

# Q fever in Australian wildlife

## Fact sheet

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### Introductory statement

*Coxiella burnetii* is the causative agent of Q fever, a highly contagious zoonotic disease. Within Australia, native and feral wildlife species implicated as likely reservoirs include bandicoots, kangaroos, small rodents, feral goats and possibly pigeons (Derrick et al. 1939; Pope et al. 1960; Beveridge 1981; Garner et al. 1997). A great deal of information is available on bacteriology of the organism, transmission, clinical manifestation of disease and laboratory diagnosis, but further research needs to be undertaken to define significant wildlife reservoirs of *C. burnetii* and the role they play in transmission to domestic reservoirs in Australia.

### Aetiology

In 1935, an outbreak of a fever of unknown aetiology in several abattoir workers in Brisbane, Queensland was termed “Q” fever, or “Query” fever (Derrick 1937; Sidky 1950). Although the organism was initially described as being of rickettsial origin, it is now clear that the organism differs from typical *Rickettsiae*, and has been reclassified as *Coxiella burnetii*.

*C. burnetii* is a pleomorphic, obligate intracellular Gram-negative bacterium (Family *Coxiellaceae*, genus *Coxiella*). Gene sequence analysis has led to placement of the *Coxiella* genus within the gamma subdivision of the proteobacteria in the order *Legionellae*, family *Coxiellaceae* (Seshadri and Paulsen 2003).

*C. burnetii* is capable of displaying various developmental cycle variants, including a large-cell variant (LCV), small-cell variant and small dense cell (SDC). The LCV and SDC survive extracellularly as infectious, spore-like particles. In this state, *C. burnetii* is highly resistant to a variety of environmental conditions (Heinzen et al. 1999). *C. burnetii* also exhibits two antigenic forms. Pathogenic Phase I, isolated from infected individuals and avirulent Phase II, isolated after serial passage in cell culture or chick embryo yolk sac (Quevedo Diaz and Lukáčová 1998; Hotta et al. 2002).

### Natural hosts

Whilst sheep, goats, cattle and domesticated animals have traditionally been associated with transmission of *C. burnetii*, the organism is known to infect mammals, birds, fish, reptiles and arthropods, namely ticks (Arricau-Bouvery and Rodolakis 2005; The Center for Food Security and Public Health 2007). No age or sex

linked predisposition in wildlife hosts have been reported. Within Australia, native and feral wildlife species implicated as likely reservoirs include bandicoots, kangaroos, small rodents, feral goats and possibly pigeons (Derrick et al. 1939; Pope et al. 1960; Beveridge 1981; Garner et al. 1997). Infection with *C. burnetii* is widespread among feral goats (seroprevalence of 52% in one study) in Australia (Parkes et al. 1996). It is usually non-pathogenic in goats.

Overseas, *C. burnetii* has been isolated from rabbits and a high antibody prevalence has been reported (Enright et al. 1971), yet no published studies have implicated these animals as significant reservoirs in Australia. The same situation is true for coyotes (*Canis latrans*) and grey foxes (*Urocyon cinereoargenteus*) in the USA (Enright et al. 1971). Surprisingly, no research has been undertaken on dingoes and foxes in Australia. Feral cats are unlikely to act as a natural host (Enright et al. 1971).

## World distribution

The only region considered to be free from *C. burnetii* is the Antarctic (Woldehiwet 2004; Arricau-Bouvery and Rodolakis 2005) and possibly New Zealand (Hilbink et al. 1993).

## Occurrences in Australia

Q fever occurs widely throughout Australia, with residents in southern Queensland, northern and western NSW reporting higher levels of infection (Garner et al. 1997). Though limited surveys have been performed in Australian wildlife (below), it is likely that Australian wildlife species will have been exposed wherever there is evidence of human or production animal infection.

## Epidemiology

Whilst infection in wildlife is more likely to stem from direct contact with contaminated fomites, such as faeces or birth products, a tick-vertebrate-tick cycle also exists. A wide variety of native marsupials and rodents show serological evidence of infection (Glazebrook et al. 1978), but clinical disease is mild or inapparent. All species infected with *C. burnetii* may act as carriers of the organism, shedding intermittently over prolonged periods of time in urine, faeces, colostrum and milk. Shedding is heaviest at parturition.

A recent study conducted in south-west and central Western Australia found sera from 115 of 343 western grey kangaroos (*Macropus fuliginosus*), that were tested by ELISA to be positive for Q fever antibodies ((Banazis et al. 2010). A second study confirmed the role of western grey kangaroos in Q fever epidemiology with 24.1% of 1017 serum samples testing positive by ELISA and 4.1% of 990 kangaroo faecal samples testing positive for *Coxiella burnetii* DNA by PCR (Potter et al. 2011).

A third study found a seroprevalence of 13% of 200 macropods sampled from southern Queensland, 30.4% of 92 macropods from northern Queensland, 3.6% of 28 macropods from western Queensland and 22.8% of 180 macropods from Western Australia. When separated by species the greatest number of positive results were found in eastern grey kangaroos (*Macropus giganteus*), western grey kangaroos, common wallaroos (*Macropus robustus*) and black-striped wallabies (*Macropus dorsalis*) (Cooper et al. 2012).

Research undertaken at Murdoch University has shown that the western grey kangaroo is a likely reservoir host of *C. burnetii* (Banazis et al. 2010). The importance of the role that this wildlife species plays in transmission of *C. burnetii* should not be underestimated. Ticks, a natural vector of the bacterium, are frequently found on kangaroos. These reservoirs are possibly responsible for contaminating pastures by

shedding the bacterium in faeces, resulting in transmission to domestic stock during grazing. Due to the asymptomatic nature of infection in wildlife, it is likely that a large number of unknown species play a similar role.

Overseas, morbidity and mortality are considered to be extremely low in wildlife species as they are thought to be asymptomatic reservoirs. The incubation period is considered variable (The Center for Food Security and Public Health 2007). Although not reported in published literature, morbidity and mortality rates in Australian wildlife are likely to be similarly low due to the asymptomatic nature of *C. burnetii* infection in these species.

## Clinical signs

Most wildlife species do not exhibit clinical signs of infection, although they are capable of shedding large numbers of the organism. Experimental infection in bandicoots (*Isodon macrourus torosus*), a likely reservoir host in Australia, produced no clinical signs of disease or febrile response (Derrick et al. 1939). However, one of two rufous bettongs (*Aepyprymnus rufescens*) experimentally infected with *C. burnetii* died with splenomegaly and focal hepatic necrosis (Derrick et al. 1940). In mammals, predominantly sheep, goats and cattle, natural infection in naïve animals can lead to abortion, stillbirth, retained placenta, endometritis, infertility and small or weak offspring (Arricau-Bouvery and Rodolakis 2005).

## Diagnosis

Diagnosis can be made through a combination of PCR, serology (immunofluorescence assay, complement fixation and enzyme-linked immunosorbent assay) and microscopic examination of infected tissue smears although routine diagnosis is most commonly achieved using immunological methods.

## Clinical pathology

Infected wildlife species develop serum agglutinating and complement-fixing antibodies against Phase I and II antigens (Derrick et al. 1939; Pope et al. 1960; Enright et al. 1971; Berri et al. 2001). Whilst livestock species remain seropositive for a number of years following acute infection (The Center for Food Security and Public Health 2007), it is unknown whether antibodies persist in Australian wildlife reservoirs.

## Pathology

Given the asymptomatic nature of infections in most wildlife species, gross pathological changes are not common. Splenic enlargement was reported in two experimentally infected bandicoots (*I. macrourus torosus*) (Derrick et al. 1939) and splenomegaly and focal hepatic necrosis in 1 of 2 experimentally infected rufous bettongs (Derrick et al. 1940). In domestic mammals exhibiting clinical signs, predominantly cattle, sheep and goats, pathology characteristically involves placentitis, with thickening, a white-yellow, creamy exudate or reddish-brown fluid. Severe vasculitis and pneumonia may be noted, but less commonly (The Center for Food Security and Public Health 2007) while lesions associated with the infection in pregnant mice are characterised by necrosis of placental tissues and microthrombosis (Baumgartner and Bachmann 1992).

As *C. burnetii* is an obligate intracellular bacterium, invasion and growth within eukaryotic cells is an essential factor of virulence (Arricau-Bouvery and Rodolakis 2005). Minimal cytopathic effects are noted in infected cells (Heinzen et al. 1999). Microscopic examination of stained, infected tissue will reveal a large number of thin, coccobacillary bacteria (OIE 2008).

## Differential diagnoses

Differential diagnoses have not been described for wildlife species. For domestic stock exhibiting reproductive disease, *Chlamydophila* and *Brucella* should be ruled out (Arricau-Bouvery and Rodolakis 2005; OIE 2008).

## Laboratory diagnostic specimens

Serum requires clotted whole blood collection in tubes permitting serum separation. Ideally, an acute and convalescent sample should be collected to detect a four-fold increase in antibody titre. A minimum of 500 µl is required for CFT diagnosis although the ELISA requires only 50 µl (Peter and Dupuis 1987). Detection of the organism requires samples to be taken from the placenta, vaginal discharge, aborted foetus (liver, lung or stomach contents), milk, faeces and urine (The Center for Food Security and Public Health 2007; OIE 2008). In asymptomatic animals, diagnosis is likely to require serum and faecal samples only. Approximately 1g of faeces is required for PCR. Samples should be frozen at -20°C as soon as possible.

## Laboratory procedures

Live *C. burnetii* requires a PC3 laboratory due to the hazardous nature of working with the organism. Until recently it has not been able to be cultivated on host cell-free media (Omsland and Cockrell 2009) and its isolation is long and difficult (Arricau-Bouvery and Rodolakis 2005). Despite being the OIE reference test for *C. burnetii*, complement fixation is weakly sensitive and detects seroconversion later than other serology tests. Immunofluorescence is not commonly used for diagnosis of Q fever in animals due to its subjective nature and inconvenience in large-scale screening. ELISA has a high level of sensitivity and good specificity, being easy to perform and favoured over the IFA and CF tests as it allows a greater number of samples to be tested at once (OIE 2008). It is important to recognise that serological evidence does not correlate with shedding of the organism. Confirmed diagnosis should couple serology with positive isolation of the organism.

Traditional and real-time PCR can be used to detect *C. burnetii* in milk, colostrum, aborted material and faeces. In wildlife, being asymptomatic carriers, faecal material will produce the best results. The advantage of this method is that samples can be heat inactivated, ensuring their safety within the laboratory. PCR is the most rapid and sensitive way of detecting animals who are shedding. Combining an ELISA with faecal PCR is the most effective method of diagnosing *C. burnetii* in wildlife species.

## Treatment

*C. burnetii* is responsive to antibiotics in humans, but little is known of the efficacy in animals (The Center for Food Security and Public Health 2007). Treatment with prophylactic antibiotics is commonly used in domestic stock in an attempt to reduce the number of abortions and shedding at parturition. Whilst it is unlikely that free ranging wildlife will receive treatment, those infected animals held in captivity may require antibiotics to prevent further transmission. In human cases of clinical disease doxycycline is most effective for acute infection. Chronic infection requires prolonged antibiotic treatment for two or more years (OIE 2008).

## Prevention and control

As Q fever is an air-borne disease, prevention is aimed at minimising exposure to animal and environmental contamination. Many of the methods of prevention and control are currently aimed at cattle, sheep and

goats. Whilst not directly related to wildlife, these measures assist in preventing transmission to free ranging species found to mix with domestic stock.

Infected manure from contaminated paddocks should never be spread to uninfected grounds. Lime or 0.4% calcium cyanamide should be used to treat animal droppings and reduce the level of environmental contamination, minimising transmission to wildlife species grazing on the same pasture. Regular disinfection of animal facilities must be undertaken for similar reasons. Quarantining of new animals will reduce chances of Q fever being introduced to the original, uninfected herd. Pregnant animals need to be separated due to increased likelihood of shedding and any placenta/aborted foetus must be removed immediately to prevent ingestion by domestic cats, dogs and wildlife. It is unknown if wildlife (foxes/ wild dogs) have a significant role to play in the epidemiology of the disease in Australia. Pasteurisation of milk is essential and particularly important in preventing transmission to people (OIE 2008).

A vaccination program for cattle in Slovakia in the 1970's and 1980's was successful in reducing the occurrence of Q fever in that country but this approach has not been widely adopted due to cost and technical factors involved in vaccine production (Arricau-Bouvery and Rodolakis 2005). A vaccination program in people received approval in Australia in 1989 and is currently used to immunise individuals occupationally exposed to Q fever. A national Q fever management program was initiated in 2001 in Victoria, South Australia and Queensland. The program was designed to screen and vaccinate people in an attempt to reduce the burden of disease. Information is available on the Department of Health and Ageing website. Given kangaroos and bandicoots are likely to play a significant role in transmission of the bacterium, (Australian Technical Advisory Group on Immunisation 2007) has advised that anyone exposed to these animals should be vaccinated. Those particularly at risk include individuals involved in the kangaroo harvesting industry, wildlife carers, zoo and wildlife park employees.

Veterinary laboratory staff, particularly those involved in necropsy work and isolation of the organism, should consider vaccination. The vaccine can have severe local reactions in previously exposed individuals. So prior assessment of humoral and cellular responses to the organism are essential. Detailed advice on vaccination is available at [www.qfever.org](http://www.qfever.org).

#### Surveillance and management

Wildlife disease surveillance in Australia is coordinated by the Wildlife Health Australia. The National Wildlife Health Information System (eWHIS) captures information from a variety of sources including Australian government agencies, zoo and wildlife parks, wildlife carers, universities and members of the public. Coordinators in each of Australia's States and Territories report monthly on significant wildlife cases identified in their jurisdictions. NOTE: access to information contained within the National Wildlife Health Information System dataset is by application. Please contact [admin@wildlifehealthaustralia.com.au](mailto:admin@wildlifehealthaustralia.com.au).

Q fever is notifiable in humans within Australia, monitored by the National Notifiable Diseases Surveillance System (NNDSS). The Laboratory Virus and Serology Reporting Scheme (LabVISE) also collates data on the laboratory diagnosis of Q fever. It is also a notifiable disease by the OIE.

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A recent Queensland study examined blood, faeces and urine from a range of species for the presence of *Coxiella burnetii* DNA by PCR. Of the blood samples tested negative results were obtained for the bilby, both dolphins, three kangaroos, one possum, one quoll, three reptiles and seven wombats. However, three of the 26 koalas tested were positive. Faecal results were positive for one of the 43 koalas tested but none of the four wallabies. Urine samples were positive for seven of the 90 flying foxes, and one of the 30 koalas, but the three possum and wombat samples and the single wallaby sample were all negative (Tozer et al. 2014).

## Research

Whilst livestock have traditionally been associated with transmission of Q fever, Derrick et al. (1939) proposed that marsupials were potentially a significant reservoir host of *C. burnetii*. This stemmed from an extensive study involving experimental infection of bandicoots (*I. macrourus torosus*), with the organism. Animals appeared to be susceptible to infection, developing antibodies, but did not develop clinical signs (Derrick et al. 1939). Pope et al. (1960) subsequently found a number of macropod species to be seropositive to *C. burnetii*. Out of 270 animals tested, 18% were found to be positive for complement-fixing antibodies, agglutinating antibodies or both. Red kangaroos (*Macropus rufus*) had a higher prevalence of complement fixing antibodies at 33%, compared to eastern grey kangaroos at 12%. Isolation of the organism was also achieved in one animal (*M. major*), suggesting that systemic infection does occur in kangaroos. The incidence of CF antibodies was similar in both males and females. Significantly, isolation of *C. burnetii* was achieved from 13 kangaroo ticks (*Amblyomma triguttatum*) and four of these were found on goats and sheep. *A. triguttatum* is a 3-host tick and thus may be able to act as a vector between the different host species (Pope et al. 1960).

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It is important to characterise the relationship between domestic and wildlife cycles of *C. burnetii* in Australia. Research in this area may provide valuable information relating to the zoonotic threat that marsupial coxiellosis poses and assist in epidemiological studies tracing human outbreaks of Q fever. Furthermore, defining the pathology of *C. burnetii* infection in Australian marsupials would clarify any tissue tropism that occurs which could have implications in regard to disease transmission risk factors and provide clues as to the overlap between wild and domestic reservoirs.

## Human health implications

The clinical manifestation of infection with *C. burnetii* is Q fever. It 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 2008). Human infection with *C. burnetii* takes on one of three forms; asymptomatic (60%), acute (flu-like, pneumonia, hepatitis) and chronic (endocarditis, post-Q fever fatigue syndrome) (Arricau-Bouvery and Rodolakis 2005). Resistant spores are able to survive for a long period of time within the environment, providing a constant source of infection. The most common route of infection is via inhalation of contaminated aerosols as well as contact with viscera, blood, afterbirth and faecal material of animals known to carry infection and ingestion of non-pasteurised milk products (Beveridge 1981). A review of Q fever in Australia (1991 – 1994) found that a pronounced male bias exists in infection due to an occupational association with livestock and meatworks (Garner et al. 1997).

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 townfolk not involved in meat preparation (Pedler 2007) (Henderson In prep.).

## Conclusions

Q fever is a highly infectious, zoonotic disease considered to be re-emerging in many countries around the world. In Australia, it is occupationally associated with individuals working closely with livestock and abattoirs. A great deal of information is available on bacteriology of the organism, transmission, clinical manifestation of disease and laboratory diagnosis, but further research needs to be undertaken to define significant wildlife reservoirs of *C. burnetii* and the role they play in transmission to domestic reservoirs. The limited work that has been performed does, however, suggest that Australian wildlife play a role in the epidemiology of this disease and need to be considered in any management activities.

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## To provide feedback on this fact sheet

We are interested in hearing from anyone with information on this condition in Australia, including laboratory reports, historical datasets or survey results that could be added to the National Wildlife Health Information System. If you can help, please contact us at [admin@wildlifehealthaustralia.com.au](mailto:admin@wildlifehealthaustralia.com.au).

Wildlife Health Australia would be very grateful for any feedback on this fact sheet. Please provide detailed comments or suggestions to [admin@wildlifehealthaustralia.com.au](mailto:admin@wildlifehealthaustralia.com.au). We would also like to hear from you if you have a particular area of expertise and would like to produce a fact sheet (or sheets) for the network (or update current sheets). A small amount of funding is available to facilitate this.

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