mason and Fleming 1999a), serving as a source of infection of domestic/commercial pigs and cattle. These authors reported 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 1999a). It has also not been detected in Western Australia or Northern Territory (Choquenot et al 1996). Brucellosis is a nationally 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 endemic 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 1999a). The incidence of human brucellosis is on the rise in Queensland (Robson et al 1993), and a recent case also occurred in the Hunter Valley region of New South Wales (Hunter New England Population Health 2006): all these people were involved with killing feral pigs. The threat to people’s health is increasing with the growth of the lucrative pig hunting industry (Robson et al 1993).

*Streptococcus suis* is another occupational hazard for piggery workers, with two recent cases reported in New South Wales (Kennedy et al 2008) — although these cases did not specify feral pig involvement.

Other bacteria associated with feral pigs include Spotted Fever Group rickettsia in ticks, reported to be endemic in south-west Western Australia (Li et al 2007). *Salmonella anatum* and *S. typhimurium* were reported in a third of 154 feral pig carcasses processed for human consumption (Bensink et al 1990). Pathogenic enteric bacteria *Lawsonia intracellularis* (in 19% of 222 pigs analysed), *Brachyspira hyodysenteriae* (in 8%) and *Brachyspira pilosicoli* (in 0.45%) were reported in feral pigs in Western Australia (Phillips et al 2009). *Pseudomonas pseudomallei* (since reclassified as *Burkholderia pseudomallei*) was reported in two thirds of feral pigs tested in north Queensland (Pavlov and Edwards 1995); melioidosis appears commonly in wet weather in the Top End (AHSQ 2001, 6.4).

Tuberculosis caused by *Mycobacterium bovis* has rarely been reported in feral pigs since the national bovine tuberculosis eradication campaign (BTEC) in cattle and buffalo was completed in 1997. *M. bovis* was reported 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 have also been reported in pigs (Pavlov et al 1992, Caley 1993, Caley et al 1994). Porcine parvovirus (PPV) antibodies were 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 can cause reproductive failure in piggeries — although it is vaccine preventable.

Antibody to the highly infectious and zoonotic Menangle virus was reported in pigs in several New South Wales piggeries, but there was no evidence of the virus in 190 feral pigs in the Northern Territory (Kirkland et al 2001). Menangle disease causes reproductive disorders in pigs, and influenza-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 et al
According to Animal Health Australia, there has been only one outbreak of Menangle disease in Australia, occurring in 1997. The disease is a nationally notifiable animal disease.

Another virus amplified in pigs is Japanese encephalitis virus (JEV), a nationally notifiable animal disease vectored by mosquitoes. In 1998, antibodies to JEV were detected in sentinel pigs on Cape York Peninsula, and a fisherman in that area contracted a JEV infection (Hanna et al 1999, Exotic Animal Diseases Bulletin 2003). These were the only known cases of JEV occurring on the Australian mainland until 2004, when sentinel pigs and feral pigs in Cape York showed serology patterns consistent with exposure to the virus (AHSQ 2006, 11.2; Van den Hurk et al 2006) and JEV was isolated from mosquitoes in the area (van den Hurk et al 2006). A study in northern Australia reported that mosquitoes preferentially feed from marsupials rather than pigs (only 9% fed from pigs; van den Hurk et al 2003). This observation suggests that feral pigs may not play a major role in JEV transmission in Australia, despite being a significant amplifier of the virus in Asia.

Another virus reported in feral pigs is Trubanaman virus (Johansen et al 2005). Antibody to this mosquito-borne virus was reported in 3.5% of feral pigs tested in south-western Western Australia (Johansen et al 2005). It is suspected as a cause of polyarthritis in people (Boughton et al 1990).

Helminths:
Hydatid cysts from the tapeworm *Echinococcus granulosus* were reported in 9% (Banks et al 2006a) 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 reported in about half the pigs examined in the Kosciuszko region of New South Wales (Jenkins and Morris 2003). These results show that feral pigs can sylvatically cycle this parasite. *E. granulosus* was also reported in feral pigs in Western Australia, where they were involved in a 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 been reported at high infection rates in feral pigs (Pavlov and Edwards 1995, Heise-Pavlov and Heise-Pavlov 2003). Sparganosis, caused by zoonotic *Spirometra* tapeworms, was reported in high prevalence in pigs of northern Queensland (Pavlov et al 1992): this parasite can infect people who eat inadequately cooked pork.

Protozoa:
Toxoplasmosis occurs in feral pigs, with the national serological prevalence for *Toxoplasma* protozoa estimated at 9.3% (AHSQ 1998 Vol 3.1). Other protozoan parasites that present human health risks, such as *Giardia, Cryptosporidium, Balantidium* and *Entamoeba* spp, were detected in faeces from feral pigs caught in metropolitan drinking water catchments in Western Australia (Hampton et al 2006). The pigs aid in transmission of diseases directly through faecal contamination of water, and turbidity from pig wallowing may also protect waterbourne pathogens from chemical disinfection (Hampton et al 2006).

Fungi:
Feral pigs carry and spread the fungus *Phytophthora cinnamomi* that causes dieback disease in native flora (Pavlov et al 1992). Infection of native plants by *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). A list of pathogens reported in foxes is in Table 1.

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

**Viruses:**
A low prevalence of Canine herpes virus has been reported in foxes (Reubel et al 2004). Trubanaman virus has also been reported (Johansen et al 2005); this virus possibly causes polyarthritis in people (Boughton et al 1990). Canine distemper virus has also been reported (K. Rose, Taronga Zoo, personal communication).

An interesting overseas study on predator-mediated spread of viral disease reported Avian influenza virus H5N1 in foxes that had eaten infected bird carcasses (Reperant et al 2008). The foxes could excrete the virus while remaining free of severe disease (Reperant et al 2008). This article highlights the potential for feral carnivores in Australia to transmit disease in a similar way.

**Helminths:**
*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 reported 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 health 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 an emerging threat (Jenkins 2006, Jenkins et al 2008).

Other helminth parasites reported in foxes include the zoonotic heartworm *Dirofilaria immitis*, in 6.4% of 125 foxes in Melbourne (Marks and Bloomfield 1998), and in 9% of 68 foxes near Sydney (Mulley 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).

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

**Ectoparasites:**
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 scurvy mange, is commonly carried by foxes, and seriously affects wombats and possibly other native wildlife (Skerratt 2004, 2005).

**Fungi:**
The only fungus reported in foxes is ringworm (*Microsporum*) (Saunders et al 1996).

### 3.7 Rabbits

A list of pathogens reported in rabbits in Australia is in Table 1. Most search results returned recent literature on rabbit haemorrhagic disease. The majority of the pathogens present a disease threat to domestic or commercially bred rabbits, although some zoonotic organisms were reported, 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 or RHDV, 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 (Cooke et al 2002, Strive et al 2009).

Antibody to zoonotic arboviruses Sindbis virus (Sindbis fever causes fever and malaise in people) and Trubanaman virus (possibly causing polyarthritis in people, Boughton et al 1990) have been reported in rabbits, at 0.8% and 2.4% prevalence respectively (Johansen et al 2005).

**Helminths:**
Various 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).

**Protozoa:**
The intestinal pathogen *Cryptosporidium* has been reported in faecal samples of rabbits (Cox et al 2005).

### 3.8 Rats and mice

A variety of pathogens and parasites have been reported in mice and rats in recent literature; a 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 reported in black rats (*Rattus rattus*), Norway rats (*R. norvegicus*) and mice (*Mus musculus* and *M. domesticus*) are discussed below.

**Bacteria:**
*Streptobacillus moniliformis* was reported in wild mice in south-east Australia (Taylor et al 1994, Singleton et al 2005). *S. moniliformis* is reported in 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 factsheet7, 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 commonly 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* was also reported in mice (Singleton et al 2005).

**Viruses:**
Mouse mammary tumour virus (MMTV) was reported in mice in south-eastern 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 people. LCMV was identified by serology in 9.6% of mice tested in north-eastern 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. The possibility was raised that ‘introduction of Mus domesticus and its viruses into Australia’s indigenous small mammal populations that had probably been isolated from such viruses for many thousands of years, cleared the way for house mice to dominate the cereal growing regions’ (Smith et al 1993).

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 years8).

**Helminths:**

One of the most significant parasites reported 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 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 south-eastern 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.

Protozoa:

A zoonotic protozoan parasite reported 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 protozoa such as Cryptosporidium and Giardia for transmission to people and other mammals.

Neospora caninum, a 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).

Other:

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.

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### 3.9 Wild dogs

A list of pathogens and parasites reported associated with wild dogs is in Table 1. The most significant of these organisms 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 south-east 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 reported on free-roaming dogs associated with Aboriginal communities (Brown et al 2006).

**Viruses:**

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

**Helminths:**

The most significant pathogenic helminth carried by wild dogs (the definitive hosts) is probably *Echinococcus granulosus*. The hydatid 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). Burdens of more than 10,000 worms are common, with 100,000 worms regularly detected (Jenkins 2006). With such large worm burdens, a low number of infected dogs can maintain a high transmission rate (Jenkins and Morris 1991).

High prevalence of hydatid infection has also been reported in native species, especially macropods: for example New South Wales studies reported 40% prevalence in macropods in the Bondo State Forest (Jenkins and Morris 1991), and up to 69% prevalence in wallabies in Kosciuszko National Park (Jenkins and Morris 2003). The main wildlife transmission cycle of *E. granulosus* is perpetuated 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).

*E. granulosus* infection affects wildlife, livestock and human health in Australia. Hydatids disease has serious consequences for wildlife, particularly macropods (Jenkins 2005, 2006). Infected native animals often have multiple cysts in the lungs, particularly affecting their ability to run from predators (Jenkins and Morris 1991). Viable hydatid 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 is reportedly due to 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 and insects (Grainger and Jenkins 1996). Wild dogs/dingoes have also been implicated in transmission of hydatids to cattle (see Grainger and Jenkins 1996 and references within). Hydatids in livestock results in production losses (Fleming et al 2001); it can for instance lead to condemnation of infected organs, affecting offal sale and live cattle trade with south-east 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 2006b).
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 caused by tapeworms from dogs and/or foxes (AHSQ 2002, 7.2). Lungworm (*Oslerus osleri*) and whipworm (*Trichurus vulpis*) are other helminths reported that can seriously affect young dogs (Fleming et al 2001).

*Zoonotic helminths reported in high prevalences in wild dogs in a study near Townsville include: Dirofilaria immitis* heartworm (at 75% prevalence), *Ancylostoma caninum* hookworm (90%) and *Diplydium caninum* tapeworm (59%) (Brown and Copeman 2003). *Spirometra erinacei* (also called *S. erinaceieuropaei*) is another zoonotic helminth reported in wild dogs (Brown and Copeman 2003).

**Protozoa:**

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 reported 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 south-east Queensland.

**Ectoparasites:**

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). *Amblyomma* and *Haemaphysalis* ticks reportedly found on wild dogs can cause disease in people (Brown and Copeman 2003).

### 4. Concluding remarks

This report has described a large variety of pathogenic bacteria, viruses, helminths and protozoa in vertebrate pests in Australia. Many of these agents present significant health threats to people, domestic animals and native species. According to Gortazar et al (2007): ‘One area that causes severe concern...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: for instance, 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 (eg for foot-and-mouth disease; 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 (Suzán and Ceballos 2004) and the potential of treating foxes in urban areas with anthelmintics to control the spread of *Echinococcus* tapeworms (Eckert and Deplazes 2004).

Although literature on management of disease in vertebrate pests in Australia was not comprehensively searched for this review, some relevant studies were found on feral pig population genetics and behaviour (Hampton et al 2004; Cowled et al 2006, 2008a) and on potential use of baits for vaccine delivery (Cowled et al 2008b). Data on abundance and distribution of key vertebrate pests have been collected nationally and this should assist in targeting management strategies (West 2008). Risk factors for swine dysentery in piggeries were assessed in a Western Australian study, highlighting the need for effective preventative management practices on-farm (Robertson et al 1992). Mason and Fleming (1999b) 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:

- 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
- National Significant Disease Investigation Program — standardising reporting of disease investigations by private practitioners
- Wildlife Event Investigations Team — supporting a national approach to disease investigation that should enhance Australia's surveillance capability.

The need for enhancements to Australia's animal health system has been recognised by the Commonwealth government, including the need to redefine the science–policy interface and refine risk analysis, surveillance and diagnostics (Black et al 2008). Further research should improve disease contingency measures by defining operational management units, identifying high-risk areas and assessing different disease control options. It may provide support for particular management strategies and conservation measures such as eradication of feral animals in high-risk/high-value areas, or vaccination programs. Research will need the integration of veterinary, ecology and wildlife management expertise and resulting literature should not be limited to reporting disease or pathogen occurrence: ideally, implications for management should also be included to improve the science–policy interface. Research and education should be supported by government and industry sectors, and will need to be continued and expanded for best disease management outcomes.

### 5. Acknowledgements

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6. References


