



Australian Government

Department of Health
and Aged Care

Guidance Note for medical practitioners and hospitals

Australian endemic tick-borne diseases:

- Queensland tick typhus
- Flinders Island spotted fever
- Australian spotted fever
- Q fever

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List of abbreviations

Abbreviations	Description
ACT	Australian Capital Territory
ASF	Australian spotted fever
ATAGI	Australian Technical Advisory Group on Immunisation
CDNA	Communicable Diseases Network Australia
DNA	Deoxyribonucleic acid
DSCATT	Debilitating Symptom Complexes Attributed to Ticks
FISF	Flinders Island spotted fever
GP	General practitioner
PPE	Personal protective equipment
QFS	Q fever fatigue syndrome
QTT	Queensland tick typhus
Q-VAX®	Q fever vaccine
RACGP	Royal Australian College of General Practitioners
RPE	Respiratory protective equipment
SCV	Small-cell variant
SFG	Spotted fever group
UV	Ultraviolet

About this Guidance Note

Purpose and objective

This Guidance Note is part of a series of Guidance Notes on ticks, tick-borne diseases, tick-induced allergies, and Debilitating Symptom Complexes Attributed to Ticks (DSCATT).

In response to the 2016 Senate Community Affairs References Committee's Final Report Inquiry into the growing evidence of an emerging tick-borne disease that causes a Lyme-like illness for many Australian patients, the Australian Government commissioned the development of educational and awareness materials related to DSCATT, as well as a clinical pathway and multidisciplinary care model to support clinicians' decision-making on differential diagnosis and referral pathways for patients presenting with DSCATT.

The purpose of the Guidance Notes is to provide evidence-based guidance for clinicians in community and hospital settings, as well as providing a reference source on DSCATT topics.

Topics covered in this Guidance Note

This Guidance Note covers known Australian tick-borne diseases:

- Spotted fever group *Rickettsia* transmitted by ticks:
 - Queensland tick typhus (QTT)
 - Flinders Island spotted fever (FISF)
 - Australian spotted fever (ASF)
- Q fever.

While acknowledging Australia has an almost complete set of rickettsial infections transmitted to humans, this Guidance Note does not cover scrub typhus (transmitted by mite bite), murine typhus or cat flea typhus (transmitted by flea bite or inhaled infected flea faeces).

Information on DSCATT is also included in this Guidance Note. The cause(s) of DSCATT in Australia is not yet established.

Information about the tick species associated with the Australian endemic tick-borne diseases, particularly the Australian paralysis tick *Ixodes holocyclus* is also provided in the Guidance Notes on Introduction to ticks, Australian ticks and tick-borne diseases and illnesses and Tick-induced allergies: tick anaphylaxis and mammalian meat allergy/anaphylaxis, and tick-associated toxicosis and paralysis.

This Guidance Note is based on information freely available to the public, from published peer-reviewed literature, and Australian and international guidance and guidelines, with a focus on literature published in the past 10 years. In this Guidance Note, where published peer-reviewed papers were not freely available to the public but are of high importance as they relate to the Australian situation, this literature was included. The percentages for various measures reported in this Guidance Note were included as they were reported by the authors of included literature; as such, there are a range of decimal places reported for percentages. Studies and publications cited by the authors of articles included in this Guidance Note are provided as in-text citations. This approach allows for articles published prior to the past 10 years and articles that are not freely available to the public to be acknowledged and provides easy access for readers who may wish to explore an article further.

In this Guidance Note and in the series of Guidance Notes on ticks, tick-borne diseases, tick-induced allergies and DSCATT, there is some repetition of content between the Guidance Notes and also within the Guidance Notes, where appropriate. This approach enables each Guidance Note to be read as a stand-alone document, rather than requiring the reader to read from start to finish. The repetition between sections within a Guidance Note allows the reader to read each section as a standalone section, rather than being referred to other sections within the Guidance Note. The Contents page of each Guidance Note is hyperlinked to sections within the Guidance Note to enable the reader to easily access information. Additionally, readers are also referred to other Guidance Notes in this series where additional information can be found.

A short video on [how to remove a tick](#) by killing the tick in situ with ether-containing sprays is available here:

Important! [Watch this video](#) about how to safely remove a tick¹
<https://www.allergy.org.au/patients/insect-allergy-bites-and-stings>

¹ An allergy project supported by the National Allergy Strategy, Australasian Society of Clinical Immunology and Allergy, Allergy & Anaphylaxis Australia, and Tick-induced Allergies Research and Awareness.

Overview and summary

Apart from the occasional local bacterial infection at the tick bite site (eschar) the only two systemic infections that are definitely known to be transmitted by tick bites in Australia are rickettsial infections from infection with *Rickettsia* spp. (Queensland tick typhus (QTT), Flinders Island spotted fever (FISF), and Australian spotted fever (ASF)), and Q fever (*Coxiella burnetii*).

The species of Australian ticks known to bite humans and transmit bacterial infection are:

- the **Australian paralysis tick** (*Ixodes holocyclus*), which is endemic on the east coast of Australia and causes QTT due to *Rickettsia australis* and causes Q fever due to *C. burnetii*
- the **common marsupial tick** (*Ixodes tasmani*), which occurs in New South Wales, Queensland, South Australia, Western Australia, Tasmania and Victoria and causes QTT due to *R. australis* and causes ASF due to *Rickettsia honei* subsp. *marmionii*
- the **southern paralysis tick** (*Ixodes cornuatus*), which occurs in New South Wales, Victoria and Tasmania and causes QTT due to *R. australis*
- the **ornate kangaroo tick** (*Amblyomma trigutatum*), which occurs throughout much of the central, northern and western Australia and causes Q fever due to *C. burnetii*
- the **southern reptile tick** (*Bothriocroton hydrosauri*), which occurs mainly in south-eastern Australia and causes FISF due to *R. honei*
- the *Haemaphysalis novaeguineae* (no common name) tick causes ASF due to *R. honei* subsp. *marmionii* (Barker & Walker (2014) in Dehghani et al., 2019; Graves & Stenos, 2017).

QTT, FISF and ASF have similar clinical and serological characteristics (Dehghani et al., 2019), with the *Rickettsia* spp. that cause each respective disease being members of the alpha subclass of proteobacteria (Graves, n.d.). The clinical presentation of these rickettsial infections in Australia include eschar, fatigue, fever, headache, myalgia and rash (macular, papular, vesicular) although the severity and duration of rickettsial diseases vary considerably (Dehghani et al., 2019). Early clinical features are often non-specific, making diagnosis challenging (Stewart et al., 2017a). Additionally, symptoms may overlap with other infectious diseases including those that are transmitted by non-tick vectors, as well as a number of chronic diseases.

QTT is an emerging public health threat (Dehghani et al., 2019; Stewart et al., 2017a) and an increasingly recognised important cause of community-acquired acute febrile illness in eastern Australia (Stewart et al., 2017a). Stewart et al. in 2017 noted acute *R. australis* infection is likely to be under-recognised, with recent evidence showing its increased disease burden with increased recognition of severe disease, including death (Derne et al. (2015), Laboratory Australian Rickettsial Reference (2015), Baird et al. (1992), and Unsworth et al. (2007) in Stewart et al., 2017a).²

While FISF is often described as a mild illness (Heymann (2015) in Willis et al., 2019), Willis et al. noted the infection can be severe and rare deaths have been reported, with the death of a middle-aged woman due to acute infection of *R. honei* described in Queensland (Graham et al. (2017) in Willis et al., 2019).

² Laboratory Australian Rickettsial Reference is the reference as cited by Stewart et al. 2017a. The laboratory is known as the Australian Rickettsial Reference Laboratory.

Rickettsial infections are typically seen in residents of endemic areas, as well as campers, travellers, and hikers to endemic areas (Dehghani et al., 2019). A transmission cycle of rickettsiae exists between ticks and species of vertebrates; humans are an accidental host (Baird et al. (1992) in Stewart et al., 2017a).

Currently, there is no effective vaccine against any *Rickettsia* species, in Australia (Derne et al. (2015) in Stewart et al., 2017a). Difficulties exist in creating a universal spotted fever group (SFG) rickettsial vaccine due to variation in the chemical configuration of the immunologically important outer membrane proteins (Stenos & Walker (2000), and Feng & Walker (2003) in Stewart et al., 2017a).

Tick-borne rickettsial diseases are not nationally notifiable diseases in Australia (Australian Government Department of Health, 2021). Rickettsial infection (including spotted fevers and all forms of typhus fever) is notifiable in Western Australia (Western Australian Department of Health, n.d.). Typhus (all forms) is notifiable in Northern Territory (Northern Territory Department of Health, 2016), and typhus (epidemic) is notifiable in New South Wales (New South Wales Ministry of Health, 2020). FISH is also notifiable in Tasmania (Tasmanian Department of Health, 2020).

Q fever is a nationally notifiable disease in Australia (Australian Government Department of Health, 2021) with a Q fever laboratory case definition (Public Health Laboratory Network, 2017), and it is included in the Communicable Diseases Network Australia (CDNA) National Guidelines for Public Health Units (Communicable Diseases Network Australia, 2018).

In Australia, Q fever is the most commonly reported zoonotic disease (Eastwood et al., 2018).

C. burnetii (Q fever) is not a true *Rickettsia*, despite being tick-transmitted, but is a member of the gamma proteobacteria (Graves, n.d.). Q fever is acquired via various modes of transmission, a minority of which are tick-borne. Two cases of tick-transmitted Q fever in Australia have been described in the literature (Beaman & Hung, 1989; Graves et al., 2020). Q fever vaccine (Q-VAX®) has been available in Australia since 1989, with efficacy estimated at 83% to 100% (Marmion (2009) in Communicable Diseases Network Australia, 2018). The vaccine is recommended for those at risk of infection with *C. burnetii*. The Australian Immunisation Handbook (<https://immunisationhandbook.health.gov.au/>) recommends Q fever vaccination for people aged ≥15 years who have close contact with animals, including abattoir workers, farmers, veterinarians, professional cat and dog breeders, zoo and wildlife workers and animal refuge workers.

Queensland tick typhus

QTT is an emerging public health threat (Dehghani et al., 2019; Stewart et al., 2017a) and an increasingly recognised important cause of community-acquired acute febrile illness in eastern Australia (Stewart et al., 2017a).

Acute *Rickettsia australis* infection is likely to be under-recognised, with evidence showing an increased disease burden with increased recognition of severe disease, including death (Derne et al. (2015), Laboratory Australian Rickettsial Reference (2015), Baird et al. (1992), and Unsworth et al. (2007) in Stewart et al., 2017a).³

QTT has similar clinical and serological features to FISF and ASF (Dehghani et al., 2019). Early clinical features are often non-specific, making diagnosis challenging (Stewart et al., 2017a). Additionally, symptoms may overlap with other infectious diseases including those that are transmitted by non-tick vectors, as well as a number of chronic diseases.

Diagnosing *R. australis* infection can be challenging. In patients presenting with fever and a rash, epidemiologic data and knowledge of high-risk exposure activities can be valuable in considering QTT. A high degree of suspicion is required as the non-specific symptoms in early QTT can lead to a delay in diagnosis (Stewart et al., 2017a).

Infectious agent

QTT was the first tick-transmitted infection recognised in Australia (Graves & Stenos, 2017). The first case of QTT was reported in 1946 (then known as Australian tick typhus) from North Queensland (Brody (1946) in Graves & Stenos, 2017). The causative agent, *R. australis*, was isolated and described in the same year from a series of cases (Anfrew et al. (1946) in Graves & Stenos, 2017). *R. australis* was later isolated by Pope in 1955 from a patient in southeast Queensland (Pope (1955) in Graves & Stenos, 2017) and in 1974 by Campbell and Domrow from *I. holocyclus* and *I. tasmanii* ticks (Campbell & Domrow (1974) in Graves & Stenos, 2017). Subsequent similar cases were reported in New South Wales and Victoria (Pinn & Sowden (1998) in Dehghani et al., 2019).

Rickettsia are a genus of nonmotile, non-spore-forming, obligate, Gram-negative bacteria that belong to the group alphaproteobacteria (Australian Rickettsial Reference Laboratory, n.d.; Dehghani et al., 2019; Graves, n.d.; Azad & Beard (1998) in Stewart et al., 2017a). *Rickettsia* obtains energy by parasitising vascular endothelial cells and macrophages in mammalian target organs (Dehghani et al., 2019). Vascular injury in the form of intramural and perivascular infiltration of lymphocytes and macrophages occurs (Feng et al. (1993) in Stewart et al., 2017a). Severe vasculitis and vessel thrombosis can then cause isolated end-organ infarction (Stewart et al., 2017a). Suppression of T cell immunity allows intracellular rickettsial survival (Feng et al. (1993) in Stewart et al., 2017a).

R. australis is endogenous to Australia and phylogenetically distinct from rickettsiae in other parts of the world (Graves (2013) and Beard et al. (1996) in Stewart et al., 2017a). *R. australis* appears to be relatively uncommon in Australia (Laboratory Australian Rickettsial Reference (2015) in Stewart et al., 2017a).

³ Laboratory Australian Rickettsial Reference is the reference as cited by Stewart et al. (2017a). The laboratory is known as the Australian Rickettsial Reference Laboratory.

Vector

R. australis has been isolated from both *I. holocyclus* (Australian paralysis tick), with the adult female *I. holocyclus* the predominant vector, and *I. tasmanii* (Sexton et al. (1991), and Barker & Walker (2014) in Stewart et al., 2017a). *I. holocyclus* has a predilection for forested areas with annual rainfall over 1,000 mm and requires the presence of appropriate vertebrate hosts for its survival (Barker & Walker (2014), and Domrow & Derrick (1964) in Stewart et al., 2017a). While the official common name is the Australian paralysis tick, it is often referred to (depending on its stage of development) as the grass tick (nymphs), seed tick (larvae) and bush tick (adult) (Australian Government Department of Health, 2015; Doggett, 2004). In Queensland, *I. holocyclus* is also known as the scrub tick, particularly in North and Far North Queensland, with the name 'scrub tick' echoing the tick's predilection for edges of wet forests (scrub) (Barker & Barker, 2018).

I. cornuatus has also been identified as a vector for *R. australis* (Dehghani et al., 2019). The southern paralysis tick (*I. cornuatus*), is found in Tasmania and Victoria (Australian Government Department of Health, 2015).

The tick vector *I. tasmanii* is distributed throughout eastern and south-eastern Australia, and has been found as far inland as Emerald and Roma, Queensland, although it rarely bites humans (Barker & Walker (2014) in Stewart et al., 2017a).

Reservoir

Mammalian species including bandicoots, rodents, cattle, wombats and companion animals are bitten by ticks harbouring SFG *Rickettsia* (Derne et al. (2015) and Sexton et al. (1991) in Stewart et al., 2017a). The bandicoot species *Isodon macrourus* (Northern brown bandicoot) and *Perameles nasuta* (Long-nosed bandicoot), which carry the highest densities of ticks, are necessary for *Ixodes* tick populations to persist through the seasons in Queensland (Barker & Walker (2014) in Stewart et al., 2017a).

I. holocyclus has been recorded from 34 species of mammals and seven species of bird (Barker & Walker (2014) in Barker & Barker, 2018), although whether it feeds successfully on all of these species is undetermined (Barker & Barker, 2018). Indeed, this tick has an extensive host range including, but not limited to, domestic animals such as dogs, cats, chickens and other fowl, as well as humans (Barker & Walker (2014) in Chalada et al., 2016). In native animals, hosts include wallabies, kangaroos, bandicoots, possums and dingoes (Barker & Walker (2014) in Chalada et al., 2016).

Where it is abundant, *I. holocyclus* will be found on most of the species of mammals present, but in south eastern Queensland the bandicoots *I. macrourus* (Northern brown bandicoot) and *P. nasuta* (Long-nosed bandicoot) have been considered the principal hosts since at least 1975 (Doube (1975) in Barker & Barker, 2018). These bandicoots may carry many ticks (Barker & Barker, 2018). In south eastern Queensland it appears that reasonable numbers of *I. macrourus* and *P. nasuta* are required for populations of *I. holocyclus* to persist from one tick season to another (Doube (1975) in Barker & Barker, 2018). However, in other parts of the geographic range of *I. holocyclus*, where there seem to be large numbers of ticks but few, if any, bandicoots, this is probably not the case (Barker & Barker, unpublished data, in Barker & Barker, 2018).

I. holocyclus has also been found in urban environments, feeding on introduced pests (Lydecker et al., 2014; Taylor et al., 2020). A recent study that investigated the potential role of rabbits in the life cycle of Australian ticks, using rabbits collected from pest control

programs in two urban forest remnants in Sydney, found the most abundant tick species on the rabbits was *I. holocyclus* (Taylor et al., 2020). A study by Lydecker et al. that questioned whether urban bandicoots were solely to blame for tick concerns noted *I. holocyclus* has also been recorded feeding on the following urban pests: the wild rabbit, the house mouse, the brown rat, and the black rat (Lydecker et al., 2014).

I. tasmanii is likely to maintain transmission of *R. australis* among small mammals (Unsworth et al. (2005) in Stewart et al., 2017a).

Mode of transmission

R. australis is transmitted by the bite of certain *Ixodes* spp. ticks (Barker & Walker (2014), and Graves & Stenos (2003) in Stewart et al., 2017a) which are present predominantly along the east coast of Australia (Laboratory Australian Rickettsial Reference (2015), and Barker & Walker (2014) in Stewart et al., 2017a). *R. australis* has been isolated from both *I. holocyclus* (Australian paralysis tick), and *I. tasmanii*, with the adult female *I. holocyclus* the predominant vector, (Sexton et al. (1991), and Barker & Walker (2014) in Stewart et al., 2017a).

I. cornuatus has also been identified as a vector for *R. australis* (Dehghani et al., 2019).

Australian paralysis ticks are not particularly mobile, and rely on passing animals for a blood meal (Australian Government Department of Health, 2015). In searching for a host, the Australian paralysis tick displays a behaviour referred to as 'questing'; whereby the tick climbs to the top of the nearest vegetation and waves its forelegs to and fro slowly, in the hope of contacting a prospective passing host (University of Sydney Department of Medical Entomology, 2003). The Australian paralysis tick undertakes this questing behaviour each time a host is required for blood. They rarely climb higher than 50 cm in their habitat, for risk of desiccation, so do not drop out of trees, despite this common belief (Australian Government Department of Health, 2015; Doggett, 2004; University of Sydney Department of Medical Entomology, 2003). However, after successfully making contact with and landing on a person or animal, they can wander over the host for some hours before attaching (Doggett, 2004), often attaching to the head area (Australian Government Department of Health, 2015; Doggett, 2004) such as on the scalp behind the ear, or other areas where skin is thinner.

I. holocyclus can attach to various sites of the body, including the conjunctiva, making removal of the tick very challenging (Teong et al. (2015) in Sukkanon et al., 2019).

While QTT infection can occur from the bite of an infected tick, it can also occur from exposure to the faeces of infected hosts, with ticks in infected areas generally surviving by feeding off other wildlife including marsupials and rodents (Thomas & Wu, 2018).

Incubation period

While *Rickettsia* spp. usually produce symptoms between one and two weeks after inoculation by an *Ixodes* tick (Sexton et al. (1991), and Pinn & Sowden (1998) in Stewart et al., 2017a), *R. australis* has a shorter incubation period averaging five days (range, three to six days) (Sexton et al. (1991), Streeten et al. (1948), and Ash & Smithurst (1995) in Stewart et al., 2017a).

Infectious period

As QTT is not transmitted directly from person to person there is no infectious period and patients do not need to isolate.

Clinical presentation and outcome

Diagnosing *R. australis* infection can be challenging and in patients presenting with fever and a rash, epidemiologic data and knowledge of high-risk exposure activities can be valuable in considering QTT. A high degree of suspicion is required as the nonspecific symptoms in early QTT can lead to a delay in diagnosis (Stewart et al., 2017a).

QTT has similar clinical and serological characteristics as FISF and ASF (Dehghani et al., 2019). The clinical presentation of these rickettsial infections in Australia include eschar, fatigue, fever, headache, myalgia and rash (macular, papular, vesicular) although the severity and duration of rickettsial diseases vary considerably (Dehghani et al., 2019). Early clinical features are often non-specific, making diagnosis challenging (Stewart et al., 2017a). Additionally, symptoms may overlap with other infectious diseases including those that are transmitted by non-tick vectors, as well as a number of chronic diseases.

In symptomatic infections, QTT is often a mild condition involving fever, chills, headache, a slight cough, malaise, myalgia, a rash, eschar and enlarged lymph nodes (Dehghani et al., 2019; Graves & Stenos, 2017; Streeten et al. (1948) in Stewart et al., 2017a; Brody (1946), and Andrew et al. (1946) in Unsworth et al., 2007).

In acute uncomplicated cases, QTT resolves within 48 hours after initiation of treatment with doxycycline (Sexton et al. (1991), and Streeten et al. (1948) in Stewart et al., 2017a).

The clinical presentation of a case of QTT in rural Queensland published by the Royal Australian College of General Practitioners (RACGP) provides advice to support general practitioners (GPs) (Thomas & Wu, 2018). The case report by Thomas and Wu is available at the following link: <https://www1.racgp.org.au/ajgp/2018/june/queensland-tick-typhus>.

The clinical features of SFG diseases in North Queensland, from a retrospective clinical audit of microbiologically-confirmed cases of SFG rickettsial infection between 1997 and 2016 at a tertiary referral hospital in North Queensland have recently been described (Stewart et al., 2019). The article (Stewart et al., 2019), which also covers the implications for patient identification and management, can be fully accessed at this link: <https://pubmed.ncbi.nlm.nih.gov/31318873/>.

Fever

High grade fever of up to 41°C (Sexton et al. (1991) in Stewart et al., 2017a) is observed in acute cases. Prolonged fever is associated with rickettsaemia, end organ dysfunction and intensive care admissions (Derne et al. (2015) in Stewart et al., 2017a).

Rash

Rash morphology is variable, and can be macular, maculopapular (Sexton et al. (1991) in Stewart et al., 2017a), vesicular or pustular, with the latter two forms sometimes confused with acute varicella (Hudson et al. (1994) in Stewart et al., 2017a). Infrequently, the rash is pruritic (Hudson et al. (1993) in Stewart et al., 2017a).

The rash usually lasts for 10 to 12 days, can appear as early as 24 hours after a tick bite, and typically follows a widespread, global eruption involving the trunk and limbs (Stewart et al., 2017a). In hospitalised patients with severe illness, evolution to a petechial or purpuric rash toward the end of the clinical course has been described (Birch & Muller (2009), and Sexton et al. (1991) in Stewart et al., 2017a).

Erythema migrans (EM) at and around the *Ixodes* attachment site is not uncommon in QTT (Stewart et al., 2017a). Of note, EM is observed in other tick-borne diseases such as *Rickettsia* and *Borrelia* spp., including Lyme disease (Barker & Walker (2014) in Stewart et al., 2017a), as well as southern tick-associated rash illness (Beaman, 2016), which are both overseas-acquired diseases. In the Australian context, QTT infection can cause EM. Hence the recommendation to seek advice from appropriate experts in vector-borne diseases.

Eschar

In approximately 50% to 65% of *R. australis* infections, an eschar is seen, with the detection of an eschar being diagnostically valuable. It is, however, often difficult to find as it can occur in sites that can be missed on examination such as in the axilla or groin (Stewart et al., 2017a).

While it is not known whether the presence of an eschar is associated with severity of disease (La Scola et al. (2009) in Stewart et al., 2017a), it may be associated with inoculum size and increased probability of disseminated disease (Stewart et al., 2017a).

Lymphadenopathy

R. australis is associated with higher rates of lymphadenopathy and eschar than other rickettsial spotted fever infections in Australia (Sexton et al. (1991) in Stewart et al., 2017a).

Tender lymphadenopathy, usually localised to the region draining the tick bite or eschar, occurs in approximately 70% of patients (Sexton et al. (1991) in Stewart et al., 2017a).

Severe and rare presentations of QTT

QTT may be severe (McBride et al. (2007), and Birch & Muller (2009) in Graves & Stenos, 2017; McBride et al. (2007), and Birch & Muller (2009) in Stewart et al., 2017a) or fatal (Sexton et al. (1990) in Graves & Stenos, 2017; Sexton et al. (1990) in Stewart et al., 2017a) and may have unusual features (Wilson et al. (2013) in Graves & Stenos, 2017).

Less common manifestations of QTT include arthralgia, splenomegaly, abdominal pain, dry cough, sore throat, conjunctivitis, and photophobia (McBride et al. (2007) in Stewart et al., 2017a).

Complications from severe QTT are reported to be usually vascular in origin, signifying endothelial dysfunction and vasculitis that can lead to end-organ dysfunction (Feng et al. (1993) in Stewart et al., 2017a). In rare cases, severe vasculitis-driven skin manifestations have been reported to complicate severe infection, causing skin necrosis, bullae, and purpura mimicking Stevens-Johnson syndrome (McBride et al. (2007) in Stewart et al., 2017a), with purpura fulminans reported in one case series (Graves & Stenos (2009) in Stewart et al., 2017a). Necrotic phenomena, while rare, have been reported sporadically in case reports with these phenomena including widespread digital necrosis and splenic infarction (Birch & Muller (2009), and Wilson et al. (2013) in Stewart et al., 2017a).

Rarer severe manifestations have also included pneumonitis requiring mechanical ventilation (McBride et al. (2007) in Stewart et al., 2017a), and myocarditis and small-volume pericardial effusions (Graves & Stenos (2009), and Wilson et al. (2013) in Stewart et al., 2017a).

While QTT is not known to directly affect the central nervous system, there have been reports of confusion, seizures and hallucinations as presentations of this disease (Sexton et al. (1991) in Stewart et al., 2017a).

Renal failure as a result of *R. australis* infection has not been reported (Stewart et al., 2017a).

There are no known identified risk factors for developing severe disease or complications of QTT (Stewart et al., 2017a).

Outcomes

There is little systematic evidence available regarding the outcomes of acute *R. australis* infection, particularly for non-hospitalised patients. In the documented severe hospitalised cases with complications, a full recovery following acute illness is expected (Stewart et al., 2017a). There is no evidence of chronic infection (Stewart et al., 2017a).

Post-infection fatigue, a well-known consequence of several infections including Ross River virus, Q fever and Epstein-Barr virus, is not yet widely recognised as a problem following rickettsial infection. However, it has been suggested by a study involving two large cohorts of fatigued and non-fatigued patients (Unsworth et al. (2008) in Graves & Stenos, 2017) and a case report (Watts et al. (2008) in Graves & Stenos, 2017). This post-infective syndrome of lethargy, malaise, and muscle pains that persists for several months or more after acute infection has been described (Watts et al. (2008), Unsworth et al. (2008), and Knyvett & Sanders (1964) in Stewart et al., 2017a).

Persons at increased risk of disease

Rickettsial infections such as QTT are typically seen in residents of endemic areas, as well as campers, travellers, and hikers to endemic areas (Dehghani et al., 2019). People who live in, or travel to, areas where QTT is regularly found and who also engage in outdoor activities that increase the risk of them being bitten by an infected tick are at increased risk of QTT.

The risk of contracting a tick-borne infection, including QTT, is determined by the overall number of ticks in the area, the proportion of those carrying disease, and human behaviour (World Health Organization, 2014).

All nonimmune people are susceptible to rickettsial infection, [including QTT], depending on environmental exposure. Long-lasting immunity probably follows infection. People are at risk of infection for as long as they remain in infected areas (Victorian Department of Health and Human Services, n.d.).

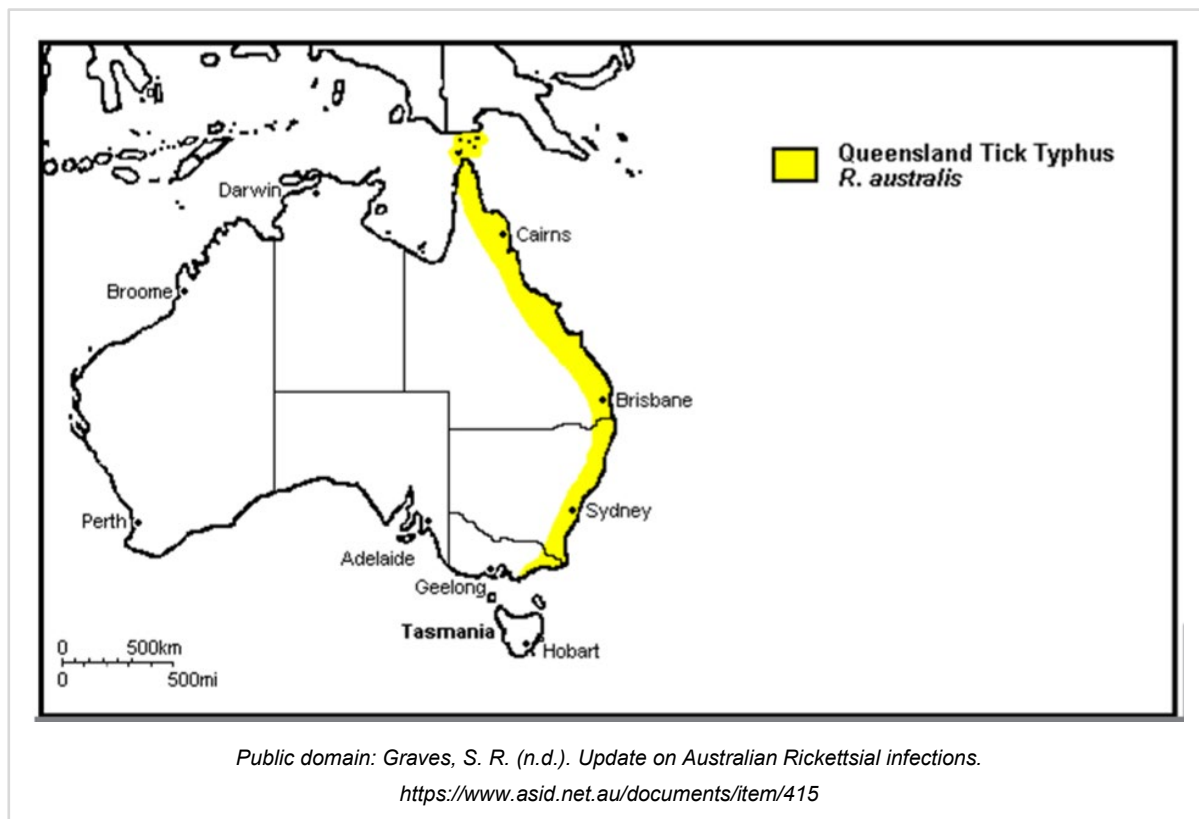
Risk areas for QTT infection in Australia

Over 95% of tick bites in humans in eastern Australia are due to the Australian paralysis tick (Australian Government Department of Health, 2015; Geary et al., 2021; Taylor et al., 2019; van Nunen (2018) in van Nunen & Ratchford, 2021), and most tick-borne illnesses in Australia are due to this species (Australian Government Department of Health, 2015). A very recent study further demonstrated that *I. holocyclus* is well-known for biting humans (Geary et al., 2021). Geary et al.'s 2021 paper reported on 30 years of samples submitted to the Department of Medical Entomology at Westmead Hospital, the New South Wales reference laboratory for arthropods of medical importance. Their research showed the most common species of all arthropods submitted for identification (n = 5655), was *I. holocyclus*, with 708 species submitted (Geary et al., 2021). Of the *I. holocyclus* submitted, 98.3% were from New South Wales, with many from the south coast of New South Wales, and a small number (n = 10) were from Victoria. Additionally, of the *I. holocyclus* specimens submitted, Geary et al. reported that specimens from child patients (aged zero to nine years) predominated and were more than twice as frequent as any other age class, even with

population-adjusted data. Age classes were described as 'in 10 year intervals' (Geary et al., 2021). The samples were mostly submitted by health professionals and pathology services from within New South Wales but also received from interstate, environmental health officers, pest control companies, veterinarians, schools, various government and industry organisations, as well as members of the public (Geary et al., 2021).

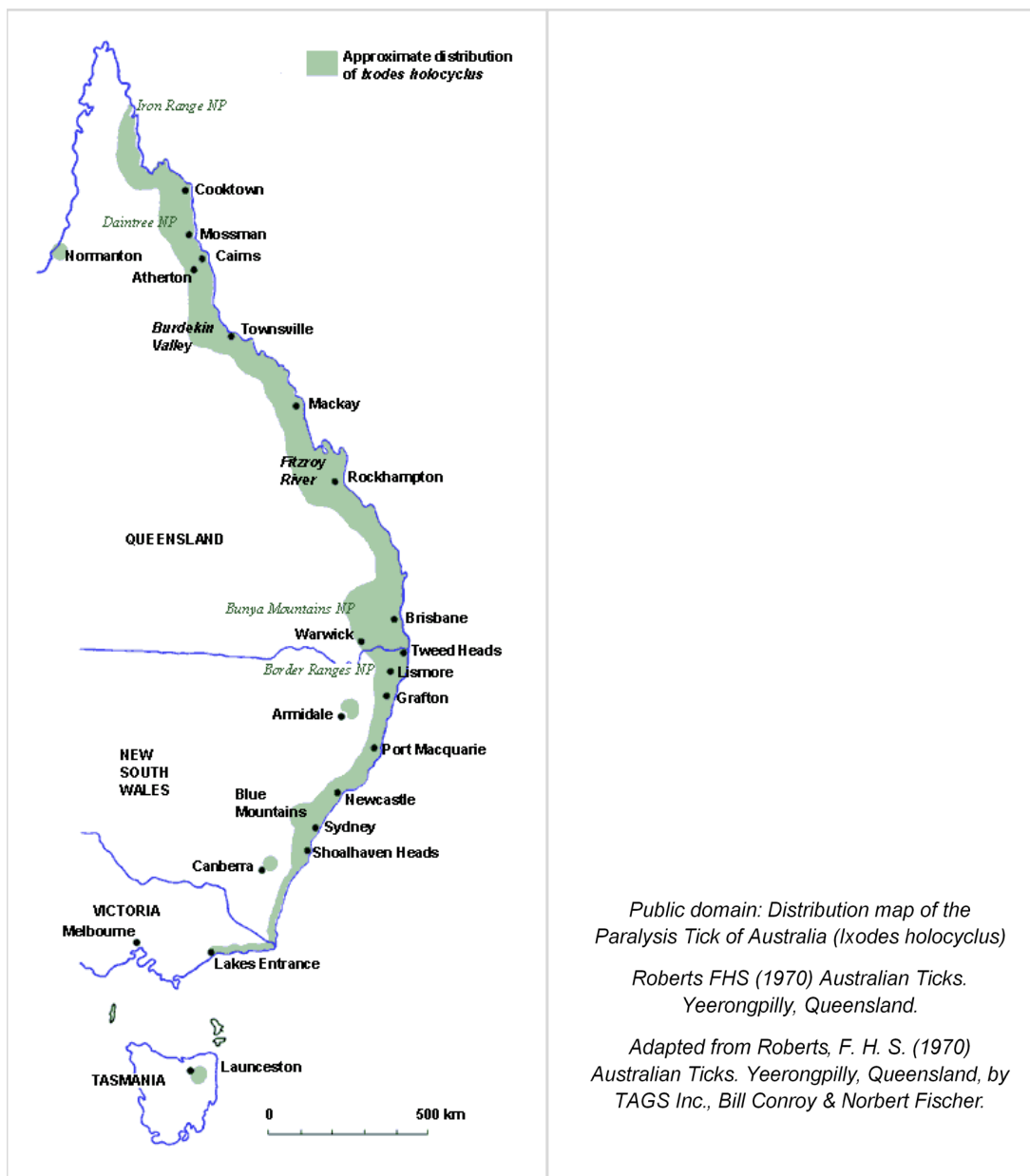
QTT is regularly seen on the east coast of Australia from the Torres Strait Islands to the south-eastern corner of Victoria, with the northern suburbs of Sydney a very common location for transmission of this infection (Campbell et al. (1979), and Hudson et al. (1993) in Graves & Stenos, 2017; Stewart et al., 2017a). Figure 1 below is from Graves' update on Australian rickettsial diseases (Graves, n.d.) and shows the distribution of QTT.

Figure 1: Distribution of Queensland tick typhus (Public domain)



In north-eastern New South Wales, 15.4% of Australian paralysis ticks (*I. holocyclus*) were found to contain *R. australis* (Graves et al. (2016) in Graves & Stenos, 2017), suggesting a one in six risk of being infected with Rickettsia if bitten by this tick in this location (Graves et al., 2016; Graves & Stenos, 2017).

Figure 2: Approximate geographic distribution of *Ixodes holocyclus* (Public domain)



The main distribution of the *I. holocyclus* tick (Figure 2 above) is within 20 km of the coast along virtually the entire eastern seaboard of Australia (Barker & Walker (2014), and Hardy et al. (2014) in Stewart et al., 2017a; Tick-induced Allergies Research and Awareness, n.d.). However, it has been isolated in areas more than 100 km inland including the Bunya Mountains, Barcardine, and Thargomindah in Queensland and the Lower Blue Mountains in New South Wales (Stewart et al., 2017a; Tick-induced Allergies Research and Awareness, n.d.). It can also be found in the Australian Capital Territory, probably having travelled from the coast (Tick-induced Allergies Research and Awareness, n.d.).⁴ It is not known to occur in South Australia, Western Australia or the Northern Territory (Australian Government Department of Health, 2015).

⁴ Probably travellers from the south coast on people and their companion animals.

While *I. holocyclus* is not known to be distributed north of Cooktown, Queensland (Hardy et al. (2014) in Stewart et al., 2017a), a case has been documented in the Torres Strait (Unsworth et al. (2007) in Stewart et al., 2017a).

The geographical distribution of *R. australis* is expanding due to changes in climate and human population demographics (Dehghani et al., 2019), with the geographical distribution and boundaries of QTT infection being pushed further along the coastline, as well as inland (Graves & Stenos (2009) in Stewart et al., 2017a). A larger distribution of QTT along eastern coastal Australia than was demonstrated in a retrospective review by Derne et al. (Derne et al. (2015) in Stewart et al., 2017a) has been identified by serological surveys (Stewart et al., 2017a).

Risk factors and risk activities for acquiring QTT infection

People living in, or travelling to, areas endemic for QTT and who also engage in activities that increase their risk of coming into contact with ticks are at greater risk of contracting QTT.

Living conditions, rural residence and tropical climate are predictors of QTT, with these conditions related to vector abundance and their proximity to humans (Derne et al. (2015) in Stewart et al., 2017a). The large number of mammalian host species for *Ixodes* spp. ticks and its predilection for wet forested areas in certain times of the year, certain human activities are high-risk for acquiring infection (Sexton et al. (1991), and Derrick (1957) in Stewart et al., 2017a).

I. holocyclus has a predilection for forested areas with annual rainfall over 1,000 millimetres and requires the presence of appropriate vertebrate hosts for its survival (Barker & Walker (2014), and Domrow & Derrick (1964) in Stewart et al., 2017a). Australian paralysis ticks are therefore found most commonly in wet sclerophyll forests and temperate rainforests (Tick-induced Allergies Research and Awareness, n.d.) in moist, humid coastal areas with abundant native animals that serve as hosts for the tick (Australian Government Department of Health, 2015). Long grasses and bushland provide ideal environments for ticks, and if people live close to these areas, it is not uncommon for there to be Australian paralysis ticks in their garden (Australian Government Department of Health, 2015).

Occupational (e.g. farming, commercial harvesting) and recreational (e.g. bushwalking) activities have been identified as strong risk factors for *R. australis* infection with up to 50% of infections acquired in this manner (Sexton et al. (1991) in Stewart et al., 2017a).

Stewart et al., in their review on QTT, notes many cases reported in the literature were preceded by exposure to bush environments during activities such as:

- gardening (Sexton et al. (1991) in Stewart et al., 2017a)
- living near bushland (Kende & Graves (2013) in Stewart et al., 2017a)
- military training exercises (Andrew et al. (1946) in Stewart et al., 2017a)
- fishing (Derne et al. (2015) in Stewart et al., 2017a)
- fieldwork (Sexton et al. (1991) in Stewart et al., 2017a).

A 2017 retrospective study on QTT infection in hospitalised patients in North Brisbane also found risk factors such as hobbies and/or occupational activities were present, with 42% of those hospitalised for QTT having hobbies and/or occupations linked to the acquisition of the disease (Stewart et al., 2017b). Cases of QTT were identified retrospectively from 2000 to 2015 from any of the five sites in Metropolitan North Hospital and Health Service in Queensland, Australia, through a local pathology database. Nearly half (47%) identified a tick bite in the days preceding presentation at hospital; however, reported exposure to a known

animal host was minimal (25%). This study also identified that slightly more women than men (1.1:1) were hospitalised for QTT (Stewart et al., 2017b). Stewart et al. noted that their finding on male-to-female ratio of 1:1.1 was in contrast to previous reports that had shown a male dominance (Sexton et al. (1991) in Stewart et al., 2017), but commented that this could represent a change in occupation and recreational activities among women over the past half century, which is known to highly influence the risk of infection (Stewart et al., 2017b).

Seasonal risk for QTT infection

People who reside in, or travel to, areas where *I. holocyclus* and QTT are endemic are most at risk of becoming infected if they are undertaking at risk activities during the winter and spring, and during periods of high humidity.

The Australian paralysis tick has a distinct seasonality; the larval stage is most active during the autumn months, the nymph during winter and the adult during the spring (Australian Government Department of Health, 2015; Geary et al., 2021; Eppleston et al. (2013) in Sukkanon et al., 2019). Geary et al.'s 2021 study of arthropod samples sent to the Department of Medical Entomology at Westmead Hospital, in Sydney, New South Wales, between 1988 and 2017, showed strong seasonal trends for *I. holocyclus*, the most common (708 specimens submitted/5655 total samples submitted) of all arthropods sent to the Department for identification. In their study, larval *I. holocyclus* ticks peaked in autumn, nymphs peaked during winter and adult ticks peaked during spring (Geary et al., 2021). Adult female *Ixodes* spp. ticks (the vector for *R. australis*) are most abundant in Queensland from October to December (Barker & Walker (2014) in Stewart et al., 2017a). This tick is most active during periods of high humidity, especially after rain, and this is when people should take particular care to avoid tick bites (Australian Government Department of Health, 2015).

Infection by *R. australis* may occur throughout the year in immunocompetent people of all ages and ethnicities although previous research had indicated 80% of documented cases have occurred in winter and spring (June to November) coinciding with increased tick densities in these months (Sexton et al. (1991), and Barker & Walker (2014) in Stewart et al., 2017a).

In Stewart et al.'s (2017b) study of patients hospitalised for QTT in North Brisbane, infection occurred throughout the year, as indicated by previous research. However, in this study, half of the cases were reported between April and July, during the seasons of autumn and winter in Australia. Stewart et al. noted their finding of increased incidence of QTT in autumn and winter contradicted a previous report of increasing incidences of QTT during summer and spring months (Sexton et al. (1991) in Stewart et al., 2017b).

Age and gender

In Stewart et al.'s 2017 study on QTT infection in hospitalised patients in North Brisbane, male-to-female ratio was 1:1.1 (Stewart et al., 2017b). Stewart et al. noted that the finding on male-to-female ratio of 1:1.1 was in contrast to previous reports that had shown a male dominance (Sexton et al. (1991) in Stewart et al., 2017b), although Stewart et al. commented that this could represent a change in occupation and recreational activities among women over the past half century which is known to highly influence the risk of infection (Stewart et al., 2017b).

Infection by *R. australis* may occur throughout the year in immunocompetent people of all ages and ethnicities (Sexton et al. (1991), and Barker & Walker (2014) in Stewart et al., 2017a). Stewart et al.'s 2017 study on QTT infection in hospitalised patients in Northern

Brisbane, confirmed that QTT infection had affected people across a wide range of ages with the age of those hospitalised for QTT ranging from three to 72 years (mean of 39.5 years) (Stewart et al., 2017b).

In Geary's 2021 study, which reported on 30 years of samples submitted to the Department of Medical Entomology at Westmead Hospital, New South Wales, of the *I. holocyclus* specimens submitted, specimens from child patients aged zero to nine years predominated and were more than twice as frequent as any other age class, even with population-adjusted data (Geary et al., 2021).

Disease occurrence and public health significance

QTT is not a nationally notifiable disease in Australia (Australian Government Department of Health, 2021). Rickettsia infection (including spotted fevers and all forms of typhus fever) is notifiable in Western Australia (Western Australian Department of Health, n.d.). Typhus (all forms) is notifiable in Northern Territory (Northern Territory Department of Health, 2016), and typhus (epidemic) is notifiable in New South Wales (New South Wales Ministry of Health, 2020; New South Wales Parliamentary Counsel's Office, 2021).

Recent serological surveys suggest a disease burden along the east coast that is greater than previously realised (Derne et al. (2015), and Faa et al. (2006) in Stewart et al., 2017a). The public health impact of rickettsial infections on lives or productivity lost is largely unmeasured, but it is suspected to be high (Victorian Department of Health and Human Services, n.d.).

QTT is an emerging public health threat (Dehghani et al., 2019; Stewart et al., 2017a) and an increasingly recognised important cause of community-acquired acute febrile illness in eastern Australia (Stewart et al., 2017a). Stewart et al. noted acute *R. australis* infection is likely to be under-recognised with recent evidence showing its increased disease burden with increased recognition of severe disease including death (Derne et al. (2015), Laboratory Australian Rickettsial Reference (2015), Baird et al. (1992), and Unsworth et al. (2007) in Stewart et al., 2017a).⁵

Routine prevention activities

Currently, there is no effective vaccine against any Rickettsia species, including *R. australis* (Derne et al. (2015) in Stewart et al., 2017a). Difficulties exist in creating a universal SFG rickettsial vaccine due to variation in the chemical configuration of the immunologically important outer membrane proteins (Stenos & Walker (2000), and Feng & Walker (2003) in Stewart et al., 2017a).

See *Prevention and management of tick bites in Australia Guidance Note* for information on personal preventive strategies to prevent tick bites on people and pets, preventing tick bites around the home and safely managing tick bites in Australia.

Also see the DSCATT Clinical Pathway (Australian Government Department of Health, 2020), for more advice.

⁵ Laboratory Australian Rickettsial Reference is the reference as cited by Stewart et al. 2017a. The laboratory is known as the Australian Rickettsial Reference Laboratory.

Diagnosis

Refer to the DSCATT Clinical Pathway (Australian Government Department of Health, 2020).

Treatment

Refer to the DSCATT Clinical Pathway (Australian Government Department of Health, 2020). The Australian Immunisation Handbook (<https://immunisationhandbook.health.gov.au/>) notes that bite wounds can lead to tetanus; however, 'bite wounds' are not intended to extend to tick bites. Therefore, clinicians do not need to check the tetanus immunisation status of patients who present with a tick bite.

Flinders Island spotted fever

FISF has similar clinical and serological features to QTT and ASF (Dehghani et al., 2019). Early clinical features are often non-specific, making diagnosis challenging (Stewart et al., 2017a). Additionally, symptoms may overlap with other infectious diseases including those that are transmitted by non-tick vectors, as well as a number of chronic diseases.

FISF was first identified on Tasmania's Flinders Island, and is most common in Tasmania, although tick vectors are also found in southern Australia. A high proportion of visitors to Tasmania undertake outdoor activities that put them at risk of tick bites. Although it is likely that some visitors to Tasmania will develop rickettsial disease, they may seek medical advice after leaving the island, and treating clinicians outside of Tasmania may not be aware of the risk and not perform the appropriate tests (Willis et al., 2019).

Infectious agents

The causative agent of FISF is the bacteria *Rickettsia honei* (Stenos et al. (1998) in Dehghani et al., 2019; Tasmanian Department of Health, 2020; Graves & Stenos (2009), and Graves et al. (1993) in Willis et al., 2019). A rickettsial infection typically associated with Tasmania, FISF was first described in 1991 by Robert Stewart, the sole GP on the island who had identified 26 cases of a spotted fever-like illness over 17 years (Stewart (1991) in Willis et al., 2019).

R. honei was isolated by Graves et al. from febrile patients (Graves et al. (1993) in Graves & Stenos, 2017), and shown to be genetically different from *R. australis* (Graves & Stenos, 2017).

Vector

The tick vector for FISF infection is the reptile-associated *Bothriocroton hydrosauri* (southern reptile tick, formerly *Aponomma hydrosauri*), which is known to bite humans (Barker & Barker, 2018; Graves & Stenos, 2017). *B. hydrosauri* is the arthropod-host of *R. honei* on Flinders Island, Tasmania (Stenos et al. (2003) in Barker & Barker, 2018), and mainland Tasmania (Whitworth et al. (2003) in Barker & Barker, 2018). A study found 63% of these ticks on Flinders Island contained bacteria (*R. honei*) that cause FISF (Stenos et al. (2003) in Graves & Stenos, 2017).

B. hydrosauri is the only known vector/reservoir of *R. honei* in Australia (Stenos et al. (2003) in Unsworth et al., 2007).

Reservoir

While FISF was first identified on Tasmania's Flinders Island, and is most common in Tasmania, tick vectors are also found in southern Australia.

The *B. hydrosauri* tick that carries the FISF bacteria *R. honei* is thought to be found most commonly on reptiles (Tasmanian Department of Health, 2020). Native blue-tongue lizards and snakes are confirmed hosts for the southern reptile tick *B. hydrosauri* (Stenos et al. (2003), and Whitworth et al. (2003) in Willis et al., 2019).

Barker and Barker noted *R. honei* is apparently sustained in populations on *B. hydrosauri* on Flinders Island by vertical, transovarial transmission which involves *R. honei* infecting the eggs of *B. hydrosauri* in situ with the next generation of *B. hydrosauri* becoming infected with

R. honei without feeding on an infected vertebrate. Therefore, vertebrates are apparently not needed for the survival of *R. honei* on Flinders Island (Barker & Barker, 2018).

B. hydrosauri may be found on the main types of reptiles in southern Australia – lizards, snakes and a terrestrial turtle (Barker & Walker (2014) in Barker & Barker, 2018).

The main host of *B. hydrosauri* in much of South Australia is *Tiliqua rugosa* (sleepy lizard); however, given the opportunity, *B. hydrosauri* will attach to and feed on humans, cattle and horses (Barker & Barker, 2018).

Mode of transmission

FISF is transmitted to humans by a bite from the tick *B. hydrosauri* infected with the bacteria *R. honei* (Tasmanian Department of Health, 2020). There is no human-to-human transmission of FISF (Tasmanian Department of Health, 2020).

Incubation period

Symptoms usually start one to two weeks after being bitten by an infected tick (Tasmanian Department of Health, 2020).

Infectious period

As FISF is not transmitted directly from person to person there is no infectious period and patients do not need to isolate.

Clinical presentation and outcome

FISF is characterised by fever, headache, myalgia, transient arthralgia, maculopapular rash and cough in some patients (Dehghani et al., 2019; Stewart (1991) in Willis et al., 2019). Willis et al. noted 46% of cases in Stewart's case series had a cough, and 46% had a skin lesion other than a rash (Willis et al., 2019).

FISF has an abrupt onset and lasts approximately 19 days without antibiotic treatment (Stewart (1991) in Unsworth et al., 2007) but can be as long as six weeks (Stewart (1991) in Willis et al., 2019), with prompt treatment likely to reduce the burden of disease.

In 2005, Dyer et al. reported on a new focus of *R. honei* spotted fever in South Australia, within the geographic range of *Aponomma hydrosauri* (now *B. hydrosauri*) (Dyer et al., 2005). The four patients presented with fever, maculopapular rash, severe malaise, muscle pains, and in two cases, respiratory symptoms and signs, together with pulmonary infiltrates. All four patients required hospital admission, but Dyer et al. noted that that it is likely that mild or symptomatic infection occurs in others and remains undiagnosed (Graves et al. (1991) in Dyer et al., 2005).

Despite FISF often being described as a mild illness (Heymann (2015) in Willis et al., 2019), Willis et al. noted the infection can be severe, with the death of a middle-aged woman in Queensland due to acute infection of *R. honei* reported in the literature (Graham et al. (2017) in Willis et al., 2019). Much is still unknown about the causes of severe disease and complications, but doxycycline may reduce the risk (Heymann (2015) in Willis et al., 2019).

Persons at increased risk of disease

Rickettsial infections such as FISF are typically seen in residents of endemic areas, as well as campers, travellers, and hikers to endemic areas (Dehghani et al., 2019). People who live

in, or travel to, areas where FISF is regularly found and who also engage in outdoor activities that increase the risk of them being bitten by an infected tick are at increased risk of FISF.

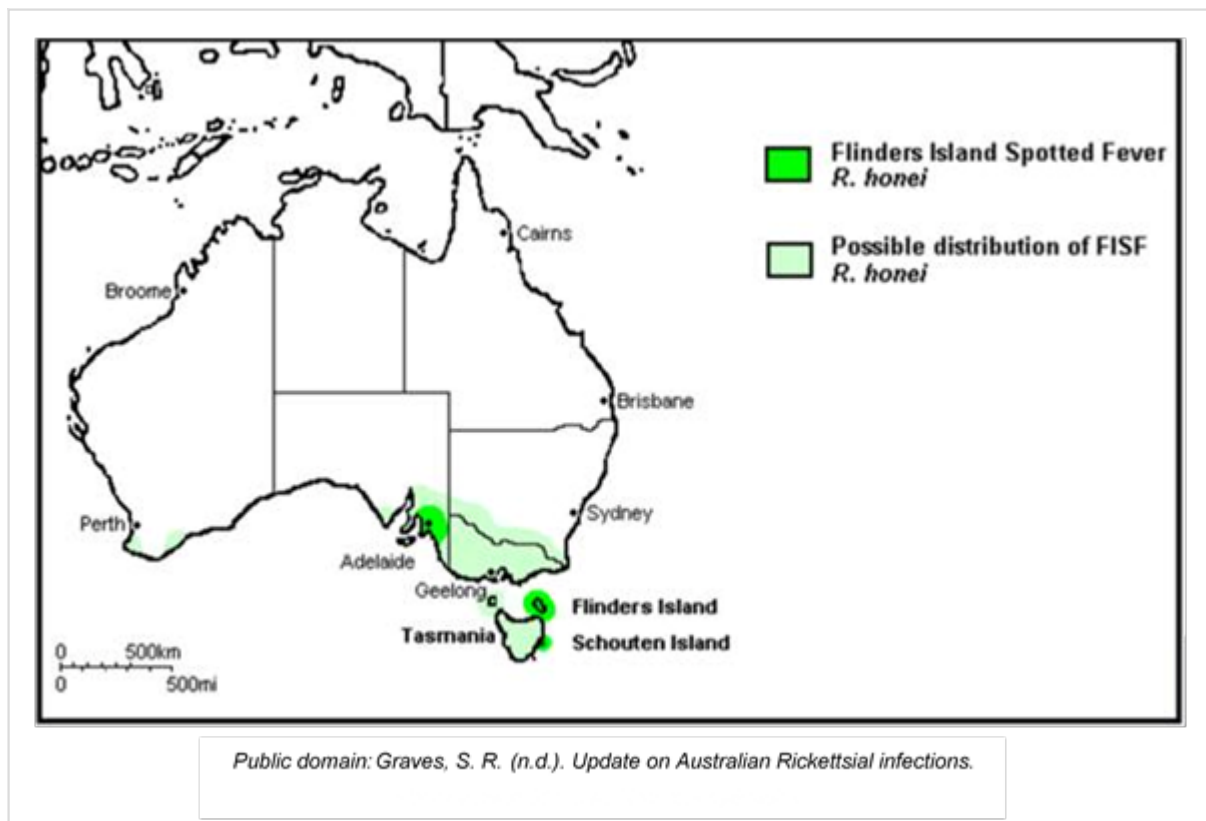
The risk of contracting a tick-borne infection, including FISF, is determined by the overall number of ticks in the area, the proportion of those carrying disease, and human behaviour (World Health Organization, 2014).

All nonimmune people are susceptible to rickettsial infection, [including FISF] depending on environmental exposure. Long-lasting immunity probably follows infection. People are at risk of infection for as long as they remain in infected areas (Victorian Department of Health and Human Services, n.d.).

Risk areas for FISF infection in Australia

FISF has been reported on Flinders Island, mainland Tasmania, south-eastern Australia, south-western coastal areas of Western Australia on Salisbury Island and in Walpole, and south-eastern coastal regions of South Australia near Adelaide (Dehghani et al., 2019; Graves & Stenos, 2017; Willis et al., 2019). Cases have been detected on Schouten Island, south of Freycinet Peninsula, Tasmania, and two were detected in south-eastern South Australia where *B. hydrosauri* are also endemic (Unsworth et al. (2005) in Willis et al., 2019). Figure 3 below is from Graves' update on Australian rickettsial diseases (Graves, n.d.) and shows the distribution of FISF.

Figure 3: Distribution of Flinders Island spotted fever (Public domain)



The Tasmanian Department of Health reported in 2019 that confirmed cases have been acquired in Tasmania, including the Midlands of Tasmania (Willis et al., 2019). These locations included around Great Oyster Bay. The study was the first account including confirmed cases acquired in the Midlands of Tasmania (Willis et al., 2019). The most recent advice (2020) from the Tasmanian Department of Health is that in Tasmania, infections have

been acquired on Flinders Island; the east coast, including Schouten Island, and possibly as far south as Kettering and the Midlands; however the ticks that carry FISF are in other parts of Tasmania and it is possible that FISF can be caught from a much wider geographical distribution (Tasmanian Department of Health, 2020).

Risk factors and risk activities for acquiring FISF

Tasmania is a popular tourist destination. Visitors who undertake outdoor activities may be at risk of tick bites (Tourism Tasmania (2018) in Willis et al., 2019). Bushwalking and other outdoor activities are known to be high-risk in tick endemic areas.

Seasonal risk for FISF infection

The Tasmanian Department of Health advises that infections can occur throughout the year, but the risk increases during the spring and summer months when ticks are most active and when camping and other outdoor activities are more common (Tasmanian Department of Health, 2020). The spring and summer months are not only when the ticks, but their hosts, snakes and blue-tongued lizards, are most active. The advice from the Tasmanian Department of Health regarding seasonal risk for FISF is applicable to other areas outside Tasmania where the tick *B. hydrosauri* is endemic, including southern Australia.

Age and gender

FISF infections can occur in people of all ages (Tasmanian Department of Health, 2020).

Disease occurrence and public health significance

FISF is not a nationally notifiable disease in Australia (Australian Government Department of Health, 2021). FISF is notifiable in Tasmania (Tasmanian Department of Health, 2020) and Rickettsia infection (including spotted fevers and all forms of typhus fever) is notifiable in Western Australia (Western Australian Department of Health, n.d.).

Rickettsial infections can be acquired in locations around Great Oyster Bay and the Midlands region, with the possibility that the distribution is wider still, given the southern reptile tick is distributed throughout Tasmania (Willis et al., 2019).

Willis et al. (2019) noted, as above, a high proportion of visitors to Tasmania undertake outdoor activities that put them at risk of tick bites. Willis et al. (2019) advised that although it is likely that some visitors to Tasmania will develop rickettsial disease, they may seek medical advice after leaving the island, and treating clinicians outside of Tasmania may not be aware of the risk and not perform the appropriate tests. Increased awareness of potential infection by clinicians is essential to accurately diagnose and appropriately treat rickettsial infections, thereby reducing the burden of the disease (Willis et al., 2019).

Routine prevention activities

No vaccine is available for FISF (Tasmanian Department of Health, 2020). Currently, there is no effective vaccine against any Rickettsia species, (Derne et al. (2015) in Stewart et al., 2017a). Difficulties exist in creating a universal SFG rickettsial vaccine due to variation in the chemical configuration of the immunologically important outer membrane proteins (Stenos & Walker (2000), and Feng & Walker (2003) in Stewart et al., 2017a).

See Prevention and management of tick bites in Australia Guidance Note for information on personal preventive strategies to prevent tick bites on people and pets, preventing tick bites around the home and safely managing tick bites in Australia.

Also see the DSCATT Clinical Pathway (Australian Government Department of Health, 2020), for more advice.

Diagnosis

Refer to the DSCATT Clinical Pathway (Australian Government Department of Health, 2020).

Treatment

Refer to the DSCATT Clinical Pathway (Australian Government Department of Health, 2020). The Australian Immunisation Handbook (<https://immunisationhandbook.health.gov.au/>) notes that bite wounds can lead to tetanus, however, 'bite wounds' are not intended to extend to tick bites. Therefore, clinicians do not need to check the tetanus immunisation status of patients who present with a tick bite.

Australian spotted fever

ASF has similar clinical and serological features to QTT and FISF (Dehghani et al., 2019).

Seven cases of ASF have been identified and were widely distributed throughout eastern Australia, including cases on the eastern seaboard of Australia (including the Torres Strait), Tasmania and South Australia (Unsworth et al., 2007). These seven cases demonstrate that emerging rickettsioses are present in Australia (Unsworth et al., 2007).

Infectious agents

The bacterium *Rickettsia honei* subsp. *marmionii* causes ASF, an infection similar to FISF (Graves & Stenos, 2017). The strain is genetically related to *R. honei* (the cause of FISF), and while a SFG Rickettsia, is less closely related to *R. australis* (Unsworth et al., 2007). The name ASF was adopted to distinguish infection caused by *R. honei* subsp. *marmionii* from FISF caused by *R. honei* (Dehghani et al., 2019).

R. honei subsp. *marmionii* has been detected in the ticks *I. tasmani* (unpublished data in Graves & Stenos, 2017), and *Haemaphysalis novaeguineae* in Queensland (Lane et al. (2005) in Graves & Stenos, 2017).

R. honei subsp. *marmionii* has been associated with several cases of human disease in eastern Australia (Unsworth et al. (2007) in Graves & Stenos, 2017; Unsworth et al., 2007). The bacterium has been detected in five patients and isolated from two patients with chronic illness in Melbourne, Australia, although it is not known whether the *rickettsiae* is causally related to the patients' chronic illnesses, or reactivation of latent *rickettsial* infection (Unsworth et al., 2008).

Vector

The only known tick host of *R. honei* subsp. *marmionii* is *H. novaeguineae*, a tick not previously recognised as a transmitter of human pathogens (Unsworth et al. (2007). There is no information currently available on the epidemiology and ecology of the *H. novaeguineae* within Australia (Dehghani et al., 2019).

R. honei subsp. *marmionii* has not been found in any *B. hydrosauri* ticks, although *H. novaeguineae* may be a vector/reservoir, as a *H. novaeguineae* tick was removed from one of the patients from Queensland in the case report series by Unsworth and colleagues (Unsworth et al., 2007). Rickettsial *rrs* and *ompA* gene sequences within the tick demonstrated 100% homology with *R. honei* subsp. *marmionii* (Lane et al. (2005) in Unsworth et al., 2007).

Researchers advise further research is required to know where exactly *H. novaeguineae*, the New Guinea haemaphysalid, lives in northern Australia and how abundant it is there (Barker & Barker, 2018).

Reservoir

The vertebrate host of the tick *H. novaeguineae* is unknown (Graves, n.d.) and the vectors and reservoirs, of *R. honei* subsp. *marmionii* in southern Australia are not known (Unsworth et al., 2007).

H. novaeguineae is known to bite numerous animals including humans and is found in both northern Australia and Papua New Guinea (Roberts (1970) in Unsworth et al., 2007).

H. novaeguineae was not previously recognised as a transmitter of human pathogens (Unsworth et al., 2007).

Mode of transmission

ASF is transmitted to humans by the bite of the tick *H. novaeguineae* infected with *R. honei* subsp. *marmionii*. Rickettsial diseases in Australia (including ASF), are not transmitted directly from person to person (Victorian Department of Health and Human Services, n.d.).

Incubation period

The one patient in Unsworth et al.'s case reports of ASF had a history of a *H. novaeguineae* tick bite which Unsworth et al. suggested may imply an incubation period of five days (Unsworth et al., 2007).

Infectious period

As ASF is not transmitted directly from person to person there is no infectious period and patients do not need to isolate.

Clinical presentation and outcome

ASF has similar clinical and serological features to QTT and FISF (Dehghani et al., 2019).

Unsworth et al. described the clinical presentation of seven ASF cases which were similar to FISF (Unsworth et al., 2007). Among these patients, the disease symptoms were consistent with a relatively mild rickettsial SFG disease. The observed frequent acute symptoms were:

- fever (100%)
- headache (71%)
- arthralgia (43%)
- myalgia (43%)
- cough (43%)
- rash (maculopapular/petechial) (43%)
- nausea (29%)
- pharyngitis (29%)
- lymphadenopathy (29%)
- eschar (29%).

In these cases, the rash did not appear on the palms or soles (Unsworth et al., 2007). This presentation was unlike previously reported cases of FISF (Stewart (1991), and Unsworth et al. (2005) in Unsworth et al., 2007).

Persons at increased risk of disease

Rickettsial infections such as ASF are typically seen in residents of endemic areas, as well as campers, travellers, and hikers to endemic areas (Dehghani et al., 2019). In the seven reported cases of ASF, adults and children were affected with people aged from nine to 55 years of age. Little is known about ASF and there is no available information on activities that put people at risk of becoming infected with ASF.

The risk of contracting a tick-borne infection, including ASF, is determined by the overall number of ticks in the area, the proportion of those carrying disease, and human behaviour (World Health Organization, 2014).

All nonimmune people are susceptible to rickettsial infection, [including ASF] depending on environmental exposure. Long-lasting immunity probably follows infection. People are at risk of infection for as long as they remain in infected areas (Victorian Department of Health and Human Services, n.d.).

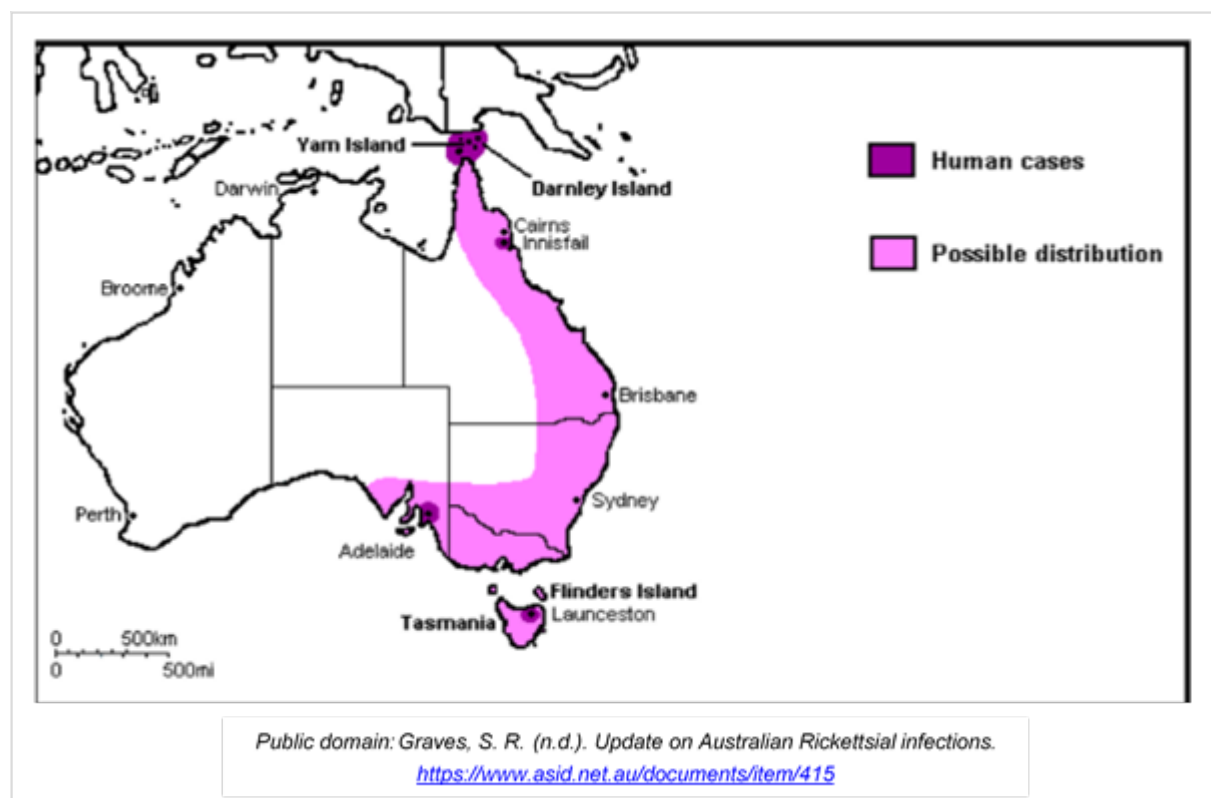
Risk areas for ASF infection in Australia

Seven cases of ASF that have been reported and described were widely distributed throughout eastern Australia, including with cases on the eastern seaboard of Australia (including the Torres Strait), Tasmania and South Australia (Unsworth et al., 2007). The specific locations of the cases described in Unsworth et al.'s (2007) paper included:

- Port Willunga, South Australia
- Darnley Island, Queensland (two patients)
- Yam Island, Queensland
- Innisfail, Queensland
- Iron Range, Queensland
- Launceston, Tasmania.

The discovery of ASF cases suggests its possible presence in Papua New Guinea (Unsworth et al., 2007). Figure 4 below is from Graves' update on Australian rickettsial diseases (Graves, n.d.) and shows the distribution of ASF.

Figure 4: Distribution of Australian spotted fever (Public domain)



Risk factors and risk activities for acquiring ASF

In Unsworth et al.'s paper on the seven cases of ASF, one patient had a history of tick bite (*H. novaeguinae*) (Unsworth et al., 2007). There was no other information on risk factors or risk activities for ASF.

Seasonal risk for ASF infection

In Unsworth et al.'s paper on the seven cases of ASF, cases were most prevalent in autumn. The reported cases of ASF occurred between February and June (late summer and autumn) (Unsworth et al., 2007). This seasonal onset was in contrast to previously described cases of FISH which have their peak onset in summer, and QTT which has its peak onset in late winter (Sexton et al. (1991), and Stewart (1991) in Unsworth et al., 2007).

Age and gender

Reported ASF cases, in Unsworth et al.'s paper, included adults and children, ranging from nine to 55 years of age (Unsworth et al., 2007).

Disease occurrence and public health significance

ASF is not a nationally notifiable disease in Australia (Australian Government Department of Health, 2021). *Rickettsia* infection (including spotted fevers and all forms of typhus fever) is notifiable in Western Australia (Western Australian Department of Health, n.d.).

The seven cases of the illness ASF were widely distributed throughout eastern Australia, including with cases on the eastern seaboard of Australia (including the Torres Strait), Tasmania and South Australia (Unsworth et al., 2007). Cases are yet to be reported in Victoria, New South Wales, the Northern Territory, or Western Australia (Unsworth et al., 2007). These seven cases, according to Unsworth et al. demonstrate that emerging rickettsioses are present in Australia (Unsworth et al., 2007).

Routine prevention activities

No vaccine is available for ASF. Currently, there is no effective vaccine against any *Rickettsia* species (Derne et al. (2015) in Stewart et al., 2017a). Difficulties exist in creating a universal SFG rickettsial vaccine due to variation in the chemical configuration of the immunologically important outer membrane proteins (Stenos & Walker (2000), and Feng & Walker (2003) in Stewart et al., 2017a).

While little is known about the risk factors for ASF, it is considered a tick-borne disease, with one patient with ASF having been bitten by *H. novaeguineae*. As such, following established advice to prevent and manage tick bites to prevent ASF is appropriate.

See Prevention and management of tick bites in Australia Guidance Note for information on personal preventive strategies to prevent tick bites on people and pets, preventing tick bites around the home and safely managing tick bites in Australia.

Also see the DSCATT Clinical Pathway (Australian Government Department of Health, 2020), for more advice.

Diagnosis

Refer to the DSCATT Clinical Pathway (Australian Government Department of Health, 2020).

Treatment

Refer to the DSCATT Clinical Pathway (Australian Government Department of Health, 2020). The Australian Immunisation Handbook (<https://immunisationhandbook.health.gov.au/>) notes that bite wounds can lead to tetanus, however, 'bite wounds' are not intended to extend to tick bites. Therefore, clinicians do not need to check the tetanus immunisation status of patients who present with a tick bite.

Q fever

In Australia, Q fever is a nationally notifiable disease (Australian Government Department of Health, 2021) with a Q fever laboratory case definition (Public Health Laboratory Network, 2017), and is included in the CDNA National Guidelines for Public Health Units (Communicable Diseases Network Australia, 2018). In Australia, Q fever is the most commonly reported zoonotic disease (Eastwood et al., 2018). As Q fever can be mistaken for other conditions, including other zoonotic diseases (e.g. leptospirosis, brucellosis), the work up should be determined by a detailed history, examination and initial screening investigation, with a useful algorithm having been developed for GPs (Gunaratnam et al. (2014) in Eastwood et al., 2018).

To ensure consistency with the current national guidance in Australia on Q fever, the majority of the information about Q fever in this Guidance Note is taken directly from the CDNA National Guidelines for Public Health Units (Communicable Diseases Network Australia, 2018). These guidelines were endorsed by the Australian Health Protection Principal Committee on 1 November 2018 and released by the Australian Government Department of Health on 27 November 2018. Some additional information and references have been added in sections relevant to the disease. This additional information does not alter the guidance in the CDNA guideline on the management of Q fever.

Infectious agents

CDNA Guideline

The infectious agent is *Coxiella burnetii*, an obligate intracellular Gram-negative coccobacillus (Communicable Diseases Network Australia, 2018). It is a highly infective and efficient pathogen, with an extremely long biological half-life (Williams et al. (1991) in Communicable Diseases Network Australia, 2018). The disease was first described as Q (for query) fever in 1937 by Edward Derrick in Queensland, with the organism subsequently identified through culture almost simultaneously by Cox in the US and Burnet (Burnet & Freeman (1937) in Communicable Diseases Network Australia, 2018) in Australia. The organism has since been found around the world (with the exception of Antarctica and possibly New Zealand) (Maurin & Raoult (1999) in Communicable Diseases Network Australia, 2018).

Additional information

C. burnetii replicates in eukaryotic cells (Eldin et al., 2017) and is a member of the gamma proteobacteria (Graves, n.d.). The establishment of its axenic⁶ culture has been a major step in the characterisation of this pathogen (Eldin et al., 2017).

C. burnetii has a low infectious dose (approximately 10 to 15 organisms for humans) (Brooke et al. (2013) in Eastwood et al., 2018). The estimated doubling time of *C. burnetii* in in vitro cell culture is between 20 and 45 hours (Angelakis & Raoult (2010) in Eldin et al., 2017).

⁶ The growth and maintenance of a single species in isolation, free from foreign or contaminating species. (<https://www.accessscience.com/content/axenic-culture/066300>).

Reservoir

CDNA Guideline

Cattle, sheep, and goats are the primary reservoirs for *C. burnetii* (Marrie (2015), and Parker et al. (2006) in Communicable Diseases Network Australia, 2018), but a wide range of domestic (Kopečný et al. (2013), and Shapiro et al. (2016) in Communicable Diseases Network Australia, 2018) and wild animals can be infected (Wildlife Health Australia (2013) in Communicable Diseases Network Australia, 2018), including camels, llamas, alpacas, rodents, cats, dogs, horses, rabbits, pigs, buffalo, foxes, some birds, bandicoots, and kangaroos. Ticks are an important vector in the transmission cycle in reservoir species (Cooper et al. (2013) in Communicable Diseases Network Australia, 2018).

Clinical signs in animals include abortion, stillbirth, retention of fetal membranes, endometritis, infertility, and pneumonia. Cattle are usually asymptomatic. Most wildlife species do not exhibit clinical signs of infection (Heymann (2015) in Communicable Diseases Network Australia, 2018). Shedding of high numbers of organisms from infected animals occurs particularly with birth products (e.g. placental tissue and birth fluids) (Heymann (2015) in Communicable Diseases Network Australia, 2018). *C. burnetii* also can be shed in the urine, faeces, and milk of infected animals. Animals may eat the placenta after giving birth, and *C. burnetii* can survive digestion and pass through an animal's intestine, leading to the organism being discharged with the faeces. With transport of manure this can lead to the organism being spread widely in the environment (Bond et al. (2015), and Hermans et al. (2014) in Communicable Diseases Network Australia, 2018).

C. burnetii not only exists in a variety of domestic and wild animal species, but also in the general environment (e.g. dust and soil) (Tozer et al. (2014) in Communicable Diseases Network Australia, 2018). It is resistant to a variety of harsh environmental conditions, including elevated temperatures, desiccation, osmotic shock, UV light, and chemical disinfectants (McCaul (1991) in Communicable Diseases Network Australia, 2018). It can survive as an infectious agent on wool at 15 to 20°C for nine months and on fresh meat in cold storage for more than a month.

Additional information

C. burnetii has two forms: a large cell variant form, which exponentially replicates; and a small-cell variant (SCV) form, which are typical of the stationary phase or non-growing 'survival' form (Eastwood et al., 2018; Eldin et al., 2017). SCV are stable in the environment and are highly resistant to osmotic, mechanical, chemical, heat and desiccation stresses. SCVs can survive:

- on fresh meat for longer than one month
- on wool at ambient temperature for seven to 10 months
- in milk for longer than 40 months (Eldin et al., 2017).

Mode of transmission

CDNA Guideline

- **Respiratory route:** the most common mode of transmission to humans is via the respiratory route following inhalation of contaminated aerosols or dust (Raoult (2005) in Communicable Diseases Network Australia, 2018), arising from for example:

- parturient, slaughtered, or necropsied animals, particularly associated with birth products (birth fluids, placental tissue, aborted/stillborn animals), and the evisceration component of butchering
 - dust residue contaminated by birth fluids, blood, faeces, or urine from infected animals, and
 - *C. burnetii* can survive in dust for months to years. Windborne spread of contaminated dust can disperse the organism over several kilometres (Tissot-Dupont et al. (2004) in Communicable Diseases Network Australia, 2018). Activities generating dust, such as herding, shearing, transport of animals, and mowing in or through areas where there are livestock or wild animals, may precipitate human infections.
- **Percutaneous route:** infection can occur through subcutaneous and intramuscular inoculation (Raoult et al. (2005), and Marmion (2009) in Communicable Diseases Network Australia, 2018), for example, following cuts with contaminated knives in the abattoir, or needle-stick injury when working with infected animals.
 - **Foodborne:** Consuming unpasteurised milk or unpasteurised milk products from infected animals has been suggested as a possible route for infection, although evidence is limited (Parker et al. (2006) in Communicable Diseases Network Australia, 2018).
 - **Vector-borne:** *C. burnetii* has been detected in numerous tick species in Australia (Cooper et al. (2013), Graves & Islam (2016) and Duron et al. (2015) in Communicable Diseases Network Australia, 2018) but human infections from ticks have been infrequently documented (Marmion (2019), Duron et al. (2015), and Graves et al. (2016) in Communicable Diseases Network Australia, 2018) possibly through tick bites or inhalation of tick excreta.
 - Person-to-person transmission is very rare but can occur through:
 - blood transfusion (Pantanowitz et al. (2002) in Communicable Diseases Network Australia, 2018) or bone marrow transplant (Kanfer et al. (1988) in Communicable Diseases Network Australia, 2018)
 - vertical or perinatal transmission (Raoult & Stein (1994) in Communicable Diseases Network Australia, 2018)
 - autopsy of infected cadavers (Maurin & Raoult (1999) in Communicable Diseases Network Australia, 2018), and
 - sexual transmission (Milazzo et al. (2001) in Communicable Diseases Network Australia, 2018).

Considering the major transmission routes of *C. burnetii* to humans, Q fever is not only thought to be a disease of occupational hazard (e.g. for farmers/abattoir workers), but also an environmental disease (Hartzell et al. (2008) in Communicable Diseases Network Australia, 2018).

C. burnetii has been listed as a Category B bioterrorism agent by the US Centers for Disease Control and Prevention (CDC) (Heymann (2015), and Khan et al. (2000) in Communicable Diseases Network Australia, 2018) due to its ease of production, survival in desiccation, and transmission through inhalation.

Additional information

Q fever is acquired via various modes of transmission, a minority of which is tick-borne.

While *C. burnetii* are present in both the Australian paralysis tick and ornate kangaroo tick, and therefore it is classified as a tick-borne disease, most cases of Q fever infection occur by inhalation of infectious aerosols from carrier (reservoir) vertebrate animals such as goats, sheep cattle, kangaroos and domestic pets or dust particles (Communicable Diseases

Network Australia, 2018; Dehghani et al., 2019; Graves & Stenos, 2017; Mackenzie, 2013) contaminated by birth fluids, faeces or urine from infected animals. The organism can remain dormant in soil and dust and be spread by vehicle movements and activities such as lawn mowing (O'Connor et al. (2015), Tissot-Dupont et al. (2004), and Flint et al. (2016) in Eastwood et al., 2018) or spread over wide areas under the influence of wind, resulting in disease outbreaks (O'Connor et al. (2015), and Tissot-Dupont et al. (2004) in Eastwood et al., 2018).

Two cases of tick-transmitted Q fever in Australia have been described in the literature (Beaman & Hung, 1989; Graves et al., 2020). In 1989, Beaman and Hung reported on a case from Western Australia, of acute Q fever with pericarditis. This case apparently occurred after multiple tick bites, most likely from the [ornate] kangaroo tick *Amblyomma triguttatum* (Beaman & Hung, 1989) which the authors noted was the predominant tick in the area (Pearce & Grove (1987) in Beaman & Hung, 1989). The 30-year-old patient had been bitten on the lower limbs and left iliac crest by five ticks while digging a well in scrubland near Toodyay (80 km northeast of Perth). At that time the authors noted that this species of tick was known to carry *C. burnetii* and transmit Q fever to kangaroos (Pope et al. (1960) in Beaman & Hung, 1989), but had not been directly implicated in transmission of Q fever to humans previously. In addition to the case being a rare complication of Q fever not previously described in Australia, they also noted that they had implicated the role of a tick vector in direct transmission of Q fever for the first time (Beaman & Hung, 1989).

More recently, in 2020, Graves et al. reported on a case of Q fever following a tick bite in a 41-year-old man who worked as a fly-in-fly-out coal miner from Mackay, Queensland who had been bitten by ticks 10 days previously (Graves et al., 2020). In this case, as the tick was damaged, it could be partially identified as *Amblyomma* spp. While full species identification (based on mouthpart morphology) was not possible, the authors reported it was most likely *A. triguttatum*, the ornate kangaroo tick. The tick was reported as strongly positive for DNA from *C. burnetii* by qPCR, based on two unique genes; com 1 and *htpAB* (Graves et al., 2020). The authors noted the one previous case of tick-transmitted Q fever reported in Western Australia, described above (Beaman & Hung (1989) in Graves et al., 2020).

A review of 2,838 Q fever notifications reported in Queensland from 2003 to 2017 reported that 49% (377 out of 774) of cases with an identified occupational group would be considered high risk for Q fever, with the most common occupational being agricultural/farming (31%) (Tozer et al., 2020). Within this review, findings were also reported for the period 2013 to 2017. Of the total cases that reported direct animal and insect contact one month prior to onset of Q fever (2013 to 2017), 16.8% (197 out of 1,170 cases) responded that they had had contact with ticks. During 2013 to 2017, the most commonly reported direct animal and insect contact in the month prior to onset of Q fever were dogs (56.5%; 661 out of 1,170 cases), Australian native wildlife (50.2%; 587 out of 1,170), cattle (48.6%; 569 out of 1,170 cases), followed by cats (28.4%; 332 out of 1,170 cases) (Tozer et al., 2020).

The CDC (Anderson et al., 2013) notes that infection may occur after direct exposure to infected animals and their products (placenta, abortion products, hides, wool, manure, etc.) particularly at the time of parturition or slaughtering (Robyn et al. (2015), Kaplan & Bertagna (1955), and Dupont et al. (1995) in Anderson et al., 2013). As *C. burnetii* may persist for prolonged periods in the soil, these aerosols may be produced long after the release of bacteria by infected animals, with evidence indicating that bacterial aerosols can be dispersed for at least 30 km by the wind (Tissot-Dupont et al. (2004) in Anderson et al., 2013), resulting in Q fever cases far away from the primary contaminated areas. Therefore,

Q fever cases are often diagnosed in persons with no recent contact with animals (Anderson et al., 2013).

Eldin et al. in their extensive 2017 review of the evidence on Q fever, noted that in tropical areas such as in Australia, examples suggest that acute Q fever incidence may be related to the rainy season (Eldin et al., 2017). A clear seasonal peak of acute Q fever cases was observed in Queensland, Australia, in May, three months after a peak February rainfall (Harris et al. (2013) in Eldin et al., 2017). Additionally, Eldin et al. noted the rainy season in Australia corresponds to an increase in the populations of macropods (wallabies and kangaroos) and other wildlife that potentially play a role in the spread of the bacterium, with rates of up to 20.8% seroprevalence for *C. burnetii* having been observed in macropods (Cooper et al. (2012) in Eldin et al., 2017).

While Graves and Stenos note that *C. burnetii* are present in both Australian paralysis ticks (Graves et al. (2016) in Graves & Stenos, 2017) and ornate kangaroo ticks (Pope et al. (1960), and Cooper et al. (2013) in Graves & Stenos, 2017), and anecdotally there are other cases of Q fever being transmitted by ticks, there is only one published case where the patient developed pericarditis, a rare presentation of Q fever, after being bitten by an ornate kangaroo tick (Beaman (1989) in Graves & Stenos, 2017). Graves and Stenos commented that while the bacterium *C. burnetii* has been isolated from the bandicoot tick (*Haemaphysalis humerosa*) from both sides of Australia (Smith & Derrick (1940), and Bennett et al. (2011) in Graves & Stenos, 2017), it is unlikely that this tick species bites humans. In north-eastern New South Wales, 6.5% of Australian paralysis ticks (*I. holocyclus*) contained the com1 gene of *C. burnetii* (Graves et al. (2016) in Graves & Stenos, 2017).

Incubation period

CDNA Guideline

Typically, the incubation period is two to three weeks, depending on the size of the infecting dose (range, four days to six weeks) (Parker et al. (2006) in Communicable Diseases Network Australia, 2018).

Infectious period

CDNA Guideline

Person-to-person spread rarely occurs (Communicable Diseases Network Australia, 2018). Immunity following recovery from clinical illness may be life-long (Heyman (2015) in Communicable Diseases Network Australia, 2018) with cell-mediated immunity lasting longer than humoral immunity. Antibodies are detectable for three to five years, but may persist for as long as 15 years (Communicable Diseases Network Australia, 2018).

Clinical presentation and outcome

CDNA Guideline

Following infection with *C. burnetii*, the majority of cases (60%) will be asymptomatic/subclinical infections (Marmion (2009) in Communicable Diseases Network Australia, 2018). Q fever may be present as an acute or chronic illness.

Acute Q fever

A person with acute Q fever can present with a variety of symptoms. The most common manifestation is an influenza-like illness which might occur in conjunction with hepatitis and/or pneumonia (Maurin (1999) in Communicable Diseases Network Australia, 2018). Commonly reported signs and symptoms include fever, chills, sweats, severe headache (especially behind the eyes), photophobia, weakness, anorexia, nausea, myalgia, cough, and weight loss (Communicable Diseases Network Australia, 2018).

Patients can present with mild hepatitis associated with *C. burnetii* infection, which is more frequently acquired in sheep and goat-breeding areas (Marrie (2015) in Communicable Diseases Network Australia, 2018).

Pneumonia is an important manifestation of acute Q fever, ranging from mild to severe. Q fever pneumonia can, however, appear similar to other aetiologies of atypical pneumonia, such as those associated with *Legionella* or *Mycoplasma*, requiring consideration of differential diagnoses. Pneumonia is less common in Australian than European cases, and upper respiratory tract involvement and tracheobronchitis seen with influenza are not typical features of Q fever (Communicable Diseases Network Australia, 2018).

A minority of infected cases ($\leq 1\%$) may develop pericarditis, myocarditis (Eldin et al. (2017) in Communicable Diseases Network Australia, 2018), or neurologic complications (e.g. meningoencephalitis, encephalomyelitis) (Maurin & Raoult (1999) in Communicable Diseases Network Australia, 2018). Infection in pregnant women (symptomatic or not) can lead to abortion, premature delivery, or low birth weight (Carcopino et al. (2007) in Communicable Diseases Network Australia, 2018). The case fatality rate for untreated acute cases is usually less than 1% (Heymann (2015) in Communicable Diseases Network Australia, 2018).

Chronic Q fever

Chronic Q fever can occur from one month to several years after acute illness, and sometimes without a history of acute illness, as a result of persistence of *C. burnetii* infection in the host after a primary infection (Communicable Diseases Network Australia, 2018).

Chronic Q fever may present as one of three major forms according to the focus of infection:

1. Endocarditis is the most serious manifestation of chronic Q fever, occurring in about 2% of acute Q fever patients (Marmion (2009) in Communicable Diseases Network Australia, 2018). The most important factors associated with progression to endocarditis following primary Q fever infection are underlying valvular heart disease/valvular prosthesis (Eldin et al. (2017), and Fenollar et al. (2001) in Communicable Diseases Network Australia, 2018). Symptoms are typically suggestive of cardiac involvement (heart failure or cardiac valve dysfunction), with histological features such as significant fibrosis and calcifications, slight inflammation and vascularisation, and small or non-visible vegetation. *C. burnetii* endocarditis is fatal if left untreated; however, for cases with treatment, the ten-year mortality rate is 19% (Heymann (2015) in Communicable Diseases Network Australia, 2018).
2. Osteoarticular infections. Bone and joint *C. burnetii* infections have been reported, occurring in less than 1% of hospitalised Q fever cases (Raoult et al. (2000) in Communicable Diseases Network Australia, 2018). Osteomyelitis appears to present more frequently in children than in adults, with evidence of granulomatous bone lesions (Eldin et al. (2017) in Communicable Diseases Network Australia, 2018). Reported joint infections involve multiple locations, including wrist, tibia, ankle, shoulder, and prosthetic joints (the knee and hip) (Eldin et al. (2017) in Communicable Diseases Network Australia, 2018).
3. Vascular infections occur in patients with pre-existing aneurysms or vascular grafts after a primary infection, and remain a severe disease with mortality rates between 18% and 26% (Eldin et al. (2017) in Communicable Diseases Network Australia, 2018). The abdominal or thoracic aorta is the most frequent site for vascular infection (Communicable Diseases Network Australia, 2018).

Other related clinical syndromes

Q fever fatigue syndrome (QFS) refers to systemic symptoms that fail to recover more than

12 months after the acute illness. Typical features of QFS include profound fatigue, arthralgia, myalgia, concentration and memory problems, sleeping problems, sweats, and headaches (Morroy et al. (2016) in Communicable Diseases Network Australia, 2018). QFS is the most common sequela following acute infection in Australia, occurring in approximately 10% to 15% of patients with acute Q fever (Marmion (2009) in Communicable Diseases Network Australia, 2018).

Additional information

Unlike rickettsial infections, Q fever is unlikely to be associated with a rash (Dehghani et al., 2019).

The two cases of tick-transmitted Q fever in Australia reported in the literature (Beaman & Hung, 1989; Graves et al., 2020) describe the clinical presentation and outcome of the respective cases. Beaman and Hung described a case of pericarditis associated with Q fever, with the authors noting pericarditis as a major manifestation of Q fever is rare (Beaman & Hung, 1989). Graves et al.'s case report published by the RACGP describes the clinical presentation of the case (described as typical even though it was transmitted by tick bite rather than aerosol transmission) and additionally has a question and answer section about Q fever (Graves et al., 2020).

A review of 2,838 Q fever notifications reported in Queensland from 2003 to 2017 reported that 40% of the total Q fever cases spent at least one day in hospital (Tozer et al., 2020). The authors noted their study findings on hospitalisation for Q fever were similar to a report from New South Wales, Australia, where 46.5% of cases from 2011 to 2015 were hospitalised; whereas it has been previously reported that only 2% of acute Q fever cases require

hospitalisation (Cutler et al. (2007) in Tozer et al., 2020). Tozer et al. commented that, without recent comparable nationwide data, it was uncertain whether the hospitalisation rates are consistent throughout Australia or are higher for Queensland and New South Wales; however, Queensland and New South Wales combined account for approximately 80% of all national Q fever notifications (Sloan-Gardner et al. (2017) in Tozer et al., 2020).

A report from Queensland Health on Q fever notifications 2016, also reported on hospitalisations among notified cases of Q fever (Queensland Health, 2016). Of the 1,041 Q fever cases notified in Queensland (2012 to 2016), 569 (54.7%) reported being hospitalised, with 494 of those hospitalised reporting the length of hospital stay; the median was five days (range, one to 46 days) (Queensland Health, 2016).

Acute Q fever

The CDC in a Morbidity and Mortality Weekly Report (MMWR) (Anderson et al., 2013) provided the following summary on acute Q fever:

Summary of Acute Q fever (Anderson et al., 2013)

- Prolonged fever (>10 days) with a normal leukocyte count, thrombocytopenia, and increased liver enzymes is suggestive of acute Q fever infection.
- Children with Q fever generally have a milder acute illness than adults.
- Children are more likely to have a rash than adults. Rash has been reported in up to 50% of children with acute Q fever.
- Women infected with Q fever during pregnancy are at increased risk for miscarriage and preterm delivery.
- Women of child-bearing age who receive a diagnosis of Q fever can benefit from pregnancy screening and counselling to guide health-care management decisions.

Additionally, Anderson et al. (2013) also provided the percentage of patients with acute Q fever with selected clinical and laboratory findings, as shown in Table 1 overleaf.

Table 1: Percentage of acute Q fever patients (Table 1 in Anderson et al., 2013)

Percentage of acute Q fever patients with selected clinical and laboratory findings	
Clinical or laboratory finding	% of patients
Clinical	
Fever	88–100
Fatigue	97–100
Chills	68–88
Headache	68–98
Myalgia	47–69
Sweats	31–98
Cough	24–90
Nausea	22–49
Vomiting	13–42
Chest pain	10–45
Diarrhea	5–22
Skin rash	5–21
Myocarditis	0.5–1
Pericarditis	1
Meningoencephalitis	1
Death	1–2
Laboratory	
Normal leukocyte count	90
Thrombocytopenia	25
Increased transaminase levels*	45–85
Increased bilirubin levels	9–14.3
Increased alkaline phosphatase levels	27.7–57
Increased γ -glutamyl transferase levels	25–75
Increased creatine phosphokinase levels	29
Increased lactate-dehydrogenase levels	33.3–40
Increased creatinine levels	29–40
Elevated erythrocyte sedimentation rate	43–87.5
Smooth muscle antibodies	65
Antiphospholipase antibodies	50

*Alanine transaminase and aspartate transaminase

Acute Q fever in children

The CDC advised in a MMWR on Q fever (Anderson et al., 2013) that children with Q fever are less likely to have symptoms than adults and might have a milder illness. In symptomatic children, the CDC noted the following findings:

- Acute Q fever is typically characterised by a febrile illness, often accompanied by headache, weakness, cough, and other non-specific systemic symptoms.
- Illness is frequently self-limited, although a relapsing febrile illness has been documented in some children (Richardus et al. (1985) in Anderson et al., 2013).
- Gastrointestinal symptoms such as diarrhoea, vomiting, abdominal pain, and anorexia are reported in 50% to 80% of paediatric cases (Richardus et al. (1985), Maltezou et al. (2004), and Terheggen et al. (2007) in Anderson et al., 2013).
- Skin rash is more common in children than adults, with a prevalence as high as 50% among children with diagnosed cases (Richardus et al. (1985), Maltezou et al. (2014), Terheggen et al. (2007), and Ruiz-Contreras et al. (1993) in Anderson et al., 2013).
- Q fever pneumonia is usually moderate with mild cough; respiratory distress and chest pain may occur (Anderson et al., 2013).
- Severe manifestations of acute disease are rare in children and include hepatitis, haemolytic uremic syndrome, myocarditis, pericarditis, encephalitis, meningitis, haemophagocytosis, lymphadenitis, acalculous cholecystitis, and rhabdomyolysis (Maltezou et al. (2001), Maltezou et al. (2002), Carrascosa et al. (1997), Rolain et al. (2003), and Foucault et al. (2004) in Anderson et al., 2013).

Eldin et al., in their extensive 2017 review on Q fever, noted that there are few specific studies on the clinical manifestations of Q fever in children (Eldin et al., 2017). They also noted that, as in adults, the clinical presentation of the primary infection is not specific and can mimic other childhood infections, with testing for Q fever rarely performed by paediatricians (Hackert et al. (2015) in Eldin et al., 2017).

Acute Q fever in pregnant women

The CDC (Anderson et al., 2013) notes the following findings regarding Q fever in pregnant women:

- Pregnant women might be less likely to have symptoms of Q fever compared with other adults (for example, a febrile illness), although they remain at risk of adverse pregnancy outcomes (Tissot-Dupont et al. (2007) in Anderson et al., 2013).
- Q fever infections in women that occur shortly before conception or during pregnancy might result in miscarriage, stillbirth, premature birth, intrauterine growth retardation, or low birthweight (Carcopino et al. (2007) in Anderson et al., 2013).
- Adverse pregnancy outcomes are likely to be caused by vasculitis, or vascular thrombosis resulting in placental insufficiency, although direct infection of the foetus has been documented (Stein et al. (1998) in Anderson et al., 2013).
- The risk for adverse effects on the foetus and the risk that the mother will develop chronic Q fever are highest when an acute infection occurs during the first trimester (Raoult et al. (2002), and Carcopino et al. (2009) in Anderson et al., 2013).
- Untreated infection in the first trimester is more likely to result in miscarriage, whereas infection later in pregnancy is more likely to cause premature delivery (Carcopino et al. (2007) in Anderson et al., 2013).
- Women infected with acute Q fever during pregnancy, including those who were asymptomatic or experienced no adverse pregnancy outcomes, might be at risk of recrudescent infection during subsequent pregnancies (Syruczek et al. (1958) in Anderson et al., 2013). As such, pregnant women with a history of Q fever infection during a

previous pregnancy should be monitored closely for recrudescence infection in all subsequent pregnancies (Anderson et al., 2013).

- None of the reports that have described outcomes in infected pregnant women have documented an increased risk for congenital malformations because of infection (Carcopino et al. (2007), and Stein et al. (1998) in Anderson et al., 2013).

More recently, Eldin et al. (2017), in their extensive review on Q fever, noted obstetrical complications have been reported mainly in cases from France, Spain, Canada, and Australia (Million et al. (2014), Langley et al. (2003), Shinar et al. (2012), Quijada et al. (2012), Jover-Diaz et al. (2001), Coste Mazeau et al. (2016), and Denman & Woods (2009) in Eldin et al., 2017) However, Eldin et al. noted that recent large-scale population-based serological studies from Germany (Boden et al. (2012) in Eldin et al., 2017), Denmark (Nielson et al. (2012), and Nielson et al. (2013) in Eldin et al., 2017), and the Netherlands (Munstyler et al. (2013), and van der Hoek et al. (2011) in Eldin et al., 2017) have shown no increased risk of adverse obstetrical outcomes in seropositive pregnant women (Giakoumelou et al. (2016) in Eldin et al., 2017). Eldin et al. (2017) noted the discrepancy can be explained by several factors, including study design.

Of the evidence on obstetrical complications of Q fever, Eldin et al. stated: “A recent meta-analysis from our team of 136 cases and seven population-based studies confirmed some key points: Seropositivity and untreated Q fever during pregnancy are associated with fetal death, and antibiotic treatment prevents this complication” (Million et al. (2014) in Eldin et al., 2017).

Chronic Q fever

The CDC in a MMWR (Anderson et al., 2013) provided the following summary on chronic Q fever:

Summary of chronic Q fever (Anderson et al., 2013):

- Persons who are at high risk for development of chronic Q fever include persons with pre-existing valvular heart disease, vascular grafts, or arterial aneurysms.
- Infection during pregnancy and immunosuppression (e.g., from chemotherapy) are both conditions that have been linked to chronic Q fever development.
- Endocarditis and infections of aneurysms or vascular prostheses are the most common forms of chronic Q fever and generally are fatal if untreated.
- Chronic Q fever is rarely reported in children.
- In contrast with adults, osteomyelitis is one of the most common findings in children with paediatric chronic Q fever.

Chronic Q fever in children

The CDC (Anderson et al., 2013) advised the following about chronic Q fever in children:

- Chronic Q fever is rarely reported in children.
- Like adults, children who are immunocompromised or have underlying heart valve disease might be at higher risk for chronic Q fever.
- Paediatric chronic Q fever manifests most frequently as chronic relapsing or multifocal osteomyelitis, blood-culture–negative endocarditis, or chronic hepatitis (McQuiston, (2009) in Anderson et al., 2013).
- Children with Q fever osteomyelitis often experience a prolonged course with recurrent episodes affecting multiple bones before diagnosis (Maltezou & Raoult (2002), and Nourse et al. (2004) in Anderson et al., 2013).

Chronic Q fever in pregnant women

The CDC (Anderson et al., 2013) advised the following about chronic Q fever in pregnant women:

- Women infected with Q fever during pregnancy are at high risk for developing chronic Q fever, possibly because of a failure to mount an appropriate immune response to acute infection or the ability of *C. burnetii* to use placental trophoblasts as a replicative niche (Carcopino et al. (2009), and Amara et al. (2010) in Anderson et al., 2013).
- The earlier a woman is infected during pregnancy, the greater her risk for development of chronic disease (Foucault et al. (2004) in Anderson et al., 2013).
- Chronic infection might be evidenced by increased phase I IgG *C. burnetii* titers that do not decrease after pregnancy and can lead to adverse outcomes during subsequent pregnancies (Raoult et al. (2002) in Anderson et al., 2013).

Q fever endocarditis

An Australian paper on the prevention of Q fever endocarditis concluded that due to its indolent progression and poor outcome when diagnosis is delayed, a thorough cardiac assessment of all patients with suspected or confirmed Q fever is important (Hess et al., 2011). Furthermore, Hess et al. advised that GPs, especially in rural and regional areas, are encouraged to conduct cardiac assessments for all patients with acute Q fever to identify patients at risk of developing Q fever endocarditis (Hess et al., 2011). Hess et al. noted the following evidence regarding Q fever endocarditis:

- Acute Q fever progresses to chronic Q fever in 5% to 30% of patients (Marmion, (2009), Islam et al. (2001), and Fournier et al. (1998) in Hess et al., 2011).
- While endocarditis commonly develops within three to six months of the acute attack (Landais et al. (2007) in Hess et al., 2011), it may only become apparent after five to 20 years (Marmion (2009), and Ayres et al. (1996) in Hess et al., 2011).
- Q fever endocarditis occurs almost exclusively in patients with pre-existing cardiac valve defects, prosthetic heart valves or an impaired immune system (Maurin & Raoult (1999), Parker et al. (2006), and Fenollar et al. (2001) in Hess et al., 2011).
- Pre-existing valve defects most commonly involve insufficiencies of the mitral and/or aortic valve; however, patients with a prosthesis seem at a greater risk of developing endocarditis (Fenollar et al. (2001) in Hess et al., 2011).
- In some cases of Q fever endocarditis, pre-existing valvulopathy has been minor, such as bicuspid aortic valve, mitral valve prolapse and trivial mitral valve insufficiency (Fenollar et al. (2006) in Hess et al., 2011).

- The risk of endocarditis in patients with valvular disease has been estimated at 39%, of which almost 75% are male with a mean age of 60 years (Landais et al. (2007), Fenollar et al. (2001), and Houpiikian et al. (2002) in Hess et al., 2011).
- If Q fever endocarditis is untreated, most patients will die, but even with appropriate therapy the mortality rate remains at 10% (Maurin & Raoult (1999), and Parker et al. (2006) in Hess et al., 2011).

Persons at increased risk of disease

CDNA Guideline

- At-risk occupational groups (including contractors within the industries) are those with contact of high-risk animals or animal products (Marmion (2009) in Communicable Diseases Network Australia, 2018) including:
 - abattoir and meat workers (e.g., workers involved in slaughtering/skinning/meat processing/rendering, by-products workers, meat inspectors/packers, administration and maintenance workers)
 - agriculture, livestock and dairy farmers/workers
 - stockyard/feedlot workers and transporters of animals, animal products and waste
 - shearers, wool classers/sorters, pelt and hide processors
 - knackery workers
 - tannery workers
 - laundry workers handling clothing from at-risk workplaces
 - pet food manufacturing workers
 - veterinarians, veterinary nurses/students/researchers, and others who work with veterinary specimens
 - agriculture college staff and students (working with high-risk animals)
 - animal shooters/hunters
 - laboratory personnel who work with materials containing viable *C. burnetii* (e.g., birth products of infected animals/humans, tissue culture)
 - wildlife/zoo workers and animal trainers (working with high-risk animals), and
 - dog/cat breeders, and anyone regularly exposed to parturient animals.
- Other people at risk of Q fever through non-occupational, environmental exposures include:
 - family members of the at-risk occupational groups described above, through exposures to contaminated clothes, boots or equipment
 - people living on or in close proximity to a high risk industry (e.g. neighbouring livestock farms, stockyards housing cattle, sheep or goats (Anderson et al. (2013), and Karki et al. (2015) in Communicable Diseases Network Australia, 2018), meatworks (Palmer et al. (2007) in Communicable Diseases Network Australia, 2018), land being fertilised by untreated animal manure)
 - visitors to at-risk environments (e.g., farms, abattoirs, animal saleyards, agricultural shows)
 - people living or working near livestock transport routes with the potential to be exposed to contaminated dust from the passing animals, and
 - people involved in mowing, which aerosolises dust potentially contaminated by animal excreta, in areas where there are livestock or wild animals (e.g., kangaroos).

- Persons at increased risk for chronic Q fever after experiencing an acute infection include (Eldin et al. (2017), and Fenollar et al. (2001) in Communicable Diseases Network Australia, 2018):
 - immunosuppressed persons
 - pregnant women
 - persons with valvular heart disease/valvular prosthesis
 - persons with aneurysms/vascular grafts.

Additional information

An ongoing prospective cohort study of adults found acute Q fever was highest for adults living on a farm in outer regional or remote areas of New South Wales (Karki et al. (2015) in Eastwood et al., 2018).

A review of 2,838 Q fever notifications reported in Queensland 2003 to 2017 identified, through improved surveillance data, that the most frequent at-risk exposures for Q fever notifications in Queensland (2013 to 2017) were environmental exposures; specifically exposure to dust from animal paddocks, living within 300 metres of bush/scrub/forest, and living or working within one kilometre of an abattoir (Tozer et al., 2020).

An analysis of age and gender distribution of Q fever in Australia from 2012 to 2016 by Sloan-Gardner et al. showed males were over-represented in the combined age group 40 to 69 years (Sloan-Gardner et al. (2017) in Eastwood et al., 2018).

Disease occurrence and public health significance

CDNA Guideline

Q fever is a zoonotic disease that occurs around the world (Communicable Diseases Network Australia, 2018).

The true incidence of disease is greater than that reported because of subclinical infection, as well as limited clinical suspicion and testing (Communicable Diseases Network Australia, 2018).

In Australia, there were around 500–800 notifications (2.5 to 5.0 per 100,000 population) annually in the 1990s (National Notifiable Diseases Surveillance System (2016) in Communicable Diseases Network Australia, 2018).

During 2001 to 2006, an Australian Government funded National Q Fever Management Program was implemented in Australia, which provided subsidised vaccination to at-risk groups, initially to abattoir workers, contractors working in abattoirs, and sheep shearers; and subsequently to sheep, dairy, and beef cattle farmers, and their employees and family members working on farms (Gidding et al. (2009) in Communicable Diseases Network Australia, 2018). The program was concluded in late 2006. This program led to a substantial decrease in national Q fever notifications over the period and beyond, from 792 cases (4.0 per 100,000 population) in 2002 to a nadir of 314 cases (1.4 per 100,000 population) in 2009. However, since cessation of the program there has been a gradual increase in annual Q fever notifications after 2010, reaching 551 cases (2.3 per 100,000 population) in 2016 (National Notifiable Diseases Surveillance System (2016) in Communicable Diseases Network Australia, 2018).

Q fever notification rates are relatively high in Australia compared with European countries (0.18 per 100,000 population in 2014) (EFSA (European Food Safety Authority) & ECDC (European Centre for Disease Prevention and Control) (2015) in Communicable Diseases Network Australia, 2018) and the US (0.04 per 100,000 population per year) (CDC (2016) in Communicable Diseases Network Australia, 2018).

The majority of Australian Q fever notifications have been reported from Queensland and New South Wales, which accounted for 48% and 39% of total national notifications, respectively, during 2011 to 2015 (National Notifiable Diseases Surveillance System (2016) in Communicable Diseases Network Australia, 2018). The notification rate remains highest in southwest/central west Queensland and northwest New South Wales (Gidding et al. (2009) in Communicable Diseases Network Australia, 2018), generally reflecting the intensity of local cattle, sheep, and goat husbandry, and associated processing industries.

Q fever outbreaks have been reported occasionally in Australia, generally related to occupational and/or environmental exposures. The largest reported Q fever outbreak in the world occurred in the Netherlands from 2007 to 2010, involving over 4,000 cases (including 28 deaths reported) (Desling et al. (2010), and Kampschreur et al. (2014) in Communicable Diseases Network Australia, 2018). The outbreak was linked to dairy goat farms situated in and around densely populated areas. In the context of this large outbreak, the Q fever notification rate peaked at 9.8 per 100,000 population per year in the Netherlands in 2009 (EFSA (European Food Safety Authority) & ECDC (European Centre for Disease Prevention and Control) (2011) in Communicable Diseases Network Australia, 2018).

Additional information

Q fever infections have been reported in all countries except for New Zealand and Antarctica (Eastwood et al., 2018; Tozer et al., 2011), and it is the most commonly reported zoonotic disease in Australia (Eastwood et al., 2018; Hess et al., 2011). While cases occur throughout Australia, rates are higher in northern New South Wales and southern Queensland (Eastwood et al., 2018). Q fever is a significant zoonotic disease in some parts of rural Australia (Hess et al., 2011).

In Australia, Q fever is a nationally notifiable disease (Australian Government Department of Health, 2021).

Notification rates are 6.3 per 100,000 population per annum in Queensland, 3.1 per 100,000 per annum in New South Wales, 1.1 per 100,000 per annum in South Australia, and less than 1.0 per 100,000 in the other states and territories; however, these figures are likely to underestimate the true burden of the disease as many cases are undiagnosed (Sloan-Gardner et al. (2017) in Eastwood et al., 2018). An analysis of age and gender distribution of Q fever in Australia from 2012 to 2016 by Sloan-Gardner et al. showed males were over-represented in the combined age group 40 to 69 years (Sloan-Gardner et al. (2017) in Eastwood et al., 2018).

Several seroprevalence studies have been conducted in Australia, with these providing an indication of the prevalence of *C. burnetii* infection in a population (Eastwood et al., 2018). In a 2011 report on the seroprevalence to *C. burnetii* in a convenience sample from the Hunter New England region of New South Wales, the overall seropositivity rate was 7%, with higher rates in rural areas compared with urban areas (Islam et al. (2011) in Eastwood et al., 2018; Islam et al., 2011). The largest proportion of seropositives (37%) was in the >60 years age group, with lower prevalence in the zero to nine years (1%) and 10 to 19 years (5%) age groups. The report indicated residents in the rural areas of the Hunter New England region had a considerable exposure to *C. burnetii*, consistent with the high notification rate for Q fever in this part of Australia (Islam et al., 2011).

Another study of specimens from a serum bank of samples from a generalised Queensland, Australia population found 109 specimens of the 2122 tested were positive, with 5.3% of samples from the rural/remote population positive (Tozer et al. (2011) in Eastwood et al., 2018; Tozer et al., 2011). In this study, and in contrast to the findings of Islam et al. (2011), the rate in participants from metropolitan Brisbane, where the area is considered considerably less risky, or not at risk, was similar at 5.0%. The study found seropositivity of children was 1.3% and increased with age, with the authors commenting that the study showed both metropolitan and paediatric populations which are considered low risk of *Coxiella* exposure have “surprisingly” high seropositivity (Tozer et al., 2011). While Eastwood et al. (2018) noted these two studies have inherent limitations, biases and confounders, the authors commented they do indicate that Q fever is likely to be more prevalent than the notification rate suggests (Eastwood et al., 2018).

Additionally, regarding seropositivity to Q fever particularly among children, Eldin et al. cited a study in Queensland, Australia, that had found a seropositivity rate of 2.5% in children younger than 15 years (Parker et al. (2010) in Eldin et al., 2017).

A review of 2,838 Q fever notifications reported in Queensland 2003 to 2017 reported that, for this period, Queensland accounted for 43% of the Australian national Q fever notifications (Tozer et al., 2020). Queensland and New South Wales account for approximately 80% of all national Q fever notifications (Sloan-Gardener et al. (2017) in Tozer et al., 2020).

Routine prevention activities

CDNA Guideline

Vaccination (Australian Government Department of Health (2013) in Communicable Diseases Network Australia, 2018)

Q fever vaccine (Q-VAX®) has been available in Australia since 1989, with efficacy estimated at 83 to 100% (Marmion (2009) in Communicable Diseases Network Australia, 2018). The vaccine is recommended for those at risk of infection with *C. burnetii*.

Immunisation of those in high-risk occupational groups is the most effective preventive measure against Q fever. This includes everyone whose work exposes them to cattle, sheep, goats, kangaroos, camels, and other high-risk animals and animal products (including products of conception). See section on 'Persons at increased risk of disease' for details of the at-risk occupations. In addition, people who are at risk of Q fever through non-occupational, environmental exposures (see section on 'Persons at increased risk of disease') are also recommended for vaccination.

Work health and safety legislation places duties on employers to ensure the health and safety of their workers, so far as is reasonably practicable. Ideally, vaccination should occur at least 15 days before the person starts working in an at-risk environment. People who visit high risk workplaces (even occasionally), such as tradespeople, labour hire workers, or occupational health staff, should also be vaccinated.

Pre-vaccination testing is imperative as a hypersensitivity reaction to the vaccine can result from previous (possibly unrecognised) exposure to the organism. A stringent pre-vaccination protocol must be followed, which includes skin testing for cellular immunity, serological testing for humoral immunity, and a detailed history looking for previous laboratory-confirmed Q fever disease and previous vaccination. Persons who have worked for some time in the livestock or meat industries or another high-risk occupational group should be questioned particularly carefully. Pre-vaccination screening tests require expertise in both administration and interpretation. See the online version of the Australian Immunisation Handbook for current, detailed recommendations for pre-vaccination screening and vaccination (Australian Government Department of Health (2013) in Communicable Diseases Network Australia, 2018).

The lower acceptable age limit for Q fever vaccination is not known; however, it is not currently recommended for use in anyone aged less than 15 years. Q fever vaccination is not recommended during pregnancy. In general, Q fever skin testing, and vaccination should be avoided in individuals with impaired immunity – if exposure is unavoidable or highly likely, expert advice on vaccination should be sought for these cohorts.

The Australian Q fever register is owned and funded by the Australian Meat Processor Corporation (AMPC). It was established in 2001 to store information about Q fever vaccination status of people who have agreed to provide information (www.qfever.org). This website has a link to lists of Q fever vaccine service providers and a searchable database of the immune status of individuals who choose to submit their details

Reducing exposures

As well as vaccination as a preventive measure, individuals, companies and employers, and government agencies can take steps to reduce the risk of exposure to Q fever through workplace design, safe work practices and town planning.

Workplace design (Workplace Health and Safety Queensland (2015), and SafeWork NSW (2016) in Communicable Diseases Network Australia, 2018)

Engineering and design controls can be used in Q fever high risk areas (e.g., kill floors, offal rooms, slink rooms, yards, and pens) to minimise exposure, for example:

- installation of appropriate ventilation and dust suppression systems to reduce aerosols and dust from spreading
- structures, surfaces, machinery, and equipment should be designed to be easily cleaned, and
- yard facilities for sheep, goats, and cattle should be situated well away from residential domestic living areas.

Safe workplace practices (Workplace Health and Safety Queensland (2015), and SafeWork NSW (2016) in Communicable Diseases Network Australia, 2018)

Require all workers, contractors, labour hire workers, and visitors to show proof of immunity to Q fever.

Persons without evidence of immunity should preferably be refused entry to the workplace or higher risk areas. However, respiratory protective equipment (RPE) can be used as an interim or short-term control measure to protect non-immune workers, contractors, and visitors. The minimum level of RPE is a properly fitted disposable P2 respirator.

Handle animal products, waste, placentas, and aborted fetuses appropriately using personal protective equipment (PPE), and dispose of birth products by deep burial. Wash animal body fluids from the work site and equipment. Where possible prevent animals from eating placental tissue and avoid using animal placental tissue in compost.

PPE and contaminated clothing/coveralls should be removed at the workplace, and appropriately bagged and washed on site, to reduce the risk of exposing non-vaccinated individuals and family members outside of the workplace. Equipment should not be removed from the workplace.

Maintain infection prevention and control principles – hands and arms should be washed thoroughly in soapy water after handling animals, animal products, and potentially contaminated materials.

Minimising dust and aerosols in slaughter and animal housing areas.

Town planning

Town planning should consider the potential for windborne spread of Q fever and limit the encroachment of residential dwellings on existing likely sources of Q fever, including abattoirs, tanneries, stockyards, and land that has historically been used for these purposes. Dust contaminated by the organisms can be carried downwind for several kilometres (Parker et al. (2006) in Communicable Diseases Network Australia, 2018). In the Q fever outbreak settings in the Netherlands, the population risk of infection was substantially higher within 5 km of infected dairy goat farms (Schimmer et al. (2010) in Communicable Diseases Network Australia, 2018).

Additional information

Eastwood et al. noted issues affecting vaccine uptake for Q fever have been reviewed for farmers, veterinarians and veterinary assistants (Lower et al. (2017) in Eastwood et al., 2018). Cost, inconvenience of the two-step vaccination process to the patient and GP, access to vaccination and safety discourage uptake. Lack of disease awareness and a misunderstanding of the breadth of risk factors associated with Q fever are the main issues for patients and clinicians in Australia (Eastwood et al., 2018).

More recently, Graves et al. commented that in the case of tick-transmitted Q fever in Australia they described in 2020, Q fever vaccination would likely have prevented this

infection (Graves et al., 2020). Regarding the administration of the vaccination for Q fever, the authors noted the following:

- Prior to vaccination with the Q fever vaccine, individuals must undergo pre-vaccination evaluation with both serum antibody testing and skin testing to ensure they have not been previously exposed to the organism.
- Administration of the Q fever vaccine after a person has been exposed to Q fever can lead to significant adverse reactions to the vaccine given the development of hypersensitivity to the organism.
- Similarly, individuals with a previous history of Q fever infection should not be vaccinated (Graves et al., 2020).

Regarding recommendations for Q fever vaccination, Graves et al. “recommended” the Q fever vaccine for persons living or working in rural and regional Australia, particularly in New South Wales and Queensland where Q fever is a problem and who have exposure to high-risk animals (Australian Technical Advisory Group on Immunisation (ATAGI) (2018) in Graves et al., 2020). They also noted that the Australian Immunisation Handbook recommends Q fever vaccination for people aged ≥ 15 years who have close contact with animals, including abattoir workers, farmers, veterinarians, professional cat and dog breeders, zoo and wildlife workers and animal refuge workers (Australian Technical Advisory Group on Immunisation (ATAGI) (2018) in Graves et al., 2020). Citing the recent article by Armstrong et al. on ‘Q fever vaccination in children in Australia: Limited experience to date’, Graves et al. noted that for at-risk children, the Q fever vaccine is also recommended, with caution (Armstrong et al. (2019) in Graves et al., 2020).

While tick-transmitted Q fever is rare in Australia, it is considered a tick-borne disease. As such, the following established advice to prevent and manage tick bites to prevent Q fever is appropriate.

See Prevention and management of tick bites in Australia Guidance Note for information on personal preventive strategies to prevent tick bites on people and pets, preventing tick bites around the home and safely managing tick bites in Australia.

Also see the DSCATT Clinical Pathway (Australian Government Department of Health, 2020), for more advice.

Diagnosis

Refer to the DSCATT Clinical Pathway (Australian Government Department of Health, 2020) and 2018 CDNA Q Fever National Guidelines for Public Health Units (Communicable Diseases Network Australia, 2018).

Treatment

Refer to the DSCATT Clinical Pathway (Australian Government Department of Health, 2020) and 2018 CDNA Q Fever National Guidelines for Public Health Units (Communicable Diseases Network Australia, 2018). The Australian Immunisation Handbook (<https://immunisationhandbook.health.gov.au/>) notes that bite wounds can lead to tetanus, however, ‘bite wounds’ are not intended to extend to tick bites. Therefore, clinicians do not need to check the tetanus immunisation status of patients who present with a tick bite.

Debilitating Symptom Complexes Attributed to Ticks (DSCATT)

DSCATT is the term used by the Australian Government to describe the group of Australian patients suffering from the symptoms of a chronic debilitating illness, which many associate with a tick bite (Australian Government Department of Health, 2018) to appropriately acknowledge this patient group and the multifaceted illness they are experiencing, and acknowledge their illness is poorly understood. The Australian Government acknowledges that many of these patients experiencing debilitating symptom complexes are living in turmoil because their illness cannot be easily diagnosed and treated. With the causes of DSCATT remaining unknown, the Australian Government urges patients and health professionals to keep an open mind about the cause of a patient's symptoms.

DSCATT was also proposed as a name to move away from the stigma and controversy associated with the terms previously used to describe this patient group such as "Lyme disease-like illness" and "Chronic Lyme Disease" (Australian Government Department of Health, 2018).

In November 2020, the DSCATT Clinical Pathway was published on the Australian Government Department of Health's website (Australian Government Department of Health, 2020). The evidence-based clinical pathway and multidisciplinary care model for patients presenting with symptoms associated with DSCATT was developed to support decision-making on differential diagnosis and referral pathways for patients presenting with either new onset or unresolved debilitating symptoms with or without a history of tick bites and that cannot be attributed to another condition (acute or chronic) (Australian Government Department of Health, 2020).

Infectious agents

The cause of DSCATT is not known.

The Australian Government has acknowledged the need for, and is supporting, research to further investigate DSCATT. In January 2019, the National Health and Medical Research Council funded research to better understand the nature, prevalence and causes of these symptoms, with the longer-term aim to obtain evidence to guide development of treatments:

- Professor Peter Irwin of Murdoch University received funding for research to determine the causes of DSCATT, as a first step towards improving diagnostic outcomes for patients through the provision of accurate and evidence-based information about their illness.
- Professor Richard Kanaan of the University of Melbourne received funding for a project to develop a new treatment for DSCATT, that will include the development of a case definition, adapting the treatment approach for unexplained syndromes to the specifics of DSCATT, and then piloting a randomised controlled trial to test the effectiveness of the new therapy (Australian Government Department of Health, 2019).

Additionally, the Australian Government Department of Health funded the Commonwealth Scientific and Industrial Research Organisation to progress two projects, both of which were completed in 2021 (Australian Government Department of Health and Aged Care, 2022).

These projects included:

- a tick survey, to better understand which bacteria, viruses and other pathogens are carried by ticks in Australia and their impact on human health

- a case study biobank, to gather and analyse samples from DSCATT patients for possible biomarkers as possible indicators of disease.

Results of these projects are available on the Australian Government Department of Health and Aged Care website at this link: <https://www.health.gov.au/initiatives-and-programs/dscatt>.

Reservoir

The aetiology and pathophysiology of DSCATT is unknown.

Mode of transmission

The aetiology and pathophysiology of DSCATT is unknown.

Incubation period

The aetiology and pathophysiology of DSCATT is unknown.

Infectious period

The aetiology and pathophysiology of DSCATT is unknown.

Clinical presentation and outcome

There are no peer-reviewed published epidemiological or clinical studies about patients experiencing DSCATT. The only relevant information available is self-reported and anecdotal. Patients have told of the symptoms they have experienced and attribute to DSCATT to the Senate Community Affairs References Committee (Senate Community Affairs References Committee, 2016a, 2016b), the DSCATT Patient Forum (TMS Consulting Pty Ltd, 2018), and the DSCATT Think Tank (Allen + Clarke, 2019).

The most common symptoms described by patients with DSCATT to the Senate Inquiry were: fatigue (62.6%); disordered thinking (51.9%); sensory disturbance (46.1%); arthralgia (45.6%); headache (44.5%); followed by myalgia; rash; mood disturbance; visual disturbance; dizziness; pain; fever; nausea; palpitations; insomnia; seizures; diarrhoea; tremor; and personality change (Brown, 2018). Patients reported having experienced symptoms for a median of 10 years and had seen a median of 13 doctors for diagnosis and treatment of their illness. An analysis of the submissions to the Senate Committee noted the unquestionably real and debilitating physical and social harm from illness reported in the submissions. Of relevance to the attribution of symptoms to ticks, over half of the submissions analysed did not comment on tick bite but of those that did, a majority (89.5%) reported a positive history. The author's conclusion suggested that patients who identified as having DSCATT displayed a symptomology similar to 'medically unexplained physical symptoms' syndromes, and also experience social and financial harms and are at risk of nosocomial harms. They may also have sought alternative and potentially non-evidence-based diagnoses and treatments (Brown, 2018).

Similarly, multiple symptoms and signs being attributed to DSCATT were identified by stakeholders who attended the DSCATT Think Tank in May 2019, with neurological symptoms (including brain fog, cognitive dysfunction, memory loss, fine motor impairment and reduced verbal fluency) and chronic fatigue being the most commonly identified symptoms and signs (Allen + Clarke, 2019).

Persons at increased risk

While the term DSCATT implies the symptom complex of DSCATT is attributed to ticks, the cause is not currently known and persons at increased risk of experiencing DSCATT has not been established.

Diagnosis

DSCATT is not a diagnosable disease, and a patient cannot be given a diagnosis of DSCATT. It is not clearly defined and has no diagnostic criteria. Associated symptoms may be the end point for several different disease processes. The symptom complexes to which the name DSCATT has been given incorporates a wide range of nonspecific symptoms.

Refer to the DSCATT Clinical Pathway (Australian Government Department of Health, 2020).

Treatment

DSCATT has no diagnostic criteria, known cause or causes, and no treatment.

Refer to the DSCATT Clinical Pathway (Australian Government Department of Health, 2020).

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