Q fever in Australian wildlife

Fact Sheet

April 2025

Key points

- *Coxiella burnetii* is the causative agent of Q fever, a highly infectious and serious zoonotic disease. Q fever is a notifiable human disease in all Australian states and territories (see *Surveillance and management*).
- People at greatest risk of contracting Q fever are those in close contact with cattle, sheep and goats, which are considered the most important reservoir species for the transmission of Q fever to humans.
- Studies have shown that Australian wildlife species can also become infected (mostly sub-clinically) with *C. burnetii.*
- In Australia, there has been a steady increase in the number of Q fever notifications attributed to sources other than livestock, with Australian native wildlife (particularly kangaroos and wallabies) implicated as potential sources of human infection.
- Australian wildlife may potentially be a source of environmental contamination (through bacteria shed in their faeces or birth material) and Q fever vaccination is recommended for people in close contact with kangaroos and bandicoots [1].

Aetiology

Coxiella burnetii is an obligate intracellular Gram-negative bacterium (Family *Coxiellaceae*, genus *Coxiella*). Many strains of *Coxiella*-like organisms have been described in ticks [2]. These organisms appear to be important for the health of ticks and are not considered to be pathogenic to vertebrates [3].

One Health implications

Wildlife and the environment: *C. burnetii* does not appear to cause disease in most Australian wildlife species and it is thought unlikely to have environmental or population level impacts, in spite of the fact that many species are susceptible to infection (see below) and may potentially be reservoir species. Recent studies suggest a possible link between *C. burnetii* and poor reproduction in Australian fur seals (*Arctocephalus pusillus doriferus*) [4].

Domestic species: cattle, sheep and goats, are considered the most important reservoir species for the transmission of Q fever to humans and occasionally suffer from reproductive-related disease. Cats and dogs may occasionally be infected.

Humans: Q fever is considered the most infectious disease in the world and is a serious zoonotic disease.

Natural hosts

Coxiella burnetii can infect an extensive range of vertebrate and invertebrate hosts including wild and domestic mammals, birds and arthropods [5-7].

Domestic ruminants (cattle, sheep and goats) are considered to be the primary source of *C. burnetii* infection for humans with the majority of Q fever outbreaks worldwide being linked to direct or indirect exposure to these species or their products [8, 9]. Infected ruminants shed the organism in high concentrations during parturition via birth products and fluids [10], resulting in massive environmental contamination. Infected animals also shed the pathogen in their milk, vaginal secretions, urine and faeces [11]. Other non-traditional domestic animal species that may be a source of *C. burnetii* for humans include cats and dogs [12, 13].

Infection and/or exposure to *C. burnetii* has been identified in over 100 species of **wild mammals** worldwide [14]. In Europe, *C. burnetii* infection has been described in wild ungulates, carnivores, lagomorphs and wild birds [15]. The wild European rabbit (*Oryctolagus cuniculus*) population has been suggested as a potential reservoir of *C. burnetii* infection for humans and livestock in Europe [16]. Evidence of *C. burnetii* infection has also been reported in wild rodents in Canada [17] and wild rodents and foxes in the UK [18], while in French Guiana the three-toed sloth (*Bradypus tridactylus*) has been identified as a reservoir of a highly virulent strain of *C. burnetii* [19]. Infection has also been described in the northern hemisphere in a range of marine mammal species, both cetaceans and pinnipeds [20-23].

In Australia, infection with *C. burnetii* is widespread among feral goats (seroprevalence of 52% in one study) [24]. Up to 10% seroprevalence has been reported in feral rabbits [25], and it has been suggested that feral pigs may also represent a potential source of *C. burnetii* infection for humans and other animals [26].

Infection has been reported from placentas of Australian fur seals from a Bass Strait colony and it has been suggested that there may be a link with poor reproductive success in this species [4].

As far back as the 1930s, it was proposed that Australian marsupials were potentially a significant reservoir host of *C. burnetii*, stemming from an extensive study involving experimental infection of *C. burnetii* in northern brown bandicoots (*Isoodon macrourus torosus)* [27]. Bandicoots appeared to be susceptible to infection, developed antibodies, but did not develop clinical signs. A number of macropod species were subsequently found to be seropositive to *C. burnetii*. Of 270 animals, 18% were 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 eastern grey kangaroo), suggesting that systemic infection can occur in kangaroos as it does in humans. Significantly, *C. burnetii* was isolated from 13 kangaroo ticks (*Amblyomma triguttatum*) and four of these were found on goats and sheep. *Amblyomma triguttatum* is a 3-host tick and it was suggested that it may act as a vector between the different host species [28].

Since these early studies, serological and molecular evidence of *C. burnetii* infection in Australian wildlife has been demonstrated in a range of studies, summarised in Tables 1 and 2 respectively (see *Appendix*).

World distribution

Coxiella burnetii has been reported in animals and humans world-wide with the exception of New Zealand [29] and the Antarctic [30, 31].

Occurrences in Australia

Q fever cases have been reported from people living in all states of Australia, however the eastern states of NSW and Qld account for most annual human Q fever notifications [32]. Adult males working in traditional high risk industries such as abattoir and meat processing, farming, shearing and livestock transportation [32-36] account for a high proportion of Q fever notifications, with slaughtering and assisting with animals births considered particularly high risk activities [34, 37].

Evidence of *C. burnetii* infection has been reported in Australian wildlife from NSW, Qld, Vic and WA (see Tables 1 & 2) however it is likely that Australian wildlife species in other states and territories will also have been exposed to *C. burnetii*, given the known distribution of human or production animal infection.

Epidemiology

Q fever is considered the most infectious disease in the world [5, 38]. *Coxiella burnetii* is highly infectious to humans [39] with the inhaled infectious dose for humans as low as one to 15 organisms [33]. Human infection with *C. burnetii* takes on one of four forms; asymptomatic, acute (flu-like, pneumonia, hepatitis), chronic (endocarditis, vasculitis, osteomyelitis) and the post-Q fever fatigue syndrome [30]. A pronounced male bias exists in infection, partly due to an occupational association with livestock and meat works [32] but also the protective effect of female hormones, as shown in many animal species.

Coxiella burnetii exists as two distinct developmental cycle variants: large-cell variants (LCVs) and small-cell variants (SCVs). The LCVs are the metabolically active intracellular form of *C. burnetii.* In contrast, SCVs are the highly infectious spore-like forms, which exist outside the cell in the environment, and are resistant to a variety of environmental conditions [40]. *Coxiella burnetii* also exhibits two antigenic forms based on their lipopolysaccharide structure known as Phase 1 and Phase 2.

Inhalation of contaminated aerosols or dust is the most common route through which *C. burnetii* is transmitted to humans [8, 9, 41] and is likely also to be the most common route for animals, although tick-transmission is probably also important in animals. People may become infected via direct contact with tissue, excreta, and fluids from infected animals, particularly during slaughtering or parturition [42]. Infection may also occur indirectly via inhalation of contaminated dust released from wool [43], clothing [44] and manure used as fertiliser [45].

The *C. burnetii* isolated from Australian fur seals is a marine-adapted genotype that is molecularly distinct from terrestrial Australia genotypes, but similar to genotypes found in northern hemisphere marine mammals [46]. Pacific gulls (*Larus pacificus*) may be involved in the spread of *C. burnetii* in Australian fur seal colonies, as they scavenge seal placentas and have been found to carry and shed the bacterium.

Owing to the ability of *C. burnetii* to persist for extended periods in soil [42] and withstand harsh environmental conditions, infectious aerosols may remain long after environmental shedding by infected animals. Environmental *C. burnetii* has the potential to be spread over large distances in contaminated dust in dry and windy conditions. Windborne spread of *C. burnetii* has been attributed to Q fever outbreaks in France [47] the UK [48], the Netherlands [49] and Australia [50].

Coxiella burnetii was originally isolated from a *Dermacentor andersoni* tick in the 1930s and it has since been reported that over 40 tick species are naturally infected with *C. burnetii* [5, 51]. However the role of ticks in the epidemiology of Q fever is poorly understood [2].

A recent survey found that Australian wildlife rehabilitators (AWRs) were almost twice as likely to be seropositive to *C. burnetii* compared to the general Australian population [52]. Unvaccinated AWRs were also more likely to self-report having had medically diagnosed Q fever [53]. Rehabilitators who self-reported medically diagnosed Q fever were more likely to be > 50 years of age and to rehabilitate wildlife whilst working in a veterinary clinic and/or have domestic ruminants residing on the property where they rehabilitated wildlife. These findings indicate that the traditionally identified exposure routes such as livestock and contaminated environment were also potential sources of infection for these people.

Clinical signs

Humans

Between 20 and 80% of *C. burnetii* infections in humans are asymptomatic [9] with this figure varying with geographical region, likely due to variations in the virulence of strains of *C. burnetii*. However, *C. burnetii* infection can manifest as a serious illness with long term health consequences regardless of the initial clinical presentation [9]. Following an incubation period of two to five weeks, acute Q fever manifests as a self-limiting influenza-like illness, characterised by high-grade fevers, chills, severe headache, fatigue and muscle aches [33]. Patients may also experience complications including hepatitis and pneumonia [9, 54]. Other sequelae may occur following both asymptomatic and symptomatic infection, including persistent focal infections (formerly chronic Q fever), most commonly endocarditis [9], and post Q fever fatigue syndrome [55].

Animals

Coxiella burnetii infection in animals (known as coxiellosis), appears to be sub-clinical in most cases. However coxiellosis has been associated with reproductive disorders manifesting as placentitis, abortion, still birth and weak offspring in ruminants (cattle, sheep, goats, deer) [5, 56, 57] and other mammals including cats [13], dogs [12], fur seals [21] and sea lions [20].

Infections in Australian fur seals may be linked to poor reproductive success [4].

The only studies of experimental infection with *C. burnetii* in Australian wildlife were undertaken over 70 years ago. One in northern brown bandicoots produced no clinical signs or fever [27]. In the second study, one of two rufous bettongs (*Aepyprymnus rufescens*) experimentally infected with *C. burnetii* died with splenomegaly and focal hepatic necrosis [58].

Diagnosis

Diagnosis of Q fever in humans is usually achieved via serology and/or polymerase chain reaction (PCR) performed by specialist human diagnostic pathology laboratories www.health.gov.au/resources/publications/q-fever-cdna-national-guidelines-for-public-health-units.

Coxiellosis is not routinely diagnosed in animals, given the largely subclinical nature of the infection in animals, and testing for *C. burnetii* in animals is mainly undertaken for research purposes or epidemiological studies. Investigations have occasionally been undertaken in animals associated with outbreaks of Q fever in humans. Combining serology with faecal PCR is likely to be the most effective method of detecting *C. burnetii* in wildlife species [59].

Culture

Microbiological culture of *C. burnetii* is rarely performed due to its slow growing nature and the requirement for physical containment level 3 (PC3) facilities resulting from the hazardous nature of working with the organism [60]. In Australia culture is undertaken by the Australian Rickettsial Reference Laboratory, based at Geelong Hospital, Vic.

Serology

Antibody against *C. burnetii* has been detected in humans and animals via the complement fixation test (CFT), enzyme-linked immunosorbent assay (ELISA) and the immunofluorescence assay (IFA). Serology requires clotted whole blood collection in tubes permitting serum separation. A minimum of 500 µl of serum is required for CFT diagnosis although the ELISA and IFA may require as little as 50 µl [61].

CFT is highly specific but poorly sensitive and detects seroconversion later than other serological tests [38].

IFA is considered the gold standard test for diagnosis of Q fever in humans and has been increasingly used for detection of previous exposure to *C. burnetii* in animals [62]. The main disadvantage of IFA is its subjectivity (requiring experience in reading IFA slides) and inconvenience in large-scale screening.

ELISA has a variable sensitivity and specificity, depending on which commercial assay is used. It is easy to perform and is favoured over the IFA and CFT because of its convenience for screening large numbers of animal samples [38].

Serological positivity does not necessarily correlate with shedding of viable organism and samples collected during early infection may produce negative results due to the lag in antibody response. Antibody against phase 2 antigen arises earlier than that against phase 1 so ideally, serological assays should be directed against both phases if available, and an acute and convalescent sample should be collected to detect a rise in antibody titre.

Molecular methods

PCR is the most rapid and sensitive way of detecting animals that are shedding the organism and will detect both viable and non-viable bacteria (but does not distinguish between dead and live organisms).

Early Q fever infections in humans can be detected via traditional and real-time PCR on samples extracted from serum or buffy coat (obtained from an EDTA blood sample) as it detects the early bacteraemic phase lasting 10-14 days from the onset of clinical signs. PCR can also be performed on biopsy or tissue samples (e.g., cardiac valve postoperatively in endocarditis cases) from persistently infected individuals.

In animals, PCR can be used to detect *C. burnetii* in milk, colostrum, vaginal secretions, placentae and other products of conception from normal birth and aborted maternal material, aborted foetus (liver, lung or stomach contents), serum or buffy coat, urine and faeces [38, 63]. Approximately 1g of faeces is required for PCR. Samples should be frozen at -20°C as soon as possible and preferably -80°C if there will be a delay in processing.

Treatment

Humans: it is strongly recommended that people who are suspected of having acute Q fever seek medical assistance promptly, as treatment commenced within the first three days of symptom onset is most effective at preventing severe long-term complications [33, 64]. For more information on Q fever treatment in humans see www.health.nsw.gov.au/Infectious/factsheets/Pages/q-fever.aspx and www.health.gov.au/resources/publications/q-fever-cdna-national-guidelines-for-public-health-units.

Animals are rarely treated for coxiellosis as infection and disease are seldom identified. Tetracycline use has been reported in livestock however the lack of scientific evidence to support the efficacy of antimicrobials in controlling *C. burnetii* infections means they are not recommended [65].

Prevention and control

Humans: due to the airborne transmission of *C. burnetii* and its prolonged survival in the environment, vaccination is regarded as the best risk control measure for the prevention of Q fever in humans [1, 66, 67]. Currently, Australia is the only country with a licensed human Q fever vaccine [68], with a protective efficacy ranging from 83 to 100% [69-71]. The Australian Technical Advisory Group on Immunisation (ATAGI; https://immunisationhandbook.health.gov.au/contents/vaccine-preventable-diseases/q-fever) recommends Q fever vaccination for high-risk occupational groups including meat and livestock industry workers, professional dog and cat breeders and veterinary personnel. In 2018, recommendations were extended to wildlife and zoo workers who have contact with kangaroos and bandicoots, and people who cull or process kangaroos [1].

Details of the appropriate biosecurity guidelines for reducing the risk of contracting Q fever are available in the *National Wildlife Biosecurity Guidelines* (https://wildlifehealthaustralia.com.au/Portals/0/ResourceCentre/BiosecurityMgmt/National_Guidelines_Management_Disease_Freeranging_Aust_Wildlife_Nov_2020.pdf) issued by Wildlife Health Australia. These guidelines recommend that when interacting with wildlife, basic biosecurity

practices should be adopted at all times regardless of the perceived risks [72]. Basic biosecurity practices include regular hand washing, the appropriate management of laundry and use of personal protective equipment (PPE) such as disposable gloves and coveralls. The main biosecurity practices listed in the guidelines specifically for Q fever are: ventilation controls, dust management, P2/N95 face mask and Q fever vaccination. Although wearing a P2/N95 face mask may reduce the risk of airborne *C. burnetii* transmission, vaccination remains the most effective means of preventing Q fever in humans given the long survival time of *C. burnetii* in the air and environment [73] .

Animals: vaccines against *C. burnetii* are not currently available in Australia, although one is available for livestock in Europe [74]. Prevention and control of *C. burnetii* infection in Australian wildlife is not considered feasible.

Research

Further research is needed to define significant wildlife reservoirs of *C. burnetii*. Research to characterise the relationship between domestic and wildlife cycles of *C. burnetii* in Australia is important as it may provide valuable information relating to the zoonotic threat that marsupial coxiellosis poses and assist in epidemiological studies tracing the source of human outbreaks of Q fever. Further work is required to understand the potential pathogenic effects of *C. burnetii* infection in Australian pinnipeds [75].

Surveillance and management

Q fever is a **notifiable human disease** in all Australian states and territories (www.health.gov.au/our-work/nndss#diseases-on-the-national-notifiable-disease-list), monitored by the National Notifiable Diseases Surveillance System (NNDSS) [76].

In Australia, coxiellosis is rarely diagnosed and not notifiable in animals at the national level. Tasmania is the only Australian state in which coxiellosis is listed as a notifiable animal disease [77].

We are interested in hearing from anyone with information on this condition in Australian wildlife, including laboratory reports, historical datasets or survey results that could be added to the National Wildlife Health Information System. Negative data are also valuable. If you can help, please contact us at admin@wildlifehealthaustralia.com.au.

Wildlife Health Australia administers Australia's general wildlife health surveillance system, in partnership with government and non-government agencies. Wildlife health data is collected into a national database, the electronic Wildlife Health Information System (eWHIS). Information is reported by a variety of sources including government agencies, zoo based wildlife hospitals, sentinel veterinary clinics, universities, wildlife rehabilitators, and a range of other organisations and individuals. Targeted surveillance data is also collected by WHA. See the WHA website for more information https://wildlifehealthaustralia.com.au/Our-Work/Surveillance and https://wildlifehealthaustralia.com.au/Our-Work/Surveillance/eWHIS-Wildlife-Health-Information-System.

Appendix

Table 1. Studies reporting serological evidence of *Coxiella burnetii* exposure in Australian native wildlife Scientific names of species are listed below table (Adapted from Mathews 2022 [78]).

Australian State	Species	Sample (n)	Seroprevalence (%)	Method	Reference
WA	Western grey kangaroo	343	33.5	ELISA	Banazis et al. 2010 [59]
		1017	24	ELISA	Potter et al. 2011 [79]
	Western barred bandicoot	35	8.6	ELISA	Bennett et al. 2011 [80]
Qld and WA	Various macropod species	500	20.8	ELISA	Cooper et al. 2012 [81]
	Brushtail possum	56	10.7	ELISA	Cooper et al. 2012 [82]
	Northern brown bandicoot	46	23.9	ELISA	Cooper et al. 2012 [82]
	Northern brown bandicoot	35	31.4	ELISA	Cooper et al. 2013 [83]
	Eastern grey kangaroo	17	41.1		
	Agile Wallaby	5	60.0		
	Red kangaroo	4	-		
	Common wallaroo	3	66.2		
	Brushtail possum	2	-		
	Rufous bettong	1	-		
	Blacked-striped wallaby	1	-		
Roma, Qld	Eastern grey kangaroo and red kangaroo	50	52.7	IFA	Tolpinrud et al. 2022 [62]
St George, Qld		35	40.5		
Look at me now headland, NSW		31	81.3		
Arrawarra, NSW	Eastern grey kangaroo	20	45.7		
Heritage Park, NSW		14	57.5		
Nelson Bay, NSW		34	94.2		
Sydney water catchment, NSW		35	58.0		
Western Sydney, NSW		24	3.1		
Anglesea, Vic		30	0		
New Zealand	Red-necked wallaby	30	0		

Western grey kangaroo (*Macropus fuliginosus*), western barred bandicoot (*Perameles bougainville*), macropod species (*Macropus* spp.), brushtail possum (*Trichosurus vulpecula*), northern brown bandicoot (*Isoodon macrourus*), eastern grey kangaroo (*M. giganteus),* agile wallaby (*M. agilis*), red kangaroo (*M. rufus*), common wallaroo (*M. robustus*), rufous bettong (*Aepyprymnus rufescens*), black-striped wallaby (*M. dorsalis*), red-necked wallaby (*Notamacropus rufogriseus*), ELISA- enzyme-linked immunosorbent assay. IFA -Immunofluorescence assay

Table 2. Studies reporting molecular evidence of *Coxiella burnetii* exposure in Australian native wildlife by PCR (Adapted from Mathews 2022 [78]).

Australian region and state	Species	Sample type	Sample number (n)	PCR prev. (%)	Reference
WA	Western grey kangaroo	faeces	343	12.3	Banazis et al. 2010 [59]
WA	Western grey kangaroo	faeces	990	4.1	Potter et al. 2011 [79]
WA	Western barred bandicoot	faeces	12	8.3	Bennett et al. 2011 [80]
Qld	Northern brown bandicoot	whole blood	6/35	25.0	Cooper et al. 2013 [83]
	Eastern grey kangaroo		6/17		
	Agile wallaby		1/5		
	Red kangaroo		1/4		
	Common wallaroo		1/3		
	Common brushtail possum		1/2		
	Rufous bettong		0/1		
	Blacked-striped wallaby		1/1		
Qld	Kangaroo	blood	0/3	5.8	Tozer et al. 2014 [84]
	Koala	blood, faeces, urine	5/99		
	Flying-fox	urine	7/90		
	Wallaby	faeces, urine	0/5		
	Wombat	blood, faeces, urine	0/10		
Qld	Eastern grey kangaroo and red kangaroo	multiple tissues from same animal	50	30	Stevenson et al. 2022 [85]
Qld	Red-necked wallaby	scat	103	0	Mathews 2022 [78]
Dubbo, NSW	Eastern grey kangaroo		24	0	
Valla, NSW			44	2.2	
Camden, NSW			4	0	
Camden, NSW	Swamp wallaby		1	0	
Camden, NSW	Common wallaroo		2	0	
Camden, NSW	Common wombat		14	0	
Camden, NSW	Koala		1	0	
Campbelltown, NSW		urogenital swabs	75	0	
Lismore, NSW			75	0	
Pt Macquarie, NSW			76	1	
Canberra, ACT	Eastern grey kangaroo		51	0	
Kanowna Is, Vic	Australian fur seal	placentas	66	10.6-40.9*	Gardner et al. 2022 [4]

Western grey kangaroo (*Macropus fuliginosus*), western barred bandicoot (*Perameles bougainville*), northern brown bandicoot (*Isoodon macrourus*), eastern grey kangaroo (*M. giganteus),* agile wallaby (*M. agilis*), red kangaroo (*M. rufus*), common wallaroo (*M. robustus*), brushtail possum (*Trichosurus vulpecula*), blacked-striped wallaby (*M. dorsalis*), koala (*Phascolarctos cinereus*), flying-fox (*Pteropus* spp.), wallaby (*Macropus* spp.), common wombat (*Vombatus ursinus*), swamp wallaby (*Wallabia bicolour*); Australian fur seal (*Arctocephalus pusillus doriferus*)

* different testing techniques revealed differing prevalence

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Wildlife Health Australia recognises the Traditional Custodians of Country throughout Australia. We respectfully acknowledge Aboriginal and Torres Strait Islander peoples' continuing connection to land, sea, wildlife and community. We pay our respects to them and their cultures, and to their Elders past and present.

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